Chapter 4

NEUROPSYCHIATRIC CASUALTIES OF NUCLEAR, BIOLOGICAL, AND CHEMICAL WARFARE

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Kerr Eby *Match Sellers, Class of '17* 1918

Kerr Eby was initially a member of the Ambulance Corps in the U.S. Army in World War I, then transferred to the Engineers, and went to the front lines in France. His painting depicts a group of soldiers blinded during a gas attack. Whereas the neuropsychiatric casualties of other forms of warfare may present as individuals, the casualties of nuclear, biological, and chemical warfare are most often seen as groups because the agent, whatever it may be, is delivered in a dispersed form to affect as many troops as possible. The title, *Match Sellers*, refers to a possible occupation, from a previous era, for these soldiers when they return home.

Art: Courtesy of US Center of Military History, Washington, DC.

INTRODUCTION

Although outlawed by both the Hague Convention[1] and the Geneva Convention,[2] chemical warfare continues to exist along with potential use of nuclear and biological warfare. In recent wars it has been alleged that the former Soviets used mycotoxins against Afghan guerrillas and that Vietnam used "yellow rain" (mycotoxins) in Cambodia and Laos. Iraq used chemical agents against Iranian soldiers and Kurdish rebels, and had the ability to use them against coalition forces in the Persian Gulf War. The long history of the use of such agents is likely to continue. This usage has usually occurred when one side has, or believes that it has, superiority in such weaponry. During World War II, the Germans had a superior capability in the form of nerve agents; but, believing the Allies to have a similar capability, they did not use them. There is, therefore, a compelling argument in favor of the deterrent effect of parity in chemical warfare capability. Tear gas and nausea-producing agents, which were not designated as prohibited agents by the Hague[1] and Geneva[2] conventions, were used by U.S. forces in Vietnam.[3–5] Exhibit 4-1 reveals the frequency of alleged usage of chemical and biological weapons since World War I. This review indicates that the U.S. armed forces or modern armies must be prepared for the possible use of such agents in future wars.

The use of tactical (battlefield) nuclear weapons is possible not only between the major powers but also between smaller industrialized and nonindustrialized nations. In addition to the nations known to have nuclear arsenals in 1992 (United States, Russia, Ukraine, Khazakstan, Great Britain, France, China, and India), a number of countries had nuclear weapons or were developing them (South Africa, Israel, Pakistan, Iraq, and possibly North Korea, Iran, and Brazil). Most industrialized European nations are capable of developing such weapons if they choose to do so. Furthermore, the potential for terrorists to steal nuclear weapons or to make primitive devices also exists. Mental health workers must plan for tactical and strategic nuclear exchanges.[6] Strategic nuclear exchanges will be addressed in terms of disaster planning. This chapter will address the psychiatric aspects of tactical nuclear weapons and nonnuclear radiation threats after first discussing chemical and biological warfare which appear more likely to exist on the battlefield than nuclear warfare.

CHEMICAL WARFARE

Ancient artwork and documents reveal the use of chemical agents. Three-thousand-year-old Assyrian bas reliefs depict the use of liquid fire. Irritant or toxic smoke or fumes were used at the sieges of Syracuse (413 BC) and Rhodes (304 BC). Such agents were used for both their asphyxiating and incendiary effects but were not very effective.[7]

At the Hague Congress in 1899, all countries represented, except the United States, pledged to refrain from using suffocating or poisonous gases. The U.S. delegate felt that it was illogical to say that gas was inhumane while bombs, bullets, and other methods of warfare were more humane.[7]

Just 15 years later, the modern use of poison gas began in earnest during World War I. In August 1914, the French used tear gas against German troops. On the pretext that this was chemical warfare, in April 1915, the Germans attacked French troops by releasing 180 tons of chlorine in a cloud north of Ypres. This attack was devastating. In December 1915, the Germans introduced phosgene and in July 1917, mustard gas. Other gases were tried, including arsenic compounds and cyanide, but with limited success. Mustard gas was the most successful. During World War I, chlorine and mustard gas killed or injured more than a million soldiers and civilians.

Although the U.S. Army entered late in the war, 31% of all American battle casualties were due to gas.[7] Russian gas casualties amounted to half a million, of which 10% were fatalities. The Russian Army suffered twice the number of casualties and 5-fold the number of deaths secondary to gas than any other combatant.[8]

The forerunners of modern chemical nerve agents were developed by German chemists during the 1930s as a by-product of insecticide research. By the end of World War II, about 12,000 tons of Tabun (Ga) and small amounts of Sarin (Gb) and Soman (Gd) were synthesized, most of which fell into Soviet hands. As mentioned, the Germans did not use these weapons because they

EXHIBIT 4-1

CHRONOLOGY OF CHEMICAL AND BIOLOGICAL WARFARE ALLEGATIONS

1899: Hague Council outlawed use of chemical warfare (CW)

1915–1918: World War I—Both sides used chemical agents extensively

1919–1921: Russian Civil War—Both sides perhaps, Whites definitely, used CW

Early 1920s: British forces in Middle East—Both sides allegedly used CW

Mid 1920s: Morocco—Spanish used mustard gas in 1925; French allegedly used it

Early 1930s: China—Governor of Manchuria used CW against insurgents

1935–1936: Ethiopia—15,000 of total 50,000 Ethiopian casualties were from CW agents used by Italy

1936: Spain—Probably only tear gas used by Fascists

1937–1945: China—CW used by Japanese but probably irritants initially, mustard later; Japanese experimented with bacteria on human prisoners

1939–1945: World War II—Poles in 1939 used mustard gas defensively; Germans used chemical and bacterial agents in crimes against civilians in concentration camps; Japanese used cyanide hand grenades on U.S. forces in Pacific; U.S. forces had mustard gas available but the only casualties from its use were Americans accidentally exposed.

1945–1949: China—Civil War—Alleged use of CW by Nationalists

1947: Indochina—Alleged use of CW by French

1949: Greece—Civil War—Sulfur dioxide allegedly used by government (first military use of sulfur dioxide was in the same area 2300 years earlier when the Peloponnesians besieged Platea)

1951–1952: Korea—United States allegedly used CW and biological warfare, admits only to tear gas

1957: Cuba—Castro forces allegedly used mustard gas

1958: Rio de Oro—French and Spanish allegedly used CW in Sharan, Morocco, area

1958: China—Nationalists allegedly used CW from Quemoy against the mainland

1963-1967: Yemen—Egyptian forces allegedly used CW (multiple gases) in Yemeni Civil War

1965–1971: United States used tear gas in Vietnam; others were alleged

1968: Guinea and Bissau—Portugal allegedly used gas

1969: Palestine—Palestine Liberation Organization accused Israel of using gas warfare

1970: Angola—Portugal was accused of gas warfare

1970: Rhodesia (now Zimbabwe)—Government was accused of poisoning a rebel water supply

1980s: Afghanistan—Soviets allegedly used CW, particularly mycotoxins, especially early in the war

Early 1980s: Cambodia and Laos—Vietnam allegedly used mycotoxins in "yellow rain"

1980s: Iraq used several chemicals, including mustard gas and nerve agents, against Iranian ground troops. In 1988, hundreds of Kurdish civilians and Iranian soldiers were killed by Iraqi mustard and cyanide gas in territory captured by Iran; Iran allegedly retaliated with CW

1991: Iraq had chemical agents but did not use them during the U.N. attack on Iraq. Iraq was close to developing a nuclear capability.

Data sources: [1899–1970] Stockholm International Peace Research Institute. *The Problem of Chemical and Biological Warfare. Vol 1: The Rise of CB Weapons*. New York: Humanities Press; 1971: 1–5, 141–212, 212–230. [1980–1991] Author research.

mistakenly believed that the Allies also possessed them.

