

Awareness-level information for veterinarians on weapons of mass destruction and preservation of evidence

James G. W. Wenzel, DVM, PhD, DACT, DACVPM

Weapons of mass destruction may be defined as chemical, biological, radiologic, nuclear, explosive, or incendiary devices intended to cause widespread injury or death.¹ Injuries and illnesses resulting from weapons of mass destruction may be overt, subclinical, or delayed. Because veterinarians may find themselves responding to emergency incidents, they should be aware of the agents that may cause mass casualties. Importantly, if such agents have been used intentionally, then the incident is a crime scene, and responders should be aware of the need to secure the scene and preserve evidence. If the incident was intended to intimidate or coerce for social or political reasons, then it is classified as an act of terrorism, and federal regulations and authorities have jurisdiction.¹ Specifically, animal enterprise terrorism has been defined as a crime that causes physical disruption to the functioning of an animal enterprise or economic damages exceeding \$10,000 to that enterprise.²

There is little published information on the effects of most weapons of mass destruction in animals,³ although some information has been published on possible dangers for search-and-rescue dogs working in postdisaster environments.⁴⁻⁸ Nevertheless, veterinarians may obtain some useful comparative information from published reports of the effects of weapons of mass destruction on humans and their treatment. Because of the potential for veterinarians to be involved in the response to an incident involving weapons of mass destruction, they should have at least some awareness-level information of the various agents that might be used.

Similarly, although law enforcement personnel are responsible for securing crime scenes, gathering and preserving evidence, and maintaining a documented chain of custody for such evidence, veterinarians should have at least some rudimentary knowledge of the principles and practices of evidence preservation because they may be involved in rendering aid at the site of a disaster or treating victims at the site or elsewhere.

Chemicals

Veterinarians are trained to recognize and treat many forms of chemical intoxication, including intoxication

with some agents that are commonly found in nature but can also be used in chemical weapons. Weaponized forms of these chemicals, however, are typically much more potent per unit volume than the natural form. Intentional chemical attacks are often only slowly recognized owing to a low index of suspicion. Common symptoms, including death, in humans, animals, and insects; a rapid onset of signs; and unusual clouds, vapors, or odors should raise the index of suspicion for chemicals.⁹

The responder community tends to classify chemical agents according to the signs or syndromes they cause as nerve agents (eg, organophosphates and carbamates), blood agents (eg, cyanide-containing chemicals), choking agents (eg, chlorine) and irritants (eg, tear gas and pepper spray), blister agents, and incapacitating agents (eg, potent anticholinergics meant to impair function without causing further harm).⁹

Nerve agents—The nerve agents include familiar organophosphates (eg, malathion) and carbamates (eg, propoxur), but also include so-called G (for German) chemicals originally developed as insecticides, including GA (tabun), GB (sarin), GD (soman), and GF.¹⁰ These nerve agents are volatile and, thus, may be inhaled, but generally do not persist in the environment. An organophosphate that does persist because of its viscous, oily nature is VX (V for venom and X for an earlier insecticidal designation).⁹ When weaponized, these agents are hundreds of times more potent than pesticides and can penetrate skin. The nerve agents inhibit the action of acetylcholinesterase and, thus, cause salivation, lacrimation, urination, defecation, gastroenteritis, emesis, and miosis.¹ Bronchoconstriction and bronchorrhea are also common and may be the leading cause of death. The onset of signs ranges from minutes to hours with these agents, but the response to treatment is typically rapid. Victims must be thoroughly decontaminated. Sodium hypochlorite (bleach) is a convenient chemical deactivator of the organophosphates, but must be properly diluted to avoid damage to skin. Unfortunately, appropriate dilutions for most animal species are not known. For humans, a 0.5% solution can be used to decontaminate skin. A 5% solution can be used to decontaminate equipment.¹¹ Treatment involves administration of atropine, pralidoxime chloride (except in carbamate intoxication), and diazepam; provision of supplemental oxygen; and aggressive removal of pulmonary secretions.⁹

From the Department of Clinical Sciences, College of Veterinary Medicine, Auburn University, Auburn, AL 36849.

