

# Management and prevention of toxicoses in search-and-rescue dogs responding to urban disasters

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It is not possible to anticipate all of the potential toxicologic hazards that search-and-rescue (SAR) dogs may encounter while searching urban disaster sites. To decrease the likelihood of a dangerous exposure for SAR dogs, it is important to have knowledge of the most common toxicologic hazards and risks<sup>1</sup> and of the specific toxicologic agents.<sup>2</sup> Despite this, problems may occur, and provisions must be made for prompt and appropriate treatment of SAR dogs exposed to toxic substances at urban disaster sites. The purpose of the present article is to describe diagnostic testing for and basic treatment of SAR dogs exposed to specific toxicants and to provide recommendations to minimize potential risks.

## Patient Assessment

Each patient should be individually assessed, and any life-threatening situation (eg, dyspnea or bleeding) should be addressed before specific treatment for a suspected toxicosis is initiated. Clinicians should not wait for confirmation of a toxicosis before starting treatment, and lack of a confirmed diagnosis should not interfere with the responsibility to “treat the patient, not the poison.” General principles of emergency medicine should be followed, and vital signs should be stabilized. Hydration should be assessed along with metabolic status. Once any urgent problems have been corrected, decontamination and antidotal treatment may be instituted as required.

## Hydrocarbons

**Diagnostic testing**—Specific methods to determine exposure to hydrocarbons, their breakdown products, or their metabolites exist but may not be universally available. Often, exposure to kerosene or gasoline can be determined by its odor on an animal's breath or hair. Lipid granulomas in the liver and spleen are thought to be a result of dietary exposure to mineral oils and waxes in humans, so their detection may be useful only as an index of past exposure to petroleum hydrocarbons.<sup>3</sup>

**Treatment**—Induction of emesis and gastric lavage are not recommended after hydrocarbon ingestion because of the risk of aspiration.<sup>4</sup> Further, it is unclear whether activated charcoal would be of any great benefit in dogs that have ingested hydrocarbons,<sup>4</sup> and administration of activated charcoal may contribute to a potential aspiration risk. Dermal decontamination generally consists of washing with a general

liquid dish detergent and clipping hair that will not wash clean.

Radiography is indicated in all dogs with clinical signs of respiratory tract involvement and in dogs in which the severity of gastrointestinal tract or neurologic signs might be sufficient to predispose the dog to aspiration. Radiologic evidence of lung infiltrations is suggestive of oral or inhalation exposure to petroleum products.<sup>3</sup> However, radiographs obtained shortly after exposure may not have evidence of aspiration pneumonia or chemical pneumonitis. Therefore, radiography should be repeated to detect the development of these abnormalities.<sup>5</sup> Dogs that are coughing have probably already aspirated and should be closely monitored. Antibiotics, oxygen, and assisted ventilation (positive end-expiratory pressure) should be administered as appropriate.<sup>3</sup> Any additional treatment should be directed at addressing specific clinical signs and providing supportive care.

## Polychlorinated Biphenyls

**Diagnostic testing**—Gas-liquid chromatography electron-capture detection has been used to detect polychlorinated biphenyls (PCBs) in canine serum, and serum PCB concentration measured with this technique has been shown to be significantly correlated with residence time in the study area for dogs exposed to PCBs.<sup>6</sup> However, this test is rarely available for use following an acute exposure.

**Treatment**—Following exposure, washing multiple times with a liquid dish detergent and water may reduce dermal absorption of PCBs.<sup>7</sup> Hydrocarbon-based solvents should never be used to clean PCB-contaminated skin, as they may not only cause their own adverse effects, but also increase the dermal absorption of fat-soluble PCBs. Induction of emesis is considered contraindicated following suspected oral exposure to PCBs because of the risk of aspiration. The value of administering activated charcoal following oral PCB exposure is unknown, but its administration with a saline cathartic or sorbitol is frequently recommended in human medicine<sup>8</sup>; administration of multiple doses may help prevent reabsorption of metabolites. Rice bran fiber has decreased intestinal absorption and stimulated fecal excretion of PCBs in rats,<sup>9</sup> so the addition of fiber to an SAR dog's diet may possibly offer a protective effect.