Many armies expect that chemical weapons will be used in any major conflict. Modern military doctrine assumes that combat operations will continue in the presence of toxic chemicals. As of 1990, training in the use of protective masks and clothing started at the grade school level in schools in the former Soviet Union and continued through adolescence and into the military service. Soviet troops were routinely trained to fight while wearing protective masks and clothing, and chemical defense personnel were integral to Soviet fighting units down to the company level.[9] Training of Soviet troops included exposure in protective gear to active diluted nerve agents. Psychological conditioning to fighting in a chemical environment was also heavily stressed.[8] Soviet chemical weapons were believed to consist predominantly of the nerve agents (particularly Soman), cyanide and mustard,[10,11] and possibly mycotoxins. With the demise of the Soviet Union and Warsaw Pact, the threat of major warfare and use of NBC warfare is lessened; however, terrorist use of such weapons remains a real threat. This is exemplified by the March 1995 use of Sarin in the Tokyo subway system terrorist attack, reportedly by the religious cult Aum Supreme Truth. The attack resulted in 10 deaths and over 1,000 injuries.[12]

Iraq used mustard and nerve agent[13] in its war against Iran after the tide of the war had turned in Iran's favor. Iraqi Republican Guard units, which had been trained to fight on the contaminated battlefield, enabled Iraq to win the limited counteroffensive which finally brought Iran to accept a grudging cease-fire. The threat of Iraqi chemical weapons was taken seriously during the 1991 Persian Gulf War. Many troops were given prophylactic pyridostigmine as pretreatment for nerve agent exposure. The U.S. units were frequently in mission-oriented protective posture (MOPP) gear. No cases of Iraqi chemical weapons' use were documented, although there were numerous chemical alarms sounded.

Since the Persian Gulf War, the media have reported that several thousand veterans have complained of miscellaneous symptoms including fatigue, trouble concentrating and remembering, pares-thesias, hair loss, joint pains, skin rashes, respiratory and gastrointestinal problems, and caustic (irritating) semen.[14] Many show severe emotional distress as they testify before Congress or the news media that similar symptoms are affecting their spouses and children (who they also fear are subject to birth defects). Many of the sufferers (and one congressman) attribute this "Persian Gulf Syndrome" (which is perhaps more appropriately labeled "Possible Persian Gulf Illnesses") on either trace quantities of Iraqi chemical or biological agents, or on the U.S. pretreatment drug pyridostigmine or the vaccinations against anthrax. Another suspect is radiation from the depleted uranium used as antitank ammunition or additional armor for tanks. The difficulty of confirming or excluding any of these hypotheses illustrates the high ambiguity of the NBC threat and the severe stress that ambiguity causes.

Physiological Effects of Nerve Agents

The "nerve agents" are derived from organophosphorus compounds related to commercially available insecticides such as parathion and diazinon. They are extremely toxic. For example, 0.8 mg of Soman or 0.4 mg of agent VX can be lethal. Other nerve agents include Sarin and Tabun, which can penetrate ordinary clothes with ease, making special suits necessary for protection. Nerve agents in the liquid state can penetrate unbroken skin, and one or two droplets on the skin can be fatal if not removed immediately.[15] The addition of thickening compounds to these agents can increase persistence, resulting in a contact hazard that may last for weeks.[11]

Nerve agents are irreversible inhibitors of acetylcholinesterase, an enzyme that is present throughout the central nervous system, the skeletal muscles, numerous glands, and other cholinergically innervated organs. Poisoning with these agents leads to an inability to break down acetylcholine. An excess of acetylcholine in the synapses results, leading to a functional denervation state or subsensitivity of the post-synaptic receptor in response to overwhelming stimulation.[16] The resulting symptoms of cholinergic overstimulation include lacrimation, salivation, nausea, hyperpnea, rhinorrhea, bronchoconstriction, vomiting, muscle twitching, progressive respiratory paralysis, and death. The usual cause of death is respiratory paralysis, which may be central in origin.[17] Autopsy studies of animals who survived exposure to nerve agents revealed extensive damage to limbic neurons.[18] This suggests that human survivors of nerve agent poisoning may suffer serious, permanent personality changes.

The detailed treatment of acute poisoning with nerve agents is beyond the scope of this book; however, the mainstay of treatment is atropine. Cur-

rently U.S. soldiers are provided with three autoinjectors, each with 2 mg of atropine, for self-administration in the field. As further therapy, atropine is administered until significant side effects appear. Atropinization is usually maintained for at least 24 to 48 hours. As much as 10 to 40 mg of atropine may be necessary in the first 24 hours.[19,20] Some studies[21] indicate that scopolamine, which apparently enters the brain more readily than atropine, may be more effective than atropine in treating the central nervous system effects of nerve agent poisoning.

Treatment protocols[20,22–24] and military manuals[25–28] all call for treatment with pralidoxime (Protopam or 2-PAM chloride), which, by removing the bound agent from the enzyme, reactivates the enzyme.[23,24] While oximes are effective antagonists to many cholinesterase inhibitors, they also produce side effects. Furthermore, some nerve agents are refractory to currently available oximes; this is particularly true of Soman, the predominant nerve agent in the former Soviet Union arsenal.[29] The current standard oxime of the U.S. Army, pralidoxime chloride, is administered by the slow intravenous route along with atropine in the Mark I autoinjector.

Plans now include pretreatment with pyridostigmine 30 mg every 8 hours prior to anticipated exposure. This does not prevent symptoms but greatly increases the efficacy of atropine and pralidoxime. Pyridostigmine is itself a "nerve agent" which temporarily protects some acetylcholinesterase enzyme from deactivation by the enemy's nerve agents. Diazepam 10 mg is also issued as an anticonvulsant to be administered with the third autoinjector or if the casualty is convulsing, with the goal of decreasing the chances of lasting brain damage. Genetic engineering may allow the development of a more effective antidote in the future. This is suggested by the discovery of an enzyme in squid nerves that hydrolyzes and detoxifies Soman.[30]

If a nerve agent is used, many soldiers will receive only low doses by virtue of location or protective measures; this low-level poisoning may be accepted as a calculated risk or may even go undetected. If a nerve agent is used, many more soldiers will receive only very low doses than will receive high doses, by virtue of their location or protective measures. Some low-dose exposure may be accepted as a calculated risk. The first symptom of minimal exposure of the eyes to nerve agent vapor (at 1/30th to 1/50th of the lethal dose) is pinpoint pupils.[25] This does not grossly impair vision in bright light, but causes dimming of vision and may impair performance in the low levels of artificial illumination inside vehicles or tents (tactical operations centers) and/or at the critical times of predawn and dusk. Soldiers with pinpoint pupils would be seriously impaired and at a dangerous disadvantage in night operations under blackout conditions. At slightly higher doses, the soldiers also have eye discomfort on focusing, blurred vision, headaches, jitteriness, and runny noses to further distract them.[25] These symptoms could involve all members of a squad, platoon, or company, to greater or lesser degree. The pinpoint pupils remain marked for 24 hours, before gradually improving over several days. The soldiers may well be fearful of further NBC or conventional attack, unwilling to venture out after dark, and prone to overreact to threats until adequate vision returns.

Experience with human exposure to the chemically related organophosphate insecticides suggests that, in cases of *chronic* exposure to low concentrations, psychiatric symptoms may predominate over physical ones. Impaired thinking, judgment, attention, and short-term memory are likely, but toxic delirium may also be present. The cognitive deficits may persist many months, even after treatment, along with irritability, mood changes, depression, and insomnia with nightmares. Acetylcholinesterase inhibitors have been found to precipitate psychotic symptoms in predisposed individuals.[31]

Behavioral manifestations of nerve agent poisoning that antedate, follow, or occur independently of somatic symptoms[19] may be most prominent in individuals who have been exposed to sublethal doses or in those who have recovered from the somatic effects of poisoning. Acute organophosphorus intoxication produces cognitive impairment with difficulty in concentration, confusion, and drowsiness.[31,32] Airplane crashes of crop-duster pilots may be due to acute intoxication with organophosphorus compounds.[33] Chronically-exposed agricultural workers have complained of forgetfulness, difficulty in thinking, visual impairment, and drowsiness. These deficits were quantified by sophisticated testing.[34] Another study[32] showed that chronically-exposed workers had higher levels of anxiety than matched controls. Chronic exposure has also been associated with increased anxiety, possibly causing misdiagnosis of combat stress reactions.[35] Memory also appears to be impaired by organophosphate poisoning.[20] Significant impairment of cognition, vigilance, and memory may make it difficult for a minimally-exposed soldier to perform the often complex tasks that will be required.

Poisoning with nerve agents may cause other psychiatric disturbances that mimic psychological as opposed to organic disorders. Depression has been observed as a prominent symptom in accidental poisoning.[22,31,36] The severity of depression seems to be related to the severity of poisoning and the degree of acetylcholinesterase inhibition. This depressogenic effect of nerve agents is suggested by the observation that the reversible cholinesterase inhibitor, physostigmine, can normalize mood in manic patients and can cause depression in normal persons.[37,38] Could the same be true for the pretreatment drug, pyridostigmine, in some soldiers? Nerve agent toxicity has also been associated with complaints of "excessive" dreaming, nightmares, insomnia, and delirium.[22,32] Treatment with anticholinergic agents seems to reduce these symptoms.[22] Nerve agents may also lower the threshold for convulsive seizures. These could be mimicked by pseudoseizures in tense, anxious persons, creating a problem for differential diagnosis.

