civil defense
Technical Bulletin

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CHEMICAL WARFARE DEFENSE SERIES—3

A DIGEST OF TECHNICAL INFORMATION

CHEMICAL WARFARE AGENTS OF SPECIAL SIGNIFICANCE TO CIVIL DEFENSE

This is the third in a series of technical bulletins on civil defense against chemical warfare agents. Later bulletins in this series will describe protective measures against chemical warfare agents and treatment of casualties.

It is the purpose of this technical bulletin to describe certain physical and chemical properties, together with the physiological action, of those chemical warfare agents that an enemy might use against the United States. Only war gases will be considered and we shall discuss here only those that have a high or low persistent effect.

While phosgene, (CG), is still one of the most effective tactical chemical agents, it is not considered a very serious threat in strategic bombing since it would be effective in vapor or aerosol form at the point of release for a relatively short period of time. This bulletin, therefore, is concerned only with those war gases that would appear to be logistically practical. Of these the so-called anticholinesterase agents, or nerve gases, are by far the most important.

Nerve Gases

Prior to World War II, German scientists were engaged in extensive research to find new and more potent insecticides. In 1937 at Elberfeld, Germany, a highly toxic compound was prepared and called Tabun, and in 1938 other German scientists prepared a compound they called Sarin. These have been designated GA and GB respectively. They were found to be so highly toxic that studies were made to determine their value as war gases.

After World War II began in 1939, Germany started large scale production of these chemicals as war gases. Throughout World War II, German research was intensified on these and similar chemicals; and in 1944, at Heidelberg, a third war gas was prepared called Soman, which was later designated as GD. Upon the collapse of the German military machine, large stocks of these war gases fell into the hands of the allies and samples were sent to the United States for analysis and study. The largest plant manufacturing nerve gases was confiscated by the Russians, dismantled, and together with the key technical personnel shipped to Russia.

While all three of the above compounds differ somewhat in molecular structure, they have the same physiological action on man in that they upset the balance between the sympathetic and the parasympathetic nervous systems, which together are the autonomic nervous system. In the body this balance is maintained normally by the enzyme, cholinesterase, reacting with the acetylcholine which is produced as a result of nerve cell stimulation. These so-called G agents were found to react with the cholinesterase in an irreversible action in tissue fluid permitting accumulation of acetylcholine and continual stimulation of the parasympathetic nervous system.

Rapid use of so-called autonomic blocking agents, which act directly on the effector nerve cell, will nullify the effect of acetylcholine. No apparent chemical reaction seems to occur between these autonomic blocking agents and acetylcholine. Atropine salts are the most commonly used autonomic blocking agents. Because of the indirect action of these war gases on the parasympathetic nervous system without any other apparent systemic effect, they are all called nerve gases. The action of the nerve gases and the blocking effect of atropine are shown in Figure 1.

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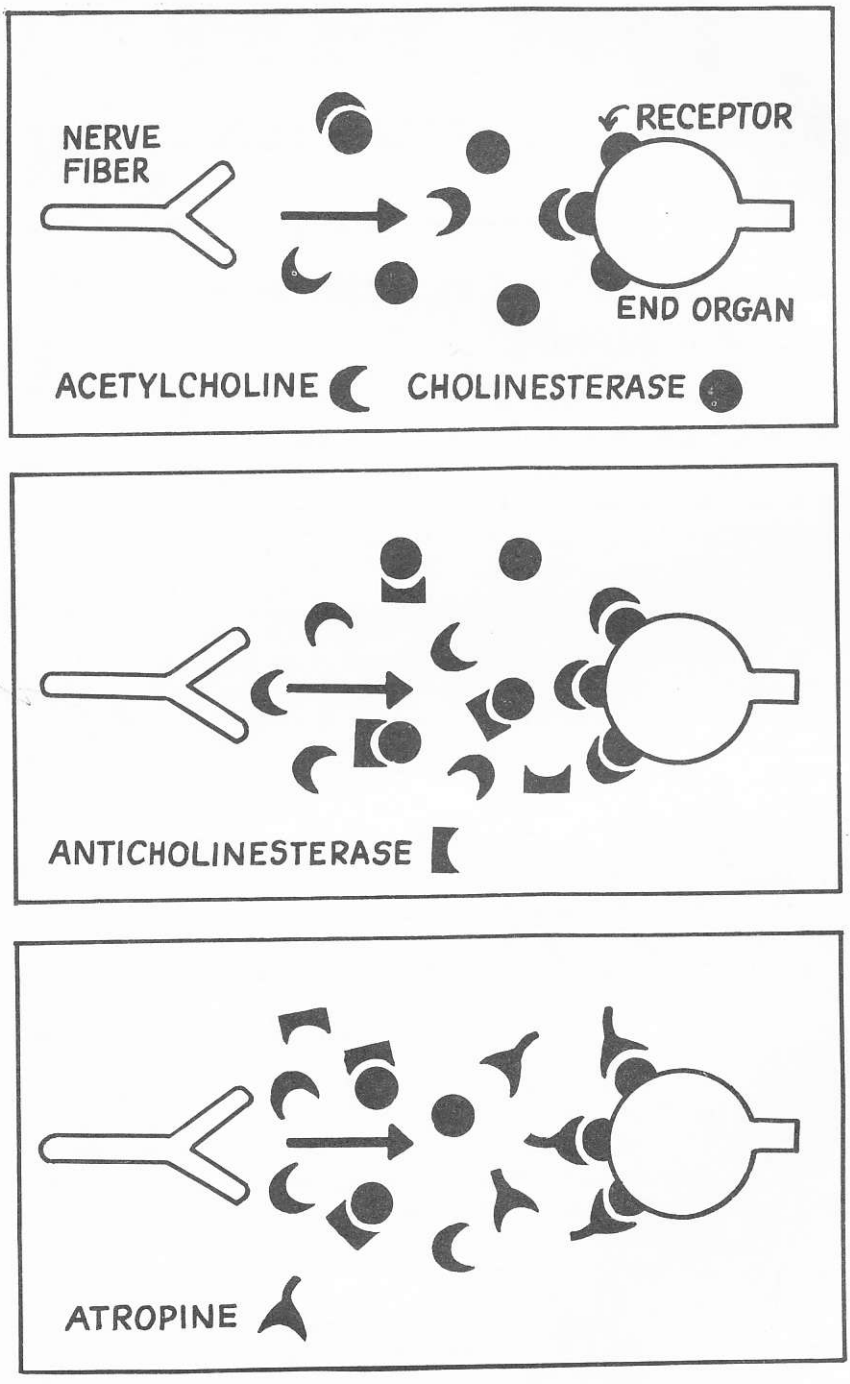


