Canines in a CBRNE Environment

Veterinary Treatment Protocols for Chemical, Biological, Radiological/Nuclear Agents of Concern and Explosive Events

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SECTION TWO

Contents

I. GENERAL APPROACH to the EMERGENT K9 PATIENT

A. ABC vs CAB or MARCHE

Triage priorities in a disaster are to stabilize life threatening conditions and transport to a referral facility ASAP. The severity of a canine's condition is determined by the primary survey, which addresses the most life threatening problems first.

Classic **ABC** uses: **A**irway, **B**reathing, **C**ardiac and **C**irculatory collapse.

Close on the heels of these emergent life systems checks are the neurologic system, followed by urogenital and musculoskeletal assessments. All conditions below necessitate transport to an emergency veterinary facility for continued intensive care and monitoring.

Canines embedded with military special operations units experience trauma from blast and gunfire. Medic and veterinary support advocate the CAB or MARCHE system, along with pain management an antibiotics, to be the most effective in those situations:

Muzzle/**M**assive hemorrhage
Airway
Respiration
Circulation/**C**ardiac
Hypothermia
Evacuation

Control bleeding
Airway
Breathing

B. HISTORY

1. Current Concern
2. History, Information

C. COMPLETE PHYSICAL EXAMINATION

1. Temperature: 100.0-102.5^{o} F, 38-39^{o} C
2. Attitude: Bright, Alert, Responsive
3. Hydration: Moist MM, Skin Elasticity
4. Mucous Membranes: Pink, Capillary Refill 1-2 Seconds
5. Eyes: Clear, Pupils Equal and Responsive
6. Ears: Clean, Dry, No Malodor, No Discharge
7. Nose: No Wounds, No Discharge, Symmetrical
8. Mouth: No bleeding, Nonpainful, Normal Closure, Symmetrical
9. Throat: No obstruction
10. Peripheral Lymph Nodes: No Prominence
11. Heart, Pulse: 70-140 Beats per Minute, Strong, Synchronous
12. Lungs, Respiration: Clear, Eupnic, 15-30 Breaths per Minute
13. Abdomen: Non-Painful, Concave in Shape (Not Bloated)
14. Urogenital: Normal Micturition, Clear Light Yellow Urine, No Wounds
15. Integument: No Wounds, Lumps/Bumps, Moist Clumps, Pain, Redness/Bruising
16. Musculoskeletal: Normal Gait and Posture, No Lameness
17. Neurologic: Head, Spine, and Peripheral Nerve Function

D. Diagnostics - Unknown and Known Exposure

1. Exposure Unknown; Exposure but Contaminant Unknown

a. ABCs and Supportive care
   i. Decontamination of visible substance (save for diagnostics)
   ii. Treat the abnormalities you observe directly and on physical exam
   iii. Monitor response to your treatments

b. Diagnostics
   i. Bloods
   ii. Urine
   iii. Expiratory gases
   iv. Radiation Detectors
   v. **Toxidromes** - these especially important for agents with specific antidotes as treating symptomatically will only take one so far. One may miss the opportunity time-wise to effectively treat and save the patient.

2. Exposure with Contaminant Identified

a. All of the above
b. Consult options: read it, speak it, web it (Poison Control Centers, Toxicology and Hazmat Literature, Internet, Apps)
c. Antidotes and continued care as applicable

II. DECONTAMINATION

A. External Decontamination

1. Principles

a. Protect yourself and others
b. Remove or decrease the exposure
c. Part of the treatment protocol
d. Copious irrigation with water or saline
e. Wash-rinse cycles with soap and water

2. Considerations

a. Eyes - saline or purified water irrigation
b. Ears, Nose, Mouth - baby wipes
c. Body - Soap and water wash-rinse cycles; baby wipes on a pile
d. Paw Pads - the most contaminated portion
   i. Most exposed area to contaminants and hazards
   ii. Can absorb agents systemically due to presence of sweat glands
   iii. Haired toe webs and rough-surfaced edges difficult to decontaminate
   iv. Ground-level position makes decon a pain in the back - literally

3. Water Concerns

a. Commercial system (Anivac)
   i. Cleansing and suction action simultaneously
   ii. Minimal water required and contaminated water is self-contained
   iii. Requires electrical power
   iv. Cost may be limiting factor

B. Internal Decontamination

1. Respiratory
- a. Supplemental oxygen; humidify for long term administration
- b. Observe for airway obstruction - physical, inflammatory, upper/lower airway
- c. Intubate, tracheotomy/tracheostomy as needed

2. Gastrointestinal
- a. Take Heed
  - i. Patient must be fully awake or intubated to avoid complications
  - ii. Aspiration most common complication
- b. Emesis
  - i. Effective if ingestion within last 60 minutes
  - ii. Contraindicated for acids, alkalis, petroleum distillates, or unknown toxin
  - iii. ***Hydrogen peroxide 3%*** (*non-expired)* @ 1-5 ml/kg PO with max 30 ml; repeat 1/2 or whole dose if no emesis within 15-20 minutes; follow dose with some water to enhance effect
  - iv. ***Apomorphine*** @ 1.5-6 mg or half a 6mg tab for small dogs, whole tab for large dogs - placed into conjunctival sac (rinse tablet from eye afterwards); @ 0.04-0.08 mg/kg IM, @ 0.03 mg/kg IV
    NOTE: reverse with naloxone if patient too sedate @ 0.02-0.04 mg/kg IV
  - v. ***NO LONGER RECOMMENDED:***
    Syrup of Ipecac - severe hemorrhagic gastritis
    Salt - severe hypernatremia; exacerbated by activated charcoal
- c. Binding Agents
  - i. Bind with toxins to limit their adsorption
  - ii. ***Activated Charcoal*** @ 1-5 g/kg (6-12 ml/kg) PO every 2-4-6 hours as needed but only first dose with sorbitol (cathartic) ; for multiple dose use product w/out sorbitol or may cause severe hypernatremia (other: ***UAA gel***)
  - iii. ***Aluminum Hydroxide*** - phosphate binder @ 10-30 mg/kg (0.5-1.5 ml/kg) PO every 8 hours (Vit D Rodenticide poisoning)
- d. Gastric Lavage
  - i. Effective only if within 1-2 hours of ingestion
  - ii. ***Advantages***: rapid removal from stomach, dilutes corrosives, can administer activated charcoal
  - iii. ***Disadvantages***: requires general anesthesia, risk of esophageal/gastric trauma, aspiration, will not remove large chunks of ingesta or toxin

3. **Enhanced Elimination** - unusual in the veterinary world, requiring intensive care facility with proper equipment and trained staff
- a. Intravenous fluids: renal vasodilation, perfusion, toxin elimination, and rehydration
- b. Urinary
  - i. Requires a functioning renal system and monitoring
  - ii. Diuresis: ***Furosemide*** @ 2-5 mg/kg IV
  - iii. Diuresis: ***Mannitol*** @ 0.25-0.5 g/kg IV over 30 minutes
  - iv. Urine acidification with ***ammonium chloride*** @ 100-200 mg/kg PO every 12 hours
  - v. Urine alkalinization with ***sodium bicarbonate*** @ 1-2 mEq/kg IV every 3-4 hours
- c. Chelation Therapy
  - i. Combines with toxin to form insoluble salt that is poorly absorbed
  - ii. BAL, calcium salts, desferoxamine, succimer, EDTA, D-penicillamine

III. TOXIDROMES

A. **Definition and Purpose** Greek roots: *toxicon* = bow; arrows often had poisoned tips
dromos = race course

A toxidrome is the syndrome caused by a specific toxin or group of toxins. Toxidromes group agents together according to the signs and symptoms they generally produce. This allows one to narrow the suspected etiology to a small group of agents and effect more specific treatments

B. Toxidrome Recipe

1. Vital Signs + Mental Status + Autonomic Signs
2. Confirmatory Diagnostics - labs and EKG
3. Other = colors and odors

C. Toxidrome Categories

1. Asphyxiants - "Blood Agents"
   a. Interfere with oxygen transport and/or utilization
   b. Displace oxygen from ambient atmosphere, decreasing oxygen available to lungs

2. Anticholinergics - "Incapacitating Agents"
   a. Competitively antagonize the neurotransmitter acetylcholine (ACh) at postganglionic parasympathetic (cholinergic) nerve fibers
   b. Also targets smooth muscles influenced by acetylcholine but lack innervation
   c. Affected: glandular secretions, intestinal motility, bronchi, cardiac rate/function
   d. "Hot as a hare, red as a beet, dry as a bone, blind as a bat, mad as a hatter"
   e. "Can't see, can't spit, can't pee, can't sh*t"
   f. The anti-sludge: lack of salivation, lacrimation, urination, defecation, GI stress/diarrhea, emesis

3. Cholinergics - "Nerve Agents"
   a. Inhibit acetylcholinesterase, the enzyme that neutralizes the neurotransmitter acetylcholine at nerve-muscle or nerve-gland junction
   b. Affects the Peripheral Nervous System where ACh acts on 2 different receptors:
      1) Nicotinic - neuromuscular junction of skeletal muscles, post-ganglionic neurons of parasympathetic NS, some brain neurons
      2) Muscarinic - neuromuscular junction of cardiac & smooth muscle, glands, post-ganglionic neurons of sympathetic NS
   c. "SLUDGE" and "DUMB BELS"
   d. Affects the Central Nervous System as well

4. Irritant Gases - "Choking/Pulmonary Agents"
   a. Mechanism via direct irritation to the respiratory tract
   b. Affected lung parenchyma: alveolar sacs fill with fluid (non-cardiogenic pulmonary edema) "dry land drowning"

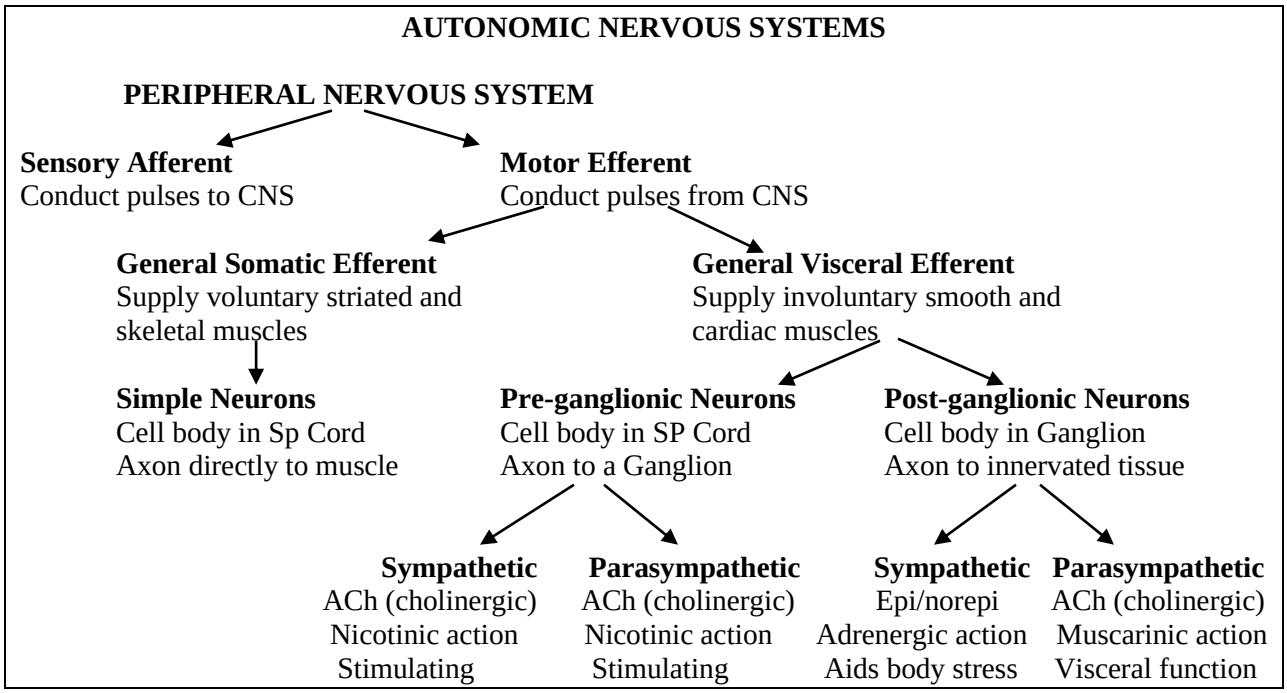
5. Vesicants - "Blister Agents"
   a. Mechanism via direct contact with liquid or high vapor concentrations
   b. Eyes, respiratory tract, and warm moist thin skin most affected
   c. Canine skin does not blister due to difference in dermal blood supply

6. **Corrosives**
    a. Acids - corrosive burns along digestive tract if ingested, on skin if dermal exposure
    b. Alkalis - deep tissue necrosis along GI Tract if ingested, on skin if dermal exposure

7. **Alcohols**
    a. Syndrome of intoxication (NS effects) and metabolic acidosis
    b. Alcohols range in their toxicity to canines
    c. More toxic forms lead to irreversible renal failure (anti-freeze products)

8. **Hydrocarbons - Petroleum Distillates**
    a. Inhalation: narcosis/stupor/coma, cardiac irritability/death, chemical pneumonitis
    b. Ingestion: nausea, vomiting, diarrhea, pain
    c. Defatting dermatitis

9. **Sympathomimetics**
    a. Increase release of catecholamines stimulates sympathomimetic receptor sites (epinephrine)
    b. Caffeine-related: tachypnea, tachycardia, hyperexcitability, tremor/seizure



Muscarinic Actions: cardiac inhibition, vasodilation, salivation, lacrimation, bronchoconstriction, GI stimulation

Cholinergic (Nerve Agents) inhibit acetylcholinesterase
muscarinic effects are prolonged

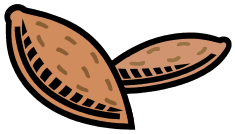
Anticholinergic (Incapacitating Agents) antagonize acetylcholine at the receptors
muscarinic actions cannot be transmitted

IV. CBRNE THREAT CHEMICALS of CONCERN

A. Asphyxiants "Blood Agents"

1. Hydrogen Cyanide (AC)

- Colorless flammable gas or liquid with characteristic odor of ***bitter almonds***
- Miscible with water and ***lighter than air***, tending to rise rapidly once released
- Also known as carbon hydride nitride, cyclone B, cyclone, evercyn, formic anammonide, formonitrile



2. Cyanogen Chloride (CK)

- Colorless nonflammable gas at normal temperatures with pungent and biting odor
- Soluble in water and ***heavier than air***
- Also known as chlorine cyanide, chlorocyanide, chlorocyanogen, cyanochloride

Characteristics

These are inhalation threats. Once inhaled, they will cause immediate effects. Respiratory protection, unavailable to working search canines, is the best protection.

Mechanism of Action

Blood agents interfere with the cellular enzyme (cytochrome oxidase) that transfers oxygen from the blood to the cells, by irreversibly binding to ferric ion on that enzyme. Blood can become oxygenated, but cells are blocked from using it. Oxygen starvation is at the cellular level. A lethal dose can cause respiratory distress leading to loss of consciousness and death within 3-5 minutes.

Clinical Signs

- Cherry red eyes, lips, mucous membranes
- Hypoxia-induced mentation changes (anxiety, confusion, weakness, dizziness)
- Irregular respiration (tachypnea, dyspnea, hyperpnea)
- Nausea, frothing, vomiting
- Irregular heartbeat
- Convulsions
- Cyanosis (blue mm)
- Unconsciousness
- Death

Treatments

1. Move animal from contaminated area immediately
2. If known cyanide-containing substance ingested within the last 15 minutes, induce vomiting (hydrogen peroxide, apomorphine)
3. If known cyanide-containing substance ingested within the last 15-60 minutes and no signs are present, perform gastric lavage
4. Activated charcoal administration may help decrease ingested absorption
5. Flush dermal wounds with copious amounts soap/water; treat irritation based on severity of wound (clip hair, ab oint/silver sulfadiazine, bandaging wet-to-dry or non-adherent)
6. Antibiotics (cefazolin/Keflex, ciprofloxacin/Baytril)
7. Analgesics (narcotics, NSAIDs)
8. Consider anti-emetics if vomiting (chlorpromazine, ondansetron (Zofran), dolasetron (Anzemet), maropitant citrate (Cerenia)

Emergency Treatments

1. Clear airway of fluid, provide oxygen
2. Secure airway PRN, ventilate if necessary, 80-100% oxygen at least 30 minutes
3. EKG monitoring
4. IV access for cardiovascular and respiratory support
5. Thorough examination, blood collection and monitor electrolytes, acid-base
6. Control seizures (diazepam, midazolam, pentobarbital)

Antidotes

1. ***Sodium thiosulfate*** @ 400-500 mg/kg IV (comes as 20% or 200 mg/ml and 25% or 250 mg/ml solutions)
2. **ONLY IF CERTAIN ABOUT CYANIDE DIAGNOSIS**: ***sodium nitrite*** @ 16-20 mg/kg IV (nitrite-induced potentially fatal methemoglobinemia if cyanide not present); comes as 3% or 30 mg/kg
3. ***Amyl nitrate*** via inhalation followed by sodium nitrite has been recommended, but use in dogs is not well documented and evidence of scientific benefit is lacking

♫ NOTE: The first three drugs above are available in a ***Cyanide Antidote Kit*** (other names: Taylor Kit, Lilly Kit, Pasadena Kit)

4. ***Hydroxocobalamin*** (vitamin B12a) has shown much promise; it forms cyanocobalamin with the cyanide and is excreted; human dose: 25 mg/hour constant rate infusion (100 mg in 100 ml 5% dextrose in water)
5. **ONLY IF CERTAIN ABOUT CYANIDE DIAGNOSIS:** ***Dicobalt edetate*** @ one 300 mg ampule IV over 1 minute followed by 50 ml glucose IV infusion; it is used in the united Kingdom, as yet not available in the US; it forms a nontoxic stable ion complex with cyanide; toxic to patient if cyanide not present!