Blood agents—The blood agents are classified as such by the responder community because of their apparent effects on RBCs. In general, these agents consist of various forms of cyanide that cause damage via anoxia of body tissues. Hydrogen cyanide and cyanogen chloride are toxic industrial chemicals that cause signs most veterinarians would recognize in animals that have consumed certain wilted plants, including *Prunus* spp (eg, cherry laurel), which contain amygdalin, a cyanogenic glycoside. Cyanide may also form as a result of combustion of certain natural and synthetic materials containing carbon and nitrogen. Weaponized forms would typically be gaseous. Venous blood and, sometimes, the skin of victims of cyanide toxicosis may be cherry red because of failure of the cells to extract oxygen from the blood. The resultant tissue hypoxia may cause progressive hyperpnea, ataxia, convulsions, coma, and death. Ingestion of or contact with liquid forms of the blood agents results in rapid absorption and development of signs. Signs occur more rapidly following inhalation; however, industrial cyanides tend to evaporate and dissipate quickly and, thus, have their greatest effects in confined spaces. Some people may detect the smell of bitter almonds when cyanide is present, but this odor may not persist as the agent dissipates.¹¹ Victims should be evacuated, decontaminated, and treated with amyl nitrite, if available, via inhalation, then with sodium nitrite by injection. These drugs cause dissociation of the cyanide, and subsequent treatment with sodium thiosulfate provides the substrate for conversion to thiocyanate, which is not toxic and is excreted by the kidneys. Hydroxocobalamin, or vitamin B12a, has also been used because it forms cyanocobalamin with cyanide, which is likewise excreted.⁹

Choking agents—The choking agents are also called pulmonary agents because the pulmonary system is the organ system most commonly affected. They are gases that combine with moisture in the respiratory tract to form strong acids or bases, causing damage that may result in life-threatening pulmonary edema. Clinical signs may not be evident for several hours to several days, and bronchitis and pneumonia are potential sequelae. The most highly water soluble agents (anhydrous ammonia) tend to affect the upper respiratory tract, and the least soluble (diphosgene) affect the alveoli. Oxides of nitrogen, hydrogen chloride vapor, some blister agents, and some products of combustion of tetrafluoroethylene may also reach the alveoli. Chlorine is intermediate in solubility and primarily affects the bronchi. Although phosgene was originally developed as a weapon, it is now, like the others, produced in large quantities for industrial or agricultural use. Thus, these agents pose risks from accidental releases or spills or as weapons of opportunity. Victims should be evacuated from contaminated areas. Secondary contamination of responders by victims is minimal, except potentially as a result of off-gassing of phosgene. Specific treatment following exposure to these agents is not available. Bronchodilators, oxygen, and positive end-expiratory pressure delivered via mask or endotracheal tube may be used as supportive measures. Diuretics have been of limited value, and disagreement exists as to the value of the use of steroids.^{1,9,11}

Blister agents—The blister agents are also called vesicants and mustards. They are usually oily liquids with odors of garlic, onion or mustard (sulfur mustard), fish (nitrogen mustard), or geraniums (lewisite); phosgene oxime is a solid at temperatures < 35°C (95°F).¹ All are persistent agents that produce vesicant vapors. Exposure of skin causes, progressively, pruritis, stinging or burning pain, erythema, edema, and formation of bullae. Ocular exposure may progress from conjunctivitis and blepharospasm to chemosis, corneal ulceration, corneal perforation, and blindness. Inhalation of vapors may cause only sinusitis or laryngitis, but can also cause sloughing of the upper respiratory tract epithelium, pseudomembrane formation, pneumonia, respiratory failure, and sepsis. Nausea and vomiting are reported in humans. Bone marrow suppression can occur, causing leukopenia first and, later, anemia and thrombocytopenia. Immediate decontamination (within 2 minutes) by thorough washing with dilute hypochlorite or alkaline soap is the best treatment, along with copious irrigation for ocular exposure. If the effects of pulmonary exposure are severe, ventilation and evacuation of pulmonary secretions may be indicated. Further treatment should be supportive, such as bronchodilators, analgesics, and fluid therapy, but should include antimicrobial administration, as infection of affected tissues is common. There are no antidotes, but antioxidants may be somewhat palliative.^{9,11}