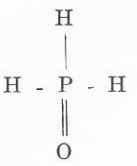
Acetylcholine mediates nerve impulses to end organs. Cholinesterase regulates the amount of Acetylcholine, prevents accumulation by hydrolyzing Acetylcholine to choline and acetic acid.

Organic phosphate (anticholinesterase) inactivates cholinesterase by combining with it. Then acetylcholine accumulates, and produces excessive stimulation of the end organ.

Atropine blocks the action of excess acetylcholine at the end organs.

FIGURE 1 — Mechanism of action of anticholinesterase compounds.

Analysis of these nerve gases disclosed the physical and chemical properties discussed in the following paragraphs. It will be seen that all of these gases may be considered to be derived from phosphine oxide:



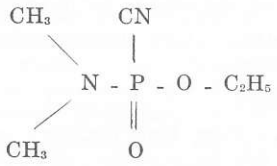
TABUN

General: Tabun is a colorless to brownish liquid.

Chemical name: Cyanodimethylaminoethoxyphosphine oxide.

Formula:

(1) Structural.



(2) Rational: $CN(CH_3)_2NPOC_2H_5O$.

Molecular weight: 162.3

Vapor density (compared with air): 5.63

Liquid density: 1.073 at 25°C. (77°F.).

Freezing point: —49.5°C. (—56°F.) to —50°C. (—58°F.).

Boiling point: Approximately 237.2°C. (459°F.) (estimated). Decomposes below boiling point.

Vapor pressure: 0.070 mm Hg at 25°C. (77°F.).

Volatility: 570 to 610 mg/m³ at 25°C. (77°F.).

Flash point: 77.7°C. (172°F.).

Decomposition temperature: About 230°C. (446°F.).

Latent heat of vaporization: 79.56 calories per gram (average value) between 25° and 50°C. (77° and 122°F.).

Rate of hydrolysis: Reacts slowly with water. Fairly rapid with strong acids or alkalies; self buffering at pH 4 to 5. Half life, 7 hours at pH 4 to 5. Hydrolysis catalyzed by phosphate.

Hydrolysis products: HCN and other products.

Stability in storage: Stable in steel containers at ordinary temperature.

Action on metals or other materials: None.

Odor: Faint, sweetish, fruity.

Median concentration detectable:

(1) By odor and chest tightness. 9 mg/m³.

(2) By eye effects. 3.2 mg/m³.

Rate of detoxification: Slight but definite.

Skin and eye toxicity:

(1) Eye effect: Very high toxicity vapor causes pupil of eye to contract; difficult to see in dim light.

(2) Skin effect: LCT_{50} 0.23 gm/70 kg man. A small drop on skin may cause death of man in short time. Liquid does not injure skin but penetrates it rapidly.

Rate of action: Very rapid: death usually within 15 minutes after fatal dosage absorbed.

Physiological symptoms: Vapors, when inhaled, may cause nausea, vomiting, and diarrhea; these effects may be followed by muscular twitching and convulsions. Even in low concentrations vapor causes eye pupils to contract. Vision becomes difficult, especially in dim light, and headache may result. After short exposure, a sense of tightness in the chest may be noticed. This sense of tightness in the chest is increased by deep breathing. Vapor does not penetrate unbroken skin. Liquid does not injure the skin but penetrates it very rapidly and poisons the body. Penetration of liquid through the eyes and the linings of the mouth and nose is even more rapid than through the skin. Contraction of the pupils may not appear as a warning sign of liquid penetration. The primary physiological action is on the central nervous system, causing a vasoparesis.