B. Anticholinergics "Incapacitating Agents"

1. 3-quinuclidinyl benzilate

- ☠ Persistent crystalline solid
- ☠ Produces a non-lethal syndrome similar to atropine or scopolamine

Mechanism of Action

This is an anticholinergic, competitively antagonizing acetylcholine at sites innervated by postganglionic, parasympathetic (cholinergic) nerve fibers and on smooth muscles that are influenced by acetylcholine but lack innervation. Therefore it affects glandular secretions, intestinal motility, bronchi, and cardiac rate and function.

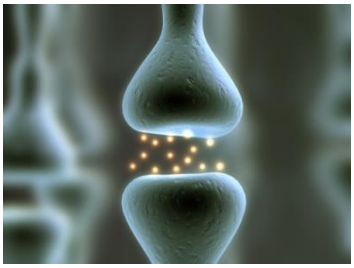
Clinical Signs - peak ~ 8 hours post exposure, slowly subside over 2-3 days

- Tachycardia
- Dry skin
- Dry MM
- Hyperthermia
- Mydriasis
- Blurred vision
- Odd behavior
- Hallucinations
- Stupor

Treatments - supportive care based on signs, symptoms, toxidrome/other diagnostics

Antidotes

1. ***Physostigmine*** @ 0.06 mg/kg IM or IV over 5 minutes
2. ***Pyridostigmine*** @ 0.1 mg/kg (IV, IM?) or 0.5-3.0 mg/kg PO q8-12 hours
3. ***Neostigmine*** @ 0.02-2.0 mg/kg IM PRN or 0.5 mg/kg PO q8-12 h



C. Cholinergics "Nerve Agents"

1. Tabun (GA)

- Colorless to brownish liquid having a colorless vapor
- Odor is faintly fruity, unless pure
- Soluble in water with a flash point of 78°C (172°F)
- Other names include ethyl dimethylphosphoramidocyanidate, dimethylaminoethoxyphosphoryl cyanide, EA 1205

2. Sarin (GB)

- Colorless liquid
- Odor of Juicy Fruit gum, but odorless in pure form
- It is miscible in water, nonpersistent (evaporates quickly), and nonflammable
- Other names include isopropyl ester of methylphosphonofluoridic acid, zarin, isopropyl methylfluorophosphonate, isopropoxymethylphosphonyl fluoride

3. Soman (GD)

- With impurities this is amber to dark brown with a camphor odor
- In pure form it is a colorless liquid with a fruity odor
- Highly soluble in water, flash point of 121°C (250°F)
- Other names include Pinacolyl methanefluorophosphonate, EA 1210, Zoman

4. Venom X (VX)

- Colorless to straw colored liquid, similar in appearance to motor oil
- Odorless if pure, sulfur odor if impure
- It is persistent (evaporates slowly) and remains on surfaces for a long time
- Can remain in clothing and dog hair for days
- The most lethal agent known, miscible with water, flash point of 94°C (201°F)
- Other names include methyl phosphonothioic acid, ethyl-S-dimethylaminoethyl methylphosphonothiolate, and others

<u>Characteristics</u>

Nerve agents are the most toxic of warfare agents. They have a rapid onset of action and can gain access to the body via multiple routes of entry.

<u>Mechanism of Action</u>

Nerve agents interfere with the normal chemistry at the nerve-muscle or nerve-gland junction. Normally the enzyme acetylcholine (Ach) is the neurotransmitter secreted at a nerve ending to effect an action (muscle contraction or gland secretion). Once completed, the enzyme acetylcholinesterase is secreted to neutralize Ach so the muscle can relax or the gland stops release, and they reset for another contraction or secretion.

Nerve agents inhibit acetylcholinesterase so Ach remains and the muscle or gland receives continuous nerve stimulation. This leads to muscle twitching and fatigue, and for the gland there is excess production (tearing, salivation). Lower respiratory fluid builds up, bronchoconstriction occurs, and eventually cardiopulmonary failure and death. A fatal nerve agent dose may be fatal within 15 minutes of absorption.

Perhaps more familiar, these affects are seen in organophosphate and organocarbamate-containing pesticides formulated for use in dogs and cats used in the veterinary field to get rid of fleas and ticks.

Clinical Signs

Early Effects are mainly muscarinic signs:

- Miosis
- Involuntary urination, defecation
- Hyperpnea
- Bradycardia
- Excessive lacrimation
- Hypersalivation
- Dyspnea (bronchoconstriction)
- Tachycardia (catecholamine release)

Late Effects are nicotinic and CNS related:

- Nausea, vomiting
- Generalized weak, drowsy
- Ataxia
- Seizures
- Cyanosis
- Respiratory arrest
- Muscle fasciculations
- Confusion, anxiety
- Hyperthermia
- Flaccid paralysis
- Collapse, unconscious
- Coma, death

Treatments

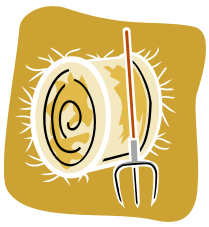
1. Move animal from contaminated area immediately
2. Flush dermal wounds with copious amounts of soap and water
3. Induce emesis only if ingestion was within last 60 minutes and patient shows no clinical signs (hydrogen peroxide, apomorphine)
4. Perform gastric lavage if possible; alternatively administer activated charcoal (activated charcoal may be indicated for dermal exposure due to absorption)
5. Technical decontamination with 0.5% sodium hypochlorite (1:10 bleach) 2-5 minutes, then rinse well

Emergency Treatments

1. Clear airway of fluid, provide oxygen
2. Secure airway if needed, ventilate, 80-100% oxygen for at least 30 minutes
3. EKG monitoring
4. IV access for cardiovascular and respiratory support
5. Thorough examination, blood collection and monitor electrolytes, acid-base
6. Control seizures (diazepam, midazolam, pentobarbital)
7. Treat hyperthermia if present

Antidotes

1. ***Atropine sulfate*** @ 0.2-2.0 mg/kg: give ¼ dose IV, rest IM or SC; repeat if necessary based on the reappearance or persistence of respiratory signs (not based on salivation or miosis); drug effects last 4-6 hours…avoid overdose!
2. ***Pralidoxime chloride*** (2-Pam) @ 20-50 mg/kg IV SLOWLY or SC every 12 hours. Start with low dose; if no response after 3-4 doses discontinue } not for carabamate
3. ***Diphenhydramine*** 1-4 mg/kg IM or PO every 8 hours to relieve muscle tremors. Start with lower dose if giving IM
4. ***Diazepam*** @ 5-10 mg IV; poorly absorbed IM and injection is painful. Midazolam at the same dose is a better alternative for IM administration w/r to absorption and less painful



D. Irritant Gases "Choking/Pulmonary Agents"

1. Chlorine (CL)
   - Greenish-yellow noncombustible industrial gas with pungent, irritating odor
   - Also known as molecular chlorine
2. Phosgene (CG)
   - Colorless nonflammable industrial gas with suffocating odor, like musty hay
   - Also known as carbon oxychloride, carbonyl chloride, carbonyl dichloride
3. Ammonia
   - Colorless nonflammable industrial chemical with distinct pungent odor
   - Anhydrous form has high affinity for water, rapidly penetrates tissues
   - Lighter than air, expands at a ratio of 850:1 in air

Characteristics

Generally gases or volatile liquids that dissipate rapidly. Their main effects are to the respiratory system via inhalation. Skin contact with concentrated material may cause chemical burns, but is not absorbed. Respiratory protection, unavailable to working search canines, is the best protection.

Mechanism of Action

Choking agents are irritating to the respiratory tract. Irritation from contact with respiratory tract mucosa causes fluid secretion. If the lung parenchyma is affected, alveolar sacs fill with fluid (pulmonary edema) and oxygen transfer from lungs to bloodstream is compromised. This is sometimes referred to as 'dry land drowning'.

Clinical Signs

- Coughing, choking
- Lacrimation (tearing)
- Foamy saliva
- Nausea, vomiting
- Dizziness
- Syncope, faint
- Skin irritation
- Hypoxia, cyanosis
- Burning eyes, nose, mouth, throat, lower respiratory tract

Treatments

1. Move animal from contaminated area immediately
2. Flush eyes, nose, mouth with water/saline, ophthalmic if available
3. Fluorescein cornea for damage; ophthalmic antibiotic ointment (no ulcer- steroid)
4. Flush wounds with copious amounts of water, treat based on severity (clip hair, antibacterial ointment/silver sulfadiazine, bandaging wet-to-dry or non-adherent)
5. Antibiotics (cefazolin/Keflex, ciprofloxacin/Baytril)
6. Analgesics (narcotics, NSAIDs)
7. Consider anti-emetics if vomiting (chlorpromazine, ondansetron (Zofran), dolasetron (Anzemet), maropitant citrate (Cerenia)

Emergency Treatments

1. Clear airway of fluid, provide oxygen
2. Secure airway as needed, ventilate if necessary, 80-100% O_2 at least 30 minutes
3. IV access for cardiovascular and respiratory support
4. Thorough examination, blood collection and monitor electrolytes, acid-base
5. Bronchodilator as needed (terbutaline, albuterol, aminophylline, metaproterenol)
6. Diuretic therapy (furosemide, mannitol)? Non-cardiogenic pulmonary edema from damaged alveolar-pulmonary membranes

E. Vesicants "Blister Agents"

1. Mustard Agents (H, HD, HN-1, HN-2, HN-3)

- Yellow to dark brown or black, oily liquids at room temperature
- Odor descriptions vary: burning garlic, horseradish, weak sweet agreeable (sulfur mustard), or fishy (nitrogen mustard)
- Similar flammability to motor oil
- Other names include Yperite (Y), Kampfstoff Lost, Iprit S-Lost, Schewefel-lost, Yellow cross liquid, senfgas, bissulfide, sulfide, bis (2-chloroethyl), EA 1033, etc

2. Lewisite (L) – an arsenical compound

- Amber to dark brown liquid with a strong penetrating *geranium odor*
- The pure compound is actually a colorless, odorless, oily liquid
- Nonflammable, dissolves in water to form solid Lewisite oxide
- Other names include (2-chlorovinyl) dichloro-arsine, arsonous dichloride, chlorovinylarsine, beta-chlorovinyldichloroarsine, EA 1034

3. Phosgene oxime (CX)

- As a solid, it is colorless; as a liquid, it is yellow-brown
- It has a strong, extremely irritating odor
- Its vapor density is greater than air, so it tends to settle in low lying areas
- Also known as dichloroformoxime

Characteristics

Irritation and blisters (in humans) are caused by direct contact with liquid or with high vapor concentrations. The eyes are the most sensitive organ. Warm, moist, thin-skinned areas (abdomen, axilla, flank) are also very sensitive as well as the entire respiratory tract. Open sores are susceptible to infection and take a long time to heal.

♫ **NOTE**: Canine dermal blood supply and skin reaction to chemicals differs w/r to human skin in that they do not develop blisters. Rather their skin becomes moist and hyperemic (reddens). Depending on the extent of the damage, skin may slough away, leaving open wounds. Because of their furry coat, meticulous physical examination of canine skin is needed to detect chemical burns early. Flinching or painful reaction when touched may be the only sign. More noticeable skin damage may not become apparent for hours to days.

MUSTARDS
Do not cause immediate symptoms or pain
Cell damage starts almost immediately

LEWISITE
Immediate intense pain on contact
Immediate cell damage

PHOSGENE
Vapor is extremely irritating
Liquid and vapor cause immediate tissue damage on contact

Mechanism of Action

Blister agent damage mechanisms of the skin and cornea are not completely understood. Exposure times and agent concentration play a role in the severity of damage. Respiratory tract exposure leads to severe mucosal tissue irritation, causing fluid secretion. This disrupts oxygen transfer within the pulmonary alveoli. Victims may also be susceptible to pneumonia.

Clinical Signs

MUSTARDS (delayed symptoms)

Eyes: Corneal irritation
Conjunctival irritation
Lacrimation (tearing)
Light sensitivity
Blindness

Skin: Red moist skin
Swelling within 2-3 hours
Erectile hair

Respiratory Tract:
Runny nose
Dry, barking cough
Hoarse vocalization
Nausea, vomiting (usually up to 24 hrs)
Fever
Respiratory distress (dyspnea)
Hemorrhage/necrosis lung tissue
May cough up blood/blood-tinged fluid

LEWISITE, PHOSGENE (acute)

Eyes: Burning, tearing
Pain, irritation, swollen lids
Corneal scarring, iritis
Severe damage/permanent blind within 1 minute

Skin: Redness in 30 minutes
Pain, itching for 24 hrs
Deep burns, pain for 2-3 days

Respiratory Tract:
Profuse nasal secretions
Violent sneezing
Cough
Frothing mucous
Pulmonary edema

Systemic Poisoning:
Restlessness, weakness
Hypothermia, low bld pressure

Treatments

1. Move animal from contaminated area immediately
2. Flush eyes with copious amounts of water or saline, ophthalmic if available
3. Immediate technical decontamination with 0.5% sodium hypochlorite (1:10 bleach) or alkaline soap
4. Fluorescein cornea for damage; ophthalmic antibiotic ointment (no ulcer- steroid)
5. Flush dermal wounds with copious amounts of soap and water, then treat irritation based on the severity of the wound (clip hair, antibacterial ointment/silver sulfadiazine, bandaging wet-to-dry or non-adherent)
6. Alkaline solutions (sodium bicarbonate, calcium carbonate) will hydrolyze CX
7. Antibiotics (cefazolin/Keflex, ciprofloxacin/Baytril)
8. Analgesics (narcotics, NSAIDs)
9. Consider anti-emetics if vomiting (chlorpromazine, ondansetron (Zofran), dolasetron (Anzemet), maropitant citrate (Cerenia)

Emergency Treatments

1. Clear airway of fluid, provide oxygen
2. Secure airway as needed, ventilate if necessary, 80-100% oxygen for at least 30 minutes
3. IV access for cardiovascular and respiratory support
4. Thorough examination, blood collection and monitor electrolytes, acid-base

Antidote for Lewisite

1. ***British Anti-Lewisite (BAL) ointment***
2. ***British Anti-Lewisite (BAL) Injectable*** is also known as dimercaprol (used to treat arsenic and lead poisoning) @ 2.5-5.0 (up to 7 mg/kg for severe cases) mg/kg IM every 4 hours for 2 days, then every 12 hours for the next 10 days
3. ***Edetate calcium disodium (CaEDTA),*** a heavy metal chelators, may be used if BAL not immediately available; 1% solution (10 mg/ml) in NS or D5W @ 27.5 mg/kg SC q 6 hours for 5 days, wait 5 days, repeat if needed

V. CHEMICAL AGENTS of CONCERN in a USAR ENVIRONMENT

A. Asphyxiants "Blood Agent Gases"

Sources:

1. **Carbon Monoxide** – Combustion, fires, smoke inhalation, auto and airplane exhaust, and poorly vented heaters
2. **Hydrogen Cyanide** – Fires, plants (apricot, peach, plum, and cherry pits), photo chemicals, plastics, laboratories, and pest baits
3. **Hydrogen Sulfide** – found in oil wells, refineries, tanneries, sulfur hot springs, asphalt fumes, mines, manure pits, septic tanks, and sludge pools
4. **Halogens** – *Chlorine* found in bleach products, plastics plants; *bromine* is a gas additive; *fluorine* (chlorofluorocarbons) in refrigerants, aerosols, solvents, fire extinguishers

Mechanisms of Action:

1. **Carbon Monoxide** – its affinity for hemoglobin is 240 times that of oxygen, replacing oxygen on the heme portion of the red blood cell to form carboxyhemoglobin.
2. **Hydrogen Cyanide** – irreversibly combines to ferric ion of cytochrome oxidase; blood becomes oxygenated but cells blocked from using it, turn to anaerobic metabolism
3. **Hydrogen Sulfide** – binds to mitochondrial cytochrome oxidase, blocking electron transport and causing cellular asphyxia
4. **Halogens** – chlorine generates to hydrochloric acid and oxygen free radicals; bromine causes a direct injury effect to mucous membranes, and fluorine causes cardiac toxicity and asphyxiation by saturating room air (as chlorofluorocarbon).

