Textbook of Military Medicine

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Part III

Disease and the Environment

Volume 2

OCCUPATIONAL HEALTH

THE SOLDIER AND THE INDUSTRIAL BASE

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OCCUPATIONAL HEALTH
THE SOLDIER AND THE INDUSTRIAL BASE

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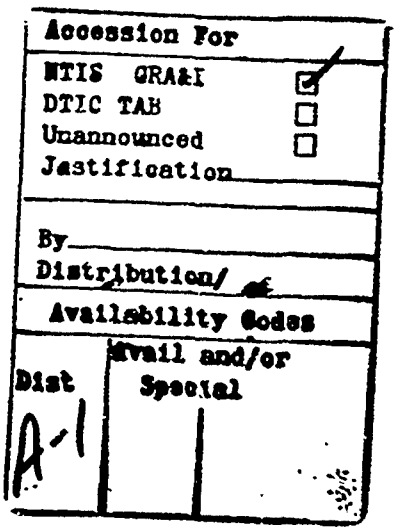
The Coat of Arms
1818
Medical Department of the Army.

A 1976 etching by Vassil Ekimov of an
original color print that appeared in
The Military Surgeon, Vol XLI, No 2, 1917

The first line of medical defense in wartime is the combat medic. Although in ancient times medics carried the caduceus into battle to signify the neutral, humanitarian nature of their tasks, they have never been immune to the perils of war. They have made the highest sacrifices to save the lives of others, and their dedication to the wounded soldier is the foundation of military medical care.



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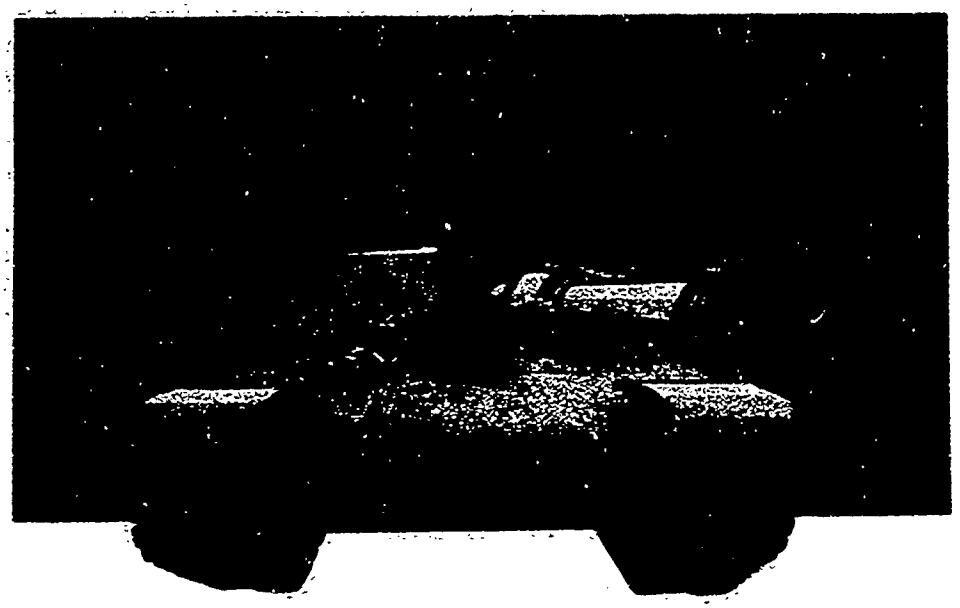

The M1 Abrams tank shown above is surrounded by five of the many, oft-times forgotten, industrial operations that support this weapon system and the soldiers who operate it. Represented clockwise from the lower left corner are (1) heavy metal-parts machining, (2) explosives fabrication, (3) vehicle-components painting, (4) electronics fabrication, and (5) ammunition inspection.

OCCUPATIONAL HEALTH
THE SOLDIER AND THE INDUSTRIAL BASE

Specialty Editors

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1993

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Foreword

Army occupational health came into being during World War I to protect our soldiers and civilian workers against the adverse effects of warfare chemicals that were being produced, stored, transported, and used on the battlefield. During World War II, army occupational health services became even more critical. These assets were absolutely essential for victory. Our civilian workforce had to perform consistently at maximum efficiency for our industrial base to keep pace with our advancing armies. Absenteeism as a result of workplace exposures was intolerable. Additionally, soldier health and performance could not be degraded by potentially harmful stressors like toxic gases in tanks.

More recently, during Operation Desert Storm in 1991, our troops used extremely technical vehicles and equipment and they performed in an outstanding fashion. The army occupational health team played a major role in that victory. During the decade of the 1980s they had worked diligently to improve the soldier–machine interface and to identify, eliminate, or control the stressors that might injure our troops or detract from their ability to fight and survive on the battlefield.

As we move into the 21st century, we must emphasize the practice of occupational medicine for all healthcare providers in the U.S. Army Medical Department. The army of the future, where soldiers will be required to use highly sophisticated and extremely powerful machines properly while also tolerating the stresses of battle, requires that occupational medicine be a core component of military medicine. As the numbers of soldiers and civilians are reduced, we must make every effort to ensure that readiness is not compromised because workplace exposures are adversely affecting our people. Everyone in the Army Medical Department must be alert to the possibility that an illness or injury may be job related, and if it is, take steps to ensure that the harmful exposure is controlled.

This volume will be extremely useful to our active, reserve, national guard, and civilian components, and to contractors, both as an instructional text and as a reference. Every person in the Army Medical Department should know about this volume so that it may be used to support our most valuable resource, our people.

Lieutenant General Alcide M. LaNoue
The Surgeon General
U.S. Army

September 1993
Washington, D.C.

Preface

A decade ago, as Commander of the Army's Training and Doctrine Command (TRADOC), General Carl Vuono tasked his command to ensure that no United States Army soldier would lose life or limb because of improper or insufficient training. Indeed, during that decade, the army experienced a revitalized and concentrated emphasis on training in all TRADOC schools. Our soldiers became the most highly skilled and trained warriors in the world. However, despite the dramatic increase in the soldier's familiarity with the proper use and capabilities of his equipment, the soldier's workplace and environment present significant threats that still exist and endanger each one of our servicemen and -women.

For the soldier, sailor, marine, and airman, the workplace may be a tank, a submarine, a missile silo, or a garrison motor pool. In the more traditional industrial setting, like the motor pool, the principles and practice of occupational medicine are the same for both civilian workers and soldiers. As our civilian and uniformed workers come into contact with more sophisticated military machines and complex hazards, military occupational specialists face a critical challenge. We must ensure that our soldiers do not suffer serious adverse effects as a result of military service and that they are afforded the opportunity to perform at maximum efficiency. This means that every medical practitioner seeing our employees as patients must be able to recognize and know how to deal with the health hazards of both the installation industrial setting and the hazards of the militarily unique setting.

This is the first textbook totally dedicated to the practice of occupational medicine within the U.S. Army. Many of these unfortunate incidents occur because the typical medical practitioner is not sufficiently aware of the potential hazard, or the preventive measures that can be taken to avoid the hazard, to inform decision makers, leaders, supervisors, and the soldiers themselves about the potential illnesses and injuries that may occur and about the means to prevent these illnesses and injuries.

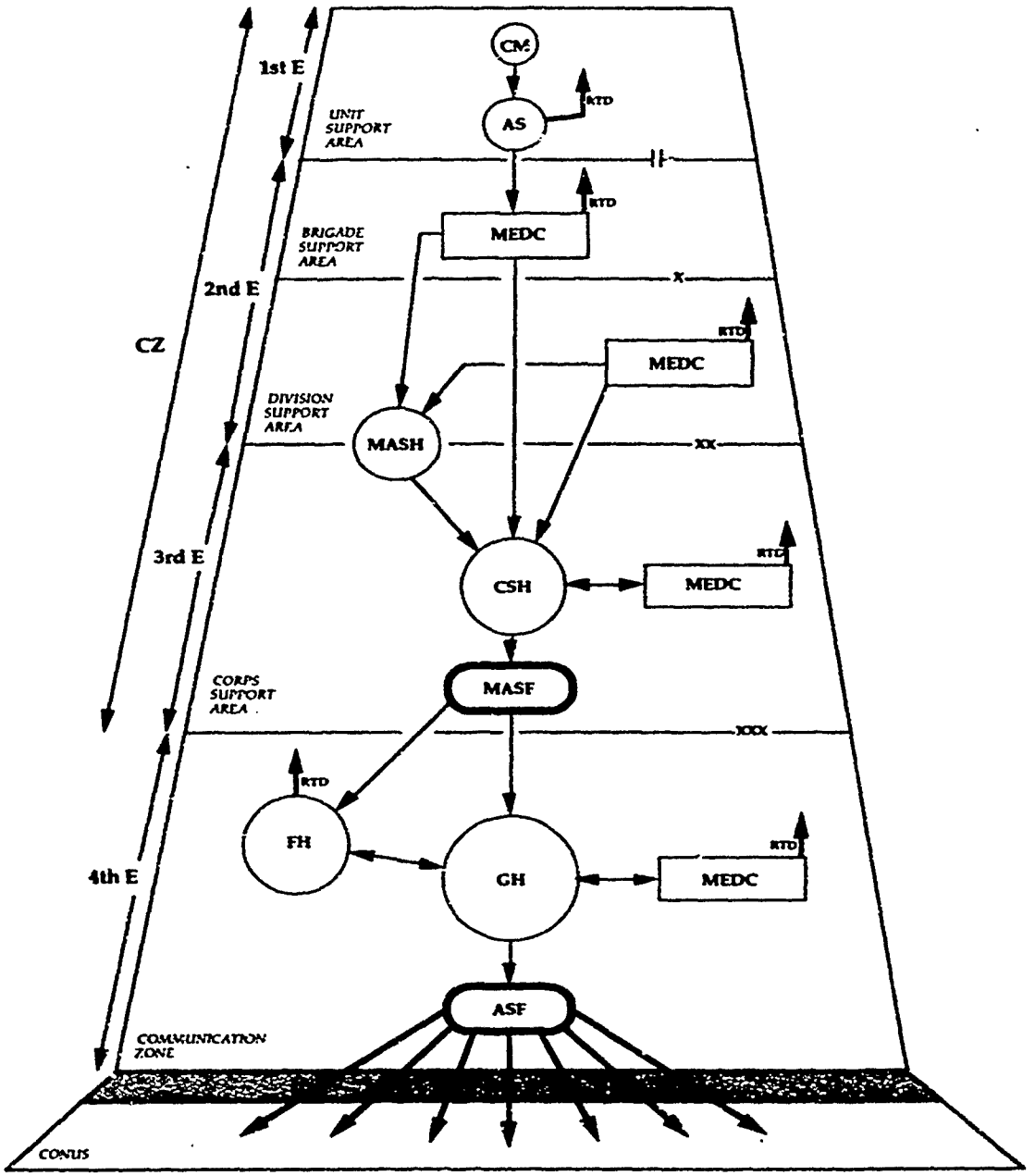
It is my hope that you will find this volume of the *Textbook of Military Medicine* series useful and that it will contribute to the reduction of injuries due to occupational health hazards. This volume became a reality due to the commitment and hard work of Colonel Joel C. Gaydos and Lieutenant Colonel David P. Deeter. Additionally, the editors gratefully acknowledge the assistance in the preparation of this volume of Ms. Barbara Weyandt and Dr. Melissa McDiarmid.

Brigadier General Russ Zajtchuk
U.S. Army

September 1993
Washington, D.C.

The current medical system to support the U.S. Army at war is a continuum from the forward line of troops through the continental United States; it serves as a primary source of trained replacements during the early stages of a major conflict. The system is designed to optimize the return to duty of the maximum number of trained combat soldiers at the lowest possible level. Far-forward stabilization helps to maintain the physiology of injured soldiers who are unlikely to return to duty and allows for their rapid evacuation from the battlefield without needless sacrifice of life or function.

Medical Force 2000 (MF2K)
PATIENT FLOW IN A THEATER OF OPERATIONS



AS: Aid Station
ASF: Aeromedical Staging Facility, *USAF*
CM: Combat Medic
CSH: Combat Support Hospital
CZ: Combat Zone
E: Echelon
FH: Field Hospital
GH: General Hospital
MASF: Mobile Aeromedical Staging Facility, *USAF*
MASH: Mobile Army Surgical Hospital
MEDC: Medical Company
RTD: Return to Duty

Chapter 1

OCCUPATIONAL HEALTH IN THE U.S. ARMY, 1775–1990

JOEL C. GAYDOS, M.D., M.P.H.*

INTRODUCTION

THE CIVILIAN WORKER IN WORLD WAR I
- Gas Production and Gas Protection Plants
- Munitions Industries

THE CIVILIAN WORKER IN WORLD WAR II
- The Army's Responsibility for Employee Health
- Expanding the Industrial Medical Program
- Organizational Advances in Providing Occupational Health Services

THE OCCUPATIONAL SAFETY AND HEALTH ACT AND THE U.S. ARMY
- Occupational Health Programs at Army Installations
- The Occupational Health Management Information System
- Overseas Programs

THE INDUSTRIAL SOLDIER

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- The Early Years
- World War I
- Between the Wars
- Special Laboratories During World War II
- Weapons Modernization in the 1980s

CHEMICAL WARFARE
- Demilitarization
- Production of Binary Chemical Weapons
- Contemporary Threats
- Medical Education
- Ethics

ENVIRONMENTAL HEALTH
- Mission and Organization
- Environmental Program Initiatives

SUMMARY

*Colonel, U.S. Army; Associate Professor and Director, General Preventive Medicine Residency Program, Department of Preventive Medicine & Biometrics, F. Edward Hébert School of Medicine, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799

INTRODUCTION

Occupational health applies the disciplines of medicine, biology, epidemiology, engineering, economics, education, politics, and the law to protect workers from hazards in the workplace.[1] The army's occupational health effort, the U.S. Army Occupational Health Program, has a set of defined goals and objectives and is accompanied by a plan and the resources necessary to achieve success.

Diseases and the injuries associated with productive labor accompany human history. Pliny the Elder (AD 23–70), a Roman scholar, recommended that workers wear masks to prevent inhaling dust and fumes. Georgius Agricola (1494–1553), a German physician, wrote a classic volume describing the diseases and accidents that befall miners and ways to prevent those diseases. The father of occupational medicine, Bernardino Ramazzini (1633–1714), studied and practiced in Italy. His treatise on working conditions and occupational diseases included diseases of military service (*De Morbis Castrensibus*) and *cautions*, or preventive measures. Ramazzini is credited with formulating the question that must be asked about every patient: "What occupation does he follow?"[2-4]

The long-standing interest in Europe in workers' health had no parallel in the United States. Massachusetts created this country's first factory-inspection office in 1867, and the Knights of Labor, a labor group formed later during the 19th century, fought for health and safety measures in mining and other industries. Noteworthy achievements, however, were few.[2,5] Alice Hamilton, M.D. (1869–1970), the pioneer who established occupational health as a specialty in medicine, described early 20th-century conditions (Figure 1-1):

> American medical authorities had never taken industrial diseases seriously, the American Medical Association has never held a meeting on the subject, and while European journals were full of articles on industrial poisoning, the number published in American medical journals up to 1910 could be counted on one's fingers.
>
> For a surgeon or physician to accept a position with a manufacturing company was to earn the contempt of his colleagues as a "contract doctor"; as for factory inspection and control, we never discovered a trace of it.
>
> This ignorance and indifference was not confined to the medical profession—employers and workers both shared it. The employers could, if they wished, shut their eyes to the dangers their workmen faced, for nobody held them responsible, while the workers accepted the risks with fatalistic submissiveness as part of the price one must pay for being poor.[6(pp3-4)]

The development of the U.S. Army Occupational Health Program and the civilian occupational health movement were closely related. Military program efforts were directed primarily at (*a*) the army's civilian worker in the industrial setting, (*b*) the soldier in the industrial setting, and (*c*) the soldier with militarily unique exposures.

THE CIVILIAN WORKER IN WORLD WAR I

U.S. Army Medical Department (AMEDD) involvement with civilian-employee health programs began during World War I because poisonous military chemicals were being produced and used on the battlefield.[7,8] After the German army launched an effective chlorine gas attack against French and Canadian troops in 1915, army medical officers were assigned to the British and French armies as gas warfare observers and reported their observations on gas defense. As a result of the army's concern about gas defense, and possibly related to the observers' reports, AMEDD was assigned the mission to furnish gas masks and other gas-defense equipment to the army. Later, this mission was transferred to the Chemical Warfare Service.[9,10]

Gas Production and Gas Protection Plants

Gas-defense equipment was procured under contract, but the U.S. Army's Office of The Surgeon General (OTSG) had to build and supervise its own plant to manufacture items that were not available commercially. To develop and test procedures for gas defense, some AMEDD personnel participated in experiments and training exercises that dealt with the use of poisonous gases in warfare. The OTSG quickly became convinced that all soldiers and civilian workers who might be exposed to poisonous gas in any setting, including gas factories, must be provided protection and medical care. Lacking expertise in providing

Fig. 1-1. Dr. Alice Hamilton was so impressed by the morbidity and mortality associated with occupational exposures that she devoted her professional life to the practice of occupational medicine. This occurred at a time when American physicians knew nothing about this field or showed little interest in it. Dr. Hamilton was also a pioneer in other respects. After she received her M.D. degree in 1893 from the University of Michigan, she studied in Europe and at The Johns Hopkins University in Maryland; taught pathology at the Woman's Medical School, Northwestern University, in Chicago; and in 1919 became the first woman faculty member at Harvard University. Photograph: Reprinted from United States Public Health Service. *Man, Medicine, and Work*. Washington, DC: USPHS; 1964. Publication 1044.

protection against war gases, the OTSG obtained assistance from the U.S. Bureau of Mines (Department of the Interior), several major universities, and the Marine Biological Laboratory at Woods Hole, Massachusetts. A collaborative program with the Bureau of Mines for supervising the *sanitary supervision* (health and safety evaluations) of government- and contractor-operated gas factories resulted from this effort.[10,11]

Munitions Industries

Because American manufacturers had previously relied heavily on the German chemical industry, they lacked experience in producing (and protecting their workers against) not only chemical warfare agents but also many of the other chemicals needed for wartime use. Additionally, even though American manufacturers were concerned about preventing explosions in their plants, they had no interest in the toxicity of industrial chemicals, particularly unfamiliar ones like picric acid and trinitrotoluene (TNT).

The first beehive coke ovens in the United States were built in southwestern Pennsylvania in 1841 to produce coke from coal for iron and steel production (Figure 1-2).[12] During the early years of World War I, American industries, especially those dealing with coke by-products, had to reconfigure to produce dyes, aniline (needed to make dyes and rubber), and nitric acid (needed to make munitions). Dr. Hamilton, working for the Department of Labor, conducted inspections of these factories.[6] Sometimes *canaries* (workers stained yellow with picric acid) led her to the plants; at other times she located the industrial sites

> by the great clouds of yellow and orange fumes, nitrous gases, which in those days of crude procedure rose to the sky from picric-acid and nitrocellulose plants. It was like the pillar of cloud by day that guided the children of Israel.[6(p184)]

Both Dr. Hamilton and army sources document occupational exposures in the munitions industry as causes of morbidity and mortality.[6,13] In a 1917 report,

Fig. 1-2. The first beehive ovens in the United States were built in southwestern Pennsylvania in 1841 to produce coke from coal for iron and steel production. Near the turn of the century, spurred by the loss of products from the German chemical industry during World War I, American industrialists began to seek better ways to make coke and to capture the chemicals necessary for manufacturing explosives and other synthetic materials. However, the severe air pollution associated with beehive coke ovens continued into the second half of the 20th century. Source: Hamilton A. *Exploring the Dangerous Trades, the Autobiography of Alice Hamilton, M.D.* Boston: Little, Brown and Co; 1943. Photograph: Reprinted with permission from Gates JK. *The Beehive Coke Years: A Pictorial History of Those Times.* Uniontown, Pa: John K. Gates; 1990.

Dr. Hamilton identified 2,432 instances of occupational poisoning. Oxides of nitrogen accounted for 1,389 cases, and 28 of 53 deaths. TNT exposure was considered to be the cause of 660 illnesses and 13 deaths.[6] Similarly, army sources reported a total of 475 fatalities—all or nearly all civilians, presumably due to occupational diseases—from exposure to TNT and related compounds during World War I.[13] According to the chief of ordnance, factories in the United States, which produced 40% of the military explosives used by the allies during World War I, reported a ratio of 230 fatalities (presumably due to occupational diseases) per billion pounds of explosives manufactured.[13]

Dr. Hamilton considered poisoning from oxides of nitrogen to be an engineering problem, which the manufacturers addressed effectively over time, but preventing TNT poisoning was more difficult because this hazard either was not recognized or simply was neglected. In England, medical scientists determined that TNT was absorbed through the skin and manufacturers addressed the need for plant cleanliness and personal cleanliness, including showers at the worksite and washable work clothing, to reduce the potential for skin contact and accidental ingestion. Unfortunately, American manufacturers did not. Furthermore, a wealth of clinical information concerning TNT poisoning had been accumulated in England, but American physicians did not know what to look for, were indifferent, or were secretive.[6]

Dr. Hamilton's attacks on the explosives industry resulted in (*a*) the National Research Council's (NRC's) appointing an expert committee to act as a consultative body and (*b*) her working to establish a code to protect TNT workers. Eventually, the expert committee made it possible for medical students to visit TNT plants to study exposures and poisonings. In April 1919, 5 months after the armistice, a code was published. However, not only was it weaker than the English code, it was also voluntary.[6]

Although Dr. Hamilton was a pacifist, she acknowledged that occupational health in America advanced as a result of World War I:

Fig. 1-3. This April 1942 photograph from the U.S. Army Ordnance Department shows workers measuring smokeless powder, which contains a nitrate or nitroglycerin compound, and pouring the powder into bags, which will later be used to propel projectiles from guns or cannons. Although the dangers of skin absorption in the munitions industry and the need for washable work clothing were recognized in the World War I era, these early-World War II workers labored in street clothes without any evidence of concern about skin contact with the powder. Source: Hamilton A. *Exploring the Dangerous Trades, the Autobiography of Alice Hamilton, M.D.* Boston: Little, Brown and Co; 1943. Photograph: U.S. Army Ordnance Department, 1942.

> The war did have a beneficial influence on industrial hygiene. If it increased the dangers in American industry, it also aroused the interest of physicians in industrial poisons. And that interest has never died down, on the contrary it has increased with the increasing complexity of methods of manufacture. A change took place also in the attitude of employers, for a large labor turnover was found to be not only wasteful but an unsatisfactory method of dealing with dangerous processes in industry. The Public Health Service had entered this field during the war and the medical journals had published articles discussing the action of the new poisons and various methods of preventing danger from the old ones. Industrial medicine had at last become respectable.[6(p198)]

When World War I ended, the medical division of the Chemical Warfare Service continued to do toxicological research and to develop treatments for chemical casualties.[11] However, the army's interest in the health of its civilian employees all but disappeared (Figure 1-3), and when war recurred, the army would again turn to the civilian community to meet its occupational health requirements.[8,13-18]

THE CIVILIAN WORKER IN WORLD WAR II

During the 1930s, first aid was the only occupational health service available to civilian employees of government-owned industrial plants. Part-time contract surgeons, nurses, and enlisted personnel provided this care whenever the War Department (which would later become the Department of the Army [DA]) recognized that a need existed. This changed on 10 August 1938, when the chief of ordnance requested that additional medical services be made available to employees of the army who were engaged in potentially hazardous jobs—particularly those handling TNT. He recognized that the army had a legal responsibility to provide diagnostic and preventive measures for occupational illnesses. The surgeon general knew of no requirement for AMEDD to furnish these services, but recommended that the matter be referred to higher authority. As a result, the adjutant general arranged for additional pay for contract surgeons to perform periodic physical examinations on civilian employees with potentially hazardous exposures.[13] It is interesting to note that it was the chief of ordnance, and not AMEDD, who led the effort to initiate preventive medical services for the army's civilian employees.

The Army's Responsibility for Employee Health

Between 1939 and 1940, the Ordnance Department expanded its activities and continued to place pressure on the surgeon general to confront occupational health issues. The questions: Who should receive occupational health services?; How should the services be implemented?; and How should the needed professional expertise be obtained? were discussed repeatedly. On 18 November 1940, the surgeon general assumed responsibility for the medical care of civilian employees, but only for those who worked at Ordnance Department arsenals. However, he realized that other technical services, like the Quartermaster Corps, also were expanding and had similar needs. (The identification and definition of these occupational health needs were accomplished, at least in part, through site visits and inspections in which the U.S. Public Health Service [USPHS] played a major role.) In September 1941, the army surgeon general requested authority to establish an armywide industrial medical program. The adjutant general responded in January 1942 by directing AMEDD to provide emergency treatment for military and civilian workers and to supervise industrial hygiene practices, but only in army-operated industrial plants (Figure 1-4). Provisions were made for space, equipment, money, and personnel to support the effort.[13]

The participants in an industrial medical conference in August 1942 estimated that the army owned and operated more than 160 industrial plants that employed approximately 400,000 civilians. The army's stated responsibility to this work force was to determine that (*a*) employees are physically fit for their work, (*b*) the conditions under which employees work are safe and sanitary, (*c*) adequate industrial medical service is provided, and (*d*) injuries that occur to employees while they are on duty are reported to the U.S. Employees' Compensation Commission, when indicated.[15]

Expanding the Industrial Medical Program

The surgeon general was under constant pressure to expand the industrial medical program to encompass *all* War Department employees, not just those at industrial installations. He resisted due to a lack of funds and trained personnel. In December 1942, he established a medical program for the approximately 40,000 civilian employees—whose duties were primarily clerical—at the Pentagon. (This reduced the absences from work that occurred when employees needed to visit their personal physicians.) Eventually,

Fig. 1-4. This photograph of an 18-year-old woman operating a lathe, with a caption stating that the "chips are flying," was proudly released by the U.S. Army Ordnance Department in May 1942 as an example of a woman operating a machine that had previously been operated by a man. The photograph itself, its original caption, and the fact that they were released demonstrate that recognition of the need for industrial hygiene controls such as eye protection was lacking. In January 1942, the adjutant general directed the Army Medical Department (AMEDD) to supervise industrial hygiene practices in army-operated industrial plants. Source: Anderson RS, ed. Special fields. In: *Preventive Medicine in World War II*. Vol 9. Washington, DC: DA Office of The Surgeon General; 1969. Photograph: US Army Signal Corps, 1942.

in June 1945, emergency medical treatment services were expanded and made available to all civilian employees of all U.S. Army Service Forces installations. (This was one of the three major commands; the other two were the U.S. Army Air Force and the U.S. Army Ground Forces, and included corps areas and technical supply services.)[12]

Special Considerations for Pregnancy

By 1944, women constituted 40% to 70% of the employees in many army industrial plants. The number of women in the work force had not only increased during the war years but their job assignments also included most types of work. Women were soon given special consideration for medical services. In July 1944, a War Department policy statement on pregnancy was issued and was considered to be the first of its kind in American industry. This policy, which commanders considered a major advance in employee relations, (*a*) limited the work period relative to weeks of pregnancy and the postpartum period, (*b*) required that the job assignment not endanger the employee's health, and (*c*) ensured that job seniority would not be lost as a result of pregnancy.[13]

Special Considerations for Infectious Diseases

In addition to medical services involving women, infectious diseases were also given special consideration. Tuberculosis and venereal diseases in civilian employees were matters of concern. At no cost to the army, the USPHS conducted tuberculosis surveys and case finding among workers of army-owned and army-operated industrial plants.

Workers exposed to TNT or other chemicals that might cause systemic illnesses received routine serological tests. However, these were required only for employees who had been hired for jobs that might jeopardize their health if they had, or were being treated for, syphilis.[13] (Because syphilis was treated with arsenicals that could injure multiple organs, the concern was that the patient would sustain multiple toxicities.)

Special Considerations for Occupational Health Overseas

Interest in and debate regarding American and foreign-national civilians working for the U.S. Army overseas centered on the army's need to do preemployment and preassignment medical screening and the rights of these employees to receive emergency medical care and compensation for job-related injuries and illnesses. There was particular concern about hiring large numbers of foreign nationals without evaluating their health status, especially if they might have communicable diseases. Although overseas civilian-employee medical programs received considerable discussion, better programs should and could have been established. The Occupational Health Division of the OTSG, in evaluating the World War II–overseas programs, recommended that

> the basic plan for future operations in foreign areas ought to include more competent means of medical and engineering control of industrial operations wherever troops are so engaged and civilian employees assist them.[13(p130)]

Ironically, however, the first U.S. Army Occupational Medicine Consultant in Europe (discussed later in this chapter) was not assigned until 1983.

Organizational Advances in Providing Occupational Health Services

The army made significant advances in occupational health in several other areas during World War II: (*a*) occupational health representation in the OTSG was firmly established, (*b*) the U.S. Army Environmental Hygiene Agency (USAEHA) was founded, and (*c*) progress was made in the effort to protect the health of workers in munitions and other wartime industries.

Representation in the U.S. Army Surgeon General's Office

Recognition that occupational health needed to be represented in the OTSG resulted in the 1941 establishment of the Industrial Hygiene Section of the Preventive Medicine Division. In 1942, a separate Occupational Health Division was formed and headed by Lieutenant Colonel Anthony J. Lanza, who developed a 15-point plan for implementing the army's Occupational Health Program. Many of his original points remain appropriate today:

- Worksites with potentially hazardous exposures should be evaluated, and a continuing program of inspection and control should be inaugurated.
- Records should be standardized and should include physical examinations, morbidity reports, and reports of absenteeism.
- Records of mortality and absenteeism should be compiled and compared for all plants.
- Surveys and inspections should be conducted, not only to determine performance and compliance but also to educate civilian and military plant medical officers, plant officials, and workers.
- Health-education programs for employees should be encouraged.[13]

Although his plan eventually succeeded, Dr. Lanza faced inadequate resources including a shortage of doctors and the lack of a policy regarding the practice of occupational health in the army. As its number of industrial employees rapidly increased, the army commissioned physicians with experience in industrial medicine in private life. However, establishing an occupational medicine training program for regular AMEDD officers was considered too time consuming. Those available physicians who had training in general public health were given duty as industrial medical officers, but a shortage of physicians still necessitated that contract surgeons be used.[13] Because trained physicians were few and policy was undeveloped, numerous questions were directed to the Occupational Health Division, U.S. Army Surgeon General's Office. At times, these inquiries showed that some army industrial medical officers simply were not familiar with the standard practice of occupational medicine. In other instances, where the physicians were adequately trained and competent, the questions indicated their difficulty in dealing with the army system. Dr. Lanza realized that both a carefully developed occupational health directive and the guidance necessary to implement the directive were needed. He knew that a document from the OTSG could possibly be more harmful than helpful. Policy that is not developed on the basis of current, accurate data may create unnecessary work, not achieve needed goals, or result in misclassification of priorities. Therefore, staff visits to installations became important vehicles for acquiring data upon which to base policy, establish priorities, and market the army's Occupational Health Program.

By February 1943, sufficient data and experience had been accumulated to warrant issuing War Department Circular Number 59, *Industrial Medical Program of the United States Army*. This document established that industrial medical services would be provided to

all employees at installations that were predominantly industrial in nature, regardless of whether or not the job exposures were hazardous. Specifically, the circular stated:

> As an employer, the Army is obligated to furnish safe and hygienic working conditions and to maintain an adequate industrial medical service. The Surgeon General will make all necessary provisions for the supervision of industrial hygiene and for the emergency treatment of military personnel and civilian employees at Army-operated industrial plants.[13(p117)]

Although Circular Number 59 encompassed numerous employees, War Department workers other than those employed in industrial facilities were excluded. By June 1943, War Department civilians not receiving medical services numbered approximately 600,000.[13] However, 730,000 employees were provided medical services under War Department Circular Number 59 at industrial installations that included depots, manufacturing plants, and repair shops of the Quartermaster Corps; arsenals and depots of the Ordnance Department; arsenals of the Chemical Warfare Service; depots and laboratories of the Signal Corps; depots of the Corps of Engineers; and ports of embarkation. In June 1945, medical services were expanded to include emergency treatment for all civilians working at U.S. Army Service Forces installations.[13]

The U.S. Army Industrial Hygiene Laboratory

The U.S. Army Industrial Hygiene Laboratory was established in October 1942 at The Johns Hopkins University School of Hygiene and Public Health in Baltimore, Maryland. Largely through the efforts of Dr. Anna Baetjer, who played a major role in the laboratory, The Johns Hopkins University fostered the development of the field of industrial hygiene before World War II. The laboratory's mission was to

> conduct surveys and investigations concerning occupational health hazards in U.S. Army–owned and U.S. Army–operated industrial plants, arsenals, and depots.[13(pp169–170)]

Of the two phases of the army's occupational health effort—the supervision of workers and the control of the working environment—the latter was centered largely at this laboratory.[8,13,18,19]

Its initial staff—five officers, one enlisted man, and three civilians—accomplished the laboratory's diverse activities. The officers belonged to the first group of civilian professionals, trained or experienced or both in industrial health, and were commissioned specifically to carry out the army's Occupational Health Program.

The laboratory's activities included both periodic and special surveys and special investigations; these industrial hygiene surveys were conducted at 98 installations. The laboratory developed new methods of industrial hygiene sample collection and analysis (eg, the charcoal-tube method, which is used in many chemical analyses). Special studies investigated the combustion products of solid fuels, the lead hazard associated with field stoves and lanterns, and the fungicides used in the manufacture of various military materials. In conjunction with the USPHS, the laboratory also participated in numerous toxicity evaluations with extensive patch testing.[8,13,18] Other services were provided in the areas of engineering design, chemistry, medicine, statistics, education and training, and toxicology.

The Industrial Hygiene Laboratory played an extremely important role throughout World War II. At the end of the hostilities, the army recognized its future value, relocated it to Edgewood Arsenal, Maryland, and renamed it the U.S. Army Environmental Hygiene Agency.[8,13,18]

Occupational Health in Munitions and Other War Industries

Early in the war, ordnance plants were identified as needing effective accident- and occupational disease–prevention programs. As a result, the Industrial Hygiene Branch in the Office of the Chief of Ordnance was established to oversee preventive-medicine efforts in ordnance plants, regardless of who owned or operated the plant. An army physician and two USPHS officers staffed this branch. The USPHS conducted periodic surveys in contractor-operated plants, and the U.S. Army Industrial Hygiene Laboratory conducted the same type of surveys in government-operated plants. The USPHS also provided further assistance by offering the services of the National Institutes of Health (NIH), and the Water and Sanitation Investigations Station. Specifically, the NIH conducted studies of explosives and gasoline additives, and the Water and Sanitation Investigations Station conducted field studies of water pollution. The USPHS and the Bureau of Mines also collaborated with the army in providing occupational health–education programs.[13] The types of plants that received assistance included (*a*) explosives manufacturing works, (*b*) miscellaneous chemical works (that manufactured basic chemicals for explosives), (*c*) small-arms ammunition plants, (*d*) bag-loading plants (that loaded artillery ammunition), (*e*) arsenals, and (*f*) proving grounds.

One hundred one government-owned explosives plants compiled quarterly occupational disease reports. Ten reporting periods, extending from 1 June 1941 through 31 July 1945, encompassed an average of more than 37 months of operation, with an average workforce of 309,000 employees. These reports demonstrated that the greatest hazards were (*a*) poisoning from TNT; (*b*) exposure to lethal concentrations of oxides of nitrogen (generated through the nitration of organic compounds in explosives manufacturing); and (*c*) contact dermatitis from exposure to compounds like *tetryl* (trinitrophenylmethylnitrosamine), a powerful sensitizer that was used as a booster charge in large-caliber ammunition.[13]

In 968,000 *man-years* of operations during World War II (a man-year is defined as the number of workers multiplied by the number of years each worked), 28 occupational disease fatalities occurred, of which 22 were attributed to TNT, 3 to oxides of nitrogen, 2 to carbon tetrachloride, and 1 to ethyl ether—fewer than three fatalities per 100,000 workers per year. Occupational illnesses that resulted in time lost from work amounted to 2.4 cases per 1,000 man-years of production. Dermatitis caused two-thirds of these, with more serious, systemic illnesses causing 0.8 cases per 1,000 man-years of production.[13]

Compared with data acquired during World War I, the army's efforts to establish a vigorous, large-scale occupational health program during World War II were effective. During World War I, American plants experienced 230 fatalities per billion pounds of explosives manufactured. During World War II, however, government-owned, contractor-operated plants (which produced 95% of all military explosives manufactured in the United States) experienced only five disease-related fatalities per billion pounds of explosives manufactured. Moreover, mortality and morbidity rates decreased as World War II progressed, despite the significantly increased production. These results were attributed to

- a coordinated effort by several governmental agencies that provided occupational health services;
- effective industrial-plant surveys that addressed actual and potential problem areas;
- follow-up and enforcement of survey recommendations through operational channels;
- the availability of technical consultation and studies;
- the fulfillment of research requirements; and
- education programs for healthcare providers, management, and workers.[13]

THE OCCUPATIONAL SAFETY AND HEALTH ACT AND THE U.S. ARMY

From the end of World War II until 1970, there were no significant developments in army occupational health. The Occupational Safety and Health Act (OSHAct) of 1970 made employers responsible for providing safe and healthful workplaces, and ensured that federal and state officials developed and enforced meaningful workplace standards. The OSHAct required record keeping and reporting procedures to monitor job-related morbidity and mortality and strongly encouraged employers to improve old programs or develop new programs to reduce, control, or eliminate workplace hazards and associated injuries and illnesses.[3]

The original OSHAct did not include federal workers, civilians employed by the Department of Defense (DoD), or the military. Title 29, Code of Federal Regulations (CFR), Parts 1910 and 1960, subsequently stated that each federal agency shall comply with the standards issued under the OSHAct.[20] The DA's responsibilities were defined clearly in (*a*) Department of Defense Instruction (DoDI) Number 6055, the *DoD Occupational Safety and Health Program*; (*b*) U.S. Army Regulation 40-5, *Medical Services, Preventive Medicine*; and (*c*) U.S. Army Regulation 385-10, *The Army Safety Program*. These documents required that the DA

- utilize and comply with the standards promulgated under the OSHAct in all operations and workplaces that are not unique to the military, regardless of whether the work is performed by military or civilian personnel;
- apply safety and health standards promulgated under the OSHAct to militarily unique equipment, systems, operations, and workplaces, in whole or in part, insofar as practicable; and
- develop and publish special military standards when compliance under the OSHAct is not feasible in militarily unique situations, or when no applicable standard exists.[21-23]

The applicability of the OSHAct to DoD and DA civilians was only mildly controversial; however, the question of its applicability in certain instances involving uniformed service members, particularly in training and research settings, generated heated contro-

versy. If the U.S. Army Safety Office determined a certain situation to be militarily unique, the OSHAct provisions did *not* apply to the soldiers involved. However, as specified in DoDI 6055.1 and U.S. Army regulations, such a determination in no way relieved the army of its responsibility to its soldiers in the areas of occupational health and safety.[20-23]

The OSHAct has changed the army's official attitude towards occupational health over the last 20 years, reflected in its leadership's greater recognition of the need for

- effective occupational health services that would reduce or eliminate the threat that the army would be cited for serious noncompliance;
- more comprehensive occupational health services at the installation level, particularly with regard to more responsibility for control of the workplace with less reliance on the USAEHA; and
- effective record keeping and reporting procedures, including current, accurate inventories of hazards.[24]

During the early 1980s, the army's leadership became aware of questions in two areas regarding the adequacy of the army's occupational health services. First, at DoD periodic briefings and in response to congressional and other inquiries, it became apparent to the OTSG, the office of the Assistant Secretary of Defense (Manpower, Reserve Affairs, and Logistics), and the office of the Assistant Secretary of the U.S. Army (Installations, Logistics, and Financial Management) that the army did not maintain current workplace-hazard inventories; furthermore, if applicable standards emanating from the OSHAct, or elsewhere, were being met, no documentation existed. And second, Office of Workers Compensation Programs (OWCP) claims for DA employees were increasing toward $100 million per year.[24] These concerns prompted army leadership to take action to improve compliance with the OSHAct and to reduce workers' compensation claims.

