
Chemical Terrorism

Fact Sheet

Vesicants/Blistering Agents – Dichloroarsine Compounds: Lewisite

Protective Equipment/Detection

Since ordinary clothing and surgical protective gear offer no protection against **lewisite**, special equipment including a respirator, NBC protective suit, gloves and overboots are required.

The detection of **lewisite** is helped by the fact that it forms colored products with many reagents. Draeger™ tubes are available which react with organic arsenicals. However, there are no automatic field detectors that will detect it.

Decontamination

Decontamination of the Skin - is based on physical adsorption or on the combination of physical adsorption and chemical inactivation. Physical adsorption is achieved by adsorbing powders (flour, talcum powder, Fullers earth), while chemical inactivation is achieved by incorporating chlorinating compounds (chloramines, household bleach) into adsorbing powders, ointments, solutions or organic solvents. **Lewisite** should not be decontaminated with water, except for the eyes, as this may spread the agent.

Decontamination of Mucous Membranes and Eyes - The substances used for skin decontamination are too irritating for use on mucous membranes and the eyes. In this case the affected tissues should be flushed immediately with water. The eyes can be flushed with copious amounts of water, isotonic sodium bicarbonate (1.26%), or saline (0.9%).

Signs and Symptoms

Like mustard agents, **lewisite** can burn and blister any part of the body it contacts, typically acting on the eyes, mucous membranes, lungs, and skin. It can be distinguished from mustards by the immediate pain upon contact, since, unlike mustards, **lewisite** has no latency period. Injuries from **lewisite** are expected to be less severe than those of the mustards, because the immediate burning and lung irritation leads to the prompt use of protective gear and decontamination.

Eyes: In animal tests, **lewisite** caused an immediate edema of the lids, conjunctivae, and cornea, with an early meiosis. There is also early, severe involvement of the iris and ciliary body, followed by depigmentation and atrophy of the iris stroma with severe exposures. Eye injuries with **lewisite** tend to be less severe than with mustard agents, because the immediate blepharospasm and edema helps prevent prolonged exposure. However, eye injuries can be serious, with pannus formation, massive necrosis, and ultimately blindness. A droplet of 0.001 ml can perforate and destroy the eye.

Skin: **Lewisite** begins to burn upon contact. Burns may be the result of either vapor or liquid exposure. Erythema is evident within 15-30 minutes, followed by painful vesication within a few hours. Unlike mustards that produce groups of small blisters over erythematous areas, **lewisite** blisters start small and expand to cover the entire erythematous area. Maximum blistering takes up to 4 days to occur. Unlike the mustard agents, pigment changes do not occur and minor lesions heal more readily. The blister roof contains the entire epidermal layer and is less fragile than mustard blisters.



Lewisite blister on wrist
Photo Courtesy Department of Defense

Chemical Overview

Lewisite is the representative chemical of a group of vesicant, or blistering, agents known as dichloroarsines. **Lewisite** is 2-chlorovinyl-dichloroarsine. In pure form, **lewisite** is a colorless, odorless, oily liquid that is heavier than mustard(HD), poorly soluble in water but very soluble in organic solvents. It usually contains impurities that give it a brown to blue-black color and the odor of geraniums. Although first synthesized late in WWI, its use in battle has never been verified. It hydrolyzes rapidly and maintaining biologically active concentrations in high humidity is difficult. It does, however, remain fluid at low temperatures, making it better than the mustards for winter dispersal. Acute toxicity levels for humans are not well defined but 0.05-0.1 mg/cm² produces erythema, 0.2 mg/cm² produces vesication and a 15-minute exposure to a vapor concentration of 10 mg/m³ produces conjunctivitis. About 30 drops (2.6 mg), applied to the skin and not decontaminated, would be expected to kill an average man through systemic toxicity. With inhalation, the LC₅₀ in man is estimated to be about 1500 mg min/m³.

Prophylaxis

There is no prophylactic treatment for **lewisite**. You must rely upon the physical protection of a protective respirator and special clothing, followed by immediate decontamination when exposed.

