World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 6.129

CHEMICAL WARFARE & NERVE AGENTS: MODERN WEAPON OF MASS DESTRUCTION

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Article Received on 21/12/2021 **Article Revised on 11/01/2022** **Article Accepted on 01/02/2022**

ABSTRACT

Nerve agents, also known as nerve gases are organophosphorus compounds used in chemical warfare and terrorism and are the most potent toxic synthetic compounds known. At ambient temperatures, nerve agents are liquids that readily penetrate the skin and enter the bloodstream; thus, dermal contact is an important route of exposure. Another important route of exposure is inhalation, since nerve agent vapors and aerosol droplets are easily inhaled and highly toxic. Cholinesterase inhibition is the primary mechanism of toxicity and clinical management includes drug therapy that restores normal cholinergic signaling as well as supportive care. Nerve agents are acutely toxic; the clinical effects of exposure may appear within seconds to hours or days depending on the level and route of exposure.

KEYWORDS: Organophosphate, Chemical warfare, Acetylcholinesterase, Tabun, Sarin, Soman, V_x.

INTRODUCTION

Nerve agents are a class of phosphorus-containing organic chemicals (organophosphates) that disrupt the mechanism by which nerves transfer messages to organs. The disruption is caused by blocking acetylcholinesterase, an enzyme that normally destroys acetylcholine, a neurotransmitter. As chemical weapons, they are classified as weapons of mass destruction by the United Nations according to UN Resolution 687 (passed in April 1991) and their production and stockpiling was outlawed by the Chemical Weapons Convention of 1993; the Chemical Weapons Convention officially took effect on April 29, 1997. The use of dangerous gases in warfare is forbidden by treaties already in the Hague Conventions of 1899 and 1907 and Geneva Protocol of 1925. Poisoning by a nerve agent leads to contraction of pupils, profuse salivation, convulsions, involuntary urination and defecation, and eventual death by asphyxiation as control is lost over respiratory muscles. Some nerve agents are readily vaporized or aerosolized and the primary portal of entry into the body is the respiratory system. Nerve agents can also be absorbed through the skin, requiring that those likely to be subjected to such agents wear a full body suit in addition to a respirator. Nerve agents are generally colorless to amber-colored, tasteless liquids that may evaporate to a gas Agents Sarin and V_x are odourless; Tabun has a slightly fruity odor and Soman has a slight camphor odor.[1]

HISTORY

The nerve agents are extremely toxic relatives of organophosphate insecticides, which have been used in agriculture since the early 20th century. The first three nerve agents: tabun, sarin, and soman, were invented in Nazi Germany between 1938 and 1944. Tabun and sarin were weaponized by the German military. Germany never used her tabun and sarin weapons in World War II, for reasons still debated today. During the Cold War, the United States and Soviet Union maintained large stockpiles of nerve agents but refrained from their use. Nerve agents were not used on the battlefield until 1984, when Iraq turned to the chemical arm to achieve victory against.

Iran. Iraq had invaded Iran in 1981, but Iraq's invasion bogged down against the numerically superior Iranian defenders. From 1984 to 1987, Iraq used nerve agents' sarin and tabun, against Iranian soldiers; Iraq later used these against Iranian cities. There were anywhere from 45,000 to 100,000 Iranian chemical casualties in the war, of whom the majority appear to have been caused by nerve agents.

Figure-1: War gas shell bombing.

The United States has fought two wars with Iraq, in 1991 and beginning in 2003. In neither war were chemical weapons used, but Iraq's incontestable record of their use played a role in justifying these wars. In 1995, Iraq admitted to the United Nations its possession of nerve agents, as well as other chemical and biological weapons. A full accounting of the quantities admitted by Iraq has not been made.

In May 2004, two U.S. soldiers serving in Iraq were exposed to liquid sarin contained in a round that formed part of an improvised explosive device (IED). The round was most likely an old one from the Saddam Hussein era, and whoever placed it in the IED may not have known that it contained sarin. These two men are the only battlefield nerve agent casualties in U.S. history. The United States signed the Chemical Weapons Convention in 1995, and it came into force in 1997. Under its terms, all nations possessing certain chemical weapons, including nerve agents, agreed to destroy their stockpiles. Nerve agents, unlike anthrax and other biological agents, cannot simply be burned in the open but must be chemically destroyed in specially designed facilities. The convention gives the United States 10 years to destroy all of its chemical munitions. The United States and Russia have successfully petitioned the Organization for the Prevention of Chemical Weapons, which oversees the convention, for permission to extend this deadline.[2]

Iraq is the only country to have used nerve agents in warfare. It is not known how many other countries have clandestine programs involving these agents. Al-Qaeda and other terrorist groups have expressed interest in these agents. U.S. forces captured literature on nerve agents in Al-Qaeda sites in Afghanistan after the 2001 invasion.