It is possible that nerve agents can cause psychosis, by altering the dopaminergic-cholinergic balance in mesolimbic structures. Anticholinesterases reportedly activate symptoms in schizophrenic patients.[36] One study[39] reported that a higher percentage of agricultural workers exposed to toxic agents developed psychotic illnesses than would have been expected. These results have not been confirmed, however.

In summary, the data on the subacute and chronic physiological effects of exposure to organophosphorus poisons reveal substantial risk of depression and sleep disturbances, decreased cognitive abilities, and a slight risk of psychosis and anxiety disorders.[40]

Added to the dangers of nerve agent poisoning are effects of antidotes, which may outweigh the danger of potential exposure to nerve agents. Self-injection of atropine by the soldier after exposure to nerve agents may be lifesaving, but inappropriate self-injection may lead to a central anticholinergic syndrome with delirium. If a false alarm (or a deliberate attempt to make oneself a casualty) leads to the administration of chemical warfare antidotes in the absence of the appropriate agent, serious psychological symptoms are likely. Atropine in low doses produces blurred vision, tachycardia, dry mouth, sweat suppression with increased risk of heat stroke, urinary retention, and perhaps impaired thinking, judgment, insight, and short-term memory. Even in the 2 to 6 mg range available for early self-administration, atropine may give some individuals a toxic anticholinergic delirium with disorientation, agitation or stupor, paranoid delusions, and visual and tactile hallucinations lasting 8 to 16 hours. TAB (TMB-4 [an oxime], atropine, and benactyzine), an antidote combination formerly used by the U.S. Air Force and U.S. Navy / Marine Corps, was virtually guaranteed to produce psychosis with visual hallucinations in its standard dose.[30] While the presence of anticholinesterase nerve agents in the body may partially counteract such psychiatric side effects, it is unlikely that agent and antidote will exactly cancel each other.

Studies conducted at the U.S. Army Aeromedical Research Laboratory demonstrated that experienced helicopter pilots can fly helicopters, although seriously impaired, after receiving 4 mg of atropine. "Effects were seen most often … in terms of aircraft control problems, vision disturbances, impaired tracking, reduced cortical activation, and decreased cognitive skills. These problems indicate helicopter tactical flight is dangerous after an unchallenged 4 mg dose. Other types of flight should also be avoided for at least 12 hours after atropine."[41(p.857)]

Following the administration of large doses of atropine, symptoms progress from tachycardia and dryness of the mouth to ataxia, hallucinosis, and confusion. In a study performed at Edgewood Arsenal,[42] subjects administered large doses of atropine became unable to pay attention, carry out instructions, speak coherently, or perform calculations. Significantly, soldiers who had impaired judgment failed to recognize their degree of general impairment and resented assistance. In this study, chlorpromazine, strongly anticholinergic itself, potentiated the adverse mental effects of anticholinergic agents. This takes on added significance in view of the widespread use of phenothiazines to treat anxiety symptoms in Vietnam.[43,44]

Treatment of atropine-type psychoses should *not* include phenothiazines, which produce anticholinergic side effects of their own, although haloperidol may be used. Current definitive treatment is with the carbamate, physostigmine, but this requires careful and prolonged intravenous titration.

The effects of atropine resemble those of another class of chemical agents, the incapacitants. Agent BZ (3-quinuclidinyl Bensylate) is a strong antimuscarinic compound that for some time was kept in the arsenal of U.S. chemical weapons. It produces hallucinations and psychological incapacitation similar to that produced by atropine, but is more specific and longer lasting (24–48 h) than atropine.

The burden of self-diagnosis is formidable, particularly considering the fact that other agents or even anxiety may mimic early symptoms of nerve agent poisoning. Under battle conditions, with

oncoming shells producing smoke or spreading tear gas, with troops dispersed and communications strained, the apprehension and ambiguities may unavoidably lead to individual or group decisions to administer antidotes when exposure to nerve agents has not, in fact, occurred.[45]

Whether due to mistaken self-administration of antidotes or exposure to antimuscarinic agents, the possibility of significant numbers of casualties with anticholinergic poisoning must be considered. One example of troops thus exposed was in 1676, when British troops in Jamestown, Virginia, ingested Jimson ("Jamestown") Weed (*Datura stramonium*) and suffered mass anticholinergic poisoning.[46] Bizarre behavior and amnesia allegedly lasted for 11 days. The soldiers, who had to be confined to prevent them from injuring themselves, were very negligent of personal hygiene. More recently, combativeness or hyperactivity was noted in 10% of a series of 212 cases of stramonium intoxication.[47] Hallucinations occurred in 99 of the 212 cases, and 5 deaths were directly attributable to psychosis.

The failure to maintain adequate self-care behavior such as keeping dry, maintaining adequate hydration, and attending to personal hygiene has been noted as a cause of soldier ineffectiveness.[48,49] Reported effects of high doses of anticholinergic agents on soldiers include impaired performance in timing and vigilance tasks, in firing rifles or running an obstacle course; ataxia; blurred vision; inability to perform calculations; and disruption of communication between individuals.[42] Possible effects in the field might include failure to observe discipline, failure to conserve drinking water, failure to wear protective clothing, misinterpretation of visual or auditory signals, misidentification of individuals, failure to maintain silence, and increased risk of heat stroke. Anticholinergic syndromes can be reversed with physostigmine,[50] but this drug is not easy to use in the field. There are storage problems and it requires careful titration in use. Furthermore, as mentioned earlier, physostigmine can produce serious depression in normal persons, and the same problem of inappropriate use described with atropine exists.

In summary, nerve agents present numerous problems in practical therapeutics for the psychiatrist. Both the agents themselves and their antidotes may have significant behavioral effects, and the military psychiatrist may be called upon to treat, and to advise other physicians on how to treat, these problems.

Physiological Effects of Other Agents

Cyanide

Other chemical agents also may be encountered. Cyanide gas, a nonpersistent agent, is especially dangerous because it may saturate the active material in gas mask filters and render them useless. A combination of cyanide and a nerve agent would be particularly deadly. The early symptoms of cyanide exposure are anxiety, confusion, giddiness, and hyperventilation. These symptoms can also be caused by stress, and could lead to unnecessary medication with cyanide antidotes. In civilian situations, antidotes to cyanide have often been administered inappropriately.[51,52] Chronic exposure to low levels of cyanide could lead to mental changes as was seen when cyanide compounds were used therapeutically.[53] The effects of administration of large doses of anticholinergic agents to soldiers who have been poisoned with cyanide is unknown. Atropinization may conceal the symptoms of cyanide poisoning or may increase its lethality.

Incapacitating Agents

Tear-gas agents have been extensively used in war as most countries have interpreted the Hague and Geneva accords against gas warfare as not applying to these agents. Intended to be used as harassing agents, their lethality is very low.

Nonlethal incapacitating agents may exert their own direct effects on psychological functioning (like the anticholinergic BZ). Those reportedly used in Afghanistan appear to cause temporary unconsciousness or immobilization. Incapacitants may also provoke inappropriate responses by mimicking the early symptoms of more lethal agents. The tear gases may be confused with the lacrimation-producing nerve gases. Vomiting agents may stimulate fears of having been irradiated or exposed to biological agents. Adamsite, in this class, reportedly also may cause depression.[11]

Mustard

Mustard gas was responsible for the majority of gas casualties during World War I. It is considered an obsolete agent by the U.S. but the former Soviets were believed to have stockpiles of it,[8] and it was used by Iraq against Iran. Mustard gas is unique for its insidious method of action and its

latency of several hours before burns and blisters appear. It can present a contact hazard for weeks. Sulfur mustard (bis[2-chloroethyl] sulfide) is a powerful alkylating agent that can produce severe skin burns and pulmonary injury if inhaled. Very low doses of mustard produce painful conjunctivitis of the eyes which lasts for days to weeks, severely impairing vision.[54] As with nerve agent, blindness on the battlefield, even if known to be temporary, can produce anxiety, dependency, and other psychological as well as management problems. Higher-dose contact burns the eyes and causes permanent blindness as well as disfiguring facial burns. The potential psychological impact of these is discussed in Chapter 14, Disabling and Disfiguring Injuries. Blistering of exposed hands could also leave long-term physical and emotional scars. In soldiers without protective overgarments, mustard tends to produce blisters at the moist creases of the body, notably the genital region. Psychological reactions to genital injuries are also discussed in Chapter 14.

