

CHAPTER 4

BLISTER AGENTS (VESICANTS)

Section I. INTRODUCTION

4-1. General

a. Blister agents (vesicants) are likely to be used to produce casualties and to force opposing troops to wear full protective equipment. Blister agents are used to degrade fighting efficiency rather than to kill, although exposure to such agents can be fatal. Thickened blister agents will contaminate terrain, ships, aircraft, vehicles, or equipment and present a persistent hazard. Vesicants include sulphur mustard (H and HD), nitrogen mustards (HN), lewisite (L) (this may be used in mixture with HD), and halogenated oximes (example, phosgene oxime (CX)). Halogenated oximes properties and effects are very different from those of the other vesicants.

b. Vesicants burn and blister the skin or any other part of the body they contact. They may act on the eyes, mucous membranes, lungs, and skin; mustards may act on blood-forming organs. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when ingested.

c. Some vesicants have a faint odor; others are odorless. They often have more serious effects than is immediately apparent. Both L and CX cause immediate pain on contact. The mustards are insidious in action, with little or no pain at the time of exposure. In some cases, signs of injury may not appear for several hours.

d. Vesicants poison food and water and make other supplies dangerous to handle.

e. Vesicants can be disseminated by artillery shell, mortar shell, rocket, aircraft spray, and bomb.

f. The severity of a blister agent burn is directly related to the concentration of the agent and the duration of contact with the skin.

4-2. Self-Aid

a. Assume MOPP 4 whenever liquid or vaporized agents are known to be present.

b. Liquid vesicants in the eyes or on the skin require immediate decontamination procedures as outlined in appendix D.

4-3. Precautions in Receiving Casualties

a. Casualties contaminated with vesicants endanger unprotected attendants. Individuals in contact with these casualties must be at MOPP 4, plus wear a butyl rubber apron.

b. Special precautions must be taken in receiving contaminated casualties to prevent injury to others. Contaminated casualties are decontaminated outside the field MTF to prevent vapor accumulation indoors. They are kept separated from clean (uncontaminated) casualties until decontamination is completed. Contaminated litters, blankets, and equipment must be left outdoors. Decontamination is necessary for equipment, vehicles, watercraft, and aircraft that have been used to transport contaminated casualties. Appendix B contains further information on decontamination.

c. Unhydrolyzed mustard on patients' skin surface can present a hazard to individuals receiving or treating these patients even after several hours. As mustard reacts with skin and subcutaneous tissue, it is hydrolyzed; however, the destroyed tissue becomes a barrier for complete hydrolyzation of excess mustard on the surface.

4-4. Protective Devices

a. The protective mask protects only the face, eyes, and respiratory tract. The mask protects against both liquid and vapor forms of vesicants.

b. Chemical protective overgarments help prevent the vesicant from reaching the skin.

4-5. Disposition of Casualties

See section V for disposition of casualties with blister agent burns.

Section II. MUSTARDS

4-6. Mustard (H and HD)

a. *Physical Properties.* Mustard is an oily liquid ranging from colorless, when pure (neat), to dark brown when plant-run (unpurified form when first produced). Mustard is heavier than water, but small droplets float on water surfaces and present a special

hazard in contaminated areas. It smells like garlic or horseradish. Distilled HD, the most common form of mustard, freezes at 57°F (14 °C) and boils at 442°F (228°C). It is only slightly soluble in water, which gradually destroys it, but undissolved mustard may persist in water for long periods. It is most soluble in

fats and oils. It is freely soluble in acetone, carbon tetrachloride, alcohol and liquid fuels (gasoline, kerosene, and diesel); however, these solvents do not destroy mustard. Mustard disappears from contaminated ground or materials through evaporation or through hydrolysis. It is rapidly destroyed by decontaminating chemicals or by boiling in water. The primary use of mustard is to cause delayed casualties by the liquid and vapor effects on the skin and the eyes and by the vapor effects through the respiratory system.

b. Persistence. The persistence of hazard from mustard vapor or liquid depends on the degree of contamination by the liquid, type of mustard, nature of the terrain and soil, type of munition used, and weather conditions. Mustard may persist much longer in wooded areas than in the open. Mustard persists two to five times longer in winter than in summer. The hazard from the vapor is many times greater under hot conditions than under cool conditions. Standard chemical agent detector kits should be used to detect the presence of HD vapor in the field.

c. Cumulative Effect. Even very small repeated exposures to mustard are cumulative in effect. For example, repeated exposures to vapors from spilled mustard can kill or produce 100 percent disability by irritating the lungs and causing a chronic cough and pain in the chest.

4-7. Effects of HD on the Eyes

a. Pathology, Symptoms, and Prognosis. In a single exposure, the eyes are more susceptible to mustard than either the respiratory tract or the skin. Figures 4-1 through 4-4 show effects of mustard on the eyes. Conjunctivitis follows an exposure time of about 1 hour to a concentration barely perceptible by odor. This exposure does not affect the respiratory tract or the skin significantly. A latent period of 4 to 12 hours follows mild exposure, after which there is lacrimation and a sensation of grit in the eyes. The conjunctival and the lids become red and edematous. Heavy exposure irritates the eyes after 1 to 3 hours and produces some severe lesions. Although temporary blindness may occur, permanent blindness is very

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rare. Casualties should therefore be reassured and a positive attitude taken. Care must be exercised to avoid transferring liquid agent from the hands to the eyes. Mustard burns of the eyes may be divided as follows:

(1) Mild conjunctivitis (75 percent of cases in World War I). Recovery takes 1 to 2 weeks.

(2) Severe conjunctivitis with minimal corneal involvement (15 percent of the cases in World War I). Blepharospasm, edema of the lids, and conjunctival occur, as may orange-peel roughening of the cornea. Recovery takes 2 to 5 weeks.

(3) Mild corneal involvement (10 percent of the cases in World War I). Areas of corneal erosion stain green with fluorescein. Superficial corneal scarring and vascularization occurs as does iritis. Temporary relapses occur and convalescence may take 2 to 3 months. Hospital care is indicated for casualties of this type.

(4) Severe corneal involvement (about 0.1 percent of mustard casualties in World War I). Ischemic necrosis of conjunctival may be seen. Dense corneal opacification with deep ulceration and vascularization occurs. Convalescence may take several months. Patients may be predisposed to late relapses.

b. Treatment.

(1) Self-aid.

(a) The risk of leaving liquid vesicant in the eyes is much greater than the risk from exposure of the eyes to vesicant vapors during the short period of decontamination. Decontamination must, therefore, be done despite the presence of vapor.

(b) Speed in decontaminating the eyes is absolutely essential. This self-aid procedure is very

effective for mustard within the first few seconds after exposure but is of less value after 2 minutes. Decontamination is done the same as for other vesicants (app D).

(2) Treatment of mustard conjunctivitis.

(a) Mild lesions require little treatment.

Although the lesions may become infected, a steroid antibiotic eye ointment, such as dexamethasone sodium phosphate-neomycin ophthalmic ointment, can be applied. Ophthalmic ointments, such as 5 percent boric acid ointment, will provide lubrication and minimal antibacterial effects. The application of sterile petroleum jelly between the eyelids will provide additional lubrication and prevent sealing of the eyelids.

(b) More severe injuries will cause enough edema of the lids, photophobia, and blepharospasm to obstruct vision. This obstruction of vision alarms patients. To allay their fears, the lids may be gently forced open to assure them that they are not blind.

(c) The pain is controlled best by systemic narcotic analgesics. Patients with severe photophobia and blepharospasm should have one drop of atropine sulfate solution (1 percent) instilled in the eye three times a day. To prevent infection, a few drops of 15 percent solution of sodium sulfacetamide should be instilled every 4 hours. Other antibacterial ophthalmic preparations may be substituted for sodium sulfacetamide.

(d) The eye must not be bandaged or the lids allowed to stick together. Sealing of the lids may be prevented as described in a above. The accumulation of secretions in the conjunctival sac or pressure on the eye predisposes to corneal ulceration.

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Figure 4-3. Casualty showing eye effects of mustard vapor.

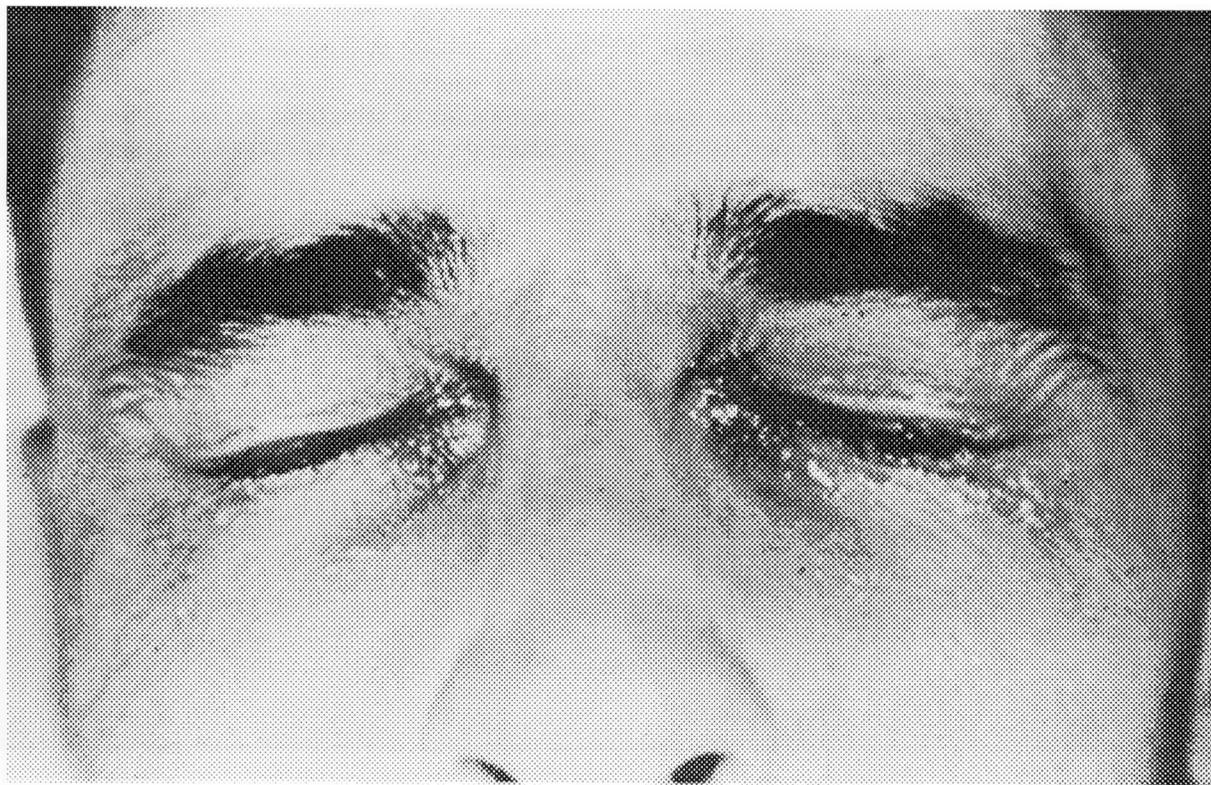


Figure 4-4. Casualty showing effects of mustard conjunctivitis.

