

Responding to the Threat of Weapons of Mass Destruction Part III & IV



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FACULTY MEMBERS

Author

Steven Cordas, D.O., M.P.H.

Clinical Assistant Professor, University of North Texas Health Science Center at Fort Worth
Bioterrorism Consultant, Texas Department of Health
Hurst, Texas

Consultants

Ronald Blanck, D.O. (Lt. Gen., Ret.)

Former Surgeon General of the United States Army
President, University of North Texas Health Science Center at Fort Worth
Fort Worth, Texas

Alexia (Alex) Hathaway, M.D., M.P.H.

Chair, BioDefense Council of the Tarrant County Medical Society
Serving as Tarrant County Public Health Authority
Fort Worth, Texas

Thomas Kurt, M.D., M.P.H.

Clinical Professor of Medicine
University of Texas Southwestern Medical School
Dallas, Texas

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Those completing this activity will receive information that should allow them to...

- List blister gasses, cyanogens and additional chemical and biological agents likely to be used in a terrorist attack;
- Describe the toxicology of each agent
- Identify clinical aspects relative to each agent; and
- Describe treatment protocols for individuals exposed to likely agents.

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EDITORS

Nicole King
Managing Editor
Tarrant County Physician
Tarrant County Medical Society

Andrew Crim
Assistant Director
Professional & Continuing Education
University of North Texas Health Science Center at Fort Worth

Pam McFadden
Assistant Vice President
Professional & Continuing Education
University of North Texas Health Science Center at Fort Worth

Responding to the Threat of Weapons of Mass Destruction

Part III Blister Gases and Cyanogens

Stevan Cordas, DO, MPH
Ronald Blanck, DO (Lt. Gen. Ret)
Alexia Hathaway, MD, MPH
Thomas Kurt, MD, MPH

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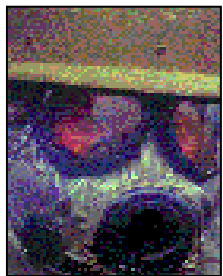
Responding to the Threat of Weapons of Mass Destruction

Part III Blister Gases and Cyanogens

Section one covered nerve gasses. Blister gases and cyanide will be covered here since we know that terrorists and countries that harbor terrorists possess blister gases.

According to the Department of Defense, the only official chemical casualty in the Gulf War was from a soldier, PFC David Fisher of the 3rd Armored Division, who entered a captured bunker and sustained skin burns after a lapse of several hours. Testing revealed evidence of blister gas. The soldier was treated and did not suffer any time off work, but did have some residual scarring.

Cyanide will be covered because there have been attempts to add cyanide to community water sources and it is one of the more common agents employed in the past 100 years with the intent to kill.



Blister Gases (Vesicants) –Mustards

History

The best-known vesicant is sulphur mustard, which was extensively utilized in World War I. This compound is termed Yperite in French, since it was used for the first time in 1917 near Ypres, France. It is called Lost in German. It is still in the chemical inventory of about 17 nations.

Sulphur mustard was first discovered in 1822, but its importance as a blistering agent was not confirmed until 1860. More recently, mustard was used by Iraq in the Iran-Iraq war. About 95 percent of these cases, many taken to Belgium to be treated, recovered but required prolonged hospital stays.

Mustard is 2,2, -di (chloro-ethyl)-sulfide. The U.S. symbol for the pure compound is HD. After World War I, it was discovered that nitrogen could be substituted for the sulfur without a loss of the blister effects. There are three nitrogen mustards.

- (1) N-ethyl-2, 2' di
(chloroethyl) amine or
HN1,

(continued on next page)

- (2) N-methyl-2, 2' di (chloroethyl) amine or HN2, and
- (3) 2,2'2' tri (chloroethyl) amine or HN3.

HN3 is the only nitrogen mustard likely to be used today for warfare or criminal acts. HN2 is Mustargen, used for years as the mainstay for anti-neoplastic therapy, until replaced by newer agents.

Toxicology

Vesicants readily penetrate cell membranes and tend to be persistent. They can be thickened or dissolved in certain solvents to make them even more persistent. With alkalinity and increasing temperature, their volatility increases and they are less persistent. When dissolved in water they are hydrolyzed to polyalcohols and hydrochloric acid. In running water their persistence is only a few days, but in stagnant water several months. No drug is available for prevention of its effects. The LCt (see Definition Part II) is 1500mg.min/m³.

Mustard vapors tend to rise as the temperature climbs. During World War I the Germans often dispersed mustards at night. In the cool of the evening, this persistent agent would cling to the earth. The next day the Allies, thinking the worse was over, would take off their gas masks only to be poisoned by the agent as the daytime temperature rose, and with it the volatility and vapor density of mustard.

Protection is not possible with ordinary clothing. In the military, NBC suits, including a respirator, gloves and overboots, are required. The military uses MOPP levels to determine the demands for personal protection in response to a threat, with the higher levels requiring more protection. There are seven MOPP levels at this time, and MOPP 4 is the level required with mustard gases, Lewisite and Phosgene Oxime.

In the civilian sector, training for eight hours is necessary for first-responders to understand when and how to wear the personal protection (PPE) equipment, including the inherent problems associated with its use and proper maintenance of the equipment. (OSHA 29 CFR 1910.120). There are four levels of protection in the civilian sector. These are best described in information available at no charge from the U.S. Department of Health and Human Services.¹

Detection is possible with special dosimeters; automatic detection methods are not available, though research is attempting to overcome this problem.

HD and HN3 are the most-feared vesicants. They are stable and persistent. They attack the skin, eyes and respiratory tract. HD can be made into a vapor more easily than HN3. The effects of mustard are more likely to be disabling than lethal, but death can occur if the Ct (see definition part II) is sufficient. The effects are mutagenic, cytotoxic and cytostatic. They rapidly convert

to cyclic agents that are alkylating agents interacting with cellular components. DNA adducts are formed and cross linkage damage occurs. As a result, DNA replication is impaired. Since actively proliferating cells are affected most readily, the hemopoietic, basal epithelial cells and gastrointestinal system are most vulnerable.