Blister agents (mustard gas, Lewisite) have been noted to produce chronic psychological symptoms of apathy and depression[55] in addition to their severe and delayed dermatologic, pulmonary, and systemic lesions. Phosgene, a suffocant gas, keeps active the traditional World War I terror of gas attack as producing a horrible sensation of the lungs filling with fluid, with the added threat that it permanently inactivates the charcoal in one's mask or overgarment. Phosgene's delay of several hours before the first symptoms, and the dangerous worsening of symptoms by physical exercise, was especially unnerving, even to those not actually exposed.[56]

Mycotoxins

The mycotoxins (thought to have been used by the Soviets and their allies in Southeast Asia and Afghanistan) produce terrifyingly rapid symptoms of vomiting, tissue necrosis, and failure of blood coagulation. Psychotic symptoms and bizarre behavior have not been mentioned prominently in current accounts; however, such psychiatric symptoms have been prominent in historical natural outbreaks of "St. Vitus' Dance" and "tarantism," which are now attributed to contamination of food grains by ergot derivatives from other fungal toxins. Mental symptoms may also become significant if mycotoxin patients are kept alive by treatment of the otherwise rapidly fatal symptoms.

Neuropsychiatric Syndromes Associated With Chemical Warfare

Large-scale gas warfare during World War I presented special problems. In addition to the "physical" casualties of chemical warfare, there were "psychological" casualties and syndromes. One of these was "gas hysteria," which usually occurred in small epidemics, threatening the integrity of entire units until remedial measures were taken.[57,58] This "epidemic" effect is seen in the following World War I episode:

> One morning a large number of soldiers were returned to the field hospital diagnosed as gas casualties. The influx continued for about eight days and the number of patients reached about 500. The divisional gas officer failed to find any clinical evidence of gas inhalation or burning.... Most of the patients had the fixed conviction that they had been gassed and would usually describe all the details with convincing earnestness and generally with some dramatic quality of expression.... It was obvious on examination that they were not really gassed. Further, it was inconceivable that they should be malingerers.[57(pp318–319)]

Acute symptoms of gas hysteria often mimicked some of the symptoms of gas poisoning and included dyspnea, coughing, and burning of the skin. Aphonia was also seen prominently.[59] For those in whom the symptoms persisted, the term "gas neurosis" was applied.[60] The degree of exact exposure to gas was unrelated to the symptoms presented. Dramatic symptoms were seen, such as tics and blepharospasm,[59] as well as "unconscious movements of the hands like clawing at the throat or removing a mask."[60(p214)] The patients presented with signs of air hunger and anxiety with alterations of consciousness, but no organic basis for the symptoms was found. One author suggested that gas neurosis patients "are particularly amenable to suggestion and rest. Optimism based on exact diagnosis and sincere faith in ultimate recovery is excellent therapy."[60(p214)] However, some of the gas neuroses continued until long after the war and were rewarded with compensation.[57] It should be remembered that during World War I there were approximately twice as many gas neurosis cases as there were gas exposure cases.[57]

Chemical warfare is still perceived as choking off one's breath. In spite of the complicated chemical properties of modern agents, respiratory symptoms are likely to predominate in conversion reactions.

This is reinforced by current military training requiring wearing protective clothing while tear gas agents are used to simulate poison gases.

More recently, similar conversion symptoms have been reported in the aftermath of chemical attacks in Afghanistan and Cambodia,[8] with aphonia and catatonic symptoms predominating. More generally, the mere act of donning protective gear will increase the soldier's sense of isolation from peers and decrease intragroup communication and support, factors that have been shown to be important in maintaining morale and probably in decreasing the incidence of psychiatric casualties.[61]

Gas warfare is perceived much as it was in 1918, as "awe inspiring" with "unbelievable horrors."[60] During World War I, units that were otherwise stressed, having spent long periods in combat under arduous conditions, were at much higher risk for "gas hysteria." This risk increased still further if relief was anticipated but did not arrive on schedule.[57] This aspect will loom large in any future mid- or high-intensity conflict, with the extremely high-pressure, continuous combat operations that are expected.[48,61]

As long as the nuclear, biological, and chemical (NBC) threat is an active, serious threat, protective masks and overgarments must be worn, certainly for brief periods and perhaps for prolonged ones. The protective gear distorts visual, auditory, and tactile perception; impairs fine motor coordination; increases physical effort, frustration, and fatigue; and increases psychiatric casualties in simulation exercises.[62,63] These effects may be especially disruptive to the performance of medical functions; however, modifications of protective gear for medical treatment[64,65] as well as approaches to triage have been proposed.[66] The protective ensembles retain body heat and increase the risk of heat exhaustion and heat stroke, which may present with mental symptoms and be difficult to diagnose in full MOPP (mission-oriented protective posture) gear. During a Scud missile attack by Iraq on Israel in January 1991, several elderly persons died of heart attacks while in gas masks.

The restrictions on eating, drinking, elimination, and hygiene functions, along with other discomforts and interference with sleep, can affect morale as well as physiological well-being. Decreased ability to recognize comrades and gauge their mood or resolve may lessen unit cohesion and increase the sense of helpless isolation (a prime factor in battle fatigue). Claustrophobic panic, premature unmasking (which may be imitated by others), spatial disorientation, and paranoid reactions to impaired sensory functioning may also occur. Such adverse reactions are exacerbated when visibility is further restricted by darkness, smoke, or vegetation. Gas mask phobia proved to be a significant problem in the Allied invasion of Iraq in 1991.[67]

Field studies and training exercises which test troops in force-on-force battles using the MILES (multiple integrated laser engagement system) to score who "shoots" whom have shown an alarming increase in friendly fire casualties ("fratricide") by insufficiently trained troops in the protective ensemble. Whereas only about 1 in 20 soldiers or vehicles is "shot" by their own side in conventional battles, the rate rises as high as 1 in 5 in full mission-oriented protective postures.[68,69] This is attributable to the combination of impaired vision and hearing plus the jumpiness this provokes. Fratricide, of course, is very disruptive of morale. Rigorous training is needed to reduce the risk.

Factors that predispose to psychiatric casualties include the rates of wounding in the unit, lack of sleep, and lack of prior combat experience.[70–72] Chemical attacks might increase psychiatric casualties by exacerbating all three of these factors.[45,73] Chemical weapons create fear of the unknown, a potent effect in inexperienced troops. Usually, troops entering combat will be, for the most part, untested in battle. Lack of sleep has produced hallucinations in troops during extended training exercises[74–76] and is a factor predisposing to combat psychiatric reactions.

With the possibility of high-intensity, continuous combat, psychiatric services will be strained to the limit. Because this country is committed against the initial use of chemical weapons, U.S. forces would be the first to suffer from chemical attacks, with the concomitant psychiatric casualties. Lack of experience in chemical warfare and the psychiatric syndromes arising from the physiological effects of chemical agents and their antidotes may lead to misdiagnosis and mistreatment. This may lead to decreased unit effectiveness at critical times. The difficulties in decontaminating large numbers of exposed soldiers may lead to removing them to centralized decontamination stations well to the rear. Many soldiers who might be medically fit to return to their units might develop an evacuation syndrome that would increase their resistance to returning to combat. In the 1982 Israeli incursion into Lebanon, some Israeli combat stress casualties

were evacuated by air to Israel, while others were appropriately treated in forward areas. Almost all of the forward-treated cases, but few of the rear-evacuated cases, returned to combat.[77]

In summary, the psychological casualties of chemical warfare may well outnumber and prove more costly in personnel losses than the physical casualties, as occurred in World War I.[78] However, it should be noted that most veteran soldiers in World War I adapted to the threats and discomforts of chemical warfare. They often "preferred" this risk of chemical attack to those of high explosive shells and machine gun bullets because the protective equipment and good training allowed them to improve their odds of survival better.[79]

BIOLOGICAL WARFARE

Reports of biological warfare go back many centuries. One example is the introduction of smallpox to the American Indians by early settlers who gave or sold them infected blankets or trinkets.[80] However, in modern times the use of biological warfare agents appears to have been rare.

The United States did not have an offensive or defensive biological warfare capability until toward the end of World War II.[80] The nascent program focused on anthrax and botulinus toxins primarily. An anthrax plant received authorization to produce a million bombs and progress was being made in short-range dispersal techniques for botulin in paste form. Figure 4-1 shows a probable descendent of the World War II anthrax dispersal bomb. Moreover, there is evidence that research was conducted with brucellosis, psittacosis, tularemia, and the respiratory disease, glanders. In addition, botanical toxins and viruses were being explored with the aim of destroying Japanese vegetable gardens and rice crops. Tactical, rather than moral, considerations prevented the use of defoliants in World War II. They were later used in Vietnam, in the form of Agent Orange.