Biological effects: Nerve agents attack the nervous system. All such agents function the same way resulting in cholinergic crisis: they inhibit the enzyme acetylcholinesterase, which is responsible for the breakdown of acetylcholine (ACh) in the synapses between nerves that control whether muscle tissues are to relax or contract. If the agent cannot be broken down, muscles are prevented from receiving 'relax' signals and they are effectively paralyzed. It is the compounding of this paralysis throughout the body that quickly leads to more severe complications, including the heart and the muscles used for breathing. Because of this, the first symptoms usually appear within 30 seconds of exposure and death can occur via asphyxiation or cardiac arrest in a few minutes, depending upon the dose received and the agent used.

Initial symptoms following exposure to nerve agents (like Sarin) are a runny nose, tightness in the chest, and constriction of the pupils. Soon after, the victim will have difficulty breathing and will experience nausea and salivation. As the victim continues to lose control of bodily functions, involuntary salivation, lacrimation, urination, defecation, gastrointestinal pain and vomiting will be experienced. Blisters and burning of the eyes and/or lungs may also occur. This phase is followed by initially myoclonic jerks (muscle jerks) followed by status epilepticus -type epileptic seizure. Death then comes via complete respiratory depression, most likely via the excessive peripheral activity at the neuromuscular junction of the diaphragm. The effects of nerve agents are long lasting and increase with continued exposure. Survivors of nerve agent poisoning almost invariably suffer chronic neurological damage and related psychiatric effects. Possible effects that can last at least up to 2–3 years after exposure include blurred vision, tiredness, declined memory, hoarse voice, palpitations, sleeplessness, shoulder stiffness and eye strain. In people exposed to nerve agents, serum and erythrocyte acetylcholinesterase in the long-term are noticeably lower than normal and tend to be lower the worse the persisting symptoms are.

Classes: There are two main classes of nerve agents. The members of the two classes share similar properties and are given both a common name (such as Sarin) and a two-character NATO identifier (such as GB).

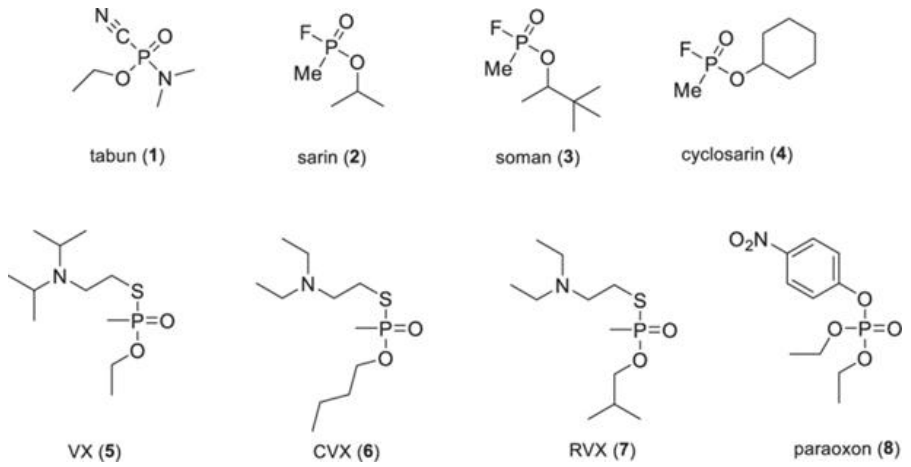

Figure-2: G-series.

Chemical form of the nerve agent tabun, the first ever synthesized

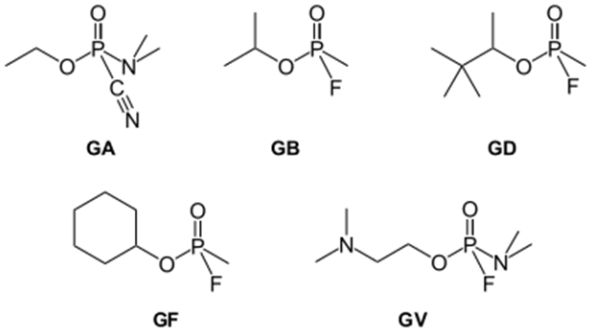

Figure-3: The G series of nerve agents.