Imperial War Museum – Mustard gas attack 1918

Clinical Aspects

Eyes

With mild brief exposure, the eyes are affected first and indicated by a mild conjunctivitis, which lasts one to two weeks. Most cases in World War I were of this nature. The respiratory tract is usually not affected. With heavier exposure, corneal involvement and iritis occur, requiring months of convalescence. In rare cases, recurrent corneal ulcer and opacification occurs. Permanent blindness is rare, but temporary blindness is common. There is a latent period of four to 12 hours

before these symptoms occur. Most eye problems resolve within 14 days after injury.

Skin

The skin can be affected by both the liquid or vapor form, and also is affected after a four to 12 hour latent period. With very mild exposure, the skin may not blister but only turn red with itching or burning. With slightly greater Ct, a striking scarlet-fever-like redness of the skin occurs first, with slight edema after the latent period. The skin itches intensely. After this, residual pigmentation similar to a sunburn remains. With higher Ct, blisters occur that are relatively painless and skin loss can be seen, especially around the penis and scrotum. The blisters break easily, but the fluid inside is not dangerous. Necrosis exists down to the dermis in many cases, healing is slow and secondary infection common. Dark brown or black hyperpigmentation then results. Late effects include skin sensitivity, contractures, scarring and fragile skin in many cases. Superficial burns will heal within 14 days, but deeper burns heal take about 60 days to heal unless there are complications.

Occasionally skin sensitization occurs so that with repeat exposures, even with low doses, a more rapid onset of symptoms occurs. These begin usually within one hour of exposure, but only last 2 or 3 days. A morbiliform rash or eczematoid

condition also occurs in sensitized individuals.

Respiratory Tract

After the latent period mentioned above, rhinorrhea occurs followed by a sore throat and hoarseness. Aponia may result from vocal cord damage. Increased thick secretions occur in the lower airways. Secondary infection is common. Abnormal pulmonary function tests are common if deeper bronchial or parenchymal damage occurs. With very high dosages, the subject dies in a few days from pulmonary edema and necrotic tissue obstructing the trachea or bronchi.

Hemopoetic

These agents can cause a depletion of the granulocyte and megakaryocyte series. At first, a reactive leukocytosis occurs followed by a decrease in white cells. A severe leucopenia or marrow aplasia indicates a poor prognosis.

Gastrointestinal

After ingestion of contaminated food or water, the clinical picture is that of acute gastroenteritis with nausea, vomiting, diarrhea and prostration. Shock may occur.

Systemic effects

Absorption of high dosages can lead to convulsions followed by CNS depression. Arrhythmias including AV block and cardiac arrest can occur.



Vesicles after a mustard attack Iran –Iraq War, courtesy Department of Defense

Treatment

- First decontaminate the eyes and mucous membrane with water, 1.26% sodium bicarbonate or saline.
- The skin should not be decontaminated with water, only the eyes. In the military, special chlorinated absorbent kits are used on the skin. They are not a substitute, however, for general decontamination. A 0.5% hypochlorite solution is the preferred agent for decontamination. Diluting Clorox one part to 10 parts of water will accomplish this. The standard 5% solution of hypochlorite is too irritating. Calamine lotion or water will reduce itching. Ointments and creams are not advised. Skin grafts have not taken well and are rarely performed. Avoid infection, as this is the most significant complication.
- There is no drug treatment available. Sodium thiosulfate

may reduce some of the systemic effects of mustard if given within 20 minutes of exposure.

- Eye- Local analgesics in the injured eye are not recommended. Systemic narcotics will be required. Antibacterials in the eye are utilized. If a corneal lesion is present using fluorescein solution, consult an ophthalmologist and use mydriatics. Don't cover the eyes with a bandage. Instead, use dark glasses or goggles.
- Respiratory – Sterile, cool mist inhalations for laryngitis and tracheitis. Use antibiotics if infection occurs. In general, treat all systems supportively.
- Systemic Effects – After the latent period, anorexia, fever, depression, nausea and vomiting can be seen. Malaise and nausea are seen first. With high doses of mustard, especially in high temperature, nausea, vomiting and collapse will occur before erythema of the skin develops.

Chronic effects

- Carcinogenesis (lungs, skin)
- Corneal scarring, blindness (rare)
- Skin pigmentation, scarring, and contractures
- Chronic obstructive lung disease, bronchial stenosis

Lewisite

Lewisite, termed L by the military, was purified by the United States after World War I. It was named for Capt. W. L. Lewis, who headed the team that synthesized it in 1918. It was not used on the battlefield. The Japanese may have used it against the Chinese. It can be mixed with mustard to lower its freezing point and permit more persistence. This compound smells like geraniums.

Lewisite is 2-chlorovinyl-dichloroarsine. ($\text{ClCH}=\text{CH}-\text{AsCl}_2$) Arsines possessing the $-\text{AsCl}_2$ group are vesicants. It is an odorless, colorless liquid, but impurities give it a brownish color. It hydrolyzes rapidly, and on a humid day an effective concentration is difficult to maintain. It is soluble in solvents. With strong alkalis it becomes a non-vesicant. It differs from other vesicants because its effects occur within seconds of exposure.

Detection is by a Draeger tube, which reacts with organic arsenicals. No automatic detectors are available.

Protection - The same rules apply as mentioned under mustard. Ordinary clothing will not be protective. Decontamination is the same as for mustard.

There is no known Lewisite being produced or stockpiled in any country, except the United States' Toole Army Depot.

Toxicology

It can penetrate the skin and then produce arsenic poisoning. It interferes with lipoic acid and a number of sulfhydryl enzymatic sites.

Clinical Aspects

Eyes - Severe damage to the eyes with instant pain and blepharospasm occurs. Edema of the lids closes the eyes within one hour. Corneal lesions may heal or progress to complete destruction, depending on Ct. A liquid arsenical vesicant produces a gray clouding of the cornea instantly.