Rumors that a 1979 mishap with anthrax in the former Soviet Union produced scores of casualties at Sverdlovsk have been replaced by verified accounts that such a mishap actually occurred.[81] Iraq was suspected of working towards developing anthrax as a weapon. That led to the decision of the United States to vaccinate many personnel in the Persian Gulf campaign with an experimental anthrax vaccine. Many of the same considerations of chemical warfare apply to biological warfare; however, a major difference is the self-perpetuating effect of live biological agents. This can produce fear of contagion, which may severely impair relationships among troops and interfere with proper care of casualties. The near-panic responses of some persons in casual contact with victims of the practically noncontagious (except through sexual contact and shared needles) acquired immunodeficiency syndrome (AIDS) illustrate the validity of this factor.

Physiological Effects of Biological Agents

A number of viral, bacterial, and rickettsial agents have been identified as potential weapons, both for their psychological effects and the terror those effects produce in exposed troops. Anticipated psychiatric casualties of biological warfare will be included in a general discussion. It is, however, possible that an enemy could develop neurotropic viral agents that could produce primarily psychiatric symptoms.[82] Tables 4-1, 4-2, and 4-3 show agents considered by Malek[80] to have a biological warfare potential.

Neuropsychiatric Casualties of Biological Agents

Contagious biological organisms, like persistent transmissible chemical agents, would require quarantine and special handling. This would surely have widespread psychological effects on buddy-care and attitudes toward strangers, stragglers, and refugees. Within the medical system, the impact of invisible, patient-borne threats to other patients and to the treating personnel would reintroduce an old but now rarely encountered psychological as well as occupational stress, which may be difficult to cope with under field conditions. In World War I, entire medical/surgical teams were temporarily incapacitated by eye damage from mustard vapor brought into the operating room on wounded patients who had not yet shown any effects of their own exposure.

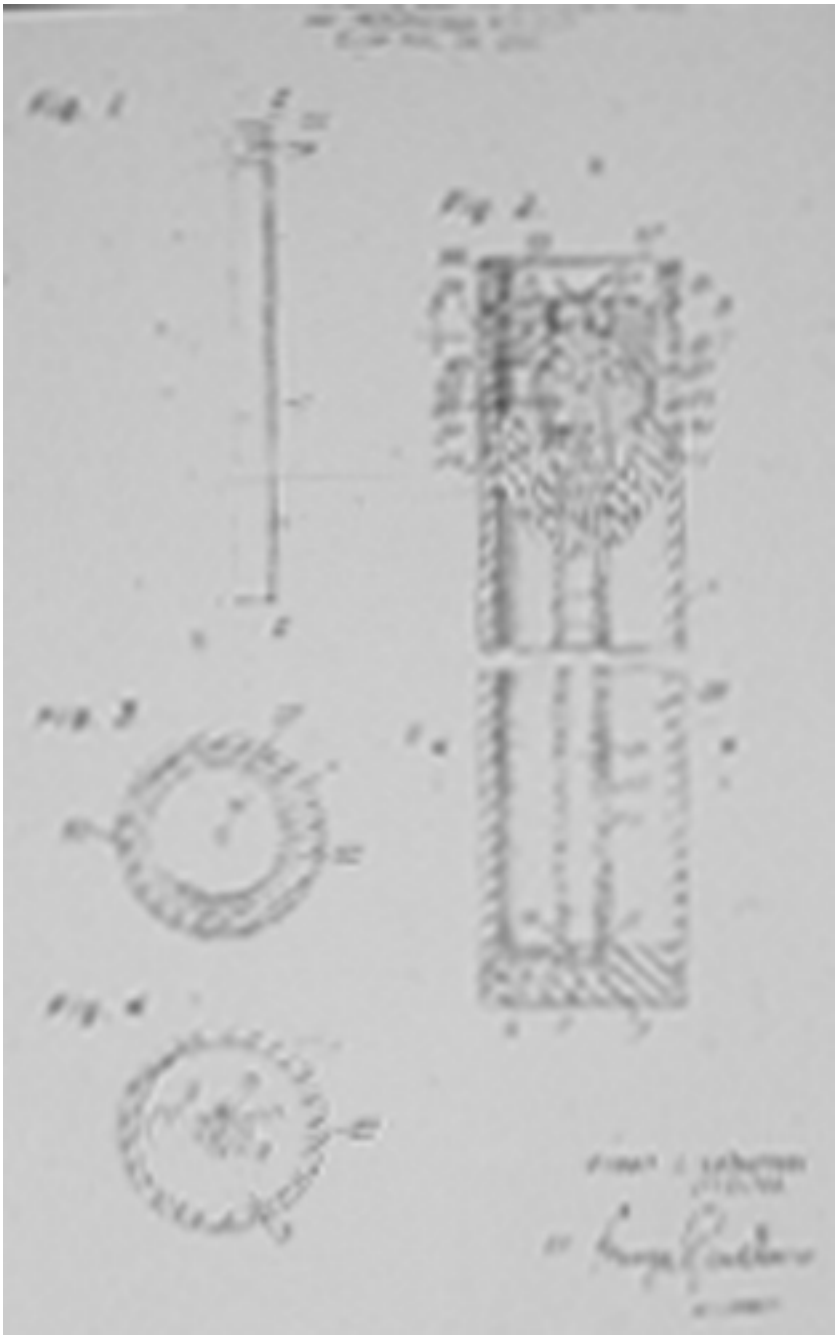

Fig. 4-1. Patent application of R. L. Le Tourneau for a light high-explosive bomb for dispersing toxic and insecticidal aerosols, 1955. Le Tourneau's patent application reveals the technological ingenuity he employed in continuing the production of gas warfare mechanisms. This technological interest continued for the next several decades with the development of safer binary weapons (ie, chemicals were inactive until combined at the time of actual use). Photograph: U.S. Patent and Trademark Office, Arlington, Va.

TABLE 4-1

POSSIBLE BIOLOGICAL WARFARE AGENTS—BACTERIAL

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TABLE 4-2

POSSIBLE BIOLOGICAL WARFARE AGENTS—VIRAL

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TABLE 4-3

POSSIBLE BIOLOGICAL WARFARE AGENTS—RICKETTSIAL, FUNGAL, TOXIC

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NUCLEAR WARFARE AND DISASTERS

In some countries the use of nuclear weapons is a practical rather than a moral issue. An attack could be launched by tactical missile forces and fighter bombers.[73] There is concern that portable tactical nuclear weapons from the stockpiles of the former Soviet Union or from unstable states might fall into the hands of terrorists who could use them against cities. Industrial disasters like that at Chernobyl could release nuclear radiation to contaminate large areas and require military intervention to minimize the damage, evacuate the population, and maintain order.

Physical Effects of Nuclear Warfare

The energy generated by a typical tactical nuclear explosion consists of blast and shock waves (45% of the energy produced), light and heat (35%), initial radiation (5%), and residual fallout radiation (about 15%).[83] A ground detonation, vaporizing soil and melting granite and clay, results in large amounts of debris drawn by vacuum into the fireball, where it is mixed with radioactive bomb debris and subsequently falls to earth to contaminate it (fallout). While an air detonation produces the greatest blast damage, few particles are drawn into the atmosphere to mix with radioactive bomb debris so there is little residual contamination from fallout. Nuclear explosions over water produce massive vaporization and an intensely radioactive rainfall as well as a 50- to 100-foot tidal wave capable of drowning a coastal city.

Electromagnetic Pulse Effects of Nuclear Warfare

A high altitude electromagnetic pulse (EMP) is a radiated electromagnetic wave caused by the detonation of a nuclear weapon above the earth's atmosphere. A 3.3 megaton nuclear weapon, detonated 400 km above the center of the United States, can produce sufficient electromagnetic radiation to cover the entire country; and, if detonated over Europe, most of the continent would be affected.[84] Vandre, et al,[84] in simulated EMP tests, showed that by creating power surges in standard field medical equipment, an EMP would render about 65% of

such equipment useless. The more modern and integrated the equipment, the greater the vulnerability to an EMP power surge.

An American Medical Association (AMA) report of the Board of Trustees indicated that civilian medical facilities would be equally devastated.[85] The AMA noted that in addition to medical equipment, telephone and other telecommunication equipment, computers and electronic equipment involved in life support systems, and diagnostic testing and other equipment utilizing solid state components are particularly vulnerable.