Protection required: Leakproof protective mask and impermeable protective clothing.

Decontaminants: Bleach slurry and dilute alkali solutions. In confined area, steam and ammonia. Hot soapy water.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Moderately persistent.

Tactical use: Quick-acting, casualty gas.

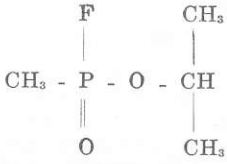
SARIN

General: Sarin is a colorless liquid; its vapor is also colorless.

Chemical name: Fluoroisopropoxymethylphosphine oxide.

Formula:

(1) Structural.



(2) Rational. $F(CH_3)_2CHOPOCH_3$.

Molecular weight: 140.10.

Vapor density (compared with air): 4.86.

Liquid density: 1.100 at 20°C. (68°F.).

Freezing point: —37.5°C. (—36°F.).

Boiling point: 147.2°C. (297°F.).

Vapor pressure: 1.57 mm Hg at 20°C. (68°F.).

Volatility: 12,100 mg/m³ at 20°C. (68°F.).

Flash point: Nonflammable.

Decomposition temperature: Not available.

Latent heat of vaporization: 84.93 calories per gram, average value between 25° and 50°C. (77° and 122°F.).

Rate of hydrolysis: Variable with pH. Half life, 7.5 hours at pH 1.8; 30 hours in unbuffered solution.

Hydrolysis products: HF under acid conditions; isopropyl alcohol and polymers under alkaline conditions.

Stability in storage: Fairly stable in steel at 65°C. (149°F.). Stability improves with increasing purity.

Action on metals or other materials: Slightly corrosive to steel.

Odor: Almost none in pure state.

Rate of detoxification: Low detoxification rate; essentially cumulative.

Skin and eye toxicity:

(1) Eye effect: Very high toxicity. Vapor causes pupil of eye to contract; difficult to see in dim light.

(2) Skin effect: LCt^{50} 0.31 gm/70 kg man. A small drop on skin can cause death of man in short time. Liquid does not injure skin, but penetrates it rapidly.

Rate of action: Very rapid; death usually within 15 minutes after fatal dosage absorbed.

Physiological symptoms: Effects on the body are the same as for Tabun. The primary physiological action, like that of Tabun, is on the sympathetic nervous system, causing a vasoparesis.

Protection required: Leakproof protective mask and impermeable protective clothing.

Decontaminants: Bleach slurry and dilute alkali solutions. In confined area, steam and ammonia. Hot soapy water.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Moderately persistent.

Tactical use: Quick-acting, casualty gas.

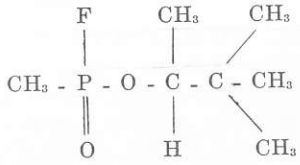
SOMAN

General: Soman is a colorless liquid which gives off a colorless vapor.

Chemical name: Fluoromethylpinacolyloxyphosphine oxide.

Formula:

(1) Structural:



(2) Rational: $FCH_3OPO(CH_3)_3CCHCH_3$.

Molecular weight: 182.178.

Vapor density (compared with air): 6.33.

Liquid density: 1.026 at 20°C. (68°F.).

Freezing point: —70°C. (—94°F.).

Boiling point: 167.2°C. (333°F.).

Vapor pressure: 0.207 mm Hg at 20°C. (68°F.).

Volatility: 2,060 mg/m³ at 20°C. (68°F.).

Flash point: High enough not to interfere with military use.

Decomposition temperature: Not available.

Latent heat of vaporization: 78.49 calories per gram, average value between 25° and 50°C. (77° and 122°F.).

Rate of hydrolysis: Varies with pH; complete within 5 minutes in 5 percent NaOH.

Hydrolysis products: Essentially HF.

Stability in storage: Less stable than Tabun or Sarin.

Action on metals or other materials: Slightly corrosive to metals.

Odor: Fruity; with impurities, odor of camphor.

Median concentration detectable (by odor): Usual field concentration detectable by most people.

Rate of detoxification: Low detoxification rate; essentially cumulative.

Skin and eye toxicity:

(1) Eye effect: Very high toxicity, vapor causes pupil of eye to contract; difficult to see in dim light.

(2) Skin effect: Extremely toxic by skin absorption. Liquid does not injure skin, but penetrates it rapidly.

Rate of action: Very rapid; death usually within 15 minutes after fatal dosage absorbed.

Physiological symptoms: Effects on the body are the same as for Tabun and Sarin but Soman acts faster and in lower concentration; in equal concentrations, the effects from Soman are much more severe than from either Tabun or Sarin. As with Tabun and Sarin, the primary physiological action is on the sympathetic nervous system, causing a vasoparesis.

Protection required: Leakproof protective mask and impermeable protective clothing.

Decontaminants: Bleach slurry and dilute alkali solutions. In confined area, steam and ammonia. Hot soapy water.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Low persistency.

Tactical use: Quick-acting, casualty gas.