The G series of nerve agents: The G-series is thus named because German scientists first synthesized them. G series agents are known as non-persistent, while the V series are persistent [persistency measures time elapsed (upon release) before evaporation]. All of the compounds in this class were discovered and synthesized during or prior to World War II, led by Gerhard Schrader (later under the employment of IG Farben). This series is the first and oldest family of nerve agents. The first nerve agent ever synthesized was GA (Tabun) in 1936. GB (Sarin) was discovered next in 1939, followed by GD (Soman) in 1944, and finally the more obscure GF (Cyclosarin) in 1949. GB was the only G agent that was fielded by the US as a munition, in rockets, aerial bombs, and artillery shells.[3]

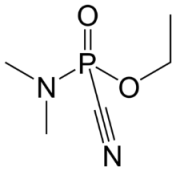

Figure-4: Tabun.

Tabun or GA [CAS: 77-81-6; (RS)-Ethyl N,N-Dimethylphosphoramidocyanidate] is an extremely toxic chemical substance. It is a clear, colorless, and tasteless liquid with a faint fruity odor. It is classified as a nerve agent because it fatally interferes with normal functioning of the mammalian nervous system.Its production is strictly controlled and stockpiling outlawed by the Chemical Weapons Convention of 1993. Tabun is the first of the so-called G-series nerve agents along with GB (sarin), GD (soman) and GF (cyclosarin). Although pure tabun is clear, less-pure tabun may be brown. It is a volatile chemical, although less so than either sarin or soman. Tabun can be destroyed with bleaching powder, though the poisonous gas cyanogen chloride is produced. The symptoms of exposure include: nervousness/restlessness, miosis (contraction of the pupil), rhinorrhea (runny nose), excessive salivation, dyspnea (difficulty in breathing due to bronchoconstriction/secretions), sweating, bradycardia (slow heartbeat), loss of consciousness, convulsions, flaccid paralysis, loss of bladder and bowel control, apnea (breathing stopped) and lung blisters. Tabun is toxic even in minute doses. The number and severity of symptoms which appear vary according to the amount of the agent absorbed and rate of entry of it into the body. Very small skin dosages sometimes cause local sweating and tremors accompanied with characteristically constricted pupils with few other effects. Tabun is about half as toxic as sarin by inhalation, but in very low concentrations it is more irritating to the eyes than sarin. Also, tabun breaks down slowly, which after repeated exposure can lead to build up in the body. Inhaled lethal dosages kill in one to ten minutes, and liquid absorbed

through the eyes kills almost as fast. However, people who experience mild to moderate exposure to tabun can recover completely, if treated almost as soon as exposure occurs. Treatment for suspected tabun poisoning is often three injections of a nerve agent antidote, such as atropine. Pralidoxime chloride (2-PAM Cl) also works as an antidote; however, it must be administered within a period of from minutes to a few hours following exposure to be effective.

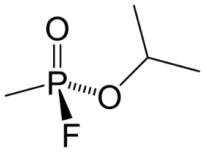

Figure-5: Sarin.

Sarin, or GB [CAS: 107-44-8; Propan-2-yl methylphosphonofluoridate], is an organophosphorus compound with the formula $(CH_3)_2CHO]CH_3P(O)F$. It is a colorless, odorless liquid, used as a chemical weapon owing to its extreme potency as a nerve agent. It has been classified as a weapon of mass destruction in UN Resolution 687. Production and stockpiling of sarin were outlawed by the Chemical Weapons Convention of 1993, and it is classified as a Schedule 1 substance. Sarin can be lethal even at very low concentrations, with death following within 1 to 10 minutes after direct inhalation due to suffocation from lung muscle paralysis, unless some antidotes, typically atropine or biperiden and pralidoxime, are quickly administered to a person. People who absorb a non-lethal dose, but do not receive immediate medical treatment, may suffer permanent neurological damage. Like other nerve agents, sarin attacks the nervous system by interfering with the re-absorption of neurotransmitters at neuromuscular junctions. Death will usually occur as a result of asphyxia due to the inability to control the muscles involved in breathing function. Specifically, sarin is a potent inhibitor of acetylcholinesterase, an enzyme that degrades the neurotransmitter acetylcholine after it is released into the synaptic cleft. In vertebrates, acetylcholine is the neurotransmitter used at the neuromuscular junction, where signals are transmitted between neurons from the central nervous systems to muscle fibres. Normally, acetylcholine is released from the neuron to stimulate the muscle, after which it is degraded by acetylcholinesterase, allowing the muscle to relax. A build-up of acetylcholine in the synaptic cleft, due to the inhibition of cholinesterase, means the neurotransmitter continues to act on the muscle fibre, so that any nerve impulses are effectively continually transmitted. Sarin acts on cholinesterase by forming a covalent bond with the particular serine residue at the active site. Its mechanism of action resembles that of some commonly used insecticides, such as malathion. In terms of biological activity, it resembles carbamate insecticides, such as Sevin, and the medicines pyridostigmine, neostigmine, and physostigmine.[4]

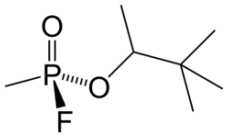

Figure-6: Soman.