Clinical Signs: severity related to amount and duration of exposure

Carbon Monoxide	**Hydrogen Cyanide**	**Hydrogen Sulfide**	**Halogens**
Bright red MM	Bright red MM	Salivation	Conjunctivitis
Cherry red blood	Cherry red blood	Blepharospasm	Corneal burns
Tachypnea, dyspnea	Tachypnea, dyspnea	Tachypnea	Tachypnea/dyspnea
Hyperpnea	Hyperpnea	Pulm edema/pnmonitis	Pulm edema, pneumonitis
Confusion, Ataxia	Confusion, Ataxia	Confusion, dizzy	Confusion, dizzy
Lethargy	Lethargy	Nausea, vomiting	Nausea, vomit, hemoptosis
Seizures, Coma	Seizures, Coma	Seizures, Coma	Cardiac arryhthmia
Agonal respiration	Agonal respiration	Respiratory Arrest	Death

General Treatments:

1. Move affected animal to fresh air immediately
2. Secure airway, ventilate if necessary, 80-100% oxygen for at least 30 minutes
3. IV access for cardiovascular and respiratory support
4. Thorough examination, blood collection and monitor electrolytes, acid-base
5. Bronchodilator and diuretic therapy as needed
6. Seizure control. Monitor temperature

Additional Antidotal Treatments:

1. **Carbon Monoxide** – Oxyglobin, fresh blood transfusion, hyperbaric oxygen
2. **Hydrogen Cyanide** – Na thiosulfate, Na nitrite if diagnosis certain, Oxyglobin
   Emesis if ingested ≤15 min, gastric lavage if 15-60 min; activated charcoal
3. **Hydrogen sulfide** – Sodium nitrite
4. **Halogens:** copious irrigation of eyes and skin, corticosteroids, antibiotics
   Fluorine - avoid adrenergic drugs (avoid cardiac stimulation)

B. Cholinergic Pesticides: Organophosphate, Orgaocarbamate

Sources
Malathion, carbaryl (Sevin), bendiocarb (Ficam), propoxur (Baygon, Sendran), chlorpyrifos (Dursban), methylcarbamate, chlorfenvinphos (Dermaton Dip), cythioate (Proban), dichlorvos (Vapona), dioxathion, fenthion (ProSpot), Golden Malrin (fly bait)

Mechanism of Action:
Organophosphates and organocarbamates competitively inhibit acetylcholinesterase and pseudocholinesterase, allowing continued presence of acetylcholine to maintain a constant state of nerve stimulation. This tends to be reversible with the carbamates, but irreversible with phosphates. They are readily absorbed from skin, GI tract, and inhalation.

Clinical Signs:

Muscarinic - dyspnea (from bronchorrhea and bronchoconstriction), bradycardia, excessive lacrimation, salivation, miosis, micturition, defecation, vomiting (SLUDGE)
Nicotinic - facial twitching, tremors, generalized muscle fasciculations, weakness, eventual paralysis
CNS - convulsions, seizures, ataxia, anxiety, depression or aggression, centrally mediated respiratory depression, respiratory failure, death

Testing:
Diagnosis based on history, exposure; blood cholinesterase depression of ≥50% of normal indicates exposure. Depression to <25% of normal is often seen with toxic exposures.

Although not definitive, and atropine trial (0.02-0.04 mg/kg IV) may be indicative: if effects occur with this low dose (tachycardia, dry mouth, mydriasis) then cholinesterase inhibitor toxin is unlikely. This dose is too low to be effective in a cholinergic incident.

Treatments

1. Move animal from contaminated area immediately
2. Flush dermal wounds with copious amounts of soap and water
3. Induce emesis only if ingestion was within last 60 minutes and patient shows no clinical signs (hydrogen peroxide, apomorphine)
4. Perform gastric lavage if possible; alternatively administer activated charcoal (activated charcoal may be indicated for dermal exposure due to absorption)
5. Technical decontamination with 0.5% sodium hypochlorite (1:10 bleach) 2-5 minutes, then rinse well

Emergency Treatments

1. Clear airway of fluid, provide oxygen
2. Secure airway if needed, ventilate, 80-100% oxygen for at least 30 minutes
3. EKG monitoring
4. IV access for cardiovascular and respiratory support
5. Thorough examination, blood collection and monitor electrolytes, acid-base
6. Control seizures (diazepam, midazolam, pentobarbital)
7. Treat hyperthermia if present

Antidotes

1. ***Atropine sulfate*** @ 0.2-2.0 mg/kg: give ¼ dose IV, rest IM or SC; repeat if necessary based on the reappearance or persistence of respiratory signs (not based on salivation or miosis); drug effects last 4-6 hours…avoid overdose!
2. ***Pralidoxime chloride*** (2-Pam) @ 20-50 mg/kg IV SLOWLY or SC every 12 hours. Start with low dose; if no response after 3-4 doses discontinue } not for carabamate
3. ***Diphenhydramine*** 1-4 mg/kg IM or PO every 8 hours to relieve muscle tremors. Start with lower dose if giving IM
4. ***Diazepam*** @ 5-10 mg IV; poorly absorbed IM and injection is painful. Midazolam at the same dose is a better alternative for IM administration w/r to absorption and less painful

♫ Avoid morphine, phenothiazines, basically any drugs that decrease respiratory drive

C. Corrosives - Acids and Alkalis

Sources:

Household cleaners, toilet bowl and drain cleaners, dishwasher detergents, cleaners, antirust compounds, alkaline batteries

Mechanism of Action:

Acids produce corrosive burns, laryngeal spasm and edema may occur. Fortunately intense pain results and most animals will not ingest very much (alkalis as well).
Alkalis produce deep tissue necrosis which continues until neutralized by the tissues.

Clinical Signs:

- Oral mucous membrane irritation, ulcers, or burns (their absence does not rule out esophageal injury) which may be gray, yellow, or black from acids
- Ptyalism is common
- Oral +/- abdominal pain, vocalization
- Dysphagia, panting
- Laryngeal edema, upper airway obstruction
- Hematemesis
- Severe tissue injury may cause perforation of esophagus or stomach, leading to additional signs of pneumothorax, peritonitis, pleuritis, sepsis, shock, collapse, death

Treatments

Oral Ingestion – mild-moderate exposure symptoms:

1. Dilute with milk or water most effective if performed early. Activated charcoal is ineffective. Gastric secretions are usually enough to neutralize acids.
2. Gastric lavage and emesis **are not** recommended because of corrosive effects
3. **Do not** give neutralizing agents that usually result in a heat-producing reaction and worsens injury to the tissues
4. Transport for further evaluation, as endoscopy is recommended to accurately assess injury. Clinical signs rarely correlate to the degree of tissue injury.
5. Monitor for signs of oral, esophageal, and gastrointestinal irritation and ulceration
6. Symptomatic treatments always includes analgesics, others include antacids, gastrointestinal protectants, and antimicrobials. Corticosteroids are based on further diagnostics and individual preference regarding esophageal stricture formation.

Oral Ingestion – emergency treatment for severe symptoms:

1. Secure airway, ventilate patient, supplemental oxygen
2. IV catheter, collect blood and urine for testing
3. Crystalloid IV fluids (LRS, Normosol, Plasmalyte) maintain BP, urine output
4. Administer large volume of water (preferred) or milk
5. Analgesics (buprenorphine, butorphanol, morphine, oxymorphone, fentanyl)
6. Antibiotics (ampicillin, cephalosporin, enrofloxacin)
7. Corticosteroid administration controversial
8. Transport ASAP

Dermal exposure

1. Bathe immediately with mild liquid hand/dish detergent or non-insecticidal dog shampoo. The area should be flushed with running water for at least 30 minutes.
2. Monitor for erythema (brush back hair to see skin), swelling, pain, and pruritis
3. For eye exposure flush with sterile saline if available (water is better than no flush) for 30 minutes, then evaluate the cornea
4. Symptomatic treatments may include analgesics, anti-inflammatory drugs, and antimicrobials

D. Alcohols - Glycol

1. Ethylene Glycol

Sources:
Antifreeze and color film processing solutions

Mechanism of Action:
Although some ethylene glycol is eliminated unchanged through the kidneys, variable amounts are metabolized by the liver into toxic substances: glycoaldehyde (CNS & respiratory depression), glycolate (metabolic acidosis), and oxalate (with calcium forms crystals → renal damage). Phosphorus rust inhibitors may cause hyperphosphatemia.

Testing:
A commercially available test kit exists. Negative results are reliable, but false positives occur with formaldehyde, metaldehyde, glycerin/glycerol, propylene glycol, and activated charcoal. Test early as it does not test for the metabolites.

Many commercial products add a fluorescein dye to allow mechanics to detect leaks with a black light. Fluorescein may be detected in urine, gastric contents, on paws, on muzzle.

Clinical Signs:
Stage 1= 30 minutes to 12 hours after ingestion

- Polyuria, polydipsia
- Depression
- Seizures
- Rarely coma and death
- Nausea, Vomiting
- Ataxia
- Hyperglycemia

Stage 2 = 12-24 hours after ingestion

- Tachycardia
- Tachypnea

Stage 3 = 24-72 hours after ingestion

- Oliguric renal failure
- Severe depression
- Vomiting, diarrhea
- Dehydration, azotemia
- Hypothermia

Treatments:

1. Emesis if ingestion <15-30 minutes
2. Consider gastric lavage
3. Activated charcoal unless treatment is with oral ethanol
4. IV access for intravenous fluids to maintain BP and perfusion
5. Control seizures and temperature regulation
6. Sodium bicarb for metabolic acidosis, enhances excretion of glycolate metabolite
7. Diuretics to enhance renal excretion (mannitol may help reverse renal damage)

Antidotes - ***best administered before azotemia develops, otherwise prognosis is poor***

1. ***Fomepizole*** (4-methylpirazole) –minimal side effects
2. ***Ethanol*** – side effects include CNS depression, hyperosmolality, and diuresis
3. ***Thiamine & pyridoxine***

2. Propylene Glycol

Sources:
This is used in antifreeze, some drugs, and soft-moist pet foods.

Mechanism of Action:
Propylene glycol metabolizes to isomers of lactic acid. It also accumulates in the CNS causing a narcotic effect.

Testing:
This will test positive with the commercial ethylene glycol test.

Clinical Signs:

- Depression
- Ataxia
- Hypotension
- Lactic acidosis
- Hypothermia
- Muscle twitching
- Seizures
- Coma

Treatments:

1. Emesis and activated charcoal if within 30-60 minutes of ingestion if no signs
2. Gastric lavage if showing signs, then activated charcoal
3. IV catheter, blood work, crystalloid fluids (LRS Normosol-R, PlasmaLyte A) for lactic acidosis
4. Most dogs will eliminate the toxin within 24 hours

Emergency Treatment

1. Secure airway, ventilate if needed, 100% oxygen
2. Venous access for IV fluids and drug administration
3. Seizure control
4. Treat hypothermia if present
5. Fomepizole (4-methylpyrazole) treatment is as yet unsubstantiated

♬ A note about 'safer' antifreeze/coolant products

Sierra® antifreeze coolant is marketed as a safer coolant in case of ingestion by people or animals because it contains ***propylene glycol*** rather than ***ethylene glycol*** used in other antifreeze products. Not only is its effect less toxic, but it also has an unpleasant taste compared to the sweet taste of ethylene glycol products.

As with any potential toxic agent, the amount ingested and the relative body weight of the victim plays a role in how they will be affected. Safer doesn't imply they won't become ill, so monitoring, diagnostics, and subsequent treatments are still needed.

E. Hydrocarbons - Petroleum Distillates

Sources:
These organic compounds, made up entirely of hydrogen and carbon, are derived from petroleum distillates. They include kerosene, gasoline, mineral spirits, diesel and fuel oil.

Mechanism of Action:
These have direct irritation effects to skin, eyes, and MM, sensitize the myocardium to catecholamines, and when aspirated potentially cause fulminant and fatal pneumonitis. Hepatic, renal, and CNS injury may also occur.

Clinical Signs:

Inhalation	**Ingestion**	**Topical**
Conjunctivitis	Fixed pupils	Conjunctivitis
Nausea, vomiting	Nausea, vomiting	Erythema
Diarrhea	Diarrhea (+/- bloody)	Dermatitis
Depression	Depression	Hypotension (long exposure)
Tachypnea	Abdominal pain	
Cyanosis	Ataxia	
Hemoptysis	Confusion	
Pulmonary edema	Dizziness	
Hypotension, weak pulse	Incoordination	
Convulsions	Coma	
Collapse	Death	

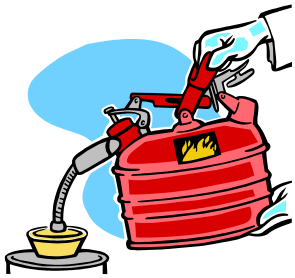
Treatments:

Dermal

1. Wash with liquid detergent & warm water, clip hair that won't clean well
2. Avoid inducing hypothermia
3. Topical dermal agents

Ingestion

1. **Do not** induce emesis
2. Gastric lavage if ingestion within 1-2 hours, but caution! risk of aspiration
3. Activated charcoal only if no aspiration risk, benefit debated
4. Saline or sorbitol cathartic (no magnesium, which worsens CNS signs)



Inhalation

1. Remove source or relocate patient to well ventilated area
2. Secure airway, ventilate if needed, 100% oxygen
3. IV access, collect blood to monitor values
4. Seizure control (diazepam, midazolam, phenobarbitol, pentobarbitol, propofol)
5. Bronchodilators for bronchospasm (albuterol, terbutaline, aminophylline, theophylline)
6. Diuretics for pulmonary edema (furosemide, dopamine)

F. Sympathomimetics - Chocolate: Theobromine and Caffeine (Methylxanthines)

Sources:

Methylxanthines are seen in chocolates and candy, baking chocolate, landscaping cacao shells, cocoa powder, coffee, tea, and soft drinks. The amount of theobromine and caffeine vary with the types of chocolate and drinks.

Mechanism of Action:

Theobromine causes the release of catecholamines (epinephrine, norepinephrine, and dopamine). Caffeine stimulates the myocardium and CNS while antagonizing benzodiazepine receptors in the brain.

Toxic Amount:

Theobromine 100-150 mg/kg causes toxic reaction.

Milk chocolate @ 44 mg of theobromine per oz. = 1 ounce/pound for toxicity
Semisweet chocolate @ 150mg/oz. = 1 ounce per 3 pounds for toxicity
Baker's chocolate @ 390mg/oz. = 1 ounce per 9 pounds for toxicity

Clinical Signs:

Theobromine	**Caffeine**
• Mild hypertension	• Tachypnea
• Brady/tachy/dysrhythmia	• Tachycardia, arrhythmia
• Nervousness, excitement	• Hyperexcitability
• Hyperthermia	• Hyperthermia
• Tremors, seizures	• Tremors, seizures
• Urinary incontinence	• Generalized congestion
• Coma, death	• Generalized hemorrhage

Treatments:

1. Emesis may be effective even after 4-6 hours since ingestion
2. Gastric lavage helps if emesis only partially productive or contraindicated
3. Activated charcoal recommended; significantly decreases half-life of theobromine
4. Diazepam for tremors, anxiety, or seizures; barbiturates if diazepam ineffective
5. Atropine for bradycardia
6. Lidocaine, metoprolol, or propanolol for tachycardia
7. Catheterize bladder to prevent reabsorption of theobromine through bladder mucosa
8. IV fluids for supportive care and to enhance excretion
9. Avoid erythromycin, corticosteroids that interfere with methylxanthine excretion.