Tabun, Sarin, and Soman are the three nerve gases developed by the Germans before and during World War II. They should be considered as examples of a potentially large group of compounds that have in common unbalancing of the central nervous system. It would be unrealistic to think that a potential enemy with a high degree of technical knowledge could not develop in the last decade more toxic compounds of the anticholinesterase type or compounds that attack other vital systems.

Blister Gases

The blister gases are used for casualty effect (or to threaten casualties) so that the use of ground may be restricted, movements slowed, supply impeded, and use for materiel or installations hampered. These war gases affect the eyes and lungs and blister the skin. During World War I, mustard gas was the only blister gas used. It was recognized by a distinctive odor and was highly persistent. Since then, blister gases have been developed that are odorless and that vary in persistency. Blister gases are insidious in their action. Most of them cause no pain at time of exposure to them, and the development of casualties is delayed. Blister gases are extremely difficult to protect against

because they attack any part of the body with which the liquid or vapor comes in contact.

Mustard Gases

The introduction of mustard gas by the Germans against the British near Ypres on July 12, 1917, marked the beginning of a new phase of gas warfare. The gas was dispersed by artillery shell and caught the Allies completely by surprise. Up until the introduction of mustard gas, development of new war gas defensive measures was not far behind development of offensive measures.

Adequate protection was not afforded against mustard gas, however, throughout the remainder of the war. The gas shell casualties admitted to British clearing stations from July 21, 1917, to November 23, 1918, totaled 160,970; of these, 77 percent were mustard gas casualties. About 12,000 tons of mustard gas were used, and 400,000 casualties resulted. This amounted to one casualty for every 60 pounds of mustard gas used, as compared with one for every 230 pounds of choking gases used during World War I. Comparing the number of toxic-gas shell casualties with the number of nongas artillery ammunition casualties, one casualty was produced for each 45 toxic-gas shells and one casualty for each 100 nongas (HE) artillery shells. Toxic-gas shells were, therefore, more than twice as effective as nongas (HE) shells in producing battle casualties.

Mustard Gas (HD)

Mustard is prepared by the Levinstein process. It contains about 30 percent sulfur impurities, which give it a pronounced odor. It is usually purified by washing and vacuum distillation and is designated HD.
Chemical name: Bis (2-chloroethyl) sulfide.
Formula: $(ClCH_2CH_2)_2S$.

Molecular weight: 159.08.

Vapor density (compared to air): 5.4.

Liquid density: 1.27 at 20°C. (68°F.).

Solid density: 1.37 at 0°C. (32°F.).

Freezing point: 14.4°C. (58°F.).

Boiling point: 228°C. (442°F.).

Vapor pressure: 0.072 mm Hg at 20°C. (68°F.).

Volatility: 22 mg/m³ at —17.7°C. (0°F.); 47 mg/m³ at 0°C. (32°F.); 958 mg/m³ at 20°C. (68°F.); 3,660 mg/m³ at 40°C. (104°F.). HD cannot be used for its vapor effect on the skin in cool or cold weather.

Flash point: 105°C. (221°F.); low enough to cause occasional ignition if explosive charges in the shell are too great.

Decomposition temperature: 148.8° to 176.6°C. (300° to 350°F.).

Latent heat of vaporization: 94 calories per gram. (This property is not of great importance in a persistent war gas, as the sustained vapor concentration is essentially a matter of the temperature of the surroundings.)

Rate of hydrolysis: Very slow at ordinary temperatures.

Hydrolysis products: Hydrogen chloride and thiodiglycol.

Stability in storage: Stable in lacquered steel or aluminum containers.

Action on metals or other materials: Very little when pure.

Odor: Garliclike.

Median concentration detectable (by odor): 1.2 mg/m³.

Median lethal dosage:

(1) Inhalation: 1,500 mg-min/m³.

(2) Skin absorption (masked personnel): 10,000 mg/min/m³.

Median incapacitating dosage:

(1) Eye injury: 200 mg-min/m³.

(2) Skin absorption (masked personnel): 2,000 mg-min/m³. Wet skin absorbs more mustard gas than dry skin. For this reason, mustard gas exerts a casualty effect at lower concentrations in hot humid weather, since the body is then moist with perspiration. The figure given above for skin absorption applies to temperatures of approximately 21.1° to 26.6° C. (70° to 80° F.), as the body would not normally be perspiring at these temperatures. Above 26.6°C. (80°F.), perspiration causes increased skin absorption. The incapacitating dosage drops rapidly as perspiration increases; at 32.2° C. (90° F.), 1,000 mg-min/m³ could be incapacitating.

Rate of detoxification: Very low. Even very small repeated exposures are cumulative in their effects. This has been shown in the postwar case histories of workers in the Huntsville Arsenal mustard gas filling plant. Exposure to vapors from spilled mustard gas causes minor symptoms, such as "red eye". Repeated exposure to such vapors produces 100 percent disability by irritating the lungs and causing chronic cough and pain in the chest.

Skin and eye toxicity: Eyes are very susceptible to low concentrations; higher concentrations are required to produce incapacitating effects by skin absorption than by eye injury.

