

## CHAPTER 2

### NERVE AGENTS

#### Section I. INTRODUCTION

#### 2-1. General

a. Nerve agents are a group of highly toxic organic esters of phosphoric acid derivatives. These agents have physiological effects (inhibition of cholinesterase) resembling those of physostigmine and pyridostigmine. However, they are more potent, longer-acting, and tend to be irreversible after a time which varies with the agent.

b. Nerve agents are among the deadliest of chemical agents and may produce rapid symptoms. They include the G- and V-agents. Examples of G-agents are Tabun (GA), Sarin (GB), Soman (GD), and GF. A V-agent is VX. In some countries, "V" agents are known as "A" agents. (Detailed descriptions of nerve agents are found in FM 3-9.)

c. Nerve agents can be dispersed by artillery shell, mortar shell, rocket, land mine, missile, aircraft spray, and aircraft bomb or bomblet.

d. Several related but somewhat less toxic compounds have proven to be useful in medicine and agriculture, as indicated below. The symptoms and treatment of poisoning by these compounds are similar to those of poisoning by nerve agents.

(1) Anticholinesterase agents have been used in the treatment of abdominal distention, urinary retention, and glaucoma.

(2) Many of the insecticides currently in use are organophosphates and are chemically related to nerve agents. Although beneficial for arthropod control, their widespread use has caused many accidental poisonings—some fatal. Organophosphate insecticides may have a slower and longer lasting effect as compared to CW organophosphates.

#### 2-2. Physical and Chemical Properties

Nerve agents are colorless to light brown liquids. Some are volatile, while others are relatively non-volatile at room temperature. Most nerve agents are essentially odorless; however, some have a faint fruity odor. In toxic amounts, aqueous solutions of nerve agents are tasteless. The G-agents tend to be nonpersistent, whereas the V-agents are persistent. However, thickened nonpersistent agents may present a hazard for an extended period of time. These agents are moderately soluble in water with slow hydrolysis; are highly soluble in lipids; and are rapidly inactivated by strong alkalis and chlorinating compounds (strong

alkalies and chlorinating compounds are used for decontaminating equipment; in diluted formulas, chlorinating compounds are used for patient decontamination).

#### 2-3. Absorption of and Protection Against Nerve Agents

a. Nerve agents may be absorbed through any body surface. When dispersed as a spray or aerosol, droplets can be absorbed through the skin, eyes, and respiratory tract. When dispersed as a vapor at expected field concentrations, the vapor is primarily absorbed through the respiratory tract. If enough agent is absorbed, local effects are followed by generalized systemic effects. The rapidity with which effects occur is directly related to the amount of agent absorbed in a given period of time. Liquid nerve agents may be absorbed through the skin, eyes, mouth, and membranes of the nose. Nerve agents may also be absorbed through the gastrointestinal tract when ingested with food or water. Local effects after skin exposure are localized sweating and/or muscular twitching. Local effects after vapor or liquid exposure to the eye include miosis and often conjunctival hyperemia. Local effects of liquid on the mucous membrane include twitching or contracting of the underlying muscle and glandular secretions. Absorption of a nerve agent by any route may result in generalized systemic effects. The respiratory tract (inhalation) is the most rapid and effective route of absorption.

b. The protective mask and hood protect the face and neck, eyes, mouth, and respiratory tract against nerve agent spray, vapor, and aerosol. Nerve agent vapor (in expected field concentrations) is absorbed through the skin very slowly, if at all, so proper masking may protect against the effects of low vapor concentrations. To prevent inhaling an incapacitating or lethal dose, hold your breath and put on your mask within 9 seconds at the first warning of a nerve agent presence.

c. Liquid nerve agents penetrate ordinary clothing rapidly. However, significant absorption through the skin requires a period of minutes. The effects may be reduced by quickly removing contaminated clothing and neutralizing liquid nerve agent on the skin (washed off, blotted, or wiped away). Prompt decontamination

of the skin is imperative. Decontamination of nerve agents on the skin within 1 minute after contamination is perhaps ten times more effective than it would be if delayed 5 minutes. A nerve agent on the skin can be removed effectively by using the M291 Skin Decontaminating Kit or the M258A1 Skin Decontamination Kit (app D). The M291 Skin Decontaminating Kit is replacing the M258A1. Upon receipt of the M291, discontinue using the M258A1 on the skin. Liquid nerve agent in the eye is absorbed faster than on the skin and is extremely dangerous; immediately irrigate the eye with copious amounts of water.

d. The chemical protective overgarment, patient protective wrap (PPW), impermeable protective gloves, and overboots protect the skin against nerve agents in liquid, aerosol, and vapor forms.

## 2-4. Effects of Nerve Agents

a. *Mechanism of Action.* The effects of nerve agents (table 2-1) are due to their ability to inhibit cholinesterase enzymes throughout the body. Since the normal function of these enzymes is to hydrolyze acetylcholine wherever it is released, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. These include the endings of the autonomic nerves to the smooth muscle of the iris, ciliary body, bronchial tree, gastrointestinal tract, bladder, and blood vessels; to the salivary glands and secretory glands of the gastrointestinal tract and respiratory tract; and to the cardiac muscle and endings of sympathetic nerves to the sweat glands (fig 2-1). The accumulation of acetylcholine at these sites results in characteristic muscarinic signs and symptoms (table 2-1). The accumulation of acetylcholine at the endings of motor nerves to voluntary muscles and in some autonomic ganglia results in nicotinic signs and symptoms (table 2-1). Finally, the accumulation of excessive acetylcholine in the brain and spinal cord results in characteristic CNS symptoms (table 2-1). The inhibition of cholinesterase enzymes throughout the body by nerve agents may be irreversible and their effects prolonged; therefore, treatment should begin promptly before irreversibility occurs. Until the tissue cholinesterase enzymes are restored to normal activity, there is a period of increased susceptibility to the effects of another exposure to any nerve agent. This period of increased susceptibility occurs during the enzyme regeneration phase which could last from weeks to several months, depending on the severity of the initial exposure. During this period the effects of repeated exposures are cumulative.

b. *Pathology.* Aside from the decrease in the activity of cholinesterase enzymes throughout the body (which may be analyzed by laboratory methods), no specific lesions are detectable by ordinary gross examination. At postmortem examination there is

usually capillary dilation, hyperemia, and edema of the lungs; there may be similar changes in the brain and the remaining organs. Neuropathologic changes have been reported in animals following severe intoxication.

c. *Effects of Vapor.* The lungs and the eyes absorb nerve agents rapidly. Changes occur in the smooth muscle of the eye, resulting in miosis (contraction of the pupil); also in the smooth muscle and secretory glands of the bronchi, producing bronchial constriction and excessive secretions in the upper and lower airways. In high vapor concentrations, the nerve agent is carried from the lungs throughout the circulatory system; widespread systemic effects may appear in less than 1 minute.

(1) *Local ocular effects.* These effects begin within seconds or minutes after exposure and before there is any evidence of systemic absorption. The earliest ocular effect which follows minimal symptomatic exposure to vapor is miosis. This is an invariable sign of ocular exposure to enough vapor to produce symptoms. It is also the last ocular manifestation to disappear. The pupillary constriction may be different in each eye. Within a few minutes after the onset of exposure, there also occurs redness of the eyes due to conjunctival hyperemia and a sensation of pressure with heaviness in and behind the eyes. Usually vision is not grossly impaired, although there may be a slight dimness especially in the peripheral fields or when in dim or artificial light. Exposure to a level of a nerve agent vapor slightly above the minimal symptomatic dose results in miosis; pain in and behind the eyes attributable to ciliary spasm, especially on focusing; some difficulty of accommodation; and frontal headache. The pain becomes worse when the casualty tries to focus the eyes or looks at a bright light. Some twitching of the eyelids may occur. Occasionally there is nausea and vomiting which, in the absence of systemic absorption, may be due to a reflex initiated by the ocular effects. These local effects may result in moderate discomfort and some loss of efficiency, but may not necessarily produce casualties. Following minimal symptomatic exposure, the miosis lasts from 24 to 72 hours. After exposure to at least the minimal symptomatic dose, miosis is well established within half an hour. Miosis remains marked during the first day after exposure and then diminishes gradually over 2 to 3 days after MODERATE exposure, but may persist for as long as 14 days after severe exposure. The conjunctival erythema, eye pain, and headache may last from 2 to 15 days depending on the dose.