Occupational Health Programs at Army Installations

The offices of the Assistant Secretary of Defense (Manpower, Reserve Affairs, and Logistics), and the Assistant Secretary of the U.S. Army (Installations, Logistics, and Financial Management) responded to these problems by authorizing 185 civilian occupational health positions and making $19.5 million available to the Occupational Health Program in fiscal year 1984. Additionally, $7.5 million per year was identified for distribution in fiscal years 1985 through 1988. Later, the annual disbursement of significant amounts of money to the army's occupational health effort was extended into the 1990s.

The new civilian positions allowed for stronger occupational health programs at army installations, particularly in the areas of developing and maintaining workplace-hazard inventories. The money purchased contract assistance, new equipment (such as industrial hygiene sampling instruments and mobile occupational health clinics), and a new, comprehensive U.S. Army Occupational Health Management Information System (OHMIS). These new resources were intended primarily to benefit civilian employees working in the United States and overseas, but they also benefited soldiers.[24,25]

The Occupational Health Management Information System

The army developed the OHMIS specifically to meet AMEDD requirements for data collection, management, and analysis. OHMIS is a three-module, integrated system consisting of (*a*) the Hearing Evaluation Automated Registry System (HEARS), which addresses audiometric testing and workplace noise data; (*b*) the Health Hazard Information Management (HHIM) system, which maintains workplace-hazard inventory data and information on control of hazards; and (*c*) the Medical Information Module (MIM), which provides assistance in the management of clinical services including medical surveillance. OHMIS was a major step toward modernizing and standardizing occupational health data collection, storage, retrieval, and use.[24,26]

Overseas Programs

The recommendation that plans for future military or industrial operations in foreign areas that include soldiers and civilians ought also to include an occupational health program emanated from World War II.[13] But by 1980, still no defined, comprehensive occupational health program existed in any army overseas command. Occupational health needs overseas had not been totally neglected, however. Both the Tenth Medical Laboratory in Germany and the U.S Army Pacific Environmental Health Engineering Agency in the Far East provided workplace evaluations, consultations, and environmental services. Additionally, the USAEHA performed routine and special studies outside the United States.[8] However, a defined program

combining medicine, nursing, and industrial hygiene, with strong installation programs supported by the USAEHA and other laboratories, did not exist. A policy that addressed overseas occupational health programs was also weak or absent.[24] Even though the OSHAct in itself did not apply outside the United States, its applicability to overseas DA civilians and soldiers was covered in regulations and in a DoDI.[20-23]

In 1983, the OTSG initiated overseas army occupational health programs. The army's first occupational health consultant in Europe was assigned to establish the policy and framework needed to support a commandwide program.[27] A major reason that establishing the position of European occupational health consultant was so long delayed was that fully trained occupational medicine physicians were in short supply in the army. During the early 1980s, however, the numbers of highly qualified applicants to the army's occupational medicine residency program at the USAEHA increased greatly, indicating that new positions in occupational medicine could be developed and staffed.[24] The fledgling program in Europe was allocated 40 civilian-position authorizations for fiscal year 1984, and $2.2 million in fiscal year 1985 as its share of the newly received occupational health resources. In 1984, the first occupational health nursing consultant in Europe was hired.[24,27-29] Similarly, but on a smaller scale, an occupational health program was launched in Korea in fiscal year 1987.[30]

Both these overseas ventures represented new approaches to providing services, since distinct occupational health clinics were not created. Instead, the new resources were integrated with existing health services, and missions were expanded to include industrial hygiene and occupational medicine. In most cases, the installation's preventive medicine service and outpatient clinics absorbed the expanded mission requirements.[24]

THE INDUSTRIAL SOLDIER

During the early 1980s, in an attempt to improve occupational health services for soldiers, AMEDD began to sponsor one physician's assistant each year to obtain a master's degree in occupational health at the University of Oklahoma. In 1984, a graduate of this program initiated a model test program for soldiers at Fort Campbell, Kentucky.[31]

Many people assume that all soldiers are combatants. As a result, soldiers engaged in garrison industrial operations, and their need for occupational health services, are overlooked. The test model began by defining all industrial operations at Fort Campbell that were not militarily unique. Of 769 industrial operations—including acid cleaning, battery charging, degreasing, spray painting, and welding—soldiers exclusively performed 530.

A survey of healthcare providers was conducted in an attempt to assess morbidity. Eye injuries were selected as an indicator because safety glasses were noticeably absent among the troops. This survey showed that (*a*) an estimated 95 eye injuries occurred each week among soldiers and (*b*) appropriate eye protection would have prevented 48% of them. The yearly time lost from work associated with preventable eye injuries was over 89,000 man-hours.[31] When the survey findings were presented to the military command, the occupational health specialists were able to demonstrate that preventable eye injuries were detrimental to combat readiness. As a result, funds were allocated not only for safety glasses but also for other items of personal protection, such as respiratory-protective devices. New procedures for procuring these items still needed to be established because the sources of funding and the responsibility for procurement were different from the existing procedures used to purchase personal protective equipment (PPE) for civilian workers.[31]

The Fort Campbell experiment stimulated other studies of occupational morbidity in troops and served as a model for army garrisons worldwide.[31,32] Aspects of the Fort Campbell model that were implemented at many installations included occupational health education efforts for troop medical-care providers, development of new data-gathering systems and forms to support epidemiological surveillance and studies, and efforts to increase command emphasis on occupational safety and health.

MILITARILY UNIQUE EXPOSURES

Soldiers should not be placed at unnecessary risk—in training or in combat—because of either their machines' shortcomings or their own ignorance of the health hazards associated with their equipment. For example, a soldier who fails to wear hearing-protective devices on the firing range today will be a deaf and

ineffective leader on the battlefield tomorrow. The tank commander who uses his vehicle's ventilation system improperly while firing his weapons will put his crew at risk of carbon monoxide poisoning.[17,33] Thus, applying the principles of occupational health to the fighting soldier's unique environment is crucial to military readiness.

The Early Years

Dr. Benjamin Rush (1745–1813), a signer of the Declaration of Independence and the surgeon general of the Middle Department, Continental Army, during the American Revolutionary War, recorded his observations on diseases in military camps and hospitals, including hearing loss from artillery fire.[7] However, for the most part, military men simply accepted hearing loss, exploding cannons, and other risks of their profession.

This acceptance changed during the Civil War (1861–1865), with the introduction of a new generation of weapons—the revolving gun turret, armored railroad artillery, and an early version of the machine gun—that significantly threatened the health of the soldiers who manned them.[34]

The union repeating gun, the early version of the machine gun, was mounted on artillery wheels with a large hopper on top of its single barrel. As the weapon's crank was turned, cartridge cases in the hopper were dropped one by one into a revolving cylinder. A firing pin struck each cartridge, which was then ejected. President Abraham Lincoln liked the weapon, but Colonel John Geary, a hero of the Mexican-American War, returned those that had been sent to him. One reason for his rejection was the danger the weapon posed to its operators. Pieces of soft metal had apparently sheared off—when the cartridges were forced against misaligned parts of the weapon—during firing and had endangered Colonel Geary's own troops. The weapon's most famous casualty was General William Tecumseh Sherman, who was wounded when a piece of metal penetrated his leg while he watched a test-firing.[34]

World War I

Several aspects of war presented unique hazards to soldiers in World War I, including tanks, highly effective machine guns, and trench warfare. Except for its response to the threat posed by gas warfare, no evidence exists to show that the United States paid any attention to the militarily unique occupational health needs of its soldiers. Our European allies, however, did.[7,9,17]

The first major use of tanks was by the British in 1917 in France (Figure 1-5). One of the first British soldiers to go into battle in a tank described his experience:

> The whole crew are at various guns, which break forth in a devastating fire....By this time, the fumes from the hundreds of rounds which we have fired, with the heat from the engines and the waste petrol and oil, have made the air quite oppressive and uncomfortable to breathe in. However, those who go down to the land in tanks are accustomed to many strange sensations, which would make an ordinary mortal shudder.[35,p40]

British and French medical officers identified and attempted to manage threats to the health of their soldiers who used military machines during World War I. The dangers of heat stress in armored vehicles, and of carbon monoxide poisoning that occurred when machine guns were fired in tanks and in enclosed gun emplacements, were well known. (Accidents that caused carbon monoxide casualties had occurred before, and during, the war.)[9,17]

Carbon monoxide was produced by incomplete combustion of the propellant in the machine gun cartridges. Machine gun emplacements, which gun crews had attempted to seal to protect themselves against the enemy's poisonous gases like chlorine, were not well ventilated and were extremely dangerous; carbon monoxide reached toxic levels inside.

Ventilation inside tanks was also poor, and carbon monoxide accumulated when the tanks' weapons were fired. In April 1918, a series of tests performed in France on a Renault tank with a Hotchkiss machine gun showed that soldiers could reduce the level of carbon monoxide inside by opening the tank's doors or running its engine (Figure 1-6).[17] Tests and observations by the French army concluded that machine gun emplacements must never be hermetically sealed, and that soldiers must be protected with adequate ventilation against carbon monoxide poisoning. The French attempted to develop an effective filtering system for carbon monoxide, although their efforts were unsuccessful.[17]

The tunnels that laced the trenches of World War I battlefields created extremely hazardous conditions underground. Defensive mining operations, consisting of digging tunnels and planting explosives, were initially used only when necessary to protect important trenches, salients, or sectors. Later, offensive mining, using large quantities of explosives, resulted in intensive crater warfare. Thousands of soldiers, many of them skilled miners in civilian life, worked underground daily. The use of compressor engines

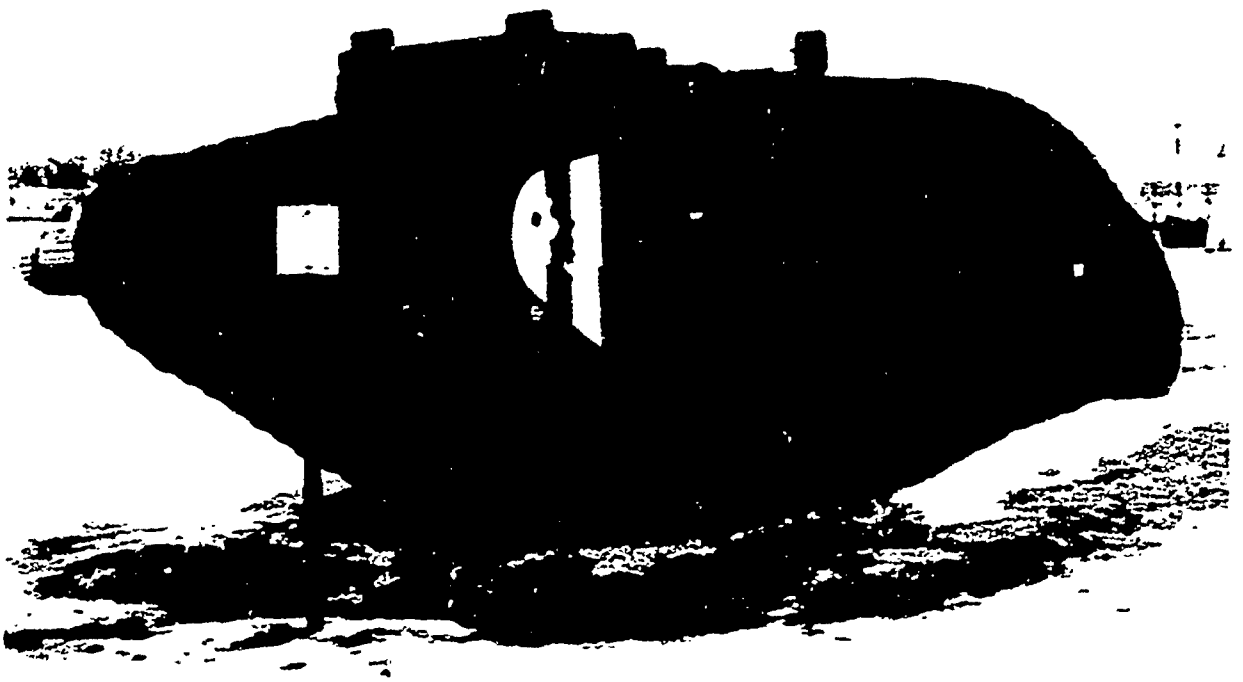

Fig.1-5. The first major tank battle, the Battle of Cambrai, took place on 20 November 1917 in northern France. From behind the British front, 378 ironclads (an early name for tanks) similar to the British Mark IV shown above moved toward the Hindenburg Line. The Mark IV had five .303-caliber Lewis machine guns and armor only thick enough to protect against small-arms fire. The inside of the tank was one large compartment in which the crew were exposed to combustion products from the weapons and exhaust from the petrol engine. Sources: (1) Gaydos JC. A historical view of occupational health for the soldier. *Medical Bulletin of the US Army Medical Department*. 1988;2:4–6. PB 8-88.(2) Cooper B. *The Ironclads of Cambrai*. London: Souvenir Press; 1967. Photograph: Courtesy of Colonel Joel C. Gaydos, Bethesda, Md.

and explosives inside the tunnels created oxygen-deficient areas that had high concentrations of carbon monoxide, nitrogen oxides, hydrogen, and methane. Gas poisonings and explosions caused large numbers of casualties. The British army evaluated existing mine-rescue equipment that was used in the civilian mining industry and adopted an apparatus containing compressed oxygen for the rescuers and an oxygen-resuscitating apparatus for treating the casualties. They also started mine-rescue schools and published an official manual, *Memorandum on Gas Poisoning in Mines*.[9]

Between the Wars

Although the AMEDD made no attempt to develop the field of occupational health and its related areas after World War I, those initiatives were made elsewhere.[8] Fortunately, governmental agencies and private industry recognized the value of such skills. Civilian programs in occupational medicine and industrial hygiene received emphasis in graduate schools of public health and were able to provide the occupational health experts that AMEDD required during World War II.

Special Laboratories During World War II

Several laboratories played a major role in militarily unique occupational exposures during World War II, including (*a*) the Harvard Fatigue Laboratory, (*b*) the Armored Medical Research Laboratory, (*c*) the Climatic Research Laboratory (CRL), and (*d*) the U.S.

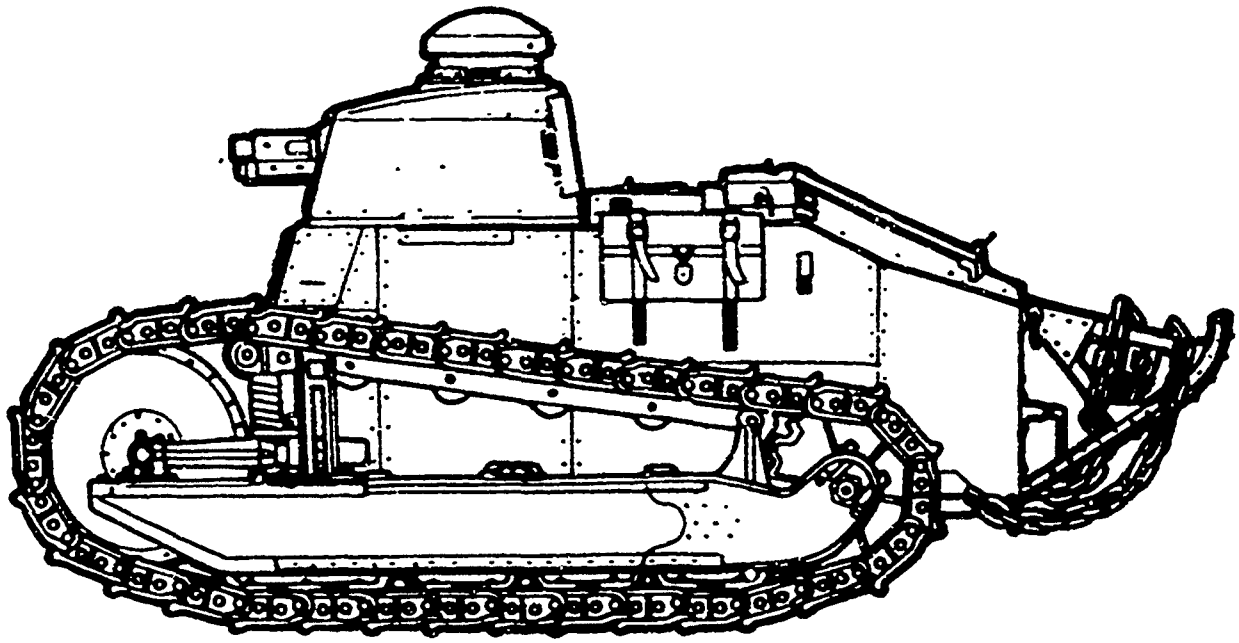

Fig. 1-6. The basic configuration of the modern tank first appeared in 1917 with the French Renault LT. Earlier tanks resembled large, armored boxes but the Renault had the driver in the front, a fully traversable turret containing the main gun in the center, and the engine in the rear. The first Renaults were armed with either an 8-mm Hotchkiss machine gun or a 37-mm gun (above). From the time they had first examined a wooden mockup in December 1916, French military leaders expressed concern that the crew space was too small and that ventilation would be inadequate to prevent asphyxiation of the crew after the gun was fired. Results from a series of tests done in April 1918, in a Renault tank with a Hotchkiss machine gun, showed that the level of carbon monoxide inside the crew compartment could be reduced by firing the weapon with the tank doors opened or with the engine running. Sources: (1) Gaydos JC. A historical view of occupational health for the soldier. *Medical Bulletin of the US Army Medical Department.* 1988;2:4–6. PB 8-88.(2) Zaloga, SJ. *The Renault FT Light Tank.* London: Osprey Publishing; 1988. Original drawing by Leon Conjour, USAEHA Illustration Shop.

Army Research Institute of Environmental Medicine (USARIEM).

The Harvard Fatigue Laboratory

The Harvard Fatigue Laboratory was established in 1927, and its distinguished faculty, students, and graduates had compiled an impressive research record by the time the United States entered World War II. Some staff members remained at the laboratory during the war to do collaborative work with the military; others left for military service elsewhere, either as civilians or as officers. From June 1940 to March 1947, the laboratory submitted 180 research reports to different U.S. governmental agencies and to the British and Canadian armed forces. Those reports addressed clothing and equipment, primarily for use in cold weather; nutrition; physical fitness, including methods of evaluation; high-altitude problems, including anoxia, oxygen masks, heated suits, and physiological response to cold; physiological adaptation to excessive heat; and blood-chemistry derangements, particularly carbon monoxide poisoning.[14,36]

The Harvard Fatigue Laboratory closed in 1947, but it left an indelible mark on the scientific community.[14,36] Several military research facilities were associated with the Harvard Fatigue Laboratory during its existence because staff members who entered the military initiated collaborative efforts with their former colleagues. These facilities, which continued their efforts after the laboratory closed, included the Armored Medical Research Laboratory, Fort Knox, Ken-

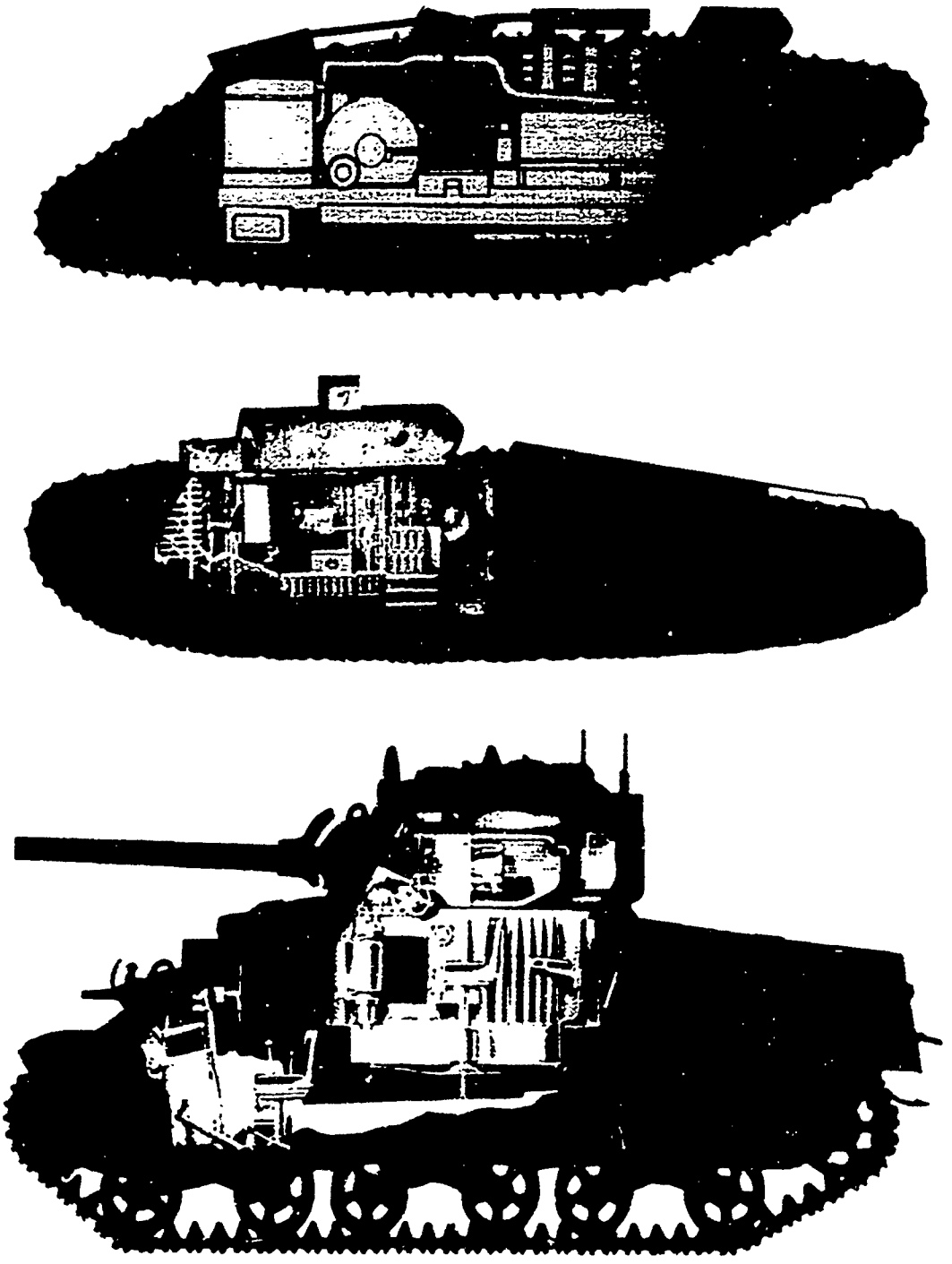

Fig. 1-7. Cutaway drawings of the British Mark IV combat tank of World War I (upper), the British Mark VIII (center), which appeared around the end of World War I, and the Sherman M4 (lower), which was used extensively during World War II. The light areas represent the crew compartments. After only a few years' experience, designers of the Mark VIII placed a bulkhead between the engine and the remainder of the tank interior that, at least theoretically, would reduce crew exposure to engine exhaust products, petrol vapors, engine heat, and engine fires. The concept of a separate engine compartment was incorporated into World War II tanks, but crew exposure to combustion products of weapons continues even to the present. Original drawings by Leon Conjour, USAEHA Illustration Shop.

tucky; the U.S. Army Climatic Research Laboratory (CRL), Lawrence, Massachusetts, and later Natick, Massachusetts; the Naval Medical Research Institute, Bethesda, Maryland; the Navy School Aeromedical Laboratory, Pensacola, Florida; and the Aeromedical Laboratory, Wright Field, Ohio.[14,36]

The Armored Medical Research Laboratory

As the threat of World War II approached and the role of armor in modern warfare unfolded, no U.S. Army organization had been assigned the responsibility for studying the soldier in this environment, particularly the exposures inside tanks (Figure 1-7).[36] The concern that crews of armored vehicles would be unable to perform and endure in hot climates was so great that achieving the technology needed for air-conditioning tanks was considered to be a major breakthrough in modern warfare. The British feared that the Germans had begun to produce air-conditioned tanks.[37] The Germans had not mastered the required technology, however, and there was no rational basis for the British fear.

Even though the initial basic tank design, exemplified by the British Mark IV, had changed considerably, U.S. Army tank commanders and medical officers still feared that limited ventilation in a confined space could result in very high internal temperatures and accumulation of toxic levels of weapons combustion products. Control of the internal temperature of a tank and control of air contaminants through filtration or dilution could be expected to greatly improve the endurance and performance of tank crews, particularly in hot climates and under other conditions that required that hatches be closed. Unfortunately, space and power were so limited in tanks that our own AMEDD officers could not convince tank developers to install even a small fan in the Sherman tank to improve ventilation.[15]

The tank environment posed several potential problems to the soldiers of all forces, including heat stress, toxic gases created by weapons fire, postural injuries, and fires in the crew compartment. With the assistance and support of the NRC, the Armored Medical Research Laboratory was established in 1942 at Fort Knox, Kentucky, to deal with the medical problems of armored-vehicle crews.[13,15] Staffed by physicians, medical and physical scientists, and engineers, the laboratory's mission was to

- identify and evaluate the stressful demands placed on operators of tanks and other weapons;
- determine the limits and the capabilities of soldiers; and
- find the proper balance between operating demand and human capabilities, to avoid breakdown or failure of the man–weapon system.

In April 1942, the OTSG began to recruit teachers and researchers from civilian sources to staff the Armored Medical Research Laboratory.[13-16,36] During its more than 3 years of wartime service, the laboratory staff produced approximately 130 reports dealing with 19 different categories of problems, including temperature extremes; rations; physical and mental standards for tank crewmen; protection against fire, dust, noise, and blast in tanks; defense against chemical warfare agents; fatigue; and toxic gases.[15,16,38] In one demonstration, general officers became the tank crew in order to experience the irritating effect of ammonia produced by main gun fire in an M4 tank with all its hatches closed (Figure 1-8).[15]

After World War II, the Armored Medical Research Laboratory was involved in reorganizations and name changes; eventually, responsibility for many of its research areas was transferred to USARIEM.[36]

The Climatic Research Laboratory

The CRL, established in 1943 in Lawrence, Massachusetts, was a Quartermaster command with some staffing from the OTSG. The Harvard Fatigue Laboratory supplemented the facilities and staff and provided a senior scientist as the first director and commander.[36] The laboratory was designed to simulate all climatic conditions to which soldiers might be exposed and to test clothing and equipment under those conditions. Between 1943 and 1954, the staff published 359 technical reports on topics including the effects of environmental temperature and physical activity on the variables related to the insulating properties of materials used in clothing, sweat production under varying conditions, cold-induced diuresis, and rewarming.

In 1954, the laboratory was reorganized and eventually became the Environmental Protection Research Division (EPRD) at Natick, Massachusetts. In 1961, sections of the EPRD became part of USARIEM.[36]

The U.S. Army Research Institute of Environmental Medicine

The army established USARIEM in Natick, Massachusetts, by consolidating research elements that had been initiated in the Armored Medical Research Labo-

Fig. 1-8. Sherman medium tank (M4A4, 1942 vintage). The M4 tank had no ventilation designed specifically for the crew. While the engine was running, air that was needed to cool the engine was drawn through the turret, allowing some air exchange to occur in the crew compartment. During World War II, a test was conducted at the Armored Medical Research Laboratory, Fort Knox, Kentucky, in which two general officers and an Army Medical Department (AMEDD) officer attempted to fire 10 rounds of 75-mm ammunition from an M4, similar to the one above, with all the hatches closed. After firing only four rounds, ammonia levels reached about 400 ppm and the "crew" were weeping copiously and ready to quit the test. During World War II, morbidity and mortality from toxic combustion products of weapons were probably averted because tank crews tended to leave the hatches open whenever possible. Source: Hatch, TF. Some reminiscences, the armored force medical research laboratory in WW II. *Medical Bulletin of the US Army (Europe).* 1985;42:22–26. Photograph: Courtesy of Colonel Joel C. Gaydos, Bethesda, Md.

ratory and the EPRD. The laboratory's main effort always was directed at the health and effectiveness of soldiers who functioned at extremes of temperature or at high altitude. More recently, USARIEM assumed research roles in physical fitness and nutrition.[36]

Data and information were acquired at Natick; the Arctic Medical Research Laboratory at Fairbanks, Alaska; the John T. Maher Laboratory at Pikes Peak, Colorado; and from numerous field studies. Using these data, USARIEM assisted military commanders and medical officers by consulting on and teaching about a wide range of topics, including tolerance times for soldiers working in the heat; coagulopathies associated with heat stroke, frostbite, and hypothermia; and injuries associated with physical-fitness training.[36]

Weapons Modernization During the 1980s

Allegations of adverse health effects in operators of new army weapons systems began to surface during the late 1970s. Early in the 1980s, the DoD launched a major weapons modernization program, which brought more allegations and criticism that the army had forgotten the soldier in the man–machine inter-

Fig. 1-9. The OH-58D Advanced Scout Helicopter was designed for enhanced aerial reconnaissance; intelligence gathering; and target detection, acquisition, and designation. It serves as an excellent example of the many different, potentially adverse exposures that may be inherent in new materiel. Under the Health Hazard Assessment Program, the OH-58D was evaluated for the following potential hazards to human health: lasers contained in the ball on top of the main rotor; heat stress resulting from the large number of electronic items carried in board, engine-exhaust products that could enter the cockpit, whole-body vibration; and the crash worthiness of the seats, which had been used on other aircraft and were reported to have been associated with excessive spinal injuries during crash landings. Source: Rowden SE, McIntosh RM. The health hazard assessment program: Occupational health for the soldier in the field. *Medical Bulletin of the US Army Medical Department.* 1988;2:7–13. PB 8-88. Photograph: USAEHA file.

face. Critics also attacked the army for failing to adequately address crew and vehicle survivability on the battlefield in the design and testing of armored combat vehicles.[17,39,40]

The Health Hazard Assessment Program

Weapons and equipment development continued after World War II, as new technologies (such as radar and lasers) produced potential threats to the health of soldiers who operated and maintained them. Although AMEDD continued to develop its expertise in occupational health, this expertise was not integrated into the U.S. Army Materiel Acquisition Decision Process (MADP), a multiyear cycle in which new military items are systematically conceived, developed, tested, reviewed, and accepted, rejected, or modified, before being fielded. Therefore, no systematic medical review occurred to identify and control, or eliminate, hazards to the soldiers who used and maintained the new tanks, guns, and other equipment.[17,39]

Problems resulting from the absence of expert medical opinion in the MADP became obvious by the late 1970s and early 1980s. Questions arose regarding the potential harmful effects from blast overpressure with the M198 howitzer and carbon monoxide poisoning in the Bradley Fighting Vehicle (BFV), when both were nearing the end of a multiyear MADP cycle. These questions should have been addressed earlier in the conceptual stages of materiel development to preclude the possibility that costly—and even unacceptable—changes would have to be made. To prevent similar problems in the future, U.S. Army Regulation 40-10 established the U.S. Army Health Hazard Assessment (HHA) Program, which was published in October 1983.[41] This regulation required medical review of items in the MADP at critical points in the multiyear cycle. The HHA process has evaluated a long list of potential hazards, including noise and vibration in helicopters, toxic gases in armored vehicles, blast overpressure from mortars, and skin irritation and sensitization caused by chemical protective masks, clothing, and other items of PPE (Figure 1-9).[39]

After the army's HHA program was initiated, the U.S. Deputy Chief of Staff for Personnel undertook the Manpower and Personnel Integration (MANPRINT)

initiative. This effort's objective was to ensure that the human component was considered first in the design and development of army systems such as weapons systems, field water-treatment systems, and communication systems. By regulation, key personnel in the MADP were required to attend MANPRINT education programs (which included orientation on medical topics) and to ensure that the human aspect of the soldier–machine interface not only was not forgotten, but was given the highest priority.[42]

Testing for Survivability and Vulnerability

Whether or not a military vehicle, particularly an armored combat vehicle (ACV), survives combat is directly related to both the vehicle's and the crew's vulnerability. Until the 1980s, vehicle vulnerability was assessed by simply evaluating the ability of the armor to withstand penetration by a specified antiarmor threat (eg, a particular rocket or artillery shell). Selective components of the vehicle were tested and data were extrapolated, using computer modeling, to determine the vulnerability and survivability of the ACV. This approach—particularly the testing of the BFV—was criticized, and as a result, the Office of the Secretary of Defense initiated the *Joint Live Fire Program* in 1984. In a closely related action in 1987, the U.S. Congress passed legislation requiring live-fire testing of all weapons platforms, like an ACV, ship, or airplane, that perform as a combat machine against realistic combat threats. The objectives of the testing were to (*a*) assess the vulnerability of both vehicle and crew, (*b*) identify design changes to improve both vehicle and crew survivability, (*c*) produce a database in order to improve computer modeling of vulnerability, and (*d*) assess the lethality of American weapons systems against foreign systems.[40,43] Because the first three of these objectives addressed the protection and survivability of American troops, AMEDD had a role in identifying and defining the potential hazards and making recommendations for improving survivability. However, AMEDD's involvement with the last objective was precluded for ethical reasons. The lead medical organization in this new undertaking was the Walter Reed Army Institute of Research (WRAIR), Washington, D.C., with support from USAEHA and the U.S. Army Aeromedical Research Laboratory (USAARL), Fort Rucker, Alabama.[43] The Armed Forces Epidemiological Board also provided assistance.[44,45]

Using current technology and developing new, the army's vehicles, containing a variety of sophisticated instruments, were subjected to antiarmor threats. Fragments, thermal effects, blast overpressure, flash, acceleration and deceleration, and toxic gases were studied. The results of this effort have been directed toward improving soldier survivability in battle and AMEDD's ability to predict, diagnose, and treat battle casualties. After considerable research and study, health risks within an ACV (including a scenario in which the crew remained in the vehicle after its armor was penetrated) and criteria for predicting injuries were defined and reviewed.[40,44]

CHEMICAL WARFARE

Just as Germany's use of poisonous gas during World War I had stimulated the U.S. Army to initiate its first occupational health program,[7,8] poisonous gas again became a center of interest in both occupational health and environmental fields as the decade of the 1980s drew to a close.

Demilitarization

In 1969, President Richard M. Nixon unilaterally halted American production of warfare chemicals, thereby eliminating the routine replacement of chemical weapons affected by aging.[46] By the late 1980s, a deteriorating American chemical-warfare stockpile was described as "90% useless for military purposes, and costing approximately $65 million per year to safeguard."[47] The 1986 Defense Authorization Act attempted to correct this situation by requiring the destruction (demilitarization) of aging munitions and chemical agents stored at eight locations in the continental United States (CONUS), Johnston Island in the Pacific Ocean, and West Germany.[47] The initial target date of 30 September 1994 for completion of the mandated destruction was later extended to 1997.[47,48] Public concern, congressional interest, and strict requirements for environmental evaluations focused considerable attention on the army's plan to incinerate the agents at the eight CONUS locations and Johnston Island.[48] The involvement of the army's occupational health community in conjunction with the USPHS and the Committee on Toxicology of the NRC included

- documenting and verifying occupational and environmental exposure standards (to include those for the general population);
- reviewing workplace and destruction procedures for compliance with standards;

- assisting with the preparation of environmental assessment and impact documents; and
- addressing inquiries and concerns of citizens, legislators, and the U.S. Environmental Protection Agency (EPA).

Production of Binary Chemical Weapons

In support of the United States government's position that a chemical weapons capability deters an enemy poisonous gas attack, the 1986 Defense Authorization Act also approved production of binary chemical weapons,[48] which began at Pine Bluff, Arkansas, in December 1987.[46] In the binary system, two toxic but sublethal component chemicals are manufactured and stored separately. When the fuze is installed in the weapon (eg, an artillery shell), separate containers of each chemical component are also installed. When the weapon is fired, the separate containers rupture and the components mix to form a new, lethal compound.[47] This weapons system produced a number of new chemicals, such as ethyl 2-[di-isopropylamino]-ethylmethylphosphonite (known as QL). To protect civilians and soldiers who might be exposed to the new chemicals, the army's occupational health community defined requirements for toxicity studies, interpreted toxicity data, developed exposure standards, and evaluated worksite procedures.

Contemporary Threats

The threat of exposure to poisonous chemicals has been considered a possibility with (*a*) armed conflict, (*b*) terrorist activities, (*c*) the destruction of our aging chemical stockpile, or (*d*) the accidental unearthing of old, forgotten underground disposal sites.

In a 1919 report to Congress, General of the Armies John J. Pershing stated:

> Whether or not gas will be employed in future wars is a matter of conjecture, but the effect is so deadly to the unprepared that we can never afford to neglect the question.[49(p11)]

Chlorine, mustard, and phosgene had been used in World War I and caused more than 1 million casualties and almost 100,000 deaths.[49] The possibility of an enemy gas attack was considered to be remote during World War II, but American military personnel received chemical-warfare training nevertheless. Few, however, took either the training or the threat seriously. The opposing sides maintained significant stockpiles, but there was no confirmed combat use of chemical weapons by major belligerents.[49]

An unfortunate incident in 1943 demonstrated that when medical personnel do not suspect and are unprepared to handle chemical casualties, the consequences can be grave.[50,51] At dusk on 2 December 1943, the Germans bombed the allied-occupied harbor of Bari, Italy. The merchant ship SS *John Harvey* was in port loaded with a large quantity of high-explosive munitions and a secret cargo of approximately 100 tons of American-made mustard bombs. The ship and her cargo exploded, and any crew members who might have known of the secret cargo were killed. Many casualties were exposed to mustard vapor and were covered with mustard mixed with oil (Figure 1-10).