Rate of action: Delayed—usually 4 to 6 hours until first symptoms appear. (Latent periods have been observed, however, up to 24 hours and even, in rare cases, up to 12 days.)

Physiological action: Acts first as a cell irritant and finally as a cell poison. The first symptoms of mustard gas poisoning usually appear in from 4 to 6 hours. The higher the concentration, the shorter the interval of time between exposure of the gas and the first symptoms.

The physiological action of HD may be classified as local and general. The local action results in conjunctivitis or inflammation of the eyes; erythema (morbid redness of the skin) which may be followed by blistering or ulceration; and inflammatory reaction of the nose, throat, trachea, bronchi, and lung tissue. Susceptibility to the toxic action of mustard

gas varies with race; white people are more susceptible than colored. Susceptibility also varies with individuals. Injuries produced by HD heal much more slowly and are more liable to infection than burns of similar intensity produced by physical means or by other chemicals. This is due to the action of HD in making the blood vessels incapable of carrying out their functions of repair, and by the fact that necrotic (dead) tissue acts as a good medium for bacterial growth.

Protection required: Protective mask and permeable protective clothing for vapor; impermeable clothing for protection against liquid.

Decontaminants: Bleach or fire.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

(1) Summer: 3 days to one week.

(2) Winter: Up to several weeks.

Tactical use: Delayed-action casualty gas.

Nitrogen Mustard Gases

The nitrogen mustard gases are a group of three related compounds which may be considered as derivatives of ammonia (NH_3) where the hydrogen atoms are replaced by various organic radicals. In each of these war gases, nitrogen is the central atom. These compounds have been discussed recently in scientific literature because of the discovery that they possess medicinal value. The three members of the group are described individually in the following paragraphs.

Nitrogen Mustard Gas (HN-1)

Chemical name: Bis (2-chloroethyl) ethylamine.

Formula: $ClCH_2CH_2)_2NC_2H_5$.

Molecular weight: 170.08.

Vapor density (compared to air): 5.9.

Liquid density: 1.09 at 25°C. (77°F.).

Freezing point: —34.4°C. (—30°F.).

Boiling point: 85°C. (185°F.) at 10 mm Hg. At atmospheric pressure, HN-1 decomposes below boiling point.

Vapor pressure: 0.17 mm Hg. at 25°C. (77°F.).

Volatility: 140 mg/m³ at —10°C. (14°F.); 329 mg/m³ at 0°C. (32°F.); 1,590 mg/m³ at 20°C. (68°F.); 6,290 mg/m³ at 40°C. (104°F.). HN-1 closely parallels HD in the variation of volatility with temperature, and is of little value as a vapor hazard when weather is cold.

Flash point: High enough not to interfere with military use of the gas.

Decomposition temperature: No data available, but high enough for storage stability.

Latent heat of vaporization: 77 calories per gram.

Rate of hydrolysis: Quite slow.

Hydrolysis products: Hydroxyl derivatives and condensation products. (Intermediate products, all of which are toxic, are produced during hydrolysis.)

Stability in storage: Adequate for military use.

Action on metals and other materials: None.

Odor: Faint fishy or musty.

Median concentration detectable (by odor): 13 mg/m³ to 17 mg/m³, depending on purity.

Median lethal dosage:

(1) Inhalation: 1,500 mg-min/m³.

(2) Skin absorption (masked personnel): 20,000 mg-min/m³.

Median incapacitating dosage:

(1) Eye injury: 200 mg-min/m³.

(2) Skin absorption (masked personnel): 9,000 mg-min/m³.

Rate of detoxification: Cannot be detoxified.

Skin and eye toxicity: Eyes are very susceptible to low concentrations; higher concentrations are required to produce incapacitating effects by skin absorption than by eye injury.

Rate of action: Delayed 12 hours or longer.

Physiological action: Irritates the eyes in dosages that do not significantly damage the skin or respiratory tract, insofar as single exposures are concerned. This irritation appears in a shorter time than that from mustard gas. In mild vapor exposures there may be no skin lesions. After severe vapor exposure, or after exposure to liquid nitrogen mustard gas, erythema (morbid reddening of the skin) may appear earlier than in mustard gas contamination. There may be irritation and itching as with mustard gas. Later, blisters may appear in the erythematous areas. The skin lesions are similar to those caused by mustard gas. Effects on the respiratory tract include irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. Fever, labored respiration, and moist rales may develop. Bronchopneumonia may appear after the first 24 hours. Following ingestion or systemic absorption, the nitrogen mustard gases cause injury to the intestinal tract. Severe diarrhea, which may be hemorrhagic, occurs. Lesions are most marked in the small intestine and consist of degenerative changes and necrosis in the mucous membranes. Ingestion of 2 to 6 milligrams causes nausea and vomiting.

Protection required: Protective mask and permeable protective clothing for vapor; impermeable clothing for protection against liquid.

Decontaminants: Bleach or fire.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

Tactical use: Delayed-action casualty gas.

Nitrogen Mustard Gas (HN-2)

General: HN-2 is highly unstable and is no longer seriously considered as a war gas. It is rated as somewhat more toxic than HN-1.

Chemical name: Bis (2-chloroethyl) methylamine.