Skin – In general, lewisite produces less pain than mustards with a more rapid onset of erythema and painful, thicker blisters. The blisters start within several hours. The blister fluid is non-toxic. The itching and pain persists for about 72 hours. The skin absorbs lewisite in two or three minutes, compared to 20 to 30 minutes for mustard. Lewisite blisters start in the center of an erythematous area and spread out to include the entire area of erythema, whereas mustard blisters start out at the edge of the blister as a “string of pearls.”

Airways – It is a strong irritant and can produce pulmonary edema. It also can cause significant protein and plasma leakage from capillaries; initially those in the lungs but general anasarca can result with hypovolemia and renal responses.

Treatment

Unlike most other agents, dimercaprol—also known as British anti-lewisite (BAL)—acts as an antidote for lewisite. It can be made into an ointment for skin burns prior to vesiculation, or an eyedrop for ocular manifestations. The ocular dimercaprol would have to be made in advance and applied to the eye within two minutes of exposure, so it is not very practical. A strong mydriatics, such as atropine, should be used if there is iritis, corneal erosions, or with marked photophobia or miosis. Dimercaprol ointment is not compatible with silver sulfadiazine.

Systemic treatment is indicated if signs of pulmonary edema are present, or a skin burn exists the size of the palm of the hand, caused by a liquid arsenical vesicant. Topical BAL is used plus BAL 10% in oil, 3 mg/kg every 4 hours for 2 days deep IM. Fifty percent of individuals receiving BAL get sick from the BAL, but the effects will clear in 72 hours.

Newer agents such as DMSA (meso-dimercaptosuccinic acid) or DMPS (2,3-dimercapto –1- propanesulfonic acid) are more active, more soluble and are less toxic than BAL. The newer products also reduce brain arsenic levels better than BAL, and if available should be used.

Phosgene Oxime

The last vesicant we will review is phosgene oxime, though there are others. It has not been used in the

battlefield since German scientists synthesized it in 1929. It has been stockpiled as it can penetrate garments and rubber at 8 percent concentrations very quickly and it produces severe and prolonged effects.

Toxicology

Phosgene oxime, termed CX by the military, is dichlorformoxime with a formula $\text{CCL}_2=\text{NOH}$. This compound is a colorless, crystalline powder that melts around 40°C and boils at 129°C . The solid material will produce enough vapor to cause symptoms. It is not a true vesicant, since it produces an urticant effect (erythema, wheals and urticaria). If you add certain other compounds to the substance, it will vaporize at room temperature with a high vapor pressure.

Clinical Aspects

As with other vesicants, the same protection rules apply. In low concentrations it irritates the eyes and skin immediately. A few milligrams to the skin will cause a burn and a necrotizing wound. It is very painful and destructive and has been termed a corrosive agent. Systemic effects are possible, probably by oximes reacting with SH and H₂N groups.

Clinically, it can cause pulmonary edema and death. It can cause corneal burns and blindness. Its effects on the skin are intense stinging, followed by the skin turning white due to coagulation necrosis. Skin necrosis, desquamation and a

purulent discharge then occur. No other agent produces such an immediate painful onset of pain in the eyes, skin and respiratory tract followed by rapid tissue necrosis.

Treatment

Recovery takes about three months and the treatment is supportive. Chlorination solutions do not work when decontaminating these patients. Alkalinization is better. Pulmonary edema must be managed as a noncardiac pulmonary edema case. Eye care is the same as any corrosive. The agent is absorbed within seconds so that, in general, decontamination is useless.

Summary

Vesicants are inhumane compounds with skin, ocular and systemic effects. They usually don't kill, but are very symptomatic and disabling. They would put a strain on the health care system of a community if a large amount of vapor was emitted. Decontamination, usually with chlorinated solutions, is important, as there is no specific antidote except for arsenicals.²

Cyanogens

During World War I, cyanide was used as a chemical weapon. The most important cyanogens agents are hydrogen cyanide (HCN) and several cyanogens halides.

Hydrogen Cyanide

Cyanides have been used extensively in this country for manufacturing purposes. It is used in electroplating, gold and silver processing, tanning, metallurgy, chemical processing and other compounds. It has been used in executions, suicides and poisonings for many years. Cyanide poisoning has been reported from eating chokecherries, bitter almonds and apricot pits. It is a component of Laetrile. Cassava, a staple, is blamed for a high incidence of tropical ataxic neuropathy due to its amount of cyanide. Cigarette smoke contains cyanide so that the smoker has about 17 $\mu\text{g}/\text{mL}$. (Controls average about 0.06 $\mu\text{g}/\text{mL}$.) The gas chamber utilizes the principle of generating Hydrogen Cyanide (HCN). Dropping a cyanide salt into a strong acid produces it.

Numerous international terror attempts have used HCN release. Most recently the Aum Shinrikyo cult used cyanide salt and acid in several restrooms in the Tokyo subway, several weeks after the sarin nerve gas attacks in March 1995.

Toxicology

Hydrogen cyanide, termed AC by the military, is a colorless, highly volatile

liquid. It is less dense than air (0.99 at 20C) and has a faint smell like bitter almonds or peach kernels. A segment of the population cannot smell this. This agent gradually oxidizes in water, losing its toxicity as it does so. Strong oxidizing agents, such as potassium permanganate, neutralize it by oxidation. This substance has an affinity for oxygen and is flammable. Sulfur-containing compounds and metals form complexes with this substance. The boiling point of AC is 25.7 C, vapor pressure is 740 mm Hg, and its LCt for vapor ($\text{mg}\cdot\text{min}/\text{m}^3$) is 2500 to 5000 and for liquid is 100.

HCN can readily diffuse through the skin, where it forms a reversible complex with respiratory cytochrome oxidase enzyme system. This impairs cellular respiration and produces a histotoxic anoxia. The brain is especially sensitive to this, and central respiratory failure is the most common cause of death.

Protection from this substance requires MOPP 4 protection. A modern NBC respirator will provide protection, but the filter must be changed after the attack. The older military respirator will not help to prevent poisoning unless the charcoal is dry and has been impregnated with metal salts in the canister.

Military M254A1 kits, Draeger tubes, automatic detectors (ICAD) and water testing kits are available to detect HCN.

