Chemical Agents of Terrorism: Preparing Nurse Practitioners

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Since September 11, 2001, the world has experienced continual threats of terrorism, witnessed the use of an incapacitating agent on terrorists and hostages in Moscow, followed news reports of the discovery of the nerve agent sarin in a train station in Paris, and watched the search for weapons of mass destruction in Iraq. First utilized during World War I, weaponized chemicals are used to accomplish military and political gain. In the hands of terrorists, chemical weapons create fear and panic, gain publicity, and disrupt government and health care systems.[1,2] Whether from the deployment of weapons of mass destruction, isolated terrorist use, or accidental exposure, clinicians must prepare to manage the effects of chemical agents.

Nurse practitioners must exercise vigilant readiness to properly care for victims of chemical injuries. In this article, appropriate clinical management of each category of chemicals is addressed, including supportive and pharmacologic care. Triage decisions are explained and decontamination concerns are identified. Attention is directed at special populations such as children and the elderly.

Chemical Agents

Chemical agents vary and are viewed by their structure and/or physical effects on victims. An example of chemical agents identified by chemical structure and effect are organophosphates, also known as nerve agents because of their effects on the central nervous system. Most scientific sources of agent identification utilize agent index numbers, class index numbers, the agent name, and chemical structure. [3]

Nerve Agents

Nerve agents are an extremely toxic group of organophosphate compounds first manufactured and utilized in 1917.[4] All nerve agents are liquids at room temperature with the capability of producing vapor that penetrates the skin, respiratory epithelium, and cornea. The liquid is absorbed through intact skin or through the gastrointestinal tract after ingestion of contaminated food.[5] Nerve agents likely to be seen in a chemical attack include "G" agents—tabun (GA), sarin (GB), soman, (GD), cyclosarin GF, and more persistent V-agents known as VX. Detecting the presence of nerve agents is extremely difficult as those freshly mixed are clear and colorless liquids. Liquid agents are heavier than water and their vapor is heavier than air, allowing them to sink into low terrains and basements. [6]

Nerve agents affect communication between nerves and organs.[6] Acetylcholinesterase is inhibited resulting in accumulated acetycholine at neural junctions. The stimulus of cholinergic conduction at the central, nicotinic, and muscarinic receptors leads to rapid onset of rhinorrhea and excessive salivation. Many substances have the same effects as nerve agents including the therapeutic drugs such as physostigmine sali-

cylate (Antilirium), neostigmine bromide (Prostigmine), and pyridostigmine bromide (Mestinon), and certain pesticides such as carbaryl (Sevin) and O-dimethyl-S-dithiophosphate (Malathion).[7]

Droplet exposure results in symptoms that may not appear for 30 minutes to 18 hours. The time frame for appearance of symptoms is determined by the amount of exposure to the toxin, winds, and humidity. One drop can cause sweating and twitching and may progress to nausea, vomiting, diarrhea, loss of consciousness, convulsions, respiratory arrest, and flaccid paralysis.[6] Large exposures result in increased likelihood of death.

Vaporized nerve agents are the most toxic to humans. Symptoms appear within *seconds* if the dosage is large and concentrated. Smaller dosages of vapor and prolonged exposure result in symptoms within *minutes* and produce immediate symptoms such as miosis, injected conjunctiva, eye pain, blurred vision, nausea, vomiting, rhinorrhea, increased salivation, chest tightness, shortness of breath, and cough.[6] Large exposures produce loss of consciousness, convulsions, flaccid paralysis, and respiratory arrest in less than 1 minute. The half-life of the agent in soil may be from 2 hours to greater than 15 days.[3]

Diagnoses and treatment are based on observations of signs and symptoms regardless of cholinesterase levels.[7] Treatment for nerve agent poisoning involves the use of three therapeutic drugs: atropine, pralidoxime, and diazepam.[8]

Cyanide

Cyanide agents, or blood agents, are compounds that stop the transfer of oxygen from the blood to the rest of the body by inhibiting cytochrome oxidase.[3] The cell dies with the oxygen remaining in the blood stream causing a very red appearance of the blood. Victims will not be cyanotic but may have a flushed appearance of the skin.