In a related paper, Vandre, et al,[86] described methods to minimize the vulnerability of equipment to EMP effects by keeping wiring near the ground, keeping it short, unplugging unused power equipment, running power cabling and tents in a north-south direction (thereby avoiding running power cabling in an east-west direction), and placing sensitive equipment in standardized shelters. By following such procedures Vandre, et al[86] estimated that 88% or more of unplugged field medical equipment could be kept functional in the event of EMP. The AMA noted that the increasing use of fiber optic components would decrease the effects of EMP because they are not vulnerable to the EMP surge. They recommended use of standby diesel generators to provide emergency power.[85]

Physiological Casualties of Nuclear Warfare

Studies of the effects of nuclear weapons on humans, with the exception of a few accidental radiation victims (particularly in Chernobyl), focus on the effects of the Hiroshima and Nagasaki bombings. A useful bibliography has been prepared by the Atomic Bomb Casualty Commission.[87]

Initial physical casualties are due to the blast and shock, heat, and nuclear radiation. Later, casualties are primarily from nuclear radiation. The initial effects are most closely related to the proximity and size of the fireball. At Hiroshima and Nagasaki more than 80% of the population within 0.6 of a mile of ground zero were casualties, and over 90% of these casualties were killed. In contrast, of individuals who were beyond approximately 1.6 miles from ground zero, less than 5% were killed.[88] These findings suggest that troop dispersal will be the primary defensive strategy in nuclear war. Burns accounted for two thirds of the initial deaths at Hiroshima and for one half of the total deaths. About 30% of those who died in Hiroshima had received lethal doses of radiation; however, this was not always the immediate cause of death.

There are two major biological effects of radiation in excess of 100 rad (a measure of the dose absorbed from ionizing radiation equal to 100 ergs per gram): (1) cell membrane damage leading to cellular and vascular leakage affecting especially brain and lungs, and (2) loss of reproductive capacity in stem cells.[87] Different tissues of the body show different sensitivities to ionizing radiation. In general, the radiosensitive cells are found in lymphoid tissue, bone marrow, spleen, organs of reproduction, and gastrointestinal tract. Of intermediate sensitivity are skin, lungs, and liver. Muscle, nerve, and adult bones are least sensitive.[88]

The medical manifestations of radiation damage can be conveniently divided into three time phases: initial, latent, and final.[88] During the *initial phase,* exposed persons may experience nausea, vomiting, headache, dizziness, and malaise. The onset time decreases and the severity of these symptoms increases with increasing dose. During the *latent phase,* exposed persons will experience few, if any, symptoms and will be able to carry on normal functions. The *final phase* is characterized by illness that requires hospitalization of people who have received the higher doses. The symptoms experienced in the initial phase will recur and be accompanied by skin hemorrhages, diarrhea, and sometimes loss of hair. At higher doses, seizures and prostration may occur. The final phase is ended by either recovery or death. These effects are summarized in Table 4-4 and Figure 4-2.

Persistent radioactive contamination is quite similar to persistent mustard gas in many of its effects. Presumed "psychological fallout" is an unseen hazard that produces delayed illness in all bodily systems that involve rapid cellular reproduction (ie, distressing gastrointestinal symptoms, impaired wound healing, and increased susceptibility to infection and hemorrhage). Early death may occur unless sophisticated medical support is given. Survivors are at increased risk of death from cancer.

Acute irradiation from a nuclear explosion (without concurrent disabling burn or blast injury) puts the military medical-ethical dilemma of these conditions into even starker relief. In the absence of quantitative dosimetry for each exposed patient (a difficult task in itself), how are the massed casualties to be triaged into those who are expected to die ("expectant") and those who can reasonably be saved with the over-used resources that remain? What is to be done with those fatally exposed who can still function? Should they be told the prognosis? Should they be discharged "RTDTD" ("Return To Duty To Die"), perhaps to carry out high-risk delaying ac-

TABLE 4-4

SUMMARY OF POTENTIAL MILITARY EFFECTIVENESS OF RADIATION CASUALTIES

Radiation in rems	Onset of initial incapacity from vomiting, etc.	Duration of effectiveness (latency)	Later incapacity (onset to duration)	Incidence of Death (time)
0–100	None	100%	None	None
100–200	3–6 h to 1 d	1 d to 2 wk	10–14 d to 4 wk	None
200–600	½–6 h to 2 d	1–2 d to 4 wk	1–4 wk to 1–8 wk	0%–90% 2–12 wk
600–1,000	15–30 min to 2 d	2 d to 5–10 d	5–10 d to 1–4 wk	90%–100% 1–6 wk
1,000–5,000	5–30 min to 1 d	0 to 7 d	2 to 10 d	100% 2–14 d
Over 5,000	Almost immediately	None	Almost immediately	100% 1–2 d

Adapted from Glasstone S, Dolan PJ. *The Effects of Nuclear Weapons.* 3rd ed. Washington, DC: US GPO; 1977: 580–581.

tions, "kamikaze" attacks, and operations in contaminated areas? What medications should they be given for symptom relief to improve their efficiency? Could those who cannot be discharged provide the answer to the shortage of whole blood in the combat zone? Will there be a place for euthanasia on the battlefield to end suffering and preserve other lives through allocation of medical resources, as suggested by Swann?[89] Such mind-chilling questions convey some of the moral and psychological implications that the Army Medical Department (AMEDD), the line military, and the exposed individuals themselves would have to deal with.

Triage by scarce medical resources is obviously the best way to preserve lives. This is critical in an operational sense in that the medical resources should be expended in helping those who are most likely to be able to return to duty.

	Organs Affected by Ionizing Radiation				
Range	0–100 rems Subclinical range	100–1,000 rems Therapeutic range			Over 1,000 rems Lethal range	
		100–200 rems	200–600 rems	600–1,000 rems	1,000–5,000 rems	Over 5,000 rems
Role of therapy		Clinical surveillance	Therapy effective	Therapy promising	Therapy palliative	
Leading organ		Hematopoietic tissue			Gastrointestinal tract	Central nervous system
Characteristic signs	None below 50 rems	Moderate leukopenia	Severe leukopenia; hemorrhage; infections; epilation above 300 rems		Diarrhea, fever, disturbance of electrolyte balance	Convulsions, tremor, ataxia, lethargy
Critical period postexposure	——		1–6 wk		2–14 d	1–48 h

Fig. 4-2. Summary of clinical effects of acute ionizing radiation. Adapted from Glasstone S, Dolan PJ. *The Effects of Nuclear Weapons.* 3rd ed. Washington, DC: US GPO; 1977: 580-581.

Neuropsychiatric Casualties of Nuclear Warfare

Although nuclear devices have been used twice in warfare, no one has actually fought a nuclear war. Neuropsychiatric casualties of nuclear warfare, therefore, must be inferred from studies of the primarily civilian victims of the nuclear explosions at Hiroshima and Nagasaki; from the disasters in Goiânia, Brazil, and Chernobyl, Ukraine; from a small number of imperfect simulations, such as the Desert Rock I, IV, and V studies; and from extrapolations from situations of extreme stress such as that during disasters or combat. The following sections will briefly review some of these studies.

Hiroshima and Nagasaki

There have only been about 20 useful studies of the psychological reactions to nuclear explosions; many of these are autobiographical accounts. Although retrospective, one of the best reviews of the physical, social, and psychological toll of nuclear devices is that of a Japanese committee commissioned by the cities of Hiroshima and Nagasaki. This report was first published in Japan in 1979; an English translation was published in 1981.[90] This review concluded that organic forms of mental illness among survivors were not prominent.

In one study[91] of 50 survivors at Omura Hospital in Japan, psychoneurological observations were made 2 to 3 weeks after exposure, the following month, and about 3 to 4 months after exposure. Only 4 of the 50 patients were diagnosed as having mental disorder in the initial stage. In the intermediate stage, some patients, especially those suffering thermal burns, showed neurasthenia-like symptoms. In the later stage, those suffering a specific diathesis tended to develop neuroses. These were considered indirect effects of physical deterioration. Acute radiation illness was considered to be the only direct effect of the atomic bomb on the human psychoneurological system. Konuma[91] has argued that complaints of "agony," lassitude, fatigability, and other symptoms constitute a "diencephalic syndrome," which can be diagnosed even in the absence of physical examination signs or abnormal laboratory findings. The presumption is that hematologic or other direct radiation effects on nerve cell membranes have damaged the vegetative nervous system. However, the severe losses (of relatives, physical capacities, and material resources) experienced by many of the victims could readily produce a depressive syndrome with many of these characteristics, particularly in a vulnerable personality.

