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Characteristics and Effects of Chemical Weapons in Chemistry

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ABSTRACT

Chemical agents should be considered in key incident planning. Consider exposure to chemical agents in any casualty with unexplained and unusual symptoms. Poisoning with many chemical agents, especially nerve agents, can be treated when diagnosed early. Protective equipment must be worn if there is suspicion that chemical agent remains in the local environment. Move casualties from contaminated environment to well ventilated area to give first aid. Decontamination of the casualty involves removal of clothing, shaving contaminated hair, and irrigation with water or dilute sodium hypochlorite to remove residual agent from skin. In this review, we have focused on the agents that pose the greatest threat, recognising chemical weapons injuries, and the principles of management.

Keywords: characteristics, chemical agents, effects, skin decontamination, terrorists agents

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INTRODUCTION

Chemical warfare has been widely condemned since it was first used on a massive scale during the First World War. Chemical weapons are cheap, can cause mass casualties, and are relatively easy to produce, even by developing nations. They have been used in many conflicts during the 20th century, most recently by Iraq during the Iran-Iraq war, as well as in terrorist attacks [1].

Experts believe that terrorist use of chemical agents is an event with low probability, but potentially high consequences. While terrorist groups may or may not have an increased interest in chemical agent acquisition and use, the domestic vulnerability of the United States to chemical attack remains an issue. Both the United States and Russia have signed and ratified the Chemical Weapons Convention (CWC), and are reducing, and eventually eliminating, their chemical weapon stockpiles [1].

Policy approaches to reducing chemical agent vulnerability have generally treated them as a group, rather than addressing specific agents. Additionally, military and civilian chemical agent detection has developed with little coordination, so that civilian toxic industrial chemical kits and military chemical weapons detectors have varying sensitivities and detection capabilities. Treatments for chemical exposure vary on a chemical by chemical basis [2]. Because comparatively few individuals have been exposed to modern chemical weapons, health care providers have limited practical experience in treatment of chemical casualties, especially among civilians. While national efforts to reduce vulnerability to terrorist chemical agent use continue, it is not clear whether these efforts address the risks from those specific agents that pose the greatest danger. This report describes the different types of chemical weapons and toxic industrial chemicals, their availability, treatment, and detection [2].

DEFINITION

The North Atlantic Treaty Organization's definition of a chemical agent is "a chemical substance which is intended for use in military operations to kill, seriously injure or incapacitate people because of its physiological effects." Classic chemical weapons and biological weapons (such as anthrax or plague) are considered to form two ends of a spectrum with "chemicals of biological origin" (such as botulinum toxin or ricin) lying between these two extremes [3]. Chemical weapons can be classified according to their mode of action or by the time they remain active in the environment (persistence) and lethality (Figure 1).