To prevent complications, the patient should be treated by an ophthalmologist as soon as possible. When possible, the patient should be kept in a darkened room, given dark sunglasses, or given an eyeshade to help his photophobia.

(3) *Treatment of infected mustard burns of the eye.* Secondary infection is a serious complication and increases the amount of permanent scarring of the cornea. If infection develops, initial treatment should be carried out with several drops of a 15 percent solution of sodium sulfacetamide every 2 hours. After appropriate cultures, specific antibacterial preparations may be applied. Irrigation should be gentle and employed only to remove accumulated exudate. Pain is controlled as described in (2) (c) above. Patients with secondary infection or other complications should be referred to an ophthalmologist. Local anesthetics should not be used.

c. *Classification of Eye Lesions.* See paragraph 4-29b.

4-8. Effects of HD on the Skin

a. *Pathology.* The severity of the lesions and the rapidity with which they develop are greatly influenced by weather conditions as well as by the degree of exposure. Hot, humid weather strikingly increases the action of mustard. Even under temperate conditions, the warm, moist skin of the perineum, external genitalia, axillae, antecubital fossae, and neck are particularly susceptible.

(1) *Latent period.* Exposure is followed by a latent period which varies with the degree of exposure. It may be as short as an hour after liquid contamination, when the weather is hot and humid, or as long as several days after mild vapor exposures. With most vapor exposures in temperate weather, the latent period is usually 6 to 12 hours.

(2) *Erythema.* Erythema gradually appears (2 to 48 hours postexposure) and becomes brighter, resembling sunburn (figs 4-5 and 4-6). Slight edema of the skin may occur. In severe burns, the edema may limit motion of the limb. Itching is common and may be intense. As the erythema fades, areas of increased pigmentation are left (this sequence is reminiscent of that seen in sunburn).

(3) *Vesication.* Except with mild vapor burns, erythema is followed by vesication (figs 4-7, 4-8, 4-9, and 4-10). This is caused by progressive development of liquefaction necrosis of the cells in the lower layers of the epidermis. Exudation of tissue fluid into the spaces so formed results in an intra-epidermal vesicle. Clinically, multiple pinpoint lesions may arise within the erythematous skin; these enlarge and coalesce to form the typical blister (which is unusually large, domed, thin-walled, yellowish, and may be surrounded by erythema). The blister is filled with a clear or slightly yellow liquid that tends to

coagulate. The blister fluid does not contain mustard and is not a vesicant. Liquid contamination of the skin usually results in a ring of vesicles surrounding a gray-white area of skin which, although necrotic, does not vesicate. As noted in paragraph 4-3 c above, unhydrolyzed vesicant on contaminated patients may pose a hazard to other individuals coming in contact with them.



Figure 4-5. Noncasualty with erythema.

(4) *Resorption.* If the blister does not rupture, resorption takes place in about a week. The roof forms a crust beneath which reepidermization takes place. However, because of their thinness and tenseness, the blisters are fragile and usually break. If the roof becomes ragged, the burn may be considered an open wound. Once the blister has broken, it is best to remove its ragged roof to decrease the possibility of secondary infection.

(5) *Healing.* Since the damage to the corium is relatively superficial, healing occurs with little scar tissue formation, except in more extensive or infected burns where scarring is more severe.

(6) *Pigmentation.* Mustard burns usually are followed by a persistent brown pigmentation except at the site of actual vesication, where there may be a temporary depigmentation due to exfoliation of the pigmented layers of the skin (figs 4-11 and 4-12).

(7) *Hypersensitivity.* Repeated burns may lead to hypersensitivity of the skin to mustard.

b. Symptoms and Prognosis.

(1) An outstanding characteristic of the action of mustard is its insidiousness. Exposures to mustard are not accompanied by immediate symptoms, nor do any local manifestations occur until erythema develops. At this time there may be itching and mild burning. This pruritus may last several days and persist after healing. The blisters may be painful.

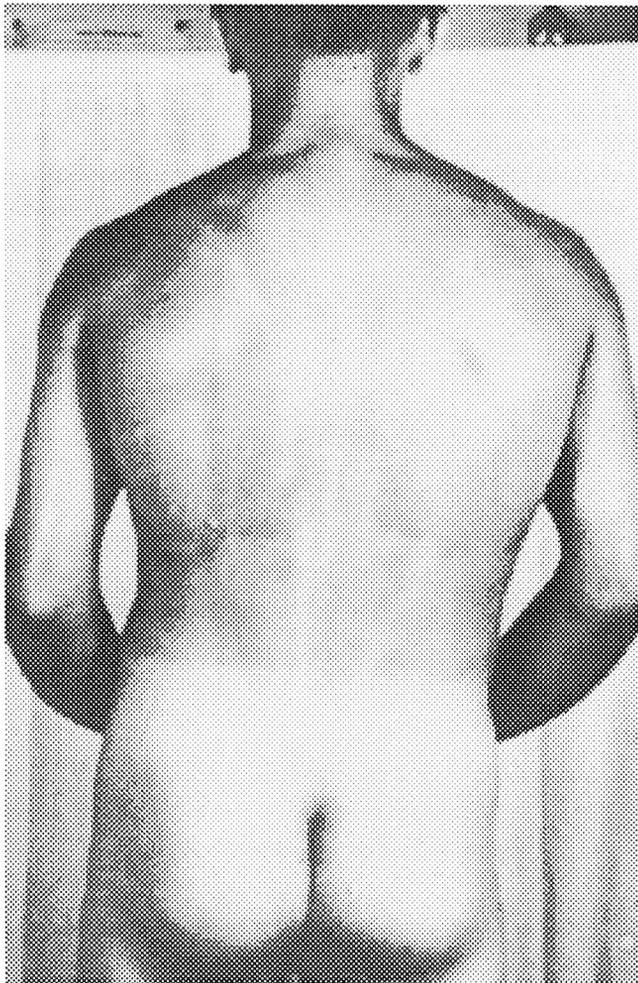


Figure 4-6. Casualty with generalized erythema and systemic intoxication.

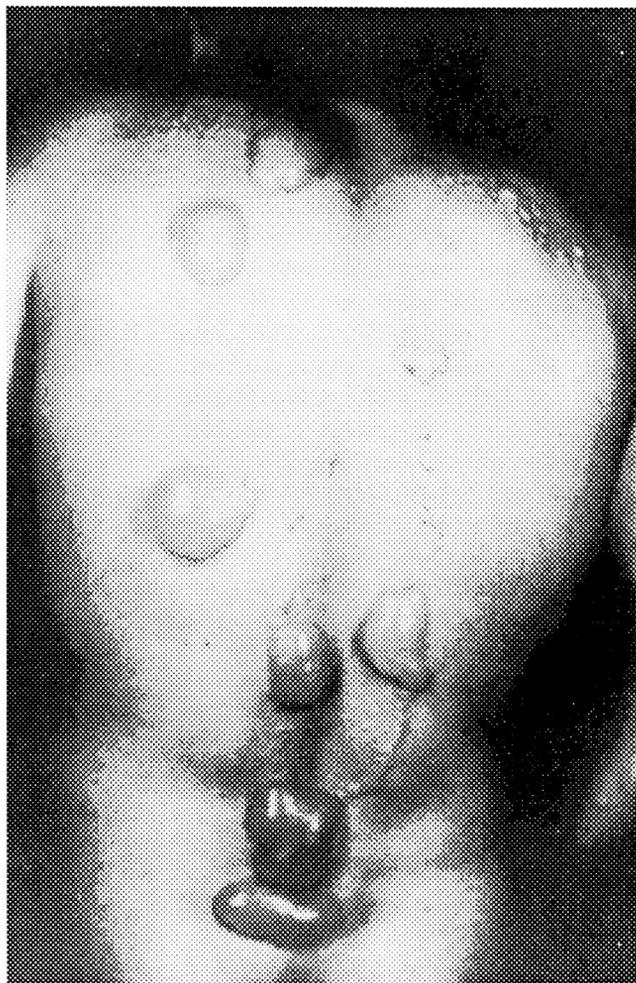


Figure 4-7. Casualty with severe vesication.

[Click here for Figure 4-6.](#)

[Click here for Figure 4-7.](#)

(2) Mustard erythema heals at about the same rate as sunburn of like severity. Areas of multiple pinpoint vesication usually heal, with desquamation, in 1 to 2 weeks. Healing times for mustard blisters vary widely with both severity and anatomical location. In general, blisters of the face heal in 1 to 2 weeks. Blisters located in other areas may take slightly longer to heal; but if protected from infection, they will heal in 2 to 4 weeks. If cutaneous injury results in full-thickness coagulation necrosis, skin grafting may ultimately be necessary. However, a mustard burn of the skin is usually limited to the epidermis and does not require grafting (fig 4-13).