Formula: $ClCH_2CH_2)_2NCH_3$.

Molecular weight: 156.07.

Vapor density (compared to air): 5.4.

Liquid density: 1.15 at 20°C. (68°F.).

Freezing point: Approximately —25.5°C. (—14°F.).

Boiling point: 75°C. (167°F.) at 10 mm Hg. At atmospheric pressure, HN-2 decomposes below boiling point.

Vapor pressure: 0.130 mm Hg at 10°C. (50°F.); 0.427 mm Hg at 25°C. (77°F.); 1.25 mm Hg at 40°C. (104°F.).

Volatility: 1,150 mg/m³ at 10°C. (50°F.); 2,580 mg/m³ at 25°C. (77°F.); 10,000 mg/m³ at 40°C. (104°F.).

Flash point: High enough not to interfere with military use of the gas.

Decomposition temperature: No data available, but the instability of HN-2 is associated with its tendency to polymerize or condense. The reaction involved could generate enough heat to cause an explosion.

Latent heat of vaporization: 78.8 calories per gram.

Rate of hydrolysis: In winter, hydrolysis is fairly rapid until 50% complete. Alkalies induce hydrolysis.

Hydrolysis products: None (Condensation or polymerization yields complex products.)

Stability in storage: Not stable.

Action on metals or other materials: None.

Odor:

(1) In dilute form: Like soft soap.

(2) In high concentrations: Fruity.

Median concentration detectable (by odor): 33 mg/m³.

Median lethal dosage (by inhalation): 3,000 mg-min/m³.

Median incapacitating dosage:

(1) Eye injury: 100 mg-min/m³.

(2) Skin absorption (masked personnel): Somewhere between the values given for HN-1 and HN-3.

Rate of detoxification: Cannot be detoxified.

Skin and eye toxicity: HN-2 is the most blistering of the nitrogen mustard gases in vapor form but is intermediate as a liquid blistering gas. Toxic eye effects are produced more rapidly than by HD.

Rate of action: Skin effects delayed 12 hours or longer.

Physiological action: Irritates the eyes in dosages that do not significantly damage the skin or respiratory tract, insofar as single exposures are concerned. This irritation appears in a shorter time than that from mustard gas. In mild vapor exposures there may be no skin lesions. After severe vapor exposure, or after exposure to liquid nitrogen mustard gas, erythema (morbid reddening of the skin) may appear earlier than in mustard gas contamination. There may be irritation and itching as with mustard gas. Later, blisters may appear in the erythematous areas. The skin lesions are similar to those caused by mustard gas. Effects on the respiratory tract include irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. Fever, labored respiration, and moist rales may develop. Bronchopneumonia may appear after the first 24 hours. Following ingestion or systemic absorption, the nitrogen mustard gases cause injury to the intestinal tract. Severe diarrhea, which may be hemorrhagic, occurs. Lesions are most marked in the small intestine and consist of degenerative changes and necrosis in the mucous membranes. Ingestion of 2 to 6 milligrams causes nausea and vomiting.

Protection required: Protective mask and permeable protective clothing for vapor; impermeable clothing for protection against liquid.

Decontaminants: Bleach or fire.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

Tactical use: Delayed-action casualty gas.

Standardization status: None.

Nitrogen Mustard Gas (HN-3)

General: HN-3 is the most stable in storage of the three nitrogen mustard gases and would seem to be admirably suited for use in shells. The German Army had planned to use HN-3 in this way in the event of chemical warfare during World War II.

Chemical name: Tris (2-chloroethyl) amine.

Formula: $N(CH_2CH_2Cl)_3$.

Molecular weight: 204.54.

Vapor density (compared to air): 6.9.

Liquid density: 1.24 at 20°C. (68°F.).

Freezing point: 3.8°C. (39°F.).

Boiling point: About 137.7°C. (280°F.) at 15 mm Hg. At atmospheric pressure, HN-3 decomposes below boiling point.

Vapor pressure: 0.0027 mm Hg at 10°C. (50°F.); 0.0109 mm Hg at 25°C. (77°F.); 0.0382 mm Hg at 40°C. (104°F.).

Volatility: 31.5 mg/m³ at 10°C. (50°F.); 120 mg/m³ at 25°C. (77°F.); 400 mg/m³ at 40°C. (104°F.). These values indicate that HN-3 would not be a satisfactory substitute for HD. With no greater vapor toxicity than HD, the volatility of HN-3 is too low to yield an effective vapor concentration even under tropical conditions.

Flash point: High enough not to interfere with military use of the gas.

Decomposition temperature: Not known; relatively high.

Latent heat of vaporization: 72 calories per gram.

Rate of hydrolysis: Very slow.

Hydrolysis products: Not identified; probably some hydrogen chloride.

Stability in storage: Stable enough for use as a bomb filling even under tropical conditions.

Action on metals or other materials: None if HN-3 is dry.

Odor: None when pure.

Median concentration detectable (by nasal irritation): 8.5 mg/m³.

Median lethal dosage:

(1) Inhalation: 1,500 mg-min/m³.