Emergency Treatments

1. Secure airway, ventilation, oxygen
2. Venous access for blood sample, urine sample, and drug administration
3. Seizure control: diazepam often less effective because caffeine antagonizes benzodiazepine receptors in the brain. Try phenobarbitol, then pentobarbitol or propofol if necessary
4. Monitor for hyperthermia and treat as needed
5. Monitor ECG and treat dysrhythmias as needed

"OTHERS" CATEGORY OF TOXIC AGENTS in a USAR ENVIRONMENT

G. Alcohols

1. Alcohols, Ethanol, Methanol

Sources:

Isopropanol antiseptics, ethanol (ethyl alcohol), methanol, disinfectants, skin and aftershave lotions, perfumes, colognes, cleaning solvents, sanitizers (especially with pine oil)

Mechanism of Action:

Isopropanol is a potent central nervous system (CNS) depressant, twice as toxic as ethanol. Ingestion causes gastrointestinal (GI) irritation. Inhalation can cause chemical pneumonia, pulmonary edema, and coma.

Clinical Signs:

- Patient appears drunk
- Emesis, hematemesis, retching
- Respiratory depression
- CNS depression (may follow CNS stimulation)
- Alcoholic odor
- Cranial abdominal tenderness
- Shock

Testing

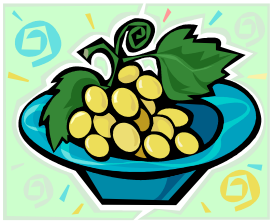
1. Blood alcohol levels can be measured at a human hospital
2. Exposure to methanol (refined petroleum product) or ethanol will have a negative result for commercial ethylene glycol test kit

Treatments

1. Gastric lavage only if large amounts were ingested within the past 2 hours
2. Emesis **is not** recommended due to potential for onset of CNS depression, increasing the risk for aspiration
3. Activated charcoal does not adsorb alcohols well and **is not** recommended
4. Nonspecific symptomatic care based on blood work
5. IV fluids and electrolytes as needed
6. Sodium bicarbonate added to fluids based on metabolic acidosis
7. Dextrose administration for hypoglycemia, a common sequela

Emergency Treatments

1. Secure airway, ventilate, 100% oxygen
2. IV access, IV fluids to maintain BP, urine output
3. Gastric lavage if large quantity within 2 hours ingested
4. Activated charcoal administration controversial w/r to effectiveness
5. Saline solution or cathartic without magnesium (Mg worsens CNS depression)
6. Blood gases may reveal respiratory and metabolic acidosis; sodium bicarbonate based on measurements



H. Foods (other than chocolate)

1. Grapes and Raisins

Mechanism of Action:
Causative agents or disease process is as yet unknown. The role of grapes or raisins is unclear. Evaluation and treatment are recommended even without clinical signs.

Testing:
Laboratory tests reveal increases in creatinine, blood urea nitrogen (BUN), phosphate and calcium. Urine production is severely diminished. Specific gravity is isosthenuric (1.010 regardless of fluid intake) and urine sediment often has renal tubular casts.

Clinical Signs:

- Vomiting
- Lethargy
- Anorexia
- Abdominal Pain
- Diarrhea
- Polyuria, oliguria, anuria

Treatments:

1. Decontamination via emesis (often patients are vomiting on their own)
2. Gastric lavage if emesis contraindicated but is often unnecessary
3. Activated charcoal administration and a saline cathartic
4. IV fluids (0.9% NaCl with hypercalcemia) maintain BP, hydration, urine output
5. Antiemetics for vomiting
6. Transport ASAP
7. Monitor urine output
8. Diuresis may be induced with mannitol, hypertonic dextrose solutions (10-20%), furosemide (also helps with hypercalcemia), or dopamine

2. Mycotoxin in Moldy Foods

Sources:
These are produced by molds that grow in foods, especially dairy, nuts (walnuts, pecans, almonds, and peanuts), stored grains, even pasta.

Testing
Laboratory analysis of the food is needed for definitive diagnosis. There is also a screen test for mycotoxins at Michigan State University.

Mechanism of Action:
Ingestion releases toxins that cause neuromuscular tremors. Mechanisms include increasing resting potentials, impulse transmissions, and depolarization duration as well as neurotransmitter inhibition.

Clinical Signs: (Ingestion of garbage containing mycotoxin causes signs usually in 2 hrs)

- Hypersalivation
- Restlessness
- Muscle tremors
- Tonic spasms
- Hyper reactive to external stimuli
- Hyperglycemia
- Seizures
- Death
- Excessive muscle activity→ hyperthermia, rhabdomyolysis, dehydration, exhaustion

Treatments:

1. Induce emesis if ingestion within 15-30 minutes and patient is not showing any signs
2. Activated charcoal and cathartic are also advised, unless signs exhibited
3. If showing signs, emesis is contra-indicated. Anesthesia and gastric lavage, activated charcoal, and cathartic are indicated
4. Methocarbamol (Robaxin) is a muscle relaxant that may control tremors

Emergency Treatment

1. Secure airway, ventilate/oxygen if needed
2. IV access for fluid administration, maintain BP
3. Control muscle tremors/seizures (diazepam, midazolam, phenobarbitol, pentobarbitol, propofol)

3. Xylitol – Sugar Alcohol

Sources:

Xylitol is used as a sugar substitute in many products: baked goods, desserts, toothpaste, and sugar-free gums and candies. It occurs in very low concentrations in fruits and vegetables.

Mechanism of Action:

In dogs, ingestion leads to a rapid severe increase in blood insulin concentration within 30-60 minutes. Acute hepatic failure occurs within 9-72 hours. The mechanism of xylitol-induced hepatic necrosis is not known.

Testing:

Blood tests are recommended to screen for liver enzyme elevations, hyperbilirubinemia, hypoglycemia, hypokalemia, hyperphosphatemia, thrombocytopenia, and prolonged clot time.

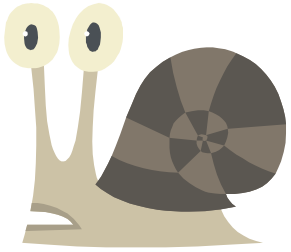
Clinical Signs:

- Vomiting
- Lethargy
- Weakness, ataxia, or seizures from hypoglycemia
- Petechiae, ecchymotic hemorrhages of the MM and skin, and bloody feces from coagulopathy

Treatments:

Aggressive treatment is recommended to avoid potential life-threatening consequences

1. Establish IV catheter for fluid and drug administration
2. Emesis only if no signs of hypoglycemia (weak, ataxic, seizures)
3. Activated charcoal administration of limited value
4. Transport ASAP
5. Monitor blood glucose, administer dextrose IV as needed; some advocate it if large amounts of xylitol ingested even if hypoglycemia has not yet developed
6. Monitor platelet count, coagulation variables, and liver enzymes
7. Plasma or whole blood transfusion for coagulopathy
8. Hepatic protectants as early as possible may help (n-acetylcysteine, S-adenosyl-L-methionine, silybin)



I. Metaldehyde

Sources:
Snail or slug bait and fuel source in small heaters. Check label for additional toxins (organocarbamate).

Mechanism of Action:
Precise mechanism is unknown. It is metabolized to acetaldehyde whose metabolism contributes to metabolic acidosis. This is worsened by muscle tremors and seizure activity.

Clinical Signs:

Early	**Next**	**Later**
Anxiety, tachycardia	Muscle tremors	Continuous convulsions
Nystagmus +/- mydriasis	Diarrhea	Hyperthermia. severe
Hyperpnea, panting	Convulsions	Acidosis
Thick frothy salivation		DIC
Stiff-legged gait, ataxia		Resp failure, cyanosis
Vomiting		Narcosis, death

Treatments:

1. Emesis if ingestion recent (<60 minutes) and no clinical signs
2. Gastric lavage with milk, activated charcoal, cathartic if emesis contraindicated
3. IV access, LRS/Normosol-R fluids, blood tests
4. Control muscle tremors with diazepam or acepromazine and methocarbamol
5. Treat for hyperthermia
6. Acidosis values pH<7.05 treat with sodium bicarbonate
7. Monitor liver values, as after surviving acute episodes liver complications may result

J. Polychlorinated Biphenyls (PCBs)

Sources: Pesticides

Mechanism of Action: These are hepatotoxic.

Clinical Signs: Anorexia, nausea, abdominal pain, coagulopathy

Treatments:

1. Dermal exposure: wash with liquid detergent and water to reduce absorption
2. **Never** use hydrocarbon-based solvents to clean skin as they not only increase the absorption but have their own toxic effects!
3. Emesis is contraindicated because of the aspiration risk
4. Activated charcoal administration is of unknown benefit but recommended along with saline or sorbitol cathartic
5. Adding fiber to diet may decrease intestinal absorption, stimulate fecal elimination
6. Monitor liver values and coagulation values
7. Treat emergency signs symptomatically

K. Metals

Sources, kinetics, and toxic effects of selected hazardous metals

Metal	Source	Kinetics	Toxic effects
Antimony	Fireproofing chemicals, manufacture of glassware and ceramics, pigments, insecticides, rodenticides, and antibilharzial medications[10]	ADME: Slow GI absorption and poor absorption via inhalation; stibine gas[a] is readily absorbed via inhalation[10] Toxicity: Low systemic toxicity; trivalent forms are more toxic than pentavalent forms; stibine gas is highly toxic following inhalation	GI: Severe vomiting, oral mucosal ulceration, hemorrhagic gastroenteritis, watery diarrhea[10] Respiratory: Metal fume fever, pulmonary interstitial fibrosis[10] Hematologic: Hemolysis (stibine gas)[62] Other: Hypovolemia, shock, anemia, myocardial degeneration, proximal renal tubular degeneration, cochlear damage, hepatic injury (stibine gas)[10,62]
Arsenic	Ground water, pesticides and other agricultural products, chemical warfare agents, microelectronics manufacturing, fossil fuel manufacturing[11]	ADME: Well absorbed orally and via inhalation; substantial dermal absorption with prolonged contact or compromised dermal integrity; arsine gas[b] is readily absorbed via inhalation; arsenic crosses placenta and can deposit in pulmonary bones; primarily excreted via the urine; other routes of excretion include sweat, saliva, milk, and incorporation into hair, epithelium, and nails[10] Toxicity: Trivalent forms are more toxic than pentavalent forms; arsine gas is highly toxic following inhalation[10]	General: Lethargy, death[10] GI: Severe vomiting, oral mucosal ulceration, hemorrhagic gastroenteritis, watery diarrhea, abdominal pain[10] Respiratory: Metal fume fever, pulmonary interstitial fibrosis[10] Hematologic: Hemolysis (arsine gas)[10] Other: Endothelial damage, myocardial degeneration, proximal renal tubular degeneration, pulmonary edema, cardiac arrhythmias, hypovolemia, shock, anemia[10]
Beryllium	Nuclear reactors, x-ray windows, aerospace equipment and fuels, automotive parts, computers and other electronics, dental supplies, telecommunications equipment, welding materials[63]	ADME: Poor oral absorption, some dermal absorption; inhalation is main route of exposure; minimal absorption from lungs, as most inhaled beryllium is sequestered in fibrotic granulomata within the lungs and pulmonary lymph nodes[10] Toxicity: GI, dermal, and pulmonary irritant; minimal systemic toxicity; humans, but no other species, develop pulmonary delayed hypersensitivity (chronic berylliosis)[10]	GI: Erosive gastroenteritis; small intestinal mucosal necrosis[10] Respiratory: Erosive tracheobronchitis, pulmonary fibrosis with granulomata[10] Other: Bone marrow erythroid hypoplasia[10]
Cadmium	Paints and pigments, electroplating, galvanizing, NiCad batteries, jewelry and manufacturing, shielding for nuclear reactor cores, fossil fuel combustion[64]	ADME: Poor oral absorption; up to 30% of inhaled cadmium is absorbed; binds to metallothionein in blood and cells; stored in kidney, liver, lung, and pancreas; excreted through urine[10] Toxicity: GI and pulmonary irritant, binds macromolecules of renal tubular epithelium, and interferes with vitamin D and calcium metabolism[10]	GI: Mild gastritis to severe hemorrhagic gastroenteritis, vomiting, diarrhea, oral esophageal ulceration[10] Respiratory: Tracheobronchitis, pneumonitis, metal fume fever, pulmonary edema[10] Renal: Proximal renal tubular degeneration and necrosis, ®2-microglobulinuria, glucosuria, aminoaciduria[10] Other: Corneal ulceration, ovarian and testicular necrosis[10]
Chromium[c]	Leather tanning materials, pressure-treated lumber, anticorrosive agent for boilers, metal plating, lithography/ photography materials, textile manufacturing, welding materials, glass manufacturing, television picture tubes[65]	ADME: Poor oral absorption of trivalent salts; hexavalent salts more readily absorbed orally; up to 85% of inhaled hexavalent chromiumis absorbed via lungs; carried in blood by transferrin or RBCs; crosses placenta; 80% excreted through urine[65] Toxicity: GI and pulmonary irritant; binds macromolecules of renal tubular epithelium;	GI: Vomiting; gastric corrosive injury with or without perforation[65] Respiratory: Tracheobronchitis, pneumonitis, metal fume fever, pulmonary edema[65] Renal: Renal tubular degeneration and necrosis[65]

		interferes with vitamin D and calcium metabolism[65]	Other: Hypovolemia, circulatory failure, hepatitis and hepatic necrosis, metabolic acidosis, methemoglobinemia, thrombocytopenia, anemia, dermal hypersensitivity[65]
Cobalt	Aircraft engine manufacturing, mining equipment and cutting tools, tire manufacturing, paints and pigments, pottery production, diamond polishing, jewelry manufacturing[66]	ADME: Absorbed via skin, ingestion and inhalation; binds to albumin; accumulates in liver and adipose tissue; excreted primarily through urine[66] Toxicity: Acute toxicity uncommon, primarily a chronic disease; oxidative injury to myocardium and hepatocytes; alters calcium channels in cells and interferes with cellular respiration at mitochondrial level	Respiratory: Hard metal disease (pulmonary interstitial fibrosis), cough, dyspnea, asthma[66] Cardiovascular: Enlarged heart, left ventricular failure, pericardial effusion[66,67] GI: Vomiting, diarrhea[66] Other: Polycythemia, hepatocellular necrosis, corneal injury, acute hypersensitivity reaction[66]
Lead	Batteries, welding materials, solders, plastic and rubber manufacturing, leaded gasoline, paints and pigments, ammunition, electrical and radiologic shieldings, radiator repair products, copper and zinc smelting[10]	ADME: Poor oral absorption in adults; inhaled lead is readily absorbed from the lungs; inorganic lead not appreciably absorbed dermally; organic leads well absorbed dermally, orally, and by inhalation[10] Toxicity: Impairs heme synthesis, inpedes vitamin D metabolism, competes with calcium ions, inhibits membrane-associated enzymes[10]	GI: Vomiting, diarrhea, anorexia (signs may be intermittent)[10,68] Neurologic: Ataxia, behavior changes, lethargy, seizures (signs may be intermittent)[10,68] Other: Anemia, weight loss, renal insufficiency, decreased fertility[10,68]

Sources, kinetics, and toxic effects of selected hazardous metals (continued)