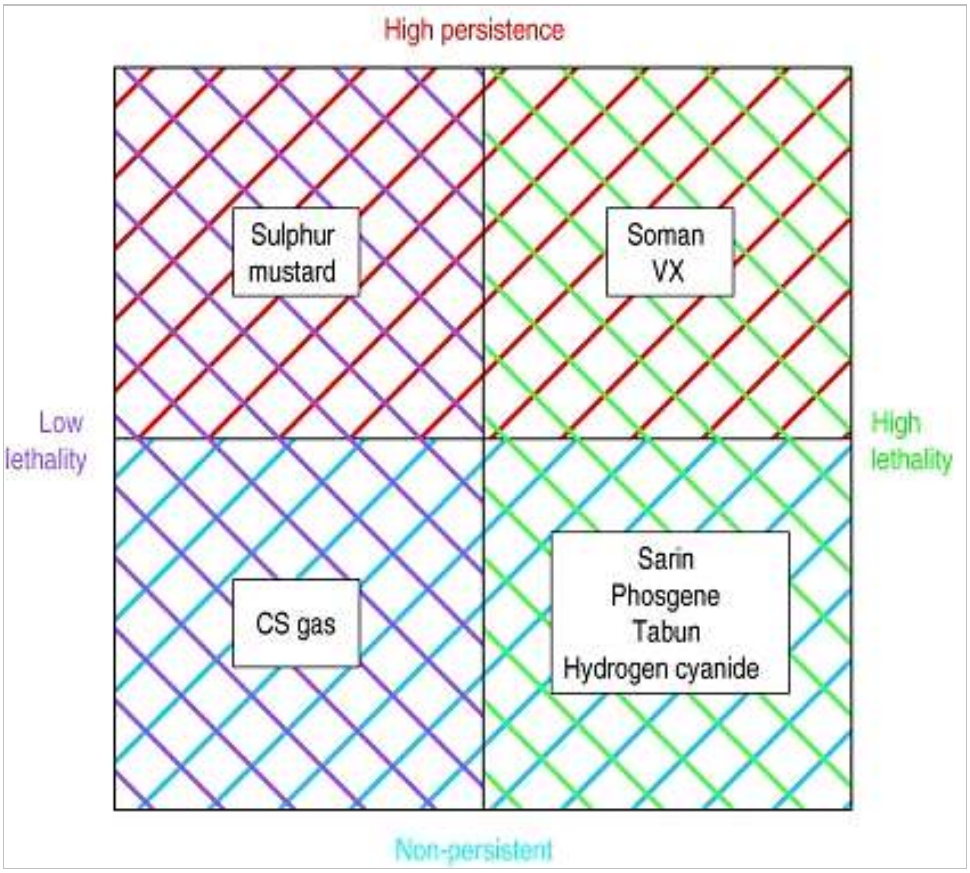

Fig. 1. *Classification of chemical weapons by persistence and lethality.*

Types of Chemical Agents

Chemical agents are, for the purpose of this report, chemicals posing exceptional lethality and danger to humans. Some chemical agents are toxic industrial chemicals used for commercial purposes, while others are chemicals developed predominantly as weapons.

Different chemical weapons cause different symptoms in and injuries to their victims. Because of this range of potential effects, identifying the chemical agent is a key step to determining the most effective treatment. Also, chemical weapons may produce their effects by multiple different exposure routes, for example by skin contact or by inhalation. As a consequence, depending on the encountered chemical, those affected must employ different protective equipment and approaches; for example, a gas mask alone does not provide sufficient protection against chemicals that can damage through skin contact.

Military planners generally categorize chemical agents into at least four classes: nerve, blister, choking, and blood agents. This method organizes chemical agents by their biological effects. Modern militaries have generally focused on nerve and

blister agents as weapons. Several choking and blood agents are chemicals widely used in industrial processes [4–6].

NERVE AGENTS

Nerve agents are a particularly toxic group of organophosphate compounds first synthesized in Germany before the Second World War. All are liquids at room temperature and produce a vapor capable of penetrating the skin, respiratory epithelium, and cornea. The liquid can be absorbed through intact skin and also through the gut after ingestion of contaminated food. VX **(**O-ethyl-S-[2(di-isopropylamine) ethyl] methylphosphoethioate) has greater potency but lower volatility than other nerve agents. All nerve agents act by inhibiting the enzyme acetylcholinesterase, which breaks down the neurotransmitter acetylcholine (Figure 2).

The route of exposure determines which clinical features appear first. After inhalation of vapor, respiratory symptoms, dimming of vision and meiosis are generally the first clinical features to appear. Local sweating and fasciculation of local muscle groups may occur initially after skin absorption of liquid or vapor. If sufficient agent has been absorbed by any route systemic effects will occur [7].