(3) Moderate contamination of mustard skin lesions with saprophytic bacteria, which causes no appreciable inflammatory reaction, does not seem to delay the healing of mustard burns. Active infection, with inflammation and purulent exudation, may increase the severity of the lesions and delay healing greatly (fig 4-14).

c. Diagnosis of Skin Lesions Due to Mustard.

Similar skin burns are produced by mustard and the nitrogen mustards. Mustard burns are also similar in appearance to those caused by arsenical vesicants. Differentiation of mustard lesions from those produced by arsenical is based upon—

- (1) History of exposure to mustard.
- (2) Absence of pain or discomfort at time of contamination (L is irritating and immediately painful).
- (3) A zone of erythema surrounding blisters (not predominant with arsenical). It should be remembered that vesicular lesions, much like mild mustard burns, may be produced in sensitive individuals by a variety of substances, notably plant poisons such as poison ivy or poison oak. However, the skin lesions of plant contact are on exposed skin and linear in configuration. The earliest affected areas of skin from mustard are the skin folds, groin, and inner aspects of the extremities.

d. Decontamination of Casualties. Casualties who have experienced liquid mustard contamination of the

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skin or clothing will seldom be received by the medical service in time to prevent subsequent blistering. Nevertheless, if erythema has not appeared, known or likely contaminated skin areas should be decontaminated as described in appendix D. Cut away and discard hair contaminated with liquid mustard. Decontaminate the exposed scalp with the M291 Skin Decontaminating Kit. If short of these substances, use 0.5 percent aqueous chlorine solution for decontamination of skin and hair. Wash off the decontaminating solutions promptly (within 3 or 4 minutes) to prevent additional skin injury, taking care that none of the solutions wash into the eyes. If erythema of the skin has appeared, soap and water is the best decontaminant. Contaminated clothing should be removed promptly from casualties outside the treatment facility to prevent more severe burns and to lessen the vapor hazard to patients and attendants.

e. Treatment of Mustard Erythema. Mustard erythema in mild cases requires no treatment. If an

annoying itch is present, considerable relief may be obtained with topical steroid creams or sprays. Severe erythema around the genitalia may become quite painful and associated weeping and maceration may ensue. Often, treatment with exposure of the area is desirable and care must be taken so that secondary infection of tissue does not occur.

f. Treatment of Mustard Blisters.

(1) Once blisters have broken, it is best to remove its ragged roof to decrease the possibility of secondary infection. Cleanse the area with tap water or saline, then apply sterile petrolatum gauze when the areas are small. Dressings should be changed and the wound inspected every 3 to 4 days. Small blisters on the face are opened and best left uncovered. Large blisters may best be treated by open methods. Apply about one-eighth of an inch thick layer of 10 percent mafenide acetate or silver sulfadiazine burn cream to the blisters as a topical antibiotic agent. Figure 4-15 shows a casualty with widespread vesication caused by mustard burns.

[Click here for Figure 4-10.](#)

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Click here for Figure 4-11.

(2) If the dressing sticks to the wound, care will be necessary to avoid pulling off the top of the blister. It is good practice to trim the edges of adherent gauze, leave it in place, and put a fresh dressing over it. If the wound needs to be examined, the dressing may be soaked off with sterile saline.

g. Treatment of Denuded Areas.

(1) Contamination of mustard burns with saprophytic bacteria is common and unless careful wound care is given, serious infection may result. If there is no inflammatory reaction, the treatment is the same as for uncontaminated burns. Figure 4-16 shows burns produced by the reaction of mustard vapor with sweat.

(2) Wounds which become infected must be treated with appropriate antibiotics after adequate cultures have been obtained. The medical officer must evaluate the infection and make the appropriate decision regarding further care.

h. Specific Antibacterial Therapy. Routine wound inspection aids in the early detection and institution of appropriate therapy for any complicating bacterial infections. Appropriate antibacterial drugs may be given either locally or systemically, as indicated. The early use of an appropriate topical antibacterial agent (such as mafenide acetate or silver sulfadiazine cream) may prevent a bacterial infection.

4-9. Effects of HD on the Respiratory Tract

a. Pathology.

(1) Inhalation of mustard vapor causes damage primarily to the laryngeal and tracheobronchial mucosa. The lesion develops slowly after exposure. A single exposure to a small amount of mustard vapor ordinarily does not produce significant injury. Repeated or chronic exposure to low concentrations of mustard vapor may lead to progressive pulmonary fibrosis, chronic bronchitis, and bronchiectasis. Moderate exposures result in hyperemia of the respiratory mucous membrane and necrosis of the lining epitheliums. With severe exposures, the necrotizing action is accompanied by exudation resulting in a diphtheritic-like pseudomembrane, which may form a cast of the tracheobronchial tree.

(2) In the more severe cases, the pulmonary parenchyma shows congestion, mild patchy edema, and focal atelectasis. Altogether, these changes may be insufficient to cause hypoxia and they are frequently complicated by bacterial infection of the lungs, which results in suppurative bronchitis and bronchopneumonia. The latter is responsible for almost all deaths following vapor exposures. The early mortality from mustard among American troops in World War I (slightly more than 2 percent) was due almost entirely to such pulmonary complications following inhalation of vapor.

[Click here for Figure 4-12.](#)

organisms with their antibiotic sensitivities should be performed, then antibiotic therapy can be limited to the specific agents.

4-10. Systemic and Gastrointestinal Effects of HD

a. Pathology.

(1) Ingestion of mustard produces vacuoles and nuclear swelling of the epithelial cells of the gastrointestinal tract, with eventual necrosis and desquamation with hemorrhage. Absorption of the mustard from the intestinal lumen results in damage to the blood-forming organs mentioned in (2) below.

(2) With lesser skin or respiratory exposures to mustard, no apparent systemic lesions develop. However, with amounts approaching a lethal dose, injury to the hematopoietic tissues (bone marrow, lymph nodes, and spleen) may result. Such hematopoietic damage is reflected in the peripheral blood by leukopenia, thrombocytopenia, and anemia. Lymphoid tissue is involved also, with consequent lymphocytopenia.

b. Symptoms.

(1) Ingestion of food or water contaminated by liquid mustard produces nausea and vomiting, pain, diarrhea, and prostration. Mustard vapor does not significantly contaminate food or water.

(2) Exposure of only the skin to mustard may cause systemic symptoms such as malaise, vomiting, and fever, coming on about the time of onset of the erythema. With severe exposures, particularly by extensive liquid contamination of the skin, these symptoms may be so marked as to result in prostration. Exceptional cases of severe systemic mustard poisoning may also present central nervous symptoms (such as cerebral depression) and parasympathomimetic effects (such as bradycardia and cardiac irregularities). (In animals, cerebral excitation and salivation have been observed, as well as bloody diarrhea with excessive loss of fluid and electrolytes.) Hemoconcentration and hypovolemic shock may occur. It must be emphasized that severe systemic effects only occur when sufficient agent has been absorbed systemically. Lesser mustard exposures do not cause severe systemic effects.

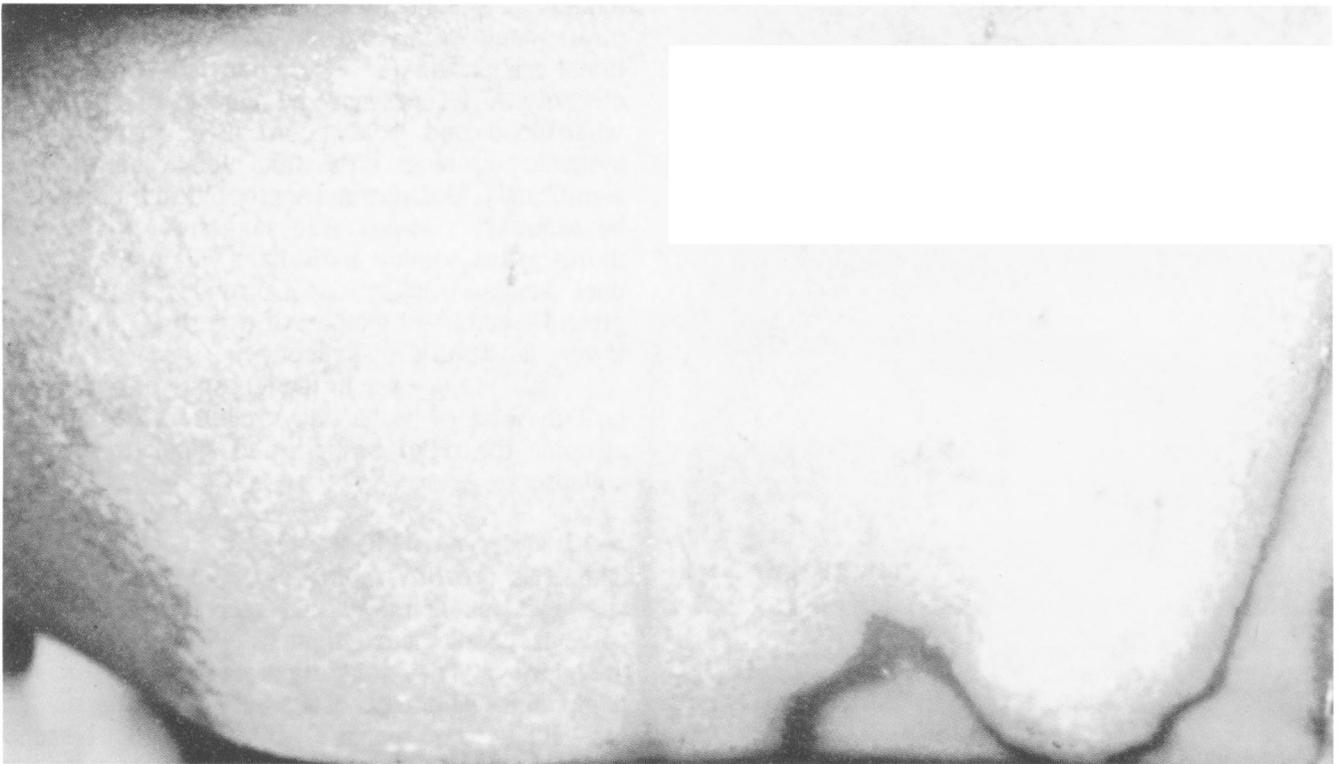
c. Prognosis.