Clinical Aspects

When exposure occurs, decontamination is not required but you must remove all contaminated clothing, wash any liquid cyanide off the skin with soap and water or water alone. Gavage and administer activated charcoal if the cyanide was ingested.

In high concentrations (Ct), death is rapid. At first, one cannot utilize oxygen properly so breathing is rapid. Then respiratory arrest occurs. Within 20 seconds, convulsions begin and then within a few minutes cardiac arrest occurs. Such cases are unlikely to make it to the ER.

With a lower concentration that is not immediately lethal, we will see weakness of the legs, vertigo, nausea and headache. This may or may not be followed by convulsions, coma and death. If the subject is comatose, residual neurological effects may occur once consciousness is regained. Temporary or permanent nerve deafness may occur.

In very mild cases, headache, nausea and vertigo are found, which will last several hours before complete recovery.

Blood cyanide levels must be performed on whole blood, since most of the cyanide rapidly invades the red blood cell. It tends to fall in stored samples due to its short half-life. Levels of 0.5 to 1.0 $\mu\text{gm/mL}$ are associated with early symptoms. Levels of 2.5 to 3 $\mu\text{g/mL}$ are

associated with coma, and levels more than 3 are associated with death.

Treatment

Speed is essential. The physical properties of this compound indicate that the agent will not remain very long in the liquid state.

Decontamination will otherwise not be required. Oxygen therapy and positive pressure resuscitation may be required. Opiates must be used with caution.

First, you must rapidly bind or fixate the cyanide ion, either by creating methemoglobin or fixing it with cobalt compounds. Any person who is conscious and breathing normally more than five minutes after being exposed to cyanide agents will recover without any treatment, as this substance is rapidly detoxified by the body. In those with symptoms, remove the patient from the source while wearing PPE. There are four methods used to fixate cyanide.

(a) Amyl nitrite is often used if there is a respiratory positive pressure present. Do not use amyl nitrate with oxygen as an explosion may occur.

(b) If there is impairment with breathing, IV sodium nitrate should be used (10 cc of a 3% solution, 300mg over three minutes). This will produce methemoglobin, which binds the cyanide. Keep the patient flat or blood pressure will fall from the nitrite. Try to obtain a little cyanosis to indicate methemoglobinemia.

(c) A newer agent, 4-Dimethylaminophenol-hydrochloride (DMAP), is very effective in treating cyanide poisoning. If available, give it instead of the nitrites at a dose of 250 Mg IV slowly every hour until sodium thiosulphate is made available. The DMAP can be stopped once the sodium thiosulphate is given. This is used by the German military and 3 mg/kg IV can produce a methemoglobin level of 15 percent in one minute. Disadvantages of 4-DMAP are necrosis in the area if given IM, increases in pain, fever and elevated muscle enzymes can be seen. Very high levels of methemoglobin are undesirable.

(d) Another alternative way to initially bind cyanide is with intravenous hydroxycyanocobalamine. This is commercially available but large amounts (4 g) IV slowly should be used as compared to the IM route. The cobalt will act to bind a portion of the cyanide and complex it until the thiosulphate is employed to finish the job. HydroxyB12 is relatively safe. Disadvantages include rare allergic reaction, high cost for the amounts required, short half-life as it decomposes in light.

(e) Follow the binding or fixating of the cyanide ion with an infusion of sodium thiosulphate. The dose must

be adjusted for children. Administer the sodium thiosulphate at a dose of 12.5 Gms (50 cc of a 50% solution over a 10 minute period of time.) Remember that sodium thiosulphate must always be given to complete the medical detoxification of cyanate by converting the free and bound cyanide to thiocyanates under the influence of the enzyme rhodenase. The relatively nontoxic thiocyanates can be metabolized. The Lilly cyanide antidote kit contains amyl nitrate, sodium nitrite and sodium thiosulfate and is familiar in the emergency room setting.

Cyanogen Halides

Due to problems of delivery of hydrocyanic gas as well as the use of more effective gas masks by the German troops, the French (who were the main proponents of cyanide during World War I, developed cyanogen chloride to be less volatile than other cyanide compounds. It proved lethal in high concentrations by paralyzing the respiratory center. Cyanogen chloride, termed CK by the military, and cyanogen bromide (used briefly by the Austrians) react to eventually release hydrogen cyanide. In addition they have local irritant effects to eyes and respiratory mucous membranes.

Toxicology

Cyanogen chloride has a strong pungent odor. It does not easily dissolve in water, but dissolves well

in organic solvents. Its vapor is heavier than air (2.10 vapor density) and very irritating to the eyes and mucous membranes, and can produce pulmonary edema. This compound is usually non-persistent. CK's boiling point is 12.9 C, vapor pressure is 1,010 mg Hg, and in a pure form it is a colorless gas or liquid. The LCt is 11,000 mg.min/m³.

This class was not readily absorbed by gas mask charcoal used in World War I and required metal salts to be added to the canisters attached to the masks. Using modern NBC masks provides short-term protection.

Water testing kits are available. Vapor and liquid detection capability for AC and CK is performed in the military using personal kits (M256A1 kits). Draeger tubes and ICAD units are available to detect cyanide.

Clinical Aspects

In high concentrations, CK causes dyspnea from its local irritant effect and intense irritation of the nose, throat and eye. Coughing, tightness in the chest and lachrymation occur, followed by failing respiration and death in a few minutes. Such cases probably will not reach the physician. If the effects are nonfatal, there is a persistent cough with frothy sputum, rales in the chest, marked cyanosis and severe dyspnea.

Treatment

See the treatment and decontamination for HN. In addition since delayed pulmonary edema is possible they must be watched for at least six hours in intensive care before being released. If pulmonary edema occurs, positive airways pressure with a PEEP mask should be provided. Intubation with positive pressure should be done if the PEEP mask fails to improve the arterial blood gases and clinical picture. Steroids have not been found to be of value, diuretics can be used, but cautiously in the presence of positive pressure respirators due to accompanying hypovolemia that may occur.