Metal	Source	Kinetics	Toxic effects
Mercury	Dental amalgams, batteries, instrumentation (eg, thermometers, barometers, calibration instruments), electroplating, jewelry, paints and pigments, photographic materials, semiconductor solar cells, paper pulp manufacturing[69]	ADME: Elemental mercury not absorbed orally; fumes from elemental mercury are absorbed via inhalation; mercury salts have low GI absorption; organic mercurials well absorbed orally; inhaled mercury vapor readily crosses lungs, placenta, and blood-brain barrier; highest levels in kidney; excretion primarily via urine[10] Toxicity: Ingested elemental mercury has low oral toxicity; inhaled mercury vapor causes respiratory irritation; absorbed mercury is damaging to renal tubular epithelium; myocardial degeneration; organic mercury may cause neuronal necrosis and axonal degeneration[10]	GI: Inorganic salts may cause severe oral, esophageal, and gastric corrosive injury; vomiting; diarrhea; abdominal pain[10,69] Respiratory: Metal fume fever, tracheobronchitis, pneumonitis, pulmonary edema, dyspnea[10,17] Neurologic: Organic mercury may cause ataxia, gait abnormalities, visual disturbances, behavior changes, muscle tremor and movement disorders[10,17] Other: Renal insufficiency, glomerulonephritis, cardiac arrhythmias, anemia (blood loss), myocardial failure, abortion, fetal cerebellar and cerebral deformities (organic mercury)[10,17]
Nickel	Electroplating, NiCad batteries, glass, jewelry coins, cutlery, dental and medical implant manufacturing, pigments, magnetic tape manufacturing, computer components, fossil fuel combustion[70]	ADME: Relatively well absorbed orally; 35% of inhaled nickel is absorbed; distributed to lungs, kidney, and skin; 90% excreted in urine[70] Toxicity: Generally only dermal and inhalation exposures are associated with significant signs; nickel carbonyl inhalation has highest potential to cause serious signs; severe respiratory irritant; myocardial damage reported experimentally[70]	Respiratory: Cough, dyspnea, cyanosis, pulmonary edema, interstitial paralysis[16,71] GI: Vomiting, diarrhea[70] Other: Lethargy, fever, ataxia, seizures, dermatitis[70]
Thallium	Rodenticides (banned in the US), photoelectric cells, lamps, semiconductors[72]	ADME: Well absorbed orally, dermally, and by inhalation; distributed widely throughout body; 60% excreted via feces, remainder in urine; extensive	GI: Nausea, vomiting, diarrhea, hemorrhagic gastroenteritis, abdominal pain; onset within hours after exposure[74]

		enterohepatic recirculation results in long half-life[10,73] Toxicity: Replaces potassium in metabolic reactions; cumulative toxicosis possible owing to long half-life; alters cell membrane function and mitochondrial activity; results in dysfunction of a variety of systems, most notably GI, neurologic, and dermatologic[10,73]	Neurologic: Disorientation, seizures, behavioral alterations, coma, peripheral neuropathy; onset within 1 week after exposure[74] Dermatologic: Erythema (within hours to days), alopecia initially at areas of friction (eg, axilla, commissures of lips), dermal necrosis, epidermal slough (within days to weeks)[74] Other: Necrotizing pneumonia, renal or hepatic injury, anemia, cardiac dysrhythmias[74]
Zinc	Galvanizing, dyes and pigments, wood preservatives, medicinal agents, televisions, x-ray and computer monitor screens; pesticides, cosmetics manufacturing, dental cements, electroplating, paper manufacturing[75]	ADME: Poor oral absorption; persistence of zinc objects in acidic stomach environment may allow for enhanced absorption; excreted primarily via urine[16,74] Toxicity: Irritant, oxidative damage to RBCs, hemolysis, nephrotoxicosis[16,74]	GI: Vomiting, anorexia, diarrhea[16] Hematologic: Intravascular hemolysis, anemia, icterus, hemoglobinemia[16] Other: Azotemia, hemoglobinuria, proteinuria[16]

[a]

Stibine gas is the toxic gas formed when acidic antimony compounds react with hydrogen gas. bArsine gas is the toxic gas formed when arsenic reacts with acids;
it is used in the manufacturing of microchips.11 cTrivalent and hexavalent salts.
ADME = Absorption, distribution, metabolism, and excretion. GI = Gastrointestinal.

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

Calcium EDTA

100 mg/kg SC, q 24 h for 2 to 5 days; divide this dose into 4 portions, each portion diluted with 5% dextrose solution to a concentration of 10 mg of calcium EDTA/mL; then be administered at a different site; do not exceed 2 g/d, do not treat for more than 5 consecutive days

Dimercaprol

2.5 to 5 mg/kg IM, q 4 h for 2 days, then q 12 h

Succimer

10 mg/kg PO, q 8 h for 10 days; administer on an empty stomach

d-Penicillamine

7.5 to 27.5 mg/kg PO, q 6 h for 7 days; may be repeated after 7 days if needed

Metals: Initial treatments, decontamination, and chelation

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[11]
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[40]
Beryllium	Fluid therapy, gastrointestinal tract protectants, oxygen, ventilatory support, other supportive care	Remove from source, bathe, induce emesis or perform gastric lavage	Not indicated[41]
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[11]; succimer, deferroxamine, and calcium EDTA have been used experimentally[42]
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[43]
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[44]
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[42]; 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 following exposure to organic mercury compounds[12]; forms that have potential for corrosive injury), dilute with egg white, administer activated charcoal, enemas, or a cathartic	Chelation is contraindicated dimercaprol may be used following acute ingestion of caustic inorganic mercury; d-penicillamine and succimer may also be used[12]; 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[11,45]; diethyldithiocarbamate has been used experimentally in animals exposed to nickel carbonyl[46]
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	Chelation generally not recommended as chelated thallium may more readily enter CNS and exacerbate neurologic signs[11]

		developed)[11]		
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[11]; calcium EDTA or d-penicillamine may be used; gastrointestinal tract must be cleared of metal prior to chelation; adequate urine output must be maintained during chelation; monitor blood zinc concentration during chelation	

L. Phenol

Sources:

Coal-tar derivatives are found in disinfectants, drugs, foods, caustics, keratolytics, and soaps.

Mechanism of Action:

Phenols denature and precipitate cell proteins, are extremely corrosive, and produce penetrating lesions. Luckily most animals will not ingest enough to cause esophageal injury. They stimulate the brain's respiratory center and some demyelinate the brain white matter.

Clinical Signs:

- Profuse salivation
- Respiratory stimulation
- Then respiratory depression
- Anorexia
- Vomiting
- Ataxia
- Panting
- Muscle fasciculations
- Hypotension
- Dark mucous membranes
- Green or Black urine
- Unconsciousness
- Seizures
- Shock

Treatments:

1. Dermal exposure: blot fur and skin, then liquid detergent and copious washing
2. Ocular exposure: irrigate eyes with 0.9% saline, treat corneal ulcers
3. Emesis is **contraindicated** because of corrosive effects
4. Dilute with egg whites or milk; gastric lavage and activated charcoal administration

Emergency Treatment

1. Secure airway, ventilate if needed, 100% oxygen
2. IV access for blood work, perfusion, BP
3. Control seizures and hyperthermia f present
4. Oxyglobin or whole blood transfusion for methemoglobinemia of present

Antidotes

1. N-acetylcysteine or SAMe (Denosyl) to avoid hepatic and renal damage

M. Rodenticides

1. Anticoagulant, Vitamin K-Antagonist

Sources:
1st Generation – warfarin, coumarin, D-Con, Ward 42, etc…
2nd Generation – brodifacoum, bromadiolone, Havoc, Talon, etc…
Indandiones – diphacinones, valones, pidones, Promar, Ramik, etc…

Mechanism of Action:
Anticoagulant rodenticides inhibit the body's ability to activate vitamin K, which is needed to activate the vitamin K-dependent clotting factors II, VII, IX, and X. Once the body's reserves of these factors are used up, coagulopathy results.

Testing:
Tests include Activated Clot Time (ACT), Prothrombin Time (PTT), Activated Partial Thromboplastin Time (APTT), and Proteins Invoked by Vitamin K Absence (PIVKA).

- Abnormal coagulation test results often develop in 1-2 days, but may be delayed up to 5-7 days after ingestion.
- Time to signs, severity, and prognosis are dependent on the type of rodenticide, amount ingested, and time elapsed before treatment.

Clinical Signs:

- Depression
- Weakness
- Pallor
- Melena
- Epistaxis
- Hematemesis
- Hematuria
- Gingival bleeding
- Small wound bleeding is profuse
- Dyspnea
- Blindness, hyphema
- Paresis, paralysis
- Seizures
- Hemothorax, hemoabdomen

Treatments:

1. Induce emesis if within 60 minutes of ingestion, patient is not seizuring or comatose
2. If within 2-4 hours perform gastric lavage
3. Administer activated charcoal
4. Thirty minutes after charcoal administer saline cathartic

Antidote

1. Vitamin K_1 is the antidote of choice

Emergency Treatment

1. Secure patent airway, intubate and ventilate 100% oxygen if needed
2. IV access, blood work
3. Treat shock with crystalloid fluids to maintain perfusion, BP
4. Note that fresh whole blood, fresh plasma, fresh frozen plasma needed to restore coagulation parameters
5. Colloids (hetastarch, dextrans) are avoided as they are known to have anticoagulation complications

2. Cholecalciferol, Vitamin D

Sources:

Rodent baits such as Ortho Mouse-B-Gone, Rampage, Rat-B-Gone and Quintox Rat are a few of the rodenticides that contain cholecalciferol.

Mechanism of Action:

Vitamin D rodenticides contain cholecalciferol which causes lethal hypercalcemia and hemorrhage by increasing intestinal absorption, renal reabsorption, and bone resorption of calcium. The half-life may be as long as 30 days.

Testing:

Monitor serum calcium, phosphorus, BUN, and creatinine.

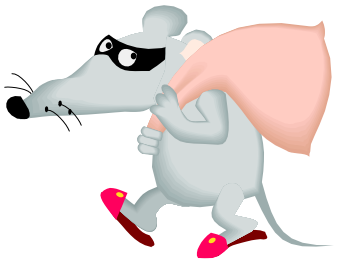
Clinical Signs:

- Anorexia
- Lethargy
- Vomiting
- Constipation
- Diarrhea
- Dehydration
- Depression
- Polyuria
- Polydipsia
- Anuric renal failure
- Petechiation
- Hematemesis
- Hematochezia
- Shock
- Dysrhythmias
- Muscle twitching
- Seizures
- Stupor
- Death

Treatments:

1. IV access for saline fluid diuresis to lower calcium levels
2. Blood work to monitor hematocrit, hypercalcemia, hyperphosphatemia, and azotemia
3. Induce emesis if within 4 hours, or gastric lavage then activated charcoal
4. Cathartic 30 minutes after charcoal, but only once
5. Furosemide to promote calciuresis
6. Calcitonin for severe hypercalcemia
7. Aluminum hydroxide to bind phosphate
8. Sodium bicarbonate if severe acidosis
9. Control seizures and treat hyperthermia
10. Monitor ECG for arrhythmias

11. Ensure patent airway, intubate and ventilate of needed with oxygen



VI. BIOLOGICAL AGENTS of CONCERN

Dogs are naturally resistant to many of the potential human biological warfare agents. They may suffer some of the same effects, but usually to a lesser degree. No approved vaccines against these agents exist for canines.

As with many agents, time exposed and contaminant concentration are factors in victim response. Also, canines may play a role as a vector and decontamination remains an important aspect in treating not only the canine but preventing further spread to others. This is usually the first step in a treatment protocol.

Awareness of a threat agent and preventing exposure or further exposure are paramount to mitigating the severity of the situation. Unfortunately, some biological releases will not be detected until victims become affected and seek care.

A. Bacteria

1. Anthrax

Etiology

Caused by contact with spores from *Bacillus anthracis*, this zoonotic (transmissible to humans) disease may affect virtually all warm-blooded animals, though livestock and horses are most susceptible. Dogs, cats, swine, mink, and captive wild animals have acquired the disease from contaminated meat. It is distributed worldwide.

Clinical Signs - Three forms of the disease occur, depending on the route of entry:

	Canines	**Humans**
Cutaneous	Not seen	Itching, sores, develop into papules, ulcers
Inhalation	Not seen Experimentally: fever	Nausea, vomit, fever, cough, resp distress Respiratory tract hemorrhage
GI	Upper GI inflammation Fever, weak, anorexia, depress Progresses to septicemia	Anorexia, nausea, fever, stomach pain, Bloody diarrhea, sepsis

Canine Susceptibility

Canines are relatively resistant (500-1000 times more resistant). Only a mild fever developed in experiments of canine respiratory exposure. They are most susceptible to ingestion of spores and show similar signs to humans. Contaminated meat is the usual source. In a terrorist situation, ingestion may occur when a dog contaminated with anthrax spores cleans itself by licking.

Diagnosis

- Microscopic exam of victim blood smear or mice injected with victim blood
- Culture growth identification or fluorescent antibody (FA) of tissues, culture

Treatment and Control

1. Decontamination - paramount to prevent spread to humans and other susceptible species
2. Penicillins: procaine 10,000-20,000 U/kg IM/SC BID; Penicillin-V 10 mg/kg PO TID; Pen G potassium 20,000 units/kg IV/IM/SC q 4 hours
3. Other: doxycycline 5 mg/kg IV/PO q12h; ciprofloxacin 5-15 mg/kg PO q12h
4. Supportive care

2. Brucellosis

Etiology

Caused by bacteria from the genus *Brucella*, this occurs in farm animals, horses, deer and elk as well. *B. suis* or *B. melitensis* are the warfare agents of choice. Although canines can be infected by these, they usually acquire *B. canis* of which they are the definitive host. Transmission is either congenital, venereal, or by ingestion of infective materials. Transmission to humans is documented, the result of contact with contaminated animals or products.

Clinical Signs

Canines	**Humans**
&Female: infertility, stillbirths, abortion	Flu-like: fever, sweats, headache, back pain, weakness
%Male: infertility, epididymitis, periorchitis, prostatitis	CNS infection - meningoencephalitis
Other: Fever, lethargy, weight loss peripheral lymphadenopathy	Cardiac infection - endocarditis
Other tissue: uveitis, glomerulonephritis, Diskospondylitis (vertebral)	

♫ **Note**: Most canines are asymptomatic or mildly affected. No mortality in adults.

Diagnosis

1. Definitive diagnosis requires isolation of organism from blood, semen, or other infected tissues. Low levels of bacteremia may make this difficult.
2. Many serologic tests are widely available and more commonly used (RSAT, TAT, AGID, ELISA)

Treatment

1. Antibiotic therapy (repeat 2 months later if cultures and titers fail to improve)
    a. Enrofloxacin 10-15 mg/kg PO BID 3 weeks, then off 3 weeks, may repeat 1-3 times (best initial choice)
    b. Minocycline 12.5 mg/kg PO BID 2-3 weeks, the off 3 weeks
    c. Tetracycline 10-20 mg/kg PO TID 3 weeks, then off 3 weeks
    d. Doxycycline 5 mg/kg IV/PO q 12 hours
2. Eliminate from breeding program
3. Neuter all infected dogs

3. Cholera

Etiology

Ingestion of contaminated water with *Vibrio cholerae* is the mechanism of infection. The bacteria are typically found in water environments such as freshwater lakes and rivers. Contamination also can occur from fecal material from infected individuals.

Clinical Signs

- Diarrhea, vomiting, dehydration, and shock.
- Most animals infected will show no signs of illness. If illness occurs, animals will have large amounts of watery diarrhea and vomiting. They can become rapidly dehydrated, which can lead to death.

Canine Susceptibility
Dogs may become infected if exposed to very large numbers of *Vibrio cholerae* in food or water. Outbreaks have been reported in bison, cattle and dogs.

Diagnosis
Fecal examination and/or culture are the best way. There is a rapid cholera dipstick test available for humans.

Treatment
- Supportive Care: fluids, electrolytes, anti-emetics
- Monitor blood values, urine production, blood pressure

4. Plague – Pneumonic, Bubonic

Etiology
This bacterial zoonotic disease caused by *Yersinia pestis* infects both cats and dogs in the western United States. It is best recalled as the 'Black Death', transmitted to humans from rats through the infected bites of fleas. Cases associated with cats and dogs have been recognized in humans since 1959.

Animal-to-human transmission occurs via the bite of an infected flea (may be carried by a canine), direct contact with infected tissue, or infected animal's bite or scratch. The organism cannot penetrate unbroken skin, but can invade mucous membranes.

Clinical Signs – three forms of the disease may be seen

	Canine	**Human**
Bubonic	Suppurative Lymphadenopathy ('bubo')	Painful lymphadenopathy
Pneumonic	Upper respiratory signs uncommon	Cough, chest pain, dyspnea, fever
Septicemic	High fever	Fever , myalgia, headache, toxemia, death

Canine Susceptibility
Dogs are fairly resistant, and naturally acquired disease rarely produces clinical signs. Experimental infection in 10 dogs resulted in transient illness. Fevers up to 105°F (40.5°C) occurred for as long as 72 hours, all recovered and were normal by day 7 after exposure. In dogs natural infection it is uncommon and not well documented.

Diagnosis
1. Bacteriologic and microscopic examination of lymph node aspirate and blood
2. Direct fluorescent antibody test on exudate
3. PCA techniques on exudate and lymph node aspirates

Treatment
1. Antibiotics
   a. Aminoglycosides: gentamicin 2-4 mg/kg IV/IM/SC BID-TID
   b. Streptomycin 10 mg/kg IM BID-every 6 hours
   c. Tetracycline 10-22 mg/kg PO BID-TID; doxycycline 5 mg/kg IV/PO BID
   d. Enrofloxacin 5-20 mg/kg PO/IM BID
   e. Chloramphenicol 25-50 mg/kg IV/PO TID
2. Flea control products
3. Supportive Care

5. Q-Fever (Query Fever)

Etiology

A rickettsial organism, Coxiella burnetii has two modes of transmission: (1) it circulates through ticks, who act as reservoirs of the disease, (2) disseminated through milk, urine, feces, placenta, and post-partum discharges. Aerosolization from infected tissues is also a potential source of infection. The organism is maintained in bird and rodent reservoirs in nature, but considered uncommon in the United States.