Soman, or GD [CAS: 96-64-0; 3,3-Dimethylbutan-2-yl methylphosphonofluoridate], is an extremely toxic chemical substance. It is a nerve agent, interfering with normal functioning of the mammalian nervous system by inhibiting the cholinesterase enzyme. It is an inhibitor of both acetylcholinesterase and butyrylcholinesterase. As a chemical weapon, it is classified as a weapon of mass destruction by the United Nations according to UN Resolution 687. Its production is strictly controlled, and stockpiling is outlawed by the Chemical Weapons Convention of 1993 where it is classified as a Schedule 1 substance. Soman was the third of the so-called G-series nerve agents to be discovered along with GA (tabun), GB (sarin), and GF (cyclosarin). It is a volatile, corrosive, and colorless liquid with a faint odor when pure. More commonly, it is a yellow to brown color and has a strong odor described as similar to camphor. It can also be deployed as a binary chemical weapon; its precursor chemicals are methylphosphonyl difluoride and a mixture of Pinacolyl alcohol and an amine. Soman is an organophosphorus nerve agent with a mechanism of action similar to Tabun. Nerve agents inhibit acetylcholine esterase (AChE) by forming an adduct with the enzyme via a serine residue on that enzyme. These adducts may be decomposed hydrolytically or, for example, by the action of some oximes and thereby regenerate the enzyme. A second reaction type, one in which the enzyme–organophosphate (OP) complex undergoes a subsequent reaction, is usually described as ‘‘aging’’. Once the enzyme–OP complex has aged it is no longer regenerated by the common, oxime reactivators. The rate of this process is dependent on the OP. Soman is an OP that stimulates the rate of aging most rapidly decreasing the half-life to just a few minutes.

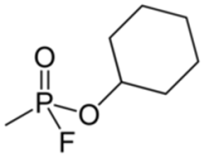

Figure-7: Cyclosarin.

Cyclosarin or GF (CAS: 329-99-7; cyclohexyl methylphosphonofluoridate) is an extremely toxic substance used as a chemical weapon. It is a member of the G-series family of nerve agents, a group of chemical weapons discovered and synthesized by a German team led by Dr. Gerhard Schrader. Cyclosarin is rapidly absorbed through skin, eyes and mucosal membranes. It will penetrate clothing and may be inhaled when in vapour form. It may also be mixed with food or water for absorption via ingestion.

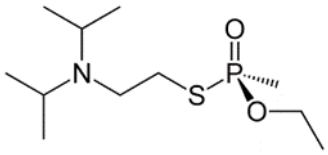

Figure-8: V-series.

The V series of nerve agents: The V-series is the second family of nerve agents and contains five well known members: VE, VG, VM, VR, and VX, along with several more obscure analogues. The most studied agent in this family, VX, was invented in the 1950s at Porton Down in the United Kingdom. Ranajit Ghosh, a chemist at the Plant Protection Laboratories of Imperial Chemical Industries (ICI) was investigating a class of organophosphate compounds (organophosphate esters of substituted aminoethanethiols). Like Schrader, Ghosh found that they were quite effective pesticides. In 1954, ICI put one of them on the market under the trade name Amiton. It was subsequently withdrawn, as it was too toxic for safe use. The toxicity did not go unnoticed and some of the more toxic materials had been sent to the British Armed Forces research facility at Porton Down for evaluation. After the evaluation was complete, several members of this class of compounds became a new group of nerve agents, the V agents (depending on the source, the V stands for Victory, Venomous, or Viscous). The best known of these is probably VX, with VR ("Russian V-gas") coming a close second (Amiton is largely forgotten as VG). All of the V-agents are persistent agents, meaning that these agents do not degrade or wash away easily and can therefore remain on clothes and other surfaces for long periods. In use, this allows the V-agents to be used to blanket terrain to guide or curtail the movement of enemy ground forces. The consistency of these agents is similar to oil; as a result, the contact hazard for V-agents is primarily – but not exclusively dermal. VX was the only V-series agent that was fielded by the US as a munition, in rockets, artillery shells, airplane spray tanks, and landmines.[5]

