TABLE 262e-2 DECONTAMINATION AND TREATMENT OF CHEMICAL TERRORISM

Agent Category	Decontamination	First Aid	Other Considerations
Nerve	Remove clothing immediately. Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline.	Atropine before other measures Pralidoxime (2-PAM) chloride	Onset of symptoms from dermal contact with liquid forms may be delayed. Repeated antidote administration may be necessary.
Asphyxiant/blood	Remove clothing immediately if no frostbite.[a] Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline.	Rapid treatment with oxygen For cyanide, use antidotes (sodium nitrite and then sodium thiosulfate).	Arsine and cyanogen chloride may cause delayed pulmonary edema.
Choking/pulmonary-damaging	Remove clothing immediately if no frostbite.[a] Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline.	Fresh air, forced rest Semiupright position If signs of respiratory distress are present, oxygen with or without positive airway pressure may be needed. Other supportive therapy, as needed	May cause delayed pulmonary edema, even after a symptom-free period that varies in duration with the amount inhaled
Blistering/vesicant	Immediate decontamination is essential to minimize damage. Remove clothing immediately. Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline.	Immediately decontaminate skin. Flush eyes with water or normal saline for 10–15 min. If breathing is difficult, give oxygen. Supportive care	Mustard has an asymptomatic latent period. There is no antidote or treatment for mustard. Lewisite causes immediate burning pain, with blisters developing later. Specific antidote—British anti-lewisite—may decrease systemic effects of lewisite. Phosgene oxime causes immediate pain. Possible pulmonary edema
Incapacitating/behavior-altering	Remove clothing immediately. Gently wash skin with water or soap and water. Do not abrade skin.	Remove heavy clothing. Evaluate mental status. Use restraints as needed. Monitor core temperature carefully. Supportive care	Hyperthermia and self-injury are the greatest risks. Hard to detect because it is an odorless and nonirritating substance Possible serious arrhythmias Specific antidote (physostigmine) may be available.

[a]For frostbite areas, DO NOT remove any adhering clothing. Wash area with plenty of warm water to release clothing.

Source: State of New York, Department of Health.

appearing hours after exposure. The organs most commonly affected are the skin (with erythema and vesicles), the eyes (with manifestations ranging from mild conjunctivitis to severe eye damage), and the airways (with effects ranging from mild upper airway irritation to severe bronchiolar damage). After exposure to large quantities of mustard, precursor cells of the bone marrow are damaged, with consequent pancytopenia and secondary infection. The gastrointestinal mucosa may be damaged, and there are sometimes central nervous system (CNS) signs of unknown mechanism. No specific antidotes exist; management is entirely supportive. Immediate decontamination of the liquid is the only way to reduce damage. Complete decontamination in 2 min stops clinical injury; decontamination at 5 min reduces skin injury by ~50%. Table 262e-2 lists approaches to decontamination after exposure to mustard and other CWAs.

Mustard dissolves slowly in aqueous media such as sweat, but, once dissolved, it rapidly forms cyclic ethylene sulfonium ions that are extremely reactive with cell proteins, cell membranes, and especially DNA in rapidly dividing cells. The ability of mustard to react with and alkylate DNA gives rise to the effects by which it has been characterized as "radiomimeti"—i.e., similar to radiation injury. Mustard has many biologic actions, but its actual mechanism of action is largely unknown. Much of the biologic damage from mustard results from DNA alkylation and cross-linking in rapidly dividing cells: corneal epithelium, basal keratinocytes, bronchial mucosal epithelium, gastrointestinal mucosal epithelium, and bone marrow precursor cells. This damage may lead to cellular death and inflammatory reactions. In the skin, proteolytic digestion of anchoring filaments at the epidermal-dermal junction may be the major mechanism of action resulting in blister formation. Mustard also has mild cholinergic activity, which may be responsible for effects such as early gastrointestinal and CNS symptoms. Mustard reacts with tissue within minutes of entering the body. Its circulating half-life in unaltered form is extremely brief.

Clinical Features Topical effects of mustard occur in the skin, airways, and eyes; the eyes are most sensitive and the airways next most sensitive. Absorbed mustard may produce effects in the bone marrow, gastrointestinal tract, and CNS. Direct injury to the gastrointestinal tract also may occur after ingestion of the compound through contamination of water or food.

Erythema is the mildest and earliest form of mustard skin injury. It resembles sunburn and is associated with pruritus, burning, or stinging pain. Erythema begins to appear within 2 h to 2 days after vapor exposure. Time of onset depends on severity of exposure, ambient temperature and humidity, and type of skin. The most sensitive sites are warm moist locations and areas of thin delicate skin, such as the perineum, external genitalia, axillae, antecubital fossae, and neck.

Within the erythematous areas, small vesicles can develop, which may later coalesce to form bullae (Fig. 262e-1). The typical bulla is large, dome-shaped, flaccid, thin-walled, translucent, and surrounded by erythema. The blister fluid, a transudate, is clear to straw-colored and becomes yellow, tending to coagulate. The fluid does not contain mustard and is not itself a vesicant. Lesions from high-dose liquid exposure may develop a central zone of coagulation necrosis with blister formation at the periphery. These lesions take longer to heal and are more prone to secondary infection than are the uncomplicated lesions seen at lower exposure levels. Severe lesions may require skin grafting.