Clinical Signs

In the canine, Q fever infection in parturient dogs may lead to early death of pups. When clinical disease occurs, reproductive failure is usually the only symptom presented. Fever, lethargy, and anorexia may be present, but are uncommon.

Canine Susceptibility

Infections in animals are usually not apparent, asymptomatic, and not considered a veterinary problem. When clinical disease occurs, reproductive failure (abortion) is usually the only symptom presented.

Diagnosis

In farm animals, complement fixation tests are most commonly used. Other tests include agglutination, isolation of organism, or visualization in stained tissue preps.

Treatment

1. Decontamination with 0.5% sodium hypochlorite (1:10 diluted bleach)
2. Tetracycline is the antibiotic of choice
3. Doxycycline +/- fluoroquinolones used in humans; unknown value in animals

6. Tularemia

Etiology

This disease, caused by *Francisella tularensis*, is a bacterial septicemia of wild rodents and lagomorphs (rabbits). It is transmitted to other animals and man by tick bites (most common), ingestion of infected rabbits or contaminated water, or inhalation of infective aerosols.

Clinical Signs

In the dog, soft nodular swellings under the skin, which drain and heal, have been linked to tularemia. Other signs may include:

- Fever
- Abscesses
- Lymphadenopathy
- Anorexia
- Splenomegaly
- Hepatomegaly, icterus

Canine Susceptibility

Dogs are generally resistant, can acquire the disease from ticks, may develop signs.

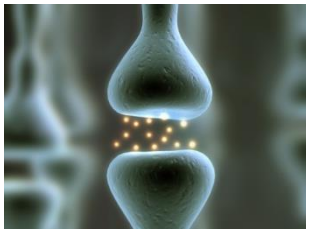
Diagnosis

Serological diagnosis is essential and accurate; culture, necropsy, and histopathlogy

Treatment

1. Antibiotics: Gentamicin 2-4 mg/kg IV/IM/SC BID-TID
   Tetracycline 10-22 mg/kg PO BID-TID
   Chloramphenicol 25-50 mg/kg IV/PO TID
2. Supportive care

B. Biological Toxins



1. Botulism

Etiology

Botulism is an intoxication, not an infection, caused by ingestion of a neurotoxin produced by the bacterium *Clostridium botulinum*. There are several serotypes of neurotoxin: A, B, and E are important in humans while types C and D are more common in dogs. Sources include food, garbage, or carrion that contains preformed neurotoxin. It is also in some rodenticides (Vengeance®, Assault®, Trounce®). The toxin prevents synthesis or release of acetylcholine (Ach) at motor end-plates, hindering passage of impulses from nerves to motor and-plates.

Clinical Signs

Onset is usually within 24-48 hours, up to 6 days of ingestion, causing progressive, symmetrical, generalized lower motor neuron dysfunction:

- Disturbed vision
- Difficulty chew, swallowing
- Ascending flaccid motor paralysis
- Respiratory or cardiac paralysis
- No loss of mental alertness
- Intact, normal pain perception
- Vomiting, regurgitation
- Death

Canine Susceptibility

Dogs are comparatively resistant to all types of ingested botulinum neurotoxin.

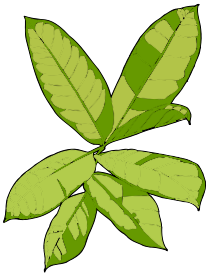
Diagnosis

Commonly this is deduced by history, suggestive clinical signs, and eliminating other causes of paralysis. Identification of toxin from serum or carrion is also possible using patient serum bioassay, ELISA antigen test, and PCR for bacterial DNA in environmental samples.

Treatment

1. Gastric lavage or induce emesis if recent ingestion
2. C. botulinum antitoxin C+D controversial; may be effective early in course of disease, but dogs often recover with good support so use is questionable
3. Increasing neuromuscular function:
    i. Physostigmine by inhibiting acetylcholinesterase @ 0.06 mg/kg IM/IV over 5 minutes; *risky treatment*! Also requires atropine @ 0.2-2.0 mg/kg ¼ dose IV and rest SC/IM to reduce muscarinic effects of the drug (bradycardia, salivation, miosis, urination, defecation)
    ii. Guanidine hydrochloride, a human treatment, may increase Ach release; *unknown value!*
4. Antibiotics: use in food-borne illness questionable
    i. Penicillin G 20,000 units/kg IM q12h
    ii. Metronidazole 10 mg/kg PO q12h
    iii. *Avoid aminoglycosides which potentiate neuromuscular blockade*
5. Supportive care
    i. Nutritional support – enteral, parenteral
    ii. Nursing care - cleanliness, turning, physical therapy
6. Respiratory support

2. Ricin



Etiology

Ricin is a potent protein phytotoxin that acts as a proteolytic enzyme to break down natural proteins. It may also act as an antigen, causing agglutination of red blood cells.

Derived from the beans of the castor plant *Ricinus communis*, the whole plant is toxic but particularly the seeds. Toxicity is much greater if the seed coat is broken, or seeds are chewed. It can be made from the mash left over from processing into several forms: powder, mist, pellet or dissolved in water or weak acid. It is 6-9 times more potent than the nerve agent sarin.

Clinical Signs

Depending on the route of exposure, dose, and whole seed versus broken:

- Stomatitis, glossitis
- Vomiting and diarrhea
- Muscle trembling
- Ataxia
- Hematuria, azotemia
- Acute anaphylaxis-like rxn
- Collapse
- Death

Canine Susceptibility

An injected dose as low as 0.0001 mg/kg is the minimum lethal dose for most mammals. The oral toxic dose is higher due to poor gastrointestinal ingestion, but still is considered a relatively low dose that can cause severe signs. Inhalation effects are not reported in canines, but based on human exposure it may be very toxic in small doses.

Diagnosis

Diagnosis is often difficult unless ingestion is witnessed or plant material is found and identified. Acute lung injury is expected with exposure to aerosolized Ricin. Serum and respiratory secretions may be submitted for ELISA antigen test.

Treatment

1. Induce vomiting if ingestion witnessed
    i. Hydrogen peroxide 3% @ 1-2 mL/kg PO, may repeat in 5-10 minutes (note: following with some water increases foaming activity in stomach)
    ii. Apomorphine @ 0.04 mg/kg IV or 0.08 mg/kg IM,SC or ¼ tablet in the conjunctival sac (flush eye afterwards)
2. Gastric lavage followed by activated charcoal @ 2 g/kg PO (sorbitol only in 1st dose)
3. Cathartic
    i. Magnesium sulfate @ 250-500 mg/kg mix with 5-10 mL/kg of water PO
    ii. Magnesium hydroxide (Milk of Magnesia) @10-150 mL PO q6-12h prn
    iii. Sodium sulfate @ 250-500 mg/kg mix with 5-10 X as much water PO
    iv. Sorbitol 4 g/kg PO
4. Supportive and symptomatic support: IV fluids, electrolytes
5. Antihistamines: diphenhydramine @ 2-4 mg/kg PO q8-12h; 1 mg/kg IM/SC q8-12h; (do not exceed 40 mg total dose) (IV can cause severe hypotension)
6. Monitor bloods for electrolytes and for onset of renal failure

3. Staphylococcal Enterotoxin B (SEB)

Etiology

Enterotoxins are food-related poisonings from ingestion of food contaminated by one of several microorganisms and their toxins. SEB is related to the exotoxin produced by *Staphylococcus aureus*. These activate intestinal epithelial secretory mechanisms, cause morphologic mucosal changes, disrupt absorptive capabilities, and alter GI biochemical pathways.

Clinical Signs

Signs may appear within 15 minutes to 6 hours, 3 hours post-ingestion is common.

- Vomiting
- Restlessness
- Weakness, ataxia
- Shock-fluid loss
- Cranial abdominal pain (hypermotility)
- Gas-distended abdomen (ileus)
- Diarrhea, may be hemorrhagic
- Hypoglycemia, leukopenia

Diagnosis

Ingestion history of garbage, carrion, or spoiled food is informative. Bacterial culture or serologic identification of *Staphyloccocal* toxins provides a definitive diagnosis.

Treatment

1. Cleansing of the gastrointestinal tract
    a. Emetics early if vomiting has not occurred
        i. Hydrogen peroxide 3% @ 1-2 mL/kg PO, may repeat in 5-10 min (note: follow with some water increases foaming activity in stomach)
        ii. Apomorphine @ 0.04 mg/kg IV or 0.08 mg/kg IM,SC or ¼ tablet in the conjunctival sac (flush eye afterwards)
    b. Gastric lavage if ingesta in stomach, then activated charcoal @ 2 g/kg PO (sorbitol only in first dose if activated charcoal is repeated)
2. Enteral antibiotics
    a. Ampicillin @ 22 mg/kg PO q8h or 11-22 mg/kg IV,SC,IM q6-8h
    b. Cefazolin @ 15-30 mg/kg IV,IM q6-8h
3. Supportive therapy
    a. Antiemetics
        i. Metoclopramide @ 0.2-0.5 mg/kg PO,SC,IV q8h or 1-2 mg/kg q24h or as CRI @ 0.01-0.02 mg/kg/hr
        ii. Chlorpromazine 0.05 mg/kg IV q4h if vomiting continue
        iii. Dolasetron (Anzemet) @ 0.6 mg/kg IV q24h
        iv. Ondansetron (Zofran) @ 0.1-1.0 mg/kg PO q12-24h
        v. Maropitant (Cerenia) @ 1.0 mg/kg SC q24h or 2.0 mg/kg PO q12h
    b. GI protectants
        i. Bismuth subsalicylate (Pepto-Bismol) 0.25-2 mL/kg PO q6-8h
        ii. Kaolin/pectin 1-2 mL/kg PO q6-12h
    c. Anti-ulceratives against lower esophagitis
        i. Ranitidine (Zantac) @ 1-2 mg/kg IV,SC,PO q8-12h
        ii. Famotidine (Pepcid) @ 0.5-1.0 mg/kg PO,IV q12-24h
        iii. Cimetidine (Tagamet) @ 4-10 mg/kg IV,IM,PO q8-12h
        iv. Omeprazole (Prilosec) @ 0.7 mg/kg PO q24h
4. Fluid therapy
5. Shock treatment

4. Tricothecene Mycotoxins (T-2)

Etiology

These mycotoxins are a group of more than 40 compounds produced by the fungi of the genus *Fusarium*. They may be found on corn, wheat, and barley and have been found in commercial cereal-based pet foods. Ingestion is the most common form of exposure in animals. As a terrorist weapon, an aerosolized form may be used ('yellow rain)' and dermal, ocular, and respiratory exposures would be expected. Inhibition of protein synthesis seems to be the primary cause of many symptoms.

Clinical Signs

Other than the ingestion section, all other signs are extrapolated from human exposure and *have not been documented in dogs.*

Ingestion

- Anorexia, weight loss
- Vomiting
- GIT irritation, diarrhea
- Bradycardia
- Low blood pressure
- Immunosuppression

Ocular Exposure

- Redness
- Pain
- Conjunctivitis
- Corneal irritation

Dermal Exposure

- Pain
- Pruritis
- Bruising, redness
- Vesicles
- Necrosis
- Epidermal Sloughing

Respiratory Exposure

- Nose and throat pain
- Nasal discharge, itch, sneeze
- Cough, hemoptysis
- Chest pain
- Dyspnea
- Wheezing

Severe poisoning results in prostration, infertility, weakness, ataxia, balance/coordination problems, collapse, shock, memory and other cognitive complications including seizures, and death.

Canine Susceptibility

?

Diagnosis

Testing of blood, tissues, and environmental samples can confirm the diagnosis. T-2 should be suspected if an aerosol attack occurs in the form of "yellow rain" with droplets of yellow fluid contaminating clothes and the environment.

Treatment

1. Eye exposure: copious saline irrigation
2. Dermal exposure: soap and water decontamination, treat wounds as needed (clip hair in dogs!)
3. Ingestion: activated charcoal; supportive care; emesis not recommended as irritation to the esophagus and oral cavity may be duplicated on the way out
4. Respiratory inhalation: remove from environment, oxygen, respiratory support as needed

C. Viruses

1. Smallpox

This is not reported in canines. Historically, man has transmitted the virus to animals but the zoonotic transmission of the vaccinia virus is not known.

A similar related virus is monkeypox. Terrorists who may not be able to obtain the variola virus may try to disseminate this one. Canines are susceptible to this. Transmission is via animal bites or direct contact with affected animals' blood, body fluid, or lesions. Person-to-person transmission is the same way. Animals get a macular, popular, vesicular or pustular rash in a localized or general distribution, conjunctivitis, coryza (rhinitis), cough, anorexia, and lethargy. Treatment is supportive. Appropriate personal protection precautions should be taken.

2. Venezuelan Equine Encephalitis

This has not been reported in canines. Another member of the *Alphavirus* genera, Eastern equine encephalitis virus (EEEV) is an *Alphavirus* that is endemic in the Southeastern United States. From 1993 to January 2005, the Veterinary Diagnostic and Investigational Laboratory in Tifton, Georgia, performed postmortem examinations on over 101 domestic canines exhibiting clinical neurological disturbances. In 12 of these dogs, brains were histologically suggestive of infection with EEEV. All dogs were less than 6 months of age, with no breed predilection. Clinical signs included pyrexia, depression, nystagmus, and lateral recumbency.

3. Viral Hemorrhagic Fevers

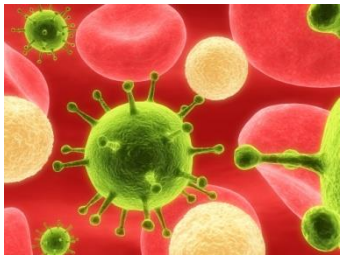
Most of these are in general not associated with canines, other than Rift Valley Fever. This is restricted to the African continent, spreading between animals primarily via mosquitoes.

Rift Valley Fever affects many species, including canines, and could be transmitted via arthropods found in the United States. IT is most severe in young animals. Dogs and cats <3 weeks old infected with the virus may range from no clinical signs to severe ones (petechiae, meningitis, myocarditis, hepatic necrosis, death). Older dogs and cats infected with the virus appear to develop little disease. Treatment is supportive care.

4. Nipah

The Nipah virus belongs to the family Paramyxoviridae. In humans infection appears associated by close contact with pigs, and has a 40% fatality rate. Over 1 million pigs from >900 farms in Malaysia were slaughtered because of it. Natural infection has been detected serologically in canines, cats, horses, goats, chickens, and bats.

Dogs develop signs similar to the distemper virus: fever, respiratory distress, conjunctivitis, mucopurulent nasal and conjunctival discharges. Researchers could isolate the virus from the urine of cats. Fruit bats and insectivorous bats are reservoirs for the virus.



VII. RADIOLOGICAL AGENTS of CONCERN

A. Forms

Radiological materials emit ionizing radiation. This radiation has enough energy to alter cells. It takes one or more forms: alpha, beta, gamma, or neutron radiation. Importantly, proper decontamination will prevent further spread of the material, limit further absorption, and remove a source for inhalation or ingestion exposure to both the canines and the humans around them.

Nuclear incidents involve detonation of a nuclear device whereas radiological incidents produce radiation without detonation of a nuclear device

Alpha Particles
- Limited penetration: stopped by superficial dead skin layer or paper sheet
- Inhalation is the primary route of entry
- Ingestion is very dangerous
- Presence may be masked by water

Beta Particles
- More penetration but generally travels just a few inches in the air
- Causes sunburn-like effect in humans (‘beta-burn’)
- Inhalation is the primary route of entry
- Stopped by inner skin layers but also dangerous if ingested

Gamma Radiation
- Not particulate, more like a high-energy x-ray with long range
- Affects all areas of the body
- Significant penetration
- Dangerous weather external or ingested

Neutrons
- Most immediately damaging to cells on contact, travel far in air
- Stopped by water, paraffin, or plastic
- Like the result of a nuclear explosion or power plant mishap

B. Dispersal

1. Radiological Dispersal Device (RDD) or "Dirty Bomb"

This is the most likely scenario as a present day terrorism threat, this is composed of conventional explosives with radioactive material added.

a. Conventional explosive likely to cause the most damage physically
b. Radiation dispersal will vary depending on the level of radiation used
c. High level radiation involves fission by-products from a nuclear facility, and is therefore more difficult for terrorist organizations to obtain
d. Low level radiation comes from industrial and medical facilities, therefore is more likely due to relative ease for a terrorist organization to obtain

2. Nuclear Weapon

The blast from a nuclear device is the result of a fission reaction.

a. It is possible, but not probable that terrorist organizations can acquire the components to make such a device
b. A more likely device is the suitcase bombs - smaller nuclear devices the equivalent of a 1 kiloton bomb
c. Russia's supply of suitcase bombs is not all accounted for, and as many as 84 may be missing

3. Non-explosive Exposure

This scenario is one where a radioactive substance is left open, contaminating people and animals in its vicinity.