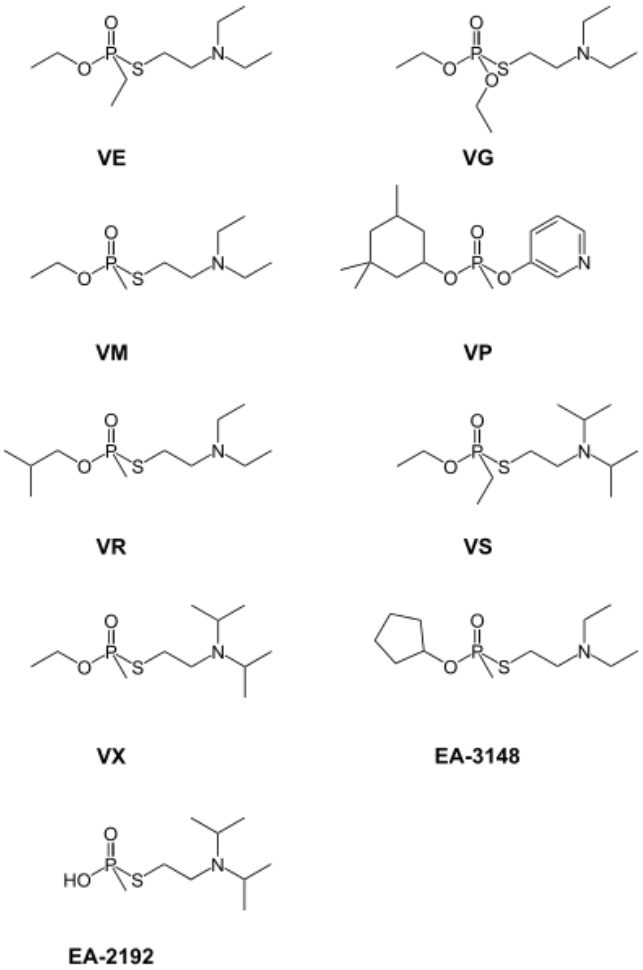

Figure-9: Chemical form of the nerve agent VX.

Methods of spreading

Many methods exist for spreading nerve agents such as:

- uncontrolled aerosol munitions
- smoke generation
- explosive dissemination
- atomizers, humidifiers and foggers

The method chosen will depend on the physical properties of the nerve agent(s) used, the nature of the target, and the achievable level of sophistication.

Detection

Detection of gaseous nerve agents:

The methods of detecting gaseous nerve agents include but are not limited to the following.

- Laser photoacoustic spectroscopy: Laser photoacoustic spectroscopy (LPAS) is a method that has been used to detect nerve agents in the air.
- Non-dispersive infrared: Non-dispersive infrared techniques have been reported to be used for gaseous Nerve agent detection.
- IR absorption: Traditional IR absorption has been reported to detect gaseous nerve agents.
- Fourier transform infrared spectroscopy: Fourier transform infrared (FTIR) spectroscopy has been reported to detect gaseous nerve agents.[6]