Summary

Cyanide compounds are rapidly lethal, but hard to manage to create mass casualties. These compounds have a long history in homicides, suicides and the death chamber. Large amounts would be required to be effective in a water supply. Treatment is possible if initiated before cardiac arrest.³



Iraqi scud missile
Department of Defense

Part IV

Additional Chemical and Biologic Agents

During World War I, tear gas, chlorine gas, diphosgene, phosgene and finally sulphur mustard were all used. The gas masks were uncomfortable and often taken off too early. They were not very effective against phosgene and not at all effective against mustard. Casualties were enormous though the mortality rates were in the range of 2 to 3 percent for both American and British forces.

Other agents used in World War I were bromacetone, benzyl bromide, chloromethyl chloroformate, chloropicrin, cyanide compounds, dichlormethylether, dibrommethylethylketone, diphenylchloroarsine, diphenylcyonoarsine, ethyldichloroarsine, ethyl diodoacetate, monobrommethylethylketone, and xylyl bromide. Many of these World War I chemicals were replaced in modern warfare with nerve gas and other more effective agents.

Incapacitants

BZ and LSD

At times a terrorist may not wish to kill, but to incite fear. One way to do this would be to cause delirium at

random. BZ, or 3-quinuclidinyl benzilate, is a white powder that is a cholinergic agent with the power to produce delirium for several days. It is effective by all routes of administration and readily crosses the blood brain barrier. Symptoms start in one hour and nervous system effects become maximal after 4 hours. The effects last 24 to 48 hours or longer, dependent upon the dose. BZ produces its central nervous system effect in the same way scopolamine and atropine does, by interfering with cholinergic transmissions at muscarinic sites. Its safety margin (between an incapacitating and a lethal dose) is 30. Usually less than 1 mg is used. Beginning about one hour after ingestion, tachycardia, ataxia, dizziness, vomiting, dry mouth, blurred vision, confusion and sedation are noticed. About four hours after the intoxication starts, there is increased inability to respond to the environment. About 12 to 96 hours, depending on the dose there is increased activity, random unpredictable behavior with delusions and hallucinations. No late residual effects are seen.¹

Treatment

Watch for and treat hyperthermia. Physostigmine is reserved for more serious cases. Give 2 to 3 mg every 15 minutes until the clinical picture is improved and then use an IV drip of about 2 mg per hour. Taper the dose as the poison leaves. Then give 2 to 5 mg po every one to two hours as the clinical condition stabilizes. Other stigmimes do not effectively cross the

blood brain barrier and should not be used.

LSD

Another incapacitant is D-Lysergic Acid Diethylamide. (LSD) – as little as 50 µg can induce dramatic psychological changes and doses above 2 mg can induce convulsions. The symptoms start shortly after inhalation or ingestion. They peak at two to three hours and gradually subside over the next four to six hours. Initial nausea is common, followed by confusion associated with delusions and hallucinations. Evidence of hypersympathotonia is present.

Treatment

Give diazepam 10 to 20 mg IV slowly or amytal 200 to 400 mg IV. No specific treatment is known.

Riot Control Agents

CS

The most common of these is tear gas, termed CS or Orthochlorobenzylidene Malononitrile. They are of low toxicity and short action. CA, CN and CR are other riot control agents. The LCt₅₀ for CS is 60,000 mg.min/m³. Exposure to CS produces a pepper-like odor with rhinorrhea, ocular irritation, coughing and dyspnea.

A terrorist may use this to create confusion, demoralize a civilian population unfamiliar with its effects or to prevent the action of rescuers.

Treatment is to remove the victim from the vapor and the symptoms will resolve with fresh air. Do not use oil-based lotions to assist or chlorinated solutions to detox the subject. Change clothes. If skin decontamination is desired, use a baking soda solution, as CS hydrolyzes more rapidly in alkaline solutions. One case of reactive airways disease (RADS) was noted after high dose tear gas exposure and individuals with asthma may have a temporary aggravation following exposure.

Toxic Inhalants

The impact of being gassed in the trenches in World War I is best described in the Victorian words of the day.

"[The] vapor settled to the ground like a swamp mist and drifted toward the French trenches on a brisk wind. Its effect on the French was a violent nausea and faintness, followed by an utter collapse. It is believed that the Germans, who charged in behind the vapor, met no resistance at all, the French at their front being virtually paralyzed."

- The use of gas at Langemarck as reported by the *New York Tribune*, April 27, 1915.



Painting by John Singer Sargent. (1856-1925) The bandages indicate that probably Mustard was utilized.

Chlorine

Used historically in World War I, chlorine gas is a dense, acrid, greenish gas with a characteristic odor. It tends to settle due to its density and is more hazardous in closed spaces.

Clinical Aspects

The first symptom caused by the inhalation of chlorine is air hunger or dyspnea. Ocular and nasal irritation occurs, followed by chest pain, coughing and a choking sensation. Preexisting bronchoreactivity or tobacco-related lung disease serve as significant risk factors. Exertional dyspnea is evident, along with increasing cyanosis. In addition to the above symptoms, aphonia or hoarseness is seen with higher doses. Pulmonary edema of a delayed type may appear two to six hours later. X-ray findings are not as good as the clinical examination at first. Characteristically, the x-ray shows pulmonary edema without cardiomegaly. With severe exposures, the pulmonary edema may appear in as little as 30 minutes with copious secretions from the tracheobronchial and nasopharyngeal regions. Sudden death can occur and is thought to be related to laryngeal spasm. Most

deaths occur in the first day and are related to respiratory failure. No residual sequelae are seen in most individuals, though reactive airways disease following heavy exposure is possible. Ordinary PFT studies may reveal obstructive changes, but may produce a false negative result in an affected group with residual dyspnea. Pulmonary stress testing with blood gases may reveal the abnormality since the oxygen-carbon dioxide exchange dramatically rises with exercise (from about 250 ml/min to 4 or 5,000 ml/min).

Treatment

Treat any superinfection that starts three to five days after exposure. The thin secretion will become thicker and colored. Prophylactic antibiotics should not be employed. Steroids are used with bronchoreactivity and bronchodilators are used as needed.