C. Acute and Chronic Radiation Syndrome

Canine >75 rad; Human >100 rad	Canine >260 rad; Human >350 rad
• Nausea, vomiting	• Fever
• Skin damage, hair loss	• Incapacitation
• Lethargy	• Convulsions
• Diarrhea	• Death

Canine Susceptibility

Dogs are comparatively more susceptible to all types of radiation, ~ 25-50%.

1. Acute Radiation Syndrome (Dr. Jerry Upp)

Situational Aspects

- For humans, ARS is usually from exposure to > 100 rad
- For canines, the number is 70 rad, though milder symptoms will occur at 30 rad
- Source usually external, though internal exposure can occur
- Radiation must be penetrating, gamma most likely
- Large portion of body exposed; ARS uncommon in localized exposure

Syndromes

a. Hematopoetic Syndrome

70-1000 rad, mild signs at 30 rad
Bone marrow suppression is usual cause of death

b. Gastrointestinal Syndrome

Irreversible damage to the GIT from exposure to 1,000-10,000 rad
Survival is unlikely, death within 2 weeks or less
Euthanasia likely chosen over such suffering

c. Central Nervous Syndrome

Doses of over 5,000 rad and is always fatal
Death in humans usually within 3 days

d. Cutaneous Syndrome

Localized inflammation, erythema, hair follicle damage, necrosis
Lesser haired areas most susceptible (muzzle, eyes, pinna, face, ventral abdomen)

Stages

These are from human studies, but one can assume canines would go through similar stages. The timing may be different (shorter)

1. **Prodromal Stage**
   Nausea, vomiting, diarrhea
   Vomiting at < 4 hours after exposure is a poorer prognosis then > 4 hours

2. **Latent Stage**
   Patient feels better, looks better, does better
   May last from hours to days to weeks

3. **Illness Stage**
   Signs become more severe, patient feels worse
   Signs may last up to several months

4. **Recovery or Death Stage**
   This may last from weeks to months, up to 2 years

2. Chronic Radiation Syndrome

Extrapolating from human chronic exposure papers described in Russia, cataract formation and tumor formation would be possible sequelae.

Other illnesses may include changes in behavior, gastrointestinal tract, and blood values (leukopenia, anemia)

D. Management - External Exposure, Decontamination

1. Decontamination

- Remove
  - Relocate to a well-ventilated upwind and upgrade area
  - Remove, replace all gear with metal or nylon disposable items
  - If in dust or powder form, wet down so as not to aerosolize agent into canine's breathing zone
- Wash
  - High-volume, low-pressure lukewarm water, soap if available
  - Don't delay if warm water or soap are not immediately available
  - Repeat three times if possible
  - Do not delay if warm water or soap is not available
  - Special attention to paws and pads that can trap material in folds and crevices; try soft-bristled brush or sponge
- Monitor
  - Alpha radiation is masked by water, so thoroughly cleanse and rinse the canine and be checked for radiation once dried off
  - Medical examination
  - Hazmat and medical monitoring

2. Medical Treatments

- Skin and pad irritation
  - Clip hair
  - Soaks/cleansing in mild antibacterial solutions (betadine diluted 1:10, chlorhexadine diluted 1:40)
  - Bandaging or leave open to heal; prevent licking

- Ocular irritation
  - Ophthalmic saline or purified water irrigation
  - Fluorescein stain corneas for lesions
  - Ophthalmic artificial tears, antibiotics, +/- steroids based on corneal diagnostics

- Respiratory problems
  - Oxygen therapy
  - Further diagnostics – radiographs, pulse oximeter, supportive care based on findings (pulmonary edema – furosemide, bronchoconstriction – bronchodilator, etc…)

E. Treatments - Internal/Ingestion Exposure to Radiological Agents

Radiological Treatment Protocols for Ingested Materials

Radionuclide	Medication	Human Protocol	Canine Protocol	Action
Iodine	Potassium Iodide (KI)	390 mg stat, then 130 mg q.d.x7-14 days if indicated		Blocking
Rare Earths Plutonium Transplutonics Yttrium	Diethylenetriamine-pentaacetate (DTPA)	1 gm Ca-DTPA in 500 ml 5% D/W IV over 60 minutes; or 1 gm (4 ml) in 6 ml 5%D/W by slow IV injection over 1 minute		Chelation
Cesium Rubidium Thallium	Prussian Blue	1 gm in 100-200 ml water PO TID for 3 weeks		Mobilization from organs/tissues; reduction, absorption
Calcium Barium	Sodium alginate	10 gm in large glass of water		Inhibits absorption
Arsenic Bismuth Gold Lead Mercury Polonium	Dimercaprol (British Anti-Lewisite or BAL)	1 ampule (300 mg) IM q4 hours x 3 days (test for sensitivity first with 1/4 ampule)	Arsenic: 3-4 mg/kg IM q8h until recovery; if severe 6-7 mg/kg IM first day only Lead: 2.5 mg/kg IM q4h days 1&2; q8h day 3; q12h after that; if severe give 5 mg/kg day 1 only **Clear GIT of metal before chelate**	Promotes excretion
Copper Gold Lead Mercury Polonium	d-Penicillamine Ethylenediamine-triacetate (EDTA)	D-pen: 1 gm IV or 0.9 gm PO q4-6 hours	D-pen for mercury/lead: 8 mg/kg PO q6h or 10-55 mg/kg q12h; EDTA for lead: 100 mg/kg q24h for 2-5 days	Chelation
Tritium	Water	Force liquid		Isotopic dilution
Strontium	Ammonium chloride	3 gm PO TID	100-200 mg/kg/day divided q8-12 hours	Demineralizing agent
Strontium-Radium	BaSO4 or MgSO4	100 gm BaSO4 in 250 ml water	4-6 mg/kg PO	Reduces absorption
Radium	Calcium gluconate	May be tried: 20% Ca-Gluconate @ 10 ml IV once or twice daily	0.25-1.5 ml/kg IV slow over 5-30 minutes; monitor for bradycardia	Displacement
Uranium	Sodium bicarbonate (NaHCO3)	Slow IV infusion of physiological solution (250 ml @ 14%)	1-2 mEq/kg IV q3-4h Bicarb = 0.3 x Base Deficit give 1/4-1/2 dose over 1 hr, rest over 6-12 hrs; monitor blood gas	Alkalinization of urine to reduce acute tubular necrosis

Protection, Decontamination, and Medical Aid for K-9 Teams; EAI Corporation, a division of SAIC; 2006

VIII. EXPLOSIVES and BLAST CONCERNS (Dr. Wayne Wingfield)

A. Devices

1. Categories

a. High-Order Explosive (HE)

- Supersonic over-pressurization shock wave
- TNT, C-4, Semtex, nitroglycerin, dynamite, amm nitrate fuel oil (ANFO)

b. Low-Order Explosive (LE)

- Subsonic explosion, lacks HE over-pressurization
- Pipe bomb, gunpowder, Molotov cocktail, aircraft as guided missiles

c. Manufactured

- Standard military issue. mass produced, quality-tested
- Exclusively High-Order Explosives

d. Improvised

- Illegally obtained, produced in small quantities
- Use of device outside its intended purpose
- IEDs made by terrorists may be HE, LE, or both

2. Types

a. Mechanical - build up of pressure inside a container, often from over-heating
b. Chemical - Instantaneous conversion of solid or liquid to gas
c. Nuclear - Fission (split atom nuclei) or fusion (join atom nuclei under force)

B. Blast Wave Patterns

1. ***The medium*** through which these sinusoidal waves travel, underwater and air, affect the severity of injury
    a. As water does not compress, an underwater blast wave will travel further than if detonated on land and cause injury at greater distances from the detonation point
    b. Injuries from underwater blast are also more severe as they are stronger for longer distances

2. Blast wave pressure

a. Expanding gases compress surrounding air causing high pressure front traveling at speeds upwards of 900 mph (1,450 kph)
b. Dense wave striking body cause injury - the sudden thump in the chest

3. ***Victim's body*** in relation to surrounding structures also plays a role
    a. If a blast wave strikes a wall, it will reflect and magnify the force of the wave
    b. An animal in front of that wall will experience devastating injury from the direct hit as well as the reflected hit

4. ***Body armor*** pros and cons
    a. Armor will protect from shrapnel and other missile debris
    b. Unfortunately, blast wave reflects off the inside of armor, causing further injury
    c. Similarly a closed room will reflect waves back and forth

C. Blast Injury Dynamics

The most vulnerable organs to blast injury are the gas-filled: ears, lungs, gastrointestinal tract

1. **Primary** - effect of blast wave or pressure wave on the body

    a. ***Spalling*** mechanism transfers blast wave through dense body tissues (liver, muscle) and into less dense tissue (GIT and lungs)
    b. ***Implosion*** of gas-filled spaces follows as surrounding high pressures compress them
    c. ***Shearing and tearing*** of affected tissues based on their vulnerability. From most to least vulnerable: Ear - tympanic membrane rupture
    Lungs - contusions, pneumothorax, air emboli
    GIT - vascular bed shearing, hemorrhage

2. **Secondary**

    a. ***Penetrating and blunt injury*** from blast-propelled 'missile' debris
    b. Since waves dissipate with distance, moving to a safe distance is best if known threat is imminent
    c. Standing structure protection may become blast debris and cause injury

3. **Tertiary -** displacement of body by the blast into other objects
    a. ***Victims becomes the projectiles*** as blast wave and associated wind throws them - injuries dependent upon what they hit
    b. ***Miscellaneous effects*** - burns, smoke inhalation

4. **Quarternary**
    a. Explosion-related injuries, illnesses, diseases from ***other than listed above***
    b. Includes exacerbation and complications of pre-existing conditions

D. Blast Patient Assessment and Treatment

1. **Approach**

    a. This is a trauma: ABCs or MARCHE
    b. Airway management paramount unless hemorrhage takes precedence
    c. Complete Physical Examination
    d. Animals will be disoriented, confused, possible hearing loss
    e. Eardrum rupture bilaterally suggest more underlying injuries likely
    **Because the K9 ear canal takes 90^o turn they are more resistant to TM rupture so lack of this injury does not discount further internal injuries*
    f. Consider possible toxin exposure (gas, irritant, etc...)
    g. Decontamination may be important part of treatment! Especially wound contamination.....

2. **Explosion Injury Summary**

    a. Auditory - Typanum rupture, ossicle and cochlear damage, foreign body

    b. Ocular - Perforated globe, foreign body, air embolism

c. Facial - Fractures, lacerations

d. Thoracic - "Blast Lung" Hemothorax, pneumothorax, pulmonary contusions/hemorrhage, air embolism, mucosal damage, aspiration pneumonitis, sepsis

e. Digestive - Bowel perforation, hemoabdomen, ruptured liver/spleen, mesenteric ischemia (air embolism), sepsis

f. Circulatory - Cardiac contusion, myocardial infarction (air embolism), shock, vasovagal hypotension, peripheral vascular injury

g. CNS - Concussion, brain injury (open, closed), spinal cord injury (fracture, luxation, paresis/paralysis), air embolism

h. Renal - Contusions, laceration, acute renal failure from shock (hypotension, hypovolemia) or rhabdomyolysis

i. Musculoskeletal - fractures (open, closed), crush injury, compartment syndrome (pressure within muscular fascia), lacerations, burns, air emboli, traumatic amputation

3. Some Clinical Pearls

a. ***Tympanum (TM) Rupture -*** not all animal blast victims will have eardrum ruptures, but if present then further underlying injury is likely; *still suspect further injury in the canine as they appear to be more resistant to the TM ruptures due to the 'L' canal shape.* With isolated rupture if thoracic radiographs are normal and all other parameters stable, prognosis is good but monitor for at least 48 hours

b. ***Hypopharyngeal petechial hemorrhages*** may be present with or without tympanum rupture, and suggest significant blast wave exposure with underlying injury to pulmonary and gastrointestinal systems likely

c. ***Pulmonary air embolism*** is suspect when disorientation and confusion (head injury), and TM rupture coincide.

d. ***Pulmonary contusions*** may be delayed in development, and may not appear radiographically for several days; thoracic monitoring should continue for at least 24-48 hours; arterial blood gases may indicate hypoxia from contusions in the absence of other detected thoracic injury

e. ***Gastrointestinal injuries*** are more difficult to identify on initial examination, and effects may be delayed for several days. Suspect until proven otherwise. Monitor: decreased bowel sounds, abdominal splinting (pain), rectal bleeding (red - lower GIT, black - upper GIT), hemoptosis (bloody vomit). Imaging: radiographs (free air), ultrasound

IX. APPENDICES

A. NERVE AGENT ANTIDOTE ADMINISTRATION USING THE MARK 1 AUTOINJECTORS IN DOGS

Introduction

The first rule of administration of nerve agent antidotes in dogs is the handlers should treat themselves first if there has been an exposure.

A second consideration is how likely is the diagnosis? The signs of nerve agent exposure include salivation, excessive tearing, urination and defecation, slowing of the heart rate, small pupils, and difficulty breathing.
Dogs may have increased salivation from many different compounds that would not warrant emergency administration of these antidotes.

Currently there is no official provision for supplying handlers with Mark 1 kits for their dogs. In light of some of the difficulties with using a human-designed antidote kit on canines, having these drugs available as separately for use in canines may be considered. However, should the need arise, these will guide Mark 1 kit use in dogs.

Medical Management

Managing a victim of nerve agent intoxication consists of 4 things:

1. Decontamination
2. Ventilation
3. Antidote administration – IM injection, epaxial muscle if possible
4. Supportive care based on patient condition

Decontamination of the skin is usually unnecessary after exposure to vapor alone, but remove any items from the body (collar, leash, pack) that may trap vapor and cause off-gassing. Soap and copious amounts of water are used for skin decontamination. Be cognizant of the ambient temperature as well as the wash water temperature. Special attention should be given to hard-to-clean areas, like the footpads.

Ventilation is important because air resistance increases (in humans up to 50-70 cm H_2O) due to bronchoconstriction and secretions. *Atropine* will alleviate resistance, but also thickens secretions, which may require suctioning. Most dogs will tolerate some form of open cone mask through which oxygen can be introduced if necessary. If the dog can remain in a sitting or sternal position (standing or lying down on the sternum), that will allow the most lung expansion of both sides of the thorax.

Antidote treatment is based on counteracting the organophosphorous cholinesterase inhibition of nerve agents. The Mark 1 self-injectors are designed for this purpose in humans, and can also be used for dogs. These injections are designed to be administered intramuscularly in the field. For dogs, it is recommended to give them in the epaxial musculature, the band of muscles on either side of the lumbar spinal cord. Familiarity with the anatomy is important.

A note of caution: Realize the injection may hurt enough that the dog may turn around and snap at the injection and/or the injector. Sudden movement may prevent the entire dose from being absorbed. The dogs should be muzzled and properly restrained during the injection to prevent injury to the handler and themselves. This does not require a manufactured muzzle but may be as simple as briefly wrapping the leash around the muzzle to keep the mouth closed during the injection. Because the dog may be having breathing difficulty as a result of exposure, the muzzle must be able to be removed quickly.

The Mark 1 kits involve 3 drugs:

1. **Atropine**

- Anticholinergic, blocks excess acetylcholine at peripheral muscarinic sites
- Common side effects: sinus tachycardia, dry mouth, thirst, mydriasis, constipation
- Adverse Effects: initial bradycardia, 2^{o} heart block, vomiting, urinary hesitancy, CNS stimulation, drowsiness, ataxia, seizures, respiratory depression, hypotension
- Mark 1 autoinjector contains 2 mg, and is connected alongside the 2-Pam injector
- Canine dosing range for treatment of cholinergic toxicity: 0.2-2.0 mg/kg
- Consider using the lower end of the dose, which can be repeated if signs persist

Weight (lb)	Weight (kg)	Dose Range (mg)	Min # Injectors
40	18	3.6 - 36 mg	2
50	23	4.6 - 46 mg	2
60	27	5.4 - 54 mg	3
70	32	6.4 - 64 mg	3
80	36	7.2 - 72 mg	3.5
90	41	8.2 - 82 mg	4

*It is very difficult to give a fraction of an autoinjector, therefore in the case of a dog weighing between 80 and 90 pounds (36-41 kg) the dog should be evaluated after the 3rd injector and a 4th considered if the heart rate is less than 140 and there are active signs of toxicity.