(1) With mild to moderate field exposures to mustard vapor, it is not anticipated that deaths will occur from the systemic effects of the absorbed mustard. However, death may occur from prolonged exposures to high concentrations of mustard vapor or, in instances of extensive liquid contamination of the skin, where decontamination is neglected or unduly delayed. The occurrence of shock or pronounced leukopenia in these cases may be regarded as grave prognostic signs. Bone marrow failure is the most frequent cause of late deaths.

b. Symptoms and Prognosis. Respiratory tract lesions develop slowly and do not reach maximal severity for several days. Symptoms begin with hoarseness, which may progress to loss of voice. A cough (worse at night) appears early and later becomes productive. Fever, dyspnea, rhonchi, and moist rales may develop. The incidence of bronchopneumonia is high. Convalescence is slow; the cough may persist a month or longer. Milder symptoms, like hoarseness, last only 1 or 2 weeks.

c. Treatment of Respirator Tract Injury Due to Mustard. Mild respiratory tract injury, with hoarseness and sore throat only, usually requires no treatment. Cough may be relieved by codeine-containing cough syrups. Laryngitis and tracheitis may be treated symptomatically with steam or sterile cool mist inhalations. If more severe respiratory tract injury is suspected, hospitalization may be advisable. If a bacterial pneumonia occurs, isolation of the specific

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SOURCE: Photograph provided by Iranian News Agency.

Figure 4-13. Casualty with mustard burn of the buttocks.



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Figure 4-14. Casualty with extensive mustard burns.

Click here for Figure 4-15.

leukopenia, hemoconcentration, and shock, every effort should be made to maintain an adequate nutritional status and to replace the loss of fluid and electrolytes. There may be a need to monitor the white blood count, hemoglobin, and platelets in severe systemic poisoning. If the white blood count decreases significantly, isolation and appropriate antibiotics may be necessary. It has been suggested by some authorities that sodium thiosulfate will prevent or reduce damage from mustard, provided that it can be given IV within 20 minutes of exposure. Its efficacy is very doubtful if given later.

(2) Injury due to the ingestion of liquid mustard in food or water may require morphine and atropine for relief of pain and shock therapy for collapse.

4-11. Nitrogen Mustards

The HNs are oily, colorless, pale yellow liquids, sparingly soluble in water but freely soluble in organic solvents. Some have a faint fishy odor, while others are odorless. Their volatility varies with the particular compound. All are persistent but not equally so. The most likely to be seen are HN1 and HN3. Nitrogen mustard (HN1) is more volatile and less persistent than HD but only one-fifth as vesicant to the skin as mustard. Nitrogen mustard (HN3) is less volatile and more persistent and about equal to HD in its vesicant effects. Nitrogen mustards are less readily hydrolyzed than HD. All their hydrolytic products, except the final ones, are toxic.

4-12. Effects of HN on the Eyes

a. Pathology and Symptoms. In single exposures, HN irritates the eye in doses which do not significantly damage the skin or respiratory tract. This irritation appears sooner than that from HD. Mild or moderate exposure causes light smarting and lacrimation within 20 minutes. Thereafter, symptoms may wax and wane until they become persistent about 2 1/2 hours later and reach the maximum in 8 to 10 hours. After more severe exposure, symptoms begin immediately and progress for 24 hours or longer. Mild exposure produces erythema and edema of the palpebral and bulbar conjunctival and superficial, steamy haziness of the cornea. Irritation, lacrimation, deep eye pain, miosis, and photophobia are usually present. After more severe exposure, these symptoms are followed by spotty hemorrhagic discolorations of the iris. The corneal epithelium shows a roughened, lusterless surface, with areas of punctate staining demonstrable by instilling fluorescein. Severe exposure may cause the corneal epithelium to exfoliate. Slit lamp examinations will reveal clouding and edema of the corneal substance extending deep below the Bowman's membrane. Local necrosis of the cornea may rupture the globe.

(2) Severe injury from ingestion of mustard is rare.

d. Self-Protection. Never drink water which has been subjected to chemical attack until it has been certified as fit to drink by the Medical Department. Never eat foods which have been exposed to liquid vesicants, unless in sealed cans or aluminum-laminated pouches (meal, ready to eat (MRE) pouches), until examined by U.S. Army veterinary personnel and certified as safe to eat. Refer to FM 3-5, FM 8-10-7, and TB MED 577 for additional information.

e. Treatment of Systemic Mustard Poisoning.

(1) In the treatment of systemic symptoms, atropine subcutaneously (0.4 to 0.8 mg; NOT the 2 mg automatic injector) may prove useful in reducing the gastrointestinal activity. General discomfort and restlessness may be treated with sedatives but may also be a manifestation of hypovolemic shock from severe systemic injury. In the exceptional cases of severe systemic poisoning with vomiting and diarrhea,

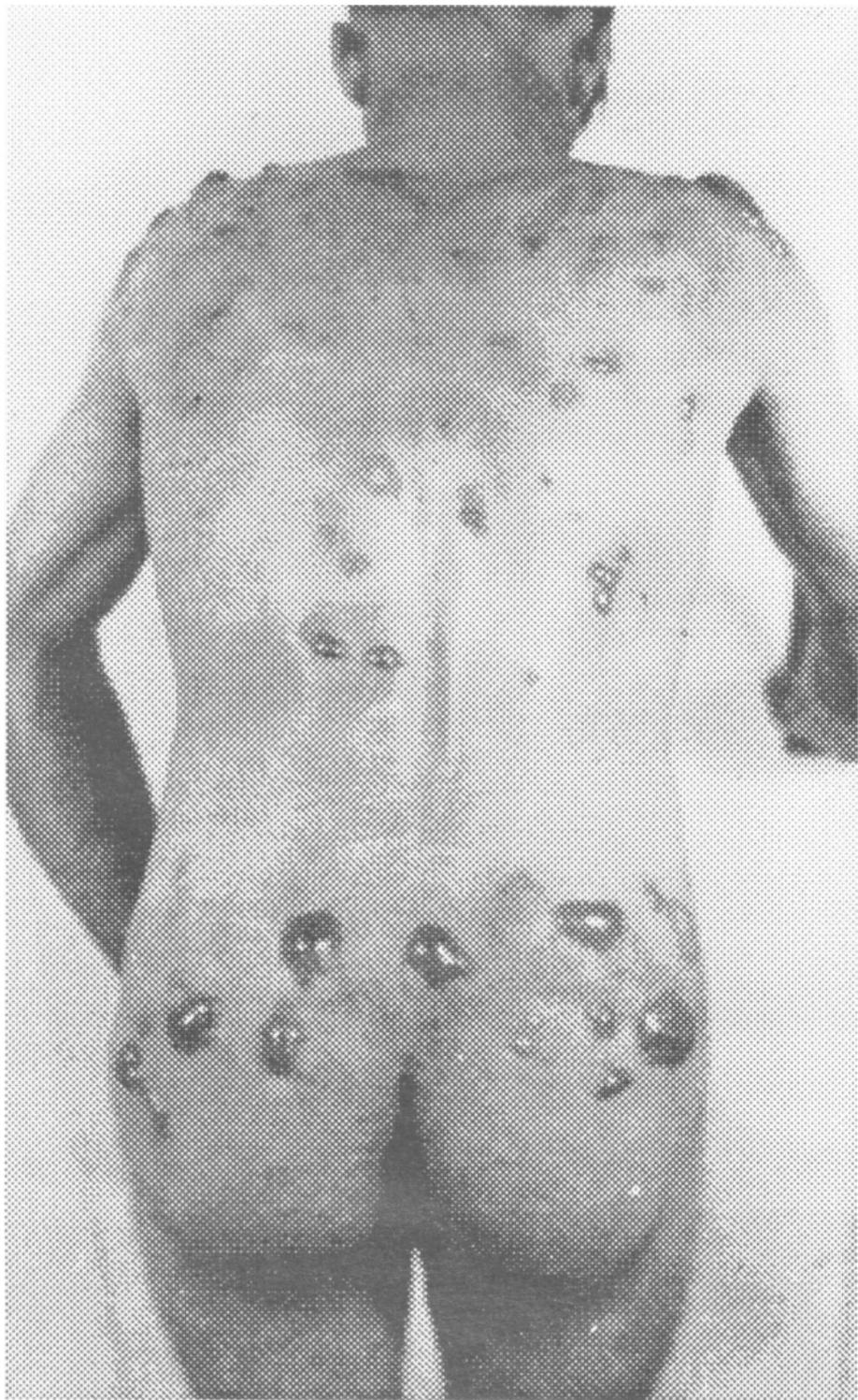


Figure 4-15. Casualty with widespread vesication.



SOURCE: Photograph provided by Iranian News Agency.

Figure 4-16. Casualty with mustard vapor burns in the legs.

[Click here for Figure 4-16.](#)

b. Prognosis. The prognosis in contamination of the eye with any liquid HN is serious unless the agent is removed by immediate decontamination. Mild injury progresses to complete recovery in about 2 weeks; severe injury requires 9 to 12 weeks or longer. The cornea heals by vascularization, and scarring may be expected in severe cases. The iris is frequently left discolored and atrophied.

c. Treatment. The treatment is the same as for HD conjunctivitis (para 4-7 *b* (2)). In general, the lesions and symptoms are more severe, requiring intensive and early treatment. Spasms of the ciliary and orbicular muscles may require frequent instillation of atropine for relief of pain.