(2) *Local respiratory effects.* Following minimal exposure, the earliest effects on the respiratory tract are watery nasal discharge, nasal hyperemia, sensation of tightness in the chest, and occasionally, prolonged wheezing expiration

Table 2-1. Signs and Symptoms of Nerve Agent Poisoning

SITE OF ACTION	SIGNS AND SYMPTOMS
<p>1. <b>Muscarinic</b> Pupils Ciliary body Nasal mucous membranes Bronchial tree Gastrointestinal</p> <p>Bronchial tree</p> <p>Gastrointestinal</p> <p>Sweat glands Salivary glands Lacrimal glands Heart Pupils Ciliary body Bladder</p> <p>2. <b>Nicotinic</b> Striated muscle Sympathetic ganglia</p> <p>3. <b>Central Nervous System</b></p>	<p><b>Following Local Exposure</b></p> <p>Miosis, marked, usually maximal (pinpoint), sometimes unequal. Frontal headache, eye pain on focusing, blurring of vision. Rhinorrhea, hyperemia. Tightness in chest, bronchoconstriction, increased secretion, cough. Occasional nausea and vomiting.</p> <p><b>Following Systemic Absorption (depending on dose)</b></p> <p>Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, pain in chest, increased bronchial secretion, cough, cyanosis, pulmonary edema. Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation. Increased sweating. Increased salivation. Increased lacrimation. Bradycardia. Miosis, occasionally unequal, later maximal miosis (pinpoint). Blurring of vision, headache. Frequency, involuntary micturition.</p> <p>Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness/flaccid paralysis (including muscles of respiration) with dyspnea and cyanosis. Pallor, transitory elevation of blood pressure followed by hypotension.</p> <p><b>Immediate (Acute) Effects:</b> Generalized weakness, depression of respiratory and circulatory centers with dyspnea, cyanosis, and hypotension, convulsions, loss of consciousness, and coma. <b>Delayed (Chronic) Effects:</b> Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG, especially on hyperventilation, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia.</p>

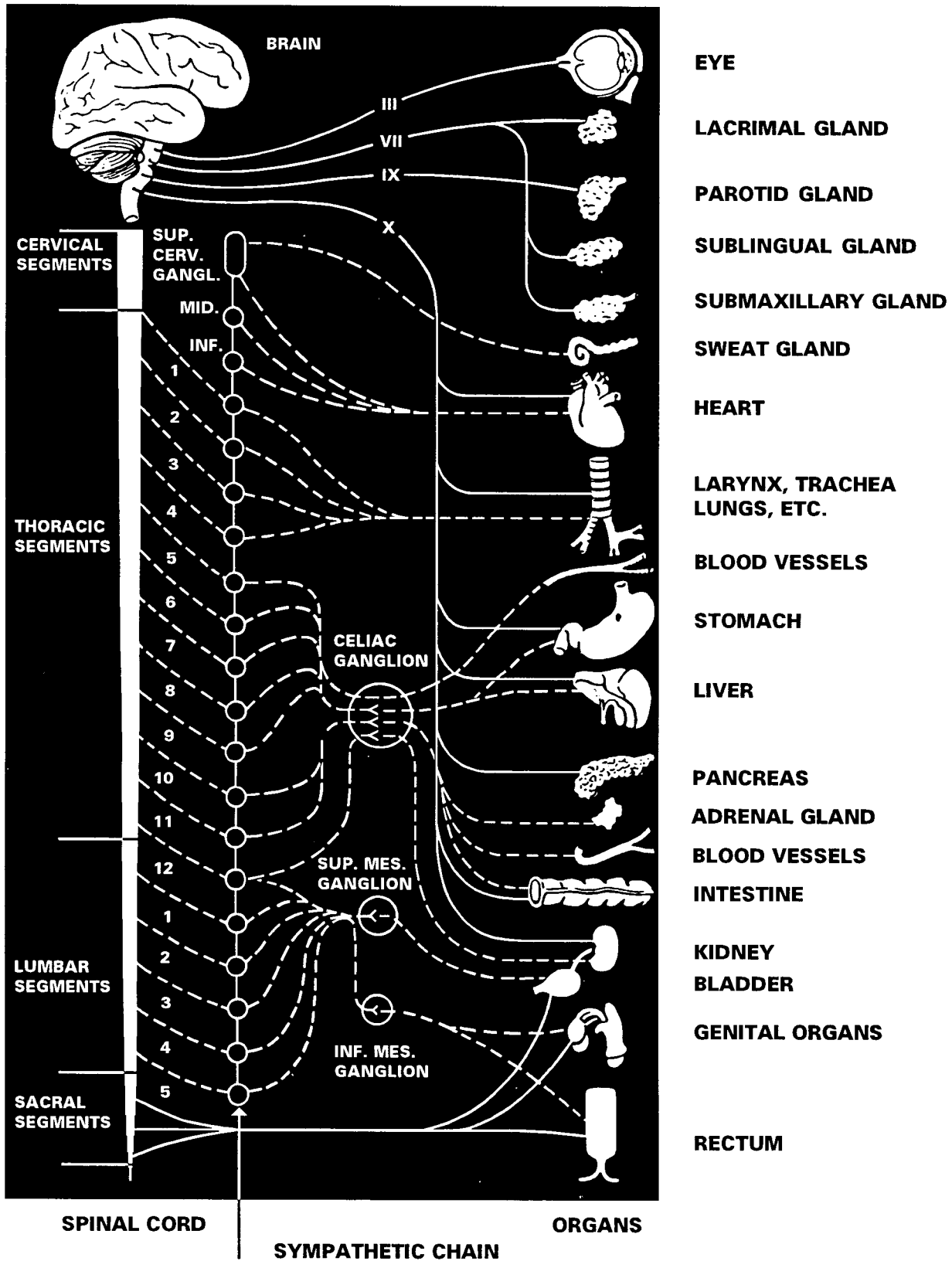


Figure 2-1. Autonomic nervous system.

suggestive of bronchoconstriction or increased bronchial secretion. The rhinorrhea usually lasts for several hours after minimal exposure and for about 1 day after more severe exposure. The respiratory symptoms are usually intermittent for several hours duration after **MILD** exposure; they may last for 1 or 2 days after more severe exposure.

(3) *Systemic effects.* The sequence of symptoms varies with the route of exposure. While respiratory symptoms are generally the first to appear after inhalation of nerve agent vapor, gastrointestinal symptoms are usually the first after ingestion. Following comparable degrees of exposure, respiratory manifestations are most severe after inhalation and gastrointestinal symptoms may be most severe after ingestion. Otherwise, the systemic manifestations are, in general, similar after any exposure to nerve agent poisoning by any route. If local ocular exposure has not occurred, the ocular manifestations (including miosis) initially may be absent. The signs, symptoms, and their time course following exposure to nerve agent are given in table 2-2. The systemic effects may be considered to be nicotinic, muscarinic, or by any action at receptors within the CNS. The predominance of muscarinic, nicotinic, or CNS effects will influence the amount of atropine, oxime, or anticonvulsant which must be given as therapy. These effects will be considered separately.

(a) *Muscarinic effects.* The tightness in the chest is an early local symptom of respiratory exposure. This symptom progressively increases as the nerve agent is absorbed into the systemic circulation, whatever the route of exposure. After **MODERATE** or severe exposure, excessive bronchial and upper airway secretions occur and may become very profuse, causing coughing, airway obstruction, and respiratory distress. Audible wheezing may occur, with prolonged expiration and difficulty in moving air into and out of the lungs, due to the increased bronchial secretion or to bronchoconstriction, or both. Some pain may occur in the lower thorax and salivation increases. Bronchial secretion and salivation may be so profuse that watery secretions run out of the sides of the mouth. The secretions may be thick and tenacious. If postural drainage or suction is not employed, these secretions may add to the airway obstruction. Laryngeal spasm and collapse of the hypopharyngeal musculature may also obstruct the airway. The casualty may gasp for breath, froth at the mouth, and become cyanotic. If the upper airway becomes obstructed by secretions, laryngeal spasm, or hypopharyngeal musculature collapse, or if the bronchial tree becomes obstructed by secretions or bronchoconstriction, little ventilation may occur despite respiratory movements. As hypoxemia and cyanosis increase, the casualty will fall exhausted and become unconscious. Following inhalation of nerve

agent vapor, the respiratory manifestations predominate over the other muscarinic effects; they are likely to be most severe. In older casualties and in those with a history of respiratory disease, particularly bronchial asthma. However, if the exposure is not so overwhelming as to cause death within a few minutes, other muscarinic effects appear. These include sweating, anorexia, nausea, and epigastric and substernal tightness with heartburn and eructation. If absorption of the nerve agent has been great enough (whether due to a single large exposure or to repeated smaller exposures), there may follow abdominal cramps, increased peristalsis, vomiting, diarrhea, tenesmus, increased lacrimation, and urinary frequency. Cardiovascular effects are occasional early bradycardia, transient tachycardia and/or hypertension followed by hypotension, and cardiac arrhythmias. The casualty perspires profusely, may have involuntary defecation and urination, and may go into cardiorespiratory arrest followed by death.