## Hazardous Metals

**Diagnostic testing**—Diagnosis of acute metal toxicosis on the basis of clinical signs alone may be difficult, as the most common manifestations of acute

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metal toxicosis tend to be nonspecific gastrointestinal and respiratory tract abnormalities. Because these signs can also occur for other reasons (eg, stress and innocuous dust inhalation), the early signs of acute metal toxicosis may be misinterpreted. For this reason, it is helpful to have an idea of the types of metals that may be encountered at an urban disaster site. Diagnosis of exposure to hazardous metals may entail monitoring blood and urine concentrations. Ideally, baseline blood concentrations of metals of concern would be obtained prior to SAR dogs being exposed to the disaster site, allowing more accurate interpretation of subsequent test results. Blood concentrations of metals such as arsenic, lead, and cadmium may be transiently elevated following acute exposure and rapidly decrease as the metal redistributes through the body.<sup>10</sup> For this reason, monitoring of concentrations during the search period and the first few weeks after leaving the search site would be recommended.

**Treatment**—Treatment of hazardous metal exposure in SAR dogs includes stabilization, decontamination, administration of supportive care, and, depending on the metal involved, chelation (**Appendix 1**). Dogs with signs of acute toxicosis should be managed symptomatically (eg, anticonvulsants for seizures, antiemetics or protectants for gastrointestinal tract signs, and oxygen administration for dyspnea). Many metals are eliminated by the kidneys and some are nephrotoxic; therefore, particular attention should be paid to ensuring that hydration is adequate. Decontamination may entail removal from the site of exposure, bathing, and administration of cathartics and enemas. Activated charcoal is considered to be a poor adsorbent of many metals, although there are some exceptions (eg, organic mercury and thallium).<sup>11,12</sup> Specific treatment should be administered if the metal to which the dog has been exposed is known (**Appendix 2**).

Chelation treatment should be undertaken with careful regard to the specific metal involved and should never be instituted at the expense of essential management of clinical signs. Chelation is contraindicated in dogs following intoxication with metals for which the chelate itself might induce substantial injury (eg, organic mercury compounds and thallium).<sup>11,12</sup> Several chelators (eg, dimercaprol and calcium EDTA) are nephrotoxic and must not be used if adequate hydration cannot be assured during the chelation period. Additionally, many chelators must not be administered until all metal of concern is out of the gastrointestinal tract, as these chelators can increase absorption of metals from the gut. Recommended dosages of commonly used chelators are available.

## Gases

The gases of greatest concern for SAR dogs at urban disaster sites include hydrogen cyanide, hydrogen sulfide, carbon monoxide, Freon, and halogenated gases (chlorine, bromine, and fluorine).

**Diagnostic testing**—Blood and urine cyanide concentrations can be measured to determine exposure to hydrogen cyanide. However, because time is of the utmost importance when treating cyanide poisoning,

treatment is often instituted before results of such testing are available.<sup>13,14</sup> Urinary thiosulfate and blood sulfide anion concentrations are considered to be useful for forensic purposes in cases of suspected hydrogen sulfide poisoning,<sup>15</sup> and carbon monoxide poisoning can be confirmed by measuring carboxyhemoglobin concentrations. Retinal hemorrhages are common in people with carbon monoxide poisoning. Unfortunately, there are no laboratory tests considered to be valuable for diagnostic use in a clinical setting to confirm hydrogen sulfide, Freon, or halogenated gas exposure or toxicosis. However, if exposure to a specific Freon is suspected, it can be detected in the blood. It is recommended that veterinarians consult with their local laboratory to determine whether specific analyses can be performed.