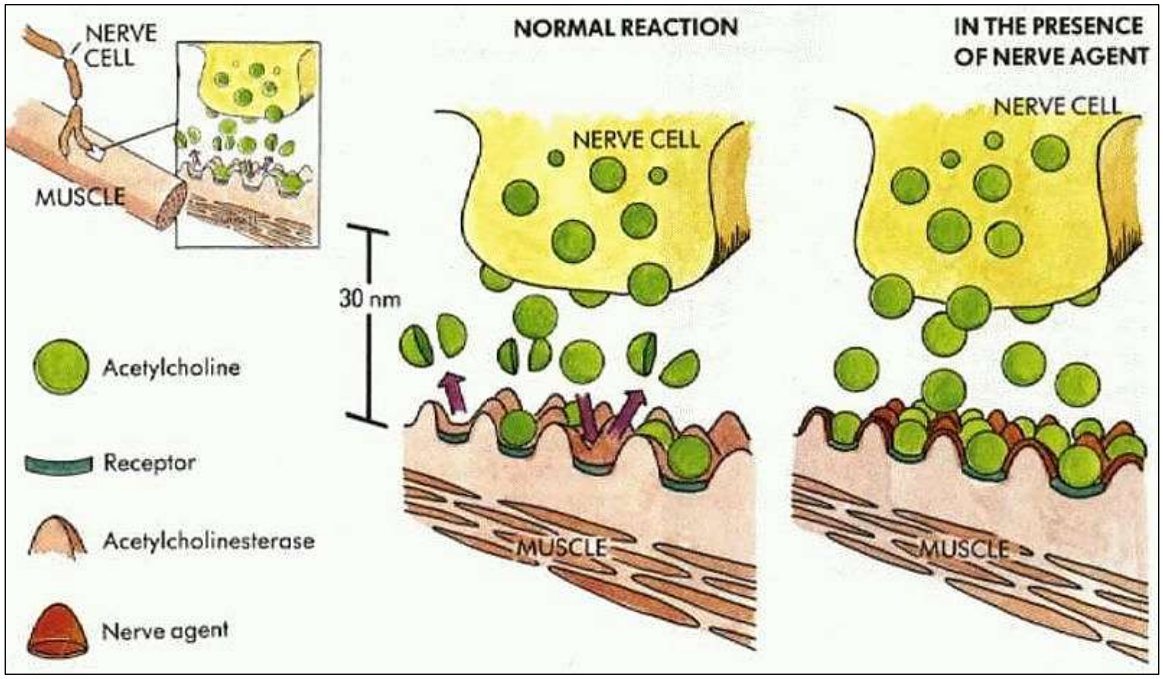

Fig. 2. *Effects of nerve agent.*

Treatment of poisoning

Poisoning with nerve agents is always a serious medical emergency because the agents act rapidly and profoundly. Patients with compromised airways and respiratory failure will require immediate endotracheal intubation and positive pressure ventilation. Aggressive suctioning may be needed to remove increased bronchial secretions. Whole blood cholinesterase concentrations give a broad indication of the degree of poisoning, but the principle should be to treat the patient not the depression of acetylcholinesterase.

Treatment includes:

- Anticholinergics to antagonise the muscarinic effects.
- Oximes to reactivate the inhibited acetylcholinesterase and antagonise the nicotinic effects.

- Prophylactic anticonvulsants to prevent seizures.

Immediate treatment with atropine is essential in patients with systemic poisoning. The total dose depends on the degree of poisoning. Intravenous atropine should be given in 2 mg aliquots every 3-5 minutes until the patient is atropinised. Signs of atropinisation include dry mouth, correction of miosis, reduced sweating, and correction of bradycardia. Atropine may need to be continued at 2 mg/hour for at least 24 hours. Complications of atropinisation include euphoria, arrhythmias, delirium, and heat stress.

Although atropine antagonises the muscarinic mediated effects of nerve agents, oximes are used to reactivate acetylcholinesterase at nicotinic sites. Pralidoxime mesilate is currently licensed for use in the United Kingdom. Oximes must be given early as their efficacy declines with time; spontaneous dealkylation (or ageing) of the inhibited acetylcholinesterase makes it resistant to reactivation. Pralidoxime mesilate 30 mg/kg should be given by slow intravenous injection at 15 minutes intervals to a maximum of 2–4 g as soon as possible.

Control of convulsions has been shown to increase survival and reduce subsequent morbidity in animal studies. Patients should be given 5 mg diazepam by any route before the onset of convulsions [8].

BLISTER AGENTS

Sulphur mustard is a yellow oily liquid at room temperature with a faint odour of mustard or garlic. It remains liquid in cold damp conditions but evaporates rapidly in a warm dry environment to produce a vapour that can penetrate ordinary clothing. Sulphur mustard is a bifunctional alkylating agent, reacting readily with most biological molecules including proteins and nucleic acids. In conflict, it has been used to incapacitate large numbers of soldiers. Injuries are usually non-fatal (mortality from exposure to sulphur mustard was only 3% during the First World War) but long term.

Sulphur mustard is a powerful blistering agent; a 0.1 ml drop of pure sulphur mustard contains 20000 times the dose required to blister skin. Symptoms may not develop until 12-24 hours after exposure. Erythema precedes the development of large, thin walled, pendulous blisters that rupture easily.[8] The burns tend to be partial thickness but are deeper in the warm, moist areas of the body such as the axillae and groin.[9] In most people with inhalation injuries, initial irritation of nasal mucosa and airways is followed by the development of bronchitis, which resolves in four to six weeks. In severe exposure, necrosis of the respiratory epithelium leads to formation of a pseudomembrane, bronchial plugging, and bronchopneumonia. Systemic absorption produces nausea, vomiting, hypotension, bradycardia, and, after initial leucocytosis, profound leucopenia. Death, although uncommon, is usually due to overwhelming infection [9].