(b) *Nicotinic effects.* With the appearance of **MODERATE** muscarinic systemic effects, the casualty begins to have increased fatigability and **MILD** generalized weakness which is increased by exertion. This is followed by involuntary muscular twitching, scattered muscular fasciculations, and occasional muscle cramps. The skin may be pale due to vasoconstriction and blood pressure moderately elevated (transitory) together with tachycardia, resulting from epinephrine response to excess acetylcholine. If the exposure has been severe, the muscarinic cardiovascular symptoms will dominate and the fascicular twitching (which usually appear first in the eyelids and in the facial and calf muscles) becomes generalized. Many rippling movements are seen under the skin and twitching movements appear in all parts of the body. This is followed by severe generalized muscular weakness, including the muscles of respiration. The respiratory movements become more labored, shallow, and rapid; then they become slow and finally intermittent. Later, respiratory muscle weakness may become profound and contribute to respiratory depression. Central respiratory depression may be a major cause of respiratory failure.

(c) *Central nervous system effects.* In **MILD** exposures, the systemic manifestations of nerve agent poisoning usually include tension, anxiety, jitteriness, restlessness, emotional lability, and giddiness. There may be insomnia or excessive dreaming, occasionally with nightmares. If the exposure is more marked, the following symptoms may be evident: headache, tremor, drowsiness, difficulty in concentration, memory impairment with slow recall of recent events, and slowing of reactions. In some casualties, there is apathy, withdrawal, and depression. With the appearance of **MODERATE** symptoms, abnormalities of the electroencephalogram

occur, characterized by irregularities in rhythm, variations in potential, and intermittent bursts of abnormally slow waves of elevated voltage similar to those seen in patients with epilepsy. These abnormal waves become more marked after 1 or more minutes of hyperventilation which, if prolonged, may occasionally precipitate a generalized convulsion. If absorption of nerve agent has been great enough, the casualty becomes confused and ataxic. The casualty may have changes in speech (consisting of slurring, difficulty in forming words, and multiple repetition of the last syllable). The casualty may then become comatose, reflexes may disappear, and respiration may become Cheyne-Stokes in character. Finally, generalized convulsions may ensue. With the appearance of severe CNS symptoms, central respiratory depression will occur (adding to the respiratory embarrassment that may already be present) and may progress to respiratory arrest. However, after severe exposure, the casualty may lose consciousness and promptly convulse without other obvious symptoms. Death is usually due to respiratory arrest and anoxia. Prompt initiation of assisted ventilation may prevent death. Depression of the circulatory centers may also occur, resulting in a marked reduction in heart rate with a fall of blood pressure some time before death.

*d. Effects of Liquid Nerve Agent.*

(1) *Local ocular effects.* The local ocular effects are similar to the effects of nerve agent vapor. If the concentration of the liquid nerve agent contaminating the eye is high, the effects will be instantaneous and marked; and, if the exposure of the two eyes is unequal, the local manifestations may be unequal. Hyperemia may occur but there is no immediate local inflammatory reaction such as may occur following ocular exposure to more irritating substances (for example, lewisite).

(2) *Local skin effects.* Following cutaneous exposure, there is localized sweating at and near the site of exposure and localized muscular twitching and fasciculation. However, these may not be noticed causing the skin absorption to go undetected until systemic symptoms begin.

(3) *Local gastrointestinal effects.* Following the ingestion of substances containing a nerve agent (which is essentially tasteless), the initial symptoms include abdominal cramps, vomiting, and diarrhea.

(4) *Systemic effects.* The sequence of symptoms varies with the route of exposure. While respiratory symptoms are generally the first to appear after inhalation of a nerve agent vapor, gastrointestinal symptoms are usually the first after ingestion. Following comparable degrees of exposure, respiratory manifestations are most severe after inhalation, and gastrointestinal symptoms may be most severe after ingestion. Otherwise, the systemic manifestations are, in general, similar after any exposure to nerve

agent poisoning by any route. If local ocular exposure has not occurred, the ocular manifestations (including miosis) initially may be absent.

*e. Time Course of Effects of Nerve Agents.* See table 2-2.

*f. Cumulative Effects of Repeated Exposure.* Daily exposure to concentrations of a nerve agent insufficient to produce symptoms following a single exposure may result in the onset of symptoms after several days. Continued daily exposure may be followed by increasingly severe effects. After symptoms subside, increased susceptibility may persist for up to 3 months. The degree of exposure required to produce recurrence of symptoms and the severity of these symptoms depend on the dose received and the time interval since the last exposure. Increased susceptibility is not limited to the particular nerve agent initially absorbed.

*g. Cause of Death.* In the absence of treatment, death is caused by anoxia resulting from airway obstruction, weakness of the muscles of respiration, and central depression of respiration. Airway obstruction is due to pharyngeal muscular collapse; upper airway and bronchial secretions; bronchial constriction and occasionally laryngospasm; and paralysis of the respiratory muscles. Respiration is shallow, labored, and rapid, and the casualty may gasp and struggle for air. Cyanosis increases. Finally, respiration becomes slow and then ceases resulting in unconsciousness. The blood pressure (which may have been transiently elevated) falls. Cardiac rhythm may become irregular and death may ensue. The individual may survive several lethal doses of a nerve agent if assisted ventilation is initiated via cricothyroidotomy or endotracheal tube, if airway secretions are cleared by postural drainage and suction, and if secretions and bronchial constrictions are diminished by the vigorous administration of atropine. However, if the exposure has been overwhelming, amounting to many times the lethal dose, death may occur as a result of respiratory arrest and cardiac arrhythmia despite treatment. When overwhelming doses of the agent are absorbed quickly, death occurs rapidly without orderly progression of symptoms.

## 2-5. Diagnosis of Nerve Agent Poisoning

*a.* Nerve agent poisoning may be identified from the characteristic signs and symptoms. If exposure to vapor has occurred, the pupils will be very small, usually pinpointed. If exposure has been cutaneous, or has followed ingestion of a nerve agent in contaminated food or water, the pupils may be normal or, in the presence of severe systemic symptoms, slightly to moderately reduced in size. In this event, the other manifestations of nerve agent poisoning must be relied on to establish the diagnosis. No other known chemical agent produces muscular twitching

Table 2-2. Time Course of Effects of Nerve Agents

AGENT DISPERSED AS	TYPES OF EFFECTS	ROUTE OF ABSORPTION	DESCRIPTION OF EFFECTS	WHEN EFFECTS APPEAR AFTER EXPOSURE	DURATION OF EFFECTS AFTER	
					MILD EXPOSURE	SEVERE EXPOSURE
Vapor	Local	Respiratory	Rhinorrhea, nasal hyperemia, tightness in chest, wheezing	One to several minutes	A few hours	1 to 2 days
Vapor	Local	Eyes	Miosis, conjunctival hyperemia, eye pain, frontal headache	One to several minutes	Miosis—24 hours	2 to 3 days
Vapor	Systemic	Respiratory or eyes	Muscarinic, nicotinic, and central nervous system effects (see table 2-1)	Less than 1 minute to a few minutes after moderate or severe exposure; about 30 minutes after mild exposure	Several hours to a day	Acute effects: 2 to 3 days CNS effects: days to weeks
Liquid	Local	Eyes	Same as vapor effects	Instantly	Similar to effects of vapor	
Liquid	Local	Ingestion	Gastrointestinal (see table 2-1)	About 30 minutes after ingestion	Several hours to a day	2 to 5 days
Liquid	Local	Skin	Local sweating and muscular twitching	3 minutes to 2 hours	3 days	5 days
Liquid	Systemic	Bronchial tree	See table 2-1	Several minutes		1 to 5 days
Liquid	Systemic	Eyes	Same as for vapor	Several minutes		2 to 4 days
Liquid	Systemic	Skin	Generalized sweating	15 minutes to 2 hours		2 to 5 days
Liquid	Systemic	Ingestion	Gastrointestinal (see table 2-1)	15 minutes to 2 hours		3 to 5 days

After lethal or near lethal exposures to nerve agents, the time to onset of symptoms and to maximal severity of symptoms is shorter; it may be extremely brief after overwhelming exposure. Following exposure to lethal concentrations, the time interval to death depends upon the degree, the route of exposure, and the agent. If untreated, exposure to lethal concentrations of nerve agents can result in death 5 minutes after appearance of symptoms.

and fasciculations, rapidly developing pinpoint pupils, or the characteristic train of muscarinic, nicotinic, and CNS manifestations.

b. It is important that all service members know the following **MILD** and **SEVERE** signs and symptoms of nerve agent poisoning. Service members who have most or all of the symptoms listed below must **IMMEDIATELY** receive first aid (self-aid or buddy aid) (paras 2-11 a and b, respectively).

(1) **MILD** poisoning (self-aid). Casualties with **MILD** symptoms may experience most or all of the following:

- (a) Unexplained runny nose.
- (b) Unexplained sudden headache.
- (c) Sudden drooling.
- (d) Difficulty in seeing (dimness of vision and miosis).
- (e) Tightness in the chest or difficulty in breathing.
- (f) Wheezing and coughing.
- (g) Localized sweating and muscular twitching in the area of the contaminated skin.
- (h) Stomach cramps.
- (i) Nausea with or without vomiting.
- (j) Tachycardia followed by bradycardia.