**Treatment**—In general, management of hydrogen cyanide, hydrogen sulfide, carbon monoxide, Freon, and halogenated gas poisoning involves supportive care. Because time can be critical, the affected animal should be moved to fresh air, and cardiovascular and respiratory support should be given as soon as possible.<sup>14,16</sup> A thorough examination should be performed, and bronchodilation therapy should be given as needed. Although no controlled clinical study has been done, corticosteroids have been used to help with respiratory tract signs caused by exposure to halogenated gases.<sup>17</sup>

If cyanide toxicosis is diagnosed, treatment must be instituted as quickly as possible. Sodium nitrite (22 mg/kg [10 mg/lb], IV) is given to form methemoglobin. Cyanide preferentially binds to the ferric iron in methemoglobin to form cyanomethemoglobin. Thus, cyanide dissociates from the iron in cytochrome oxidase, allowing oxidative phosphorylation to resume. A second drug, sodium thiosulfate (660 mg/kg [300 mg/lb], IV), is given to convert cyanide to thiocyanate with the help of the rhodanese enzyme found in skeletal muscle and liver. Thiocyanate is excreted in the urine.<sup>16,18</sup> A thorough history and physical examination are recommended to properly diagnose cyanide toxicosis and prevent unnecessary adverse effects that can accompany the use of these 2 medications. A third drug, amyl nitrite, has been given by inhalation for treatment of cyanide poisoning in humans. However, amyl nitrite use is considered controversial, and it is a poor methemoglobin producer.<sup>16</sup>

Because the mechanism of hydrogen sulfide toxicosis is similar to that with cyanide toxicosis, induction of methemoglobinemia may be beneficial.<sup>19,20</sup> In fact, methemoglobinemia has been shown to have protective and antidotal effects in laboratory animals poisoned with hydrogen sulfide.<sup>21</sup> Hyperbaric oxygen has also been shown to be beneficial in humans poisoned with hydrogen sulfide.<sup>22</sup>

## Soaps and Detergents

**Treatment**—Oral dilution with milk or water is recommended following ingestion of soaps and detergents. Emesis and gastric lavage are not recommended because of the corrosive effects of cationic detergents. Activated charcoal is ineffective for caustic agents.

## Acids and Alkalis

**Treatment**—Acid and alkali exposures are treated in the same manner. With oral ingestion, dilution with milk or water is most effective if it is performed early. Gastric lavage and emesis are not recommended because of possible corrosive effects, and activated charcoal is ineffective for caustic agents. The dog should be monitored for signs of oral, esophageal, and gastrointestinal irritation or ulceration. Following dermal exposure, bathing immediately with a mild liquid hand or dish detergent or a noninsecticidal pet shampoo is indicated, and the dog should be monitored for erythema, swelling, pain, and pruritis. Treatment is symptomatic and may include analgesics, anti-inflammatory drugs, gastrointestinal tract protectants, and antimicrobials.

## Ethylene Glycol

**Diagnostic testing**—If exposure to ethylene glycol is suspected, the diagnosis can be confirmed or supported with various blood tests. A commercially available test kit<sup>8</sup> can be used as a screening tool. Negative test results are fairly reliable indicators that dogs have not been exposed; however, false-positive results can occur because of formaldehyde, metaldehyde, glycerin or glycerol (eg, cough syrup and liqueurs), propylene glycol found in certain injectable drugs (eg, diazepam, barbiturates) or activated charcoal slurries, diethylene glycol, and other *cis* 1,2-diols.<sup>23</sup> There is no interference from ethanol, fomepizole, methanol, or polyethylene glycol.<sup>23</sup> Serum ethylene glycol concentrations can be measured at most human hospitals. A negative test result with the commercial test kit is equivalent to a concentration < 50 mg/dL.<sup>23</sup>

Measurement of serum osmolality or anion gap may also be used for diagnostic purposes. Serum osmolality significantly increases by 1 hour after exposure to ethylene glycol, whereas the anion gap typically begins to increase by 3 hours but may not increase until 6 hours after exposure. Blood urea nitrogen and serum creatinine concentrations usually are not increased until 12 hours after exposure and cannot be depended on to assist with an early diagnosis.<sup>24</sup>