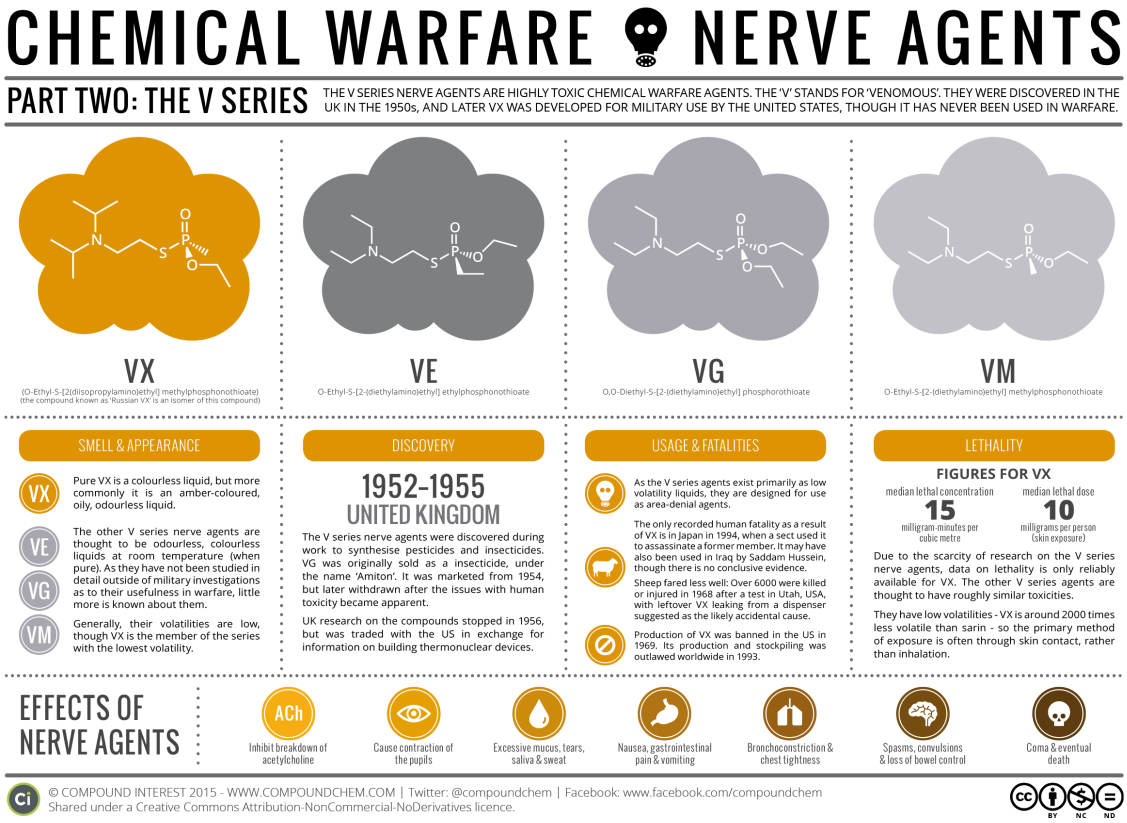

Figure-10: Nerve gas effects.

Treatment: Often the most important first step in treating nerve agent exposure is to decontaminate the patient. This is normally done by removing contaminated clothing, thoroughly washing body and hair with soap and water, and flushing eyes with large amounts of water or saline solution. Contaminated clothing should be double-bagged after removal to prevent further exposure. It is important that anyone treating a contaminated person should wear appropriate personal protective equipment to avoid exposure. Nerve agent poisoning can be treated with the antidotes atropine and pralidoxime chloride (2-PAM chloride).

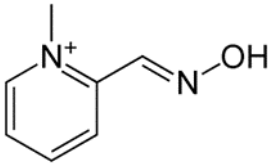

Figure-11: Pralidoxime and auto-injector.

Atropine has anticholinergic properties that are particularly effective at peripheral muscarinic sites, but are less effective at nicotinic sites. 2-PAM chloride cleaves the nerve agent from the cholinesterase enzyme and restores the enzyme's activity. In contrast to atropine's action, the effects of 2-PAM chloride are most noticeable at tissues with nicotinic receptors; muscarinic effects are not observably altered. The efficacy of 2-PAM chloride for treating patients decreases as time elapses due to the strengthening or "aging" of the nerve agent-enzyme bond. This so-called "aging" occurs most rapidly with soman, and 2-PAM chloride may be ineffective for exposures to soman unless administered within several minutes of exposure. Repeated administration of both atropine and 2-PAM chloride may be needed to reverse the effects of nerve agents on patients. Benzodiazepine administration may also be necessary to control seizures, and phentolamine may be needed to treat 2-PAM chloride-induced hypertension. Both atropine and 2-PAM chloride are available to medical professionals as spring-loaded auto-injector syringes for intramuscular administration. Mark, I kit consist of one syringe containing 2 mg atropine and another containing 600 mg 2-PAM chloride.[7]

Nerve Agent Antidote Recommendations

Patient	Mild/Moderate Effects[1]	Severe Effects[2]	Other Treatment
Child	Atropine: 0.05 mg/kg IM, IV (minimum 0.1 mg, maximum 5 mg); **and** 2-PAM chloride: 25 mg/kg IM, IV (maximum 2 g IM, 1g IV)	Atropine: 0.1 mg/kg IM, IV (minimum 0.1 mg, maximum 5 mg); **and** 2-PAM chloride: 50 mg/kg IM, IV (maximum 2 g IM, 1g IV)	Assisted ventilation after antidotes for severe exposure. **Repeat atropine** at 2-5 minute intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near normal. **Repeat 2-PAM chloride** once at 30-60 minutes, then at one-hour intervals for 1-2 doses, as necessary. **Diazepam** for seizures: Child — 0.05 to 0.3 mg/kg IV (maximum 10 mg); Adult — 5 mg IV **Other benzodiazepines (e.g. lorazepam, midazolam)** may provide relief. **Phentolamine** for 2-PAM chloride-induced hypertension: 1 mg IV for children; 5 mg IV for adults.
Adult	Atropine: 2 to 4 mg IM, IV; **and** 2-PAM chloride[3]: 600 mg IM, **or** 25 mg/kg IV slowly	Atropine: 6 mg IM; **and** 2-PAM chloride[3]: 1,800 mg IM, **or** 50 mg/kg IV slowly	

Figure-12: Antidote for nerve agents.

The doses provided by the Mark I kits are designed for adults and these kits have not been approved by the U.S. Food and Drug Administration for use of children. Atropine alone in auto-injector form is available as the AtroPen in amounts of 0.5, 1.0 and 2.0 mg. 2-PAM chloride alone is available in auto-injector form as the 600 mg ComboPen. Although the spring-loaded design of the auto-injectors can cause tissue damage in children and smaller patients, these devices can be useful when intravenous administration of antidotes would be too time consuming or not practical.