(2) Skin absorption (masked personnel): 10,000 mg-min/m³.

Median incapacitating dosage:

(1) Eye injury: 200 mg-min/m³.

(2) Skin absorption (masked personnel): 2,500 mg-min/m³. This information is based on estimates and indicates that HN-3 closely approaches HD in toxicity and that it is the most toxic of the nitrogen mustard gases.

Rate of detoxification: Cannot be detoxified; cumulative.

Skin and eye toxicity: Eyes are very susceptible to low concentration; higher concentrations required to produce incapacitating effects by skin absorption.

Rate of action: Most symptoms delayed 4 to 6 hours, as after exposure to HD; but in some cases lacrimation, eye irritation, and photophobia develop immediately.

Physiological action: Irritates the eyes in dosages that do not significantly damage the skin or respiratory tract, so far as single exposures are concerned. This irritation appears in a shorter time than that from mustard gas. In mild vapor exposure there may be no skin lesions. After severe vapor exposure, or after exposure to liquid nitrogen mustard gas, erythema (morbid reddening of the skin) may appear earlier than in mustard gas contamination. There may be irritation and itching as with mustard gas. Later, blisters may appear in the erythematous areas. The skin lesions are similar to those caused by mustard gas. Effects on the respiratory tract include irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. Fever, labored respiration, and moist rales may develop. Bronchopneumonia may appear after the first 24 hours. Following ingestion or systemic absorption, the nitrogen mustard gases cause injury to the intestinal tract. Severe diarrhea, which may be hemorrhagic, occurs. Lesions are most marked in the small intestine and consist of degenerative changes and necrosis in the mucous membranes. Ingestion of 2 to 6 milligrams causes nausea and vomiting.

Protection required: Protective mask and permeable protective clothing for vapor; impermeable clothing for protection against liquid.

Decontaminants: Bleach or fire.

Munitions suitable for use: Shell, guided or other missile,, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

Tactical use: Delayed-action casualty gas.

Arsenical War Gases

The arsenical war gases are a group of related compounds in which arsenic is the central atom. In these gases the hydrogen atoms of AsH_3 are replaced by various organic radicals. The arsenical war gases discussed under blister gases include L, HL, and HT

Weaknesses of these gases are that they hydrolyze rapidly and can be neutralized by BAL ointment.

LEWISITE (L)

General: Lewisite was America's principal contribution to the chemical warfare efforts of World War I. It was first prepared in 1917 by Dr. W. L. Lewis in an attempt to create a compound that would combine the blistering action of mustard gas with the systemic poisoning effect of arsenic. The first lot manufactured was ready for shipment in November 1918. When the armistice was signed, the lewisite was destroyed at sea. The Germans claim that they not only knew of lewisite before the American discovery, but had actually manufactured it during 1917 and 1918. Whether or not their claim is true, they did not use lewisite in World War I.

Chemical name: Dichloro(2-chlorovinyl)arsine.

Formula: ClCH: $CHAsCl_2$.

Molecular weight: 207.32.

Vapor density (compared to air): 7.2.

Liquid density: 1.89 at 20°C. (68°F.).

Freezing point: —17.2°C. (1°F.).

Boiling point: 190°C. (374°F.).

Vapor pressure: 0.40 mm Hg at 20°C. (68°F.).

Volatility: 4,702 mg/m³ at 20°C. (68°F.).

Flash point: None.

Decomposition temperature: Above 100°C. (212°F.).

Latent heat of vaporization: 58 calories per gram from 0°C. (32°F.) to 190°C. (374°F.).

Rate of hydrolysis: Rapidly hydrolyzed in liquid or vapor state.

Hydrolysis products: Hydrogen chloride and chlorovinylarsenious oxide. The latter is a nonvolatile vesicant solid not readily washed away by rains.

Stability in storage: Stable in steel or glass containers.

Action on metals or other materials: None if L is dry.

Odor: Usually geraniumlike; very little odor when pure.

Median concentration detectable:

(1) By odor: 14 to 23 mg/m³.

(2) By irritation: 8 mg/m³.

Median lethal dosage:

(1) Inhalation: 1,200 to 1,500 mg-min/m³.

(2) Skin absorption (masked personnel): 100,000 mg-min/m³. While the toxicity of L by inhalation is slightly higher than that of HD, the systemic poisoning effects due to the arsenical nature of lewisite can be prevented completely by BAL ointment applied to the skin and in the eyes. When the humidity is high lewisite hydrolyzes so rapidly that it is difficult to maintain a concentration sufficient for blistering of bare skin. This difficulty is increased by the high vapor pressure and low persistency of lewisite.

Median incapacitating dosage:

(1) Eye injury (from vapor): Below 300 mg-min/m³.

(2) Skin absorption (masked personnel): Over 1,500 mg-min/m³. L irritates the eyes and skin and gives warning of its presence. For this reason it is less likely to cause casualties than HD, which gives no warning.

Rate of detoxification: The body does not detoxify L but detoxification can be brought about by intramuscular injection of BAL in oil.