(2) **SEVERE symptoms** (buddy aid). Casualties with **SEVERE** symptoms may experience most

or all of the **MILD** symptoms, plus most or all of the following:

- (a) Strange or confused behavior.
- (b) Increased wheezing and increased dyspnea (difficulty in breathing).
- (c) Severely pinpointed pupils.
- (d) Red eyes with tearing.
- (e) Vomiting.
- (f) Severe muscular twitching and general weakness.
- (g) Involuntary urination and defecation.
- (h) Convulsions.
- (i) Unconsciousness.
- (j) Respiratory failure.
- (k) Bradycardia.

Casualties with severe symptoms **WILL NOT** be able to treat themselves and **MUST RECEIVE** prompt buddy aid (para 2-11 b), CLS aid (paras 2-9 e and 2-12 c), and prompt follow-on medical treatment (paras 2-15 and 2-16) if they are to survive.

c. Casualties with **MODERATE** poisoning will experience an increase in the severity of most or all of the **MILD** symptoms. Especially prominent will be fatigue, weakness, and muscle fasciculations. The progress of symptoms from **MILD** to **MODERATE** indicates either inadequate treatment or continuing exposure to the agent.

## Section II. PREVENTION AND TREATMENT OF NERVE AGENT POISONING

### 2-6. Essential Elements of Prevention and Treatment

The essential prevention and treatment elements of nerve agent poisoning are—

- a. Donning the protective mask and hood at the first indication of a nerve agent attack.
- b. Administering the **MARK I** (para 2-11) as soon as any signs or symptoms are noted.
- c. Administering the **CANA** to **MODERATE** to severely poisoned casualties (para 2-12).

#### NOTE

The U.S. Navy does not use the **MARK I**. Instead, the Navy issues three atropine and three pralidoxime chloride (**2 PAM C1**) auto injectors per person.

d. Removing or neutralizing any liquid contamination immediately.

e. Removing airway secretions if they are obstructing the airway. Airway suction may be needed.

f. Establishing a patent airway (for example, with a cricothyroidotomy or endotracheal tube) and administering assisted ventilation, if required. Oxygen is desired, if available.

### 2-7. Prevention of Poisoning

a. The respiratory tract absorbs nerve agent vapor very rapidly. The protective mask must be put on **IMMEDIATELY** when it is suspected that nerve agent vapor is present in the air. Hold the breath until the mask is on, cleared, and checked. If the nerve agent concentration in the air is high, a few breaths may result in the inhalation of enough nerve agent to be incapacitating or even lethal. When the concentration in the air is low, a longer exposure may precede the onset of symptoms and the detection of nerve agent poisoning. Since the effects of a nerve agent are progressive and cumulative, the prevention of further absorption is urgent once symptoms have begun. Protective masks should be worn until the “all clear” signal is given.

b. **DO NOT** give nerve agent antidotes for preventive purposes **BEFORE** contemplated exposure to a nerve agent. To do so may enhance respiratory absorption of nerve agents by inhibiting bronchoconstriction and bronchial secretion. Atropine will degrade performance when taken in doses of more than 2 milligram (mg) without nerve agent exposure, especially when maximal visual acuity is required. Also, atropine will degrade an individual’s ability to

perform duties in a hot environment. Atropine is rapidly used up in the treatment of nerve agent poisoning. A person incapacitated by nerve agent poisoning will likely remain incapacitated since atropine will not reverse all the signs and symptoms of poisoning, even in large doses.

c. Nerve agents (liquid or vapor) can poison food and water. For details on management and decontamination of food and water, see FM 8-10-7.

## 2-8. Effects of Nerve Agent Antidotes

### a. General.

(1) *Atropine*. Atropine sulfate remains an essential drug in the treatment of nerve agent poisoning. It acts by blocking the effects of acetylcholine at muscarinic receptors and produces relief from many of the symptoms previously listed. If given in large doses, some therapeutic effects are also produced within the CNS although atropine does not readily penetrate the blood-brain barrier as does diazepam (para 2-8 a (3)), and central muscarinic receptors are thought not to be identical with those in the periphery. It is thought to counteract the respiratory depression in the medulla oblongata. Used alone, it has little influence on the mortality rate in the potentially fatal apneic cases for which assisted ventilation is many times more effective. However, the combination of adequate atropinization *plus* assisted ventilation is several times more effective in saving lives than assisted ventilation alone.

(2) *2 PM Cl. 2 PAM Cl* is an oxime which increases the effectiveness of drug therapy in poisoning by some—but not all—cholinesterase inhibitors. Unlike atropine, *2 PAM Cl* acts by blocking the nerve agent inhibition of cholinesterase and/or reactivating the inhibited acetylcholinesterase clinically at muscarinic sites. Thus *2 PAM Cl* relieves the skeletal neuromuscular block, as well as reactivating the acetylcholinesterase clinically at muscarinic sites. The role of *2 PAM Cl* is to block and reverse the bonding of the nerve agent to the acetylcholinesterase. Oximes must be given early in the poisoning; after a short period of time (different for each type of nerve agent), they may no longer be effective.

### NOTE

*2 PAM Cl* varies in its effectiveness against nerve agents. It is least effective against GD.

(3) *Diazepam*. Diazepam readily crosses the blood-brain barrier to block the effects of acetylcholine on the CNS, in contrast to the partial protection of atropine at best. Diazepam antagonizes the convulsive action of nerve agents. The addition of diazepam to the basic antidotes prevents or ameliorates convulsions in MODERATE to severe nerve agent poisoning.

### b. Rate of Absorption.

(1) *Atropine*. A 2-mg intramuscular (IM) injection will reach peak effectiveness in 3 to 10 minutes, then blood concentrations will decline. If the system is unchallenged by a nerve agent, a 2-mg IM injection will cause atropine effects for several hours. In the presence of a nerve agent challenge, the effectiveness of atropine is markedly reduced and the duration of the agent is significantly shortened. More frequent doses of atropine will be required to achieve and maintain atropinization.

(2) *2 PAM Cl*. Depending on the degree of intoxication, a 600-mg IM will be effective in 6 to 8 minutes and will maintain peak effectiveness for 1 hour or more. If the system is unchallenged by a nerve agent, a 600-mg IM will remain in the circulatory system for several hours without apparent effect.

(3) *Diazepam*. A 10-mg IM injection in the thigh ordinarily produces significant plasma levels in 10 minutes; peak plasma concentrations are obtained in about 1 hour. The concentrations will then decline over a prolonged period. Rapid administration of diazepam by IM autoinjector after nerve agent exposure may more effectively prevent or ameliorate convulsions. SEVERE nerve agent toxicity may require multiple 10-mg doses given at about 10 minute intervals for a maximum of three (3) injections (a total of 30 mg diazepam) to control convulsions.

### c. Symptoms Produced by the Antidotes.

#### (1) *Atropine*.

(a) The administration of a single dose of 2 mg (one autoinjector) of atropine to an individual who has absorbed minimal or no nerve agent produces MILD symptoms, including dryness of the skin, mouth, and throat, with slight difficulty in swallowing. The individual may have a feeling of warmth, slight flushing, rapid pulse, some hesitancy of urination, and an occasional desire to belch. The pupils may be slightly dilated but react to light. In some individuals, there may be MILD drowsiness and slowness of memory and ability to recall. Recipients of atropine may have the feeling that their movements are slow and their near vision is blurred. Some individuals may be mildly relaxed. These symptoms should not interfere with ordinary activity, except in the occasional individual who proves to be unusually reactive to the "sensation" effects of atropine (particularly the feeling of drowsiness). However, mental reaction may be slightly slowed down (for this reason, aviators must not fly an aircraft after taking atropine until cleared by the flight surgeon). If the administration of 2 mg of atropine is repeated within an hour without nerve agent challenge, the symptoms become MODERATE. In most of these individuals, there will be some CNS symptoms (such as drowsiness, fatigue, slowness of memory and ability to recall, the

feeling that body movements are slow, and blurred near vision); but they can continue ordinary activity with some loss of efficiency. Near vision may be impaired for as long as 24 hours. After repeated injections of atropine, heat-stressed individuals will become casualties ((b) below). A third 2-mg dose of atropine (again without nerve agent challenge) administered within an hour will result in severe symptoms which will not permit ordinary activity—in fact, most individuals will be incapacitated. **SEVERE** incapacitating symptoms of overatropinization (nerve agent antidote poisoning) are a very dry mouth; swelling of the tongue and oral mucous membranes; difficulty in swallowing; thirst; hoarseness; dry and flushed skin; dilated pupils; blurred near vision; tachycardia (rapid pulse); urinary retention (in older individuals); constipation; slowing of mental and physical activity; restlessness; headache; disorientation; hallucinations; depression; increased drowsiness; extreme fatigue; rapid respiratory panting; and respiratory distress. Abnormal behavior may require restraint. The effects of atropine without nerve agent challenge are fairly prolonged, lasting 3 to 5 hours after one or two injections and 12 to 24 hours after marked overatropinization. Overatropinization may be incapacitating but presents little danger to life in a temperate environment for the nonheat-stressed individual. A single dose of 10 mg of atropine has been administered intravenously to *normal young adults* without endangering life—even in the absence of any prior absorption of a nerve agent—although it has produced very marked signs of overdose.