Most commercial ethylene glycol-containing antifreeze products are green, blue, or pink. Sodium fluorescein dye is added to most commercial ethylene glycol products to allow automotive service technicians to visualize radiator leaks with a black light. Therefore, a Wood's lamp may be used to detect fluorescence in urine or gastric contents, on the paws, or around the muzzle in dogs suspected of having been exposed to ethylene glycol. In humans, sodium fluorescein dye is excreted in the urine within 3 to 6 hours.<sup>25</sup>

**Treatment**—The goal of treatment in dogs with ethylene glycol intoxication is to stop conversion of ethylene glycol into its more toxic metabolites. Emesis should be induced if oral exposure has occurred < 15 minutes previously; gastric lavage can also be considered. Blood samples should be collected for baseline serum biochemical testing, a CBC, and ethylene glycol testing before activated charcoal or any medication is

given. Activated charcoal may be given, but it is not likely to be effective.<sup>26</sup> High infusion rates of crystalloid fluids are required to correct severe dehydration and hypoperfusion. Sodium bicarbonate increases renal excretion of the glycolate metabolite of ethylene glycol; pyridoxine and thiamine are also proposed to hasten elimination of metabolites.<sup>27</sup>

Two specific antagonists for ethylene glycol have been identified. Ethanol acts as a competitive substrate for alcohol dehydrogenase, whereas fomepizole inhibits alcohol dehydrogenase. Peritoneal dialysis is also effective in removing ethylene glycol and its metabolites from the body.<sup>25,28</sup> Detailed treatment for ethylene glycol intoxication is widely available.<sup>23,29</sup>

## Propylene Glycol

**Diagnostic testing**—Analysis of propylene glycol concentrations in blood, urine, stomach contents, and tissue samples (liver, kidney) may help determine exposure. Exposure to propylene glycol will also result in positive test result for the commercial ethylene glycol test kit.<sup>23</sup>

**Treatment**—Treatment consists of supportive care. If exposure was recent (< 15 minutes), emesis should be induced. However, emesis should not be induced in dogs with clinical signs of intoxication because of the risk of aspiration. Activated charcoal may be given. Intravenous administration of fluids is important for replacing fluid lost as a result of osmotic diuresis. Acid-base status and body temperature should be monitored. In dogs, if the dose ingested is equal to or more than the LD<sub>50</sub> (22 g/kg [10 g/lb]),<sup>30</sup> fomepizole may block conversion of propylene glycol to lactate via alcohol dehydrogenase. However, the usefulness of fomepizole in treating severe propylene glycol toxicosis needs to be determined. Most animals recover within 24 to 72 hours with supportive care.

## Phenol

**Diagnostic testing**—A history of exposure to phenol along with its disinfectant odor is usually all that is needed for a diagnosis of phenol toxicosis; however, multiple analytic methods may be used to detect phenol in the body. The measurement of phenol requires special laboratory equipment and techniques that are not routinely available in most human hospitals or veterinary clinics. Because of phenol's short half-life (30 to 60 minutes in humans<sup>31</sup>), testing can detect only exposures that have occurred within 1 to 2 days. Urine is the most commonly tested substance, but phenol concentrations can also be determined in blood, stomach contents, and tissue samples. Urine from a healthy dog can yield false-positive results, because phenol is a common metabolite of dietary proteins.<sup>32</sup> Testing can be used to determine recent exposure to phenol or to a substance that is converted to phenol in the body (eg, benzene).

The presence of phenols may be nonspecifically detected through the use of 10% ferric chloride reagent or can be specifically determined by high-performance liquid chromatography,<sup>33</sup> nuclear magnetic resonance (NMR) urinalysis,<sup>34</sup> or gas chromatography-mass spectrometry.<sup>35</sup>

**Treatment**—Dogs should be treated for phenol exposure in a well-ventilated area, and all attending personnel should wear gloves, gowns, and masks. With dermal exposure, areas of contaminated skin should be blotted with paper towels before washing. Liquid dish detergent and copious amounts of water should be used until phenol can no longer be smelled on the animal. Using smaller amounts of water only dilutes the phenol and expands the area of exposure, resulting in greater risk of complication.<sup>36</sup> Emesis is contraindicated following oral exposure because of phenol's corrosive effects and the potential for rapid CNS depression and seizures. Small amounts that are ingested may be diluted with egg whites or milk; gastric lavage and administration of activated charcoal are recommended. Management of systemic toxic effects is nonspecific and supportive.