The medical personnel who received the casualties had no reason to suspect chemical agents. They made no attempt to protect themselves or to decontaminate their patients; the mixture of mustard and oil remained on their skin for many hours, perhaps even days. The patients' undiagnosed clinical states aroused suspicion among the medical personnel that something unusual had happened, and some kind of chemical exposure was suspected. Eye injuries, skin erythema, and blisters were noted 12 to 14 hours after the bombing. Several days later, an investigating medical officer who had been flown in from England used clinical, epidemiological, and pathological data to show that exposure to mustard had occurred. Of the 617 known mustard casualties among military and merchant marine personnel, 83 (13.6%) died. An unknown number of civilian casualties also occurred. Of the military personnel and merchant seamen, the significant casualties—and all the deaths—occurred among those who had been completely covered with the mustard-and-oil mixture.[50,51]

Fifteen nations were thought to possess chemical weapons in 1989 and eight more were suspected of possessing them.[49] Even though the risk that a superpower might be the first to use chemical weapons was thought to be remote, some nations, like Iraq, had demonstrated a willingness to employ them. Furthermore, the ease of concealment of chemical grenades or canisters, and the psychological impact if they were used, made the possibility of a terrorist chemical attack worthy of consideration.[49]

Also, with the legally mandated destruction of the United States' old chemical stockpile, the likelihood that an accident could occur increased whenever the agents were moved, manipulated, and incinerated.[46,47] Chemical agents have been manufactured, stored, and used in anger, research, and training since World War I, both in the United States and abroad. Many times disposal was by burial in unmarked and unrecorded sites; there have been anecdotal and documented reports of unearthing containers of chemical agents at CONUS

Fig. 1-10. The harbor of Bari, Italy, on 3 December 1943. A German air raid late in the day on 2 December damaged or sank a large number of ships in the allied-occupied harbor, including the SS *John Harvey*, which carried about 100 tons of mustard bombs. Mustard vapor in the smoke, mustard agent mixed with oil from the damaged ships, and lack of preparedness for dealing with mustard casualties resulted in hundreds, or even thousands, of casualties. 13.6% of the 617 military and merchant marine casualties died. Sources: (1) Infield GB. *Disaster at Bari*. New York, Macmillan, 1971. (2) Alexander SF. Medical report of the Bari Harbor mustard casualties. *Military Surgeon*. 1947;101:1–7. Photograph. Reprinted with permission from Infield GB. *Disaster at Bari*. New York: Macmillan; 1971.

installations and American installations overseas. Recently, hundreds of vials of chemical agents, dating back to the early 1950s or earlier, were found at Fort Polk, Louisiana, when a firing range was excavated.[37]

Medical Education

Enhanced respect for the chemical threat and interest in the adequacy of training in the medical management of chemical casualties increased dramatically in the late 1980s. The American program to demilitarize outdated chemical stocks began as an increasing number of countries became able to wage a chemical war.[47,48] In February 1987, the Technical Inspections Division, U.S. Army Inspector General Agency, published the report of an evaluation of medical support at chemical storage sites. The report identified a lack of doctrine and lack of institutionalized training, which extended beyond the storage and demilitarization mission.[51] In response, The Surgeon General of the army authorized a full-time position for a medical consultant for *surety programs*, and tasked the consultant to address all the identified deficiencies and to develop and coordinate all the needed corrective actions with the various commands involved.[51] Emphasis was immediately placed on developing doctrine and improving the level of medical readiness through training.

Work on doctrine and official guidance for battlefield and nonbattlefield exposures to chemical threats was invigorated and The Surgeon General launched and financed a consolidated program of training for medical personnel.[51] The U.S. Army Medical Research Institute of Chemical Defense at Edgewood, Maryland, had offered an outstanding training course in the medical management of chemical casualties for many years. The course's impact was limited because it was not funded and staffed separately, but was taught as an additional mission on a time-available basis by people who had other primary duties. As a result, the numbers of students trained were too small to meet the army's needs. However, using this course as a nucleus, training at Edgewood was expanded and plans were initiated for a high-quality, exportable

course; training of certified instructors; special-application training packages to meet special needs (such as the demilitarization mission); and augmentation blocks of instruction to be used in other existing AMEDD courses (such as the Officer Basic Course).[53]

Ethics

Why are army occupational health professionals involved with chemical warfare agents? The question has been asked repeatedly. Occupational health addresses the protection of workers and the prevention of morbidity and mortality from all hazardous exposures, including chemicals. The principles are the same, regardless of *why* an individual is exposed. Defense against chemical weapons—including physical protection, exposure standards, prophylaxis, and treatment—have been the impetus for, and a continuing part of, army occupational health.

From World War I to the present, the offensive and defensive aspects of chemical production and warfare have never been clearly distinct.[7] At times, this has created a moral dilemma. For example, a medical professional's assessments of chemical exposures are required to determine the need for, or the adequacy of, physical protection, prophylaxis, and treatment. But unfortunately, weapons developers may also use such assessments for destructive purposes. AMEDD has maintained two positions:

- First, identification and definition of potential human health hazards and development of recommendations for promoting health and preventing morbidity and mortality are AMEDD responsibilities.
- And second, assessments accomplished for the purpose of promoting health and preventing morbidity and mortality are a part of the AMEDD mission.

We may argue that medical assessments should be classified or controlled so that access is restricted. However, except in cases involving national security, restricting access can cause and has caused difficulties. For example, the right to know about actual or potential exposures may be violated, and acceptable peer review of the assessment may not occur. An individual's right to know the nature and extent of any exposure has been well established, both in our workplaces and in our communities. After the 1986 Defense Authorization Act was passed, interest in potential exposures from the production, storage, transport, and destruction of chemical agents increased dramatically among scientists and the lay public.[48] The army was criticized because much of the data relating to health had neither been published in the open scientific literature nor been made available to the public through established information channels. The institution of an expert-panel review process by the Centers for Disease Control in Atlanta, Georgia, blunted this criticism.[54] Additionally, all unclassified reports were submitted to the Defense Technical Information Center and the National Technical Information System to facilitate public access.

ENVIRONMENTAL HEALTH

The definition of occupational health, slightly modified, also includes *environmental health*: the application of medicine, other scientific disciplines, and the law to protect all people from environmental hazards and to preserve the environment.[1] Occupational health usually deals with workers and workplace exposures, while environmental health concerns the total population and their exposures in the environment. These two closely related fields differ regarding

- the demographic features and the health status of their populations of interest,
- the knowledge of the exposures and the related risks in those potentially exposed,
- the acceptance of the risks associated with exposure, and
- the application of control measures.

Mission and Organization

In general, AMEDD's role in environmental health, as with occupational health, has been to (*a*) identify and define hazards and to assess the risk to human health; (*b*) use medical means (such as medical screening, diagnostic evaluations, and risk assessment) to help prevent injury and illness and to treat and rehabilitate those already afflicted; and (*c*) assist commanders and managers in communicating risk information, eliminating hazards, and reducing morbidity. Industrial hygienists and safety personnel identify and define hazards inside the traditional workplace and develop recommendations for their control; environmental scientists and engineers accomplish these tasks outside. Often, occupational health professionals in the army have been expected to make the transi-

tion from occupational to environmental health whenever the need occurred. Even though this expectation may seem to be reasonable, the transition is usually difficult. Medical surveillance and evaluation measures for worker populations are unlikely to be directly applicable to a general population in whom not only the dose, duration, and route of exposure, but also the demographic features are quite different. Additionally, even though risk assessment and risk communication have long been considered an important part of employee health programs, they took on different perspectives and greater meaning in the 1980s, as concerned citizens and legislators demanded more precise information on environmental pollutants and their associated disease risks. Army physicians, toxicologists, and other occupational health professionals recognized and accepted their environmental health roles much earlier than their civilian counterparts did, and acquired their expertise through on-the-job and formal training. For example, environmental health has been an important component of residency training programs for physicians at the USAEHA for decades.

AMEDD's role in occupational—and particularly environmental—health were questioned during organizational realignments during the 1980s. Proposals were made to remove industrial hygienists and environmental engineers from AMEDD and place them in the U.S. Army Safety Community and the Corps of Engineers. In every instance, the final decision was to maintain AMEDD's team intact, since an improvement could not be identified.

Environmental Program Initiatives

The army's interest in protecting the environment, paralleling the similar interest in the civilian sector, has increased greatly during the past 20 years. This interest—due to legislation, initiatives by army leadership, public pressure, and special events such as the demilitarization of our aging chemical stockpile and Pershing missiles under the Intermediate-Range Nuclear Forces (INF) Treaty—resulted in the following programs: (*a*) the U.S. Army Environmental Program, (*b*) the Installation Restoration Program (IRP), and (*c*) the Defense Environmental Restoration Program (DERP).[47,48,55,56]

The U.S. Army Environmental Program was initiated during the early 1970s to protect natural resources (air, water, and land) while the army performed its missions. The scope of the program increased, with some shifts in emphasis, to encompass hazardous waste (including toxic and medical waste), noise, radioactive waste, radon control, and asbestos control.[55]

The IRP was developed in 1974 to deal with environmental contamination from toxic and hazardous materials where these were known to exist and where environmental control efforts of some type already existed.[55] The IRP was linked closely to the Resource Conservation and Recovery Act (RCRA) and was primarily the responsibility of the Office of the Chief of Engineers (OCE).[57] The Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) of 1980,[58] also known as the *Superfund Act*, and the Superfund Amendments and Reauthorization Act (SARA) of 1986[59] prompted efforts to clean up much older, hazardous-waste sites on active army installations. With the passage of these two acts, the IRP's mission was greatly expanded and the army's environmental effort became part of DERP. The funding that was available through the Defense Environmental Restoration Account (DERA) facilitated this effort.

Army sites identified for major environmental cleanup were required to have a Health Risk Assessment (HRA) Report.[60] The OTSG, with support from the USAEHA and the U.S. Army Medical Research and Development Command (USAMRDC), was involved in the preparation of selected HRA reports and reviewed OCE contractor-generated reports. The USAEHA also provided help and conducted on-site studies to deal with special problems.[60] During the period November 1988 through January 1990, the army documented savings of $19.6 million as a result of AMEDD's involvement in the environmental effort; the potential savings may reach $128.4 million.[60,61] Approximately 200 OCE contractor-generated HRA reports were reviewed; nearly one-half of them were unsatisfactory, containing errors that would have been extremely costly because unnecessary or inappropriate work would have been performed in cleaning up the environment. Additionally, quick responses to potential problems (eg, workers' complaints regarding illnesses) averted serious outbreaks of diseases and consequent adverse publicity.[60,61]

The INF Treaty, an agreement between the USSR and the United States, became effective on 1 June 1988. This treaty required that Pershing missiles be eliminated and specified that solid-fuel rocket motors be destroyed by demolition, burning, or launching. *Static firing* (ie, firing the rocket motors while they are bolted horizontally to a fixed firing stand on the ground) was selected as the primary means of destruction. The air emissions from the perchlorate-based propellant were primarily hydrogen chloride, aluminum oxide, nitrogen, carbon monoxide, and carbon dioxide.[56]

Coordination of and discussions regarding preliminary testing and eventual destruction of the Pershing missiles involved several groups. AMEDD partici-

pated in the early testing to ensure that the data collected would be adequate for making health assessments. With assistance from the Committee on Toxicology of the National Academy of Sciences, guidelines for exposure of the general population to rocket-motor combustion products were established, and inquiries from governmental agencies and concerned citizens about the basis for and acceptability of the guidelines were answered.[61,62] Since the INF Treaty required verifiable destruction, both sides exchanged observers. The AMEDD was also involved with the INF Treaty by providing health-risk information, education, and prophylaxis, when indicated, to American observers stationed on Soviet soil.

SUMMARY

Occupational health in the army evolved with America's interest in occupational health. Before World War I, American disinterest in employee health in civilian industries was mirrored in the military, but the threats posed by the manufacture and enemy use of chemical warfare forced AMEDD to become involved with the health and safety of workers in gas-producing and gas-defense plants. The war effort stimulated the development of the American chemical industry, with a concomitant increase in attention to the workers' health.[63]

Between World Wars I and II, army leadership lost sight of the value of employee health programs. As World War II approached, however, they quickly recognized that industrial medical services were needed in the wartime industries and that new equipment like the modern tank exposed soldiers to unusual and militarily unique hazards. The civilian sector furnished trained experts and facilities to the ill-prepared army.

Aided by the USPHS, AMEDD implemented and monitored many successful programs that supported both soldiers and civilian employees, produced pioneering contributions to both military and civilian occupational health, and documented the effectiveness of the wartime industrial medical services. Fortunately, the army did not forget about occupational health after World War II.[64]

More workplace services became available after the 1970 OSHAct was passed. The massive equipment-modernization program during the 1980s called new attention to soldiers' militarily unique exposures, and as antiquated chemical weapons were demilitarized and new chemical warfare agents were produced, AMEDD was required to identify and describe environmental hazards and to protect soldiers and civilians with potential exposures to poisonous military chemicals.

The army directed critical resources and great effort to occupational health programs because the need for them was significant and often related to national defense. History has demonstrated that the army's occupational and environmental health programs must be able both to meet the needs of the time and to serve as the framework for rapid expansion during mobilization. Relying on nonmilitary experts in future national crises requires that AMEDD develop and maintain strong ties with the civilian occupational and environmental health communities and foster those experts' professional growth and development.

Equally important, AMEDD's peacetime occupational and environmental health programs must never be weakened to the degree that they no longer provide a framework for mobilization.

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REFERENCES

1. Robbins A. Foreword to the first edition. In: Weghman DH, Levy BS, eds. *Occupational Health: Recognizing and Preventing Work-Related Disease*. 2nd ed. Boston: Little, Brown and Co; 1988.

2. Rosen G. *A History of Public Health*. New York: MD Publications, Inc; 1976.

3. Felton JS, Newman JP, Read DL. *Man, Medicine and Work, Historic Events in Occupational Medicine*. Washington, DC: US Dept Health, Education, and Welfare; 1964 (reprinted). PHS publication 1044.

4. Wright WC, trans. Diseases of Workers [in Latin]. *De Morbis Artificum Bernardini Ramazzini Diatriba.* Chicago: University of Chicago Press; 1940.

5. Levenstein C. A brief history of occupational health in the United States. In: Weghman DH, Levy BS, eds. *Occupational Health: Recognizing and Preventing Work-Related Disease.* 2nd ed. Boston: Little, Brown and Co; 1988.

6. Hamilton A. *Exploring the Dangerous Trades, the Autobiography of Alice Hamilton, M.D.* Boston: Little, Brown and Co; 1943.

7 Bayne-Jones S; Anderson RS, eds. *The Evolution of Preventive Medicine in the United States Army, 1607- 1939.* Washington DC: Office of The Surgeon General, US Department of the Army; 1968.

8. Kneessy AD. *Army Occupational Health and AEHA.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1981. Defense Technical Information Center Access No. A207987.

9. MacPherson WG, Herringham WP, Elliott TR, Balfour A, eds. Medical services, diseases of the war. In: *History of the Great War.* Vol 2. London: His Majesty's Stationery Office; 1923.

10. Bradley HC. Organization in the United States. In: Weed FW, ed. *Medical Aspects of Gas Warfare,* Vol 14. *The Medical Department of the United States Army in the World War.* Washington, DC: Office of The Surgeon General, US Department of the Army; 1926: Chap 1.

11. Brophy LP, Miles WD, Cochrane RC. The Chemical Warfare Service: From laboratory to field. Unnumbered volume. In: *United States Army in World War II: The Technical Services.* Washington, DC: Office of the Chief of Military History; 1959 (reprinted 1970).

12. Leech WA Jr, Speare ME. Coke. In: *Encyclopedia Americana.* Vol 7. New York: Americana Corp; 1966: 228–232b.

13. Anderson RS, ed. Special fields. In: *Preventive Medicine in World War II.* Vol 9. Washington, DC: Office of The Surgeon General, US Department of the Army; 1969.

14. Horvath SM, Horvath EC. *The Harvard Fatigue Laboratory, Its History and Contributions.* Englewood Cliffs, NJ: Prentice-Hall, Inc; 1973.

15. Hatch TF. Some reminiscences, the armored force medical research laboratory in WW II. *Medical Bulletin of the US Army (Europe).* 1985;42:22–26.

16. Bean WB. The ecology of the soldier in World War II. *Medical Bulletin of the US Army (Europe).* 1985;42(5):20–25.

17. Gaydos JC. A historical view of occupational health for the soldier. *Medical Bulletin of the U.S. Army Medical Department.* 1988;2:4–6. PB 8-88.

18. Baetjer AM. The US Army Environmental Hygiene Agency. *Medical Bulletin of the US Army (Europe).* 1985;42:27–29.

19 Rose E. *Disease and Discovery: A History of the Johns Hopkins School of Hygiene and Public Health, 1916–1939.* Baltimore, Md: The Johns Hopkins University Press; 1987.

20. The Occupational Safety and Health Act of 1970, Pub L No. 91-596 *amended by* 29 U.S.C. § 651 et seq (1976).

21. *Department of Defense Occupational Safety and Health Program.* Washington DC: US Department of Defense; 1984. DoD Instruction 6055.1.

22. US Department of the Army. *Preventive Medicine* Washington, DC: DA; 1986 Army Regulation 40-5.

23. US Department of the Army. *The Army Safety Program.* Washington, DC: DA; 1988. Army Regulation 385-10.

24. Ranadive MV. Army occupational health program—current concerns. Presented at Occupational Health and Toxicology Update, April 1987; Garmisch, West Germany.

25. Defense Resource Board. Occupational health program resource increases FY 84–88. 1982. *Defense Resource Board Decision*. Handout.

26. Office of The Surgeon General. *Approval of System Decision Paper (SDP) for Occupational Health Management Information System (OHMIS)*. US Department of the Army; 1985. Action Memorandum for Assistant Secretary of the Army (Financial Management), 29 November 1985.

27. Deeter DP, Prier RE, Schmith JM. Occupational health program development. *Am J Prev Med*. 1987;3(3):128–133.

28. Office of The Surgeon General. *Occupational Health Staffing*. US Department of the Army; 1983. Action Memorandum fo. Assistant Secretary of the Army (Installations, Logistics, and Financial Management). 15 September 1983.

29. US Department of the Army. *Program/Budget System (PBS) Dictionary Report*, 17 May 1985. Washington, DC: 1985. PDIP-6T4P, p1301.

30. Office of The Surgeon General. Memorandum, *Occupational Health Funding Policy for FY87*. US Department of the Army; 1986. Memorandum to Commander (18th Medical Command). 5 December 1986.

31. Lott RJ. Occupational health for the soldier. *Medical Bulletin of the US Army Medical Department*. 1988;2:28–30. PB 8-88.

32. Ward DL, Gorie C. Occupational eye injuries in soldiers. *J Occup Med*. 1991;33(5):646–650.

33. Dalton BA. Carbon monoxide in US Army tactical vehicles. *Medical Bulletin of the US Army Medical Department*. 1988;2:11–13. PB 8-83.

34. Davis B. *The Civil War: Strange & Fascinating Facts*. New York: Fairfax Press; 1982.

35. Cooper B. *The Ironclads of Cambrai*. London: Souvenir Press; 1967.

36. Francesconi R, Byrom R, Mager M. United States Army Research Institute of Environmental Medicine: First quarter century. *Physiologist*. 1986;29(5 Suppl):58–62.

37. Crew FAE, ed. The army medical services: Administration. In: *History of the Second World War*. Vol. 2. London, England: Her Majesty's Stationery Office; 1955.

38. Eichna LW, Bean WB, Ashe WF, Nelson N. Performance in relation to environmental temperature, reactions of normal young men to hot, humid (simulated jungle) environment. *Bull Johns Hopkins Hosp*. 1945;76:25–58.

39. Rowden SE, McIntosh RM. The health hazard assessment program: Occupational health for the soldier in the field. *Medical Bulletin of the US Army Medical Department*. 1988;2:7–13. PB 8-88.

40. Phillips YY III, Ripple GR, Dodd KT, Mundie TG. Medical evaluation of live fire test injuries, predicting medical effects behind defeated armor. *Army Research, Development & Acquisition Bulletin*. 1989;(Nov-Dec):16–18.

41. US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process*. Washington, DC: DA; 1983. Army Regulation 40-10.

42. US Department of the Army. *Manpower and Personnel Integration (MANPRINT) in the Materiel Acquisition Process*. Washington, DC: DA; 1987. Army Regulation 602-2.

43. Biddle W. How much bang for the buck? *Discover*. 1986;(September):50–63.

44. Armed Forces Epidemiological Board. *Recommendation on the Draft Technical Bulletin on Nonfragmentary Injuries Behind Defeated Armor*. US Department of the Army, The Surgeon General; US Department of the Navy, The Surgeon General; US Department of the Air Force, The Surgeon General. Memorandum to the Assistant Secretary of Defense (Health Affairs). 30 May 1989.

45. Woodward TE. *The Armed Forces Epidemiology Board: Its First Fifty Years*. Washington, DC: Center of Excellence, Office of The Surgeon General, US Department of the Army, and Walter Reed Army Medical Center; 1990.

46. Garelik G. Toward a nerve-gas arms race. *Time*. 1988;131:28.

47. Boyle D. An end to chemical weapons: What are the chances? *International Defense Review*. 1988;9:1087–1089

48. Burton C. Army restudies chemical weapons disposal. *Army Times*. 9 March 1987:3.

49. Kroeson FJ. *Special Report: Chemical Warfare—A Real and Growing Threat*. Arlington, Va: Association of the United States Army; 1989.

50. Infield GB. *Disaster at Bari*. New York: Macmillan; 1971.

51. Alexander SF. Medical report of the Bari Harbor mustard casualties. *The Military Surgeon*. 1947;101:1–17.

52. Darby B. 280 chemical agent vials found. *Beauregard Daily News*. 1987;41(74):1.

53. Office of The Surgeon General. *Medical Management of Chemical Casualty Training for AMEDD Personnel*. Washington, DC: US Department of the Army; 1988. Information paper.

54. US Department of Health and Human Services, Public Health Service. Recommendations for protecting human health against potential adverse effects of long-term exposure to low doses of chemical warfare agents. *MMWR*. 1988;37(5):72–74,79.

55. Office of the Deputy Assistant Secretary of the Army for Environment, Safety, and Occupational Health. *Army Environment, Safety, Occupational Health: 1985 Accomplishments Report*. Washington, DC: US Department of the Army; 1986.

56. Department of the Army. *Supplemental Environmental Assessment for the Proposed Elimination of Intermediate-Range and Shorter-Range Missiles at Longhorn Army Ammunition Plant, Pursuant to the INF Treaty*. Washington, DC: US Department of the Army; 1988.

57. Resource Conservation and Recovery Act (RCRA), Pub L No. 94-580. 21 October 1976.

58. Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA). Pub L No. 96-510. 11 December 1980.

59. Superfund Amendments and Reauthorization Act (SARA). Pub L No. 99-499. 17 October 1986.

60. Broadwater WT. *Health Risk Assessment (HRA) Performance in Support of the Installation Restoration Program (IRP)*. US Department of the Army, Office of The Surgeon General; 1990. Information paper.

61. US Army Environmental Hygiene Agency. *Health Risk Assessment Performance*. US Department of the Army; 1990. Memorandum for Headquarters, 23 February 1990.

62. Office of the Under Secretary. *Environmental Support for Pershing Demilitarization Options*. US Department of the Army; 1987. Action Memorandum, 19 November 1987.

63. Hatch T. Major accomplishments in occupational health in the past fifty years. *Am Ind Hyg Assoc J*. 1964;25:108–113.

64. Editorial: A half century of service in occupational health. *Am Ind Hyg Assoc J*. 1964;25:104–107.

Chapter 2

MOBILIZING THE INDUSTRIAL BASE

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- Armament, Munitions, and Chemical Command
- Office of the Army Materiel Command Surgeon

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SUMMARY

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INTRODUCTION

The U.S. Army developed occupational health services during World War II in response to the need to maintain productivity in the civilian workforce. Programs were continued after the war, based primarily on the army's recognition that the manufacture and use of modern weapons pose significant and unique hazards to the health of those who use them. The army also recognizes that its civilian workforce is little different from the workforce in other industries, and that the health of its employees must be protected—both to safeguard productivity and to meet its legal and moral obligations to those employees.

Two major factors influence the occupational health needs of the army: the hazards associated with being a soldier, traditionally the basis for defining military medicine; and the nature of the army as a large, multifaceted industry, the basis for that specific part of military medicine that is occupational health. However, before occupational health in the army can be described, the army's occupational health needs must first be defined. This can only be done by examining the army not as military organization—with its missions of *readiness* (being ready for war), *mobilization* (assembling and preparing for war), *deployment* (assuming a strategic position in war) and *sustainment* (supplying the materiel and manpower necessary to prosecute a continuing war)—but as an industry. It is even more important to understand how mobilizing the industrial base influences the army's occupational health needs.

As an industry, the U.S. Army is a multifaceted conglomerate with over 1 million employees. All types of industry are represented: manufacturing, engineering, maintenance, supply, construction, energy, service, and knowledge. While base-support operations must be considered in determining the occupational health needs for a given installation, far more important is the installation's mission (its *product*) The nature of the product often has little to do with the base's industrial operations. For example, the mission of the Training and Doctrine Command (TRADOC) installations includes training (a major product of Fort Benning, Georgia, is trained paratroopers); the mission of the Forces Command (FORSCOM) installations includes readiness (a major product of Fort Campbell, Kentucky, is an air-assault division ready to go to war). Usually, an installation's mission is the major determinant influencing the healthcare services that the Army Medical Department (AMEDD) provides to that installation. Most FORSCOM installations are large and have correspondingly large dependent and active-duty populations, and therefore require a full-service, military-community hospital. The occupational health services provided to the civilian and military workers involved in traditional industrial operations are a very small part of these installations' total healthcare system. Usually, the state of readiness dictates the level of healthcare that is provided to any installation.

But every rule has at least one exception. In the case of the relative importance of industrial operations to an army installation's mission, that exception is the U.S. Army Materiel Command (AMC). The army's "industrial base" refers, for the most part, to the AMC. Because only two AMC installations, Redstone Arsenal and Fort Monmouth, have sufficient active-duty populations to require community hospitals, relatively few army medical personnel are ever assigned to AMC installations; therefore, few in AMEDD are familiar with the AMC. Before the occupational health needs of the army can be understood, the AMC, its operations, and its products —the army's industrial base—must be described.

THE U.S. ARMY MATERIEL COMMAND

The AMC, headquartered in Alexandria, Virginia, was formed in 1962 with the amalgamation of the army's technical services: ordnance, transportation, the quartermaster corps, the signal corps, the chemical corps, and the Corps of Engineers. (The reorganization did not change the identity of the individual corps; it combined the services that had previously been provided by the individual units within those corps.) The overall mission of the command is to support the U.S. Army, the U.S. Army Reserve, and the U.S. Army National Guard through materiel systems research, development, acquisition (including manufacturing of some items), and maintenance of materiel readiness and reserve materiel storage. Although the AMC has undergone major reorganization since the Command Briefing used in May 1992 was written, the briefing papers still detail specific portions of the AMC's mission (Table 2-1).

The AMC workforce is composed primarily of civilian employees, with approximately 10 Department of

TABLE 2-1

ARMY MATERIEL COMMAND MISSIONS

Mission	Function	Sample Output/Accomplishments
1. Equip and sustain a trained, ready army	Determine supply and maintenance needs of soldiers	$3.2 billion annual army stock fund program
	Field new systems	35 PEO and 71 AMC systems fielded
	Manufacture and procure ammunition	695,000 rounds
	Procure, store, and distribute spares, tools, and other supplies to maintain equipment readiness	Fill 3.2 million requisitions
	Manage and perform depot maintenance	97,000 major items rebuilt and repaired
		416,000 secondary items rebuilt and repaired
		$1.5 billion funded depot maintenance program
	Perform postdeployment software engineering	$90 million program for software engineering
		52 million lines of code
	Calibrate and repair test equipment	685,000 items calibrated
		74,000 items repaired
	Provide technical assistance and training to the field	1,300 logistics assistance specialists worldwide
	Dispose of unusable equipment, supplies, and munitions	Screen $1.7 billion in supplies for non-DoD users
		Dispose of $8 million in screened excess supplies
	Support other U.S. government agencies	Law enforcement, treaty compliance, and so forth
2. Provide equipment and services to other nations through the security assistance program	Prepare and present FMS total package letters of offer	7,000 projects currently managed for other countries
		500,000 requisitions per year
	Manage acquisition and delivery of materiel and services	$44.5 billion value current program
		$24.1 billion undelivered orders
	Manage system fielding, training assistance, and follow-on logistics support for allies	142 training teams in FY 1990
		Follow-on support cases worth over $3 billion to support what the U.S. sells
	Manage customer program, perform financial control, and conduct reviews	124 countries and international organizations
	Develop, negotiate, and monitor coproduction agreements	$23 billion value for 28 programs
	Manage commercial export licenses for the army	6,000 export licenses reviewed in FY 1990
3. Develop and acquire nonmajor systems and munitions	Develop, test, and evaluate equipment, systems, and munitions	75 systems (does not include munitions)
	Manufacture, procure, and test equipment and systems	197 systems currently funded and at AMC IPR level
	Provide software engineering support	4 software centers support over 300 systems
	Provide for management, engineering, production, and testing infrastructure	10 test centers conduct technical testing
	Support other government agencies	Drug Enforcement Agency communication and night-vision equipment
		Ammunition procurement for other services
4. Provide development and acquisition support to program managers (PEOs, PMs)	Facilitate the transition of future systems and munitions to and from PEO management	9 systems from PEO to AMC in 1991
	Provide functional support to augment PEO/PM offices	10 PEOs and 117 PMs
	Provide general functional support	Procurement, legal, engineering, integrated logistic support (ILS)
	Provide software engineering support	3 life-cycle software engineering centers support PEO ASM
	Provide for management, engineering, production, and testing infrastructure	AMSAA, White Sands Missile Ranges
5. Define, develop, and acquire superior technologies	Identify technology requirements for the future army	Technology-based master plan
	Demonstrate advanced technologies; facilitate technology transition/insertion	Rotorcraft pilot's associate
	Conduct and sponsor basic research and exploratory development	657 university contracts and grants—$89 million
	Influence/leverage independently funded U.S. and foreign basic research and exploratory development	45 IR&D reviews
		MOUs with 120 countries
	Support PEO/PM and Army/OSD with technology and technical assessments	610 technology-based works packages
		161 PMs supported
	Provide technical advice and counsel	Soldier-as-a-system technology assessment
		AMC FAST program

Table 2-1 (*continued*)

Mission	Function	Sample Output/Accomplishments
	Support other national initiatives	Outreach programs to minorities and women Youth-in-science research and engineering apprenticeship program
	Provide a research and development infrastructure	Ensure adequate training of scientists and engineers
6. Maintain the mobilization capabilities necessary to support the army in emergencies	Develop and test mobilization and contingency plans	Developed and shipped 3 theater-level sustainment packages for Desert Storm that involved 5,870 requisitions for a total of $135 million
	Maintain standby supply depot capacity	Ammunition storage capacity plants 169,000 ft^2 total covered Depots 419,000 ft^2 total covered
	Maintain standby depot maintenance capacity	390,000 items for depot-level work for the remainder of FY 1991
	Maintain inactive ammunition plants and test facilities	Maintenance of inactive industrial facilities—$67.8 million FY 1991 10 inactive army ammunition plants
	Maintain standby arsenal capacity	Rock Island Arsenal has 15% layaway capacity Watervliet Arsenal has 3% layaway capacity Number of pieces of equipment being retained is 1,986
	Maintain war reserve stocks	66% of worldwide ammunitions requirements on hand 106,000 secondary items for war reserves
	Maintain a manufacturing capacity to meet the threat of war	Layaway of industrial facilities WTCV $33.2 million FY 1991 AMMO $22.2 million FY 1991
	Augment government production facilities to meet wartime needs	Production base support projects to provide production facilities not commercially available for a total of $443.2 million
	Provide logistics training for active and reserve units	Provide training sites for approximately 285 reserve component units that involved 65,000 person days
7 Continue to improve productivity and quality of life	Make productivity-enhancing capital investments	LOGSPARS ($2.7 million) with $150 million annual savings SATCOM mission facility ($6.4 million) with $2.4 million annual savings
	Modernize industrial and RDT&E facilities	$61.6 million MCA program Toxicology research facility ($33.0 million) at APG Directed energy weapons lab ($14.4 million) at Redstone Arsenal
	Implement total quality management	Over 55% of HQAMC managers have received TQM training Over 800 process action teams within AMC and the number is growing daily
	Comply with EPA and state regulations	$53 million for environmental compliance FY 1990 (clean water, clean air, waste water) $124 million for environmental restoration FY 1990 (HAZMIN, site cleanup)
	Achieve quality conditions in the workplace and living quarters	Yuma and Selfridge winners in Army Communities of Excellence competition
	Operate installations and facilities efficiently	$68 million productivity investment fund (PIF) at TOAD $268.4 million RPMA programs (RDTE, OMA) 0.8% energy-reduction goal
	Maintain a valued and respected workforce Provide command and control	

AMSAA:	Army Materiel Systems Analysis Agency	IPR:	in-process review	PM:	Program Manager
APG:	Aberdeen Proving Ground	IR&D:	in-process research and development	RPMA:	real property maintenance activity
ASM:	automated system management	LOGSPARS:	Logistics, Planning, and Requirements Simplification System	RDT&E:	research, development, test, and evaluation
FAST:	field assistance in science and technology	MCA:	military construction, Army	TOAD:	Tobyhanna Army Depot
FMS:	foreign military sales	MOU:	memorandum of understanding	TQM:	total quality management
HAZMIN:	hazardous waste minimization	OSD:	Office of the Secretary of Defense	WTCV:	weapon and tracked combat vehicle
		PEO:	Program Executive Officer		

Source: Briefing Papers. Army Materiel Command Headquarters, Alexandria, Va: 1991.

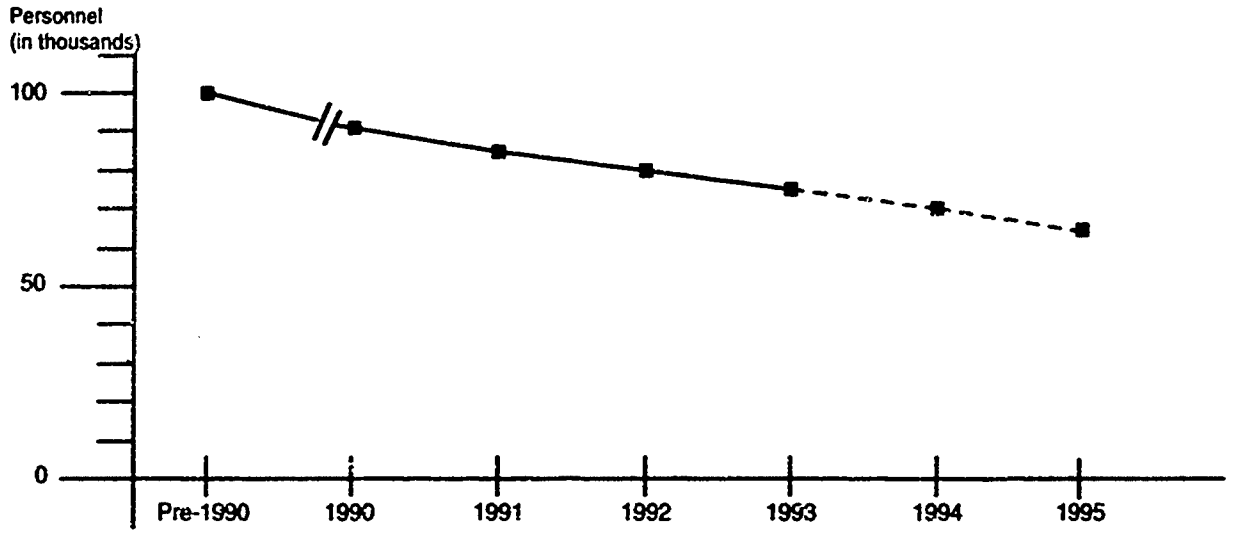


Fig. 2-1. Since it was organized in 1962, the number of employees of the Army Materiel Command has steadily decreased (except of course during the Vietnam era). This decline is projected to continue through 1995.

the Army Civilians (DACs) for each active-duty soldier. However, the actual size of the command is changing rapidly under the army's planned down-sizing (Figure 2-1). For example, in May 1990, AMC personnel consisted of approximately 100,000 DACs and 10,000 active-duty soldiers; by May 1992, the workforce had shrunk to fewer than 90,000 DACs and 8,000 active-duty.

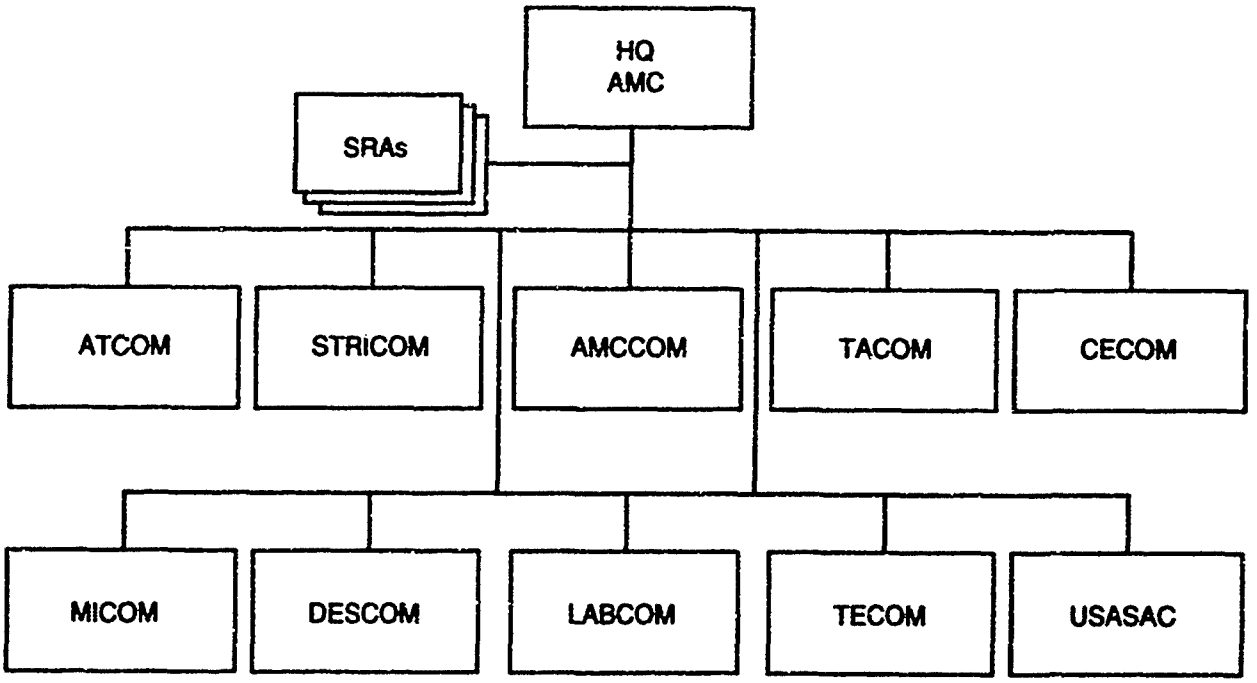
Defining the size of the AMC by looking at the size of the Department of Defense's (DoD's) peacetime workforce can be misleading. There are actually three types of AMC industrial activities:

- Government-owned, government-operated plants. These are staffed by federal employees, and are owned and operated by the AMC. Anniston Army Depot in Anniston, Alabama, is an AMC government-owned, government-operated plant.
- Government-owned, contractor-operated plants. These physical facilities and their missions and products are owned by the AMC, but the plant is operated by employees who work for a plant contractor. Sunflower Army Ammunition Plant, at DeSoto, Kansas, (2 military, 24 DACs, and 697 contract employees) is an active government-owned, contractor-operated plant.
- Contractor-owned, contractor-operated operations. Private-sector manufacturers build, own, and operate these plants, and sell the specified products directly to the AMC. An example of a contractor-owned, contractor-operated operation is the Defense Logistics Agency (DLA) contract with BMY Harsco Corporation, Maryville, Ohio, for the manufacturing of the M914 series heavy utility trucks.

These distinctions are very important because the type of industrial plant is a major determinant of the type of occupational health services that AMEDD is obligated to provide to plant employees. This distinction is discussed in detail in Chapter 3, Army Health Programs and Services. AMEDD provides medical support only to government-owned, government-operated installations.

During the summer and fall of 1992, the AMC underwent a major reorganization. The AMC is currently organized into a headquarters element and 10 major subordinate commands. Figures 2-2 and 2-3 and Exhibit 2-1, which are from the September 1992 AMC Command Brief, depict the command organization, location of subcommittee headquarters, and major missions.