Skin and eye toxicity: An exposure of 1,500 mg-min/m³ produces severe and probably permanent corneal damage to the eyes if BAL ointment is not used. L is about as blistering to the skin as HD is, even though the lethal exposure for L is much higher.

Rate of action: Rapid.

Physiological action: L has effects very similar to HD. It also acts as a systemic poison, causing pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure. In order of appearance of symptoms it is a blister gas; a toxic lung irritant; and, when absorbed in the tissues, a systemic poison. L produces an immediate though mild stinging sensation. Reddening of the skin starts in 30 minutes, although blistering does not appear until after about 13 hours. Like HD, it is a cell poison. When inhaled in high concentrations it may be fatal in as short a time as 10 minutes.

Protection required: Protective mask and protective clothing.

Decontaminants: Bleach, BAL ointment, or fire.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

(1) Summer:
  (a) Rural and open: 24 hours.
  (b) Urban or woods: 2 or 3 days.

(2) Winter:
  (a) Rural and open: 1 week or more.
  (b) Urban or woods: 2 weeks or more.

Tactical use: Moderately delayed-action casualty gas.

Mustard Gas — Lewisite Mixture (HL)

General: This is not a standard war gas but a variable mixture of two standard war gases that provides a low-freezing mixture for use in cold weather operations or as high altitude spray. Properties are listed for the eutectic mixture (the mixture having the lowest possible freezing point) that is 63 percent L and 37 percent HD by weight. Other mixtures such as 50-50, may be prepared to meet predetermined weather conditions and have advantages over the eutectic mixture because of the increased HD content. Mixtures of Levinstein mustard gas and lewisite are not satisfactory because of poor storage characteristics.

Chemical name: None; see components.

Formula: None; see components.

Molecular weight: 178.5 (on basis of eutectic mixture).

Vapor density (compared to air): 6.0.

Liquid density: Between the densities of the components; approximately 1.66.

Freezing point: —13.8°C. (7°F.).

Boiling point: Indefinite, but below 190°C. (374°F.).

Vapor pressure: 0.02 mm Hg at —11.1°C. (12°F.); 0.248 mm Hg at 20°C. (68°F.); 1.03 mm Hg at 40°C. (104°F.).

Volatility: 240 mg/m³ at —11.1°C. (12°F.); 2,730 mg/m³ at 20°C. (68°F.); 10,270 mg/m³ at 40°C. (104°F.).

Flash point: High enough not to interfere with the military use of the gas.

Decomposition temperature: Above 100°C. (212°F.).

Latent heat of vaporization: Intermediate between the heats of vaporization of the components.

Rate of hydrolysis: L is rapidly hydrolyzed in the liquid or vapor state; HD hydrolyzes slowly at ordinary temperatures.

Hydrolysis products: Hydrogen chloride, thiodiglycol, and chlorovinylarsenious oxide.

Stability in storage: Satisfactory in lacquered steel containers.

Action on metals and other materials: Little or none if dry.

Odor: Garliclike.

Median concentration detectable (by odor): Probably about 2.0 mg/m³.

Median lethal dosage:
(1) Inhalation: About 1,500 mg-min/m³.
(2) Skin absorption: Above 10,000 mg-min/m³.

Median incapacitating dosage:
(1) Eye injury: About 200 mg-min/m³.
(2) Skin absorption: 1,500-2,000 mg-min/m³.

Rate of detoxification: Cannot be detoxified.

Skin and eye toxicity: Very high.

Rate of action: Produces immediate stinging of skin and redness within 30 minutes; blistering delayed about 13 hours.

Physiological action: Liquid causes severe damage to the eyes. Contamination of the skin is followed after a short time by reddening, then blistering that tends to cover the entire area of the reddened skin. The respiratory lesions are similar to those produced by mustard gas, except that in the most severe cases pulmonary edema may be accompanied by pleural effusion. Liquid on the skin, as well as inhaled vapor, is absorbed and may cause systemic poisoning. This change is manifested in capillary permeability that permits loss of sufficient fluid from the blood stream to cause blood thickening, shock, and death.

Protection required: Protective mask and protective clothing.

Decontaminants: Bleach or fire.

Munitions suitable for use: Shell, guided or other missile, from submarine, marine, or land-based launching platform, bombs.

Persistency effect: Highly persistent.

Tactical use: Delayed-action casualty gas.

MUSTARD GAS — T MIXTURE (HT)

General: HT is a mixture of 60 percent HD and 40 percent T. T, a sulfur and chlorine compound similar in structure to HD, is a clear yellowish liquid with an odor similar to HD. HT has a greater blistering effect, is more persistent and more stable, and has a lower freezing point than HD. Its low volatility makes effective vapor concentrations in the field difficult to obtain. Properties are essentially the same as those of HD.

Physiological action: Vesicant, eye irritant, toxic by inhalation. HT does not produce more severe lesions than either H or T alone, but the mixture does tend to confuse and make diagnosis difficult.

Protection required: Protective mask and permeable protective clothing for vapor; impermeable clothing for protection against liquid.

Decontaminants: Bleach or fire.

Persistency effect: Highly persistent.

Tactical use: Delayed-action casualty gas.

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