## Alcohols

**Diagnostic testing**—For dogs suspected of toxic exposure to alcohol, blood alcohol concentration can be measured at a human hospital. Exposure to methanol or ethanol will result in a negative result for the commercial ethylene glycol test kit.<sup>23</sup>

**Treatment**—Treatment of a dog with alcohol toxicosis consists of nonspecific supportive care. Fluids and electrolytes should be given as needed. Sodium bicarbonate should be added to the fluids if metabolic acidosis is present. Hypoglycemia is commonly seen, and dextrose administration may be needed.

## Prevention

Dermal absorption is one of the most important routes of exposure to toxicants among SAR dogs responding to urban disaster. Some dogs have worn booties to reduce the risk of dermal exposure, but the booties decrease traction, and some dogs will not tolerate them. Bathing and rinsing dogs off on a regular basis will help to eliminate particulates and dermal absorption of toxicants. Ocular exposure is also a concern. Goggles designed for dogs are available,<sup>b</sup> but dogs must be acclimated to them before wearing them for an SAR operation. Regular flushing of eyes with saline solution will help to remove ocular irritants.

Search-and-rescue dogs also have a much higher potential for oral exposure to toxicants than do their handlers, as they commonly lick their noses while working. Having handlers routinely wipe their dog's nose and mouth will help reduce ingestion of toxicants. Also, keeping dogs well hydrated will decrease their chance of drinking from standing water that contains poisonous substances.<sup>37</sup>

Handlers should meet with the disaster site Safety and Health Officer before starting each shift. The Safety and Health Officer will have information about airborne toxicants and may have information on particular hazards present at specific site locations. Armed with this information, handlers can monitor their dogs for specific signs associated with significant exposures. Many times the Safety and Health Officer will have on-site equipment to monitor for hydrocarbons, carbon monoxide, hydrogen cyanide, and methane.

<sup>a</sup>Ethylene glycol test kit, Allelic Biosystems, Pensacola, Fla.

<sup>b</sup>Doggles, MidKnight Creations LLC, Los Gatos, Calif.

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## Appendix 1

Dosages of commonly used chelators for treatment of heavy metal toxicosis in dogs

Chelator	Dosage
Calcium EDTA	100 mg/kg (45 mg/lb), SC, q 24 h for 2 to 5 days; the calculated dose should be divided into 4 portions, and each portion should be diluted with 5% dextrose solution to a concentration of 10 mg of calcium EDTA/mL; each portion should then be administered at a different site; do not exceed 2 g/d and do not treat for more than 5 consecutive days <sup>38</sup>
Dimercaprol	2.5 to 5 mg/kg (1.1 to 2.3 mg/lb), IM, q 4 h for 2 days, then q 12 h <sup>38</sup>
Succimer	10 mg/kg (4.5 mg/lb), PO, q 8 h for 10 days; administer on an empty stomach; may be administered per rectum <sup>39</sup>
d-Penicillamine	7.5 to 27.5 mg/kg (3.4 to 12.5 mg/lb), PO, q 6 h for 7 days; treatment may be repeated after 7 days if needed <sup>38</sup>

Continued on next page.