- Mild/Moderate effects of nerve agents include localized sweating, muscle fasciculations, nausea, vomiting, weakness, dyspnea.
- Severe effects of nerve agents include unconsciousness, seizures, apnea, flaccid paralysis.
- Dose selection of 2-PAM chloride for elderly patients should be cautious (usually starting at 600 mg IM, or 25 mg/kg IV slowly) to account for the generally decreased organ functions in this population.[8]

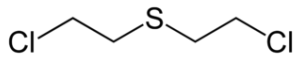

Figure-13: Sulfur mustard.

Mustard gas or sulfur mustard [CAS: 505-60-2; 1-Chloro-2-[(2-chloroethyl)sulfanyl]ethane] is a chemical

compound belonging to a family of cytotoxic and blister agents known as mustard agents. The name mustard gas is widely used, but it is technically incorrect: the substance, when dispersed, is often not actually in a vapor, but is instead in the form of a fine mist of liquid droplets. Mustard gas has a long history of being used as a blister agent in warfare and is one of the most well-studied of such agents. It can form large blisters on exposed skin and in the lungs, often resulting in prolonged illness ending in death.

Figure-14: Mustard gas hazards.

Sulfur mustards are viscous liquids at room temperature and have an odor resembling mustard plants, garlic, or horseradish, hence the name. When pure, they are colorless, but when used in impure forms, such as in warfare, they are usually yellow-brown. As a chemical weapon, mustard gas was first used in World War I, and has been used in several armed conflicts since then, including the Iran–Iraq War, resulting in more than 100,000 casualties. Mustard agents are regulated under the 1993 Chemical Weapons Convention. Three classes of chemicals are monitored under this convention, with sulfur and nitrogen mustard grouped in Schedule 1, as substances with no use other than in chemical warfare (though since then, mustard gas has been found to be useful in cancer chemotherapy). Mustard agents could be deployed by means of artillery shells, aerial bombs, rockets, or by spraying from warplanes or other aircraft.[9]

In World War I, hydrogen cyanide [HCN; CAS: 74-90-8; Formonitrile] was used by the French from 1916 as a chemical weapon against the Central Powers, and by the United States and Italy in 1918. It was not found to be effective enough due to weather conditions. The gas is lighter than air and rapidly disperses up into the atmosphere. Rapid dilution made its use in the field impractical. In contrast, denser agents such as phosgene or chlorine tended to remain at ground level and sank into the trenches of the Western Front's battlefields. Compared to such agent's hydrogen cyanide had to be present in higher concentrations in order to be fatal. A hydrogen cyanide concentration of 100–200 ppm in breathing air will kill a human within 10 to 60 minutes. A hydrogen cyanide concentration of 2000 ppm (about 2380 mg/m^3) will kill a human in about one minute. The toxic effect is caused by the action of the cyanide ion, which halts cellular respiration. It acts as a non-competitive inhibitor for an enzyme in mitochondria called cytochrome c oxidase. As such, hydrogen cyanide is commonly listed among chemical weapons as a blood agent. The Chemical Weapons Convention lists it under Schedule 3 as a potential weapon which has large-scale industrial uses. Signatory countries must declare manufacturing plants that produce more than 30 metric tons per year, and allow inspection by the Organisation for the Prohibition of Chemical Weapons. Perhaps its most infamous use is Zyklon B (German: Cyclone B, with the B standing for Blausäure – prussic acid; also, to distinguish it from an earlier product later known as Zyklon A), it was used in Nazi German extermination camps during World War II to kill en masse as part of their Final Solution genocide program. Hydrogen cyanide was also used in the camps for delousing clothing in attempts to eradicate diseases carried by lice and other parasites. One of the original Czech producers continued making Zyklon B under the trademark "Uragan D2" until recently. Hydrogen cyanide was also the agent employed in judicial execution in some U.S. states, where it was produced during the execution by the action of sulfuric acid on sodium or potassium cyanide. Under the name prussic acid, HCN has been used as a killing agent in whaling harpoons, although it proved quite dangerous to the crew deploying it, and was quickly abandoned. From the middle of the 18th century, it was used in a number of poisoning murders and suicides. Hydrogen cyanide gas in air is explosive at concentrations above 5.6%. This concentration is far above a toxic level.[10]