Sulphur mustard lesions

No specific treatment exists for mustard lesions. Treatment is therefore aimed at relieving symptoms, preventing infection, and promoting healing. Eye lesions are usually mild and heal completely in about two weeks. Treatment consists of topical antibiotics and careful cleaning with saline to prevent the eyelids sticking. Inhalation of vapour may cause severe bronchopneumonia in the worst cases. Physiotherapy, oxygen, antibiotics, and mechanical ventilation are the mainstays of treatment.

Patients with large burns should be resuscitated in line with thermal burn protocols (Figure 3). However, in contrast to thermal burns, the fluid losses do not

occur until the blisters form and the fluid lost is a transudate, which means protein losses are less.

Tense or broken blisters should be deroofed and dressed with silver sulfadiazine. Partial thickness sulphur mustard burns heal much more slowly than thermal burns of similar depth, taking at least 12 weeks to heal if treated conservatively.

This is thought to be due to cellular alkylation affecting normal wound healing processes. Standard excision and grafting of sulphur mustard burns does not reduce healing time in an animal model, but early dermabrasion and laser ablation may be beneficial. Pain can be severe, and patients may benefit from chlorpromazine in addition to standard analgesics [10].

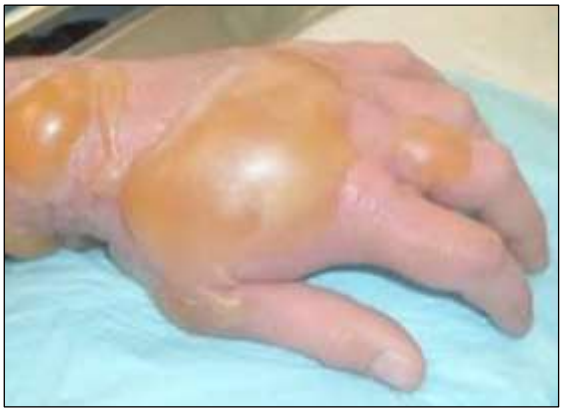

Fig. 3. *Effects of Blister agent.*

CHOKING AGENTS

A pulmonary agent, or choking agent, is a chemical weapon agent designed to impede a victim's ability to breathe. They operate by causing a build-up of fluids in the lungs, which then leads to suffocation. Exposure to the eyes and skin tends to be corrosive, causing blurred vision and severe deep burns (Figure 4) [11].

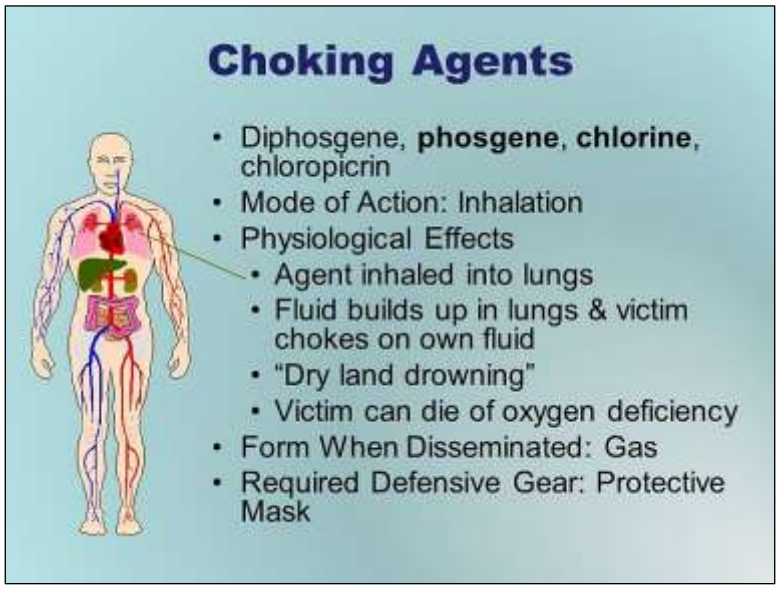

Fig. 4. *Effects of choking agents.*

In fact, exposure to as little as 15 parts per million (PPM) of chlorine gas can trigger respiratory irritation, coughing, and chest constriction. In addition to thoracic and respiratory distress, the immediate effects of choking agent exposure also include burning of the eyes, nose, and throat.

If a choking agent is inhaled, it usually causes:

- Burning in the throat
- Coughing
- Vomiting
- Headache
- Chest pain

BLOOD AGENTS

A blood agent is a toxic chemical agent that affects the body by being absorbed into the blood. Blood agents are fast-acting, potentially lethal poisons that typically manifest at room temperature as volatile colourless gases with a faint odour. They are either cyanide- or arsenic-based. Blood agents work through inhalation or ingestion. As chemic al weapons, blood agents are typically disseminated as aerosols and take effect through inhalation. Due to their volatility, they are more toxic in confined areas than in open areas.