## Appendix 2

### Treatment of hazardous metal toxicoses in dogs

Metal	Initial treatment	Decontamination	Chelation
Antimony	Fluid therapy, gastrointestinal tract protectants, blood replacement therapy, oxygen, ventilatory support, other supportive care	Remove from source, bathe, induce emesis (antimony often causes emesis), lavage	Not generally required because of poor oral and inhalation absorption <sup>11</sup>
Arsenic	Fluid therapy, gastrointestinal tract protectants, blood replacement therapy, oxygen, ventilatory support, antiarrhythmic therapy, other supportive care	Remove from source, bathe, induce emesis or perform gastric lavage	Succimer, d-penicillamine, or dimercaprol may be used; gastrointestinal tract must be cleared of metal prior to chelation; adequate urine output must be maintained during chelation <sup>40</sup>
Beryllium	Fluid therapy, gastrointestinal tract protectants, oxygen, ventilatory support, other supportive care	Remove from source, bathe, induce emesis or perform gastric lavage	Not indicated <sup>41</sup>
Cadmium	Fluid therapy, gastrointestinal tract protectants, oxygen, ventilatory support, other supportive care	Remove from source, bathe, induce emesis (controversial because of potential for oral or esophageal ulceration) or perform gastric lavage	Of questionable benefit with acute exposures; contraindicated with chronic toxicosis because of nephrotoxicity <sup>11</sup> ; succimer, deferoxamine, and calcium EDTA have been used experimentally <sup>42</sup>
Chromium (trivalent and hexavalent salts)	Fluid therapy, gastrointestinal tract protectants, oxygen, ventilatory support, blood replacement therapy, correction of acid-base imbalances, other supportive care	Remove from source, dilute with milk or water, bathe; induction of emesis is contraindicated because of corrosive effects	Dimercaprol and calcium EDTA have been used experimentally but have not been shown to be of definite benefit <sup>43</sup>
Cobalt	Manage cardiac insufficiency	Remove from source, dilute with milk or water, bathe; induction of emesis is contraindicated because of corrosive effects	Dimercaprol and calcium EDTA have been suggested, but efficacy is dubious <sup>44</sup>
Lead	Seizure control, fluid therapy	Remove source, induce emesis or perform gastric lavage, administer enema or cathartic	Dimercaprol, calcium EDTA, d-penicillamine, or succimer may be used <sup>45</sup> ; gastrointestinal tract must be cleared of metal prior to chelation (except with succimer); adequate urine output must be maintained during chelation
Mercury	Seizure control, fluid therapy, gastrointestinal tract protectants, oxygen, blood replacement therapy, other supportive care	Remove source, induce emesis or perform gastric lavage (contraindicated with forms that have potential for corrosive injury), dilute with egg white, administer activated charcoal, enemas, or a cathartic	Chelation is contraindicated following exposure to organic mercury compounds <sup>12</sup> ; dimercaprol may be used following acute ingestion of caustic inorganic mercury; d-penicillamine and succimer may also be used <sup>12</sup> ; gastrointestinal tract must be cleared of metal prior to chelation; adequate urine output must be maintained during chelation
Nickel	Oxygen, fluid therapy	Remove from source; induction of emesis not considered necessary	Generally not required; use of dimercaprol increases toxic effects of nickel carbonyl <sup>11,46</sup> , diethyldithiocarbamate has been used experimentally in animals exposed to nickel carbonyl <sup>46</sup>
Thallium	Fluid therapy (forced diuresis enhances urinary excretion of thallium), blood replacement therapy, gastrointestinal tract protectants	Early and aggressive decontamination is required; induce emesis or perform gastric lavage, administer activated charcoal. Administer ferric ferrocyanide (Prussian blue) to aid adsorption of thallium in gastrointestinal tract (minimal benefit once signs have developed) <sup>11</sup>	Chelation generally not recommended as chelated thallium may more readily enter CNS and exacerbate neurologic signs <sup>11</sup>
Zinc	Blood replacement therapy, fluid support, gastrointestinal tract protectants	Removal of zinc object from gastrointestinal tract	Rarely necessary as blood zinc concentration generally decreases rapidly following removal of zinc objects from gastrointestinal tract and chelated zinc may exacerbate renal injury; chelation is indicated when signs are progressing despite removal of zinc from gastrointestinal tract <sup>11</sup> ; calcium EDTA or d-penicillamine may be used <sup>11</sup> ; gastrointestinal tract must be cleared of metal prior to chelation; adequate urine output must be maintained during chelation; monitor blood zinc concentration during chelation