Each major subordinate command has its own areas of responsibility, and is further divided into subordinate commands, which comprise the depots, arsenals, proving grounds, laboratories, ammunition plants, tank factories, and activities. The AMC is also responsible for a number of separate reporting activities (SRAs), which include schools such as the U.S. Defense Ammunition Center and School; and centers for packaging, storage, and containerization. Discussing the AMC organization as of September 1992 is fraught with difficulties. Quite simply, due to army down-sizing, the AMC organization structure is constantly changing. Major



AMCCOM: Armament, Munitions, and Chemical Command
ATCOM: Aviation and Troop Command
CECOM: Communications-Electronics Command
DESCOM: Depot System Command
LABCOM: Laboratory Command
MICOM: Missile Command
SRA: Separate Reporting Activity
STRICOM: Simulation Training Instrumentation Command
TACOM: Tank-Automotive Command
TECOM: Test and Evaluation Command
USASAC: Security Assistance Command

Fig. 2-2. The organizational structure of the Army Materiel Command as of September 1992.

restructuring activities include combining the seven laboratories under the former LABCOM into the Army Research Laboratory, similar to the combining of DESCOM and AMCCOM (which is discussed later in this chapter). As of September 1992, AMC activities consisted of 139 organizations (10 major subcommands), 43 program management offices, 29 separate reporting activities, 3 arsenals, 17 depots, 4 proving grounds, 7 laboratories, and 26 ammunition plants, which are located at 66 installations in 40 states and 6 countries.

In 1973, army leadership almost totally redirected its mechanisms for weapons development, manufacture, and acquisition, giving virtually all these responsibilities to the AMC. TRADOC became responsible for the development of doctrine and combat concepts for new materiel. TRADOC is, therefore, responsible for recognizing a perceived enemy threat and determining what weapon capabilities are needed to address that threat. These needs are then communicated to AMC, the command with the responsibility for translating requirements into acquired materiel.

Once TRADOC formulates the materiel requirements, the concept can progress through the research and development stages. Until 1987, the commanding general of the AMC was the authority for weapons development; however, a major shift was made giving decision-making authority to an individual in the civilian defense acquisition sector, who would have undisputed authority over the process. The oversight for the development of weapons systems has been merged into a single Industrial Operations Command, which will oversee base realignment and structures (Exhibit 2-2). The AMC is projected to be reorganized in 1996 (Figure 2-4).

The oversight for the development of weapons systems has been divided: the assistant secretary of the army for research, development and acquisition (SARDA) administers major systems, which are defined as those costing more than $200 million for research, development, testing, and evaluation; or $1 billion for procurement; the AMC administers the minor weapons systems.[1] The research, development,

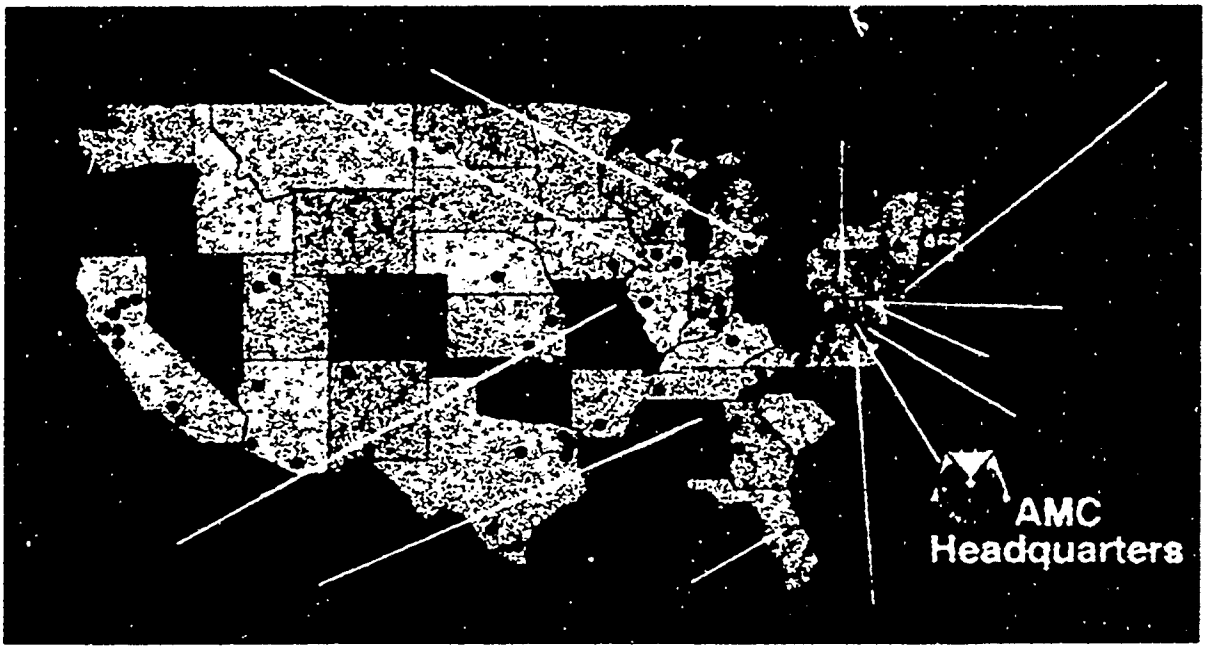


Fig. 2-3. The location of the Army Materiel Command Headquarters as of September 1992.

and testing needed to produce a weapons system may be accomplished by a subordinate command of the AMC, by a contractor, or by a combination of the two. Once the system is fielded, the AMC provides maintenance assistance, major overhaul, and modernization.

Although AMEDD is responsible for providing occupational health services to all of the AMC, two major subordinate commands, DESCOM and AMCCOM, have required more medical services due to their size and the nature of their missions. These two warrant detailed discussion, as does the AMC Command Surgeon's Office.

Depot Systems Command

DESCOM, headquartered at Letterkenny Army Depot, Chambersburg, Pennsylvania, administers an annual operating budget of approximately $2.5 billion. These resources are dedicated to enhancing the army's readiness through overhauling, repairing, modifying, converting, storing, and distributing assigned commodities for the AMC, the Defense Logistics Agency, the General Services Administration, and other suppliers. DESCOM's maintenance mission helps to sustain the readiness not only of every unit in the army but of the other services as well (Figure 2-5).

DESCOM is the largest major subordinate command of the U.S. Army Materiel Command. It comprises almost 33% of AMC personnel assets. With more than 28,000 employees—most of them civilians at 17 installations, DESCOM is equivalent to the country's 85th largest corporation. DESCOM has command and control of 11 depots, 6 depot activities, and 13 other locations throughout the continental United States (CONUS), Germany, and South Korea (Figure 2-6).

As part of the Base Closure and Realignment Process, DESCOM will be transformed into the U.S. Army Industrial Operations Command during the mid-1990s. It will merge with the munitions portion of the U.S. Army Armaments, Munitions, and Chemical Command at Rock Island, Illinois, to streamline functions for industrial operations within the army. Other changes to the organic industrial base are likely.

Anniston Army Depot

Anniston Army Depot, Anniston, Alabama, is the prime depot for the maintenance of tracked combat vehicles and their components, small arms, and land combat missiles, the Army Tactical Missile System, and the Land Combat Support System. Anniston's early involvement as the Center of Technical Excellence made it the lead depot for the M1 Abrams tank. The depot also stores, maintains, distributes, and demilitarizes ammunition, missiles, and toxic chemical munitions. Anniston personnel also repair electro-optics and small arms (Figure 2-7).

EXHIBIT 2-1

MAJOR MISSIONS OF ARMY MATERIEL COMMAND HEADQUARTERS*

AMC Headquarters, Alexandria, Virginia
Provides functional support such as legal assistance and infrastructure management

CECOM, Fort Monmouth, New Jersey
Proponent for all Communication-Electronic Programs

TECOM, Aberdeen Proving Ground, Maryland
Tests and evaluates all types of equipment, from small soldier items to major weapons systems

Army Research Laboratory (previously LABCOM), Adelphi, Maryland
Exercises command and control over all army research laboratories; focuses on research, exploratory development, and analysis

USASAC (collocated with AMC Headquarters)
Proponent for all foreign military sales; receives functional support from the Program Manager (PM) and Program Executive Officer (PEO) offices on specific weapon systems

STRICOM (previously PM; Trade and PM; Instrumentation, Target, and Threat Simulation [ITTS]); Orlando, Florida
The army's focal point for simulation, training, and instrumentation

MICOM, Redstone Arsenal, Huntsville, Alabama
Manages the development, acquisition, fielding, and sustainment of all army missile systems

ATCOM, St. Louis, Missouri
Focuses on development, acquisition, and fielding of aviation systems and individual troop items

AMCCOM, Rock Island, Illinois
Primarily responsible for development of weapons, munitions, and chemical equipment

TACOM, Warren, Michigan
Equips the army with both tracked and wheeled ground mobility equipment

DESCOM, Chambersburg, Pennsylvania
Oversees all depot operations

*As of September 1992

EXHIBIT 2-2

ARMY MATERIEL COMMAND RESTRUCTURING ACTIVITIES*

Close seven depots

Close seven ammunition plants

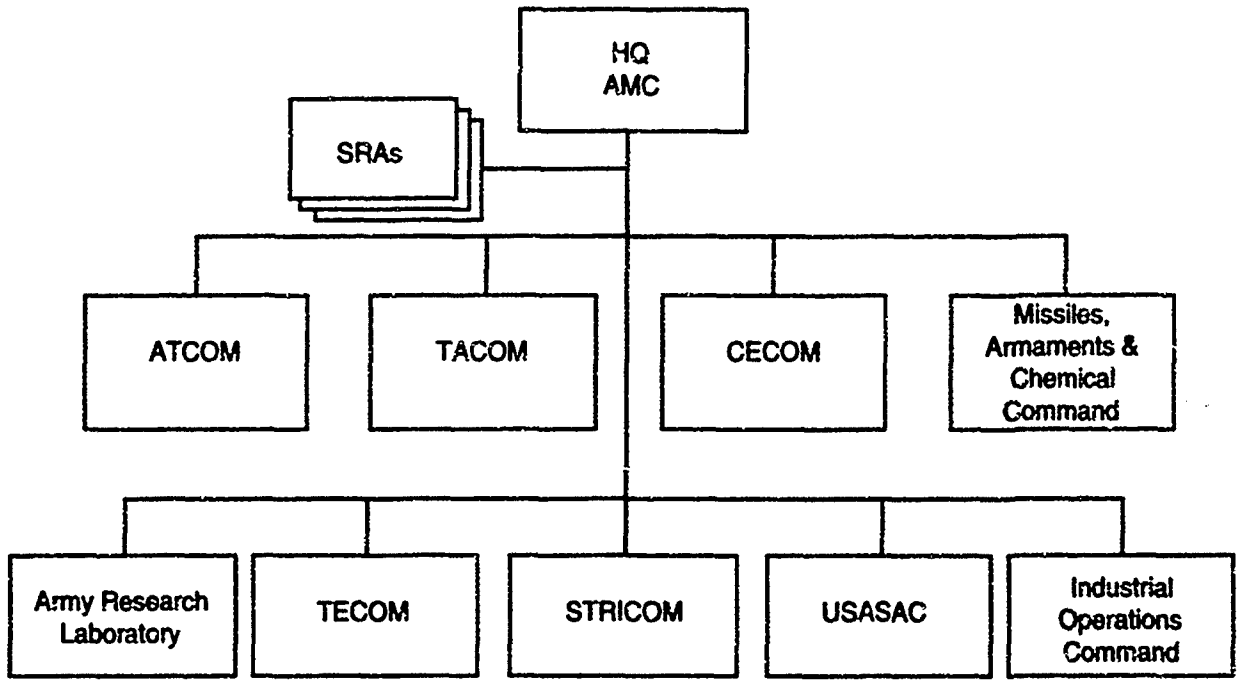
Create the Army Research Laboratory

Create the Army Industrial Operations Command

Create the Aviation and Troop Command

Create the Missiles, Armaments, and Chemical Command

*As of September 1992



ATCOM: Aviation and Troop Command
CECOM: Communications-Electronics Command
SRA: Separate Reporting Activity
STRICOM: Simulation Training Instrumentation Command
TECOM: Test and Evaluation Command
USASAC: Security Assistance Command

Fig. 2-4. The Army Materiel Command organizational structure projected for 1996 assumes that the restructuring activities shown in Exhibit 2-2 are implemented.

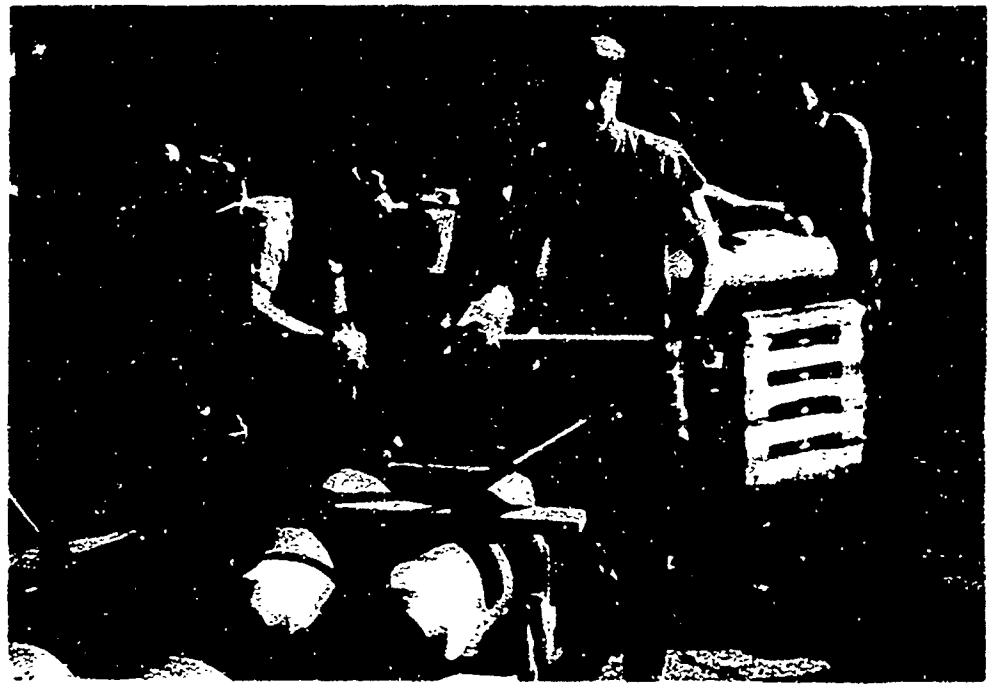


Fig. 2-5. U.S. Army reservists at a DESCOM depot activity ready U.S. Air Force bombs for shipment.

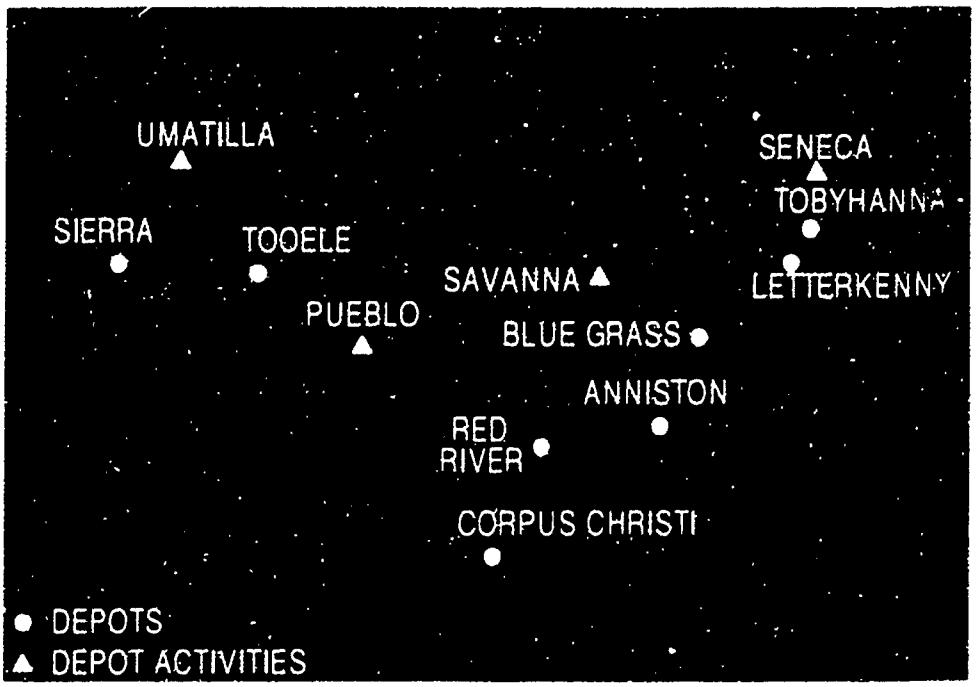


Fig. 2-6. U.S. Army Depot System Command installations. Source: Headquarters, DESCOM, Chambersburg, Pennsylvania.

Fig. 2-7. Abrams tanks damaged during Operation Desert Storm were repaired on this shop floor at Anniston Army Depot, Alabama.

Blue Grass Army Depot

Blue Grass Army Depot, located near Lexington, Kentucky, overhauls communications security equipment and has an extensive mission for assembly of communications-electronics sets, kits, and outfits. The Blue Grass facility, 2 miles south of Richmond, Kentucky, is a storage site for conventional and chemical munitions and clothing and textiles. It also runs the army's only environmentally approved "washout" facility, which recovers explosives from munitions for future use. A government-owned, contractor-operated maintenance facility for communications and electronics materiel is also located at Lexington.

Corpus Christi Army Depot

Corpus Christi Army Depot, located on the U.S. Naval Air Station in Corpus Christi, Texas, repairs, overhauls, and maintains the army's helicopters, including the UH-1 Huey, AH-1 Cobra, and UH-60A Black Hawk; the depot is DESCOM's Center of Technical Excellence for the AH-64 Apache attack helicopter. The depot's aircraft mission covers not only maintenance of helicopters but also associated aeronautical equipment. Corpus Christi Army Depot is the largest industrial employer in south Texas, with more than 4,900 employees. It was the first depot to establish a 24-hour aircraft maintenance "hotline" providing immediate, worldwide maintenance.

Letterkenny Army Depot

Letterkenny Army Depot, Chambersburg, Pennsylvania, is the prime maintenance depot for self-propelled and towed artillery, light recovery vehicles, and air-defense guided missile systems such as the Improved Hawk, the Forward Area Alerting Radar, and the Patriot Air Defense Missile System. Employees there work on equipment supporting the Army's Patriot and Hawk air defense missile systems. Additionally, the depot assembles Sparrow and Sidewinder missiles for the U.S. Air Force. Letterkenny is also the prime overhaul depot for upgrades to the self-propelled howitzer. Letterkenny also provides procurement support to U.S. Army Europe and maintains, stores, and demilitarizes conventional ammunition in CONUS (Figure 2-8).

Savanna Army Depot Activity. Savanna Army Depot Activity, Illinois, under the command and control of Letterkenny Army Depot, stores and ships conventional ammunition and war reserve materiel. The U.S. Defense Ammunition Center and School, a tenant at Savanna, is the only DoD school that provides ammunition training to civilian, military, and foreign students.

Seneca Army Depot. Seneca Army Depot, Romulus, New York, also under the command and control of Letterkenny Army Depot, stores, maintains, and demilitarizes ammunition. It is the army's only site for overhauling and upgrading DoD owned industrial plant equipment.

Red River Army Depot

Red River Army Depot, a multimission depot located in Texarkana, Texas, is the primary depot for the overhaul and repair of light armored vehicles (M113 family), M2 and M3 Bradley Fighting Vehicles, M901A1 Improved TOW (tube-launched, optically tracked, wire command, link guided) vehicles, Chaparral, Multiple Launch Rocket System, and M981 Fire Support Team Vehicles (Figure 2-9). In addition to its maintenance mission, Red River is an Area Oriented Distribution depot, providing supply support to the central United States. It also stores and maintains conventional ammunition.

Tobyhanna Army Depot

Tobyhanna Army Depot, Pennsylvania, is the largest communications and electronics repair facility in the army. The depot maintains equipment such as radio and radar systems, airborne surveillance and navigation equipment, signal intelligence, and satellite communications systems used by all DoD activities, and is the U.S. Army's Center of Technical Excellence for tactical and strategic satellite communications systems, automatic test systems, and space communications. It is the only depot with an approved Ground Control Approach radar antenna pattern range able to support the radar requirements of all the armed forces.

Tooele Army Depot

Tooele Army Depot, Utah, overhauls army tactical wheeled vehicles, power generators, rail locomotives, and Redeye antiaircraft missiles. Tooele stores and maintains both chemical and conventional munitions and is the home of the $64-million prototype Chemical Agent Munitions Disposal System, which safely disposes of lethal chemical munitions. The Ammunition Equipment Directorate designs and fabricates special-purpose ammunition handling and production equipment. Additionally, Tooele has two unique missions: it is the only DoD installation with the capability to repair and overhaul topographic equipment and military locomotives.

Pueblo Army Depot Activity. Pueblo Army Depot Activity, Colorado, is commanded and controlled by Tooele Army Depot. Pueblo stores chemical munitions and is scheduled to be the location of a chemical

Fig. 2-8. Environmental specialists at Letterkenny Army Depot, Pennsylvania, seal a hazardous waste container.

Fig. 2-9. VDLSCOM worker upgrades a Bradley Fighting Vehicle from the M2A2 to the M2A3 configuration.

weapon demilitarization facility to destroy these weapons. The Pershing II missile system was demilitarized by the depot activity under the Intermediate-Range Nuclear Forces Treaty. The depot operates a repository for the storage of precious metals received from states west of the Mississippi River.

Umatilla Army Depot Activity. Umatilla Army Depot Activity, Oregon, is also under the command and control of Tooele Army Depot. Umatilla stores chemical weapons and is scheduled to be the location of a chemical weapon demilitarization facility to destroy those weapons.

European Redistribution Facilities

The European Redistribution Facilities (ERFs) are located at Boeblingen, Hausen, and Nahbollenbach, Germany. The ERFs' mission is to receive class IX (spare and repair parts) excess, serviceable and unserviceable, economically repairable materiel, including Automatic Return Items, and to distribute them to in-theater repair and storage sites, Defense Reutilization and Marketing Offices, or CONUS repair or storage depots.

DESCOM Support Activity, Far East

Located in South Korea, the DESCOM Support Activity–Far East (D-SAFE) contracts with Korean industry to perform maintenance on combat vehicles, tactical vehicles, and communications-electronics equipment. It performs a variety of missions in the Far East including management of repair and overhaul contracts with private industry, coordination of the army's warranty and modifications work-order programs, and total-package fielding. D-SAFE also fabricates and repairs all sizes of pneumatic tires.

Armament, Munitions, and Chemical Command

The AMCCOM, headquartered at Rock Island Arsenal, Illinois, is the second-largest of the AMC subordinate commands. It has command and control of 38 installations and activities in 25 states and employs more than 500 military, 18,000 DAC, and 20,000 contractor personnel (Figure 2-10). With an annual operating budget of over $5 billion and with assets worth over $44 billion, AMCCOM is equivalent to the United States's 72nd largest production corporation and the 8th largest in assets.[1]

AMCCOM's mission is to develop and produce the most advanced guns, bullets, and chemical weapons for United States and allied forces. This mission, accomplished through research, development, and engineering, has three levels of responsibility: total, primary, and support. When AMCCOM has *total* responsibility, the command manages the entire development, production, fielding, and support for the system. Examples of projects totally supported by AMCCOM include the M198 howitzer, the M16-series rifle, and the M40 chemical-protective mask. When AMCCOM has *primary* responsibility for a program, the command manages the overall program but obtains sup-

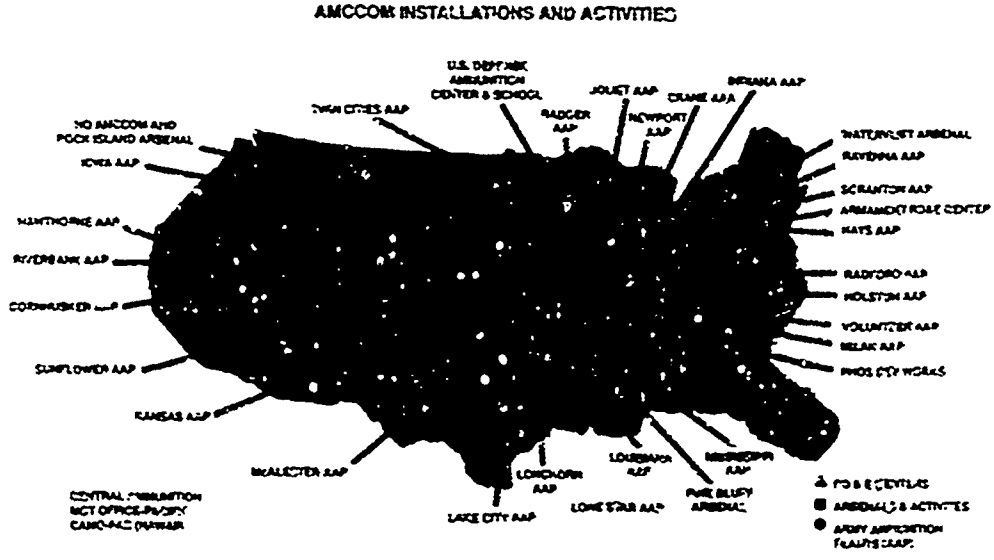


Fig. 2-10. The Armament, Munitions, and Chemical Command (AMCCOM) installations.

port from other commodity commands. The M109 self-propelled howitzers and the lightweight chemical decontamination system are two examples of weapons developed under AMCCOM's primary support. Finally, when AMCCOM has *support* responsibility, the command assists other AMC commodity commands in appropriate areas of expertise. Thus, for the Bradley Fighting Vehicle and the Abram's main battle tank, AMCCOM assisted the tank-automotive command by supplying the armament, fire control, ammunition, and related support equipment needed on these systems.[1]

Installations and Activities

Two of AMCCOM's centers—the Chemical, Research, Development, and Engineering Center (CRDEC) and the Armament Research, Development, and Engineering Center (ARDEC) develop new weapons, the other AMCCOM installations sustain the readiness of combat forces. AMCCOM's three government-owned, government-operated arsenals—Rock Island, Watervliet, and Pine Bluff—perform a variety of manufacturing missions that are central to that effort.

The Chemical Research, Development, and Engineering Center. CRDEC, located at Aberdeen Proving Ground, Maryland, is one of two facilities that incorporates advanced technology into the materiel that AMCCOM maintains. CRDEC develops chemical programs and smoke and other obscurant systems within the DoD. Current CRDEC projects include developing sensors that will detect and identify all known and projected types of biochemical agents, and will sound a warning alarm whenever such an agent is present. Specialists at CRDEC are also working on new protective gear that will be easier to wear and use, and will be fully compatible with the combat systems of the 21st century. In 1991, AMCCOM provided more than 290,000 chemical protective masks and the Fox Nuclear, Biological, Chemical Reconnaissance System to United States troops during Operation Desert Storm.

The Armament Research, Development and Engineering Center. ARDEC, located at Picatinny Arsenal, New Jersey, is the second AMCCOM-maintained, advanced-technology facility. ARDEC performs research, development, and engineering on direct-fire, close-combat systems ranging from bayonets to tank cannons. The squad automatic weapon; Bushmaster 25-mm chain gun, used on Bradley Fighting Vehicles; and the M16A2 rifle are all systems developed by ARDEC. The center also works on indirect fire-support systems such as artillery and mortars, weapons and ammunition, mines, demolitions, and fire control for army weapons. ARDEC is the leader in the development of precision and "smart" munitions.

A ballistic test range at ARDEC is a safe, all-weather, environmentally acceptable facility for testing large-caliber ammunition. Technicians at the range can photograph a fired projectile, which aids in the study of its free-flight characteristics.

ARDEC's mission includes developing propellants, explosives, pyrotechnics, and fuzes, and supports the army and other services in armament science and technology. The center is also involved in liquid propellant and electromagnetic gun technology.

Rock Island Arsenal. Rock Island Arsenal, located at Rock Island, Illinois, is the largest weapons manufacturing arsenal in the North Atlantic Treaty Organization (NATO) alliance. It produces recoil mechanisms and gun mounts for most of the howitzers and tanks now fielded and manufactures the carriage for and completely assembles the M119 and M198 howitzers. During Operation Desert Storm in 1991, the arsenal received orders for nearly 18,000 components, 60% of which supported the M198 howitzer.

The arsenal produces a prototype 105-mm recoil mechanism and cradle for the air force's AC130U gunship, and manufactures critical castings and forgings for the navy. It also assembles and ships most of the army's common and special tool kits and basic-issue-item sets to troops worldwide.

Watervliet Arsenal. Watervliet Arsenal, located near Albany, New York, is the center for the production and procurement of cannons for tanks, howitzers, recoilless rifles, mortars, breech mechanisms, and spare parts. It houses a $15-million, computerized, flexible manufacturing system that is one of the most modern heavy-manufacturing facilities in the United States. This series of interconnected machining tools produces a finished breech block from a block of raw steel, using a unique rotary forge that turns out rough forgings in 10 minutes—rather than the previous 10 hours. Investments such as this have yielded the efficiency necessary for cost-effective, low-rate, peacetime production.

Pine Bluff Arsenal. Pine Bluff Arsenal, located at Pine Bluff, Arkansas, has primary responsibility for the production of smoke, incendiary, and riot-control munitions. Its mission also includes the assembly, production, and renovation of various protective masks used by all services. This arsenal was the only source of protective masks during Operation Desert Storm; it rebuilt and refurbished more than 3,500 masks per week. Pine Bluff Arsenal has also been the site for both the production and the demilitarization of chemical weapons.

Subsidiary Activities

AMCCOM's responsibilities continue after systems are produced or procured. The command remains

responsible for fielding, maintaining, and repairing materiel. Support in these areas is provided by the National Inventory Control Points and the National Maintenance Points located at command headquarters. The National Inventory Control Point for armament and chemical systems performs a variety of supply management functions ranging from data and inventory management to security assistance. More than 800,000 customer requisitions are processed yearly for equipment, repair, and secondary items (eg, a nonstandard radio antenna that was developed for special operations); the average processing time is less than 1 day. The National Maintenance Point for armament and chemical systems provides maintenance and engineering support, produces technical publications, conducts new-equipment training, prepares fielding plans, and manages maintenance and support activities. AMCCOM developed and shipped more than 78,000 copies of technical manuals to United States forces during Operation Desert Storm. The National Inventory Control Point, separate from the National Maintenance Point, manages ammunition and serves as the operational element of the *single manager* for conventional ammunition. (The single manager is a multiservice DoD concept; for example, AMEDD is the single manager for medical supplies.) These two organizations integrate materiel management, inventory management, maintenance engineering, fielding, malfunction investigation, and demilitarization of ammunition.

As the principal field-operating agency for the single manager for conventional ammunition, AMCCOM is responsible for the development and oversight of the budget for ammunition hardware, and for the procurement, production, supply, maintenance, and renovation of conventional ammunition for the other military services. For 1992, 80% of the AMCCOM production budget was allocated for ammunition.

The command is also responsible for the storage of 2.2 million short tons (ie, 2,000 lb; a long ton is 1,000 kg, or 2,240 lb) of ammunition, and for shipment and mobilization planning for all services. During Operation Desert Storm, AMCCOM (as the single manager) shipped more than 453,000 short tons of ammunition to troops in southwest Asia. Ammunition production is largely accomplished by AMCCOM's ammunition plant system. There are currently 27 such facilities, 16 of which are active. During the next 5 years, seven more plants will be inactivated At some inactive plants, private contractors are being granted permission to use portions of the facilities for non-DoD work.

Two plants are government owned and government operated: Crane Army Ammunition Plant (CAAP) in Indiana and McAlester Army Ammunition Plant (MAAP) in Oklahoma. CAAP produces ammunition for the navy and is the only facility capable of machining 16-in. battleship projectiles. It also has the world's only plant for converting obsolete white phosphorus munitions into phosphoric acid. MAAP produces ammunition (primarily for the navy and air force); renovates, maintains, and demilitarizes ammunition; and operates depots. The plant is capable of producing explosive-loaded bombs ranging from 500 to 2,000 pounds. During Operation Desert Storm, McAlester shipped more than 45,000 short tons of bombs to southwest Asia.

Most AMCCOM ammunition plants, however, are government-owned and contractor-operated, and perform four functions: small-arms production; propellant and explosive production; metal-parts production; and loading, assembling, and packing (LAP) of finished rounds. Technology at the command's ammunition plants ranges from hand assembly to state-of-the-art modern automated systems.

Some plants perform unique functions for the DoD. For example, Lake City Army Ammunition Plant (LCAAP) in Missouri currently produces virtually all the army's small-caliber ammunition. LCAAP shipped over 320 million rounds of ammunition to troops in southwest Asia during Operation Desert Storm. Longhorn Army Ammunition Plant (LAAP) in Marshall, Texas, was one of the demilitarization sites for Pershing missile rocket motors. This mission, performed in accordance with a Soviet-American arms-control treaty, was accomplished September 1988 to June 1991.[3]

Another AMCCOM mission is to support the central management of significant acquisition programs. Two project managers report directly to the commander of CRDEC and four others report directly to the commander of ARDEC. *Matrix* support (specialists from many organizations working together as a team) is provided to many Department of the Army (DA) program executive officer PEOs). Since the PEO concept's inception, AMCCOM has supported the PEO armaments, which are located at Picatinny Arsenal. Recently, AMCCOM expanded its support to PEOs, project managers, major subordinate commands, and other military services by establishing an industrial base acquisition advocate. As the command's focal point, the advocate assists in formalizing the use of the industrial base—the arsenals and ammunition plants—within acquisition strategies.

AMCCOM's other major, field-operating elements perform varied services. For example, the Central Ammunition Management Office, Pacific, located at Fort Shafter, Hawaii, provides centralized ammunition logistics management for the Pacific Theater. The U.S. Army Defense Ammunition Center and School

(USADACS), located at Savanna Army Depot Activity, Illinois, provides training and career management for ammunition managers and quality assurance specialists ammunition surveillance (QASAS), and technical assistance and engineering services for the storage and transportation of ammunition. During Operation Desert Storm, QASAS personnel ensured that ammunition was transported, stored, issued, and used safely in southwest Asia. They also provided surveillance during the *retrograde* of ammunition (the return to CONUS of forward-deployed ammunition). More than 100 QASAS personnel served OCONUS to support United States troops in southwest Asia, and another 42 provided stateside support at ports, camps, and stations.

USADACS administers the Technical Center for Explosives Safety, which executes the army's explosives safety policies, programs, standards, and procedures to provide maximum protection to people and property.

AMCCOM's Customer Support Directorate's mission is to provide customer and materiel support in all issues that affect the field readiness of weapons systems. The Readiness Analysis and Customer Feedback Center gathers, assembles, and analyzes readiness information received from the field. They maintain an accurate, current, technical-support database, and track and report emerging trends (which are then coordinated with functional directorates for resolution). Customer Support also plans and executes materiel findings, and provides training on new equipment to support force-modernization programs. They distribute new equipment, which is delivered ready for immediate field use. State-of-the-art video equipment enables new-equipment training teams to provide soldiers with maintenance training programs.

Soldiers and civilians from the U.S. Army Technical Escort Unit, located at Aberdeen Proving Ground, Maryland, can provide emergency response for chemical-munitions and -agent incidents worldwide. Unit members also escort all DoD chemical agents. During the return of forces from Europe after Operation Desert Storm, Technical Escort Unit members were instrumental in planning for and safely removing more than 100,000 chemical artillery projectiles from Germany to Johnston Island in the Pacific.

Office of the Army Mate' ommand Surgeon

AMEDD supports the AMC in three major areas: technical and professional input for specific AMC program areas, staff management of AMC medical programs, and delivery of healthcare services to military and civilian employees. Of the 41 AMEDD personnel assigned to the AMC, 33 have responsibilities primarily related to providing medical expertise for specific AMC program areas (Table 2-2). For example, the major duties of the occupational medicine officer assigned to the Program Management Office for Chemical Demilitarization include providing medical expertise for chemical demilitarization (eg, of surety agents). This occupational medicine officer is not responsible for providing healthcare services or managing health programs. Of the eight remaining medical personnel assigned to AMC headquarters staff, six are assigned to the Office of the AMC Command Surgeon, and one each is assigned as Command Surgeon for DESCOM and AMCCOM. Their duties are the same as the Office of the Command Surgeon, but on a smaller scale.

The AMC Surgeon is the principal officer responsible for advising the commanding general of the AMC and his or her staff on all health issues that affect the command (Figure 2-11). The command surgeon's office is also responsible for developing and implementing command policy, which assures the preservation of the health of the workforce and the prevention of disease and injury, addresses health issues within the health hazard assessment (HHA) process, provides for the control of pests and the preservation of war stocks, and ensures the medical portion of nuclear and chemical surety operations.

Most medical support for the AMC is provided by medical units not organic to the command. For example, the U.S. Army Medical Materiel Activity (USAMMA) provides logistics; the Medical Research and Development Command (MRDC) provides medical research and development; the U.S. Army Environmental Hygiene Agency (USAEHA) provides health consultation; and the Health Services Command (HSC) provides direct support (clinical, pharmaceutical, preventive medicine, and industrial hygiene). The creation of the HSC in 1973 greatly lessened the control of medical resources of both the AMC surgeon and installation commanders. All medical personnel and equipment were placed under the command of the newly designated medical centers (MEDCENs) and medical department activities (MEDDACs). Through these activities, the HSC operates 36 clinics and two hospitals on AMC installations (Table 2-3). The U.S. Navy supports one installation (Crane Army Ammunition Activity) and a contractor operates one clinic located at a depot (Corpus Christi Army Depot). Although on-site contractors may operate the clinics at the ammunition plants, the USAEHA provides occupational and environmental health services to these plants. However, the quality of healthcare that the

TABLE 2-2

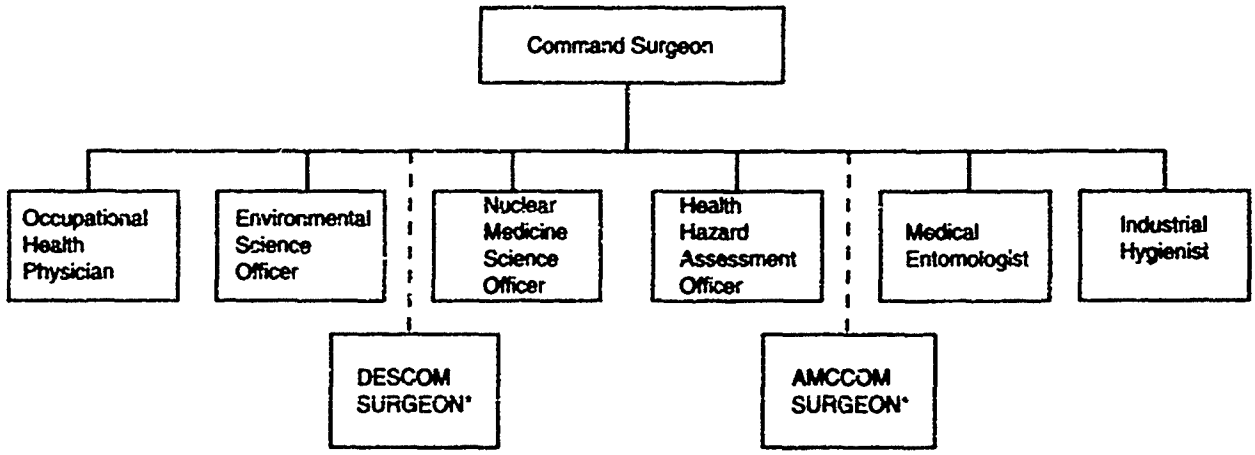
ARMY MEDICAL DEPARTMENT STAFF ASSIGNED TO THE ARMY MATERIEL COMMAND

Location	Number on Staff
HQ AMC	
Office of the Surgeon	6
Environmental Quality	3
AMCCOM	2
Edgewood Research, Development, and Engineering Command	5 (3 authorized)
AVSCOM/TROSCOM	1
Natick Research, Development, and Engineering Command	5 (7 authorized)
Belvoir Research, Development, and Engineering Command	1
CECOM	1
DESCOM	1
LABCOM	4
Materiel Testing Laboratory	1
TECOM	1
Cold Regions Test Center	1
White Sands Missile Range	1
Science Technical Center, Far East	1
Science Technical Center, Europe	1
Program Manager, Saudi Arabia National Guard	5
US Army Chemical Materiel Destruction Agency	1
TOTAL	41

contractor provides to his workforce is not a responsibility of the army. The physicians, nurses, industrial hygienists, and medical technicians, who provide direct patient care in clinical medicine and occupational health to the AMC workforce, report to a healthcare facility within the HSC. By monitoring these services and providing guidance regarding the command's medical policies and health priorities to HSC medical personnel and installation commanders, the AMC Surgeon is integral to occupational health.