Two forms of cyanide with United States military designations are hydrogen cyanide and cyanogen chloride.[3] Cyanide is found in many common foods such as lima beans and the seeds of apples. In the U.S., it is produced in massive quantities each year for use in the manufacturing of paper, textiles, and plastics. Burning plastics and other burned synthetic material also produce cyanide.[6]

Mild symptoms appear after brief exposure to low concentrations, and victims experience rapid relief of symptoms within moments of exposure to fresh air. In moderate amounts, cyanide produces nausea, dizziness, weakness, and anxiety. Large concentrations, even with brief exposure, produce loss of consciousness within seconds followed by convulsions and respiratory arrest. To differentiate cyanide agents from nerve agents, victims with cyanide convulsions have normal to large pupils or muscular fasciculations.[6]

Chemical Matrix Indicator

	A	B	C	D	E
Appearance					
Prostration		NA		NA	NA
Twitching		NA		NA	NA
Convulsions		NA		NA	NA
Coma		NA		NA	NA
Bleeding from mouth		NA		NA	NA
Coughing		NA			
Sneezing		NA		NA	
Vomiting				NA	
Fasciculations (muscle twitching)		NA	NA	NA	NA
Skin					
Cyanosis (skin is blue or purple)		NA		NA	NA
Gray area of dead skin	NA		NA	NA	NA
Pain, irritation	NA				
Clammy		NA		NA	NA
Sweating, localized or generalized		NA	NA	NA	
Eyes					
Small pupils		NA	NA	NA	NA
Normal, large pupils					
Involuntary closing	NA	NA		NA	
Tearing		NA			
Burning, irritation	NA				
Headache, pain around eyes		NA	NA	NA	NA
Dim vision		NA	NA	NA	NA
Blurred vision		NA	NA	NA	NA
Burning pain in eyes	NA				
Redness		NA	NA	NA	
Respiratory					
Coughing		NA			
Runny nose		NA			
Tight chest (shortness of breath)		NA			
Burning, irritation in nose	NA				
Cardiovascular					
Slow heart rate		NA	NA	NA	NA
Fast heart rate		NA			
Digestive System					
Defecation		NA		NA	NA
Nausea				NA	
Total					
Total Indicators					

To Use the Agent Indicator Matrix: For each symptom that is present place a check mark in the open box. Symptoms that are absent for a particular agent have the box unavailable to check. Total the number of check marks in each column.The column with the highest number of indicators represents the most likely agent utilized.

A = Nerve Agents
B = Blister Agents
C = Cyanide
D = Pulmonary Agents
E = Riot Control Agents

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Hydrogen cyanide, an industrial fumigant, is lighter than air with great potential to dissipate across large areas. It has a bitter, almond-like odor. Cyanogen chloride, a cyanide metabolite, is heavier than air and settles in low-lying places. Exposure results in immediate irritation to the eyes and upper airway.

Differentiating cyanogen chloride from riot control agents is difficult. Victims exposed to large doses of cyanogen chloride convulse within seconds. If exposure to cyanogen chloride is small, upper airway irritation may be the only effect and no further action is necessary.[6] With proper treatment, persons surviving an initial exposure will recover within hours.[9] Treatment for cyanide poisoning is nasal inhalation of amyl nitrate with subsequent treatment of IV sodium nitrite and IV sodium thiosulfate.[8]

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Two blood agents not classified as military agents are arsine and hydrogen sulfide. Both are colorless gases and rapidly decompose in light, heat, and contact with various metals. In gas form these agents are heavier than air and accumulate in low-lying areas or poorly ventilated regions. Arsine has a strong odor of garlic while hydrogen sulfide has a strong odor of rotten eggs. The odor of these gases can be detected at levels of less than one part per million, but the sense of smell is paralyzed at higher levels.[10] Inhaled, these two agents are highly toxic.

The mechanism of action of hydrogen sulfide is similar to carbon monoxide poisoning. Treatment consists of moving victims to fresh air. If ventilatory and/or hemodynamic support is required, the potential for recovery is minimal.