#### NOTE

While an unchallenged dose of atropine may allow individuals to continue normal duties, they must be closely monitored for possible heat injury. This is especially important when at **MOPP 4** and the individuals' ability to perspire is reduced due to atropine.

(b) In hot, desert, or tropical environments or in heat-stressed individuals, doses of atropine tolerated well in temperate climates may be seriously incapacitating by interference with the sweating mechanism. This can sharply reduce the combat effectiveness of troops who have suffered little or no exposure to a nerve agent. In hot climates or in heat-stressed individuals, one dose (2 mg) of atropine can reduce efficiency. Two doses (4 mg) will sharply reduce combat efficiency, and 6 mg will incapacitate troops for several hours. In hot, humid climates, individuals who have inadvertently taken an overdose of atropine and are exhibiting signs of atropine intoxication should have their activity restricted. In addition, these casualties must be kept as cool as

possible for 6 to 8 hours after injection to avoid serious incapacitation. Usually, the casualties will recover fully in 24 hours or less from a significant overdose of atropine.

(c) Experience in chemical operations has shown that when troops become alarmed, some believe they have been exposed to more chemical agents than they actually have been. Hence, it is important that service members **NOT** give themselves more than one atropine injection (2 mg). Casualties who are able to ambulate and know who they are and where they are **WILL NOT** need any more atropine injections. If the symptoms do recur additional atropine, up to two more injections for a total of three (3), can be administered to these casualties. A service member must consult with a buddy to determine if he or she needs additional injections of atropine. If an individual's heart rate is above 90, breathing appears normal, bronchial secretions have diminished, and the skin is dry, the individual does not need anymore atropine at this time. Additional atropine is given by a **buddy** since casualties requiring more will be unable to administer additional injections to themselves. The additional administration of atropine to a service member with only **MILD** symptoms must be approached cautiously with at least 10 to 15 minutes elapsing between successive injections. If the signs of nerve agent poisoning (para 2-5) disappear, or if signs of atropinization, such as a heart rate above 90, diminished bronchial secretions, and dry skin, appear during one of these 10- to 15-minute periods, no further injections should be administered. These casualties should remain under observation without further injections of atropine unless signs of nerve agent intoxication reappear.

#### NOTE

Although one means of determining the casualty's need for additional atropine is the heart rate, assessing his or her respiratory effort is important in the evaluation. Labored breathing, including coughing, noisy breathing, wheezing, and gasping for air, indicates the need for administering additional atropine. When the heart rate is not obtainable, the need for additional atropine may be based on the degree of respiratory impairment. When adequate atropine has been given, labored breathing efforts will be relieved. This assessment must be performed without compromising the protective posture of **MOPP**.

(d) Patients with severe symptoms due to systemic absorption of a nerve agent have increased tolerance for atropine. Multiple doses maybe required before signs of atropinization appear, such as heart

rate above 90, diminished bronchial secretions, and dry skin. Large doses are required to ameliorate the muscarinic effects of nerve agent poisoning. The absence of increased tolerance for atropine indicates that nerve agent poisoning probably is not present or is **MILD**. In the presence of severe nerve agent poisoning, as much as 50 mg of atropine may be required for treatment in a 24-hour period. More than three injections of atropine will be administered only by the CLS or medical personnel.

(2) **2 PAM Cl. MILD** visual changes may be a side effect of **2 PAM Cl.** After the administration of three injections of **2 PAM Cl.**, generally no further

oxime benefit is attained by additional injections of **2 PAM Cl.**

(3) **Diazepam.** The administration of a single dose of 10 mg (one autoinjector of **CANA**) to an individual who has absorbed minimal or no nerve agent produces significant performance decrements for about 2 to 5 hours. The individual will have impaired vision and decision-making functions over this time period. Overall alertness may be impaired. There could also be breathing difficulty. For this reason, casualties should be lying on their side until they are alert again. There may be transient irritation, as well as pain, at the injection sites.

### Section III. SELF-AID, BUDDY AID, COMBAT LIFESAVER PROCEDURES, AND COMBAT MEDIC/CORPSMAN TREATMENT

#### 2-9. Principles of Self-Aid and Buddy Aid

a. The protective mask and hood must be put on **IMMEDIATELY** at the first signs of a chemical attack. (The protective overgarment should have already been put on prior to the use of chemicals on the battlefield.) Stop breathing, put on your mask, clear and seal the mask, and resume breathing. Secure the mask hood. The mask and protective clothing are worn continually until the "all clear" signal is given.

b. **IMMEDIATELY** mask any casualty that does not have a mask on if the atmosphere is still contaminated.

c. The appearance of nerve agent poisoning symptoms calls for the immediate IM injection of the nerve agent antidote (paras 2-11 and 2-12). Since inhalation will be the most common route of exposure, the most likely initial symptom will be rhinorrhea (runny nose), then miosis (dim vision), followed by a feeling of tightness or constriction in the chest. After ocular (eyes) splash, there will be immediate miosis. After cutaneous (skin) splash, the initial systemic symptoms may be localized sweating and localized muscular twitching, followed by nausea and abdominal cramps. After ingestion, the first symptoms are likely to be nausea and vomiting. In any case, use the nerve agent antidotes as directed (paras 2-11 and 2-12).

d. Promptly remove any liquid nerve agent on the skin, on the clothing, or in the eyes.

(1) If a liquid nerve agent gets on the skin, decontamination must be accomplished within 1 minute (see app D). Then continue the mission. Examine the contaminated area occasionally for local sweating and muscular twitching. If these occur, the nerve agent antidote should be administered. Combat duties should be continued, as systemic symptoms of nerve agent poisoning may not occur or may be **MILD** if the decontamination was done immediately and successfully.

(2) If a drop or splash of liquid nerve agent

gets into the eye, instant action is necessary to avoid serious effects. Irrigate the eye immediately with water as described in appendix D. During the next minute, the pupil of the contaminated eye should be observed by a buddy. If the pupil rapidly gets smaller, a nerve agent antidote should be administered. If the pupil does not get smaller, the ocular contamination was not caused by a nerve agent and atropine is not needed.

e. If good relief is obtained from one set of **MARK I** injections and breathing is normal, carry on with combat duties. Dryness of the mouth is a good sign—it means enough atropine has been taken to overcome the dangerous effects of the nerve agent. If symptoms of the nerve agent are not relieved, the service member should be administered two more sets of the **MARK I** injections plus one injection of **CANA** by a buddy, in accordance with the provisions of paragraph 2-11. If symptoms still persist and the pulse (heart rate) drops below 90 per minute, bronchial secretions persist, or the skin remains moist, then the service member can be administered additional atropine injections by the CLS or medical personnel (who carry additional atropine for the treatment of nerve agent casualties) to maintain adequate atropinization. The CLS and combat medic/corpsman also carry extra **CANA** for administration to nerve agent casualties (para 2-12). The CLS or combat medic/corpsman can administer additional **CANA** up to a maximum of three before evacuating the casualty. Evacuate the service member to a field MTF as soon as the combat situation permits.

f. Atropine and **2 PAM Cl.** by injection do not relieve the local effects of nerve agent vapor on the eyes. Although the eyes may hurt and there may be difficulty in focusing and a headache, the service members should carry on with their duties to the best of their ability. These symptoms are annoying but not dangerous.

g. Exposure to high concentrations of a nerve agent may bring on incoordination, mental confusion, and/or collapse so rapidly that the casualty cannot perform self-aid. If this happens, the nearest able service member must render buddy aid.

h. **SEVERE** nerve agent exposure may rapidly cause unconsciousness, muscular paralysis, and the cessation of breathing. When this occurs, antidote alone will not save life. **IMMEDIATELY** after a buddy administers three sets of the MARK I and CANA, assisted ventilation must be started by medical personnel, if a resuscitation device is available. Assisted ventilation should be continued until normal breathing is restored.

## 2-10. The Nerve Agent Antidote Kit, MARK I

The Nerve Agent Antidote Kit, MARK I (fig E-1), is an antidote used by the Army and the Air Force in the treatment of nerve agent poisoning.

a. *Description.* The MARK I kit consists of four separate components: the atropine autoinjector, the 2 PAM CI autoinjector, the plastic clip, and the foam carrying case.

(1) The atropine autoinjector consist of a hard plastic tube containing 2 mg (0.7 milliliter (ml)) of atropine in solution. It has a pressure activated coiled spring mechanism which triggers the needle for injection of the antidote solution. The container is white plastic with yellow lettering on green identification and directions labels. The safety cap is yellow plastic attached to the clip at the rear of the container. The needle end is a green plastic cap which, when pressure is applied, activates the spring mechanism.

(2) The 2 PAM CI autoinjector is a hard plastic tube which dispenses 600 mg/2 ml of 2 PAM CI (300 mg/ml) solution when activated. It has a pressure activated coiled spring mechanism identical to that in the atropine autoinjector. The container is clear plastic with black lettering on a brown identification label. Directions are in black lettering on a white background. The safety cap is gray plastic attached to the clip at the rear of the container. The needle end is black plastic.