It is detrimental to occupational health to use the same healthcare professional to provide both occupational health and clinical patient-care services. This doubling of responsibilities requires that a healthcare professional function both as staff officer and action officer. Because staff and action services are quite different, the mission would be better performed if the responsibilities were separated. Specifically, civilian and military physicians and nurses assigned to duties within the AMC usually have no special training in occupational health; however, they usually are very experienced in providing out-patient healthcare. Personnel untrained in professional occupational health who must perform staff functions (such as monitoring the effectiveness of occupational health programs and quality of healthcare) are put in the position of serving several masters (the installation commander, the MEDDAC commander, and the patient) whose interests could conflict. Furthermore, the current DoD system for assessing workload fails to provide any incentive for a medical staff to emphasize occupational health. Certainly, when the budget is stringent, personnel resources for direct patient care are more easily justified than those for occupational health; but then the installation commander, who is responsible for protecting the health of his or her employees, will have no one monitoring the programs related to occupational health.

Historically, the healthcare provided at AMC installations has been insufficient and fraught with problems. Most clinics are small and often are not staffed sufficiently to assure that both the primary healthcare



Position	Duties
Occupational Health Officer	Medical advice and consultation • Occupational and preventive medicine • Clinical medicine • Prophylactic immunizations • Human use research protocols Monitor health services provided by HSC for adequacy of services and staffing Training of resident physicians
Environmental Science Officer	Health consultation • Child development services • Food service and personal sanitation • Field and garrison water supply • Wastewater, solid and hazardous waste disposal • Health risk assessment in environmental contamination control • Hazardous waste minimization Occupational health partnership unit cost review AMEDD health services support AMEDD personnel management issues Administrative officer for AMC surgeon
Nuclear Medical Science Officer	Radiological health consultation • Ionizing and nonionizing radiation, including lasers • Exposure control • Waste disposal and contamination control • Decontamination • Health risk assessments • Human use research protocols Monitor AMEDD health services support Compliance oversight Nuclear surety inspections
Health Hazard Assessment Officer	Manage AMC Health Hazard Assessment program Health consultation • Health risks of developmental items • Health risks of fielded items • MANPRINT • Health representation of system safety and technical integration working groups Medical function area analysis
Medical Entomologist	Health consultation • Pest surveillance and control • Pesticide storage and disposal • Pesticide war reserves • Subsistence items development and storage Monitor AMEDD health support Monitor handoff and deployment of medically developed items
Industrial Hygienist	Industrial hygiene consultation • Workplace exposures • Hazard information • Hazard identification and control • Health risk Monitor AMEDD health services support Monitor compliance with OSHA, state, and army regulations Technical oversight of compliance monitoring Chemical surety hazard control

*The dotted connecting line denotes a noncommand technical supervisory relationship

Fig. 2-11. Structure of the Army Materiel Command AMEDD staff.

TABLE 2-3

HEALTH SERVICES COMMAND MEDICAL FACILITIES WITHIN THE ARMY MATERIEL COMMAND

Command	Facility	Command	Facility
AMCCOM	Rock Island Arsenal	DESCOM	Anniston Army Depot
	Pine Bluff Arsenal		Corpus Christi Army Depot
	Watervliet Arsenal		Letterkenny Army Depot
	McAlester Army Ammunition Plant		Savanna Depot Activity
			Red River Depot Activity
	Picatinny Arsenal		Lexington Blue Grass Army Depot
AVSCOM/TROSCOM	St. Louis (2)		Sacramento Army Depot
	Natick Research, Development, and Engineering Center		Seneca Army Depot
			Tobyhanna Army Depot
CECOM	Ft. Monmouth (hospital)		Tooele Army Depot
LABCOM	Harry Diamond Laboratories		Ft. Wingate Depot Activity
	Materials Technology Laboratory		Pueblo Depot Activity
			Umatilla Depot Activity
MICOM	Redstone Arsenal (hospital)	TECOM	Aberdeen Proving Ground
TACOM	Detroit Arsenal		Dugway Proving Ground
	Selfridge Air National Guard Bureau		Jefferson Proving Ground
			White Sands Missile Range
			Yuma Proving Ground

and the occupational health missions can be accomplished. During the spring of 1992, the commanding generals of the AMC and the HSC decided that entering into a new joint partnership was the only way to ensure that the occupational health services available are both appropriate and sufficient. This partnership provides for the AMC to contract with the HSC for specific occupational health services and then to transfer sufficient operating funds to the HSC clinic to provide adequate staffing. Brooke Army Medical Center (BAMC) at Fort Sam Houston, Texas, and the four AMC installations within the BAMC region (Corpus Christi and Red River Army Depots, McAlester AAP, and Pine Bluff Arsenal) will test this initiative.

MOBILIZATION AND DEPLOYMENT

Mobilization is the process of preparing for war or other emergencies by assembling and organizing national resources. It encompasses all activities necessary to move, systematically and selectively, from a normal state of peacetime preparedness to a wartime fighting posture. Deployment is the physical movement of a military force to the site of an actual or potential conflict. Both mobilization and deployment are resource intensive, especially of personnel. Any personnel-intensive action, especially one that must be performed expeditiously, requires medical support. Adequate and timely medical support can only be provided with appropriate planning.

How does the study of occupational health relate to mobilization and deployment? AMEDD personnel must understand the impact that total mobilization for war would have on occupational medicine. Mobilization during Operation Desert Storm was only partial; the United States has not been totally mobilized since World War II. Then, raw materials and food were rationed; jobs previously performed by healthy, experienced men were performed by women and men who were categorized 4F (partially disabled); and all major industries developed and produced materiel as rapidly, and in as great quantities, as possible. The army's current occupational health stance was developed during this period as a response to the need to keep the industrial workforce as healthy as is practicable.

Mobilization as a Deterrent

The capability of the U.S. Army to mobilize and deploy its active force rapidly and efficiently is essential to deterring potential enemies and ensuring our

allies of our support. Coordination of mobilization planning is crucial to achieving such a goal. Potential enemies must be convinced that the United States can quickly mobilize and deploy active- and reserve-component combat forces in sufficient time to influence the early stages of conflict. The reserve components must be visibly able to mobilize in the required time, at the right place, and in the state of readiness necessary to meet the threat. The DoD has the responsibility for developing and executing plans for national defense. The secretary of defense and the joint chiefs of staff provide guidance to the commanders of the unified and specified commands charged with developing the capability plans to meet various potential threats.

Mobilization includes options for deterring war and, should deterrence fail, for enhancing force readiness, deployment, and sustainment. Force sustainment includes both transporting and maintaining materiel and personnel (providing medical care). The complexity and magnitude of the mobilization process make sound planning essential. The DoD's Master Mobilization Plan provides the framework for making and implementing mobilization decisions to support military operations. It also provides guidance for detailing plans to support the mobilization process for the DoD. This plan assigns responsibilities and describes activities and organizational relationships and the DoD's planning and execution process. Simply stated, this plan describes what is to be done and who is to do it. How the various tasks are to be done is explained in detail in the subsequent levels of planning, except that tasks that require the secretary of defense's decisions are in the DoD plan. The joint chiefs of staff, the uniformed services, and the defense agencies all develop their own planning systems, consistent with DoD guidance. The entire mobilization planning process will eventually be linked by a mobilization planning management system.

Of course, mobilization planning is only one small part of the planning process. Of the several unified commands with geographical responsibility (the European Command [EUCOM], the Central Command [CENTCOM], the Southern Command [SOUTHCOM], and the Pacific Command [PACOM]), a large part of the headquarters responsibilities is to prepare the operation plans for the most probable war scenarios in their regions. These plans are comprehensive: they address not only tactical operations but also the deployment, movement, and sustainment of troops and materiel. Many military operations plans require personnel and materiel resources greater than the active forces can field. This is where mobilization plans come into play: plans to mobilize reserve units, field war-reserve equipment, and mobilize all or part of the United States's industrial base to build new equipment.

Mobilization of the Reserve Component

The deterrent value of the U.S. Army's reserve component is predicated on its combat power, readiness, and ability to mobilize and be deployed rapidly enough to influence the early stages of a conflict. An effective deterrent must be clearly evident to a potential enemy. The capability to mobilize rapidly is crucial to effective deterrence; it is the means by which political or military intent is translated into usable combat power. A ready mobilization capability demonstrates resolve and national commitment; it not only deters enemies but also assures allies that the United States has both the capacity and the will to stand by its commitments.

Readiness

The deterrent value of mobilization resides not only in the readiness of the active and reserve components but also in the resolve and preparedness of military and civilian leaders to rapidly expand the military capability of the United States. Civilian manpower must readily be converted into military units, and civilian industrial production must readily be converted to wartime industrial capacities.

The United States must, first, have the will to sustain an expanded or protracted war against numerically superior forces and, second, be able to conclude the conflict on terms favorable to this country and its allies. To accomplish these goals, the military establishment places great confidence in the capacity of this nation to redirect its civilian economy to expand and sustain military power through the readiness of its transportation system, war reserve stocks and strategic stockpiles, personnel, and support base.

Ready Transportation System. The United States must maintain civil and military land, sea, and air transportation systems that not only fulfill the nation's peacetime economic and military needs but are also capable of meeting and sustaining a mobilization surge. Constant study and planning must be conducted to ensure that new transportation facilities are provided and old systems rehabilitated in a manner that will enhance the nation's wartime mobilization transportation demands. The United States must have sufficient ground transportation to transport personnel and materiel to ports of embarkation within CONUS, and sufficient air and sea assets to transport them OCONUS to the area of conflict.

War Reserve Stocks and Strategic Stockpiles. The United States must maintain enough *war reserves* (military equipment) and *strategic stockpiles* (raw materials that are not produced domestically in sufficient quantity to meet wartime demands) of critical resources in peacetime to last from the time that initial stocks are consumed until the industrial base can convert to wartime footing and sustain the needed flow of weapons, ammunition, equipment, and other essential implements of war.

Personnel Resources. The United States must provide adequately for manned reserve components that are thoroughly trained and ready, with little or no notice, to engage in high-intensity warfare. It also needs enough pretrained individuals to fill active- and reserve-component units to wartime strength and to provide casualty replacements until the draft and the training base can provide trained personnel to sustain the wartime forces.

Ready Support Base. Mobilization, deployment, and sustainment depend on an effective support base in CONUS. The United States's capability to convert to a wartime footing must be continuously assessed, and remedial action must be taken where necessary to ensure the most-rapid conversion to a wartime posture. The support base includes

- support from federal agencies (such as the Selective Service System),
- expansion of the CONUS transportation,
- expansion of the CONUS medical base,
- expansion of the personnel system,
- expansion of installations,
- support to and expansion of the training base,
- industrial expansion, and
- increased capabilities of the reserve component.

The Mobilization Process

Premobilization

Premobilization is a general term, apparently without specific definition. Premobilization can be considered to reflect a state of readiness only after a degree of mobilization has been achieved. Military planners think of the U.S. Army as always in a state of premobilization. If international conditions or national emergencies warrant, the president of the United States can activate the *Presidential 200K Call-up*. This authorizes that individuals of the selective reserve—up to 200,000 members from all services—be mobilized for up to 90 days to meet the requirements of military contingency. Premobilization does not have a direct impact on the industrial base.

Levels of Mobilization

The magnitude of the emergency governs the level of mobilization. As authorized by law or congressional resolution, and when directed by the president, the DoD mobilizes all or part of the armed forces. Concurrently, the DoD and other federal agencies marshall the national resources necessary to sustain the armed forces.

Selective Mobilization. For a domestic emergency, either congress or the president can order expansion of the active armed forces by mobilization of reserve component units or individual ready reservists (IRRs), or both, if necessary to protect lives and federal property and functions, or to prevent disruption of federal activities. A selective mobilization normally would not be associated with the requirement for contingency plans involving internal threats to the national security.

Partial Mobilization. For a contingency operation or war plan, or upon the declaration of a national emergency, congress or the president can order augmentation of the active armed forces, short of a full mobilization, by mobilizing up to 1 million members of the ready reserve for up to 24 months.

Full Mobilization. Full mobilization requires that congress pass a public law or joint resolution declaring war or a national emergency. It involves the mobilization of all reserve component units, all IRRs, all skeleton units within the existing force structure, and the material resources needed to support and equip the expanded force structure.

Total Mobilization Total mobilization involves expanding the active forces by organizing or activating, or both, (*a*) additional units or personnel beyond the existing force structure and (*b*) all the national resources needed, including production facilities to complement and sustain such forces. The last time that the United States was totally mobilized was during World War II.

Legal Basis for Mobilization of Reserve Components

The authority to order mobilization resides with the president, the congress, or both. The secretary of defense, on the advice and recommendation of the service secretaries and the joint chiefs of staff, recommends that mobilization authority be granted to support a contingency (a situation for which no specific plan exists), an operation plan (a plan previously developed by an unified command and approved by the National Command Authority), or other national emergency. The secretary of defense directs the mobiliza-

tion of reserve component units and manpower through the respective military departments.

Extension of Appointments, Enlistments, and Periods of Service (Stop Loss)

Stop loss is the process by which the period of active-duty service of any member of the armed forces of the United States is extended involuntarily for the duration of a national emergency or declared war plus 6 months. There are three principal situations in which stop loss might be invoked:

- First, stop loss is automatically invoked and immediately effective if congress declares war or a national emergency. Enlistments are automatically extended. Reserve-component officers are appointed for an indefinite period and are held at the pleasure of the president. If congress is in session, the president must request congressional authority to extend enlistments in any situation other than a congressionally declared war or national emergency. If congress is not in session and the president decides that the national interest so requires, the president may authorize the secretary of defense to extend, for not more than 6 months, any individual's military status that expires before the 30th day after congress next convenes. (This process occurred during Operation Desert Shield.)
- Second, the joint chiefs of staff will recommend that stop loss be invoked for active-duty and reserve components when an involuntary mobilization of reserve components is being executed. Exceptions to this policy would be a presidential call-up of the national guard in a domestic emergency or a 90-day order for operational missions. Both these exceptions envision a very short term of active duty. State governments, however, may call up national guard units for local and state emergencies.
- Third, the need to draft soldiers through the Selective Service System must be considered in consonance with the mobilization of reserve components. Currently, the Military Selective Service Act provides that individuals can only be inducted as a result of an act of congress.[4]

Phases of Mobilization

For ease in describing the process, the phases of mobilization can be described as (*a*) peacetime planning and preparation (premobilization), (*b*) alert, (*c*) mobilization, (*d*) deployment, and (*e*) sustainment. As a continuous process, mobilization can proceed at a deliberate pace from peacetime preparation to mobilization (M-day), deployment (C-day), and the beginning of the contingency operation or hostilities (D-day). The pace would be dictated by the deteriorating international relations. Alternatively, the process can proceed at an accelerated pace, collapsing the interval between peacetime planning and preparation and D-day. In the event of a surprise attack on the United States or her NATO allies, the process would proceed rapidly and D-day would precede or coincide with M-day.

Alert, Mobilization, and Deployment

On receiving the order to mobilize, the army alerts the active force and simultaneously begins the selective, partial, or full mobilization of reserve-component units, manpower, and materiel within the approved force. The force, or portions of it, can augment an established theater of operations such as the European commands or, alternatively, can augment an emergency force such as the rapid deployment force (RDF) in contingency operations. In any case, under the general supervision of headquarters, DA, and using more than 50 available mobilization stations as necessary, active- and reserve-component units are brought to combat-ready status and are then deployed by air and sea to areas of operation. The services are responsible for providing healthcare support to the active mobilization stations. An already active medical unit can provide this support, or a reserve medical unit can be activated and assigned the task. This medical support is addressed in detail in the mobilization plan.

The initial resources sustain the deployed force until reinforcement and resupply can be affected, until the emergency is resolved, or until the industrial and training base assume a wartime footing. Active-component units that are in place in the theater of operations are called *forward deployed units*. Other active-component units, most of them CONUS-based, are earmarked to support one or more operational plans. Reserve-component units that are ordered to active duty can be earmarked either to support one or more operational plans or to become part of the CONUS base.

THE MOBILIZED INDUSTRIAL BASE

Current mobilization and operations plans primarily address the AMC's and AMEDD's support of deployed combat and combat-support units. It is possible that detailed documentation of contingency plans for AMC installations during total mobilization do not exist. Few if any plans describe how AMEDD will support the mobilized industrial base. What is known is that (*a*) most of the army's medical assets will be either deployed within the combat zone or assigned to CONUS medical facilities to care for evacuated casualties and (*b*) AMEDD personnel assigned to the AMC installations will be responsible for supporting the industrial workforce. If plans to support the mobilized industrial base—especially in the fields of preventive medicine and occupational health—are inadequate, then medical support to these installations is likely to be inadequate. Inadequate medical support could well make the difference between a healthy, efficient workforce and an unhealthy, inefficient workforce; it could, potentially, even make the difference between effective industrial operations that provide needed materiel support to the war fighters—and defeat. To provide this support we must know two things: what the mobilized industrial base will look like and what AMEDD can do to protect the health of the force.

Of this entire volume, the sections that follow were perhaps the most difficult to prepare. The difficulty was due, primarily, to the lack of pertinent, authoritative sources of information. Only two fairly adequate sources were found: (1) the transcript of a U.S. Army Preventive Medicine symposium held in September, 1987, which dealt with occupational health support for the mobilized industrial base; and (2) the report of a survey conducted by USAEHA industrial hygiene personnel at an inactive army ammunition plant in 1988. What follows is not a factual, detailed discussion of what the mobilized industrial base would be, nor what specific occupational health program services would be required. Rather, it is a general discussion of those factors that are likely to be important for consideration if AMEDD is called upon to support a fully mobilized industrial base.

For purposes of this discussion, the term *industrial mobilization* refers to the conversion of peacetime production capabilities to full-scale production of military items. Industrial mobilization would affect not only the AMC industrial base but also private-sector manufacturers of every type. Mobilization, if required today, would be characterized by

- radical changes in the qualitative and quantitative nature of processes and products;
- the use of old, out-dated, and possibly unsafe production processes;
- the development of new, untested methods and products; and
- major demographic alterations in the workforce.

Changes in Industrial Processes

Upon mobilization, AMC's government-owned, government-operated plants and government-owned, contractor-operated plants would increase production as rapidly as possible. Full production would require the lengthening of workshifts to as much as two 12-hour shifts, 7 days per week. Production would also be increased significantly by adding key production lines. A number of the plants already have floor space designated for these additional lines, and the production machinery is kept in storage.

Increased production would be accompanied by four important occupational health concerns. First, engineering controls already in place (especially exhaust ventilation) are probably designed for current production levels. Increasing the rate of production or the number of lines might exceed the capability of the controls to maintain airborne levels of potentially hazardous substances within acceptable limits. Second, increasing the number and length of workshifts would place workers at increased risk from both safety hazards (tired workers are more likely to be careless) and toxicological hazards (the workers are exposed for longer times). For example, during World War II, the push for increased production in ammunition plants led to increased exposure to toxic hazards (such as TNT) and significant increases in job-related diseases from that exposure. Third, stored equipment is less likely to have up-to-date safety features, and equipment operators would be less likely to be adequately familiar with its operation. And fourth, increased production and increased numbers of shifts would require a larger workforce, most of whom would be inexperienced and possibly inadequately trained, thereby increasing their risk of injury.

Mobilization would also have an impact on private-sector manufacturers. For example, automobile manufacturers in the United States have agreements with the United States government that they will produce military vehicles if requested. A mobilized automotive plant might begin to produce tanks. Some industries would be asked to make even more radical changes in production. For example, seven complete plant-equipment packages for the production of cannons are stored at various locations throughout the United States. During mobilization, these packages would be brought out of storage and set up at private-sector factories. Such radical changes in product would be likely to pose significant risks to the workforce.

The AMC maintains a number of mothballed ammunition and other production plants. These plants would probably be reopened if the United States were to enter into a protracted, full-scale war. The occupational health concerns associated with reopening older facilities are addressed in detail later in this chapter.

Development of New Technology and Products

The drive for increased production has always led to advances in technology and modernization of factories. When an adversary employs new weapons systems, nations either perish or respond by developing similar or better weapons and adequate defenses. Often, the need to develop new technologies quickly overrides the need to assure that safe and adequate processes and products are developed. For example, in response to Operation Desert Shield, the AMC stepped up the development of, and prepared to manufacture and deploy, more than 40 new major items. One of these was a lightweight, chemical-protection overgarment, developed in response to the possibility that chemical weapons would be used in a hot, desert environment.

Fortunately, processes were in place to thoroughly review the product's efficacy and safety before the garment was purchased and deployed (the HHA process, which is the subject of Chapter 6, Health Hazard Assessments). Had the garment been fielded without an HHA, we would have learned quickly that not only did it not provide adequate chemical protection, but in the desert heat it also provided no heat-stress advantage. Specifically, lightweight protective clothing needed to meet two requirements: (1) the protection provided needed to equal or exceed the currently fielded Battle Dress Overgarment and (2) the garment needed to provide at least a 20% reduction in heat stress in the desert environment. The recommended garment did neither.

The Expanded and Inexperienced Workforce

Total mobilization will change the nature of the workforce, much as it changed during World War II. Obviously, the size of the workforce would have to increase dramatically to staff additional shifts, new productions lines, and newly opened plants. Most of these new employees would be inexperienced and require job training. The degree of urgency for the product would be the primary factor influencing the time available to assure adequate job training. A largely untrained, inexperienced workforce would be likely to experience significant injury and illness rates if the occupational safety and health staff are not sufficiently prepared and vigilant.

The demographic characteristics of the workforce would also be changed. The selective service draft would probably be reinstituted, and during its initial stages, young, healthy males would be entering military service and not be available to the industrial base. A large number of older men and women, not necessarily eligible for the draft, would probably volunteer for military service, which would remove this population from the industrial base also. Thus, demographically, the remaining industrial workforce would be older, less healthy, and contain more women who, at least temporarily, would be additionally burdened by being single heads of households.

Medical Support of the Mobilized Industrial Base

All the characteristics of the mobilized industrial base described above would affect the need for quantitative and qualitative changes in the medical support provided by the AMEDD. Ideally, every AMC installation would have an up-to-date mobilization plan, as is required by Army Regulation (AR) 700-90, *The Army Industrial Base Program*.[5] Included in this plan would be a detailed discussion of anticipated changes in both processes and the workforce. Attached to this plan should be the HSC clinic's plan for supporting the mobilized installation. Unfortunately, this is probably not the case.

Although the nature of the occupational health services provided might change, the greatest impact would probably be in the amount of services needed. Each HSC clinic has what is known as a mobilization Table of Distribution and Allowances (TDA), which describes required staffing levels. Because the planning at AMC and HSC has not been well coordinated, some TDAs for clinics are likely to be inadequate, if not wholly unrealistic.

- The mobilization mission at one AMCCOM installation requires that it increase its patient load up to 3-fold. Its mobilization TDA calls for increasing medical officers by five. However, the same TDA increases the clerk typists by *only two.*
- Another HSC clinic, which could also be expected to increase its workload up to 3-fold, has a mobilization TDA that is *exactly the same* as its peacetime staffing.

Clearly, if total mobilization were to be implemented under these TDAs, it is highly probable that healthcare services provided by the installation medical facility would be substantially inadequate.

U.S. ARMY ENVIRONMENTAL HYGIENE AGENCY MOBILIZATION STUDY

To ascertain the potential for occupational health problems during mobilization, the USAEHA conducted a survey of an inactive U.S. Army ammunition plant in May 1988.[6] The survey is included in this textbook because it is the only report of its kind available. The findings that follow may or may not be representative of the problems that exist throughout the army's industrial base, or even at plants similar to the one surveyed. Even so, the report offers interesting insights.

Although the plant was classified as inactive, three production lines were in operation at that time. The primary mission of the plant was to manufacture metal parts for various types of ammunition, which most recently had consisted of 40-mm and 60-mm projectiles, 81-mm mortar bodies, and grenade bodies. The secondary missions included the operation and maintenance of active and layaway facilities; the procurement, storage, and issue of necessary supplies and materials; and industrial preparedness and mobilization planning.

The process flow of individual production lines was divided into two major areas—mortar shells/case cartridges and grenade bodies—although the production processes were very similar. The metal bar stock was first cut into billets, blasted with an abrasive material, and then heated in an induction furnace. The billets were then surface-treated in processes such as acid pickling or alkaline cleaning before they passed through a series of progressive forging operations. The metal parts then progressed through metal-machining and heat-treating operations before the final painting, inspection, and packaging phases. In addition to the process outlined, the grenade-body production line included machining operations with chlorinated oil, wet-vibratory deburring, and nondestructive testing, such as ultrasonic crack testing. The USAEHA survey team ascertained the potential health hazards associated with each phase of the ammunition production process (Table 2-4).

Survey Findings

To project problems in mobilization, the USAEHA had to make several assumptions before reaching any conclusions. The agency assumed that the ammunition plant's mission during mobilization would continue to be the manufacturing of metal parts for various types of ammunition. Of course, production rates would increase during mobilization from an 8-hour workday and 40-hour work week to a 10-hour workshift with two shifts per day. To adequately protect workers from overexposures, permissible exposure levels (PELs) had to be calculated to compensate for the increased production rate. The agency also assumed that federal and DA occupational safety and health regulations would not be waived during mobilization. These assumptions directed the USAEHA to make several discoveries relating to (*a*) personal protective equipment (PPE), (*b*) the respiratory protection program, (*c*) the hazard communication program, (*d*) the asbestos management plan, (*e*) the hearing conservation program, and (*f*) occupational health and industrial hygiene support.

Personal Protective Equipment

The survey reported that those who worked on the active production lines were provided applicable PPE such as safety glasses, steel-toed shoes, and hearing protection. If mobilization were to occur, the increased workforce would also require PPE, the assignment of which presumes medical evaluations and clearances and necessitates a formal training and fitting program.[7] Without the services of adequate planning and support personnel such as occupational health professionals, the potential for mobilized workers to be assigned tasks without first receiving proper PPE, training and fitting instructions, or medical evaluation increases.

TABLE 2-4

ANALYSIS OF HAZARDS IN AN AMMUNITION PLANT

Operation	Potential Health Hazard
Abrasive Blasting	
Blasting parts with steel shot	Total nuisance dust Total respirable dust Various metals and oxides Noise
Dip-Tank Cleaning	
Treating parts in H_3PO_4 solution	H_3PO_4 mist
Surface Preparation	
Surface treating parts in H_2SO_4 or NaOH	H_2SO_4 NaOH
Plating	
Treating parts in H_2CrO_4 solution	H_2CrO_4 mist
Metal Finishing	
Vibrating parts in abrasive stone and weak alkaline water solution	Various metals and oxides Noise
Painting	
Painting parts (electrostatic, brush, or roller) with lead and chromate primer, top coat (lacquer, enamel), and drying in oven	Chromates Lead Noise Various solvents
Forging (Cold or Hot)	
Forging parts with hydraulic presses	Noise Heat stress Oil mists
Heat Treating	
Annealing parts in liquid-propane gas furnace containing an N_2 atmosphere, and may also contain various salts of $CaCl_2$, Ba, Na, or K	NOx Heat stress CO Soluble Ba compounds Asbestos Crystalline silica
Induction Heating	
Heating parts in electric furnace	Heat stress
Machining	
Machining parts using cutting fluids	Noise Oil mists Metal Ox
Shearing	
Shearing or cutting metal bar stock	Noise
Part Lubrication	
Coating parts with graphite lubricant	Graphite
Phosphate coat	
Coating parts with $Zn_3(PO_4)_2$ or Na_3PO_4	None noted
Nondestructive testing	
Ultrasonic testing of metal parts	None noted

Source: US Army Environmental Hygiene Agency. *Industrial Hygiene Mobilization Survey. Riverbank Army Ammunition Plant, Riverbank, Calif.* Aberdeen Proving Ground, Md: USAEHA; 1988. Survey 55-71-0033-891.

Respiratory Protection Program

The ammunition plant maintained a respiratory protection program, which would require drastic expansion in resources on mobilization. However, if engineering controls similar to those on active production lines were to be operational throughout the facility, respirators would probably not be required. Prior to this determination, sampling would be imperative to document airborne contaminant exposure levels. No comprehensive airborne sampling had ever been conducted to demonstrate workplace exposures. During the survey, the local exhaust ventilation systems on the inactive production lines were found to be either absent or not in operable condition due to frozen bearings, removed belts, or disconnected electrical power. Much of the equipment had been cannibalized or removed and placed in storage.

Hazard Communication Program

In accordance with Occupational Safety and Health Administration (OSHA) regulation 29 CFR 1910.1200, *Hazard Communication*, employers are required to fully inform every employee who is potentially exposed to a workplace hazard of all that is known about that hazard.[8] This is the essence of hazard communication. The ammunition plant contractor had ensured that the air-pollution-control technician provided plant workers with hazard communication training. However, just as the respiratory protection program would require more attention during mobilization, so the hazard communication program must also expand with increased workforce and production rates. To maintain compliance with OSHA's hazard communication standard, frequent training must be conducted both as new workers enter the workplace and as increased production increases the possible hazards.

Asbestos Management Plan

No written asbestos management plan was available at the plant during the survey. Although material suspected of containing asbestos was observed within both active and inactive areas, it appeared to be contained and nonfriable. However, suspect asbestos-containing material found outdoors on above-ground steam and water lines was damaged and friable.

Hearing Conservation Program

No master list or diagram of hazardous noise areas within the plant was available during the survey. This was unfortunate because this document would have aided significantly in estimating the resources required for a hearing conservation program (for example, the type, amount, and cost of hearing protective devices necessary; and the number of potentially exposed workers requiring training and medical surveillance). The accumulation of noise data to establish a historical record of noise levels must be accomplished over time, as inactive lines are activated. In the interim, extrapolations can be made between similar active and inactive operations to predict hazardous noise areas.

Occupational Health and Industrial Hygiene Support

The agency discovered that the contract between the DA and the contractor, as well as the industrial readiness plan (IRP), the industrial preparedness plan (IPP), and the mobilization master plan (MMP) lacked stipulations for industrial hygiene and occupational health services.[9,10]

Survey Recommendations

Based on survey findings and predicted mobilization requirements, the agency provided several recommendations for the ammunition plant to help protect both contractor and government personnel, and to ensure regulatory compliance. Although government contractors are obligated to adhere to federal regulations, the USAEHA maintains that all applicable U.S. Army regulations, as specified in the recommendations that follow, be incorporated in the contract:

- Address the availability, procurement, and issue of applicable PPE, and required training, fitting and medical evaluation of personnel in mobilization plans and timetables, as required by Title 29, Code of Federal Regulations (CFR), Section 1910.132(a), *Personal Protective Equipment*[11]; and AR 385-10, *The Army Safety Program*.[7]
- Perform industrial hygiene evaluations and document air sampling of the inactive production lines, once they are activated for mobilization or premobilization, to identify the need for PPE and engineering controls, as maintained by Title 29, CFR, Section 1910.1000(e), *Air Contaminants*[12]; AR 40-5, *Preventive Medicine*[13]; and Technical Bulletin, Medical (TB MED) 502, *Occupational and Environmental Health Respiratory Protection Program*.[14]
- Perform occupational noise evaluations of inact. production lines, once they have been

activated for mobilization or premobilization, to establish a master list and a diagram of hazardous noise areas and to evaluate PPE and engineering controls, as maintained by Title 29, CFR, Section 1910.95(c) and (d), *Hearing Conservation*[15]; AR 40-5[13]; and TB MED 501, *Occupational and Environmental Health Hearing Conservation*.[16]

- Establish an asbestos-management program to preclude both worker exposure and asbestos-fiber release into the environment, as maintained by Title 29, CFR, Section 1910.1001, *Asbestos, Tremolite, Anthophyllite, and Actinolite*[17]; and TB MED 513, *Occupational and Environmental Health Guidelines for the Evaluation and Control of Asbestos Exposure*.[18]
- Modify the existing contract between the DA and the contractor to include occupational health support, as maintained by AR 40-5.[13]
- Maintain engineering controls that protect the workers' health.
- Modify the AMCCOM 319-R, *Current or Backlog of Deficiency Identification and Industrial Preparedness Measure of the IPP*,[19] to include a more specific breakdown of occupational safety and health deficiencies into two separate categories: occupational safety and industrial hygiene.
- Modify the IPP mobilization manpower requirements to include clerical support for the government safety office, and staffing requirements for industrial hygiene, optometry, occupational health nursing, clinical laboratory X-ray technicians, and occupational health professionals.
- Modify the responsibilities of the government safety office and the contractor's health and safety administration office in the IPP to include industrial hygiene services.

Survey Implications for Mobilization Preparation

According to the USAEHA, occupational health support is absolutely necessary during mobilization. The need to maintain a viable workforce to meet production quotas, while complying with federal and DA health and safety regulations, requires a professional staff supporting a comprehensive occupational health program. The survey found potential occupational health problems that are common for recently mobilized depots and army ammunition plants For example, the ammunition plant's industrial preparedness plan indicated that safety and health mobilization manpower requirements for government personnel included a chief safety manager and two safety specialists. However, manpower for contract personnel included a safety and health administrator and a nurse. Surprisingly, the plan did not identify the need for an occupational health physician, an occupational health nurse, industrial hygiene support, or even clerical support for the safety office.[10]

Occupational Health Manpower Indicators

The amount and type of occupational health professionals necessary to support mobilization are partly dependent on the installation's population (Table 2-5). The occupational health professionals necessary to support mobilization of an ammunition plant include occupational health physicians, occupational health nurses, optometrists, and industrial hygienists. Quantifying the exact number of occupational health professionals necessary to support operations also depends on other variables, including (*a*) the estimated workforce, (*b*) the occupational health regulations in effect, (*c*) the production and control processes, and (*d*) the requirements for raw materials and *end products* (the finished products).

Workforce. The composition of the workforce determines the amount of time per worker required of each occupational health profession. Industrial operators, for example, will require more preemployment medical screening than a clerk-typist, whose physical condition to withstand the physical and chemical insult of the metal-working industrial environment is not a determinant of the ability to perform clerical duties. Likewise, the industrial hygienist will spend more time with industrial and production workers than with administrative personnel.

Occupational Health Regulations. Occupational health regulations tend to become more numerous and complex; there is no indication that this trend will slacken in the future. The more stringent and numerous the regulations become, the more occupational health support will be required to maintain existing programs such as hazard communication, medical surveillance, evaluation of engineering controls, and PPE, and any programs dictated by future regulations. Because these regulations cannot be waived during mobilization, the number of occupational health professionals needed to maintain the regulations' directives are a factor in determining manpower requirements.

Production and Control Processes. Production and control processes at an ammunition plant may range from inactive production lines with cannibalized or malfunctioning engineering controls to active production lines with adequate engineering controls. The condition of the production and control processes also, in part, determine the level of occupational health

TABLE 2-5

OCCUPATIONAL HEALTH PROFESSIONALS REQUIRED PER AMMUNITION PLANT

Position	Installation Population	Installation Population Increase Determining Additional Position
Part-time occupational health physician*	< 2,000	—
Full-time occupational health physician*	> 2,000	3,500
Full-time occupational health nurse*	> 350	750 – 1,000
Part-time optometrist*†	< 10,000	—
Full-time optometrist*†	> 10,000	—
Full-time clinical laboratory/X-ray technician‡	> 2,500	—
Full-time industrial hygienist‡	> 1,000	1,000 – 1,250
Full-time industrial hygiene technician‡	> 750	750 – 1,000
Full-time clerk/typist	—	> 1,000

*Source: Zenz C, ed. *Occupational Medicine, Principles, and Practical Applications*. Chicago: Year Book Medical Publishers, 1984: 77–82.
†Source: Brown ML. *Occupational Health Nursing, Principles, and Practices*. New York: Springer Publishing Co; 1981: 224–226.
‡Source: USAEHA unpublished data.

support necessary. With continuing developments in new end products and manufacturing techniques, the level of occupational health support will be influenced by the production and control processes existing at the time of mobilization.

Raw Materials and End Products. Like production and control processes, the types of raw materials and end products affect the level of occupational health support required. Manufacturing a new end product means changes in raw materials and processing techniques, which, in turn, alter the types of contaminants generated. The types and levels of health hazards associated with the raw materials and contaminants generated in manufacturing help to determine occupational health manpower requirements.

Mobilization Duties

Not only are occupational health professionals necessary, the USAEHA also suggests that they be incorporated into the mobilization workforce as early as possible. If full-time occupational health services are not necessary at a specific installation, or are time and cost prohibitive, the USAEHA recommends using independent, or fee-for-service, occupational health professionals from the local community. It is also imperative to consider, even before mobilization, the clerical and technical support, equipment, analytical and chemical laboratory support, X-ray capability, installation transportation, and other services and supports necessary for occupational health professionals to accomplish their jobs. Even before operations begin, an industrial hygienist should make a preoperational inspection of the industrial facility to determine the occupational health hazards and requirements. This process would include a facility walk-through, where the industrial hygienist—with a list of raw materials, intermediate products, final products, byproducts, and hazardous materials—scrutinizes product flow, equipment placement, and every other aspect of the industrial operation. Occasionally, all machinery is in place and operable, but at other times, some of the equipment is in storage or is inoperable; then the industrial hygienist must attempt to visualize it as a fully functioning operation with all its equipment in place. Nevertheless, the industrial hygienist must first determine the adequacy of engineering controls to protect workers from occupational exposures and then, if necessary, select the necessary PPE. While the facility is being readied for production, occupational health professionals need to (*a*) provide new employees with preemployment medical evaluations where baseline data are required, (*b*) train and fit-test for necessary PPE, and (*c*) train for chemical hazard communication, as authorized by OSHA.

Once the facility is operational, it will still take time for the occupational health professionals to assimilate the specifics of the industrial operation. Industrial hygiene personnel, for example, will require firsthand knowledge of the operation's physical-agent sources such as noise, vibration, and heat energy; and chemical-agent sources such as gases, mists, and vapors. This knowledge is necessary to evaluate the effectiveness of existing engineering controls, develop abatement actions, and analyze administrative controls to further reduce exposure.

SUMMARY

The U.S. Army must be able, at a moment's notice, to defend the nation and support its foreign policy; the army must also be able to mobilize and deploy its reserve component. This mission cannot be taken lightly. The U.S. Army operates one of the largest industrial bases in the nation, most of which is under the command and control of the AMC. Divided into 10 subordinate commands, the AMC has more than 100,000 employees, and operates more than 40 industrial installations throughout the world.