Arsine does not interfere with enzyme activity but actually enters into the blood cell, leading to hemolysis. Initial symptoms include headache, malaise, thirst, abdominal pain, dyspnea, and shivering. Onset of symptoms occurs within 30 minutes to 24 hours with large exposures. Victims will have hemoglobinuria within a few hours and jaundice within 2 days.[11]

Incapacitating Agents

Incapacitating agents are not meant to kill, but interfere with the victim's ability to function by inhibiting acetylcholine and thus allowing more lethal agents to be used without resistance. Incapacitating agents are classified as depressants or stimulants. Two recognized incapacitating agents are 3-quinulidinyle benzilate (BZ) and Agent 15 (a glycolate anticholinergic).[2,3]

Victims are exposed through inhalation, ingestion, or skin absorption producing symptoms similar to high dose atropine. Symptoms may begin immediately or be delayed for days with a median onset between 30 minutes and 4 hours. Symptoms include blurred vision, feeling hot, and dry mouth. Peripheral effects can be summed up by the pneumonic "dry as a bone, hot as Hades, red as a beet, and blind as a bat." Decreased secretions in the oral pharynx, GI tract, and eccrine and apocrine glands make victims "dry as a bone". "Hot as Hades" refers to hyperthermia from decreased sweating. Cutaneous vasodilatation, resulting from compensatory attempts to regulate the body's temperature, defines "red as a beet", and "blind as a bat" refers to the mydriasis and inability of the pupils to accommodate.[12] Mental status changes follow peripheral changes including delirium, impaired consciousness, erratic behavior, difficulty with speech, and poor coordination. Victims may also display clawing or climbing motions. The two greatest risks to victims are hyperthermia and mental changes.[2,12] The antidote for BZ or Agent 15 is physostigmine (eserine sulfate, Antilirium).[2]

Vesicant/Blister Agents

Weaponized vesicants include nitrogen mustards, sulfur mustards, phosgene oxide, and lewisite (an arsenic compound). Vapors from these toxic liquids have distinctive odors such as onions, garlic, and mustard for sulfur mustards; phosgene smells like moldy hay; and lewisite has the scent of geraniums. Inhaled vesicants result in burning pain, nasal irritation, sneezing, and interstitial fluid shifts leading to pulmonary edema.[13]

Blistering agents are inhaled, ingested, and absorbed through skin or eyes. When skin is exposed, stinging pain occurs immediately. Mustard agent symptoms may not develop for up to 24 hours. Erythematous areas of skin appear with large thin walled blisters. Burns are usually partial thickness, but in the groin or axilla burns can be deeper.[6,13]

Eye exposure to blister agents results in immediate symptoms including tearing and eye pain from corneal and conjunctival edema. Permanent damage ranging from corneal scarring to blindness can occur. Ingestion of blister agents results in nausea, vomiting, hypotension, and bradycardia. Exposure to large amounts of blistering agents causes bone

marrow depression and possibly death as a result of immunosuppression and respiratory compromise.[3,5,6]

When the patient suffers from mustard exposure, pulmonary care is supportive, including tracheostomy, oxygen therapy, and mechanical ventilation. Skin lesions are treated as burns with intravenous fluid replacement, IV antibiotics, pain relief, debridement, and topical treatment with silver sulfadiazine. Eye injuries are irrigated and treated with ophthalmic anticholinergic drugs and antibiotic eye ointment.[2,6,14]

Phosgene poisoning requires supportive therapy for pulmonary complications. Fluid restriction, close monitoring of chest x-rays, and arterial blood gases are indicated. As pulmonary edema progresses, ventilatory management may be needed. Lewisite toxicity is treated with British AntiLewisite (BAL), skin care, and ophthalmic ointments.[2,6,14]

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Pulmonary/Choking Agents

Pulmonary or choking toxins (phosgene, chlorine, and bromine) damage alveolar membranes leading to pulmonary edema. Vapors from phosgene and diphosgene have the scent of freshly cut grass or mown hay, while chlorine gas smells like bleach. Exposure in massive amounts can lead to corrosive skin and eye injuries.[13]

Effects of exposure can be delayed 2 to 24 hours. Rapid onset of symptoms indicates a poor prognosis. Symptoms seem minor initially and include blurred vision, tearing, shortness of breath, coughing, sore throat, wheezing, and the appearance of a common cold. Later, symptoms increase with chest tightness, pain, dyspnea and tachypnea, which may first appear to be anxiety.[3,5,6,13]

