

# Chemical Warfare Primer

## ***Chemical warfare***

Chemical warfare may include a list of many possible agents including Nerve Agents, Mustard Agents, Hydrogen Cyanide, Tear Gasses, Arsine, and Psycotomimetic agents. However, the most common chemical warfare agents are the Nerve Agents and Mustard Agents.

## ***Nerve agents***

Nerve agents acquired their name because they affect the transmission of nerve impulses in the nervous system. They belong to the organophosphate chemical classification. Organophosphates may be absorbed through the skin and/or via respiration. In 1936 a German chemist by the name of Gerhard Schrader created a pesticide called tabun, this later became known as the first nerve agent. From 1942-1945, Schrader and colleagues synthesized 2000 new organophosphate compounds including sarin (1938) and soman (1944). These three agents (tabun, sarin, and soman) are known as G agents. Further studies and research of these agents led to the development of the V agents in the 1950's. V-agents are approximately 10 times more toxic than sarin and among the most toxic agents ever synthesized. Death may occur within 1 to 10 minutes of inhalation exposure to a minute amount of sarin. Immediate decontamination of the smallest drop is essential. As little as 1 drop of VX on the skin, and 1-10 mL of GA, GB, or GD can be fatal.

Toxic effects are produced by nerve agents by binding to and inactivating acetylcholinesterase. This binding leads to the accumulation of acetylcholine at synapses and causes interruption of transmission at the nicotinic and muscarinic receptors, thus resulting in symptoms consistent with a "cholinergic crisis". Symptoms of a cholinergic crisis or the otherwise commonly known "SLUDGE" Toxidrome include Salivation, Lacri-mation, Urination, Diaphoresis, GI distress (cramp-ing, vomiting and diarrhea) and Emesis. In addition, there is bronchorrhea and bronchoconstriction. High dose exposure may also cause convulsions, loss of consciousness, and muscle paralysis (resulting in respiratory arrest). This bond is reversible with pharmacological intervention. After a certain time period though, these nerve agents undergo a process called "aging", in which they become irreversibly bound (despite pharmacological intervention) to acetylcholinesterase.

Treatment usually consists of decontamination (removal of clothing, bathing, fresh air) and treatment with atropine and oximes. Atropine is used to dry up secretions. It treats the symptoms but does not treat the cause. Oximes are used to break the bond of the nerve agents to the acetyl-cholinesterase enzyme. The only oxime available in the U.S. at this time is Pralidoxime (2-PAM).

## ***Mustard agents***

Mustard agents, also known as "blistering agents" or "mustard gas," produce wounds resembling burns or blisters when they come into contact with the skin. These agents may also cause severe damage to other organs such as the eyes, the respiratory system, and internal organs. They got the name "mustard agents" from an early production method that yielded a mustard-smelling agent.

Mustard gas was produced in 1822 and was first used as a chemical warfare agent in WWI. Iraq also used large amounts of mustard gas in the war against Iran from 1979-1988.

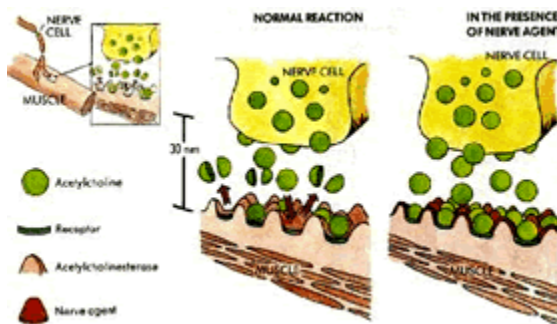
Symptoms are usually delayed between two and 24 hours resulting in severe cell damage before the patient may even know they have been exposed. Mild toxicity will result in symptoms such as eye pain, lacrimation, irritation of the mucous membranes, inflammation of the skin, hoarseness, coughing and sneezing. Severe toxicity may result in blistering, blindness, nausea, vomiting and respiratory complications. The leading cause of death after mustard agent exposure is lung injury. Lung injuries start with mild symptoms and gradually increase and ultimately result in chemical pneumonia and pulmonary edema. A drastic reduction in the number of white blood cells is seen approximately 5-10 days after a large exposure and it's effects on the bone marrow and lymphatic tissue look similar to radiation exposure. This leaves the patient at significant risk of infection.

Decontamination is the most important treatment that can be done for a mustard exposed patient. Removal of clothing, bathing, flushing of the eyes, and washing of the hair are key initial management steps. Some people go as far as to say you should shave hair completely off if it has been exposed. Treatment beyond this is primarily supportive and includes antibiotics and pain medication.

<b>Chemical Warfare Agent</b>	<b>American Denomination</b>	<b>Odor</b>	<b>Additional Comments</b>
Tabun	GA	Fruity	Easiest to make
Sarin	GB	Odorless	Used in a Japanese subway in 1994, volatile mainly taken up by inhalation
Soman	GD	Fruity, oil of camphor	Moderately volatile, taken up by inhalation or skin contact
Cyclohexyl methylphosphonofluoridate	GF		Low volatility, taken up by skin contact and inhalation of substance as gas or aerosol
O-ethyl S-diisopropylaminomethyl methylphosphonothiolate	VX	Odorless	Persistent substance which can remain on material, equipment and terrain for long periods of time. Uptake mainly through skin, but may occur through inhalation of gas or aerosol

## Pearls

- The four nerve agents, GA, GB, GD, and VX are liquid at room temperature; therefore “nerve gas” is a misnomer.
- The G agents presents a definite volatile hazard, VX is less of a volatile hazard unless the ambient temperature is high.
- There are four modes of chemical release:
  1. Explosive – release devices (probably method of choice;
  2. Bulk-release munitions spill agent into the airstreams of the projectile;
  3. Base-ejection devices are relatively uncommon owing to their cost and complexity. Like explosives and bulk-release devices, these munitions can be carried on longer-range missiles.
  4. Spray delivery is generally limited to application on undefended territory or against a poorly defended foe. It is more efficient than the other three methods
- Blistering agents include Sulfur mustard (H/HD), Nitrogen Mustard (HN), arsenical (Lewisite (L)), and phosgene oximes (CX).



In the presence of nerve agent the enzyme acetylcholinesterase, which is responsible for breaking down acetylcholine, is inhibited. The receptors keep on sending signals to the muscle cell, which leads to muscle cramp.

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