2. **2-Pam CL**

- Pralidoxime chloride, or Protopam chloride is an oxime
- Oximes attach to the nerve agent that is inhibiting the cholinesterase enzyme and they break the agent-enzyme bond to allow cholinesterase to return to normal activity
- Used as an antidote in organophosphate poisoning (NOT in carbamate poisoning) along with atropine
- Adverse side effects: neuromuscular blockade, acetylcholinesterase inhibition, tachycardia, weakness
- Mark 1 autoinjector contains 600 mg, and is connected alongside the atropine injector
- Canine dose range for OP toxicity: 20 mg/kg 2-3 times a day as needed
- Veterinary attention should be sought for any dog exposed to nerve agent, and continued administration should be determined by a veterinarian

Weight (lb)	Weight (kg)	Dose Range (mg)	Min # Injectors
40	18	360 - 900 mg	0.5
50	23	460 - 1150 mg	0.5 - 1
60	27	540 - 1350 mg	1
70	32	640 - 1600 mg	1
80	36	720 - 1800 mg	1
90	41	820 - 2050 mg	1.5

*It is very difficult to give a fraction of an autoinjector; therefore 1 autoinjector should be used for the 40-50 pound dogs, and 2 injectors for dogs > 90 lbs. While the safety margin is pretty wide, be aware that an overdose can cause signs similar to the signs of the original nerve agent such as muscle weakness, vomiting, increased respirations, or seizures.

3. Diazepam

- A benzodiazepine anticonvulsant drug also known as valium
- Serum half-life in dogs is 2.5 – 3.2 hours
- Common side effects include sedation, ataxia, excitement, occasionally paradoxical aggression
- Overdose causes CNS depression
- Mark 1 injector contains 10 mg
- This injector is separate from the atropine and 2-PAM

Weight (lb)	Weight (kg)	Dose Range (mg)	Min # Injectors
40	18	5 - 10 mg IV	1
50	23	5 - 10 mg IV	1
60	27	5 - 10 mg IV	1
70	32	5 - 10 mg IV	1
80	36	5 - 10 mg IV	1
90	41	5 - 10 mg IV	1

*****These doses are published for IV injection. Absorption of diazepam from an IM route of administration is slower than oral and incompletely absorbed. It may also be painful due to the propylene glycol used to solubilize the drug. If available, midazolam, a water soluble drug of the same class and dosing, is readily absorbed intramuscularly.

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B. Poison Formulary

POISON FORMULARY

ASPCA Animal Poison Control Center 1-888-426-4435

4-Methypyrazole (Fomepizole, Antizol-Vet, 4MP) – ethylene glycol treatment: 20 mg/kg IV load dose, 15 mg/kg IV @ 12 & 24 h, 5 mg/kg IV @ 36, 48, 60 hours after start of treatment
Acepromazine - sedative: 0.025-0.25 mg/kg IV, IM, SC PRN; max 3 mg; 0.55-2.2 mg/kg PO
Activated Charcoal - GIT adsorbent: 1-5 g/kg (6-12 ml/kg) PO q2-4-6h; only 1st dose with sorbitol
Albuterol (Proventil, Ventolin) - bronchodilator: 0.02-0.05 mg/kg PO q6-12h; Nebulization 0.5% solution in 4 ml saline @ 0.1 ml/5kg
Aluminum Hydroxide (Amphogel, Basagel) - phosphate binder: 10-30 mg/kg (0.5-1.5 ml/kg) PO q8h
Aminophylline – bronchodilator: 5-10 mg/kg PO, IM, IV very slowly q8-12h
Amoxicillin: 10-30 mg/kg PO, IM, SC q8-12h
Amoxicillin-clavulanate (Clavamox, Augmentin): 12.5-25 mg/kg PO q8-12h
Ampicillin sodium: 10-40 mg/kg PO, IM, SC, IV q6-8h
Apomorphine - emetic: 1.5-6 mg in conjunctival sac; 0.04 mg/kg IV; 0.04-0.08 mg/kg IM, SC
Atropine: antiarrhythmic (sinus block, AV block, bradycardia) 0.02-0.04 mg/kg IV, IM, SC, PO q4-8h; CPR dose 0.05 mg/kg IV or 1 mg/kg intratracheal; organophosphate toxicity 0.2-2.0 mg/kg IV, IM, SC and give ¼ dose IV and remainder IM or SC PRN
Buprenorphine (Buprenex) – partial opiate agonist: 5-30 .g/kg (0.005-0.03 mg/kg) IM, SC, IV q4-12h
Butorphanol (Torbutrol, Torbugesic) – partial agonist opiate: 0.1-0.5 mg/kg IV q1-4h; 0.2-0.8 mg/kg IM, SC, PO q1-6h
Calcitonin (Calcimar) – treat hypercalcemia: 4-6 IU/kg SC, IM q2-12h
Calcium EDTA (Versenate) – 25 mg/kg SC q6h x 20 doses by making a 1% solution (1 gram Versenate in 100 mL D5W) and giving 2.5 mL/kg q6h for 20 doses
Carprofen (Rimadyl) – NSAID: 4 mg/kg IV, IM, SC once; 0.5-2.2 mg/kg IV, IM, SC, PO q12h
Cephalosporin – antibiotics: 1st gen 10-30 mg/kg IV, IM q4-12h and 22 mg/kg PO q8-12h; 2nd gen and 3rd gen check inserts
Chlorpromazine (Thorazine) – anti-emetic: 0.05-0.1 mg/kg IV q4-6h; 0.2-0.5 mg/kg IM, SC q6-8h
Cimetidine (Tagamet) – H_2 blocker: 4-10 mg/kg IV, IM, PO q6-12h
Ciprofloxacin (Cipro) – fluoroquinolone: 5-15 mg/kg PO q12h; 10-20 mg/kg PO q24h
Crystalloid Fluids (LRS, Normosol, PlasmaLyte, NaCl): shock 50-90 ml/kg 1st hour with re-evaluation q15 minutes for need; maintenance 40-60 ml/kg/day
D-Penicillamine – 7.5-27.5 mg/kg PO q6h x 7 days; repeat after 7 days if needed; other: for copper 10-15 mg/kg PO q12h; for lead 8 mg/kg PO q6h or 10-55 mg/kg PO q12h
Dexamethasone Sodium Phosphate – shock steroid (controversial): 2-8 mg/kg IV slow
Dextran 70 – colloid fluid: shock 10-20 ml/kg/day IV
Dextrose 50%: 0.25-2.0 ml/kg IV slow; 2.0 ml/kg PO to effect
Diazepam (Valium) – benzodiazepine, anticonvulsant: status epilepticus 0.5-3.0 mg/kg IV in increments of 5-20 mg to effect or 2.5-20 mg intratracheal; Preanesthetic 0.1 mg/kg IV; restraint 0.2-0.6 mg/kg IV
Dimercaprol (BAL, British Anti-Lewisite) – 2.5-5.0 mg/kg IM q4h x 2 days, then q12h; or 3-4 mg/kg IM q8h until recovery; if severe can give 5 mg/kg day 1 only
Diphenhydramine (Benadryl) – H_1 blocker, anti-histamine: 1-2 mg/kg IM, 2-4 mg/kg PO q8h
Dolasetron (Anzemet) – serotonin antagonist anti-emetic 0.6 mg/kg IV q24h
Enrofloxacin (Baytril) - fluoroquinolone: 2.5-15 mg/kg PO, IV, IM, SC q12-24h
Epinephrine 1:1000 – anaphylaxis: 0.01 mg/kg IV, IM; CPR 0.1-0.2 mg/kg IV or 0.2-0.4 mg/kg intratracheal; bronchodilation 0.02 mg/kg IV, IM
Ethanol 7% – Ethylene glycol treatment: 600 mg/kg IV load dose then 100-200 mg/kg/h IV
Ethanol 40%, 80 proof (alcohol)- Ethylene glycol treatment: 2.25 ml/kg PO q4h
Etodolac (EtoGesic) – NSAID: 5-15 mg/kg PO q24h
Famotidine (Pepcid) – H_2 blocker: 0.5-1.0 mg/kg PO, IV, IM q12-24h
Fentanyl – opiate agonist: 3-10 .g/kg IV q30-120 min or 5-10 .g/kg/hr CRI to effect

Furosemide (Lasix) - diuretic: 1-6 mg/kg IV, IM, PO q1-2h or q6-12h
Glycopyrrolate (Robinul-V) – anti-muscarinic: bradycardia 0.005-0.01mg/kg IV, IM; 0.01-0.02 mg/kg SC q8-12h
Hespan (Hetastarch) – colloid: 16-20 ml/kg IV, may repeat
Hydrogen Peroxide 3% - emetic: 1-2 ml/kg PO, max dose 30 ml; repeat ½ dose only once of no emesis in 15 minutes

Hypertonic saline 7.5%: 4-5 ml/kg IV over 2-5 minutes
Ketoprofen (Ketofen, Orudis-KT) – NSAID: 1-2 mg/kg IV initial, then 1 mg/kg IV, IM, SC, PO q24h max 5 days
Magnesium HCl (Milk of Magnesia) - cathartic, avoid if renal or CNS compromise: 10-150 ml PO
Magnesium sulfate (Epsom salt) - cathartic, avoid if renal or CNS compromise: 250-500 mg/kg PO
Maropitant (Cerenia) – central-acting anti-emetic: 1 mg/kg SC q 24h or 2 mg/kg PO q24h
Meperidine (Demerol) – opiate: 2-5 mg/kg IM, SC q2-4h
Meloxicam (Metacam) – NSAID: 0.1-0.5 mg/kg PO q24h
Meso-Dimercaptosuccinic acid (Succimer, DMSA, Chemet) – 10 mg/kg PO q8h x 10 days; give on empty stomach; may be given per rectum
Methocarbamol (Robaxin) **–** muscle relaxant: 44.4-222.2 mg/kg IV administer half dose then titrate to effect; do not exceed 330mg/kg/day; 44.4 mg/kg PO q8h 1st day then 22.2-44.4 mg/kg PO q8h
Metoclopramide (Reglan) **-** anti-emetic: 1.0-2.0 mg/kg/24 hours in a CRI
Midazolam (Versed) **–** benzodiazepine: 0.1-0.25 mg/kg IV, IM or 0.1-0.3 mg/kg/h CRI IV
Morphine – opiate agonist: 0.25-1.0 mg/kg IV slow q1-4h; 0.2-2.0 mg/kg IM, SC q2-6h; 0.3-3.0 mg/kg PO q4-8h
Naloxone - opiate reversal: 0.02-0.04 mg/kg IV; or 11-22 micrograms/kg SQ, IM, IV
Omeprazole (Prilosec) – proton pump inhibitor: 0.2-0.7 mg/kg PO q12h; 0.5-1.0 mg/kg PO q24h
Ondansetron (Zofran) – serotonin antagonist anti-emetic 0.1-1.0 mg/kg PO q12-24h
Oxyglobin (HBOC) **–** oxygen-carrier: 15-30 mg/kg IV
Phenobarbitol – barbiturate: seizure control 2-16 mg/kg IV repeated q30min to effect; 15-200 mg/animal IV to effect
Pentobarbitol – barbiturate: seizure control: 2-15 mg/kg IV to effect; 3-10 mg/kg/h IV CRI
Propofol – anesthetic: 3-6 mg/kg IV then 0.1-0.4 mg/kg/min
Ranitidine (Zantac) – H_2 blocker: 0.5 mg/kg IV q 12 h, or 0.5-2.0 mg/kg PO q 12 h
Saline Flush 0.9% - as needed for eye and wound irrigation
Sucralfate (Carafate) – GI protectant, ulcer treatment: 250-1000 mg PO q6-8h
Sodium Bicarbonate – alkalinizer: 1-2 mEq/kg IV q3-4h; add 3 mEq/kg to IV drip
Sodium Nitrite – cyanide treatment: 16 mg/kg IV slow, repeat in 30 minutes
Sodium sulfate (Glauber's salts) - 250-500 mg/kg mixed with 5-10 times as much water PO
Sodium Thiosulfate - cyanide treatment: 1.65 ml/kg 25% solution IV; 16 mg/kg IV slow, repeat in 30 min
Sorbitol 70% - cathartic: 4 g/kg (3 ml/kg) PO; may repeat in 2-4 hours; may cause nausea, cramping, vomiting
Tramadol – opiate: 1-2 mg/kg PO q12h, extreme pain 1-4 mg/kg PO q6h
Vitamin K_1 – 2.5-5 mg/kg SC load dose, 0.25-2.5 mg/kg SC, PO q12h (give with fatty meal)

C. Antidote Chart

Poison Category	Antidote
Acetaminophen	Acetylcysteine (Mucomyst)
Alcohols - Ethylene glycol, methanol, folinic acid	Ethanol, folic acid (Folvite), 4-methyl pyrazole (Fomepizole
Anticholinergics - Diphenhydramine, benztropine	Physostigmine (Antilirium)
Benzodiazepines	Flumazenil (Romazicon)
Beta-adrenergic blockers	Glucagon
Botulism	Botulinum antitoxin
Calcium channel blockers	Calcium chloride
Carbon monoxide	Oxygen
Carbon tetrachloride	Acetylcysteine (Mucomyst)
Chelating agents/cholinergics - Organophosphates, carbamates	Atropine, pralidoxime
Coumadin derivatives	Vitamin K1 (AquaMEPHYTON, Mephyton)
Cyanide	Amyl nitrate, sodium nitrite, sodium thiosulfate, methylene blue, hydroxocobalamin
Digoxin	Digoxin immune fab (Digibind)
Heparin	Protamine
Hydrofluoric acid	Calcium gluconate
Iron	Deferoxamine mesylate (Desferal)
Isoniazid	Pyridoxine (Aminoxin)
Jellyfish	No antivenin - remove remaining tentacles, apply topical hydrocortisone cream, acetaminophen or ibuprofen for pain sculpin. No antivenin-immerse the stung extremity in water that is as hot as can be tolerated without producing a burn, for 60 to 90 minutes
Lead	Edetate calcium disodium (calcium disodium versenate)

Opioids	Naloxone (Narcan), nalmefene (Revex), naltrexone (ReVia)
Organophosphates, anticholinesterases	Atropine, pralidoxime (2-PAM, Protopam)
Salicylates	Sodium bicarbonate (Neut)
Scorpion	No antivenin-treat with acetaminophen and Benadryl
Snake - Central American pit viper	Crotalidae antivenin
Snake - Copperhead	Crotalidae antivenin
Snake - Eastern coral snake	Elapidae antivenin
Snake - North American rattlesnake	Crotalidae antivenin
Snake - South American pit viper	Crotalidae antivenin
Snake - Texas coral snake	Elapidae antivenin
Snake - Water moccasin	Crotalidae antivenin
Spider - Black widow spider	Calcium gluconate, Latrodectus antivenin
Spider - Brown recluse spider	No antivenin-treat with pain medications
Stingray	No antivenin-immerse the stung extremity in water that is as hot as can be tolerated without producing a burn, for 60 to 90 minutes
Tricyclic antidepressants	Sodium bicarbonate (Neut)

Chemical	Antidote
Anthrax	Ciprofloxacin (Cipro)
Botulism	Trivalent equine antitoxin
Brucellosis	Doxycycline, ofloxacin
Lewisite	British anti-lewisite, dimercaprol (BAL in Oil)
Nerve agents - Hydrogen cyanide	Amyl nitrate, sodium nitrate, sodium thiosulfate, vitamin B12
Nerve agents - Sarin	Atropine, pralidoxime chloride (Protopam)
Nerve agents - VX gas	Atropine, pralidoxime chloride (Protopam)
Plague	Streptomycin, chloramphenicol, gentamicin, doxycycline
Q fever	Tetracycline, erythromycin, azithromycin (Zithromax)
Ricin-inhaled toxin	No antidote-supportive therapy, gastric lavage, activated charcoal smallpox cidofovir (Vistide)
Tear gas	No antidote-treatment consists of thorough flushing of eyes, bronchodilators, assisted ventilation, oxygen when necessary, soothing lotions and blister care for skin
Tularemia	Streptomycin, gentamicin
Vesicants - Sulfur mustard	No antidote-treatment consists of supportive care and keeping damaged organs free from infection

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