Pulmonary symptoms are aggressively treated with oxygen, bronchodilators, ventilatory support, and hemodynamic monitoring. Secondary bacterial infections are treated with antibiotics. Bedrest is mandatory to lessen complications. Patients with bronchospasm or history of asthma may benefit from corticosteroids.[2,6,14]

Riot Control Agents

Riot control agents are the least likely to be used in chemical terrorism, but they are classified as chemical weapons and addressed by the Centers for Disease Contral and Prevention (CDC) as such. Most likely, they would be used to cause confusion, disperse crowds, and perhaps allow other weapons to be used. Riot control weapons cause temporary eye pain, mucous discharge from the nose, coughing, labored breathing, and stinging of the skin. Pepper spray and tear gas are the most common and are dispersed into the air as aerosols or powders. Symptoms appear very rapidly (20 to 30 seconds) and usually improve quickly after the victim is placed in a fresh air environment for 5 to 10 minutes. High skin exposures may produce dermatitis and blistering.[15] Persistent redness can be treated with water irrigation, lotion (calamine), or topical steroid agents, according to the severity. Blisters should be left intact and treated as second-degree burns.[16] Permanent damage from these agents is unlikely if victims receive prompt treatment. Antidotes for these agents are not known.[15]

Triage

Triage decisions are based on the severity of the exposure, the amount of staff and resources available, and the number of casualties involved. Patients are triaged as *immediate, delayed, minimal,* or *expectant.* Victims triaged as *immediate* require lifesaving procedures if they are to survive. Those triaged *delayed* are severely injured, however, treatment can be postponed until they can be transported to an acute care facility without an adverse effect. Victims with minor injuries are classified *minimal,* while those not likely to survive as *expectant* (see Table: "Triage for Chemical Exposure").[2] If unknown, clinicians may quickly identify the category of the toxic agent used by observing the physical effects victims are experiencing (see Table: "Chemical Matrix Indicator").[6]

Decontamination

Clinicians must approach contaminated sites with caution and be prepared to self decontaminate. Secondary explosions/devices may cause additional contamination or trauma and personal protective equipment (PPE) must be worn at all times until directed otherwise. If the chemical has been dispersed by vapor, those exposed should move upwind to an area with a downwind breeze. If the vapor was released in a building, the intake ducts and vents should be closed with air exhausted out. Liquid or solid chemicals require further decontamination. Obvious remaining chemicals should be scraped off with an object such as a stick, edge of a book, or blunt side of a knife.[6]

Clothing should be removed and the skin washed with a 0.5% chlorine solution for 10 minutes. In the absence of a

chlorine solution, absorbent materials such as dry powders, dirt, and flour can be used to reduce the amount of material absorbed. The dry powder should be removed with wet tissues. Fresh water and seawater are reasonable options in the absence of a chlorine solution. In the absolute absence of 0.5% chlorine solution, bleach may be used if rinsed within 10 minutes.[6] For victims exposed to blistering agents, the skin may be so damaged that soap and water are preferable. Removing a victim's clothing can lessen contamination 80% to 90%.[7]

Decontamination of pulmonary toxins involves moving victims to a fresh air environment, eliminating contaminated clothing, and washing skin with large amounts of water.[17]

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Special Populations

Implications for Pediatrics

Pediatric concerns focus on issues of respiratory distress, skin integrity, body surface area to mass ratio, seizures, psychological impact, and correct dosages for antidotes or other medications.[3,6,18] Careful consideration of normal vital signs for infants and small children will be necessary. Short stature puts children closer to the ground where the greatest concentration of chemical is, leading to greater exposure. Primary management for respiratory failure includes supplemental oxygen, positioning, decreased energy expenditure, stimulation, suctioning, and early intubation.[18,19,20]

Vesicant agents quickly penetrate newborn skin resulting in blistering effects at smaller doses. Children's increased body surface area requires complete decontamination even if vapor exposure is the only hazard. Radiant warmers lower risk for hypothermia during decontamination. Avoid scrubbing to preserve skin integrity. Agents causing nausea and vomiting increase the risk for dehydration requiring close monitoring and vigorous treatment.[3,6,18]