PEEP therapy may be required in more severe cases and can be used even with the rare complication of subcutaneous emphysema. Nontoxic pulmonary edema or severe dyspnea should be treated with intubation oxygenation to maintain a pO_2 of 60 mm Hg. Most cases start improving in about 72 hours.²

Phosgene

Termed CG by the military, phosgene produces a white cloud when released and is readily subjected to hydrolysis. In low doses, it smells like freshly mown hay but in higher doses is acrid and pungent. It was also used in World War I as a choking gas.

Clinical Aspects

In milder doses, it is an irritant to the mucosa and eyes. Smoking tobacco becomes unpleasant due to an objectionable taste. Also chest pain, dyspnea and a cough develop within a few minutes. With higher doses, the characteristic symptoms of acute pulmonary edema occur two to six hours later. Laryngospasm can occur and produce sudden death.

Phosgene hydrolyzes into hydrochloric acid, as does mustard. Diffuse perihilar, fluffy and diffuse interstitial infiltrates are seen with pulmonary edema. A non-hemorrhagic pulmonary edema with few inflammatory cells is seen on biopsy. Physiologically, there is damage to the alveolar-capillary junction. An exposed individual may be only mildly symptomatic, but can be converted into acute pulmonary edema by physical exertion for the first 72 hours after an exposure.

The effects of phosgene are short lived (usually three to four days) without serious late effects.

Treatment

Watch all patients with any toxic inhalation injury carefully. Auscultate carefully for signs of pulmonary edema. Do chest x-ray and ABG. If they appear to be normal, keep him at least six hours and repeat these studies. If abnormal keep longer. If normal in six hours, he can be released. There is no specific therapy. For toxic pulmonary edema, use O₂, PEEP, or intubation with a ventilator as needed. Diuretics may help a little, but may cause hypovolemia. Steroids have not been

found to be useful. Complete rest is valuable with this agent, since even minimal exertion will increase the likelihood of pulmonary edema.³

Zinc Oxide (HC)

The military used a mixture of zinc oxide and hexachloroethane and 7% grained aluminum to produce an obscurant or concealing smoke. It produced a thick white smoke when the byproducts of combustion hydrolyze with water vapor in the air. Mild exposure to HC smoke, a mixture of zinc chloride and various hydrocarbons, carbon monoxide and phosgene, produced dyspnea in the absence of any abnormalities with blood gas, chest x-rays or clinical auscultation. There are no residual problems but they must be watched for at least six hours.

With moderate amounts of exposure to HC, severe dyspnea, in the absence of x-ray findings are seen. Also they are often febrile. If released because of a lack of objective findings at six hours, this patient usually returns up to 36 hours later with obvious pulmonary edema, abnormal ABG and abnormal chest ray showing a dense bilateral hilar infiltrate thought to represent toxic pulmonary edema.

Bronchopneumonia may appear as a complication. Unlike the previous toxic inhalants, diffuse interstitial fibrosis can occur. A very high dose may be fatal quickly, but those who live can have hemorrhage of the upper airways due to ulcerations. A paroxysmal cough in more severely affected patients produces hemoptysis. The therapy for this disorder is the same as mentioned

for toxic pulmonary edema. Steroids are felt to be of value. About 10 to 20 percent of survivors will develop pulmonary fibrosis. ⁴

Other toxic gases include white, red or black phosphorus, nitrogen oxides, titanium tetrachloride, and organofluoride polymers (perfluoroisobutylene).

Ricin

This agent (Compound W by the military) is found in the Castor plant or *Ricinis communis*. Ricin is one the most toxic and easily produced plant toxins. It was used by the Bulgarian secret police to kill the Bulgarian defector Georgi Markov by placing a tiny amount on the tip of an umbrella. Small amounts of Ricin toxins by mouth are much less toxic than if given by injection. It is very difficult to diagnose and detect. It produces death by cleaving a portion of RNA and inhibiting protein synthesis. Oral ingestion produces a 1 to 2 percent death rate. The Kupffer cells and macrophages are especially sensitive to ricin due to certain binding sites. Inhalation of ricin causes severe necrosis of the airways and increased permeability of the alveolar-capillary membrane. Death after the inhalation of this agent is caused by hypoxemia from massive pulmonary edema and alveolar flooding.

Clinically, all of the serious or fatal cases with ingestion had a rapid onset of nausea, vomiting and abdominal pain. Following this, they had diarrhea, hemorrhage from the anus, dilated pupils, fever, thirst, sore throat, headache, vascular

collapse and shock. By injection, it has been reported that victims experience local pain and, depending on the dose, a feeling of weakness in about five hours. In the case of Mr. Markov, where the dose was estimated to be about 500 µg, 15 hours later he had high temperature, nausea and vomiting. Later, he suffered from gastrointestinal hemorrhage, vascular collapse, and complete arterioventricular conduction block followed by death.

Ricin, really four distinct toxins, is immunogenic and enzyme-linked immunosorbant assays of tissues or body fluids will confirm the diagnosis. Usually, antibodies will be found in approximately two weeks in survivors. There is no specific antidote. One must use supportive care only. Active prophylaxis with vaccination with the formalin-treated toxoid is possible with animal experiments. Some chemical agents have shown efficacy in laboratory experiments to specifically protect the animal from the effects of ricin.

Ricin is currently being utilized as a component of some antitumor drugs.

Contact the CDC for more information if ricin becomes known as a terror weapon in this country in the future. ⁵

Staphylococcus B Enterotoxin

Staphylococcus B

Enterotoxin, the best understood of the seven enterotoxins produced by

coagulase positive *Staphylococcus aureus*, is described as a superantigen, as small amounts avidly bind to T-cell antigen receptors and class II molecules of the major histocompatibility complex, causing rapid T-cell proliferation and massive cytokine release. SEB, an exotoxin, is the most frequent cause of food poisoning and account for about 60 percent of the food poisoning cases in the United States. If exposed to the toxin in a nonenteric route, a toxic shock syndrome that is potentially life threatening can be produced. This toxin, codenamed PG, was developed by the United States as a biologic warfare agent during the 1960s before we terminated our offensive capabilities. Russia and its allies have produced it. The most relevant route of exposure with this agent for a terrorist would be as an aerosol.

Terrorist may use this agent to contaminate food or a low volume water supply.