(3) The clip is made of clear hard plastic constructed to hold the pair of autoinjectors together while attached to their safety caps. The safety caps are held flush to the bottom of the plastic clip by a movable metal retaining flange. The clip container recesses are labeled with black numbers: "1" for the atropine and "2" for the 2 PAM CI autoinjector.

(4) The foam envelope is a charcoal gray form-fitting case with pressed seams and is designed to carry both autoinjectors. The envelope is used for shipping purposes only and is removed by service members prior to putting the MARK I kits in their mask carrier.

b. *Issue to Service Members.* In the U.S. Army and the U.S. Air Force, each person is authorized to carry three sets of the MARK I kit for the treatment of nerve agent poisoning. The U.S. Navy, however, does not use the MARK I but, rather, its antidote components are issued as three atropine and three 2 PAM CI autoinjectors per person.

c. *Protection Against Freezing.* The atropine and the 2 PAM CI solutions freeze at about 30°F (1°C). Therefore, when the temperature is below freezing, the MARK I injectors should be protected against freezing. Autoinjectors issued to the individual service member are normally carried in the protective mask carrier. During cold weather when the temperature is below freezing, the injectors should be carried in an inside pocket close to the body. (Should the MARK I injectors become frozen, they can be thawed and used.)

## 2-11. Principles in the Use of the Nerve Agent Antidote Kit, MARK I

The following are principles to be followed in the administration of the MARK I (fig E-1).

a. *Self-Aid.* If you experience most or all of the **MILD** symptoms of nerve agent poisoning (para 2-5), you should **IMMEDIATELY** hold your breath (**DO NOT INHALE**) and put on your protective mask. Then administer *one* set of MARK I injections into your lateral thigh muscle (or buttocks). (Self-aid procedure for administering the autoinjectors is found in app E.)

(1) Wait 10 to 15 minutes after giving yourself the *first* set of injections since it takes that long for the antidote to take effect. If you are able to ambulate, know who you are, and where you are, you **WILL NOT** need a second set of MARK I injections.

### WARNING

**Giving yourself a second set of injections may create a nerve agent antidote overdose, which could result in incapacitation.**

(2) If symptoms of nerve agent poisoning are not relieved after administering one set of MARK I injections, seek someone else to check your symptoms. A buddy must administer the second and third sets of injections, if needed.

b. *Buddy Aid.* If you encounter a service member suffering from **SEVERE** signs of nerve agent poisoning (para 2-5), render the following aid:

(1) Mask the casualty, if necessary. Do not fasten the hood.

(2) Administer, in rapid succession, three sets of the MARK I. Follow administration procedures outlined in appendix E.

**NOTE**

Use the casualty's own antidote auto-injectors when providing aid. Do not use your injectors on a casualty. If you do, you may not have any antidote available when needed for self-aid.

*c. Combat Lifesaver.* The CLS must check to verify if the individual has received three sets of the MARK I. If not, the CLS performs first aid as described for buddy aid above. If the individual has received the initial three sets of MARK I, then the CLS may administer additional atropine injections at approximately 15 minute intervals until atropinization is achieved (that is a heart rate above 90 beats per minute; reduced bronchial secretions; and reduced salivation). Administer additional atropine at intervals of 30 minutes to 4 hours to maintain atropinization or until the casualty is placed under the care of medical personnel. Check the heart rate by lifting the casualty's mask hood and feeling for a pulse at the carotid artery. Request medical assistance as soon as the tactical situation permits.

*d. Combat Medic/Corpsman.* A casualty has received three sets of MARK I; however, atropinization has not been achieved. Administer additional atropine at approximately 15 minute intervals until atropinization is achieved (that is a heart rate above 90 beats per minute; reduced bronchial secretions and reduced salivation). Administer additional atropine at intervals of 30 minutes to 4 hours to maintain atropinization or until the casualty is evacuated to an MTF. Check the heart rate by lifting the casualty's mask hood and feeling for a pulse at the carotid artery. Provide assisted ventilation for severely poisoned casualties, if equipment is available. Monitor the patient for development of heat stress.

## 2-12. Principles in the Use of Convulsant Antidote for Nerve Agents

The following are principles to be followed in the administration of CANA (fig E-1).

*a. Self-Aid.* The CANA is NOT for use as self-aid. If you know who you are, where you are, and what you are doing, you do not need CANA. If symptoms do not subside after self-administering one MARK I, seek assistance from a buddy.

*b. Buddy Aid.* In addition to administering the MARK I antidotes for nerve agents as buddy aid, also administer the CANA.

(1) Mask the casualty, if necessary. Do not fasten the hood.

(2) Administer the CANA with the third MARK I to prevent convulsions. **DO NOT** administer more than one CANA. Follow administration procedures outlined in appendix E.

**NOTE**

**DO NOT** use your own CANA on the casualty. You may not have any antidote for your own treatment, if needed.

*c. Combat Lifesaver and Medic/Corpsman.* The CLS or medic/corpsman should administer additional CANA to casualties suffering convulsions. Administer a second, and if needed, a third CANA at 5 to 10 minute intervals for a maximum of three injections (30 mg diazepam). Follow the steps and procedures described in buddy aid for administering the CANA. **DO NOT** give more than two additional injections for a total of three (one buddy aid plus two by CLS or medic/corpsman).

## 2-13. Effects of Atropine

The effect of atropine administration on **MILD** and **MODERATE** cases of nerve agent poisoning may help confirm the diagnosis. Atropine injection alleviates most of the muscarinic manifestations. It has little effect on the CNS symptoms and no effect on the nicotinic symptoms. If the casualty has absorbed little or no nerve agent, the administration of a single dose of 2 mg of atropine produces symptoms of mild atropinization (tachycardia, dry mouth) in most individuals and repetition of this dose within 1 or 2 hours produces **MODERATE** symptoms of atropinization in almost all individuals. In contrast, a casualty with **MODERATE** symptoms of nerve agent poisoning will not develop symptoms of atropinization after administration of 2 mg of atropine. A casualty with severe symptoms of nerve agent poisoning may tolerate—indeed may require—considerably more than 4 mg of atropine (as much as 50 mg in 24 hours).

## 2-14. Effects of Convulsant Antidote for Nerve Agents

Diazepam (CANA) is intended to prevent or ameliorate convulsions in **MODERATE** to severe nerve agent poisoning. A casualty with severe nerve agent poisoning may require multiple doses of CANA (30 mg or more).

## Section IV. TREATMENT IN THE FIELD (MEDICAL TREATMENT FACILITY)

### 2-15. Administration of the Nerve Agent Antidotes

Upon arrival at the MTF a casualty is still presenting signs/symptoms of nerve agent poisoning (para 2-5). The casualty has received self-aid, buddy aid, CLS care, or treatment by the combat medic/corpsman, or other medical personnel in the field before and during evacuation. Additional injections of the nerve agent antidote(s) must be administered at the field MTF.

*a. Atropine.* Atropinization should have been achieved before the casualty is evacuated to an MTF; if not, then atropine is administered as follows:

(1) **MILD** symptoms should be treated by administering 2 mg of the atropine every 15 minutes until signs of atropinization (dry mouth and skin, with cleared pulmonary secretions) are achieved. Maintain atropinization until muscarinic signs disappear.

(2) **MODERATE** symptoms should be treated by administering 2 mg of the atropine every 10 to 15 minutes until atropinization is achieved. Maintain atropinization by injecting 2 mg of atropine as often and long as needed.

(3) **SEVERE** symptoms should be treated by administering 2 mg of atropine as frequently as required until atropinization is achieved. Maintain atropinization by injecting 2 mg of atropine every 10 to 30 minutes as long as needed.

#### NOTE

Smoking should be prohibited until the symptoms of nerve agent poisoning have subsided.

*b. 2 PAM Cl.* Specifically as an adjunct to atropine, **2 PAM Cl** may be used to increase the effectiveness of therapy in poisoning by some, but not all nerve agents. An important facet of the activity of **2 PAM Cl** in such therapy is the reduced duration of required assisted ventilation.

(1) **MILD** symptoms should have been treated by administering at least one 600-mg **IM** injection of **2 PAM Cl**.

(2) **MODERATE** symptoms should have been treated by administering *one* or more 600-mg **IM** injections of **2 PAM Cl**.

(3) **SEVERE** symptoms should have been treated by administering *three* 600-mg **IM** injections of **2 PAM Cl**. Repeat the dose at least every hour if respiration has not improved. Generally, no increased oxime benefit is obtained after three injections of **2 PAM Cl**.

*c. Diazepam.* Diazepam is used specifically as a prevention of or treatment for convulsions in nerve

agent poisoned casualties. If brain damage is to be prevented in **MODERATE** to severe nerve agent poisoned casualties, **CANA** must be administered early. Seizures should be anticipated in all **MODERATE** to severe cases and treated with the **CANA** and repeated as necessary.