Signs and Symptoms (Continued)

With exposure to liquid **lewisite**, deep necrotic lesions may be expected. The necrosis that occurs is a coagulative one with deeper injury to the connective tissue and muscle, greater vascular damage, and a more severe inflammatory reaction than is seen in mustard burns. In large, deep, **lewisite** burns, there may be considerable necrosis, gangrene and slough.

Respiratory Tract: **Lewisite** causes irritation and congestion from the nasal cavity to the lower airways. Symptoms start with rhinorrhea, burning pain in the throat and hoarseness of the voice. Airway secretions and fragments of necrotic epithelium may cause obstruction, with resultant rales and marked dyspnea. Pseudomembrane formation and pulmonary edema are seen with severe cases. Bronchopneumonia is a major complication to be avoided. If the inhaled dose is sufficiently high, the victim dies in a few days from pulmonary edema, mechanical asphyxia due to obstruction, and/or sepsis.

Systemic Action: Systemically absorbed **lewisite**, by any route, may cause liver toxicity and systemic arsenic toxicity with diarrhea, neuropathy, nephritis, hemolysis, a true hemolytic shock, and encephalopathy. "Lewisite shock" has also been described, and is the result of protein and plasma leakage from the capillaries, with subsequent hemoconcentration and hypotension. Subnormal body temperature, restlessness, hypotension, and T-wave elevations are occasionally seen. Gastrointestinal symptoms include nausea, vomiting, diarrhea, anorexia, and abdominal pain. Generalized weakness, muscle cramping, and red or green colored urine have also been reported.

The indications for systemic treatment, following exposure by any route, are: (1) cough with dyspnea and frothy sputum, which may be blood tinged, or other signs of pulmonary edema; (2) a skin burn the size of the palm of the hand or larger, which was not decontaminated within the first 15 minutes, and (3) skin contamination covering 5% or more of the body surface, with evidence of immediate damage (grey or dead-white blanching of the skin), or where erythema develops over the area within 30 minutes.

Treatment

The aim of therapy is to relieve symptoms, prevent infections, and promote healing. Since **lewisite** is an arsenical compound, treatment of systemic arsenic poisoning is achieved using Dimercaprol (British Anti-Lewisite, BAL, 2,3-dimercaptopropanol), which acts as a chelator by binding arsenic. It is available for deep, intramuscular injection, which itself may be painful, and has significant systemic toxicity. The standard dosing regimen is 3-5 mg/kg IM every 4 hours for four doses. Dosing depends on body weight and the severity of the symptoms. Recently, two water-soluble analogues of dimercaprol have become available, meso-2,3-dimercaptosuccinic acid (DMSA) and 2,3-dimercapto-1-propanesulfonic acid (DMPS). They are less toxic, water soluble, and can be given orally. Dimercaprol treatment is generally reserved for severe exposures exhibiting pulmonary edema or shock.

Caveats:

Eyes: Do not use topical anesthetics for pain relief, as they may increase corneal damage. Use systemic, narcotic analgesics when needed. Similarly, do not bandage the eyes; use dark, protective goggles.

Skin: Involvement of greater than 20% of the skin, or the presence of deep, severe burns, requires hospitalization. (NB. Dimercaprol ointment for the skin is no longer available.)

Ingestion: Do not induce vomiting or attempt to neutralize. Activated charcoal is of no use, but milk, as an alkali, will help hydrolyze the agent.

Additional information and references available at <http://www.bioterrorism.slu.edu>



Long-term Medical Sequelae

There is no real data from which to predict long-term medical consequences of **lewisite** exposure. It is believed that chronic pulmonary disease and the persistence of acute, severe injuries to the eye are the main long-term effects. **Lewisite** does not appear to be mutagenic, teratogenic, or carcinogenic.

Environmental Sequelae

Lewisite is 7 times less persistent than the mustard agents in the environment. It hydrolyzes rapidly and is not found in soil. Lewisite oxide, however, when found in the soil demonstrates legacy contamination.

Disclaimer

Information contained in this fact sheet was current as of August 2002, and was designed for educational purposes only. Medication information should always be researched and verified before initiation of patient treatment.

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