Seizure management includes phenobarbital, lorazepam and diazepam. Compared to diazepam, intravenous lorazepam has a longer duration of action and causes less respiratory distress in children over 2 years of age. Rectal diazepam may be used in the absence of venous access; however, proper airway support must be available as children receiving rectal valium frequently experience severe respiratory suppression. Intranasal Versed may be used.[19,21]

Psychological trauma for children will be great. Incomplete concepts of body integrity, ill-defined concepts of death,

Triage for Chemical Exposure [2,6,27-29]

Triage Level	Immediate	Minimal	Delayed	Expectant
Injury				
Eye Injury		X	X	
Burns < 20% of Body Surface		X		
Burns > 20% of Body Surface	X			
Burns of Body Surface > 50%				X
Lesions < 50% of Body Surface		X	X	
Slight Respiratory Irritation		X	X	
Mild Dyspnea			X (Retriage hourly)	
Pulmonary Toxin Exposure No Symptoms		X (Retriage every 2 hours)		
Severe Respiratory Distress/ Pulmonary Edema	X			
Severe Pulmonary Edema/ Cyanosis/Hypertension				X

Pharmacologic Management of Chemical Exposure [2,3,6,7,21]

Drug	Indication	Action	Adult Dose	Pediatric Dose
Atropine	Nerve	Blocks cholinomimetic effects of organophosphates	1-6 mg I.V. May repeat every 5-60 minutes until atropinization. Also administer pralidoxime.	0.05 mg/kg May repeat every 10 to 30 minutes until atropinization. Also administer pralidoxime
Pralidoxime [2-PAM chloride, 2-pyridine aldoxime methochloride] (Protopam)	Nerve	Reactivates cholinesterase that has been inactivated by phosphorylation due to exposure to organophosphates	1-2 g I.V. in 100 ml of 0.9% over 15-30 minutes. Repeat in 1 hour if muscle weakness continues. Administer concurrently with atropine.	20-50 mg/kg/dose I.V. in 100 ml of 0.9% over 15-30 minutes. Repeat in 1 hour if muscle weakness continues, then at 10 to 12 hours if symptoms recur. Administer concurrently with atropine
Amyl Nitrate (Cyanide Kit)	Cyanide (Step 1)	Converts hemoglobin to methemoglobin, which reacts with cyanide to form cyanmethe-moglobin.	0.3 ml by inhalation for 15-30 seconds; repeat every 60 seconds until sodium nitrite and sodium thiosulfate available	0.3 ml by inhalation for 15-30 seconds; repeat every 60 seconds until sodium nitrite and sodium thiosulfate available.
Sodium Nitrite (Cyanide Kit)	Cyanide (Step 2)	Combines with hemoglobin to form methemoglobin.	300 mg in 10-ml solution given I.V. over 10 min. If symptoms reappear 1/2 the dose may be repeated in 2 hours.	Hemoglobin (Hgb) Dependent Hgb 7 – 5.8 mg/kg Hgb 8 – 6.6 mg/kg Hgb 9 – 7.5 mg/kg Hgb 10 – 8.3 mg/kg Hgb 11 – 9.1 mg/kg Hgb 12 – 10.0 mg/kg Hgb 13 – 10.8 mg/kg Hgb 14 – 11.6 mg/kg
Sodium Thiosulfate (Cyanide Kit, Versiclear)	Cyanide (Step 3)	Provides extra sulfur group to rhodanase, increasing the rate of detoxification of cyanide	12.5 g in 50 ml solution given over 10 min. May repeat one-half the dose in 2 hours if symptoms reappear	Hemoglobin (Hgb) Dependent Hgb 7 – 0.95mL/kg Hgb 8 – 1.10mL/kg Hgb 9 – 1.25 mL/kg Hgb 10 – 1.35 mL/kg Hgb 11 – 1.50 mL/kg Hgb 12 – 1.65 mL/kg Hgb 13 – 1.80 mL/kg Hgb 14 – 1.95 mL/kg
Physostigmine salicylate (Antilirium) Physostigmine sulfate (Eserine, Isopto Eserine)	Incapacitating	Inhibits destruction of acetylcholine by acetylcholinesterase	0.5 – 2 mg I.V. slowly over 2-3 minutes. Repeat every 20 minutes as needed. Repeat 1-4 mg dose if life-threatening symptoms reappear.	0.01-0.03 mg/kg/dose slow I.V. over 2-3 minutes. May repeat in 15 minutes. Maximum dose 2 mg.
British Anti-Lewisite (BAL, dimercaprol)	Vesicants	Combines with lewisite ions to form excretable chelates	3 mg/kg IM every 4 hours x 2 days, then every 6 hours on the 3rd day, and every 12 hours for 10 days.	3 mg/kg IM every 4 hours x 2 days, then every 6 hours on the 3rd day, and every 12 hours for 10 days.