Clinical aspects

If exposed to the inhalational form of SEB, a rapid onset of disease develops three to 12 hours after exposure, which is severely incapacitating. In some workers who had accidental exposure, the toxic response lasted three to four days. A fever sometimes up to 106° F was seen early. A shaking chill may accompany this. Headache, myalgia and a non-productive cough with negative findings on a chest x-ray would be present. The cough may persist for four weeks. In larger doses, shock and death may occur

with massive release of cytokines. It is unlikely to be fatal, but will produce a large number of casualties that will take weeks to recover.

Be suspicious if a febrile illness with respiratory symptoms occur suddenly in a group of people who were otherwise healthy, that were in a similar area, or exposed to a similar aerosol.

There are no specific tests at first, and the diagnosis is dependent upon a clinical evaluation and comprehensive assessment of the total circumstances. Urine should be collected, since SEB can linger in the urine for several hours but is in the blood only transiently. Antibodies to SEB will eventually appear, but not until several weeks later. A blood sample should be drawn so that a later convalescent sample can be obtained for comparison.

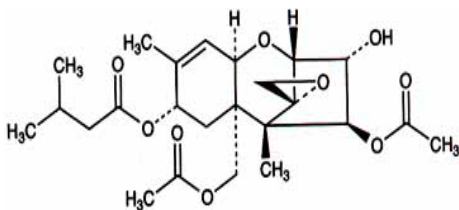
At first it would be easy to suspect influenza, adenovirus or mycoplasma. A large number of cases, either detected by the physician or the local health department, would increase the suspicion for a bioterrorist type weapon. Anthrax, plague and tularemia would worsen with time, but SEB tends to become stable rapidly. Tularemia, Q fever and plague pneumonia would be expected to have infiltrates on x-ray, whereas SEB usually does not. (Rarely, in severe late cases one might see ARDS or pulmonary edema.) Nerve agents would cause copious secretions and fasciculations, whereas SEB does

not. Mustard agents would give you a clue by having cutaneous erythema and vesiculations.

Treatment

Treat supportively. Decontaminate with .5% hypochlorite for 10 minutes then wash off with soap and water. A vaccine or antitoxin is undergoing evaluation at this time but is not released.⁶

T2 Mycotoxin



Mold toxins include aflatoxins, fumonisins, rubratoxins, trichothecenes and ochratoxins. These are natural plant toxins that can produce disease if heavily infested plants are consumed. Reports of illness occurring due to the ingestion of mycotoxins are well recorded and have been termed alimentary toxic aleukia. *Fusarium* is a fungus that has been incriminated in those cases. This fungus is also a suspected cause of the anencephaly cases along the Rio Grande, since the toxins of *Fusarium* interfere with folic acid metabolism. Fusariosis in a natural state usually affects immunocompromised individuals and can be fatal in HIV patients.

More recently, we have had to contend with a rash of cases of sick building and home syndrome from suspected mycotoxins. These are related to trichothecene production

from certain species of aspergilla, penicillium and *Stachybotrus Atrata* (*chartarum*). The trichothecenes are closely related family of about 150 compounds known chemically as sesquiterpenoids. They are of low molecular weight, 250 to 350 mw, that are lipophilic and relatively insoluble in water but not solvents. They are heat resistant and require 900 °F for 10 minutes in inactivate this chemical. They can also be inactivated by 5% hypochlorite.

One of the best-characterized trichothecenes is T2, famous for the implications of the use of this agent in the yellow rain attacks in Southeast Asia during the 1970s. T2 is strongly suspected of having been used in the past, either in a pure form or mixed with mustard or nerve gas, by North Vietnam, Russia, Laos and Egypt against various tribal groups that resisted government control.

Clinical Aspects

In small amounts, trichothecenes, once they enter the system from any route, inactivate rapidly proliferating tissue. They are toxic to most eukaryotic cells. The skin is particularly sensitive to the effects of fungi. They produce erythema swelling and even necrosis. They also cause vesicular reactions are about 400 times more potent per dose than mustards on the skin. Corneal damage, nausea, vomiting and diarrhea are also seen. It can be lethal within minutes to hours. Its lethality is similar to mustards. T2 has an LC₅₀ of 200 to 5800 mg.min/m³, whereas mustards range from 1500 to 1800 mg.min/m³. If

ingested, rapid irritation of the intestinal cells is noted, but if inhaled there is no apparent pulmonary edema or objective effect.

Considerable variation of the clinical response can occur depending on host factors such as preexistent liver disease, nutritional status, route of administration and stress. With all routes, gastric lesions occur with diarrhea and vomiting. With skin contact, necrosis and inflammation are prominent symptoms.

With an acute aerosol attack, the victim would experience lassitude, nausea, superficial skin irritation, vomiting and a loss of coordination. Within three to 12 hours, this would be followed by diarrhea eventually becoming bloody, hematemesis, dyspnea, bleeding gums, sore mouth and epistaxis. The skin would show a combination of vesicles and ecchymosis, and later areas of necrosis. Hematopoietic and immunologic suppressive effects mimic the effects of radiation. Shock with hypotension, preceded in many cases by convulsions and tremors, may occur. Animal studies indicate that pancytopenia is present. The Protime and PTT will be elevated. Detection in humans would require sending blood or bodily fluid samples to a reference national laboratory for gas-liquid and thin layer chromatographic analysis with mass spectroscopy. Immunoassays are

available to detect biomedical samples.

Treatment

Supportive. Treat similar to a mustard burn. Copious amounts of saline for skin decontamination and ocular irrigation. Remove of all clothing. Decontaminate with soap and water or diluted bleach.

Superactivated charcoal if any T2 was ingested. Apply calamine lotion to skin. Treat pulmonary edema if it occurs. Steam inhalation, cough suppressants for upper and bronchial irritation. Early use of steroids is indicated. The military used sodium bicarbonate, metoclopramide, and magnesium sulfate in addition to the charcoal.