4-13. Effects of HN on the Skin

a. Pathology and Symptoms. In mild vapor exposures, there may be no skin lesions. After severe vapor exposure or after exposure to liquid HN, erythema may appear earlier than in HD contamination. There may be irritation and itching as with HD. Later, blisters may appear in the erythematous areas. The skin lesions are similar to those caused by HD.

b. Prognosis. Prognosis is similar to that of HD burns (para 4-8 *b*).

c. Treatment. If early decontamination has not been done, late decontamination should be performed even if erythema is already present and no liquid HN is visible on the skin. The rate of absorption of liquid HN through the skin is slower than that of HD. Therefore, to prevent systemic toxicity, decontamination should be done as early as possible (within 2 or 3 hours after exposure even if it increases the severity of the local reaction).

4-14. Effects of HN on the Respiratory Tract

a. Pathology. The lesions caused by HN are similar to those caused by HD. The lesions decrease in severity down the respiratory tract from the point of entry. In the nose, larynx, and trachea, there may be swelling, erythema, and necrosis of the mucosa, followed by sloughing and fibrinous exudation. Small multiple ulcerations are commonly seen in the pharynx and tonsillar areas. Laryngeal edema and necrosis may lead to respiratory obstruction. In severe cases, the damage may extend to the bronchiole and alveoli.

Although pulmonary edema usually is not massive, secondary pulmonary infection is common.

b. Symptoms. The symptoms are the same as those due to HD; namely, delay in appearance, irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. Fever, dyspnea, rhonchi, and moist rales may develop. Chemical pneumonia may appear after the first 24 hours.

c. Prognosis. Mild inflammation of the trachea is likely to result in a persistent cough. Low-grade fever may persist a week or longer. The prognosis is grave if there is severe respiratory tract involvement. Late deaths due to pneumonia may occur.

d. Treatment. The treatment of respiratory tract involvement is the same as for HD (para 4-9 c).

4-15. Effects of HN on the Gastrointestinal Tract

Following oral administration or systemic absorption, HN injures the intestinal tract. The ingestion of 2 to 6 mg of HN causes nausea and vomiting.

4-16. Systemic Effects of HN

a. Pathology. The most specific effects of HN are on the hematopoietic and lymphoid tissues. These effects follow absorption from the intact skin, respiratory or gastrointestinal tract. In bone marrow, the degenerative changes can be detected within 12 hours and may progress to severe aplasia. The thymus, spleen, and lymph nodes involute rapidly,

with necrosis and phagocytosis of their lymphocytes. This injury is demonstrable in the blood as a transient leukocytosis of a few hours duration, followed by severe lymphopenia, granulocytopenia, thrombocytopenia, and a moderate anemia. The blood picture may show little change other than lymphopenia for 5 to 10 days after exposure, when the white count may fall to 500 cells per cubic millimeter (mm³) or lower. The various HNs differ in their ability to produce these changes.

b. Diagnosis. Diagnosis is based upon a history of exposure, a faint fishy odor on the skin and clothing, and the signs and symptoms described in paragraph 4-15 and a above.

c. Prognosis. Severe leukopenia, thrombocytopenia, and a hemorrhagic diathesis are grave manifestations.

d. Treatment. Frequent white blood cell and hematocrit determinations and examination of peripheral blood smears are necessary to institute proper treatment if anemia and thrombocytopenia occur. Severe vomiting and diarrhea may necessitate IV supplementation with balanced salt solutions or volume expanders. Sedatives, opiates, and atropine are to be used judiciously. The probability of infection with severe leukopenia may be significant and isolation of the patient to protect against infection is appropriate. If infection does occur, it should be vigorously treated with antibiotics.

Section III. ARSENICAL VESICANTS

4-17. Properties

a. These agents are organic dichloroarsines. The main ones are phenyldichloroarsine (PD) and chlorovinylchloroarsine (L). Ethyldichloroarsine and methyldichloroarsine have also been used.

b. All arsenical vesicants are colorless to brown liquids, soluble in most organic solvents but poorly soluble in water. In general, they are more volatile than mustard and have fruity to geranium-like odors. They react rapidly with water to yield the corresponding solid arsenoxides, with concurrent loss of volatility and most of their vesicant properties. As liquids they gradually penetrate rubber and most impermeable fabrics.

c. They are much more dangerous as liquids than as vapors. The liquids will cause severe burns of the eyes and skin, while field concentrations of the vapors are unlikely to cause permanent significant injuries. *Immediate* decontamination is required to remove the liquid agents in time to prevent severe burns, but decontamination is not required for vapor exposure unless pain is experienced. When inhaled, the vapors cause sneezing and may produce irritation of the upper

respiratory tract. More significant respiratory injury is unlikely from ordinary field concentrations of vapor.

4-18. Effects of Arsenical Vesicants on the Eyes

a. Pathology, Symptoms, and Prognosis. Arsenical vesicants cause severe damage to the eye. On contact, pain and blepharospasm occur instantly. Edema of the conjunctival and lids follows rapidly and closes the eye within an hour. Inflammation of the iris usually is evident by this time. After a few hours, the edema of the lids begins to subside, while haziness of the cornea develops and iritis increases. The corneal injury, which varies with the severity of the exposure, may heal without residuals, induce pannus formation, or progress to massive necrosis. The iritis may subside without permanent impairment of vision if the exposure was mild. After heavy exposure, hypopyon may ensue, terminating in necrosis, depigmentation of the iris, and synechia formation. Arsenical vesicants instantly produce a gray scarring of the cornea, like an acid burn, at the point of contact. Necrosis and sloughing of both bulbar and palpebral

conjunctival may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild conjunctivitis due to arsenical vesicants heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

b. Treatment. Treatment is largely symptomatic. In severe cases, the systemic use of morphine may be necessary for control of pain. When the conjunctival edema subsides enough to permit ophthalmic examination, the cornea should be stained with fluorescein to detect erosions, and the iris should be examined for iritis. Atropine sulfate ointment should be instilled to obtain and maintain good mydriasis in all cases with corneal erosions, iritis, cyclitis, or with marked photophobia or miosis. Sodium sulfacetamide solution may be used to combat infection after the first 24 hours. Sterile petrolatum applied to the lid margins will help prevent their sticking together. Irrigations of the eye should be sparing, employing only isotonic or slightly hypertonic solutions (example, 1 percent sodium chloride). Occlusive dressings or pressure on the globe must be avoided.

4-19. Effects of Arsenical Vesicants on the Skin

a. Pathology. Liquid arsenical vesicants produce more severe lesions of the skin than liquid mustard. Contamination of the skin is followed shortly by erythema, then by vesication which tends to cover the entire area of erythema. The surrounding halo of erythema is less noticeable than with mustard blisters, although the two are often indistinguishable. Microscopically, the blister roof is slightly thicker than the mustard blister roof, consisting of almost the complete thickness of the epidermis and showing more complete coagulation necrosis and less disintegrative necrosis than that of the mustard blister. The yellowish blister fluid is slightly more opaque than that of the mustard blister and, microscopically, contains more inflammatory cells. It contains a trace of arsenic but is nontoxic and nonvesicant. Within the corium and subcutaneous tissue, there is deeper injury to the connective tissue and muscle, greater vascular damage, and more severe inflammatory reaction than is exhibited in mustard burns. In large, deep, arsenical vesicant burns, there may be considerable necrosis of tissue, gangrene, and slough.

b. Symptoms. Stinging pain is felt usually in 10 to 20 seconds after contact with liquid arsenical vesicants. The pain increases in severity with penetration and in a few minutes becomes a deep, aching pain. Pain on contact with liquid arsenical vesicants usually gives sufficient warning so that decontamination may be begun promptly and deep burns avoided in conscious victims. After about 5 minutes of contact, there appears a gray area of dead epitheliums resembling that seen in corrosive burns. Erythema is like that

caused by mustard but is accompanied by more pain. Itching and irritation persist for only about 24 hours whether or not a blister develops. Blisters are often well developed in 12 hours and are painful at first, in contrast to the relatively painless mustard blister. After 48 to 72 hours, the pain lessens.

c. Prognosis. The erythema of arsenical vesicants usually recedes more rapidly than the erythema of mustard and with less pigmentation. Small blisters heal in about the same time as those due to mustard. Large lesions may involve deep injuries which heal slowly and require skin grafts. After repeated burns, sensitization to arsenical vesicants occurs, as with mustard.

d. Treatment.

(1) Dimercaprol (British anti-lewisite (BAL)) ointment should be tried on contaminations of the skin which are seen before actual vesication has begun. Any protective ointment already on the skin must be removed before application of the BAL ointment because it destroys the latter. British anti-lewisite ointment is spread on the skin in a thin film, rubbed in with the fingers, allowed to remain at least 5 minutes, and later washed off with water. Occasionally, BAL ointment causes stinging, itching, or urticarial wheals. This condition lasts only an hour or so and should not cause alarm. Mild dermatitis may occur if BAL ointment is frequently applied on the same area of skin. Because of its dermatitis properties, BAL should not be used as a protective (barrier) ointment on unaffected skin.

(2) Some blistering is inevitable in most arsenical vesicant cases which come to the Medical Services. The treatment of the erythema, blisters, and denuded areas is identical with that for similar mustard lesions. A severe third degree burn involving a large surface area is similar to a thermal injury and must be managed by IV resuscitation to correct potential hypovolemic shock. Morphine and splinting of the affected parts may be necessary to relieve pain. Hospitalization is indicated when the involved body surface area is greater than 20 percent. Hospitalization may be indicated when the involved area is less than 20 percent but the depth of the skin involvement appears to be significant. The wound is debrided and treated with 10 percent mafenide acetate burn cream, or silver sulfadiazine topical burn cream.