and procedural fear cause children to cry inconsolably, resist decontamination, attempt to run away, or experience nightmares and anxiety postdecontamination. Children are susceptible to acute and post traumatic stress disorder requiring ongoing assessment.[18,22]

Implications for Older Adults

Older adults (usually defined as over age 65) are particularly vulnerable to chemical agents due to changes related to aging, the presence of comorbidities, and a lack of physiologic reserves to recover from sequelae. Consider age-related changes of the respiratory system when providing airway management and ventilatory support. When ordering drugs for older adults, changes in metabolism and excretion need to be considered because confusion, with or without cognitive impairment, is likely.[23]

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Mental Health Concerns

Terrorism, the trafficking in fear and terror, involves violence upon unsuspecting noncombatants. Normal experiences cannot prepare one for the horror. It destroys one's sense of security and, in some instances, an important place or even a loved one. One's world and values are threatened so that nothing is ever the same again.

Normal reactions include a range of powerful feelings. Victims often fear the terrible pain will never cease. Those attempting to aid victims must realize intense reactions affect the physical, emotional, cognitive, and interpersonal realms of experience. Emotional numbness, shock, anger, and difficulty experiencing positive feelings are prevalent. Physical reactions include fatigue, increased startle response, insomnia, and reduced immune response. Cognitive responses such as impaired concentration and decision-making, decreased memory, confusion, and nightmares are common. People withdraw, become isolated and suspicious, are overprotective, and suffer impaired work performance or increased conflicts with others.[24]

Emotional reactions occur in phases. The Impact Phase occurs during the disaster. People act disorganized, fearful or stunned, and try to protect the lives of themselves and others. In the Post-Disaster Phase, victims experience numbness, flashbacks, denial, and anger. Frequently, there is elation at survival despite the death of others, leading some to feel guilty. Recovery begins with elation related to altruism and rescue attempts. Disillusionment may follow when the disaster leaves the front page. People often feel unsatisfied but hesitate to express distress in the face of offered help. Strong emotional reactions present as sleep disturbance, GI tract difficulties, or relationship and work difficulties.[25]

There are numerous reactions to traumatic events, including the debilitating condition known as posttraumatic stress disorder (PTSD).

The significant criteria include: exposure to a traumatic stressor, re-experiencing symptoms of avoidance or numbing, re-experiencing the event, significant distress or impairment of function, and the duration of symptoms for a minimum of 1 month.[26]

Evidence suggests symptoms of stress that occur almost immediately following trauma may predict the development of PTSD. This is commonly called Acute Stress Disorder (ASD). Important symptoms include: 1) dissociation, 2) intrusive memories and /or thoughts, 3) avoidance and arousal symptoms, 4) the occurrence of the symptoms between 2 days and 4 weeks. When a diagnosis of ASD is made, the possibility of PTSD developing is considered. [26]

Conclusion

Preparing to care for victims of chemical terrorism is no longer an option for clinicians. Events within the last 2 years indicate no community can consider themselves risk-free. In the event a chemical attack becomes reality, the local health care providers and community must be prepared to respond appropriately. NP

DISCLOSURE

The authors have disclosed that they have no significant relationship or financial interest in any commercial companies that pertain to this education activity.

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CE Test

Chemical Agents of Terrorism: Preparing Nurse Practitioners

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