The responsibility for providing adequate medical support to the army's industrial base falls squarely on the shoulders of AMEDD. Adequate support can only be provided if the services are based on a thorough evaluation of installation needs. Medical personnel must understand the organizational structure of the army's industrial base and the nature of the industrial processes. At the installation level, assigned medical-staff personnel must know who the workers are and what they do. Only through a full understanding of both worksite and workers can cost-effective, appropriate occupational health services be provided.

Should the United States ever again be totally mobilized for war, the army's industrial base will also mobilize. This means that government-owned and -operated plants will dramatically increase production, mothballed plants will be brought into production, and private-sector industries will begin to produce military equipment. All these actions will result in a workforce that is substantially increased, inexperienced, and demographically different—a workforce quite likely to be exposed to new and more significant job-related hazards. If AMEDD is to fulfill its mission of providing adequate occupational health services and protecting the health of army workers, we must anticipate the needs of the mobilized industrial base, know how to respond to those needs, and develop superior occupational health plans and programs that ensure adequate services.

REFERENCES

1. US Department of the Army. *US Army Armament, Munitions, and Chemical Command (AMCCOM) Briefing*. 05 March 1992.
2. US Department of the Army. *US Army Depot Systems Command (DESCOM) Fact Sheet*. US Army Depot System Command. 01 May 1991.
3. US Senate. *USA–USSR Intermediate Range Nuclear Forces (INF) Treaty*. Washington, DC: 100th Congress, 2nd Session. 1988. US Senate Treaty Document No. 100-11.
4. *Military Selective Service Act of 1967*. Pub L No. 90-40. Washington, DC: 90th Congress, 1st Session.
5. US Department of the Army. *The Army Industrial Base Program*. Washington, DC: DA; 1992. Army Regulation 700-90.
6. US Army Environmental Hygiene Agency. *Industrial Hygiene Mobilization Survey* [55-71-0033-891]. *Riverbank Army Ammunition Plant*. Riverbank, Calif: USAEHA; 1988.
7. US Department of the Army. *The Army Safety Program*. Washington, DC: DA; 1988. Army Regulation 385-10.
8. 29 CFR, Part 1910 § 1200.
9. US Army Materiel Command. NI Industries, Inc. Riverbank, Calif: Riverbank Army Ammunition Plant; 1984. DA Contract No. DAAA 09-85-Z-0001
10. Riverbank Army Ammunition Plant. *Industrial Preparedness Plan*. Riverbank, Calif: Riverbank AAP; 1987.
11. 29 CFR, Part 1910 § 132(a). Subpart I.
12. 29 CFR, Part 1910 § 1000(e).
13. US Department of the Army. *Preventive Medicine*. Washington, DC: DA; 1990. Army Regulation 40-5.
14. US Department of the Army. *Occupational and Environmental Health Respiratory Protection Program*. Washington, DC: DA and Defense Logistics Agency; 1982. Technical Bulletin MED 502.

15. 29 CFR, Part 1910 § 95(c).

16. US Department of the Army. *Occupational and Environmental Health Hearing Conservation.* Washington, DC: DA; 1980. Technical Bulletin MED 501.

17. 29 CFR, Part 1910 § 1001.

18. US Department of the Army. *Occupational and Environmental Health Guidelines for the Evaluation and Control of Asbestos Exposure.* Washington, DC: DA; 1986. Technical Bulletin MED 513.

19. US Department of the Army. *Current of Backlog of Deficiency Identification and Industrial Preparedness Measure (IPM).* DA, AMCCOM 319-R; 1986.

Chapter 3

U.S. ARMY HEALTH PROGRAMS AND SERVICES

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SUMMARY

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INTRODUCTION

Soldiers are considered to be military employees; when they perform work that is separate from their combat duties, they frequently encounter potentially hazardous exposures that are similar to civilian industrial exposures. When soldiers are thought of in this context, the military must address the potentially harmful effects of the work that soldiers do, and the conditions under which they work, just as these are addressed for civilian employees in the federal government and private-sector work force.

Employee health programs and services focus on two interrelated aspects: (1) to prevent or reduce negative interactions between a job and an employee's health, and (2) to provide clinical services to employees. However, differences exist between military and civilian employees in both their eligibility for health services and the manner in which the services are provided. Soldiers are eligible for health services at a medical treatment facility (MTF) for all illnesses and injuries regardless of the cause. Department of the Army (DA) civilians are eligible only for job-related health services and emergency and palliative treatment. Although the Army Medical Department (AMEDD) provides all of the healthcare to soldiers (including employee health services), the only services that are available to civilian employees are those specifically provided in the context of occupational health.

Before 1974, assorted health clinics, occupational health sections, and nursing offices were established by most army installation commanders to provide health services to civilian employees of the army. However, in 1974, the Health Services Command (HSC) was activated and all of the installation clinics, nursing offices, and occupational health sections and programs were incorporated into the local HSC activity. The responsibilities for managing all the aspects of civilian employee health were given to army Preventive Medicine Services within the various HSC activities.

LAWS, REGULATIONS, AND GUIDANCE

Several laws and regulations protect the health and promote the effectiveness of all federal employees—military and civilian—including (*a*) Public Law 79-658, (*b*) the Occupational Safety and Health Act (OSHAct) of 1970, and (*c*) DA regulations. Technical guidance does not carry the same weight as a law or a regulation; it is professional advice published to aid occupational health providers and managers.

Public Law 79-658

The first law that authorized health services for federal civilian employees was Public Law 79-658, entitled *Health Promotion for Government Employees* (also known as 5 United States Code 7901, 1946 as amended). This law authorized, but did not require, federal agencies to establish health-service programs to promote and maintain the physical and mental fitness of their employees within the limits of their appropriations. Public Law 79-658 limited employee health services to

- the treatment of on-the-job illnesses,
- the treatment of dental conditions that require emergency attention,
- preplacement and other job-related health-maintenance examinations,
- the referral of employees to private physicians and dentists, and
- preventive programs related to health.

The Occupational Safety and Health Act

The Occupational Safety and Health Act of 1970 (Public Law 91-596, 29 United States Code 651 *et seq.*) succeeded Public Law 79-658 in addressing occupational health for federal employees. This law requires that all employers provide a safe and healthy working environment for all of their employees. Although the law originally exempted federal employees (both within and without the Department of Defense [DoD]), later provisions included them. For example, Executive Order 12196 (*Occupational Safety and Health Programs for Federal Employees*) and Title 29, Code of Federal Regulations Part 1960, *Basic Program Elements for Federal Employee Occupational Safety and Health Programs* state that (*a*) OSHAct applies to all federal government agencies and (*b*) the Occupational Safety and Health Administration (OSHA) requires federal agencies to establish an occupational safety and health program. Department of Defense Instructions (DoDIs) also direct implementation of the occupational safety and health program for military and civilian DoD employees.

Federal Employees' Compensation Program

The Federal Employees' Compensation Act was passed in 1916; it provides compensation benefits to federal civilian employees for disabilities caused by personal injury or disease that are sustained while job-related duties are being performed. The 1916 Act also provides for the payment of benefits to the employee's dependents if the work-related injury or disease causes the death of the employee. The provisions of the Act and information concerning the administration of the workers' compensation program are contained in *Title 20, Code of Federal Regulations.*[1]

The Office of Workers' Compensation Programs of the U.S. Department of Labor is responsible for administering workers' compensation programs for federal civilian employees. However, two divisions of this office actually administer the program: (*a*) the Division of Federal Employees' Compensation administers the program for *appropriated-fund* employees (that is, those employees who are primarily paid from the monies that Congress appropriates to run the agency); and (*b*) the Division of Longshoremen's and Harbor Workers' Compensation administers the compensation program for *nonappropriated-fund employees* (that is, employees—generally civilians—who work for activities that create their own income by providing services).

Workers' Compensation for Appropriated-Fund Employees

The *Civilian Personnel Office* at military installations is usually responsible for administering the workers' compensation program at the local level for appropriated-fund employees. However, commanders, supervisors, safety personnel, physicians, and nurses all play a role in administering the program. The DA's main objective for the program is to provide—as promptly as possible—all of the benefits to which an ill or injured civilian employee is entitled. The army also is concerned that employees who have recovered from their injuries, either partially or completely, return to work. All army installations are required to establish a light-duty program to accommodate those injured employees who may not be able to work at their regular jobs, but who may perform light-duty tasks. In addition, procedures must be established to bring long-term disabled employees back to work.[2]

The workers' compensation program has other benefits for the appropriated-fund employee. It (*a*) provides for the medical care necessary for the treatment of job-related injuries or illnesses, (*b*) contains a stipulation for the continuation of pay following a traumatic injury, and (*c*) allows for the injured employee to choose a physician.

Under the Federal Employees' Compensation Act, an employee's regular pay can be continued for up to 45 calendar days after a traumatic injury when disability, medical treatment, or both, occur. (A traumatic injury is defined by the Office of Workers Compensation Programs as a wound or other condition of the body that is caused by an external force, including stress or strain.) After the entitlement to continuation of pay is exhausted, the employee may apply for compensation if additional time is needed to recover from the traumatic injury.

The Federal Employees' Compensation Act also entitles an employee to choose among all licensed physicians in private practice or physicians at a federal medical treatment facility. Under this stipulation, the term *physicians* also includes podiatrists, dentists, clinical psychologists, optometrists, and chiropractors; each medical officer must practice within his or her specialty as it is defined by state law. In an effort to reduce civilian workers' compensation claims, the army encourages injured civilian employees to be evaluated initially at a federal MTF and to be treated there if the resources are available. These government facilities are staffed by active-duty medical officers and civilians, and include hospitals of the army, navy, air force, and Veterans Administration. However, when an employee chooses to be treated by a private physician, the staff of the Employee Health Program should monitor the progress of treatment and perform a follow-up examination when the employee returns to work.

The workers' compensation program also provides medical care benefits. These benefits include the examinations, treatments, hospitalizations, medications, appliances, supplies, and transportation that are necessary to obtain adequate medical care.

Workers' Compensation for Nonappropriated-Fund Employees

The Nonappropriated Fund Instrumentalities Act of November 1958 (now known as United States Code 8171-8173) provides workers' compensation coverage for nonappropriated-fund employees under the Longshoremen's and Harbor Workers' Compensation Act. Workers' compensation benefits are provided by a self-insured workers' compensation program managed by the U.S. Army Central Insurance Fund.[3] The services that are provided to nonappropriated-fund employees are similar to those that are provided to appropriated-fund employees under the Federal Em-

ployees' Compensation Act. However, the army MTF responsibilities are limited to providing initial and emergency care without charge; referral to community medical resources occurs if and when further medical care is required as determined by the physician.

Workers' Compensation Claims Procedures

Employees and federal agencies must use specific forms and procedures to initiate claims for traumatic injury, occupational disease, recurrence of disability, and death.[4] Most of the forms include a statement of the purpose of the form, directions for completing and submitting the form, the party responsible for its preparation, the date by which it must be submitted, and to whom it must be submitted.

Regulations and Guidance Applicable to Employee Health

The primary Employee Health Program laws and regulations are listed in Exhibit 3-1. Other publications that pertain to the program are listed in the recommended reading at the end of this chapter.

To ensure effective management of the Employee Health Program, the staff must also develop and maintain administrative documents. A good administrative structure is basic to providing effective employee health services and managing the program.

In addition to the laws and regulations that have already been discussed, other army directives include (*a*) an installation occupational health regulation, (*b*) an occupational health program document, (*c*) standing operating procedures (SOPs), and (*d*) medical directives for occupational health nurses.

Installation Occupational Health Regulations

The installation occupational health regulation, based on the documents in Exhibit 3-1 and published army policies, defines policy and instructions as they apply to that particular installation.[5] At a minimum, this regulation should define the extent of the occupational health program, the eligibility for its services, and the responsibilities of (*a*) the occupational health staff to provide employee health services at the local level, (*b*) all installation occupational health participants such as the safety and civilian personnel officers, and (*c*) the employees who receive the services.

During the development of the regulation, the draft must be passed through all the activities and divisions that have a designated occupational health responsibility. After the local regulation is published, it must be kept current through periodic reviews and updates. Either the occupational health physician or the occupational health nurse or both must contribute to any installation directive that involves occupational health, from its initial development through the staffing stages.

Program Document

Commanders of Medical Centers (MEDCENs) and Medical Department Activities (MEDDACs) are responsible for publishing an occupational health pro-

EXHIBIT 3-1

PRIMARY EMPLOYEE HEALTH PROGRAM REGULATIONS

Department of Labor, Occupational Safety and Health Administration
Title 29, Code of Federal Regulations, Part 1910, *Occupational Safety and Health Standards*[*]

Department of the Army and the Health Services Command
Army Regulation 40-5, *Preventive Medicine*
Army Regulation 40-3, *Medical, Dental, and Veterinary Care*
Health Services Command Regulation 40-30, *HSC Operating Program—Preventive Medicine Program for MEDCEN/MEDDAC*
Health Services Command Regulation 10-1, *Organizations and Functions Policy*

Office of Personnel Management
Federal Personnel Manual, Chapter 339, Medical Qualification Determinations
Federal Personnel Manual, Chapter 792, Federal Employee Health and Counseling Programs
Federal Personnel Manual, Chapter 810, Injury Compensation
Federal Personnel Manual, Supplement 293-31, Basic Personnel Records and Files System

[*]Published annually

gram document that identifies the preventive medicine services that are available.[6] The document should include the program objectives, target dates for the accomplishment of each objective, methods to achieve the objectives, and an evaluation plan. As an addendum to the Preventive Medicine Services general program document, good management practice dictates that the employee health staff develop and review the Employee Health Program section of the document annually. The Employee Health Program plan and objectives must be consistent with the overall installation and MTF mission, priorities, and resources.

Frequently, the manager of the Employee Health Program will be called upon to (*a*) defend the program against reductions in the budget, personnel, and space; (*b*) justify requests for more resources; and (*c*) assure the commander that the Employee Health Program is helping to accomplish the installation's mission. An up-to-date, meaningful program document, representing both a plan for the future and a report on past performance, will be invaluable in these situations. The successful development and management of a continuing Employee Health Program depends not only on the quality of the program document but also on the extent to which it is followed.

Standing Operating Procedures

The basic management tools for the Employee Health Program, SOPs consist of a written set of instructions and detailed step-by-step operational procedures for accomplishing an organization's specific tasks. An SOP is an internal document and includes only the steps that employees in the immediate organization perform. SOPs assist in training new employees and serve as continuity tools in instances when regular personnel are absent, enabling others to carry on the operation. To be of value, SOPs must be used by the personnel for whom they were intended and updated at least annually.

Medical Directives

Medical directives are the physician's written orders to the occupational health nurse for administering treatment in the physician's absence. Directives must include the steps to follow in providing emergency care and must list the treatments of occupational and nonoccupational illnesses and injuries. Written medical directives are required to assure that emergencies are properly handled in the absence of a physician, to direct medical care for minor incidents that do not require the services of a physician, and to authorize other nursing staff activities such as changing a dressing.

The written medical directives must include instructions for the occupational health nurse to administer *only* noncontrolled pharmaceuticals as a one-time dose (when a nonprescription drug is the treatment of choice). The drugs, selected from a list of only nonprescription drugs, are authorized by the local Therapeutic Agents Board. Instructions for prescription drugs are not included in medical directives because it is not within the purview of usual occupational health nursing practice to administer prescription drugs in the absence of a physician.

Medical directives should be consistent with the anticipated requirements for employee health services and the capabilities of the nursing staff. Thus, the physician should coordinate with the occupational health nurse manager and supervisor to prepare the directives. The physician must sign and date these directives.[5] Occupational health nurses who participate in the preparation of medical directives should cosign them.

Clinical guidelines require periodic review and revision of the directives both as medical knowledge increases and as other changes occur at the installation. At a minimum, the occupational health physician and nurse must review the medical directives annually, and indicate this review with their signatures and the review date.[5]

ORGANIZATION OF THE ARMY'S OCCUPATIONAL HEALTH PROGRAM

The army operates more than 130 individual Employee Health Programs. These programs' missions and structures vary from installation to installation because each installation has specific employee-health needs. There are, however, a few basic designs for these programs.

The position of the Employee Health Program within an organization depends on whether it is collocated with a MEDDAC or MEDCEN, both of which include a hospital and a Preventive Medicine service, or with an Army Health Clinic (AHC), which is located at an installation without a hospital and has no Preventive Medicine service. HSC Regulation 10-1, *Organization and Functions Policy*, prescribes the organization and the functions of AMEDD activities under the command Headquarters, U.S. Army Health Services Command; HSC Regulation 40-5, *Ambulatory Patient Care*, further defines the operations of the AHCs and occupational health clinics. Compliance with these regulations allows the Employee Health Program to operate

within either the Preventive Medicine Service or the health clinic system.

Preventive Medicine Service Employee Health Program

In those installations that have an army hospital, the occupational health section of the Preventive Medicine Service conducts the Employee Health Program (Figure 3-1). Often the occupational health section has no clinical capabilities, but it manages all the administrative aspects of occupational health for both civilian and military employees who work at the installation. The staff of the collocated MEDDAC or MEDCEN performs all of the preventive and clinical services for patients, including treatment and examinations. However, at installations where a mobile occupational health vehicle (MOHV) is assigned, the staff of the occupational health section uses the vehicle to provide preventive services at the worksite (Figure 3-2).

The staff of an occupational health section usually consists of one to three civilian occupational health nurses, a clerk, and often an industrial hygienist, depending on both the size of the population that is to be served and the health services that are required. The occupational health nurse serves as the Employee Health Program manager, except when a full-time physician is assigned to the section.

Physician's assistants with 2 years of postgraduate training in occupational health are assigned to several of the larger U.S. Army Forces Command (FORSCOM) installations, where they serve as program managers for those employee health services that are provided to soldiers.

In the absence of a full-time physician, the chief of the Preventive Medicine Service (if he or she is a physician), or a physician who is assigned to the MEDDAC or MEDCEN in another capacity, may provide occupational medicine support. This type of support is most commonly found at FORSCOM and

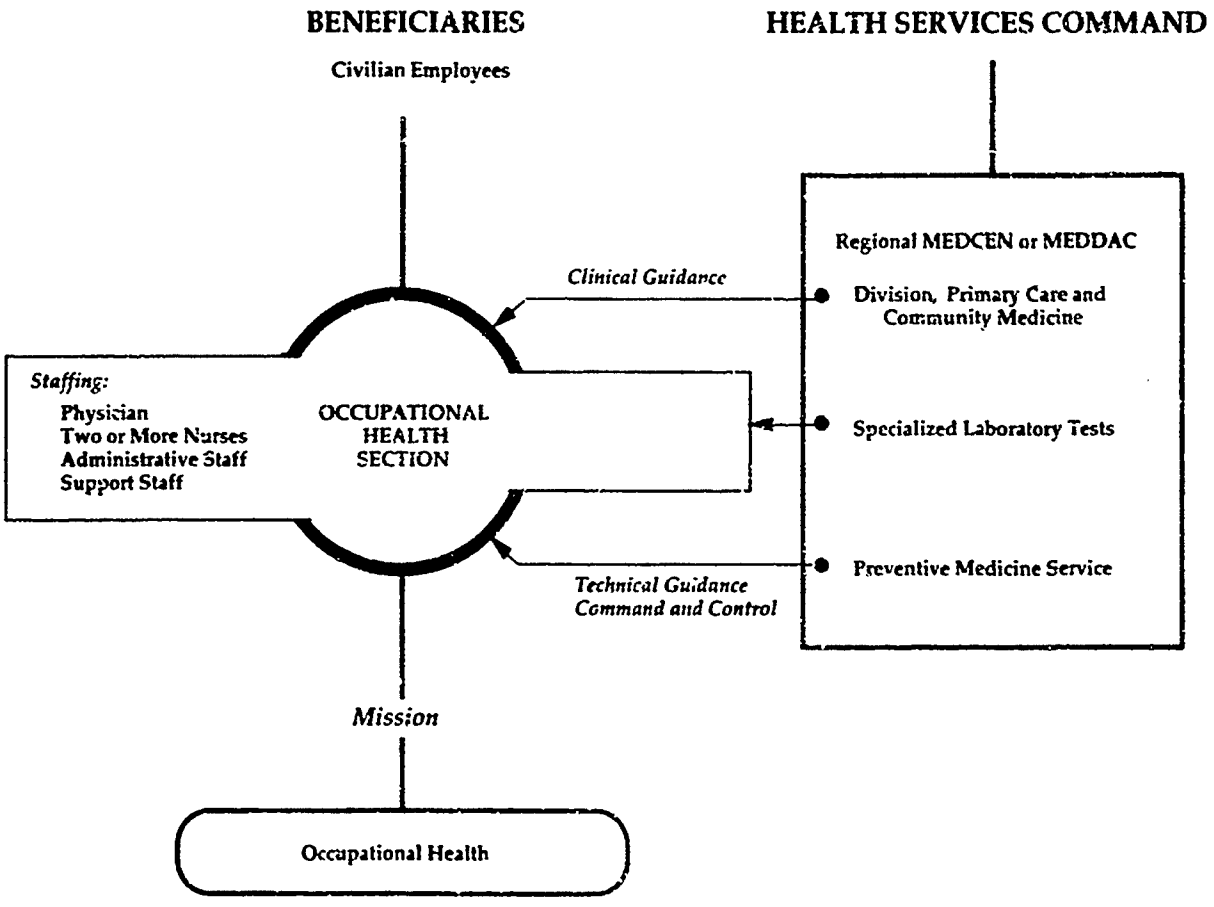


Fig. 3-1. The typical organizational structure for occupational health services on a larger army Training and Doctrine Command (TRADOC) or army Forces Command (FORSCOM) installation with a post U.S. Army hospital.

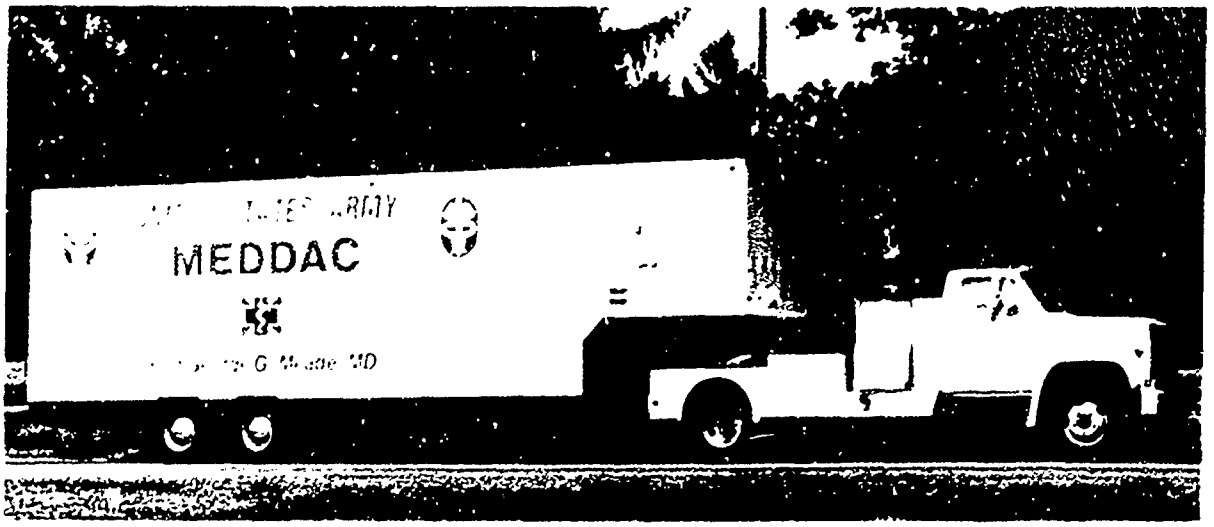


Fig. 3-2. The mobile occupational health vehicle (MOHV). The staff of the occupational health section use the MOHV to provide preventive health services at the worksite. Often power stations are set up at different locations so the vehicle can be moved from site to site according to a prearranged schedule. The U.S. Army deployed 11 MOHVs to Saudi Arabia during Operation Desert Storm.

U.S. Army Training and Doctrine Command (TRADOC) installations.

Army Health Clinic Employee Health Program

The Employee Health Program is a part of the AHC at U.S. Army Materiel Command installations. The AHC is organizationally located in the Department of Primary Care and Community Medicine of the regional MEDDAC or MEDCEN (Figure 3-3). The staff of the AHC usually includes one or two physicians (either military or civilian), several civilian nurses, and other administrative and support personnel. A physician is the director and possibly also the commander of the clinic. An AHC usually has general radiology and laboratory capabilities. With these staff and tools, the AHC's mission is to deliver both the civilian Employee Health Program and outpatient healthcare services to active-duty soldiers, their dependents, and retirees. The regional MEDDAC or MEDCEN also provides support such as specialized laboratory tests or audiology consultation when it is needed.

Although providing employee health services to civilian employees who are assigned to the installation can be a major part of the clinic's mission, managing the civilian Employee Health Program is often an additional, secondary responsibility. The Employee Health Program is usually integrated into the AHC's overall services, and the entire staff provides the employee health services. Often, one of the civilian physicians and several of the civilian nurses will be designated with the prefix *occupational health* on the Table of Distribution and Allowances, the official staffing document that identifies all positions, including specific clinic positions.

Each Employee Health Program should have a designated program manager; this is usually the senior occupational health nurse. In clinics where there is no actual position for an occupational health physician, one of the clinic physicians is assigned the responsibility of providing occupational medical consultation to the Employee Health Program. Other staff members may be assigned particular occupational health responsibilities, such as administering the hearing conservation program. The Department of Primary Care and Community Medicine manages the clinical aspects for most AHCs, but the supporting MEDDAC or MEDCEN Preventive Medicine Service provides technical guidance and support for occupational health.

Occupational Health Clinics and Nursing Offices

Occupational health clinics are medical treatment activities whose primary missions are to provide occupational health services to military and civilian employees who work at the installation, and sick call services to service members located in the immediate area (Figure 3-4). A physician heads this separately established operation, with a staff including civilian nurses and support personnel. The army has nine designated occupational health clinics. They are lo-

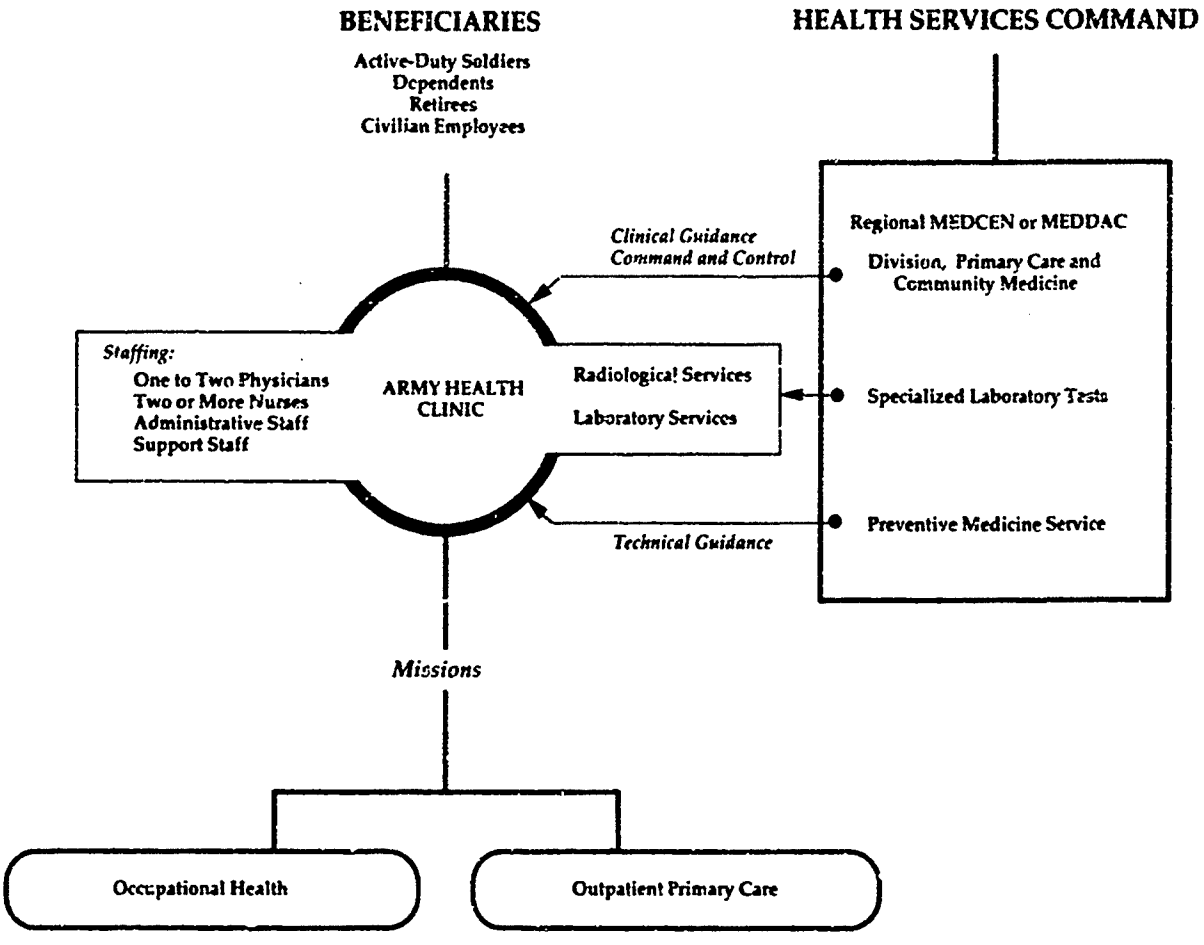


Fig. 3-3. The typical organizational structure for a U.S. Army health clinic at an installation without a post U.S. Army hospital, where primary care is the predominant healthcare mission.

cated at small installations such as the U.S. Army Natick Research Development and Engineering Center, where most workers are civilian and few military medical beneficiaries are in the area.

Occupational health nursing offices are similar to occupational health clinics but they lack a full-time physician (Figure 3-5).[7] These occupational health nursing offices are usually organizationally a part of the Preventive Medicine Service; most are located in leased federal office buildings in and around Washington, D.C.

Health Program Staffing

According to regulations of the army and the Office of Personnel Management, Employee Health Programs must be adequately staffed.[5,8] The size and experience of the staff of an occupational health section (or Employee Health Program, occupational health clinic, occupational health nursing office) depend on the population to be served, type of installation, range of employee health services provided, and availability of the resources.

All Employee Health Programs require at least one full-time civilian occupational health nurse, either a full-time or part-time physician, and clerical support. DA Pamphlet 570-557 provides guidance for determining staff requirements.[9] The pamphlet defines the civilian or military staffing levels that the army recommends for the Employee Health Program, regardless of the program's administrative structure.

Army Regulation (AR) 611-101 describes the commissioned officer's qualifications according to the *specialty skill identifier* (that is, a two-component numeric-alpha description that identifies the skills needed for a particular job).[10] For example, an active-duty occupational medicine officer is a 60-D. The *Office of Personnel Management Handbook X-118* describes the qualifications for each civilian General Schedule (GS) job series.[11]

The Civilian Personnel Office provides guidance and establishes local procedures for all civilian person-

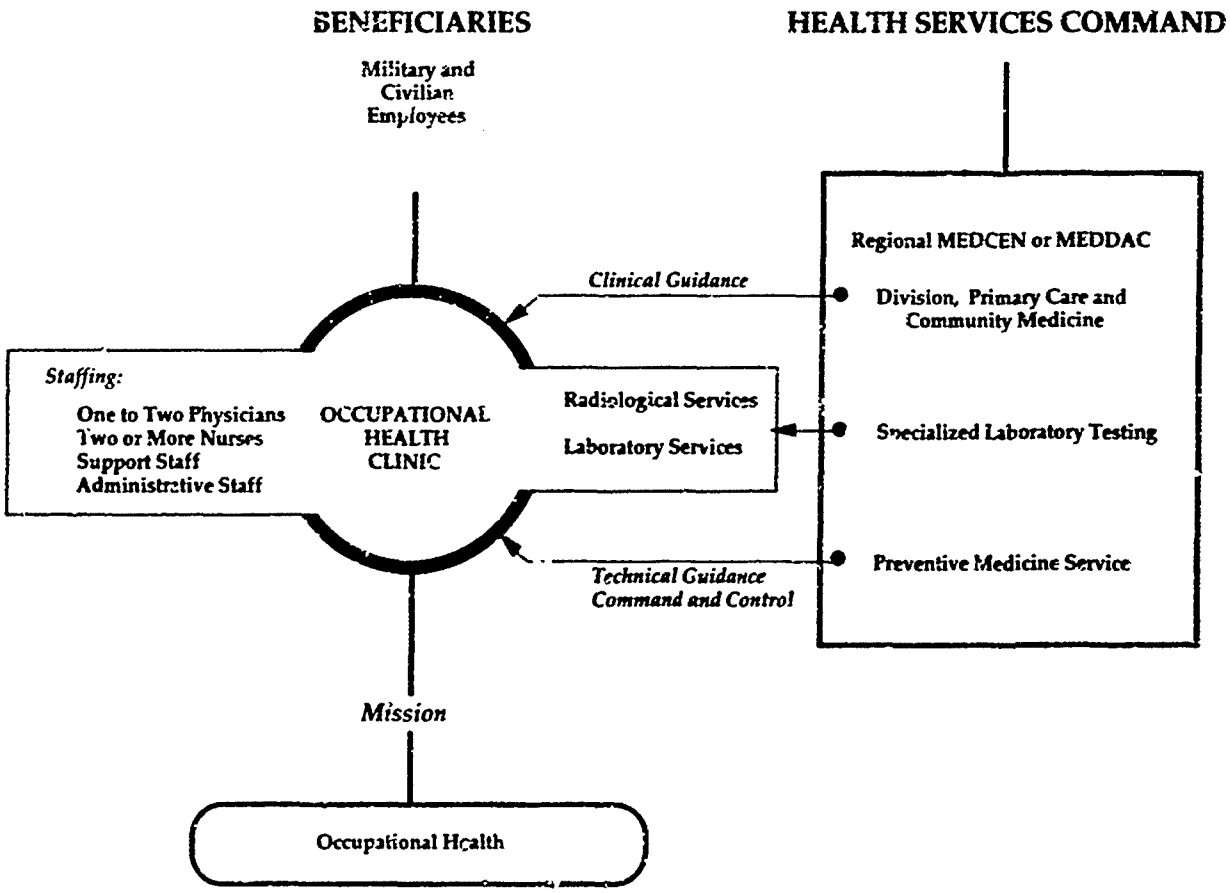


Fig. 3-4. The typical organizational structure for a U.S. Army occupational health clinic at an installation without a post U.S. Army hospital, where occupational health is the predominant healthcare mission.

nel administrative requirements. For example, a job description must accurately specify the functions of each position. Both the Civilian Personnel Office and the job supervisor are responsible for preparing each civilian employee's job description. The Civilian Personnel Office defines the technical aspects of the job description and the supervisor defines the job functions and supervisory controls. In addition, performance standards, which are based on the duties delineated in the job description, are required for all civilian staff members. The supervisor defines the acceptable level of performance for major duties, and these standards are used as a basis for evaluating an employee's job performance.

Once professional and technical personnel are assigned to the occupational health area, they are responsible for maintaining their own current licensure and certification according to legal and professional requirements. Each individual is responsible for his or her own continuing education.

The Occupational Health Physician

Either a military or civilian physician may fill the position of occupational health physician; however, their qualifications are different. While the MEDDAC or MEDCEN credentialing committee must approve both military and civilian occupational health physicians for clinical privileges as an occupational health physician,[12] civilian physicians must also meet the Office of Personnel Management's minimum qualifications for the position. In addition, training or prior experience in the field of occupational health is desirable for occupational health physicians.

Whether a military officer or a civilian, the occupational health physician can be assigned as a part-time or full-time member of the Employee Health Program staff. Usually a military physician is assigned part-time occupational health duties. (This arrangement is usually found at FORSCOM and TRADOC installations.) This part-time participation is seldom adequate

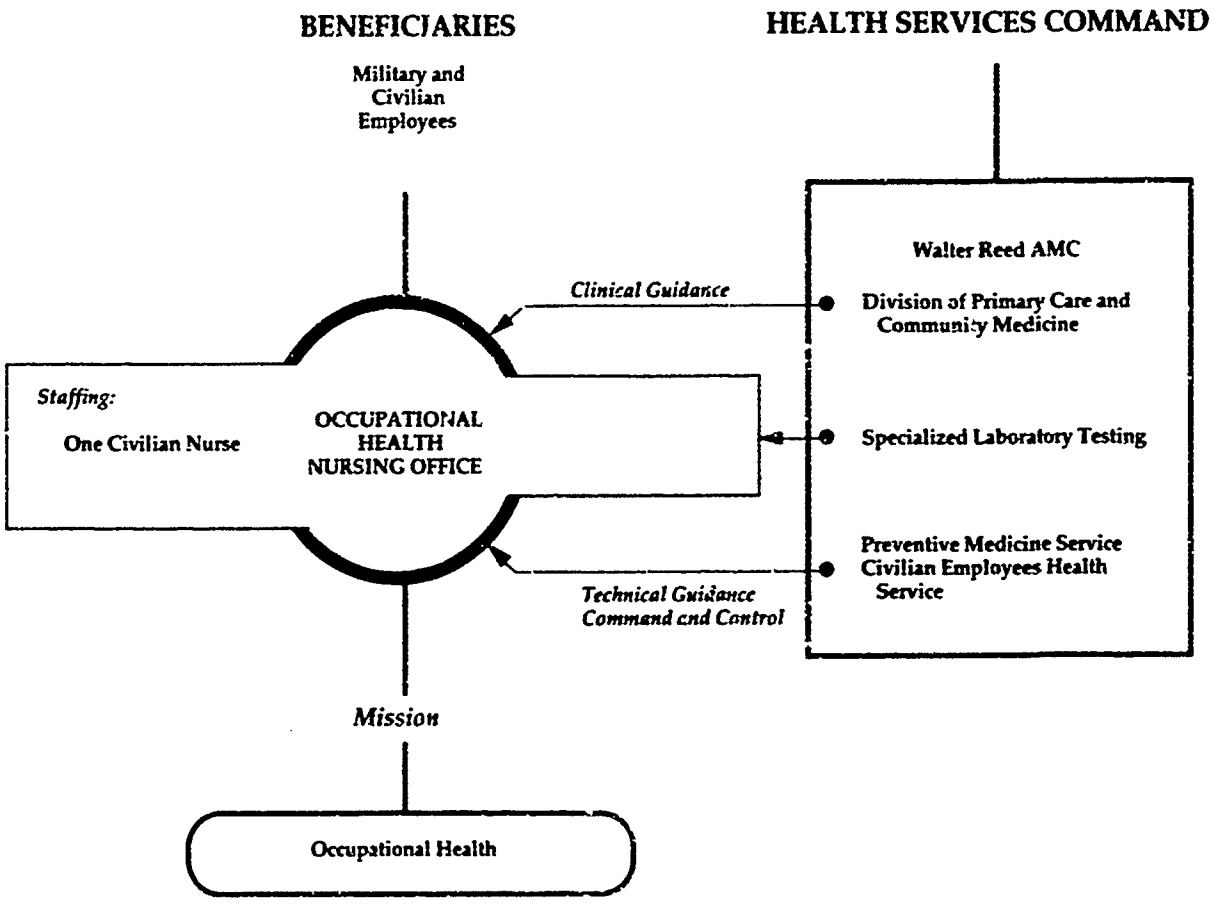


Fig. 3-5. The organizational structure of U.S. Army occupational health nursing offices in the Washington, D.C., metropolitan area.

because providing occupational medicine support for the Employee Health Program is added to the physician's primary assignment as a medical officer at the MEDDAC or MEDCEN.