A gas chamber is an apparatus for killing humans or animals with gas, consisting of a sealed chamber into which a poisonous or asphyxiant gas is introduced. The most commonly used poisonous agent is hydrogen cyanide; carbon dioxide and carbon monoxide have also been used. Gas chambers were used as a method of execution for condemned prisoners in the United States

beginning in the 1920s and continue to be a legal execution method in 3 states. During the Holocaust, large-scale gas chambers designed for mass killing were used by Nazi Germany as part of their genocide program and also by the Independent State of Croatia at the Jasenovac concentration camp. The use of gas chambers has also been reported in North Korea. When executions by gas chambers are conducted in the United States, the general protocol is as follows. First, the executioner will place a quantity of potassium cyanide (KCN) pellets into a compartment directly below the chair in the chamber. The condemned person is then brought into the chamber and strapped into the chair, and the airtight chamber is sealed. At this point the executioner will pour a quantity of concentrated sulfuric acid (H_2SO_4) down a tube that leads to a small holding tank directly below the compartment containing the cyanide pellets. The curtain is then opened, allowing the witnesses to observe the inside of the chamber. The prison warden then asks the condemned individual if he or she wishes to make a final statement. Following this, the executioner(s) throws a switch/lever to cause the cyanide pellets to drop into the sulfuric acid, initiating a chemical reaction that generates hydrogen cyanide (HCN) gas: $2KCN(s) + H_2SO_4(aq) \rightarrow 2HCN(g) + K_2SO_4(aq)$. The gas is visible to the condemned, and he/she is advised to take several deep breaths to speed unconsciousness in order to prevent unnecessary suffering. Accordingly, execution by gas chamber is especially unpleasant for the witnesses to the execution due to the physical responses exhibited by the condemned during the process of dying. These responses can be violent and can include convulsions and excessive drooling. Following the execution, the chamber is purged of the gas through special scrubbers and must be neutralized with anhydrous ammonia (NH_3) before it can be opened. Guards wearing oxygen masks remove the body from the chamber. Finally, the prison doctor examines the individual in order to officially declare that he or she is dead and release the body to the next of kin. One of the problems with the gas chamber is the inherent danger of dealing with such a toxic gas. Anhydrous ammonia is used to cleanse the chamber after cyanide gas has been used: $HCN + NH_3 \rightarrow NH_4 + + CN^-$. The anhydrous ammonia used to clean the chamber afterwards, and the contaminated acid that must be drained and disposed of, are both very poisonous. Nitrogen gas or oxygen-depleted air has been considered for human execution, as it can induce nitrogen asphyxiation. It has not been used to date.[11]

Figure-15: Gas chamber.

Chemical weapons in World War I were primarily used to demoralize, injure and kill entrenched defenders, against whom the indiscriminate and generally slow-moving or static nature of gas clouds would be most effective. The types of weapons employed ranged from disabling chemicals, such as tear gas and the severe mustard gas, to lethal agents like phosgene and chlorine. This chemical warfare was a major component of the first global war and first total war of the 20th century. The killing capacity of gas was limited, with four percent of combat deaths caused by gas. Gas was unlike most other weapons of the period because it was possible to develop effective countermeasures, such as gas masks. In the later stages of the war, as the use of gas increased, its overall effectiveness diminished. The widespread use of these agents of chemical warfare, and wartime advances in the composition of high explosives, gave rise to an occasionally expressed view of World War I as "the chemists' war

Figure-16: Tear gas.

Tear gas: The earliest military uses of chemicals were tear-inducing irritants rather than fatal or disabling poisons. During the First World War, the French army was the first to employ gas, using 26mm grenades filled with tear gas (ethyl bromoacetate, $BrCH_2COOC_2H_5$) in August 1914. The small quantities of gas delivered, roughly 19cm³ per cartridge, were not even detected by the Germans. The stocks were rapidly consumed and by November a new order was placed by the French military. As bromine was scarce among the Entente allies, the active ingredient was changed to chloroacetone ($ClCH_2COCH_3$). In October 1914, German troops fired fragmentation 335 shells filled with a chemical irritant against British positions at Neuve Chapelle, though the concentration achieved was so small that it was barely noticed. None of the combatants considered the use of tear gas to be a conflict with the Hague Treaty of 1899, which prohibited the launching of projectiles containing asphyxiating or poisonous gas.[12]

CONCLUSION

The very rapid onset of neuromuscular symptoms after an exposure should lead the clinician to consider nerve agent intoxication. The nerve agents (tabun, sarin, soman, and VX) are organophosphate analogues of common pesticides that act as potent inhibitors of the enzyme acetylcholinesterase. They are hazardous via ingestion, inhalation, or cutaneous absorption. When nerve agents bind to AChE, they prevent hydrolysis of acetylcholine (ACh). When ACh accumulates in the synaptic space of neurons, this leads to overstimulation of muscarinic and nicotinic receptors. This overstimulation is often termed ``cholinergic crisis''. Also, it is important to note that the nerve agent–AChE bond undergoes a reaction called ``aging''. Once this process is complete, the enzyme becomes irreversibly inactivated. This aging process dictates the need for prompt therapy to prevent irreversible toxicity.

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