### 2-16. Administration of Follow-on Medical Treatment

The following medical treatment may also be administered in a CPS or a clean (uncontaminated) environment, depending on the patient's needs. Modifications of these procedures may be used in a contaminated environment although an increase in exposure will occur. The alternative of not performing these procedures is death of the patient.

*a. Administration of Additional Atropine.* For patients who are in severe respiratory distress or are convulsing, all three sets of their **MARK I** auto-injectors should have been given. (Convulsions are treated with diazepam, as described in *c* below.) If relief does not occur and bronchial secretions and salivation do not decrease, administer additional atropine as often as needed. In severe nerve agent poisoning, the effect of each 2-mg atropine injection may be transient, lasting only 5 to 15 minutes. Therefore, these patients must be closely observed and atropine repeated at intervals that relieve (or counteract) the muscarinic effects of the nerve agent and maintain mild atropinization for as long as necessary.

*b. Management of Bronchial Secretions and Salivation.* Patients having excessive airway secretions and salivation (an indication for additional atropine) should be lying on their side, with the foot of the litter or bed elevated, if possible, to promote drainage. If airway obstruction is occurring, the collar should be loosened, the tongue pulled out, and the saliva and mucus cleared periodically from the mouth and pharynx by suction. Then an oropharyngeal airway may be inserted and suction carried out intermittently, as needed (through and around the airway). If, despite concentrated efforts to carry out assisted ventilation, the upper airway remains obstructed and adequate exchange of air does not occur, insert an endotracheal tube. High airway resistance because of bronchial constrictions and secretions may be decreased with the administration of additional atropine.

*c. Management of Convulsions.* Severely poisoned casualties that develop convulsions usually progress rapidly to unconsciousness and generalized muscular weakness or flaccid paralysis, at which point external evidences of convulsions cease. Seizures should be anticipated in all **MODERATE** to severe

cases and expectantly treated with CANA/diazepam and repeated as necessary. Seizing is a prominent feature of nerve agent poisoning, especially GD. Administer CANA until seizures are controlled.

**d. Treatment of Ocular Symptoms.** Ocular symptoms produced by local absorption of a nerve agent do not respond to the systemic administration of atropine. However, minimal pain relief may be obtained by the local instillation of atropine sulfate ophthalmic ointment (1 percent), repeated as needed at intervals of several hours for 1 to 3 days. If local ocular effects of a nerve agent are present, the size of the pupils cannot be used as an indicator of the systemic effects of the nerve agent or the atropine.

**e. Gastric Lavage.** If water or food contaminated with a nerve agent has been ingested, colicky abdominal pains, substernal tightness, increased salivation, and perhaps nausea and vomiting will occur 1/2 hour or later. If ingestion is known to have occurred, early gastric lavage with water should be done.

**f. Removal of Liquid Nerve Agent.** Any liquid nerve agent on the skin or in the eyes should be removed immediately.

**g. Assisted Ventilation.** If respiration is severely impaired or if it ceases after administration of atropine,

cyanosis will ensue and death will occur within minutes unless immediate effective assisted ventilation is begun and maintained until spontaneous respiration is resumed. Far forward in the field, a cricothyroidotomy is the most practical means of providing an airway for assisted ventilation, using a hand-powered ventilator equipped with an NBC filter. When a casualty reaches an MTF where oxygen and a positive pressure ventilator is available, these should be employed continuously until adequate spontaneous respiration is resumed. An endotracheal tube will most likely be required.

**NOTE**

Treatment outlined in paragraphs 2-15 and 2-16 is based on the U.S. Army doctrine on the use of the MARK I and CANA (diazepam). These procedures do not address the uniqueness of other environments (such as the threat in naval operations) where alternatives may be more constrained, requiring modification in the procedures. Procedures to address these variations should be issued by the services concerned in accordance with their specific needs.

**Section V. NERVE AGENT PYRIDOSTIGMINE PRETREATMENT**

**2-17. Purpose**

a. This section prescribes the use of nerve agent pyridostigmine pretreatment as an adjunct to the MARK I. Studies in many different types of animals indicate that when pyridostigmine is used in conjunction with the MARK I (para 2-10 and app E), the survivability of nerve agent poisoned casualties may be enhanced. Also covered in this section are the individual, unit, and command responsibilities for the pretreatment regimen.

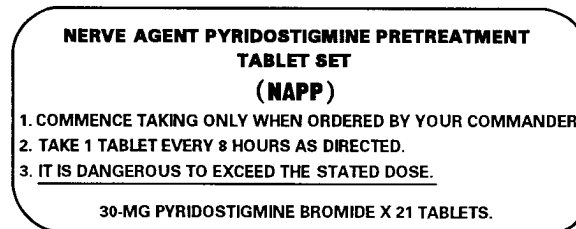
b. Animal data suggest that any potential benefits that may be derived from use of this pretreatment regimen will be realized only in nerve agent poisoned casualties who have been treated with the Mark I at the time of nerve agent exposure, and who have taken their pretreatment medication within 8 hours prior to nerve agent exposure.

c. Minimal detrimental effects are expected at the recommended dosages. Adverse effects and contraindications are described in paragraph 2-22 below.

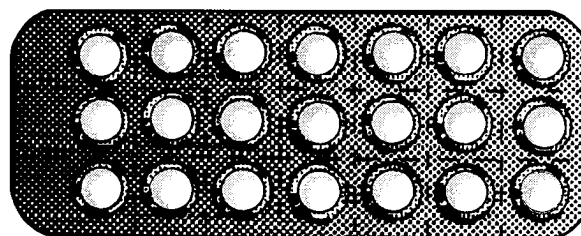
**2-18. The Nerve Agent Pyridostigmine Pretreatment Tablet Set**

a. The Nerve Agent Pyridostigmine Pretreatment (NAPP) Tablet Set (fig 2-2) contains the pretreatment medication to be taken within 8 hours prior to exposure to nerve agents at which time the MARK I

is used. The NAPP consists of a blister pack containing 21 tablets. Each tablet consists of 30-mg pyridostigmine bromide. Each blister pack (NAPP) contains enough tablets for 7 days (1 taken every 8 hours).



(A) SAMPLE OUTER WRAPPER.



(B) SAMPLE PYRIDOSTIGMINE BROMIDE TABLETS.

Figure 2-2. Nerve Agent Pyridostigmine Pretreatment Tablet Set.

b. Service members are initially issued one **NAPP** when the chemical protective ensemble is expected to be opened for use. They are responsible for carrying the **NAPP** and safeguarding it against loss. Service members will secure the blister pack in the sleeve or breast pocket of the chemical protective ensemble (or in another part of the ensemble, as directed by local standing operating procedure (SOP)).

**NOTE**

In conjunction with the **NAPP**, service members should be issued an additional M291 Skin Decontaminating Kit (fig E-1). The M291 kit will be carried in the protective mask carrier or as specified in unit SOP.

c. Orders to start taking the **NAPP** will be issued by the proper authority within the chain of command.

d. Resupply will be provided by combat, combat support, and combat service support units.

**2-19. Effects of Pyridostigmine Bromide**

a. Pyridostigmine bromide protects an enzyme (known as acetylcholinesterase) in the body from the

action of nerve agents. Muscles function as a result of nerve impulses and the release of specific chemical substances. A chemical transmitter, acetylcholine, acts at the neuromuscular junction (where the nerve interfaces with the muscle) (fig 2-3). When a nerve impulse reaches the neuromuscular junction, acetylcholine is released, thereby causing the muscle to contract. The enzyme, acetylcholinesterase, stops the action of acetylcholine on the muscle after the muscle has contracted. Nerve agents block the acetylcholinesterase; there is an accumulation of excessive acetylcholine at the neuromuscular junction resulting in nerve agent poisoning and its accompanying symptoms. Pyridostigmine protects acetylcholinesterase against nerve agents, thus preventing the accumulation of excessive acetylcholine when the MARK I is administered.

b. Pyridostigmine is not a “true” pretreatment. A true pretreatment would, by itself, provide some protection against chemical agents. Pyridostigmine is an *antidote enhancer*. Though **NOT** providing protection by itself, pyridostigmine significantly **ENHANCES** the efficacy of the MARK I within 1 to 3 hours after taking the first tablet. Maximal benefit develops with time and is reached when a tablet is taken every 8 hours.