Psychological studies were not conducted until years or decades after the event. Furthermore, disability compensation, social ostracism, and other factors colored the survivors' remembrances. Nevertheless, a fairly common response pattern was described in the 1952 study by Kubo,[90] who studied 54 victims from 1949 to 1952. Most of them had been 1 to 3 km from the bomb hypocenter. Kubo found that most were "startled" by the initial flash and fell down and covered their eyes in an "instinctive" withdrawal from the stimulus, while some turned to face the flash.

The blast, causing widespread damage, produced a feeling of "mental blankness" lasting for a few minutes. This was followed by attempts to escape the area of destruction and an inability to make clear judgments. Many of the victims aimlessly followed fleeing crowds until eventually they reached places where they felt safe; there they were given food and shelter. Most experienced a degree of recovery by the second day, but from 1 to 3 weeks later radiation sickness began to appear. This was, to most, a totally mysterious illness with high fever; bleeding from gums, throat, nose, and uterus; loss of hair; and fatigue. This produced high levels of anxiety, particularly as apparently uninjured persons began to die from intercurrent illness or other causes. The need for early treatment to prevent irreversible damage and death has been expressed.[92]

Goiânia and Chernobyl

Although the focus here is on short-term radiation effects, recognition of the longer-term effects of increased risk for cancer, sterility, and diminished life span may play an acute role in demoralizing the combatants. The Goiânia, Brazil, tragedy in which a small town was exposed to cesium isotopes from a medical device, reveals the potential for social disruption from radiation exposure. Many of the exposed individuals were shunned by friends and neighbors. Exports of produce from Goiânia dropped precipitously.

Similar effects were reported in the Chernobyl disaster in the Ukraine. Of 148 persons subjected to radiation effects of the Chernobyl atomic energy electrical plant accident and treated at Kiev Mental Hospital from 1986 through 1990, reactive psychoses were observed in only 11 cases and this was mainly during the 2 to 4 months after the disaster when the production of stress was maximal. It appears that stress, resulting in various psychopathological conditions including, rarely, psychosis, is paramount in producing psychiatric symp-

toms, rather than biological effects of ionizing radiation on central nervous system tissue.[93]

Extrapolation Studies

William James[94] appears to have been the first psychologist to systematically document the reactions of persons to disasters in his report of responses to the 1906 San Francisco earthquake and fire. It was not, however, until the late 1940s that Tyhurst[95] did field studies of two apartment house fires, a marine fire, and a flash flood from which detailed clinical and actuarial reports could be made. He formulated reactions during three overlapping phases: (1) impact, (2) recoil, and (3) post-trauma. During impact, reactions are automatic and can be separated into three main groups. The first of these is an "effective" group of about 12% to 25% who remain "cool and collected," and who appreciate and respond appropriately to the situation. The second group is characterized as a "normal" group, because this is the largest number (three fourths of the survivors), who are stunned and bewildered. Individuals in this group show a restricted field of attention and lack of awareness of subjective feelings or emotion; however, they display the physiological concomitants of fear, and automatic or reflex behavior. The last group of 10% to 15%, the "ineffectives," display manifestly inappropriate responses: confusion, paralyzing fear, inability to move or "freezing," and "hysterical" crying or screaming. During recoil there is a gradual return to normalcy; however, excessive dependency is common during the first day or two. The post-trauma reactions usually involve withdrawal, nightmares, anxiety, and pressured, repetitious recounting of the traumatic event. These responses are well known to psychiatrists and will not be addressed here.

Caldwell, Ranson, and Sacks[96] discussed potential reactions of a civilian population under atomic attack and applied Tyhurst's[95] formulations of the impact of disasters on communities. High among their considerations were expectations of group panic. Panic, however, occurs only rarely and in circumstances such that an overwhelming trauma is experienced or expected and all avenues of possible escape but one are closed.

Simulation Studies: Desert Rock I, IV, V

In Desert Rock I[97] an augmented airborne battalion combat team experienced an atomic bomb burst from a position in the open about 7 miles from the aerial explosion in October 1951. Attitude assessment, factual information questionnaires, and polygraph examinations at various times before and after the explosion were given to the participants and to control populations at the same base (Fort Campbell, Kentucky) and at another distant base (Fort Lewis, Washington).

The most significant finding was that anxiety concerning some of the bomb's effects persisted at a high absolute level throughout the entire experiment. It was also found that the better-educated were better informed, expressed more self-confidence, and experienced less anxiety. A disproportionate number of soldiers who experienced physiological disturbances on the day of the maneuver were in the lower-educated group. The few higher-educated men with physiological reactions differed from other higher-educated men only in having had greater difficulty adjusting to their roles in the U.S. Army.[97]

Desert Rock IV,[98] staged in May 1952, differed from Desert Rock I in that the participants were armored infantry troops who were stationed 4 miles from ground zero in trenches, and they witnessed the explosion of an atomic bomb emplaced on a tower rather than an aerial bomb. In all, the reactions of about 1,200 men from the 1st Armored Division, including those not involved in the maneuver, were tested from mid-May to mid-June 1952. Findings included the following:

1. Troops showed marked improvement in knowledge about atomic effects as the result of a 4-hour indoctrination at Desert Rock on the day before the atomic bomb explosion.
2. The soldiers were most successful in learning the kind of information that had to do with personal injury (eg, blindness, sterility, impotence). They tended, just after the indoctrination, to overestimate the potential dangers of the atomic bomb more than they did earlier or later.
3. The indoctrination appeared to lessen the fear the troops felt about being on an atomic bomb maneuver.
4. In contrast to the changes noted above, soldiers' attitudes other than fear toward atomic maneuvers and the U.S. Army were not appreciably altered as a result of the indoctrination.
5. There was no evidence that fear made any troops incapable of carrying out their duties just after the detonation; in fact, no

grossly disorganizing fear was observed at any time during the research. The spectacle of the blast apparently had complex effects upon soldiers' fears: In comparison to the previous night, less fear was expressed by the soldiers in answer to direct questions 15 minutes after the explosion, whereas more fear was revealed at this time by indirect (projective) tests.

6. The troops apparently were neither more nor less afraid of the effects of the bomb after they had seen the damage it had done in the forward area.
7. A material increase in the proportion of troops who would volunteer to go on another atomic bomb maneuver, and to occupy a position as close or even closer to ground zero, was recorded just after the bomb explosion.
8. The high point of troops' confidence in themselves and their outfits and in the experts' ability to use the atomic bomb safely apparently was reached just after they had completed the maneuver.

Desert Rock V found similar responses among officers.[99]

Vineberg Report

The Vineberg Report"[100] has remained the standard reference concerning potential psychiatric casualties in a nuclear war. Vineberg reviewed the relevant literature from a number of stressful situations including aerial bombing of cities (including Hiroshima and Nagasaki), the disasters' literature, the behavior of personnel in combat, and psychological mechanisms involved in coping with extreme stress such as major surgery or terminal illness. Based on these studies he formulated a model for nuclear combat.

Vineberg was reasonably optimistic about the ability of a well-trained, highly-motivated soldier to hold up, even when facing inevitable death. His general conclusion was that a soldier would act in tactical nuclear combat much as he had always acted in combat. He did feel that because of the greater stress there would be greater numbers of psychological casualties, but he did not foresee a sharp quantitative change in responses to such stress. As in conventional warfare, psychiatric casualties would arise as a function of cumulative stress related to the duration of exposure to combat and the intensity of combat. Vineberg did not believe that nuclear warfare would result in a breakdown of authority relationships resulting in amoral, lawless, and asocial behavior in civilian or military populations. Altogether, the Vineberg Report was reassuring to military commanders. This optimism had been reflected in the earlier "Clark Report,"[101] in which Clark had estimated total casualties of enlisted men in a unit to range from 4% to 23% in an infantry battalion under atomic attack and concluded that losses ranging from 40% to 70% would have to occur before the unit would be completely demoralized. Perhaps in keeping with this optimism, the NATO handbook, *Emergency War Surgery*,[102] while addressing mass casualties in thermonuclear warfare, was silent about psychiatric casualties.

The author does not share the optimism of Vineberg and others that while psychiatric casualties must be considered, they will not prove decisive in nuclear warfare. Such factors may well have been decisive in the defeat of France early in World War II.[103] Since 1965, neutron weapons capable of surgically killing living beings in precisely designated targets have been developed by the United States. Conventional weapons of near-nuclear capability in terms of blast and overpressure effects now exist, and these may be mistaken for nuclear devices.