4-20. Effects of Arsenical Vesicants on the Respiratory Tract

a. Symptoms. The vapors of arsenical vesicants are so irritating to the respiratory tract that conscious casualties will immediately put on a mask to avoid the vapor. No severe respiratory injuries are likely to occur except among the wounded who cannot put on masks and the careless who are caught without masks. The respiratory lesions are similar to those produced

by mustard except that, in the most severe cases, pulmonary edema may be accompanied by pleural effusion.

b. Prognosis. The prognosis is unknown because there have been no known human cases of poisoning by vapors of arsenical vesicants. Extrapolating from animal experiments, the prognosis probably is similar to that for respiratory injury by mustard.

c. Treatment. The treatment is a combination of that for mustard respiratory injury (para 4-9 *c*) and that for the systemic effects of arsenical vesicants (para 4-21 *c*).

4-21. Systemic Effects of Arsenical Vesicants

a. Pathology and Symptoms. Liquid arsenical vesicants on the skin, as well as inhaled vapor, are absorbed and may cause systemic poisoning. A manifestation of this is a change in capillary permeability, which permits loss of sufficient fluid from the bloodstream to cause hemoconcentration, shock, and death. In nonfatal cases, hemolysis of erythrocytes has occurred with a resultant hemolytic anemia. The excretion of oxidized products into the bile by the liver produces focal necrosis of that organ, necrosis of the mucosa of the biliary passages with peribiliary hemorrhages, and some injury of the intestinal mucosa. (Acute systemic poisoning from large skin burns causes pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure in animals (hypovolemic shock)).

b. Prognosis. Burns severe enough to cause shock and systemic poisoning are life-threatening. Even if the patient survives the acute effects, the prognosis must be guarded for several weeks.

c. Treatment.

(1) *Indications for treatment.* The indications for systemic treatment, following exposure to arsenical vesicants by any route, are—

(a) A cough with dyspnea and frothy sputum, which may be blood tinged, and other signs of pulmonary edema.

(b) A skin burn the size of the palm of the hand, or larger, caused by a liquid arsenical vesicant which was not decontaminated within the first 15 minutes.

(c) Skin contamination by an arsenical vesicant covering 5 percent (about 1 square foot) or more of the body surface, in which there is evidence of immediate skin damage (gray or dead-white blanching of the skin), or in which erythema develops over the area within 30 minutes.

(2) *Types of treatment.* The following two types of treatment may be used:

(a) Local neutralization on and within the skin by a liberal application of BAL ointment. The affected skin is to be left covered with a layer of ointment. Remove any other protective ointment before treatment with BAL ointment.

(b) Intramuscular injection of BAL in oil (10 percent).

(3) *Dose of BAL in oil.*

(a) An immediate IM injection of BAL in oil (10 percent) is given deep into the muscles of the buttocks. Take every precaution against injecting into a blood vessel. Dosage must be adjusted to the estimated weight of the patient (0.5 ml per 25 pounds, up to a maximum of 4.0 ml) as follows:

125 pounds	2.5 ml
150 pounds	3.0 ml
175 pounds	3.5 ml
200 pounds and over	4.0 ml

(b) Intramuscular injection of BAL in oil should be repeated at different sites in the buttocks at 4, 8, and 12 hours after the initial injection, for a total of four equal doses.

(c) If pulmonary symptoms or other evidence of severe arsenical poisoning are present, the interval between the first and the second dose may be shortened to 2 hours. In severe cases, subsequent half doses should be given at the rate of one injection per day for 3 to 4 days.

(d) In toxic patients, liberal fluids by mouth (or IV if necessary) and high-vitamin, high-protein, high-carbohydrate diets are indicated. If shock is present, the usual supportive measures (such as IV administration of electrolyte solutions, blood transfusions, or other vascular volume expanders) are indicated.

(4) *Symptoms caused by BAL in oil.* Symptoms caused by BAL in oil appear 15 to 30 minutes after injection and last about 30 minutes. Unless unduly severe or prolonged, they do not contraindicate the full course of treatment. The symptoms may include—

- A feeling of constriction in the throat.
- A sense of oppression in the chest.
- Burning sensation of the lips.
- Mild lacrimation.
- Slight reddening of the eyes.
- Dryness of the mouth and throat.
- Generalized muscular aching.
- Abdominal pain.
- Mild to moderate tenderness and increased muscle tonus at the injection site.
- Mild restlessness and nervousness with sweating of the hands.
- Apprehension.
- Mild nausea and vomiting on eating.
- A transient rise in blood pressure.

4-22. Mixtures of Blister Agents

Arsenical vesicants such as L or PD are often mixed with mustard. These mixtures do not produce more severe lesions than either agent alone, but tend to confuse and make diagnosis difficult.

Section IV. PHOSGENE OXIME

4-23. Properties

a. Phosgene oxime (CX) (chemical name dichloroformoxime) is an example of the class of chemical agents called urticants (or nettle gases). These agents are primarily irritants to skin and mucous membranes, but they differ from mustard by producing an immediate sensation of pain. This pain may vary from a mild prickling to a feeling resembling that caused by a severe bee sting.

b. Phosgene oxime has a disagreeable, penetrating odor. It may appear as a liquid or as a colorless, low-melting point (crystalline) solid, readily soluble in water. Phosgene oxime has an appreciable vapor pressure. A powerful irritant, it is especially effective as a liquid.

4-24. Symptoms and Course of Lesions of Phosgene Oxime

Phosgene oxime is violently irritating to the mucous membranes of the eyes and nose. Even very low concentrations of it can cause lacrimation. Any exposure to liquid or vapor which produces pain will also produce skin necrosis at the site of contact. Within 30 seconds, the area of contact becomes blanched and is surrounded by an erythematous ring. This is followed by the appearance of a wheal within the next half hour. At about 24 hours, the original

blanched area acquires a brown pigmentation. At 1 week, an eschar forms in the pigmented area; and at about 3 weeks, the eschar generally sloughs. Itching may be present throughout the course of healing. Some 20 percent of those exposed to CX may be expected to show healing delayed beyond 2 months.

4-25. Protection from Phosgene Oxime

A properly-fitting protective mask protects the respiratory system. The field protective mask, hood, and chemical protective overgarment protect the body.

4-26. Self-Aid

Because of the rapid reaction of CX with tissue, decontamination will not be entirely effective after pain has been produced. Use the M291 Skin Decontaminating Kit for skin decontamination. If the M291 kit is not available, flush the contaminated area as rapidly as possible with copious amounts of water to remove any CX which has not yet reacted with tissue.

4-27. Treatment for Phosgene Oxime Injury

Treat as any other ulcerated necrotic skin lesion with due consideration of other supportive measures, as indicated.

Section V. DISPOSITION OF PERSONNEL WITH BLISTER AGENT BURNS

4-28. General

a. *Applicability.* This information should be used as guidance by medical personnel confronted with casualties produced by blister agents. It assists medical personnel in the forward area in the disposition, rather than the treatment, of casualties. Therapy for blister agent burns is given in Sections II through IV.

b. *Locations of Burns in World War I Allied Troops.* During 1917-1918, many Allied troops with mustard burns were evacuated needlessly from the front lines. The analysis below shows the locations of mustard burns among 6,980 cases from World War I. (The incidence of permanent blindness among the eye cases was low.)

<i>Location of burns</i>	<i>Percent</i>
Eyes.....	86
Respiratory.....	75
Scrotum.....	42
Face.....	27
Anus.....	24
Legs.....	11
Buttocks.....	10
Hands.....	4
Feet.....	1.5

c. *World War II Experience.* The effects of blister agent burns on the ability of troops to carry out usual military duties were investigated during World War II. In the United States, Canada, Great Britain, and Australia, volunteers ranging from recruits to troops with combat experience were exposed to blister agents. The degree of disability was evaluated on assault courses, route marches, or in simulated combat exercises lasting several days. These observations defined the limitations of casualty production according to type of lesion and are the basis for this guidance. It should not be considered an adequate substitute for clinical observations of blister agent burns in the orientation of medical personnel.

d. Types of Blister Agent Burns.

(1) Two broad types of blister agent patients will not offer a problem in disposition.

(a) The first type consists of those who have sustained burns too minor to impair military effectiveness significantly. These individuals will be classed as noncasualties and returned to their units as soon as possible with or without treatment.

(b) The second type consists of the totally disabled who are incapable of either offensive

or defensive activity regardless of the urgency of the military situations. These individuals will be classed as casualties, promptly treated, and evacuated. Examples are injuries causing total disability and blindness, vesication of extensive areas of the trunk, or vesication of an entire limb.

(2) The intermediate types are partially disabled individuals who can perform only certain kinds of military duties but not others. The disposition of such cases is likely to constitute the main problem. This section is confined to typical injuries within this group. In disposing of these cases, the medical officer will be influenced not only by the severity of the lesions, but also by the military situation, plus the general physical and mental condition of the individual and his military occupational skill.

e. Differentiation Among Injuries According to Agent. For simplicity, no effort is made here to differentiate between the several blister agents that may be used by an enemy. While there are differences between the typical mustard and arsenical vesicant lesions, it is not recommended that the medical officer in the field try to dispose of such cases separately. The diagnostic features of the various blister agent lesions and the therapy peculiar to each are described in Sections II through IV.

4-29. Eye Injuries

a. Sensitivity to Mustard. The eye is more sensitive and more vulnerable to the action of mustard than any other part of the body. About 86 percent of the mustard casualties in World War I had eye lesions to some degree. Exposure for 2 hours to a concentration of mustard, barely perceptible by odor, will produce eye lesions but may not affect the respiratory tract or the skin. There is no immediate symptomatic or local reaction to the absorbed agent. A latent period (which varies with the degree of exposure) precedes the onset of symptoms. This period ranges from 4 to 12 hours after mild exposure and 1 to 3 hours after severe exposure.

b. Classification of Lesions of the Eyes. Eye lesions produced by mustard are divided into the following types.

(1) *Mild.* Of all the cases in World War I, 75 percent had mild burns of the eyes. The mild symptoms include itching, lacrimation, and a sensation of grit in the eye, followed by burning and sometimes by photophobia. There is a hyperemia of both the palpebral and bulbar conjunctival. The reaction in the latter usually begins as a band-shaped area running transversely across the eye with normal white bulbar conjunctival above and below it. Edema of the lids may also be present. Hospitalization is seldom required and recovery occurs in 1 to 2 weeks. Such cases are not classified as casualties.