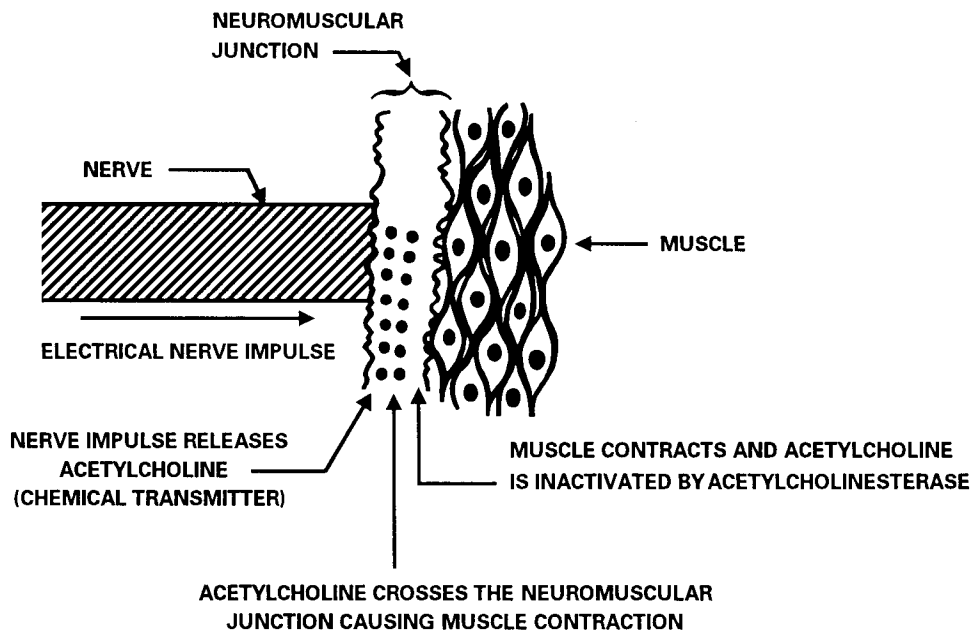


Figure 2-3. Schematic neuromuscular junction (not to scale).

## 2-20. Principles in the Use of the Nerve Agent Pyridostigmine Pretreatment Tablet Set

a. To be maximally effective, one pyridostigmine bromide tablet should be taken every 8 hours on a continuous basis prior to exposure to a nerve agent until all 21 tablets in the blister pack have been taken, or the individual has been directed to discontinue taking the medication. If pyridostigmine is to be continued, another blister pack of the medication must be issued. This regimen maintains an effective blood level of the medication. If a tablet is not taken every 8 hours, the beneficial effect of pyridostigmine as a pretreatment significantly diminishes after 8 hours from the last tablet.

b. The use of the pyridostigmine pretreatment medication does not change the administration of MARK I.

### NOTE

Do not attempt to give a NAPP tablet to a casualty with nerve agent symptoms.

c. At times a commander may have to make a decision to defer administration of the NAPP on schedule. Examples of this would be when service members—

(1) Have experienced sleep deprivation. The commander would have to decide whether the service members should be allowed to sleep or be awakened to take the pretreatment.

(2) Are in a contaminated environment. The commander would have to decide whether or not to delay administration of the medication until the unit is safely out of the contaminated area (para *d* below). In any case, the benefits versus the risks should be carefully weighed before a decision is reached.

d. When the order to take pyridostigmine has been given, it should be taken as directed (para 2-21). As long as the environment is contaminated, it is desirable to continue the pretreatment. The pretreatment should continue regardless of MOPP level since the protective posture could be breached at any time. Command guidelines should be developed for situations such as—

(1) Providing collective protection or rest and relief shelters so that personnel can remove their protective mask and take the tablets, or relocate small groups to an uncontaminated area, if possible.

(2) Taking the tablets while in MOPP 4 would be hazardous. (Examples: Troops are operating at night without lights or are in a chemical agent vapor environment.) In either case it would be more appropriate to delay taking the medication for a few hours until the tablets can be taken in a less hazardous environment.

e. The NAPP should not be taken during pregnancy.

## 2-21. Administration of Pyridostigmine Pretreatment in an Uncontaminated Environment

One 30-mg tablet is to be taken by mouth, with sufficient water to assist in swallowing the medication, every 8 hours as directed by your commander. *If a dose is missed, do not make it up. Do not take 2 tablets at once because of a missed dose—merely start again with 1 tablet every 8 hours.* Taking 2 tablets at once could result in adverse side effects. Taking more than 1 tablet at a time **DOES NOT** provide additional protection—in fact, it may be more hazardous if there is exposure to a nerve agent.

a. When the order to take pyridostigmine pretreatment has been given, it should be taken as directed, even though the protective mask is worn.

b. During hours of darkness while in an uncontaminated environment, the NAPP will be administered using the above schedule.

## 2-22. Signs and Symptoms of Pyridostigmine Bromide Overdose, Adverse Reactions, and Contraindications

Although no detrimental effects are expected at the recommended dosage, depending on the length of time and the amount of medication taken, as well as individual physiologic variations, some individuals may have contraindications for taking pyridostigmine bromide while others may experience adverse reactions.

a. Signs and symptoms of overdose, adverse reactions, or side effects are—

(1) Abdominal cramps.

(2) Nausea and vomiting.

(3) Diarrhea.

(4) Blurring of vision, miosis.

(5) Increased bronchial secretions.

(6) Cardiac arrhythmias, hypertension.

(7) Weakness, muscle cramps, and muscular twitching.

(8) Skin rash.

b. Since pyridostigmine bromide may increase bronchial secretions and aggravate bronchiolar constriction, caution should be used in its administration to personnel with bronchial asthma.

c. Pyridostigmine bromide may cause urinary obstruction.

d. Additional contraindications include hyperthyroidism, sensitivity to bromide, peptic ulcer disease, and low serum acetylcholinesterase.

e. If any of the above signs/symptoms occur, the service member should consult unit medical personnel as soon as possible.

**2-23. Emergency Medical Treatment for Pyridostigmine Adverse Side Effects, Allergic Reactions, and Overdose**

Ordinarily, discontinuing pyridostigmine should be adequate to alleviate the signs and symptoms of adverse side effects, allergic reactions, and overdose. Pyridostigmine may persist in the blood for as long as 24 hours; however, after the blood level peaks in about 4 hours, the effects of the medication diminish gradually.

a. Emergency treatment for an overdose of pyridostigmine requires the administration of atropine in adequate doses to overcome the cholinergic crisis. Initially, the 2-mg atropine autoinjector found in the MARK I kit should be used. In most cases, this will be sufficient. Further administration of atropine may be necessary to control the cholinergic effects of pyridostigmine. If additional atropine is required, 2 mg should be administered by medical personnel every 15 to 20 minutes, thereby permitting the previous injection of atropine to exert its anticholinergic effect prior to the next injection.

b. **SEVERE** cases may require assisted ventilation because of weakness, but would be unusual when the pretreatment medication was administered every 8 hours as directed.

c. When stabilized, the patient should be evacuated for further observation and treatment.

**2-24. Responsibilities**

a. The corps/division/wing commander will—

(1) Decide whether to begin, continue, or discontinue the administration of **NAPP** based on the threat. The intelligence officer, chemical officer, and the surgeon act as advisors to the commander in making his decision if a chemical nerve agent threat exists (for example, the enemy having nerve agents in the combat zone or the probability of their use). After 3 days of self-administration of **NAPP** by the service member, combat conditions should be reevaluated by the commander and his staff to determine whether to continue the medication or not. However, orders to discontinue the pretreatment **CAN** and **SHOULD** be made at any time, depending on the situation. If the pretreatment is to be continued, then a second blister pack must be ordered while the service member completes the administration of the 7 days (21 tablets) and is issued the second pack on the 7th day. *Administration of the medication beyond 14 days is not*

*recommended without a thorough evaluation of the situation and recommendation of the medical authority. However, the magnitude of the threat may outweigh any possible adverse side effects and indicate continuance of the pretreatment.*

(2) Train the service members to faithfully take the **NAPP** as directed to enhance their survivability if they are exposed to a nerve agent. Service members must be trained to take the **NAPP** during the day, at night, and while in MOPP 4, should these procedures become necessary.

(3) Issue unit **SOPs** for the retention and decontamination of the **NAPP** blister pack during personnel decontamination and overgarment exchange.

b. Units will—

(1) Obtain the supplies of **NAPP** through medical supply channels.

(2) Maintain at least a 2-week supply of **NAPP** per member of the unit. One **NAPP** is issued to each member of the unit. An additional week's supply of **NAPP** for each individual in the unit will be maintained in the unit area. Authorized quantities will be commensurate with the latest doctrine for its use.

(3) Store the **NAPP** for individual issue and request replacements as the items are issued, or as they exceed their labeled shelf life. The **NAPP** should be stored (refrigerated) in temperatures ranging from 350 to 46°F (2° to 8°C). If the medication is removed from refrigeration for a total of 6 months, it should be assumed that it has lost its potency and should not be used.

(4) Issue the **NAPP** to the service members at the time the chemical protective ensemble is expected to be opened for use.

c. Unit medical personnel will—

(1) Recognize the signs and symptoms of pyridostigmine overdose, adverse reactions, and side effects (para 2-22 above) for determining, on an individual basis, whether or not a service member is to continue the **NAPP** based on any adverse reaction to the medication.

(2) Advise the commander if any serious problems occur.

d. The individual service member will—

(1) Take the **NAPP** as directed and in accordance with the provisions of paragraph 2-20 above.

(2) Secure the **NAPP** against loss.