Cyanide compounds occur in small amounts in the natural environment and in cigarette smoke. They are also used in several industrial processes and as pesticides. Cyanides are released when synthetic fabrics or polyurethane burn, and may thus contribute to fire-related deaths. Arsine gas, formed when arsenic encounters an acid, is used as a pesticide and in the semiconductor industry; most exposures to it occur accidentally in the workplace.

The symptoms of blood agent poisoning depend on concentration and duration.

Cyanide-based blood agents irritate the eyes and the respiratory tract, while arsine is nonirritiating. Hydrogen cyanide has a faint, bitter, almond odour that only about half of all people can smell. Arsine has a very faint garlic odour detectable only at greater than fatal concentrations.

Exposure to small amounts of cyanide has no effect. Higher concentrations cause dizziness, weakness and nausea, which cease with the exposure, but long-time exposure can cause mild symptoms followed by permanent brain damage and muscle paralysis. Moderate exposure causes stronger and longer-lasting symptoms, including headache that can be followed by convulsions and coma. Stronger or longer exposure will also lead to convulsions and coma. Very strong exposure causes severe toxic effects within seconds, and rapid death (Figure 5) [12].

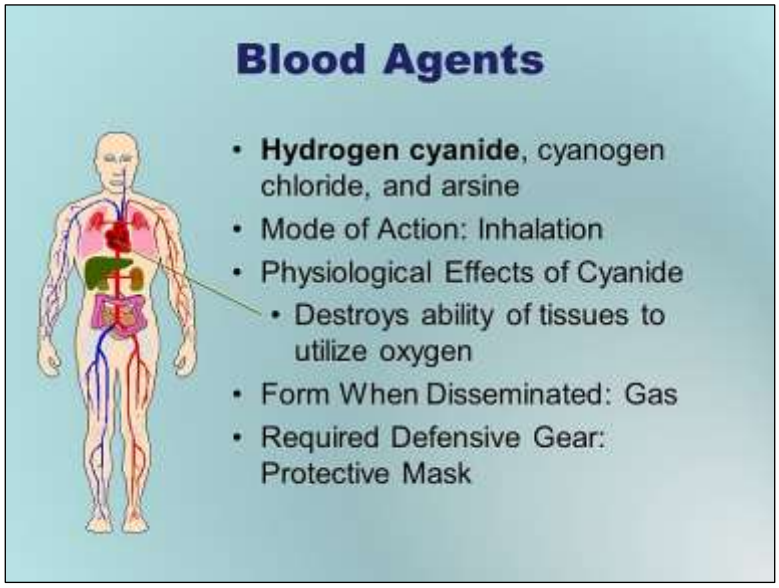

Fig. 5. *Effects of blood agents.*

The blood of people killed by blood agents is bright red, because the agents inhibit the use of the oxygen in it by the body's cells. Cyanide poisoning can be detected by the presence of thiocyanate or cyanide in the blood, a smell of bitter almonds, or respiratory tract inflammations and congestions in the case of cyanogen

chloride poisoning. There is no specific test for arsine poisoning, but it may leave a garlic smell on the victim's breath [13].

At sufficient concentrations, blood agents can quickly saturate the blood and cause death in a matter of minutes or seconds. They cause powerful gasping for breath, violent convulsions and a painful death that can take several minutes. The immediate cause of death is usually respiratory failure.

Blood agents work at the cellular level by preventing the exchange of oxygen and carbon dioxide between the blood and the body's cells. This causes the cells to suffocate from lack of oxygen. Cyanide-based agents do so by interrupting the electron transport chain in the inner membranes of mitochondria. Arsine damages the red blood cells which deliver oxygen throughout the body [14].

CONCLUSION

Treatment Damage from blister agent exposure, lesions and other skin irritations, is symptomatically treated. Hospitalization may be required for respiratory tract injuries. Victims who suffer severe lung damage may require mechanical ventilation. Exposure to large amounts of mustard agent may weaken the whole immune system, requiring special precautions to avoid opportunistic infections during recovery.

International treaties such as the Chemical Weapons Convention should help control proliferation of chemical weapons and verify disarmament but it would be naive to assume the threat will disappear. Military and emergency services must maintain their ability to manage large scale chemical weapons attacks, and that requires continued education, training, and forethought.

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