Incapacitating agents—Many compounds have been studied for their ability to produce a nonlethal syndrome similar to that seen with high doses of atropine or scopolamine, but with small amounts whose effects are felt for longer periods of time. One such chemical is 3-quinuclidinyl benzilate, a persistent, crystalline solid. Signs peak about 8 hours after exposure and subside slowly over 2 to 3 days, with the most common signs being tachycardia, dry skin and mucous membranes, mydriasis and blurred vision, hyperthermia, odd behavior, hallucinations, and stupor. Physostigmine, pyridostigmine, or neostigmine will reverse the peripheral effects of 3-quinuclidinyl benzilate, but only physostigmine crosses the blood-brain barrier.⁹

Biological Agents

Like many chemical weapons, most bioweapons have been developed as aerosols to allow for efficient dissemination. Unlike chemicals, the microorganisms and toxins that make up bioweapons may have no detectable color or odor, and the onset of signs typically is delayed, with the result that victims may be some distance from the site of exposure at the time they develop signs. The CDC lists 21 agents or groups of related organisms as potential bioweapons.¹² Most of these are zoonotic, and many veterinarians will have had first-hand experience with at least some of them (eg, *Bruceella* spp). Several have been weaponized, with the only weaponized pathogen on the list that is not zoonotic being the virus that causes smallpox.

The first goals for the responder community during a disease outbreak, whether the outbreak was caused accidentally or intentionally, are detecting the outbreak itself and identifying the causative agent.¹³ Accredited

veterinarians should be familiar with their role in surveillance for foreign animal diseases and the requirements for notification of state and federal veterinary officers. By virtue of the animals they treat and the venues in which they work, veterinarians may be the first to encounter unusual or unexpected diseases.

Some of the characteristics of the agents or diseases of concern (**Appendix**) include availability or ease of acquisition, ease of dissemination or transmission, public health impact (high morbidity and mortality rates), means of control or treatment or lack thereof, and potential for social disruption or panic. Some of these may be modified naturally (eg, through genetic drift, shift, or reassortment) or artificially (eg, through recombinant technology or selection for resistance) or emerge from unrecognized reservoirs or in previously unaffected species. Regardless, recognition of unusual signs or circumstances and a heightened index of suspicion are initial steps in disease detection. Veterinarians in the United States should be cognizant of the potential for incursion or dissemination of foreign, emerging, and zoonotic diseases.

Radiologic and Nuclear Agents

Radiologic materials may bring about a mass casualty incident in several ways. Detonation of a large nuclear device seems unlikely given the time, skill, and resources required, but special atomic demolition munitions were produced in the former Soviet Union.¹ Accidents at or attacks on legitimate nuclear facilities also pose the risk of releasing radiologic materials. An overt attack that spreads radiologic materials via a conventional explosion (ie, an explosive dispersion device or dirty bomb) is considered a possible scenario for a terrorist attack. Radiologic materials might also be disseminated in a covert manner. In any case, veterinarians possess the knowledge and clinical skills to assist in the management of such incidents.

About 50% of the energy dissipated in the detonation of a nuclear device is in the blast wave. Although covering an area larger than that associated with conventional explosives, the blast wave of a nuclear device is otherwise similar. Thermal energy constitutes about 35% of the explosive energy of a nuclear device, so many victims of flash burns and flash blindness would be expected. Additional energy is expended in an immediate wave of radiation from the blast (5%), and the remaining 10% results in radiologic contamination of the environment. Neutron emission causes some fixed materials (eg, aluminum) and smaller, more mobile particles to become radioactive (fallout), resulting in the potential for delayed radiation toxicosis.¹⁴

In the absence of direct injuries from a nuclear detonation or explosive dispersion device, the risks are almost all related to delayed radiation toxicosis, regardless of the origin or size of the release. Avoiding or minimizing exposure, especially inhalation or consumption of contaminated materials, is the most obvious safety measure. Guidelines for limited exposure are 5 rem for all responders, 10 rem if substantial amounts of property are at stake, up to 25 rem if many people will be protected or saved, and > 25 rem only if large numbers of people are at risk and only for fully informed

responders on a voluntary basis.¹ In regard to potential radiologic exposure, less time means less absorption, exposure decreases as a function of the square of the distance from the source, and use of a barrier or shielding reduces the risk of exposure.