Various other treatments are being explored but have not yet been verified for clinical use in humans. Theoretically, large doses of antioxidants would be of value. A number of antifungal drugs are being investigated in Phase 2 and 3 trials and their efficacy and safety in treating those agents that produce mycotoxins remains to be seen. Remember that T2 is a heat-stable, hardy toxin that can be weaponized. It is less toxic than ricin, Staph B or Botulinum toxin, but can still produce mortality.^{7,8,9,10}

Summary

There are a number of biologic and chemical agents that can be utilized as agents of bioterrorism. Thus far anthrax, and controlled, suicidal explosive destruction have been the method of choice. There is mention of cyanide use as well. If new agents of destruction occur, they will be thoroughly reviewed by the CDC as well as this Web site.

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Examination for Parts III and IV

(A score of 70% or better must be achieved to successfully complete this activity)

- 1. Mustard and radioactivity have several things in common clinically. Which one of these facts is false?**
 - Both usually produce blisters.
 - Both depress the white count.
 - Both cause gastrointestinal symptoms.
 - Both can damage DNA
 - Both have a latent period

- 2. A 54-year-old male presents with redness and burning of the skin, A few blisters are seen on the back. He has irritation of the eyes and blepharospasm. Rhinitis and cough are present. He was one of several individuals brought to the emergency room after being exposed to an odiferous gas 5 hours before. The most likely agent to do this is:**
 - Lewisite
 - HD
 - Cyanide
 - Sarin
 - Phosgene

- 3. A 33-year-old college student is one of three people brought to your office after being exposed to an unknown gas that killed her roommate within a few minutes. She was not as close to the gas release source as her roommate. She is extremely air hungry but without cyanosis. She also complains of nausea, lightheadedness and a headache. What agent is most likely the cause of this clinical situation:**
 - Lewisite
 - HD
 - Cyanide
 - Sarin
 - Phosgene

- 4. The treatment of choice for cyanide poisoning initially should be:**
 - Decontamination
 - Thiocyanates
 - Epinephrine
 - Cyanocobolamin
 - Nitrates

Please continue to next page

- 5. The basic pathophysiology of cyanide poisoning is:**
- Alkylation
 - Neurotransmitter inactivation
 - Interference with DNA replication
 - Histotoxic anoxia
 - Blockade of B₁₂ metabolism
- 6. HD refers to:**
- Nitrogen mustard
 - Nitrogen sulfur
 - Dichloro-ethyl sulfide
 - Phosgene Oxime
 - Lewisite
- 7. The Eli Lilly Cyanide kit contains _____, sodium nitrate and sodium thiosulfate:**
- Amyl nitrite
 - Oxygen
 - Nitroglycerin
 - Epinephrine
 - Hydroxycyanocobalamin
- 8. Arsenic poisoning is likely to be associated with:**
- Sulfur Mustard
 - HN₃
 - Lewisite
 - Phosgene Oxime
 - Cyanide Halogens
- 9. The characteristic smell of cyanide is only detectible by a portion of the population due to genetics and is described as:**
- A faint germanium smell
 - Garlic like odor
 - Musty odor
 - Bitter almond odor
 - Onion odor
- 10. Chokeberries and cassava are associated with which poison?**
- Cyanide
 - HN₄
 - HN₂
 - Lewisite
 - Cyanogen Bromide

Please continue to next page

11. A 21-year-old black male was one of 34 individuals brought to the emergency room with similar symptoms. All their manifestations started within 30 minutes of each other. About an hour after the ingestion of a punch at a political fundraiser tachycardia, ataxia, dizziness, vomiting, dry mouth, blurred vision, confusion and sedation were noticed. The most likely agent to cause such symptoms is:

- A nerve gas
- An anticholinergic such as BZ
- CS
- Cyanide
- Zinc oxide

12. The treatment of choice for severe BZ intoxication is:

- Atropine sulfate
- Diazepam
- Decontamination
- Epinephrine
- Pyridostigmine

13. Regarding CS, all of the following are true except.

- The gas is an ocular irritant
- The effects are rapidly reversible once one is removed from the gas.
- The agent is odorless and colorless
- The agent produces a pungent whitish gas.
- Rare cases of bronchoreactivity can occur after exposure.

14. Which agent causes an objectionable taste with smoking? In addition it produces respiratory irritation within minutes of exposure and can develop a delayed pulmonary edema.

- Zinc Oxide
- Chlorine gas
- Ricin
- Phosgene
- CS

15. After exposure to a yellow green vapor, a patient present with redness, pruritis and vesicles of the skin. In addition he has conjunctivitis symptoms, vomiting, a headache, incoordination, a cough and fatigue. Which agent is responsible?

- Nitrogen mustard
- T2
- Phosgene
- Chlorine
- Staph B

Please continue to next page

16. A toxic gas exposure victim was brought into your office. What agent was most likely if he expired a short time after exposure with laryngeal spasm? In addition he developed pulmonary edema (non-cardiac) in 45 minutes after exposure according to the history.

- Nerve gas
- Cyanide
- Phosgene
- Chlorine
- HC

17. Which of the following is not usually present with Staphylococcus enterotoxin B inhalation?

- Shock if large amounts of cytokines are released.
- Headaches
- Non-productive cough
- Fever
- A positive chest x-ray for pulmonary edema

18. Which of the following regarding trichothecene mycotoxins are false?

- Less toxic than botulism
- Less toxic than ricin
- Only T2 produced by Stachybotrus has ever shown a problem in humans.
- Resembles the effects of radiation on the human body.
- Produces inflammation and necrosis if contact with the skin occurs.

19. Which of the following fact is not true about zinc oxide?

- Usually afebrile.
- Short of breath with a clear x-ray at first.
- Can develop pulmonary edema after a latent period.
- Can produce interstitial fibrosis in 10 to 20% of cases
- Can cause hemoptysis and ulceration of the respiratory tract.

20. An agent from the Castor plant has already been used as a terrorist weapon. Which fact is true about this agent?

- It is difficult to produce and thus unlikely to be used again.
- It is more effective orally than by injection.
- It usually produces a milder, afebrile illness with diarrhea.
- If inhaled or injected it will usually be fatal.
- There is a toxoid available currently to prevent intoxication symptoms.

Please continue to next page

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