(2) *Moderate.*

(a) In this group there is complete closure of the eyes from a combination of spasm and swelling of the lids about 3 to 6 hours after exposure. Blepharospasm and blurring of vision develop. There is marked hyperemia and edema of the conjunctival with a prominent interpalpebral band, edema of the lids, mild iritis, and edema of the epitheliums of the cornea producing a roughened appearance like that of an orange peel. The blepharospasm and edema of the lids may be too severe to enable the patients to open their eyes and, so, they may believe they are blind. Miosis occurs early.

(b) A mucoserous discharge is usually present and, although sterile in the early stage, it may cause the lids to stick together, resulting in accumulation of secretions in the conjunctival sac and predisposition to infection. Personnel in this condition are temporarily blind and will be evacuated as casualties. Early and prolonged hospitalization is required, with transfer to the care of an ophthalmologist when possible. Recovery occurs in 1 to 6 weeks, usually without loss of vision. Return to duty will depend upon the extent of residual corneal injury, photophobia, and blepharospasm.

(3) *Severe.*

(a) In this group the latent period is short, lasting 1 to 3 hours. There is deep ocular pain and headache, both of which may be severe, in addition to severe blepharospasm and blurred or dimmed vision. There is marked hyperemia and edema of the conjunctival with a blanched area of ischemic necrosis in the interpalpebral portion, chemosis, and edema of the lids, which the patient cannot open. The epitheliums and stroma of the cornea are damaged. Surface epitheliums is hazy in the early stage and will stain extensively or, in a punctate manner, with fluorescein within 24 hours. After 24 to 48 hours there is also edema of the stroma of the cornea and a deeper haze becomes apparent. Iritis and mucoserous discharge are also present. If the damage is progressive, there may be dense corneal opacification with deep ulceration and vascularization from the limbus. The cases with corneal ulcer heal slowly and may have relapses, some may present perforations into the anterior chamber. These casualties require hospital care and should be evaluated at the earliest possible moment.

(b) Droplets of a liquid blister agent contaminating the eye may produce similar effects. One eye alone may be involved or may be affected more severely than the other. In contrast to droplets of mustard alone, droplets of L or mixtures of L and mustard cause immediate, painful spasm of the lids.

(c) In disposing of eye casualties, medical officers must assure themselves that mild symptoms will not develop into severe inflammation

and temporary blindness within a few hours. Reference to time of exposure and rate of development of symptoms will guide them. If the effects are increasing rapidly, it is advisable to evacuate the case in anticipation of development of disability within the next few hours. Symptoms usually reach a maximum within 6 to 12 hours following exposure.

c. Disposition. The correct disposition of personnel with eye lesions caused by blister agents is less of a problem to the medical officer than those with lesions involving the trunk and limbs. Several hours following exposure of the eyes to mustard, it may be possible to determine whether personnel can remain on duty or will require evacuation.

4-30. Effects on the Respiratory System

a. Circumstances of Exposure. Most respiratory lesions are the result of prolonged exposure to relatively low concentrations of vapor. Severe casualties may result from unrecognized exposure to strong concentrations of mustard vapor. A fatiguing effect on the sense of smell may follow exposure to low concentrations of mustard, thus minimizing detection. Severe lesions occur in individuals who cannot mask, such as unconscious casualties and those with severe injuries to the face or upper extremities.

b. Latent Period. The effects of exposure upon the respiratory tract are characterized by a long latent period before the onset of symptoms, 18 to 36 hours intervening. Because the eyes are more sensitive to the agent than the nasal mucosa but are exposed simultaneously, respiratory effects should be expected to follow vapor burns of the eyes (and face) in unmasked personnel.

c. Mucous Membranes. The local action of mustard vapor on skin and eyes is matched by a similar effect on the mucous membranes of the respiratory tract. Most of the inhaled vapor is absorbed in the larger respiratory passages, including the bronchi, and very little remains to injure the pulmonary parenchyma.

d. Nose. In the nose the earliest visible effect is hyperemia and edema of the mucosa. This is associated with a profuse, thin, mucopurulent discharge. Degenerative changes in the epitheliums, varying in degree according to the extent of exposure, range from small discrete ulcerations to extensive sloughing. Nosebleeding is rare. Nasal injury seldom occurs alone and, as such, is usually a cause for hospitalization.

e. Pharynx. Acute inflammation of the pharynx usually appears 1 to 3 days after exposure to mustard vapor, although there may be a delay of as much as a week. There is mild dryness and soreness of the throat, aggravated by swallowing but rarely accompanied by regional lymphatic enlargement. Pharyngeal and laryngeal lesions may develop without

significant nasal involvement, especially in mouth breathers. Upon inspection, the palate, uvula, tonsils, and pharynx are swollen. Multiple whitish ulcerations may appear, varying in size according to the severity of exposure. Pharyngeal injury is unlikely to occur alone. Secondary infection generally results in regional adenitis.

f. Larynx. Laryngeal involvement commonly results from inhaling mustard vapor, the lesions resembling those of the pharynx. Hoarseness, sometimes progressing to loss of voice, may last 3 to 6 weeks, and in rare instances, longer. This type of lesion alone may not require hospitalization, but it is almost invariably associated with more extensive injury to the respiratory tract.

g. Trachea and Bronchi. In the trachea and bronchi, a similar necrotizing and inflammatory process follows contact with mustard vapor. The exudative process results in the formation of a fairly thick, tenacious pseudodiphtheritic membrane in the larynx, trachea, and large bronchi. It may form a cast of the involved parts sufficient to prove fatal. This condition requires early hospitalization. Milder cases, however, have small ulcerations with hyperemia and edema of the mucosa and hypersecretion of mucous. Respiratory embarrassment, cough, tachypnea, and cyanosis are signs warranting prompt hospitalization.

h. Pulmonary Parenchyma. The action of mustard on the lung may cause chemical pneumonitis. Secondary infection may lead to lobular or lobar consolidation, the course being dominated by the characteristics of the type of pneumonia. Injury due to mustard in no way affects the treatment of the secondary infection. Antibiotics and supportive measures should be used as appropriate.

4-31. Cutaneous Burns

a. Evaluation of Cutaneous Burns. The following observations resulted from evaluation of lesions that have most generally led to disability of personnel exposed to blister agents during field trials and then participated in simulated combat exercises, obstacle course tests, and marches.

(1) Widespread vesication of the trunk produced casualties.

(2) Vesication localized in particular areas of the body produced casualties.

(3) Burns produced by high doses of the blister agent vapor to masked personnel (especially in tropical climates) cause severe casualties. These casualties are produced partly by edema and vesication of the skin and partly by constitutional reactions such as nausea, vomiting, and prostration.

(4) Burns produced by doses of vapor low enough to cause only such skin reactions as mild erythema, edema, burning, and itching usually do not produce casualties.

(5) The stage of development of the lesion must be considered when classifying an individual as a casualty or noncasualty.

b. Trunk and Neck.

(1) *Extensive vesication of the trunk.* All the cases considered under this heading should be evacuated promptly.

(a) Extensive vesication may occur over a large part of the trunk. Intervening areas of skin may be erythematous with pinpoint vesication. These burns are more likely to occur on the back than anteriorly. Some protection is afforded anteriorly by equipment such as webbing and ammunition pouches. The front of the uniform also gives some anterior protection because it does not cling to the body.

(b) Extensive vesication may be followed by fever, nausea, and vomiting. These conditions tend to occur more readily in tropical climates.

(c) Secondary bacterial infection may complicate the clinical course. The medical officer in a forward position is not likely to see infection of vesicated areas because such cases will have been evacuated before secondary infection develops.

(2) *Localized vesication of the trunk.*

(a) Vesication occurring within the natal cleft (between the buttocks) usually requires evacuation of the casualty. Walking becomes difficult, defecation is painful, and dressings require frequent changing. The lesion is usually most intense at the upper end of the cleft. Vesication of the buttocks usually results from sitting on contaminated ground or wearing contaminated trousers for prolonged periods. The vesicated area may extend forward across the perineum to involve the scrotum and the penis.

(b) Trivial burns, such as mild erythema affecting the natal cleft, are not of casualty severity. However, these burns require careful attention because walking or running aggravates the lesions and may break down injured skin.

(c) Single discrete blisters on the buttocks away from the natal cleft do not produce casualties.

(d) Blisters on the trunk generally require protective dressings to avoid friction of clothing. Medical officers must decide whether or not dressings should remain in position during regular duty.

c. Burns Caused by High Doses of Vapor. After exposure to a high dose of mustard vapor, especially under tropical conditions, nausea, vomiting, and symptoms of collapse are usually evident before erythema completely develops. It is important to note that this occurs also among personnel who are masked during exposure. Constitutional symptoms may persist several days, during which burns will increase in severity. Cases of this type should be classed as casualties. Severe vapor burns of the trunk give a generalized erythema but include pale gray areas that eventually vesicate or become necrotic. It is common to see

patches of unaffected skin as a result of protection by overlying equipment.

d. Burns Caused by Low Doses of Vapor. Mild vapor burns cause erythema, itching, and irritation but do not produce casualties. The medical officer should always consider the interval after exposure in relation to the severity of the burn. Mild lesions may represent early phases of severe exposure to vapor. When the period of lapse since exposure is uncertain, rapid development and presence of constitutional symptoms may help to determine the severity.

e. Sensitization Due to Multiple Exposures to Mustard.

(1) Watch for the characteristic appearance of "reexposure" burns. This manifestation may occur in individuals as a result of exposure to mustard 1 to 3 weeks (or more) previously. A small percentage of these casualties will become sensitized to the agent and will react differently, both qualitatively and quantitatively, upon reexposure.