The most important treatment for those at risk of delayed radiation toxicosis is oral administration of potassium iodide within 4 hours of potential exposure. After 12 hours, this prophylaxis is of no value. Exogenous potassium iodide occupies thyroid iodine receptors, which blocks thyroid binding of radioactive iodine. Radioactive iodine is common in the vicinity of large radiologic events and is unwittingly internalized through inhalation or ingestion. The suggested daily dose of potassium iodide is 130 mg for adult humans and younger individuals weighing > 69 kg (152 lb), 65 mg for children and adolescents between 3 and 18 years old that weigh < 69 kg, 32 mg for infants 1 month to 3 years old, and 16 mg for neonates < 1 month old.¹⁵ Although appropriate dosages have not been established for animals, potassium iodide is relatively innocuous. Therefore, veterinarians may consider extrapolating from these dosages when treating exposed animals.

Treatment of injuries sustained from the immediate effects of a radiologic event (ie, injuries resulting from the blast wave and fireball, if applicable, and immediate radiation injuries) may proceed after the scene is secured or the victims are decontaminated and evacuated. Pharmaceutical strategies exist to hasten the elimination of some radionuclides.¹⁶ However, the capacity for providing immediate treatment may be limited in incidents involving mass casualties, and difficult decisions must be made in triage. Three relatively simple measures provide the highest prognostic capability: the percentage of burned body surface is inversely correlated with survival rate; the primary sign of radiation toxicosis is nonpsychogenic emesis, and shorter times from exposure to onset carry poorer prognoses; and a decrease in or extremely low blood lymphocyte count and the rapidity with which the decrease occurs are associated with increased mortality rate.

Explosive Devices

Explosives are categorized according to the speed with which their chemical reactions occur, which correlates with their rate of expansion. Low explosives (eg, gunpowder) undergo rapid burning, or deflagration, at < 1,000 m/s (3,300 feet/s), but cause an explosion if confined. High explosives, on the other hand, produce explosions without confinement at speeds of 1,000 to 9,100 m/s (3,300 feet/s to 5.6 miles/s) through the process called detonation.¹⁷ One characteristic of the resultant blast wave, which is also known as the positive phase impulse, is brisance, or shattering ability.^{17,18}

The potential for injury from detonation, in particular, varies according to the size of the explosive charge, the distance from it, the medium in which it occurs (air, water, or earth), and the location of nearby reflective surfaces. Peak overpressure decreases as a cubic function of increasing distance from the source. Water and soil transmit energy further and more quickly because they cannot be as easily compressed and, thus, cannot

dissipate force as easily as can air. Precautions are indicated to avoid injury as a result of loss of structural integrity occurring via ground shock. Objects, including bodies, near solid surfaces (walls) receive more damage because of reflection of the blast wave, and in corners, the effects are exponentially worse. The pressure wave is followed by a less forceful vacuum phase called the negative phase impulse.¹⁸

The blast wave can tear or otherwise disrupt tissues. Gas-filled spaces may implode during the positive phase impulse and explode during the negative phase impulse. On a submicroscopic level, molecules (eg, water) may be driven from areas of high density (tissue) to areas of low density (eg, pulmonary alveoli) in a process called spalling. Missiles (flying objects) produced by the blast may injure victims through blunt or penetrating trauma, or the victims may be propelled into other objects. Flash burns occur in those closest to the device during detonation.¹⁸

Injuries relatively unique to blast victims include several sorts of trauma to the pulmonary system, collectively known as blast lung. Gross and microscopic damage resulting in intrapulmonary hemorrhage and hemothorax, hemothorax or pneumothorax leading to hyperpnea, and traumatic emphysema with wheezes are some of the possibilities. Arterial air embolism is a possible sequela if communication occurs between the pulmonary vasculature and any air spaces. Supplemental oxygen is indicated for all of these conditions. However, because of the possibility of air emboli, positive-pressure ventilation must be carefully considered and controlled. Rupture of the bowel or other viscus may be immediate or delayed as a result of failure of a compromised section. In attempting to communicate instructions, first responders must remember that tympanic membrane rupture is common among blast victims. Other auditory system damage may cause vertigo or tinnitus.¹⁸