The Occupational Health Physician's Assistant

The MEDDAC or MEDCEN credentialing committee must approve the military or civilian occupational health physician's assistant for clinical privileges.[12,13] Once physician's assistants have been approved to practice, they must be supervised by a physician.[12]

In recent years the army has provided additional education—a master's degree in occupational health—to selected military physician's assistants. The original purpose of training physician's assistants in occupational health in the army was to provide health services at the unit level to ensure soldiers' combat readiness. The role of the military occupational health physician's assistant has yet to be clearly defined; however, physician's assistants have functioned in various capacities (for example, as managers of the Occupational Health Program for soldiers or, infrequently, as managers of the overall Occupational Health Program for civilians and military personnel) when assigned to the MEDDAC at FORSCOM installations.

The Occupational Health Nurse

Occupational health nurses are civilian registered nurses who meet the minimum qualifications as mandated by the Office of Personnel Management. MEDDAC or MEDCEN clinical privileges are not required for occupational health nurses who practice within the usual limits of occupational health nursing. In addition to the basic nursing preparation, the nurse should have specialized training such as formal academic programs or short courses in occupational health. These educational programs should emphasize management principles; industrial toxicology; the cause, prevention, control, and treatment of occupational diseases; the principles of industrial hygiene and epidemiology; the concepts and practices of job-related

medical surveillance; and the legal and regulatory aspects of occupational health.

Ancillary Staff

The organization of the occupational health section determines whether ancillary personnel (such as licensed practical nurses, nursing assistants, medical technicians, occupational health technicians, laboratory technicians, and X-ray technicians) need to be assigned. MEDDAC or MEDCEN optometric and audiometric technicians may also provide significant support to the program. Depending on the technician's level of education, he or she could manage selected elements of the occupational health program such as hearing conservation.

MEDICAL RECORDS MANAGEMENT

Medical records and reports include (*a*) occupational medical records (cumulative individual medical records), (*b*) workers' compensation records (medical), and (*c*) administrative records and reports. The occupational medical record (civilian employee medical records and the military outpatient treatment records are types of cumulative individual medical records) and administrative reports are required by the Employee Health Program

An occupational medical record is the chronological, cumulative record of information about the developing health status of an employee with respect to his or her employment. Occupational medical records must contain personal and occupational health histories, employee exposure records, and the healthcare professional's written opinions and evaluations during the course of employment-related examinations, diagnoses, and treatments. In the military outpatient treatment record, the occupational health record consists of entries that are related to the soldier's work.

Occupational Medical Records

Civilian employee medical records and outpatient treatment records (for military personnel) serve as the occupational medical record. The purpose of any medical record is to document a complete medical history of the patient and his or her care, medicolegal support for the therapy that was given, and a basis for research and education. In instances when a civilian employee has dual status—such as a retired military member or the dependent of a retired or active-duty military member—that employee will also have both a civilian employee medical record and an outpatient treatment record. Each record will be marked or coded clearly to indicate this dual status and to facilitate the identification and reporting of job-related injuries and diseases.

The MTF commander is the official custodian of the medical records at the facility, but the chief of the patient administration division acts on behalf of the commander in matters that involve medical records. Both the civilian employee medical records and the outpatient treatment records should be maintained, by personnel trained in record keeping, in an MTF area designated for medical records.

The *Federal Personnel Manual Supplement 293-31, Basic Personnel Records and Files System*, is the major regulation that pertains to civilian employee medical records; AR 40-66 applies to outpatient treatment records; entries into all occupational medical records are made in accordance with AR 40-66.[14] This regulation contains guidance on recording injuries and includes a requirement to identify occupational injuries or illnesses as *occupational* in the medical record.

The *Report of Medical History* (SF 93) is used to obtain a health history from all civilian employees and to initiate a medical record on employment. (Exceptions can be made for transient nonappropriated-fund employees such as food-handlers.) Once the medical record is initiated with the SF 93, it is to be kept in the terminal digit series folder, *Alphabetical and Terminal Digit File for Treatment Record* (DA Form 3444), or in the *Employee Medical Folder* (SF 66-D). When an employee either transfers to another federal agency or is separated from federal service, the civilian employee medical record is transferred or retired in the SF 66-D. If the civilian employee medical record has been maintained in a DA Form 3444 during the individual's employment, the personnel responsible for maintaining the civilian employee medical record must ensure that a signed *Privacy Act Statement-Health Care Records* (DoD Form 2005) is transferred or retired with the record.

In general, only standard forms (that is, those forms that are authorized by HSC, DA, and DoD) are used in medical records. The MTF commander must approve any locally-developed form or overprint before it is placed in the medical record. In addition to standard forms, a copy of the Office of Workers' Compensation forms related to medical treatment must be maintained in the medical record.[15] These forms include

- Federal Employee's Notice of Traumatic Injury and Claim for Continuation of Pay/Compensation (CA-1).

- Federal Employee's Notice of Occupational Disease and Claim for Compensation (CA-2), and
- Request for Examination and/or Treatment (CA-16).

When a civilian employee separates from federal service or transfers to another federal agency, the civilian employee medical record is usually forwarded to the Civilian Personnel Office in the SF 66-D. The Civilian Personnel Office retires the record to the National Personnel Records Center when an employee separates from federal service, and forwards it to the gaining agency when an employee transfers.

Transferring and maintaining job-related X-ray films require special procedures. If it is 8½ x 11 in. or smaller, the film is placed in the medical folder (SF 66-D) and retired or forwarded as part of the medical record. However, if the job-related X-ray film is larger than 8½ x 11 in. (such as a roentgenogram of a patient's chest and torso), it must be maintained separately from the SF 66-D. When an employee separates from federal service, the large films are maintained in their original state at the last employing agency for 30 years beyond the termination of employment. The medical record must also contain a notation on the radiologist's findings, the location of the radiograph, and how it can be obtained. If the employee is transferring to another federal agency, the large films are simply forwarded to the gaining agency.[15,16]

Like any other medical record, the civilian employee medical records must be maintained in strict confidence. However, OSHA regulations allow the employee, his or her representative as designated in writing, and OSHA representatives (compliance officers and National Institute for Occupational Safety and Health [NIOSH] personnel) to examine or copy medical records or medical information that bears directly on the employee's exposure to toxic materials and harmful physical agents such as radiation and noise.[16,17] This access is strictly limited and does not include access to any health information that is unrelated to exposure.

Medical Reports for the Employee Health Program

The MTF commander is responsible for submitting several recurrent reports that require occupational health data. The chief of the patient administration division is responsible for establishing procedures for retrieving data for all required reports, and must coordinate with the chief of the Employee Health Program to identify and ensure that all occupational health reporting requirements are met. While the staff of the Employee Health Program may or may not actually compile these periodic reports, they should be aware of the data that are required and the purpose for their collection. The primary MTF reports that require occupational health data include the *Army Occupational Health Report* (DA Form 3076); the *Medical Summary Report* (MSR), RCS (Requirement Control Symbol): MED-302; the *Management Indicators for Occupational Health (MIOH) Feeder Report* (RCSSAOSA-220); and the Command health report. Two additional reports to which Employee Health Program personnel may contribute include the *Special Telegraphic Reports* [RCS MED-16(R4)] and the *Log of Occupational Injuries and Illnesses.*

The installation, MEDDAC personnel, and higher headquarters (HSC and the U.S. Army's Office of The Surgeon General [OTSG], and the installation's major command) review and evaluate the Employee Health Program using the data in DA Form 3076, which the Employee Health Program staff must prepare and submit biannually.[6] DA Form 3076 contains information about Employee Health Program staffing, the civilian and military population at risk for potential health hazards in the work environment, and the number and types of occupational health services that the Employee Health Program provides. DA Form 3075 is used to collect these data. Instructions for completing both DA Forms 3075 and 3076 are on the reverse sides of the forms.

The MED 302 Report is one of the components of the DA medical information system that the MTF, intermediate headquarters (such as MEDDACs or MEDCENs), The U.S. Army Surgeon General, and other government agencies use for evaluating budget requests, planning programs, controlling management, analyzing manpower requirements, making authorizations, and planning facilities. Each army MTF must submit the MED 302 Report every month to provide a timely and accurate medical summary of the services that the entire MTF provides. A deficiency of this report is that it does not fully recognize the workload of the preventive medicine services, but concentrates only on the services that are provided to ill patients. The chief of the patient administration division usually requests specific data from the Employee Health Program staff to formulate this report.

Local program managers and higher headquarters use the specific data in the *MIOH Feeder Report* when they analyze and review occupational health in the army. The report reflects the numbers of occupational health staff, the population at risk, and the program evaluations that have been performed. Data concern-

ing the Industrial Hygiene and Hearing Conservation programs are provided, in addition to data regarding the Employee Health Program. All MEDCENs and MEDDACs that provide health services for active-duty soldiers and civilian employees must prepare an *MIOH Feeder Report* for their health-service area biannually.[18]

Each month, every installation's Medical Authority (that is, the senior physician assigned to the MEDDAC at the installation) is required to provide the installation commander with the Command health report.[5] Although there is no specific format required for this report, it summarizes the conglomerate health status of the command and the people who comprise the command. The Command health report provides

- information regarding health conditions within the command;
- recommendations for, or descriptions of, actions taken to improve health conditions;
- advice to higher headquarters regarding the support needed to implement the recommended actions; and
- information regarding outstanding accomplishments, new developments, and trends.

The *Special Telegraphic Reports* [RCS MED-16(R4)] are unique reports that provide epidemiological data and the results of epidemiological investigations on selected health conditions, disease outbreaks, deaths, and attempted suicides. These reports include the *Special Telegraphic Report of Selected Condition*, the *Special Telegraphic Report of Reportable Outbreak*, and the *Special Telegraphic Report of Reportable Death*.[19] Preventive medicine personnel have used the MED-16 report extensively to document outbreaks of infectious disease. However, it is equally important to notify higher headquarters of outbreaks of injury or disease, or unusual occupation-related health events, such as one or more cases of overexposure to ionizing or nonionizing radiation, or if two or more persons have been removed from their jobs as a result of abnormal job-related medical surveillance tests. The submission of any of these reports requires liaison among Preventive Medicine staff (including the Employee Health Program staff), the patient administration division, and other medical staff.

All federal agencies are required to collect occupational injury and illness data and to record these in the OSHA Log of Federal Occupational Injuries and Illnesses.[20,21] Although the installation's safety officer usually has the primary responsibility for collecting data and maintaining the log, coordination with the occupational health staff and the local Federal Employees' Compensation Act Program administrator is essential to ensure that the data are complete. The safety office should report all of the following in the log:

- occupational illness,
- job-related injuries that resulted in death or disability,
- job-related injuries that caused employees to lose time at work (other than the day on which the injuries occurred), and
- job-related injuries that required medical care.

MEDICAL SURVEILLANCE

The two principal missions of occupational health in the army are not distinctly separate, but they are very different: (1) reducing negative job-health interactions is *preventive* and applies to the general population of workers, as well as to the individual worker; and (2) providing healthcare services is *clinical* and applies to the individual employee as a patient. These elements of the Employee Health Program can be discussed separately. A basic tool for the preventive aspects of occupational health is *medical surveillance*. Job-related medical surveillance in the field of occupational health consists of systematically and periodically collecting and analyzing health data on groups of employees for the purpose of early detection of the increased risk, or the actual presence, of negative job-health interactions. Medical surveillance can be used to achieve

- primary prevention, which is oriented to preventing the risk;
- secondary prevention, which is oriented to reducing or preventing the exposure; and
- tertiary prevention, which is oriented to reducing or preventing long-term impact of the health effect.

When using medical surveillance as a primary or secondary prevention tool, we view the individual as an employee. When we use medical surveillance for tertiary prevention, we view the individual both as an employee and as a patient. The surveillance results are used for the diagnosis and treatment of the clinical condition and to indicate that a change (for example, light duty) is needed in the workplace.

Primary prevention is defined as

> a means of preventing the occurrence of illness or injury; for example, by immunization against infectious disease and by using safety equipment to protect workers in hazardous occupations.[22]

It seeks to reduce or eliminate risk through intervention before exposure to that risk. Thus, when the risks that are associated with a particular job and the health characteristics of the employee that place the employee at increased susceptibility to those risks can be determined, then medical surveillance can be used to identify those employees who will be at the greatest risk, and prevention can be directed at these employees. Of course, prevention cannot be effective until the data from surveillance are used to enforce a change, thereby reducing the occupational risk.

Primary prevention seeks to reduce or eliminate the risk by avoiding exposure. For example, it can be as simple as performing glucose-6-phosphate dehydrogenase (G6PD) tests on all who might be exposed to nitrate-containing compounds, such as the explosives RDX and M6 (a propellant used for artillery). Exposure to nitrates can cause methemoglobin to form; the G6PD enzyme converts methemoglobin back to hemoglobin. Individuals with the genetically determined G6PD-enzyme deficiency (such as those of Mediterranean heritage) may be unable to make the conversion rapidly enough to prevent signs of methemoglobinemia. Therefore, a one-time preplacement surveillance for G6PD levels can prevent the worker from being exposed, and thus avert the risk of methemoglobinemia by taking preventive action based on the findings.

Secondary prevention includes both detecting deleterious health effects and intervening before an illness becomes clinically apparent, with the goal of retarding, halting, or reversing the progress of the illness. Medical surveillance can be used for secondary prevention to detect job-related health effects prior to the onset of clinical disease. Surveillance becomes a secondary prevention tool only when the data collected are used to guide interventions that are effective. Other than in hearing conservation, there have been no systematic efforts to conduct medical surveillance for soldiers, with the exception of the routine physical examinations required to remain on active duty.

During the demilitarization of the chemical agent BZ (one of the incapacitating chemical warfare agents), the workers were enrolled in a very strict medical surveillance program to assure that early signs and symptoms of exposure to BZ would be detected. Important to this program was the measurement of the size of the workers' pupils before and after they entered the chamber. BZ exposure causes mydriasis; while mydriasis is not in itself incapacitating, it is one of the most sensitive indicators of exposure. Not until a worker was actually seen with mydriasis one afternoon was it learned that the procedures used by the workers to decontaminate themselves after their workshifts might be inadequate. Not realizing that her hands could have been contaminated, the worker passed her hands over her hair and over her eyes as she showered, transferring the BZ directly. The amount of BZ was so small that only direct contact with her eyes could have caused her pupils to enlarge. Therefore, showering procedures were changed; the workers were instructed to assume that their hands were potentially contaminated, and to wash them first. In this instance, medical surveillance was an effective secondary prevention tool; it did not prevent exposure, but it detected the physiological effect of exposure before an important clinical event occurred.

Tertiary prevention includes both the detection and treatment of an illness, or the rehabilitation of an injured or ill person, sufficiently early in the course to decrease the long-term impact that the illness or injury may have on that individual. Medical surveillance is a tertiary prevention tool when it is used to (*a*) document that the job has already affected an employee's health or (*b*) demonstrate that an employee is not fit for duty after an event has occurred.

The following example demonstrates medical surveillance in its primary, secondary, and tertiary prevention roles. Carpal tunnel syndrome (CTS) is a condition associated with certain repetitive-motion occupations such as keyboarding. A medical surveillance program for keyboard operators could include disqualification of any individual who reports previously existing CTS on preplacement history. For this job, exclusion serves as primary prevention. The program would also include limited annual histories for all keyboard operators to detect the early symptoms of CTS. Any positive findings (for example, subclinical tingling in the fingers) would be followed up by actions to determine workplace practices and to rule out clinical disease. This medical surveillance program serves as secondary prevention when it has detected subclinical findings and the Employee Health Program staff has taken action to prevent further exposure. The same program serves as tertiary prevention when a worker presents with clinical signs of CTS and the actions taken in the workplace are done to prevent further exacerbation of the condition

Medical surveillance is prospective and ongoing in nature. Effective, efficient, and economical job-related medical surveillance must encompass (*a*) the design of

the surveillance program, (*b*) the performance of medical screening, and (*c*) the use of the surveillance data. The occupational healthcare professional must fully understand that medical surveillance and medical screening are two different concepts (Table 3-1). Medical *screening* is the one-time determination of the presence or absence of a health characteristic in an individual or a group at risk. Medical screening is cross-sectional and periodic. Only rarely have one-time abnormal screenings demonstrated job–health interaction.

Lead exposure and its medical indicators illustrate the concepts of medical screening and medical surveillance. Blood-level determination is an excellent screening tool for lead exposure because an elevated value of lead in the blood is related both to exposure and to lead's biological effects. There are two action levels for lead. The first is the environmental exposure (currently, 30 $\mu g/m^3$ as a time-weighted average), which determines when blood-lead screening must be initiated. The second is a blood-lead screening level of 60 $\mu g/dL$, which requires removal from the lead exposure and more frequent screening until the individual's blood lead level drops below 40 $\mu g/dL$.

Clearly, screening for lead is effective in situations where excessive exposure to lead, and elevated blood levels of lead, occur. One-time, *normal* blood levels of lead are of little use. However, if screening is conducted over time (for example, 5 years), even though measured workplace levels of lead do not exceed the action level, slowly rising normal levels can be detected. Steps can then be taken to control lead exposure *before* any individual worker is adversely affected. Careful screening together with directed actions constitute effective medical surveillance. Therefore, determining a blood level of lead is medical screening, whereas comparing blood lead levels over time, whether the results are normal or abnormal, is medical surveillance.

Determining the Need for Surveillance

Designing an effective surveillance program and tailoring it to a given group of workers begins with determining (*a*) the reason for the surveillance and (*b*) the health characteristics that should be monitored. For these purposes, all employees can be divided into three basic groups.

Group 1

The first group of employees perform jobs that require minimal physical exertion and have little potential for exposure to job-related hazards. Medical surveillance for these employees is limited to periodic screening to ensure that the workplace remains safe and the employees remain healthy.

TABLE 3-1

MEDICAL SCREENING VERSUS MEDICAL SURVEILLANCE

Characteristics	
Screening	**Surveillance**
Cross-sectional	Prospective
Singular	Longitudinal
Focuses on absolute values (normal or abnormal)	Focuses on trends

Group 2

The second group of employees are those whose health or fitness status must be sufficient for them to be able to (*a*) perform their work safely and effectively, or (*b*) wear the personal protective equipment (PPE) required at a worksite. For example, a driver should not have any condition that could incapacitate him while he transports hazardous cargo. Thus, medical standards for this job might include the absence of insulin-dependent diabetes mellitus, and a medical surveillance program would screen the driver for diabetes. (Discrimination against the medically handicapped is not at issue here, if it has been documented that the medical condition will indeed compromise the job.)

The key to designing medical surveillance for these employees is to maintain clearly defined medical standards. In the army, the staffs of Employee Health Programs do not have the authority to publish medical standards, especially if employees must meet these standards if they are to retain their jobs. The sole authority for medical standards belongs to the Office of Personnel Management through the Civilian Personnel Office. However, physicians should work closely with personnel officers to develop medical standards when appropriate.

Performance-related medical standards that address required capabilities are preferred to *specification*-related medical standards, which require documentation of the absence of specific results of screening tests. For example, a medical surveillance program should be designed to ensure that employees who are required to wear respiratory protective equipment are physically capable of doing so. Disqualifying factors for wearing a respirator safely could include wearing

a beard (which would compromise the seal) and abnormal or borderline pulmonary function.

An appropriate performance-related medical standard could state that any employee required to wear a respirator (*a*) not wear a beard and (*b*) have no evidence of compromised pulmonary function. In this case, the occupational health physician might decide that he or she will determine the presence or absence of compromised pulmonary function, especially in a younger population, merely by talking to and observing the employee periodically.

On the other hand, a specification-related medical standard could indicate that pulmonary function tests must be performed annually, and that an employee with an FEV_1 (forced expiratory volume in 1 sec) lower than 70% of that expected for the employee's age group would be removed from the job. This specification-related medical standard requires unnecessary testing and also raises insurmountable quality assurance, medicolegal, and ethical difficulties. Fortunately, few published medical fitness standards exist. The army's Employee Health Program staffs have the opportunity and the responsibility to work closely with management in developing local medical fitness standards that are tailored to individual installations.

Group 3

This group of employees works with potential chemical, physical, or biological hazards and require medical surveillance for the following reasons:

- to meet requirements where workplace exposures exceed one of the federal exposure standards,
- to detect exposure-related health effects early,
- to monitor the effectiveness of the controls, and
- to monitor the extent of the exposure.

Several acceptable means exist for tailoring job-related medical surveillance for this group of employees. Medical surveillance can be based on job titles, worksites, or documented exposures and individual susceptibility.

Designing Medical Surveillance

A medical surveillance program that is based solely on the assumption that exposure has occurred is unlikely to be cost effective. Numerous factors contribute to determining the need for and content of medical surveillance:

- The assumption that a worker might have been exposed does not mean that a biologically significant dose will also have been received.
- The assumption that a biologically significant dose has been received does not necessarily mean that the dose will have been sufficient to cause a negative health effect.
- Likewise, the assumption that a negative health effect has occurred does not mean that an appropriate screening test also exists.

The level of exposure sufficient to trigger concern for a group of employees is sometimes difficult to determine. However, if an obviously uncontrolled exposure is occurring, preventive medicine dictates that action be taken to alleviate the exposure without screening for health effects. Where uncontrolled exposure has not been documented, the probability that health effects can be demonstrated through surveillance is very low. Basing the need for surveillance on the assumption that any exposure will cause negative health effects is neither cost effective nor sound practice of occupational medicine.

Other factors further complicate surveillance design. We cannot assume that all subjects in a given population will be exposed identically; also, we cannot assume that they will respond to exposure identically. For example, in an army ammunition plant, two workers standing side by side at an assembly line may be exposed to completely different levels of propellant for so simple a reason as a difference in the air flow. Even if they were exposed identically, the two might not demonstrate the same toxic effects. Nitroglycerin unintentionally inhaled or ingested in the workplace has exactly the same biological effect, vasodilation, as nitroglycerin taken as a prescribed medication. One worker might be very susceptible, due to his cardiovascular status, while a coworker could be completely unaffected.

Once an exposure justifies medical surveillance, other assumptions must be made concerning the effects on health. First, most toxic substances affect specific organ systems in specific ways. Often the toxicity of the substance depends on the route of exposure, and health effects vary with the level and the duration of exposure. Standard toxicology textbooks usually list all the effects on health that are associated with a given poison. If the staff of the Employee Health Program designs a screening program to demonstrate the absence of every toxic effect a particular agent might cause, the staff will, in effect, be trying to prove a negative. Such a program will be neither cost effective nor useful.

Instead, the occupational health physician must use professional judgment to determine the organs most likely to be affected if a negative job-health interaction has occurred. The OTSG has accepted criteria for determining the most likely sentinel toxic effects, including

- the health effect on which the OSHA permissible exposure limit (PEL) or the American Conference of Governmental Industrial Hygienist (ACGIH) Threshold Limit Value (TLV) was originally based,
  any health effect demonstrated to occur after exposures to airborne levels up to twice the PEL or the TLV, and
- any health effect in animals, if the exposure was up to twice the PEL or TLV, and if other related chemicals are known to cause similar health effects in humans.

Some employees may require job-related medical surveillance to meet minimum medical standards, as in the case of the second group of employees described above, and due to potentially hazardous exposures, as in the case of the third group. Although it makes good sense to conduct all required surveillance concurrently, the occupational health practitioner must assure that the principles outlined for both groups of employees are applied.

Determining the Screening Content

After the need for medical surveillance has been determined, the content of the surveillance must be designed.

Medical Surveillance Screening Tools

Four medical screening tools are available: (1) the interim history, (2) a limited physical examination, (3) diagnostic laboratory screening, and (4) biological monitoring. The effectiveness of these tools is measured by their sensitivity, specificity, cost, acceptability, ease of use, accuracy, and reproducibility (Table 3-2). Few screening tools meet all criteria for widespread use.

Figure 3-6 provides a visual presentation of the relationships among sensitivity, specificity, and positive predictive value. *Sensitivity* is the percentage of positive test results in a population that has the characteristic being tested (true positives). *Specificity* is the percentage of negative test results in a population that does not have the characteristic being tested (true negatives). *Positive predictive value* depends on the specificity of the test and the prevalence of the condition in the population. Quite simply, positive predictive value is the percentage of true positives among all positives. However, in the case of job-related medical surveillance, positive predictive value and true positives must also take into account the specific etiology of the specific effect (that is, the job-relatedness). A patient's liver-function test may be abnormal, but when considering the patient as an employee, this abnormality is relevant to occupational health only if the abnormality is caused by a job-related exposure.

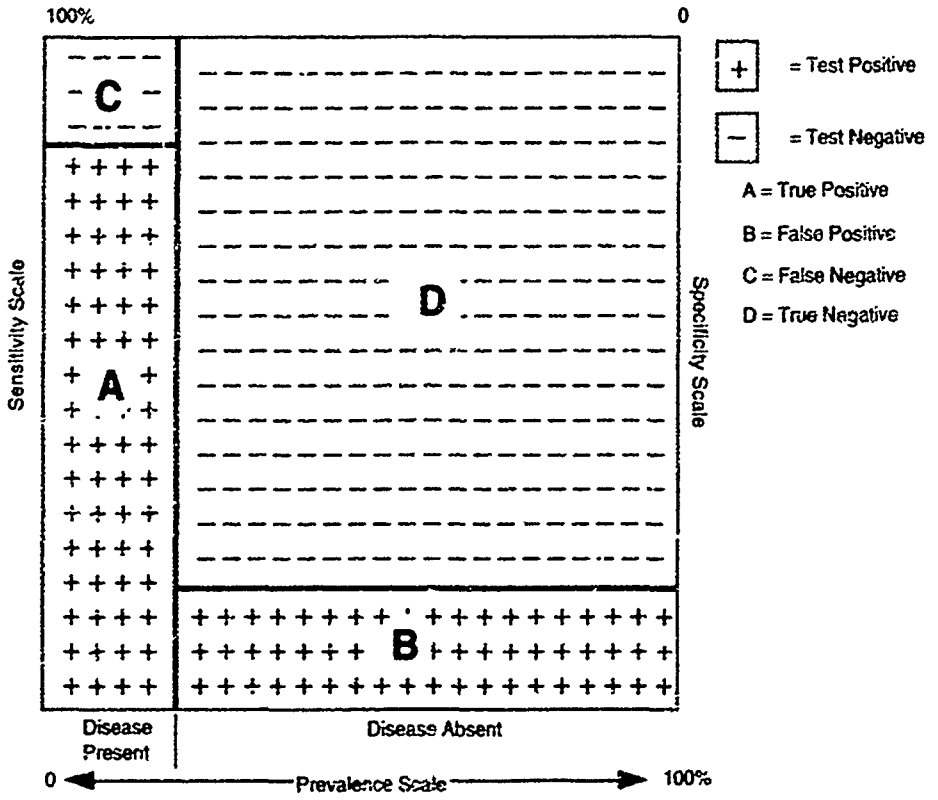
To expand further on this example, liver-function tests can be demonstrated to have very low positive predictive value for job-related medical surveillance. Assume that a worker population has a 5% prevalence of job-related liver disease, and that liver-function tests have 100% sensitivity. Also assume that liver-

TABLE 3-2

STRENGTHS AND WEAKNESSES OF MEDICAL SCREENING TOOLS*

	Interim Medical History	Limited Physical Examination	Laboratory Procedures	Biological Monitoring
Sensitivity	+	+/−	++	−+
Specificity	+	+/−	−−	++
Low Cost	++	++	+/−	+/−
Acceptability	++	++	+/−	+/−
Ease of Performance	++	++	+/−	+/−
Accuracy	−/−	+/−	++	++
Reproducibility	+/−	+/−	++	++

*Assumes adequate clinical skills and state-of-the-art technology



Sensitivity: the probability that if someone has the disease, he or she will test positive. Sensitivity = $\frac{A}{A+C}$

Specificity: the probability that the test will be negative given the absence of the disease. Specificity = $\frac{D}{B+D}$

Positive Predictive Value: the probability that someone with a positive test will actually have the disease. Positive Predictive Value = $\frac{A}{A+B}$

Negative Predictive Value: the probability that someone with a negative test will not have the disease. Negative Predictive Value = $\frac{D}{C+D}$

Fig. 3-6. A visual representation demonstrating the mathematical relationships among positive and negative test results and disease presence or absence. The relationships are dependent on test sensitivity and specificity and on disease prevalence. Figure. Courtesy of David F. Cruess, PhD, Professor, Department of Preventive Medicine and Biometrics, F. Edward Hébert School of Medicine, Uniformed Services University of Health Sciences, Bethesda, Md; 1993.

function tests have a specificity of 90%. The results of 100 liver-function tests in this population would be 5 true positives and 10 false positives. The positive predictive value would be 5 true positives out of a total of 15 positives, or 33%. This example does not include the prevalence of non–job-related liver disease (certainly not an uncommon entity), which would reduce the positive predictive value for job-related disease even further. Using low-specificity screening (such as liver-function tests) often results in expensive, time-consuming follow-up. Obviously, selecting a more specific screening tool is one solution to this problem. Being very specific in the population selected for screening (that is, doing liver-function tests on those workers who report changes in alcohol tolerance and are known to be exposed to hepatotoxins) is another.

The most valuable medical screening tool is the interim occupational and medical history, which is used throughout the employee's participation in the Employee Health Program. In most cases, the interim history constitutes the only screening necessary for medical surveillance because the army's workforce is relatively healthy and job-related illnesses are rare. A well-constructed interim history can

- confirm information about an individual's job,
- confirm the employee's use of personal protective devices,

- confirm exposure history,
- determine possible changes in health status,
- determine the need for further screening,
- establish an appropriate relationship with the employee,
- observe the employee for signs of decreased fitness, and
- provide the employee with job-specific health education.

A limited physical examination directs the physician to look for specific findings that the interim history or published medical standards have already suggested. By limiting the examination to target organs, the specificity increases and the examination becomes useful for medical surveillance. But simply instructing a physician to administer an examination with emphasis on a specific organ system does not always direct the physician sufficiently. For example, published medical surveillance guidance for workers who are exposed to isocyanates recommends an annual examination "with emphasis on the respiratory system."[23] Such guidance is inadequate. If the examining physician is unaware that isocyanates are pulmonary sensitizers that cause job-related asthma, he or she may examine the patient on Monday morning (before exposure has occurred) and note normal findings on chest auscultation. But if the physician had been advised to listen for wheezing after exposure to isocyanates, the examination could have been conducted after the worker was exposed on Wednesday afternoon. This examination, directed toward a specific finding, might also be quicker and easier. Just examining the chest is completely different from specifically listening for wheezing after an exposure to a known pulmonary sensitizer.

Diagnostic laboratory screening tests are the third medical screening tool. For laboratory screening to be effective, the test's specificity is of paramount importance. A battery of tests on a group of employees based on their job description, or on the presumption of exposure, can be costly (the cost of initial and follow-up testing) and quite likely will not discriminate between *true* positive results (which could indicate a job-related health effect) and *false* positive results (which are either spurious or have nothing to do with the job). Diagnostic laboratory screening is not able to detect all of the sentinel target organ effects on health (such as most early cancers), and one-time screening values, regardless of whether they are normal or abnormal, are often useless.

Even though published medical surveillance guidance (for example, DoD Manual 6055.5-M) for specific hazards includes a comprehensive list of laboratory tests, this information—because it is so broad and nonspecific—may or may not be valuable. Many medical personnel conclude, erroneously, that because the lists are published by experts, physicians are therefore obligated to perform all the tests listed for each specific exposure. However, the guidelines address all potential exposures; *professional judgment is essential.* The only professionals who can determine which tests are necessary are the physician, the nurse, or the physician's assistant, who have talked to the employee, visited the employee's workplace, and determined the possible health effects for which a diagnostic screening test is indicated and available.

Biological monitoring includes all of the same characteristics as diagnostic laboratory screening and can also measure a toxic agent or its metabolites in a bodily fluid. Biological monitoring can be an excellent medical surveillance tool because of its high specificity, but such monitoring is available for very few occupational hazards (for example, for lead, blood lead; for benzene, urine acetone; and for polycyclic aromatic hydrocarbons, specific DNA adducts). Audiograms can also be used as medical surveillance tools to monitor for exposure to noise.

Timing the Screening

The timing of medical screening is essential. Screening is conducted before job assignments are made, periodically during employment, and on termination of employment. It also can be conducted in conjunction with the preplacement examination in order to provide baseline data for comparison with future surveillance data, and to document the preexposure health status. Periodic medical screening is conducted at routine intervals and should always include an interim history. The content of periodic screening need not always include the entire spectrum of screening tests, but the appropriate tests should be selected based on the interim history, the latency period of the most likely health effect, and the time required for physiological or physical changes to be of sufficient magnitude for detection.

To determine the appropriate time of the day or the week for screening, the occupational health physician must consider the reason for the screening. For example, in searching for long-term cumulative effects, or in documenting fitness to work, screening might best be conducted early in the day and early in the week, to decrease the likelihood that test results will reflect transient effects. Hearing tests, for example, are done after a 48-hour quiet period (such as a weekend). However, if acute effects are being sought, or if the extent of exposure is being documented, screening

both later in the day and later in the week may be more appropriate. Thus, the easiest way to ensure that screening is appropriate is to (*a*) conduct the initial interview at any convenient time, and then (*b*) conduct further screening at a specifically appropriate time.

Using Medical Surveillance Results

The success of a medical surveillance program depends on how the collected data are used. Whether the data show normal or abnormal findings, the results must be used to prevent any negative job-health interaction. Normal medical screening results, for example, can be used as a basis for comparison with future screening, for altering the content of future screening, for documenting that the employee is fit for work, and for ensuring that control measures are adequate.

Abnormal screening results require more action. First, the physician, physician's assistant, or nurse must inform the employee of the results and recommend appropriate medical follow-up. If, after the first-level screening, a diagnosis or relationship to the job cannot be determined, the staff must conduct more comprehensive testing until a negative job–health relationship is either verified or eliminated. Furthermore, when abnormal findings demonstrate that an employee is unfit for work, according to either medical standards or physical requirements, the staff must work with the Civilian Personnel Office and recommend a job change.

When published medical standards do not exist, the staff must use their professional judgment about the possible relationship between screening results and the job. However, if surveillance results indicate that the job has affected an employee's health, then the staff cannot assume that only one employee is at risk. They must assume that a sentinel event has been detected and vigorously pursue surveillance of the potentially at-risk population. In addition, the staff, together with the industrial hygienist, safety officer, and engineer, must intervene at the job site to

- substitute nonhazardous substances;
- reduce exposure with engineering controls;
- reduce exposure with administrative controls;
- limit internal doses by using PPE, and;
- when necessary, remove employees from the job.

Far too often, occupational health staff are uninformed. They equate medical surveillance with occupational health. Furthermore, they equate medical screening with medical surveillance. While both are clearly very important to the Employee Health Program, if they are not (*a*) performed with insight and planning and (*b*) followed up with prevention-oriented actions, not only are time and money wasted, but the job-related health effects that *are* occurring may be undetected until it is too late to help the workers.

OCCUPATIONAL HEALTHCARE SERVICES

A number of Employee Health Programs, including those for medical, dental, and veterinary personnel, provide preventive medicine services based on the concept of surveillance, including (*a*) the Employee Health Program for healthcare workers, (*b*) health education, (*c*) health promotion, (*d*) administrative medical examinations, (*e*) monitoring absences due to illness, (*f*) job-related immunizations, (*g*) reproductive-system surveillance, (*h*) surveillance of personnel with chronic diseases or physical disabilities, and (*i*) epidemiological studies.

Other health services are clinical, including (*a*) emergency treatment of illness and injury and (*b*) prevention and control of alcohol and drug abuse. All of these preventive measures generate a great deal of information about the employee's health status that must not only be documented in records and reports but also be used to support the ultimate goal of the prevention of negative job-health interactions.

Preventive Medicine Services

The Employee Health Program for Healthcare Workers

Due to the wide range of potential health hazards present in healthcare facilities, healthcare personnel must also be included in the installation's Employee Health Program. Compared to the total civilian workforce, hospital workers have a higher rate of workers' compensation claims for sprains and strains, infectious and parasitic diseases, dermatitis, hepatitis, mental disorders, eye diseases, influenza, and toxic hepatitis.[24]

Some of the health hazards encountered in the healthcare environment differ from the hazards at installations, but the Employee Health Program for healthcare personnel is the same as that for other workers. Furthermore, the same staff provides em-

ployee health services to both populations. The program attempts to achieve two objectives: first, to provide a safe and healthy working environment; and second, to assist the employee in maintaining optimal health and efficiency in his or her job.

Infectious diseases are a major concern for healthcare personnel. Not only can infections be transmitted to healthcare personnel, but through them, *secondary transmission* to patients can also occur. Close coordination with the infection-control nurse and the infection-control committee, continuing education, and environmental control, are required in the MTF. Strict compliance with regulations and with the guidance of the infection-control program is essential. These regulations include

- DoD 6055.5-M, *Occupational Health Surveillance Manual*;
- U.S. Army Environmental Hygiene Agency (USAEHA) Technical Guide 143, *Evaluation of Occupational Exposure to Ethylene Oxide in Health-Care Facilities*, which will be published in Technical Bulletin Medical (TB MED) 512;
- USAEHA Technical Guide 149, *Guidelines for the Preparation, Administration, and Disposal of Cytotoxic Drugs*;
- TB MED 510, *Interim Guidelines for the Evaluation and Control of Occupational Exposure to Waste Anesthetic Gases*; and
- AR 40-14, *Control and Recording Procedures for Exposure to Ionizing Radiation and Radioactive Materials*.

Employees who work in hemodialysis units, blood banks, and biological laboratories face significant health risks as the result of occupational exposures to blood and other potentially infectious materials that contain blood-borne pathogens. Blood-borne pathogens include the hepatitis B virus, which causes hepatitis B, and the human immunodeficiency virus (HIV), which causes the acquired immunodeficiency syndrome (AIDS). *Hepatitis B is the major infectious occupational health hazard in the healthcare industry.* Both the hepatitis B virus and the HIV virus can lead to a number of life-threatening conditions including cancer. (The risks associated with these blood-borne pathogens are discussed further in Chapter 5, Health Hazards to Healthcare Workers.) OSHA is currently developing an exposure standard for occupational exposure to blood-borne pathogens.

Certain aspects of the Employee Health Program are important to healthcare personnel because they can help prevent nosocomial infections, including immunizations, monitoring absences due to illness, and pregnancy surveillance, which are discussed later in this chapter. Monitoring absences due to illness is especially important in controlling the spread of infections from healthcare personnel to patients, and to identify and document the occupational diseases that are transmitted from patients to employees. The transmission of infections signals that prevention mechanisms may not be operating optimally and may dictate that an epidemiological investigation be initiated.

Healthcare personnel are at risk for potential exposure to chemical, physical, and biological hazards. As in any other work area, the industrial hygienist must develop a comprehensive health-hazard inventory that identifies the hazards in the MTF. (These hazards are addressed in Chapter 4, Industrial Hygiene.)

Employee Health Education

Job-related health education, which trains employees about the health hazards associated with their occupations, is mandated by law and is primarily the responsibility of supervisors.[20,25] They need the support of several disciplines—safety, nursing and medicine, industrial hygiene—and the civilian and military personnel office training staff to adequately train their employees regarding the diverse hazards of the work environment.

The nurses and physicians educate the employees regarding the health consequences of workplace hazards. The information they provide should include the signs and symptoms of exposure to each hazard, the appropriate emergency medical treatment for acute exposure to each hazard, the required medical surveillance for each hazard, and the necessity for PPE or clothing for each hazard. The Employee Health Program staff may provide job-related health education individually during job-related health evaluations, or to groups of employees who are exposed to the same hazards. Documentation of the education is required in the occupational medical record.[5]

Job-related health education also encompasses other areas that need the support of the Employee Health Program staff. These include

- orientation and guidance of supervisors regarding their responsibilities for employee health,
- orientation of employees to the services that the Employee Health Program provides, and
- modification of the work practices that can be changed by health education to prevent injury or illness.