(2) Sensitization will be followed by a more rapid onset of symptoms upon reexposure. Erythema, with or without edema, and pronounced itching and burning usually appear within 1 hour. Lower concentrations of mustard are required to produce effects in a sensitized individual than in a nonsensitized. When erythema and edema result from exposure to a low dose, they generally develop rapidly and subside within 2 to 3 days. Also, vesication heals more rapidly in the sensitized individual.

(3) One of the most frequent manifestations of reexposure in sensitized personnel is the development of a morbilliform rash. Another characteristic reaction is the appearance of eczematoid dermatitis surrounding old lesions, whether or not they are healed. This may last for several days and resembles dermatitis venenata (from poison ivy). Similar phenomena due to sensitization have been known to occur with L and with the nitrogen mustards.

f. Arms.

(1) After treatment, most service members with blister agent injuries of the arms are permitted to continue with their duties. Vesication, when localized, produces little or no disability.

(2) Extensive vesication involving the axillae and the elbows, volar or dorsal aspects, partially impairs the movement of the limbs at those joints. Edema of the surrounding tissue tends to further immobilize the extremities. The dorsal aspects of the elbow and forearm are common sites of severe burns because these parts touch contaminated ground when service members are firing in the prone position. Cases of this type should be evacuated.

(3) Widespread vesication of the arms results in partial disability. Cases of this type should be evacuated.

g. Hands.

(1) Blister agent burns of the hands are often

encountered. These burns tend to cause a degree of disability out of proportion to the size of the lesions. Considerable care and judgment are required in correct disposition.

(2) The palms are more resistant to vesication but not entirely. Blisters affecting the palms are characteristically painful and slow to heal.

(3) A solitary lesion of limited extent may result in little or no disability if treated properly.

(4) Burns from liquid vesicant on the dorsum of the hand result in severe local reactions characterized by intense edema of the backs of the hands and fingers. Pain is characteristic and is intensified by movement of the fingers or wrist. These cases should be regarded as casualties. An individual exposed within the previous 24 hours and reporting for treatment, with apparently tribal blisters, may be totally incapacitated the following day. Sharp erythema of the dorsum of the hand, with beginning vesication 12 to 24 hours after exposure, indicates a lesion that will progress to extensive vesication and edema. Under such circumstances, the individual should be evacuated when first seen.

(5) More commonly, the lesions consist of scattered small vesicles and limited areas of erythema. These lesions can be protected satisfactorily and the individuals returned to duty.

(6) Exposure to vesicant vapor produces diffuse erythema of the dorsum of the hand and wrist. Higher doses cause edema and vesication as well; cases of this type require evacuation.

h. The Lower Extremities.

(1) When the lower extremities are involved, the knees are the most common sites of burns from liquid vesicant. These lesions and those of the ankles often result in incapacitation by interfering with locomotion. Movement of the joints tends to aggravate existing lesions by increasing edema. A further disabling factor is introduced by the wearing of firm dressings applied to mobile joints.

(2) Vesication often spreads over the kneecaps, upward onto the thighs, and down toward the feet. These burns tend to be extensive and are associated with edema often extending halfway up on the thigh and down the leg. Medical officers should evacuate casualties presenting such lesions.

(3) In general, burns of the leg are more incapacitating than burns of the thigh.

(4) It has been shown that the presence of many superficial blisters on the legs and thighs alone is not enough to make a service member incapable of carrying out routine military duties. Individuals with such lesions, having protective dressings, were able to take part in daily marches and routine gun drills. In disposing of these cases, the medical officer will consider the mental and physical status of these individuals, their motivation, their military occupational

specialty, and the tactical situation at the time. Such cases are in the category of partial disability. After suitable dressings have been applied, service members with high morale and robust physiques may be returned to duty.

(5) A relatively small blister or group of blisters situated in the popliteal area may reduce the efficiency of service members so much that they may require evacuation. This is due to aggravation of the lesions by movement of the limbs and interference with ambulation. However, blisters affecting this area are not necessarily casualty producing. (Inflammation, edema, infection, and lesions on other parts of the body should be considered when deciding upon the disposition of an individual.) Available evidence indicates that the mustard blister, size for size, is potentially more incapacitating than a blister from L. This results from the tendency of mustard blisters to be associated with erythema and edema, while the L blisters usually cause less local reaction.

(6) Vesicant lesions develop also near the ankles at the tops of the shoes. Blistered areas occurring at such unprotected points are associated with severe pain due to circulatory impairment and tense edema of the leg. These cases should be evacuated.

(7) Vapor burns of the legs tend to be most aggravated in the popliteal spaces. Pinpoint vesication is often found here. Higher doses cause intense erythema with scattered areas of vesication over the entire surface of the leg. Such lesions are invariably casualty producing and are generally accompanied by severe burns elsewhere, frequently with severe systemic effects.

(8) Mild vapor burns of the legs produce irritation and itching common to all widespread vapor burns. While these effects are troublesome, they are not casualty producing. Service members with mild vapor burns should be returned to duty.

(9) Extensive vesication of the feet is uncommon. The soles are protected by shoes and are more resistant to vesication. Burns on the dorsal aspect of the foot are often associated with local reactions like those seen on the backs of the hands. Individuals with these burns, especially if widespread over the foot, find it difficult or impossible to wear shoes and will require evacuation. Small discrete blisters may be of noncasualty significance. These blisters may be effectively protected so as to allow wearing of shoes and walking with little discomfort.

i. The Genitalia.

(1) The genital region, in addition to the eyes and the respiratory tract, is highly sensitive to blister agent burns. In World War I such burns produced many casualties. The majority of these burns were caused by vapor. Despite present methods of protection against blister agents, medical officers (especially in tropical areas) may be confronted with many such burns.

(2) Vapor is a more common cause of burns affecting the male genitalia than a liquid agent. Erythema may not be conspicuous. The most prominent feature of the burn is the edema involving the penis and scrotum. Fluid accumulates most readily in the prepuce, distending its entire circumference and forming a characteristic semitranslucent ring around the corona. In more severe cases, the entire body of the penis becomes edematous. Female genitalia are affected in a similar manner, the most prominent feature being edema of the labia. In severe burns, fluid may also accumulate in the labia.

(3) These lesions cause apprehension as well as physical discomfort. Occasionally, vesication is superimposed on the edema. Spotty ulceration is not infrequent at the tip of the prepuce where it may become secondarily infected. In severe cases associated with marked edema, retention of urine may result from both mechanical and reflex effects.

(4) In mild cases, objective changes of the scrotum often tend to pass undetected due to the normal pigmentation, elasticity, and looseness of the skin. Even considerable edema may not be enough to reveal its presence. In severe cases the scrotum may become grossly enlarged. The rugae may be partly or completely obliterated. Pinpoint vesication may occur, usually after a lapse of a few days. The scrotum tends to break down resulting in small, painful ulcers and fissures.

(5) Burning is the most prominent subjective symptom in involvement of the genitalia. Apprehension and anxiety are distressing during the presence of the objective changes described in (3) and (4) above. As edema decreases, itching starts and may persist long after the acute effects have subsided. Sometimes this itching is intolerable. The scrotum may continue to crack and ulcerate for a considerable period, causing pain and irritation.

(6) Mild exposure of the genital region typically is followed by a delay in the development of symptoms, often for as long as 4 to 10 days.

(7) Patients with mild burns without edema or vesication, but who complain of irritation and burning, may be safely returned to duty following treatment. In disposing of mild burns of the genitalia, medical officers must assure themselves that the symptoms are not too early to be judged with finality. Severe cases should be evacuated on the basis of the apprehension that may be suffered as well as the physical discomfort involved.

j. Systemic Effects of Cutaneous Burns.

(1) Severe systemic effects due to blister agents probably will be encountered only with disabling skin lesions. The medical officer should be familiar with the signs and symptoms. These include anorexia, nausea, vomiting, depression, and fever, and are far more prone to occur in hot than in

temperate climates. Malaise and nausea generally are the first reactions and may progress either to mild, transient vomiting or to severe, persistent vomiting and retching. Anorexia may be the only complaint in mild reactions. The actual time of onset of symptoms is 4 to 12 hours after exposure, and symptoms often occur before skin injury is manifest. No rule can be given for the duration of systemic symptoms, although casualties usually have recovered from severe vomiting within 24 to 36 hours. Anorexia and nausea may persist for a longer time.

(2) The temperature may remain elevated for several days. Mental depression may follow mustard burns and persist for several days.

(3) Service members with systemic reactions will generally be casualties, particularly in view of the probability of associated extensive skin burns. Such cases should be evacuated quickly.

k. Secondary Bacterial Infection in Blister Agent Burns. This paragraph considers the problem of secondary bacterial infection after blister agent injuries only as it influences the disposition of affected personnel in forward positions. For management and treatment of such cases, see sections II through IV.

(1) Secondary bacterial infection may result if adequate wound care is not given. Compared to the incidence of infection in thermal and traumatic wounds, the incidence of sepsis in mustard lesions is remarkably low, according to observations made at experimental installations.

(2) Secondary infection becomes manifest several days after injury. Medical officers are not likely to see secondary infection with extensive blister agent burns in the front lines because severe cases will have been evacuated early.

(3) Infection of small lesions does not require evacuation. Infection of multiple lesions is likely to be an indication for evacuation, particularly if constitutional effects are associated. Infection is particularly disabling when it involves the feet, hands, genitals, or tissue overlying the joints of the limbs.

(4) Secondary infection is more likely to occur in severe, rather than mild, vapor injury to the respiratory tract. Severe respiratory symptoms will almost always be associated with severe eye effects. Respiratory lesions may not develop for several days, and by then the individual should have been evacuated as an eye casualty.

(5) Secondary infection is uncommon as a sequel to mild degrees of mustard conjunctivitis and ordinarily would not prevent an individual from continuing duty.

(6) Mild conjunctival burns may be associated with pharyngitis, laryngitis, and tracheitis, increasing in severity for several days. Occasionally, more extensive respiratory infection may ensue.