The Postdisaster Environment

The postdisaster environment poses various risks to humans and animals,^{4,5} particularly dogs working at scenes of urban disasters.⁶⁻⁸ The most common injuries among dogs working at the scene of the 1995 Oklahoma City bombing involved the feet, and such injuries were more common among dogs used for searching than among dogs used for patrolling or detection of explosives. Behavioral changes in some animals were also reported by handlers.⁴ These hazards were shared by working dogs at the site of the 2001 collapse of the World Trade Center. Evacuation, protective footwear, adequate rest and recreation, rehydration therapy, and decontamination were the treatments most commonly mentioned.^{4,5}

Toxicologic hazards at sites of urban disasters include solids, liquids, particulates, and gases, such as hydrocarbons, polychlorinated biphenyls, hazardous metals, cleaning products, ethylene and propylene glycol, other alcohols, and phenols. Potential routes of exposure include respiratory, dermal, oral, and ocular.⁶ Knowledge of the agents and routes of exposure would be valuable to veterinarians in the prevention and treatment of potential intoxication.⁶⁻⁸

Preservation of Evidence

First responders to potential crime scenes, especially those involving weapons of mass destruction, should be aware that some types of criminals are increasingly using multiple devices, that is, 2, 3, or more devices designed to attack successive waves of responders. At the scene, therefore, responders must protect themselves from risks posed by the primary device or harmful agent, recognize and avoid additional devices, and accomplish their goals of evacuation and treatment while trying to disturb the scene as little as possible and preserve potential evidence. Early responders may notice important things about the crime scene, such as movement of people or vehicles, and this should be recorded and reported. Investigators should try also to obtain statements from victims, including those who may be dying, and other witnesses to the incident. Criminal investigators will try to videotape, photograph, or record in some way all of the contents of the scene and their positions. A goal of crime scene preservation is to change as little as possible until this is done, but providing first aid to and evacuating the injured and making the scene safe must be accomplished first.¹⁹

While decontaminating, evacuating, and treating victims, personnel should collect and seal items from the scene in appropriate containers. These should be labeled, catalogued, decontaminated if necessary, and signed over to investigators in such a way as to maintain a verifiable chain of custody. Responders may request accompaniment by or communication with investigative personnel to obtain advice on collection of such evidence. If sufficient personnel are available, investigators will often accompany victims through treatment to record statements and obtain physical evidence.¹⁹ Medical personnel can gather another sort of evidence by recording and reporting lesions and their locations on victims and, perhaps, by obtaining the cause of such lesions (eg, shrapnel). If no investigators are present to take possession of such items, the items should be documented and preserved with a clear chain of custody.

Veterinarians who might be asked to gather evidence (eg, biological or toxicologic samples) for legal proceedings should be aware that most jurisdictions have exacting procedures for gathering evidence to make it most defensible in court. Details of practices such as packaging duplicate samples and control samples, applying labels and tamper-proof seals, packaging, and cataloguing may differ, so assistance and instruction should be obtained from the appropriate authorities.¹⁹

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Appendix

Diseases and agents of concern because of their potential for use in bioweapons.

Anthrax (<i>Bacillus anthracis</i>)
Botulism (toxin of <i>Clostridium botulinum</i>)
Brucellosis (<i>Brucella</i> spp)
Cholera (<i>Vibrio cholerae</i>)
Cryptosporidiosis (<i>Cryptosporidium parvum</i>)
<i>Escherichia coli</i> O157:H7
Emerging diseases (eg, Nipah virus and hantavirus)
Epsilon toxin of <i>Clostridium perfringens</i>
Glanders (<i>Burkholderia [Pseudomonas] mallei</i>)
Influenza
Melioidosis (<i>Burkholderia [Pseudomonas] pseudomallei</i>)
Plague (<i>Yersinia pestis</i>)
Psittacosis (<i>Chlamydophila psittaci</i>)
Q-fever (<i>Coxiella burnetii</i>)
Ricin toxin from castor beans (<i>Ricinus communis</i>)
Rift Valley fever
Salmonellosis
Severe acute respiratory syndrome
Shigellosis (<i>Shigella</i> spp)
Smallpox (variola major)
Tularemia (<i>Francisella tularensis</i>)
Typhoid fever (<i>Salmonella typhi</i>)
Typhus fever (<i>Rickettsia prowazekii</i>)
Viral encephalitis (<i>Alphavirus</i>)
Viral hemorrhagic fevers (eg, Ebola, Lassa, and Marburg)