Furthermore, it would be naive to believe that nuclear weapons would not be accompanied by chemical and biological warfare. A particularly horrible dilemma might face the irradiated soldier in a chemical environment who must remove his protective mask or die from aspiration of his own radiation-induced vomitus.

Laser and Microwave Radiation

One other form of radiation injury deserves mention because it will be encountered today on any conventional battlefield. The nonionizing electromagnetic radiation of current laser range finders and designators (U.S. forces' as well as the enemy's) will produce injuries ranging from covert retinal burns to sudden catastrophic blindness at distances of several kilometers in unprotected eyes (and further in those using optical instruments). Lasers of great power may be developed as direct weapons as well. This invisible threat with its especially frightening consequences could affect the willingness of troops to look toward the enemy and use weapons' sights, and might stimulate conversion reactions of hysterical blindness as an expression of battle fatigue.

Treatment of Neuropsychiatric Casualties of Nuclear Warfare

The nature of modern combat with its high noise levels, burning flesh, random death, and sleep and sensory deprivation suggests the kinds of psychiatric casualties to expect even in the absence of nuclear weapons.[104] The largest group, unless evacuation is possible, is likely to be the "psychological shock" or "disaster-fatigue" cases encountered in mass casualty incidents. The disaster-fatigue casualties will probably occur primarily in the first few hours and days of the initiation of hostilities. Glass and colleagues[105,106] applied Tyhurst's formulations to the atomic battlefield and established the concept of treating such casualties similarly to combat fatigue; hence, their use of the term "disaster fatigue." This treatment consists of replenishing physiological needs (rest, nutrition, sleep) as necessary, and giving the individuals simple tasks to perform. In this setting, the expectancy that they are "normal" and effective is just as important as on the nonnuclear battlefield.

The treatment of disaster-fatigue casualties must emphasize education and preparation as the critical elements in minimizing these casualties. Obviously, the main target of psychiatric intervention should be the 50% to 80% of "normal" dazed persons who can be given simple tasks to aid their recovery from the psychological shock. The small hysterical group may require sedation; however, this may fixate symptoms. Glass[105] has pointed out the need in these cases for a positive expectancy just as in traditional combat fatigue. The most important element in minimizing these casualties, however, is prevention. It is well known that psychological trauma can be minimized by decreasing the suddenness of the traumatic event; for example, a sudden bereavement, as in the death of a spouse in an accident, is more likely to produce psychiatric morbidity than death of the spouse following a long illness. It is as if time were available for a cognitive desensitization. To prevent these casualties, repeated exposure to as realistic a battle experience as possible must be part of the soldier's training.

Aside from their destructive potential both from blast and radiation effects, nuclear weapons produce in most people a not unwarranted fear verging on hysteria, a fear conditioned by hundreds of media exposures to nuclear holocaust. The mere threat, therefore, of nuclear weapons may result in psychiatric casualties with a primarily psychological etiology. Cataclysmic, unconventional warfare introduces new forms of psychopathology. Long periods of anxious waiting in shelters or in hermetic tanks, and long wearing of masks and NBC protection cause psychic vulnerability[106–109] as reported in the Persian Gulf War.[67,110]

In the event of actual nuclear attack, the direct effect of nuclear flash and blast would have a terrorizing psychological consequence. The extended material destruction and numerous victims create a sense of helplessness and vulnerability that undermines the fighter's morale. Observations of Hiroshima and Nagasaki survivors reveal a collective behavior of "shock-inhibition-stupor" followed by attempts to escape.[111] This is similar to behaviors in earthquakes and similar catastrophes.

Even in the case of threatened use of unconventional weapons, psychopathological behaviors should be expected. The terror of an unknown death with the mythic fantasy of disappearing in the nuclear flash (like the man from Hiroshima reduced to his shadow) and the conviction that one can neither be protected nor cured from radiation sickness are powerful psychological factors. The normal ambiguity of the battleground will be magnified by dispersal and loss of communications due to the destruction of all but hard-wire communications by the nuclear EMP. In such circumstances rumors may magnify in a contagious manner leading to collective panic. Even well-controlled fighters will experience increased levels of anxiety, leading at the minimum to increased numbers of combat fatigue casualties, already expected to be as high as one-to-one, that is, one combat fatigue to one wounded in action, due to the stress of modern continuous warfare. Added to this stress would be concern about family (especially when accompanying U.S. forces in Europe and Korea), a factor found to be important in Israeli casualties during the 1973 Yom Kippur War, increasing the likelihood of breakdown.

While realistic training and strong unit cohesion fostered by good leadership leading to high levels of fitness and morale will minimize psychiatric casualties in even this hellish combat environment, concrete steps should be undertaken now to minimize such casualties and make best use of all resources in the event of nuclear war. Information on the diagnosis and management of neuropsychiatric casualties of NBC warfare should be widely disseminated. Realistic training in a partially contaminated NBC environment should continue. This training should involve not only combat arms troops but also combat-support troops, who may

be even more likely than combat troops to suffer chemical and nuclear attacks as the enemy attempts to disrupt logistical support. Education to combat rumors and fear of the unknown must be emphasized.

Fatally irradiated soldiers should receive every possible palliative treatment, including narcotics, to prolong their utility and alleviate their physical and psychological distress. Depending on the amount of fatal radiation, such soldiers may have several weeks to live and to devote to the cause. Commanders and medical personnel should be familiar with estimating survival time based on onset of vomiting (see Table 4-4). Physicians should be prepared to give medications to alleviate diarrhea, and to prevent infection and other sequelae of radiation sickness in order to allow the soldier to serve as long as possible. The soldier must be allowed to make the full contribution to the war effort. He will already have made the ultimate sacrifice. He deserves a chance to strike back, and to do so while experiencing as little discomfort as possible.

SUMMARY AND CONCLUSION

In summary, a wide range of chemical, biological, and nuclear threats exist. Many are invisible, persistent, have delayed effects, or are contagious. Delivery means may be nonspecific, ubiquitous, or covert. Detection methods are often inadequate. Early signs and symptoms of exposure may be nonspecific and common. The consequences of delaying treatment may be irreversible and fatal. Since Pavlov's studies with dogs, scientists have known that a requirement to discriminate between ambiguous stimuli typically produces severe anxiety, stereotyped, sometimes bizarre behavior, or both. This behavior must be expected to some extent in any combat against a foe known to be capable of using NBC weapons, even if those weapons are not actually employed.

A variety of maladaptive psychological reactions may be evoked by the threat of NBC warfare. Anxiety may lead to heightened susceptibility to rumors of the use of NBC warfare. This, in turn, could provoke undue concern, not only for self and unit, but also for the safety of dependent families if they are in the area at risk. Preoccupation with early warning symptoms may encourage individual hypochondriasis and increase the baseline demand for medical attention. Group amplification of stress and hyperventilation symptoms may cause epidemics of "gas hysteria," as seen in World War I. Even panic flight may occur if units feel completely unprepared or if protective measures seem inadequate (as they may in a hysterical epidemic). There may also be excessive concern with decontamination (perhaps leading to dermatologic problems), and overavoidance of possible sources of exposure (possibly causing dehydration, malnutrition, or even refusal to obey orders). On the other hand, there may be inappropriate fatalism or overconfidence leading to abandonment of NBC protective equipment and of sensible precautions.

Even without "gas hysteria," the added fear and uncertainty about NBC use will probably increase the incidence of acute stress reactions ("battle fatigue"). Rates as high as one case of transitory battle fatigue per one wounded in action have been projected based on historical data. Medical personnel are not immune to battle fatigue and must become familiar not only with its various presentations and basic treatment but also with its prevention.

Psychological stresses that seem unendurable have been described, but history proves that well-trained, cohesive units can come to accept such horrors as commonplace. Effective deterrence requires not only thinking about the unthinkable but obviously being prepared to deal with it. The psychological threats of NBC warfare have implications for psychological preparation that are especially relevant to the medical departments of the armed services, and to National Guard and reserve units. For example, training should avoid the traditional one-shot CS (tear gas) exposure exercise, which classically conditions somatic and anxiety symptoms to the protective mask. Such training resulted in significant gas-mask phobia cases in the Persian Gulf War. Instead, frequent practical exercises are needed to foster familiarity, confidence, and the special skills required to function in an NBC battlefield. Future warfare has aspects similar to mass-casualty disasters, which produce large numbers of psychological casualties among uninjured witnesses. Perhaps military units should train in naturally occurring disaster incidents. NBC warfare is an exceedingly unpleasant prospect to consider; however, failure to prepare for it only increases the risk of its use.

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