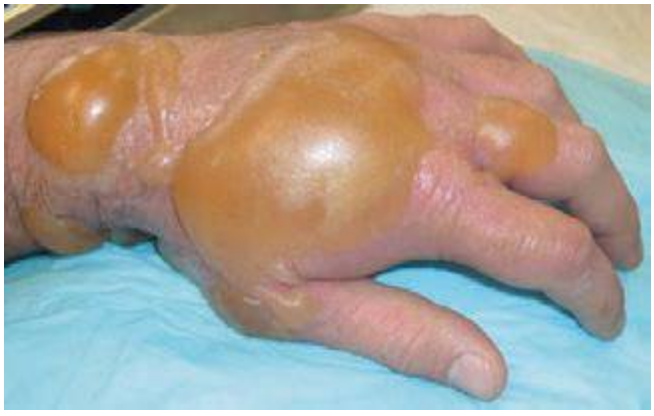

FIGURE 262e-1 Large bulla formation from mustard burn in a patient. Although the blisters in this case involved only 7% of the body surface area, the patient still required hospitalization in a burn intensive care unit.

FIGURE 262e-3 World War I photograph of troops exposed to sulfur mustard vapor. The vast majority of these troops survived with no long-term damage to the eyes; however, they were rendered effectively blind for days or weeks.

The primary airway lesion is necrosis of the mucosa with possible damage to underlying smooth muscle. The damage begins in the upper airways and descends to the lower airways in a dose-dependent manner. Usually the terminal airways and alveoli are affected only as a terminal event. Pulmonary edema is not usually present unless the damage is very severe, and then it becomes hemorrhagic.

The earliest effects of mustard—and perhaps the only effects of a low concentration—involve the nose, sinuses, and pharynx. There may be irritation or burning of the nares, epistaxis, sinus pain, and pharyngeal pain. As the concentration increases, laryngitis, voice changes, and nonproductive cough develop. Damage to the trachea and upper bronchi leads to a productive cough. Lower airway involvement causes dyspnea, severe cough, and increasing quantities of sputum. Terminally, there may be necrosis of the smaller airways with hemorrhagic edema into surrounding alveoli. Hemorrhagic pulmonary edema is rare.

Necrosis of airway mucosa causes "pseudomembrane" formation. These membranes may obstruct the bronchi. During WWI, high-dose mustard exposure caused acute death via this mechanism in a small minority of cases (Fig. 262e-2).

The eyes are the organs most sensitive to mustard vapor injury. The latent period is shorter for eye injury than for skin injury and is also dependent on exposure concentration. After low-dose vapor exposure, irritation evidenced by reddening of the eyes may be the only effect. As the dose increases, the injury includes progressively severe conjunctivitis, photophobia, blepharospasm, pain, and corneal damage (Fig. 262e-3). About 90% of eye injuries related to mustard heal in 2 weeks to 2 months without sequelae. Scarring between the iris and the lens may follow severe effects; this scarring may restrict pupillary movements and predispose victims to glaucoma. The most severe damage is caused by liquid mustard. Extensive eye exposure can be followed by severe corneal damage with possible perforation of the cornea and loss of the eye. In some individuals, latent chronic keratitis, sometimes associated with corneal ulcerations, has been described as early as 8 months and as late as 20 years after initial exposure.

The mucosa of the gastrointestinal tract is susceptible to mustard damage from either systemic absorption or ingestion of the agent. Mustard exposure in small amounts will cause nausea and vomiting lasting up to 24 h. The mechanism of the nausea and vomiting is not understood, but mustard does have a cholinergic-like effect. The CNS effects of mustard also remain poorly defined. Exposure to large amounts can cause seizures in animals. Reports from WWI and from the Iran–Iraq war described people exposed to small amounts of mustard acting sluggish, apathetic, and lethargic. These reports suggest that minor psychological problems could linger for ≥1 year.

The cause of death in the majority of mustard poisoning cases is sepsis and respiratory failure. Mechanical obstruction via pseudomembrane formation and agent-induced laryngospasm is important in the first 24 h, but only in cases of severe exposure. From the third through the fifth day after exposure, secondary pneumonia due to bacterial invasion of denuded necrotic mucosa can be expected. The third wave of death is caused by agent-induced bone marrow suppression, which peaks 7–21 days after exposure and causes death via sepsis.

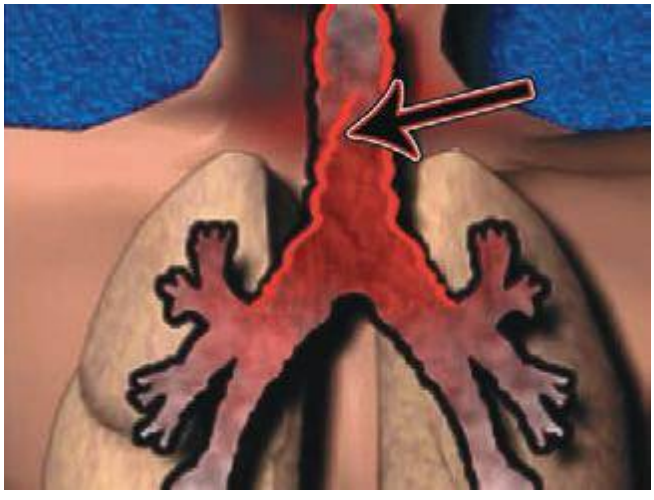

FIGURE 262e-2 Schematic diagram of pseudomembrane formation as is seen in high-dose sulfur mustard vapor inhalation exposure. In World War I, severe inhalation exposure often caused death via obstruction of large airways.

TREATMENT SULFUR MUSTARD INTOXICATION

A patient severely ill from mustard poisoning requires the general supportive care provided for any severely ill patient as well as the specific care given to a burn patient. Liberal use of systemic analgesics, maintenance of fluid and electrolyte balance, provision of nutrition, administration of appropriate antibiotics, and other supportive measures are necessary (Table 262e-2).

The management of a patient exposed to mustard may range from simple (as in the provision of symptom-based care for a sunburn-like erythema) to complex (as in total management of a severely ill patient with burns, immunosuppression, and multisystem involvement). Before raw denuded areas of skin develop, especially with less severe exposures, topical cortisone creams or lotions may be of benefit. Some very basic research data point to the early use of anti-inflammatory preparations. Small blisters (<1–2 cm) should be left intact. Because larger bullae eventually will break, they should be unroofed carefully. Denuded areas should be irrigated three or four times daily with saline, other sterile