Physicians and nurses also provide general health education to assist the employee to achieve optimal health. This education is usually given at the time that the employee receives healthcare or approaches the employee health staff with questions or problems. Small-group sessions may address specific conditions or habits when a group of workers is identified to have a common problem and help is requested. Group education may be especially effective in dealing with health problems or habits such as diabetes (or other chronic diseases), obesity, and smoking.

Healthcare personnel may distribute educational pamphlets to employees as a supplement either to group sessions or to individual education. This will help to reinforce the training provided. They may also publish health information as articles of interest within the installation, which can be disseminated to the employee population.

Health Promotion

The U.S. Army Health Promotion Program, an installation command program, is designed to promote and maintain the physical well-being and fitness of both military personnel and civilian employees.[26] Although civilian employees are encouraged to participate in fitness and exercise programs, only employees in jobs with physical fitness requirements, like firefighters, will be granted regular time off from work to participate in physical fitness training.

The commander may approve up to 3 hours of administrative leave per week to allow other civilian employees to participate in command-sponsored physical fitness exercise training, monitoring, or education. These activities must be an integral part of a total fitness program and be limited to 6 to 8 weeks in duration. Employees may, however, participate in these programs in their off-duty time.

The physical fitness program responsibilities of the Employee Health Program staff are minimal. They may provide medical examinations for the employees in jobs that have physical fitness standards in order to determine their ability to participate in an exercise program. The occupational health nurse may refer employees or may seek consultation from the health promotion program staff for certain programs such as nutrition or smoking cessation, in order to provide these in the Employee Health Program.

The Employee Health Program services include health promotion activities such as (*a*) small-group counseling, (*b*) mass disease screening, and (*c*) voluntary health maintenance examinations. All health promotion activities for civilian personnel are the lowest priority and are offered only when the resources are available.

Small-Group Counseling. The occupational health nurse may conduct small-group counseling sessions on specific problems or habits that affect employee health. Before planning these programs, it is essential to perform a *needs assessment* (that is, to determine the perceived medical needs and interests of the employees). Small-group counseling programs require their participants to make a lifestyle or a behavioral change. The programs must be designed to help individuals adopt positive health-related behaviors, such as smoking cessation, stress management, and good nutrition, to help them attain a higher level of well-being. The planners of these programs should optimally utilize available MTFs, installations, or local community health resources such as the American Heart Association or the American Cancer Society.

Mass Screening for Diseases. Another voluntary health promotion activity that the Employee Health Program may perform is mass screening for diseases—either single-disease or multiphasic-disease screening. Single-disease screening may detect one medical condition such as high blood pressure, while multiphasic-disease screening offers tests such as those for blood sugar, hearing, and vision. In planning a mass screening, coordination with the MTF and other community resources can be advantageous. In addition, the following questions need to be considered:

- Do the prevalence and seriousness of the disease or condition justify the cost of intervention?
- Is the procedure appropriate to the health goals of the age group, and is it acceptable to the population?
- Is the procedure relatively easy to administer, easy to interpret by healthcare professionals, and available at reasonable cost?
- Are resources available for follow-up diagnosis or therapeutic intervention?
- Will appropriate referrals be made as indicated?
- Is there an evaluation of the mass screening program's effectiveness?

Health Maintenance Examinations. While health maintenance examinations can be offered to civilian employees, they are not a requirement for the job; however, employees are encouraged to participate. The examinations should be tailored to specific health goals and professional health services that are appropriate for different age groups, rather than performing identical tests and examinations for the entire employee population. Regardless of the scope of the examinations, appropriate follow-up is crucial.

Administrative Medical Examinations

A basic premise of employment by the federal government is that employees must be fully qualified to perform the essential duties and responsibilities of their positions safely and efficiently, without undue risk to themselves or others. However, employees need to have only the minimum physical abilities that are necessary to fulfill this requirement. The purpose of performing an administrative medical examination is to determine the employee's physical and mental ability to perform the job. Management needs this information to make employment decisions: virtually all employment-related decisions involving an applicant's health status are made by management, not by physicians. The role of the examining physician in employment decisions is limited to determining the stability of the individual's medical condition and whether the individual meets the medical requirements of the job.

The Office of Personnel Management has established *medical standards* and *physical requirements* for a few specific jobs.[27] Medical standards describe (*a*) the minimum health status or fitness level that has been determined to be necessary for safe and efficient performance and (*b*) the medical conditions that are considered to be disqualifying for certain jobs. Physical requirements differ from medical standards in that they describe the physical abilities that an employee must have to perform the tasks included in a job, such as the ability to lift 50 lb.

The types of physical examinations that are administrative include (*a*) preplacement examinations, (*b*) periodic examinations, (*c*) fitness-for-duty examinations, and (*d*) disability retirement examinations.

Preplacement Examinations. Preplacement examinations (that is, examinations that are performed before the employee commences working) enable the occupational health physician to assess the employee's physical abilities and limitations with respect to job requirements, and to document baseline data for future use in the evaluation of potential workplace exposures. Preplacement examinations also identify susceptible individuals who might be at higher risk for developing diseases related to specific occupational exposures; for example, people of Mediterranean heritage can develop hemolytic anemia after being exposed to reducing agents, whereas their similarly exposed but non-Mediterranean coworkers will not. This helps to ensure that employees' job placements are safe.

Office of Personnel Management policy limits mandatory preplacement medical examinations to personnel who are applying for positions that have specific physical fitness standards, potentially hazardous duty exposures, or when the examination is required by the employing agency.[27] The local Civilian Personnel Officer determines the positions that require preplacement health evaluations. Generally, most wage-grade positions are considered arduous or hazardous and require a preplacement examination.[28] By comparison, GS positions are considered light duty and do not require a preplacement examination, except for a few occupational series.[11]

The local occupational health physician determines the specific content of preplacement examinations. The physician must consider the duties and requirements of the position including environmental factors, legal and regulatory requirements, and any other factors that are directly relevant to determining the applicant's ability to perform the job safely and efficiently. Additionally, if the applicant is assigned to a work environment that is potentially hazardous to his or her health, the occupational health physician must administer baseline screening tests and examinations (an audiogram for noise exposure or an electrocardiogram for nitroglycerin exposure) that are specific to the hazards. All assessments should include

- a personal and family health history, including a reproductive history for both men and women, and a smoking and alcohol history;
- a detailed work history, including the length of employment in each job and the nature and duration of exposure to hazardous conditions; and
- a general medical history.

Certain forms must be completed to document the administration and findings of the preplacement examination. The SF 78, *Certificate of Medical Examination*, and the SF 93, *Report of Medical History*, are required for all appropriated-fund applicants who are required to undergo a preplacement examination. Similarly, DA Form 3437, *Certificate of Medical Examination*, and the SF 93 are required for nonappropriated-fund applicants. The Civilian Personnel Office usually forwards the SF 78 and DA Form 3437 to Employee Health Program staff members, indicating the specific job's physical requirements and environmental factors.

Although the Office of Personnel Management does not require a preplacement examination for applicants for positions with duties that are usually sedentary or only moderately active, these applicants must complete forms prior to employment to establish their medical qualifications. Appropriated-fund applicants must complete the SF 177, *Statement of Physical Ability for Light Duty Work* and nonappropriated-fund appli-

cants must complete DA Form 3666, *Statement of Physical Ability for Light Duty Work*. Even though a preplacement examination is not required for light-duty workers, Employee Health Program personnel must obtain a general medical history for these applicants using the SF 93, *Report of Medical History*. Baseline screening, such as for blood pressure, vision, and hearing, is recommended if resources are available. Additionally, hazard-specific baseline screening tests must be done if the light-duty applicant will be assigned to a potentially hazardous work environment.

Periodic Examinations. The purpose of administrative periodic examinations is to verify an employee's continuing ability to perform the job. However, the purpose of periodic job-related examinations is to determine the effect of the job on the health status of the employee. Although the Office of Personnel Management specifically requires that only a few positions have periodic examinations, it is good occupational health practice to perform annual examinations on all employees whose positions have medical standards or physical requirements.[27,29,30] Several ARs address periodic examinations, including AR 420-90, *Fire Protection;* AR 190-56, *The Civilian Police and Army Security Guard Program;* and AR 600-55, *Motor Vehicle Driver and Equipment Operator Selection, Training, Testing, and Licensing.*

The administrative periodic examination requires that the Employee Health Program staff record the results. For appropriated-fund civilian employees, either the SF 78, *Certificate of Medical Examination*, or SF 88, *Report of Medical Examination*, is used to record results. The results of administrative periodic examinations for military personnel are recorded only on SF 88.

Fitness-for-Duty and Disability Retirement Examinations. Fitness-for-duty and disability retirement examinations determine the employee's continued capability to meet the physical or medical requirements of a position.[27,31] A federal agency may offer a medical examination that includes a psychiatric evaluation or psychological assessment, or it may request the employee to submit medical documentation in any situation in which it is in the government's interest to obtain medical information that is relevant to an individual's ability to perform safely and efficiently. Whenever an agency either orders or offers a medical examination, or requests medical documentation, the employee must be informed in writing of the following:

- the reasons for the examination,
- the individual's right to submit medical information from his or her own physician, and
- the agency's obligation to consider information from the individual's physician.[27]

The employee must submit medical documentation when a change in duty status, working conditions, or any other benefit or special treatment for medical reasons is requested. The agency requesting the medical examination or documentation must pay the reasonable costs that are associated with obtaining the examination or information. Should the employee request the examination while in the process of applying for a benefit or special consideration (such as extended sick leave or reassignment), the employee is responsible for paying the cost of the examination.[27]

The supervisor is responsible for making requests for fitness-for-duty examinations through the Civilian Personnel Office. The Civilian Personnel Office then forwards all background data that pertains to the request to the Employee Health Program staff. This information is essential for the physician to make a valid medical determination.

Monitoring Absences due to Illness

Supervisors and the Civilian Personnel Office are responsible for absentee personnel. However, the Employee Health Program staff should contribute significantly to keeping employees healthy and present on the job, to minimize the cost of the time lost because of absences due to illness. Medical monitoring ensures that the employee is well enough to perform his job safely, but it is not a means to monitor the use of sick leave. At no time should the Employee Health Program staff be requested or used to check on suspected malingerers when they are out of work on personal, sick, or workers' compensation leave. The identification and control of the abuse of leave benefits is a supervisory and Civilian Personnel Office responsibility. Management, employees, and unions should understand this for the monitoring of absences due to illness to be effective.

The requirement and responsibilities for monitoring absences due to illness must be included in the installation occupational health regulation so that the medical aspects can be implemented. The medical monitoring of absences due to illness includes evaluating, treating, or referring employees who become ill or are injured during duty hours. The purposes of this service include the appropriate disposition of ill employees, the staff's provision of health education, and the staff's increased awareness of the types of health problems in the employee population. The monitoring program also increases the staff's capability to detect sentinel job-related health effects by knowing

the reason for employees' absences from work. (Man-hours can also be conserved if palliative treatment is provided for minor illnesses or injuries and employees are able to quickly return to their jobs.)

The Employee Health Program staff should evaluate the health status of civilian employees who are returning to work after an absence due to illness.[5] This evaluation is essential whether or not the absence was occupationally related: it ensures that the employee is well enough to work. The results of the evaluation may reveal that the employee cannot return to his or her regular job, but is able to perform some other type of work. The occupational health physician can then recommend work limitations. An evaluation after an absence due to job-related illness is also essential to document the accident or illness accurately in the occupational health record.

The occupational health physician and the Civilian Personnel Office determine the duration of the absences and the illnesses that require an evaluation for all personnel except those who are off due to job-related cases, personnel in patient-care positions, and personnel who handle food. These employees are required to report to the staff for evaluations after any illness.[5] As a rule, instructing all other employees to clear through the Employee Health Program after a nonoccupational absence due to illness that is longer than 5 working days will meet the medical monitoring objective without overburdening the Employee Health Program staff.

Monitoring absences due to illness includes performing medical evaluations when they are necessary in support of the Federal Employee's Compensation Act claim *controversions* (that is, legal actions that are taken when the employer does not agree with the employee that a compensable illness or injury has occurred). Medical evaluations are also necessary for employees who are expected to be absent from work for 2 or more weeks due to a job-related illness or injury.[5] These evaluations may require only a review of medical reports or they may call for an appropriate examination. The occupational health physician should request specialty consultation when it is indicated.

Immunizations

Some occupational settings may increase an employee's risk of infection. For example, the potential for increased exposure to biological hazards exists in hospitals, medical and dental clinics, animal-care facilities, child-care centers, biological research laboratories, and waste-disposal facilities. Employees who travel to foreign countries in an official capacity are also considered to have an increased risk of infection.

The immunization program for healthcare personnel provides those who are at risk with appropriate immunizations and chemoprophylaxis. An OTSG policy letter mandates hepatitis B immunizations for all active-duty members of AMEDD.[32] This policy letter also advises that civilian healthcare personnel at risk should individually be encouraged to be immunized, unless immunization is specifically mandated in their work agreements or job descriptions. In addition to hepatitis B immunizations, the Employee Health Program may provide

- rubella, tetanus, and influenza immunizations for medical and dental personnel;
- rabies prophylaxis for veterinary staff and animal handlers;
- specific vaccinations for the staff who work with pathogens in research and medical laboratories; and
- tuberculosis screening for high-risk dental, medical, and veterinary personnel.

The staff provides or arranges for appropriate immunizations, chemoprophylaxis, and other preventive measures against communicable diseases such as tetanus, rubella, and tuberculosis. Before determining which immunizations and chemoprophylaxes should be offered to civilian personnel, the occupational health physician must assess the specific biological hazard and the potential effectiveness of a vaccine to control the problem. The occupational health physician should use the current *Recommendations of the Immunization Practices Advisory Committee*, which is published by the U.S. Public Health Service, Centers for Disease Control, as a reference in making these determinations. Immunizations for military personnel must comply with AR 40-562[33] and guidance from the OTSG.

The staff may also offer immunizations that are not job-related. For example, influenza immunizations are offered to civilian personnel in an effort to reduce absenteeism due to illness.

Surveillance of Employees Exposed to Reproductive Hazards

Surveillance minimizes occupationally engendered risks to the health of the pregnant employee and her fetus, and to the health of the reproductive system of the male employee. Therefore, this surveillance must include both male and female civilian and military employees, because the biology of reproduction demonstrates clearly that both males and females are at risk from workplace hazards to the reproductive system,[34] and is emphasized for nursing, dental, medical, and veterinary personnel.

In addition to evaluating pregnant women for potential exposures to chemical, physical, and biological hazards, exposures to nosocomial hazards (such as cytomegalovirus, herpesvirus, and rubella) in the MTF must also be evaluated. Pregnant women should avoid unnecessary or regular contact with patients who either have hepatitis or who could be carriers of the hepatitis virus: maternal infection with hepatitis B virus in the latter stages of pregnancy may cause significant illness and death to the newborn.

A pregnancy surveillance program is the responsibility primarily of the occupational health nurse, in accordance with guidelines that the occupational health physician develops. The NIOSH Research Report is an excellent reference for pregnancy surveillance.[35]

One of the first steps in implementing a pregnancy surveillance program is for the occupational health nurse to coordinate with the Civilian Personnel Office and supervisors in instituting a policy and procedure that ensures that civilian workers contact the Employee Health Program staff as soon as they know they are pregnant. The program for military women should be coordinated with the obstetrical or gynecological nursing staff or the community health nurse.

Work areas or occupations that contain potential hazards to the reproductive system must be identified through the health-hazard inventory developed by the industrial hygienist. All female employees who are of childbearing age and all male employees who are assigned to these areas must not only be informed that potential hazards to their reproductive systems exist, but also be informed of the effects of those hazards during their preplacement, periodic, and termination examinations.[5] Any history of infertility should also be evaluated from an occupational standpoint. Women must be provided information on the availability of job accommodation or transfer in the event of pregnancy, if this is necessary to protect the mother or her fetus. In addition, the potential hazards to the reproductive system that are identified and the education and counseling that are provided must be documented in the individual's occupational medical record.

When an employee contacts the Employee Health Program staff to inform them of her pregnancy, a nurse or physician must interview the employee and document a health and work history. The staff must determine at that time if the employee may continue working safely in her current job. In addition, the staff must obtain and record data in the employee's occupational medical record. These data include the expected date of confinement, previous pregnancies, home exposures, hours of work, and other pertinent information.

If the environment is safe, the Employee Health Program staff need not periodically follow up this initial evaluation. However, the interviewer should advise the employee during the initial evaluation that the occupational health nurse should be contacted immediately if any changes occur in her work environment.

Coordination between the Employee Health Program staff and the pregnant employee's attending physician is essential. The occupational health physician is familiar with the demands and exposures of the job, but the attending physician might be unfamiliar with the potential hazards that are present in the employee's work environment. When questions that are related to the safe job placement of a pregnant employee arise, a discussion will lead to a more valid mutual decision as to whether the employee may continue to work in her present position or will require a transfer for job accommodation.

The coordination between the employee's physician and the Employee Health Program staff continues even after the employee's pregnancy. While the employee's attending physician must clear the employee's return to work after maternity leave, the occupational health nurse must also evaluate the employee. The occupational health nurse should evaluate the pregnancy outcome and document the results in the employee's medical record.

Surveillance of Employees with Chronic Diseases or Physical Disabilities

Surveillance of personnel with chronic diseases or physical disabilities ensures that the employees' optimal health status is maintained and that no adverse effects result from interactions of the job with the chronic illness or disability. If this surveillance is effective, these employees can remain active members of the workforce.

The staff should identify employees who have chronic diseases or disabilities that may affect or be affected by their work assignments. This can be accomplished by

- reviewing SF 177, *Statement of Physical Ability for Light Duty Work;*
- reviewing SF 93, *Report of Medical History;*
- performing preplacement, periodic, and return-to-work examinations; and
- performing mass screening programs.

Once employees with chronic diseases or physical disabilities have been identified, standard medical practice dictates that they be medically evaluated to determine their work capabilities and to ensure safe

job placement. If the evaluation indicates that the employee is unable to perform all of his or her job duties, the Employee Health Program staff should notify the employee's supervisor.

The frequency of follow-up evaluations varies depending on the employee's specific condition. A follow-up evaluation may consist of either a telephone call by the Employee Health Program staff to the employee to inquire about the health status and to offer counsel, or a visit by the employee to the employee health service for further evaluation and counseling. During these evaluations, the employee should inform the staff of any changes in his or her condition.

The staff should document medical evaluations in the employee's medical record. Documentation should include clinical data regarding the disease or physical disability, its current treatment, and the name of the employee's personal physician. DA Form 5571, *Master Problem List*, is used to document current diagnoses and medications used currently or recently. The staff should update this information whenever the employee is examined or counseled.

Epidemiological Investigations of Occupational Illnesses and Injuries

Epidemiological investigations must be conducted after a suspected or proven occupational illness has occurred and after excessive numbers of occupational injuries have been identified.[5] For example, CTS among workers in specific job categories such as clerks and computer operators has recently been a topic of concern (Figure 3-7):

> CTS results from repetitive flexing and bending of the wrist, which causes tendons to swell, increasing pressure in the carpal channel and pinching the median nerve.[5]

The association between the illness or injury and the potential presence of an unhealthy or unsafe work environment is evaluated in an epidemiological investigation. Inquiries must extend beyond individual cases to the identification of the total population at risk for the same illness or injury. The investigation must be coordinated with the industrial hygienist and other preventive medicine personnel as needed, safety officials, and other personnel who may have a responsibility or an interest in the nature and the magnitude of the problem.

Epidemiological investigations range from a simple review of the individual's work and health history with an evaluation of the exposure at the worksite, to a detailed study involving sampling and laboratory analysis of suspected agents, medical examinations and tests, and a literature review. Frequently, investigations require the assistance of the supporting medical center. If the MEDCEN's capabilities are limited, epidemiological consultation is available through command channels from the USAEHA and the Walter Reed Army Institute of Research.[5]

Clinical Services

Clinical services are also provided to civilians who work for the army, including (*a*) emergency treatment of illnesses and injuries and (*b*) alcohol and drug-abuse prevention and control programs. (The latter is included in this section because it can be clinically relevant; however, the cause is not considered to be job related.)

Emergency Treatment of Illnesses and Injuries

Civilian employees are generally not eligible for definitive diagnoses and treatments of nonoccupational injuries and illnesses under the Employee Health Program. However, there are two exceptions to this. First, in an emergency, the civilian employee will be given the medical attention that is required to prevent the loss of life or limb, or to relieve suffering until he or she can be cared for by a private physician. And second, civilian employees may also be treated for minor disorders, including first aid or palliative treatment, when the employee would not reasonably be expected to seek attention from a private physician. This reduces absences due to illness by enabling the employee to complete the current workshift before consulting a private physician. However, requests for recurrent treatment of the same nonoccupational disorders are discouraged. If continued care is necessary, the employee should be referred to his or her private physician.

Minor treatments or services for nonoccupational conditions, such as administering allergy injections, monitoring blood pressure, and changing dressings, can be provided at the discretion of the occupational health physician in charge. Requests for allergy injections must be submitted in writing and signed by the employee's private physician, and the employee must provide the medications that are required. These services are provided so that lost work time can be avoided.

Arrangements for emergency medical care during nonduty hours of the MTF should be made for those employees who work hours other than the normal day

Fig. 3-7. The section through the wrist at the distal row of carpal bones shows the carpal tunnel. Increase in size of the tunnel structures caused by edema (trauma), inflammation (rheumatoid disease), ganglion, amyloid deposits, or diabetic neuropathy may compress the median nerve. The person with carpal tunnel syndrome will develop atrophy of thenar muscles due to long-standing compression of the median nerve, will oftentimes experience gradual numbness of the fingers while driving, and may be awakened at night by tingling and/or pain in the thumb, index, and middle fingers. Reprinted with permission from Netter FH. The CIBA Collection of Medical Illustrations. Vol 1, *Nervous System*. Part 2, *Neurologic and Neuromuscular Disorders*. West Caldwell, NJ: CIBA-Geigy; Slide 3485; 212.

shift. Firefighters or guards who are appropriately trained may provide emergency care, or employees might be referred to nearby community hospitals or physicians.

Prevention and Control of Alcohol and Drug Abuse

The U.S. Army Alcohol and Drug Abuse Prevention and Control Program (ADAPCP) is an installation command-sponsored program that addresses alcohol and other drug abuses and related activities in a single program. Military employees are not just eligible for the program; participation is mandatory for all soldiers who are enrolled in the ADAPCP by their commanders. Medical services and clinical support for soldiers being treated in ADAPCP are the responsibilities of the MEDCEN-MEDDAC commander. While civilian employees are eligible for the program, their enrollment is voluntary. Employees with an alcohol- or drug-abuse problem are encouraged to seek assistance and counsel from ADAPCP.[37,38] In the program for civilians, the functions of the Employee Health Program staff include initial counseling, referring employees to treatment resources, and performing the initial medical evaluations of employees who enter the program.

SUMMARY

The U.S. Army operates over 130 Employee Health Programs worldwide for its military and civilian employees. Although these programs' administrative structure, professional staffing, and areas of emphasis are quite diverse, they all are based on the same set of laws, DoDIs, and ARs. All have the same basic missions: to prevent negative interactions between the job and the workers' health and to provide job-related, clinical healthcare services.

The health program for the army's civilian employees is composed of (*a*) preventive medicine services and (*b*) clinical services. The preventive elements include job-related medical surveillance, administrative medical examinations, surveillance of employees exposed to reproductive hazards, monitoring of absences due to illness, surveillance of personnel with chronic diseases or physical disabilities, job-related immunizations, health education, epidemiological investigations, and health-promotion programs. The clinical elements include treatment of job-related illnesses and injuries, palliative treatment of minor illnesses, and alcohol- and drug-abuse programs. Because military personnel receive routine healthcare through MTFs, job-related medical surveillance is the only Employee Health Program element that is usually provided to soldiers.

REFERENCES

1. 20 CFR, Part 10(a).
2. US Department of the Army. *Insurance and Annuities*. Washington, DC: DA; 1983. Army Regulation o90-800, Subchap 1-1.
3. US Department of the Army. *The Administration of Army Morale, Welfare, and Recreation, Activities and Nonappropriated Fund Instrumentalities*. Washington, DC: DA; 1988. Army Regulation 215-1.
4. US Office of Personnel Management. *Injury Compensation*. Washington, DC: OPM; 1988. Federal Personnel Manual, Chap 810.
5. US Department of the Army. *Preventive Medicine*. Washington, DC: DA; 1990. Army Regulation 40-5.
6. USA Health Services Command. *HSC Operating Program—Preventive Medicine Program for MEDCEN/MEDDAC*. Fort Sam Houston, Tex: HSC; 1989. HSC Regulation 40-30.
7. USA Health Services Command. *Organization and Functions Policy*. Fort Sam Houston, Tex: HSC; 1989. HSC Regulation 10-1.
8. US Office of Personnel Management. *Federal Employees Health and Counseling Programs*. Washington, DC: OPM; 1986. Federal Personnel Manual, Chap 792.
9. US Department of the Army. *Staffing Guide for US Army Medical Department Activities*. Washington, DC: DA; 1974. DA Pamphlet 570-557.
10. US Department of the Army. *Commissioned Officer Classification System*. Washington, DC: DA; 1990. Army Regulation 611-101.
11. US Office of Personnel Management. *Qualification Standards for Positions Under the General Schedule*. Washington, DC: OPM; 1986. Handbook X-118.
12. US Department of the Army. *Quality Assurance Administration*. Washington, DC: DA; 1989. Army Regulation 40-68.
13. US Department of the Army. *Nonphysician Health Care Providers*. Washington, DC: DA; 1985. Army Regulation 40-48.

14. US Department of the Army. *Medical Record and Quality Assurance Administration. Washington,* DC: DA; 1987. Army Regulation 40-66.

15. US Office of Personnel Management. *Basic Personnel Records and Files System.* Washington, DC: OPM; 1987. Federal Personnel Manual, Suppl 293-31.

16. 29 CFR, Part 1910 § 20.

17. 29 CFR, Part 1913.

18. US Army Health Services Command. *Preventive Medicine.* Fort Sam Houston, Tex: HSC; 1985. Army Regulation 40-5, HSC Suppl, App H.

19. US Department of the Army. *Patient Administration.* Washington, DC: DA; 1983. Army Regulation 40-400.

20. *Occupational Safety and Health Act* of 1970, Pub L No. 91-596.

21. 29 CFR, Part 1960.

22. Last JM, ed. *Maxey-Rosenau's Public Health and Preventive Medicine.* 11th ed. New York: Appleton-Century-Crofts; 1980.

23. US Department of Defense. *Occupational Surveillance Manual.* Washington, DC: DoD; 1985. DoD Manual 6055.5-M.

24. US Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health. *Guidelines for Protecting the Safety and Health of Health Care Workers.* Washington, DC: US Department of Health and Human Services; 1988.

25. 29 CFR, Part 1910 § 1200.

26. US Department of the Army. *Army Health Promotion.* Washington, DC: DA; 1987. Army Regulation 600-63.

27. US Office of Personnel Management. *Medical Qualifications Determinations.* Washington, DC: OPM; 1989. Federal Personnel Manual, Chap 339.

28. US Office of Personnel Management. *Job Qualification System for Trades and Labor Occupations.* Washington, DC: OPM; 1969. OPM Handbook X-118C.

29. US Office of Personnel Management. *Physical Requirements.* Washington, DC: OPM; 1979. Civilian Personnel Regulation 950, App W.

30. US Office of Personnel Management. *Physical Standards for Motor Vehicle Operators and Incidental Operators.* Washington, DC: OPM; 1986. Federal Personnel Manual, Chap 930, App A.

31. 5 CFR, Part 831 § 502.

32. Office of The Surgeon General. *Mandatory Hepatitis B Immunization Policy.* Falls Church, Va: US Department of the Army, 17 April 1989. Memorandum.

33. US Department of the Army. *Immunizations and Chemoprophylaxis.* Washington, DC: DA; 1988. Army Regulation 40-562.

34. Rom WN, ed. *Environmental and Occupational Medicine.* Boston: Little, Brown; 1983: 75–83.

35. US Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health. *Guidelines on Pregnancy and Work.* Rockville, Md: USDEW; 1977. NIOSH Research Report. DHEW (NIOSH) Publication 78-118.

36. Ehrlich A. A Pain in the wrist. In: *The Baltimore Sun.* 29 September 1991; 8.

37. US Department of the Army. *Alcohol and Drug Abuse Prevention and Control Program.* Washington, DC: DA; 1988. Army Regulation 600-85.

38. US Office of Personnel Management. *Alcoholism and Drug Abuse Programs.* Washington, DC: OPM; 1980. Federal Personnel Manual, Suppl 792-2.

RECOMMENDED READING

US Department of the Army. *Employment.* Washington, DC: DA; 1979. Army Regulation 690-300. This AR provides details concerning the employment of personnel with chronic diseases or disabilities.

US Department of the Army. *Nonappropriated Funds and Related Activities Personnel Policies and Procedures.* Washington, DC: DA; 1984. Army Regulation 215-3, Chap 16. This AR lists forms used for nonappropriated-fund employees for initiating compensation claims.

US Department of the Army. *Medical, Dental and Veterinary Care.* Washington, DC: DA; 1985. Army Regulation 40-3.

US Department of the Army. *Accident Reporting and Records.* Washington, DC: DA; 1987. Army Regulation 385-40. This AR summarizes reporting requirements for the OSHA Log of Occupational Injuries.

US Office of Personnel Management. *Employment of the Physically Handicapped.* Washington, DC: OPM. 1978. Federal Personnel Manual, Chap 306, Subchap 4. This chapter of the FPM provides details concerning the employment of personnel with chronic diseases or disabilities.

US Army Environmental Hygiene Agency. *Guide for Developing Medical Directives for Occupational Health Nurses.* Aberdeen Proving Ground, Md: USAEHA; 1989. Technical Guide 004. This technical guide provides definitive guidance in the preparation and use of medical directives for the nursing staff.

US Army Environmental Hygiene Agency. *How to Write and Manage Standing Operating Procedures (SOP).* Aberdeen Proving Ground, Md: USAEHA; 1990. Technical Guide 176. This technical guide provides useful guidance in developing an SOP

US Army Patient Administration Systems and Biostatistics Activity. *Medical Summary Report MED 302 User's Manual.* Falls Church, Va: Office of The Surgeon General; 1987. ADSM 18-HAI-RUD-MUS-UM.

Chapter 4

INDUSTRIAL HYGIENE IN THE U. S. ARMY

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INTRODUCTION

HISTORY

THE INDUSTRIAL HYGIENE PROGRAM

RECOGNIZING HAZARDS
- Sources of Information
- Knowledge of the Installation
- Sources of Occupational Illness

EVALUATING HAZARDS
- Monitoring Methods
- Assessing Measurements
- Worksite Sampling Strategy
- Interpreting the Findings

CONTROLLING HAZARDS
- Primary Controls
- Secondary Controls

SUMMARY

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INTRODUCTION

From its inception, the U.S. Army has led in developing and using new technologies. This leadership has certainly been true concerning the recognition, evaluation, and control of potential health hazards at army worksites. Keeping up with the diverse mix of potential hazards associated with army operations in the field and in garrison has allowed army industrial hygienists to maintain a nationally recognized role in both the identification of hazards and the implementation of controls. Soldiers in fighting units are commonly exposed to hazardous materials; industrial hygienists recognize and help to control these potentially hazardous occupational exposures. Controlling even such common chemical hazards as degreasing solvents or carbon monoxide helps to ensure that soldiers' health is in a state that maximizes their ability to project combat power. Similarly, identifying and controlling noise hazards helps to protect a sentry's hearing acuity. The ability to recognize subtle enemy approach signals such as breaking twigs or jingling rifle cartridges helps keep entire units safe. At the army's industrial base installations, industrial hygienists help to prevent the loss of experienced civilian personnel who have been exposed to potentially hazardous materials during the production and repair of ammunition and other equipment.

Both the American Industrial Hygiene Association (AIHA)[1] and the American Conference of Governmental Industrial Hygienists (ACGIH)[2] have defined *industrial hygiene* as

> that science and art devoted to the recognition, evaluation, and control of those environmental factors or stresses, arising in or from the workplace, which may cause sickness, impaired health and well-being, or significant discomfort and inefficiency among workers or among the citizens of the community.[1(p5)]

AIHA and ACGIH also define an *industrial hygienist* as

> a person having a college or university degree or degrees, in engineering, chemistry, physics, medicine, or related physical and biological sciences who, by virtue of special studies and training, has acquired competence in industrial hygiene. Such special studies and training must have been sufficient in all of the above cognate sciences to provide the abilities: (1) to recognize the environmental factors and to understand their effect on man and his well-being; (2) to evaluate, on the basis of experience and with the aid of quantitative measurement techniques, the magnitude of these stresses in terms of ability to impair man's health and well-being; and (3) to prescribe methods to eliminate, control, or reduce such stresses when necessary to alleviate their effects.[1(p5)]

To meet the scope of the definition, a fully competent industrial hygienist requires an interdisciplinary education covering not only the basic sciences, toxicology, ergonomics, and physiology but also real-world experience with people and the occupational hazards they encounter daily. Army industrial hygienists are generalists; when they couple their scientific knowledge with the art of industrial hygiene, they perform true preventive medicine in the army: eliminating hazards before they cause harm. In this chapter, the term *industrial hygienist* denotes a qualified professional; the broader term *industrial hygiene personnel* includes members of the profession and supporting technical personnel (technicians). The U.S. Army's military industrial hygienists are either Environmental Science Officers (68N) or Sanitary Engineers (68P); army civilian industrial hygienists are classified by the Office of Personnel Management (OPM) as general schedule (GS) 690 or general manager (GM) 690; industrial hygiene technicians are classified in the general OPM series as GS 640, Health Aide and Technician.

HISTORY

The U.S. Army became seriously involved in industrial hygiene during World War I, when workers in military gas-mask manufacturing plants needed protection not only from chemical agent gases but also from typical industrial—chemical and physical—hazards: varying (and various) gas concentrations, solvents, dust, and noise.[3] Both government- and contractor-operated factories received industrial hygiene evaluations from the army during World War I, but those efforts continued only until the war's end.

During the rapid expansion of war materiel production in the late 1930s, the army's chief of ordnance requested medical care for civilian workers. The surgeon general of the army responded by providing the medical care for hundreds of thousands of ordnance workers. However, full identification, evaluation, and control of worksite health hazards was not emphasized until the United States became involved in World War II.[4]

Soldiers who operated the weapons systems and who were exposed to potential hazards of the ordnance itself also received support from the Army Medical Department (AMEDD). An interdisciplinary team of physicians, engineers, and scientists at the Armored Force Medical Research Laboratory, formed in early 1942 at Fort Knox, Kentucky, did pioneering work on heat stress, exposure to toxic gas from weapons firing, the relationship of fitness and fatigue to performance, ergonomics, and human factors engineering. Working in cooperation with other health professionals, industrial hygienists studied equipment systems, predicted potential hazards, and formulated protective responses, as the following examples from that era demonstrate:

- Because dehydration had decreased their ability to function, a tank crew in the Pacific theater failed to engage the enemy, even under the pressure of war.[5] Industrial hygienists did not respond to the actual medical event—in this example, treating soldiers suffering from dehydration. The role of industrial hygienists included attempting to predict and preempt the hazard. For dehydration from heat, increased water intake based on predicted need, not on thirst, is one of several techniques used to protect soldiers and prevent performance degradation. Others include increased air flow with cooler or drier air to cool by convection and evaporation, and enforcing work-rest cycles to reduce metabolic heat loads.
- General officers were convinced to support tank gun ventilation by having them act as gunner and loader in a test-firing of 75-mm shells in an M-4 tank. After four of the planned 10 rounds had been fired, the ammonia levels reached 400 ppm. The generals, weeping copiously and ready to quit the test, realized firsthand the importance of exhaust ventilation:

> The M-4 tank of 1942 had no ventilation provided to specifically meet the needs of the crew. Engine-cooling air was drawn into the turret and through a heat-exchanger to the engine compartment. But in a stationary tank with the engine not operating, the men received no exchange air. Since the 75-mm gun released considerable carbon monoxide and ammonia as the gun breech opened after firing, there was a clear toxic gas hazard that needed to be corrected. This had not been done, I think, because it was usual to practice gun-fire with the turret hatch open.... [S]ystematic measurements of carbon monoxide and ammonia concentrations under various conditions of firing gave convincing proof of the hazard. This led to development of a compact fan to provide the necessary exhaust ventilation. The report recommending installation of such fans [had previously been disapproved] on the grounds that the tank already had too many gadgets![6p24]

During the laboratory's 3 years of operation, researchers at the Armored Force Medical Research Laboratory produced 130 detailed reports of this nature and recommended many improvements to reduce potential adverse health effects and therefore improve the soldiers' fighting capabilities. This laboratory heralded AMEDD's current interest in the interdisciplinary medical consideration of the human component first during the design and development of army systems.[6]

In October 1942, the Department of the Army (DA) established the U.S. Army Industrial Hygiene Laboratory at The Johns Hopkins University to conduct occupational health hazard surveys and investigations in army industrial plants, arsenals, and depots.[4,7] Workers at these facilities had potentially hazardous exposures to militarily unique and common maintenance operations at their worksites. This new laboratory concentrated on four technical and scientific areas: field survey, chemical sampling analysis, engineering design, and medicine and toxicology.

Throughout World War II, personnel at the U.S. Army Industrial Hygiene Laboratory developed and applied industrial hygiene technology to the new and greatly expanded army operations. While the Industrial Hygiene Field Surveys Section did its work at the production and repair plants, the Chemistry Section developed new and improved methods to sample and analyze worksite hazards, the Engineering Design and Development Section conceived innovative controls for industrial hazards, and the Medical Section became more involved in toxicological evaluation of fungicides, insecticides, repellents, flame retardants, and other items with military and industrial applications. The Industrial Hygiene Field Surveys Section and the Engineering Design and Development Section of this laboratory are the specific forerunners of today's U.S. Army Industrial Hygiene Program.

The Industrial Hygiene Field Surveys Section used early, portable, direct-reading instruments to determine carbon monoxide and benzene exposure levels. When potentially hazardous exposures at production facilities involved particulate matter such as toxic dusts or vapors from chlorinated solvents, these early industrial hygienists collected air and bulk samples to be analyzed by the Chemistry Section. They developed the principle that recommendations to control hazard-

ous exposures had to be as practical, fully described, and inexpensive as possible, and could interrupt neither operations nor individual productivity.[8] Simple, low-cost control tactics to reduce the number of people exposed evolved from this principle: physically moving all personnel unrelated to the operation to other, less hazardous locations; isolating essential operational personnel from the hazards (by enclosing the operations); and using less hazardous techniques such as wet grinding or sanding to keep airborne toxic dust levels low.

Because most situations required some additional exhaust ventilation, the Engineering Design and Development Section prepared original designs, reviewed the Field Surveys Section's ventilation proposals, and conducted performance tests of existing ventilation systems. Much of the work involved controlling carbon monoxide from internal combustion engines, firing-range lead fumes and dust, metal fumes from welding operations, toxic pigments from spray finishing, pneumoconioses-producing dusts from abrasive blasting, acid mists from plating, and solvents from degreasing.[8] Interestingly, over 60% of the exposures studied were in these categories.

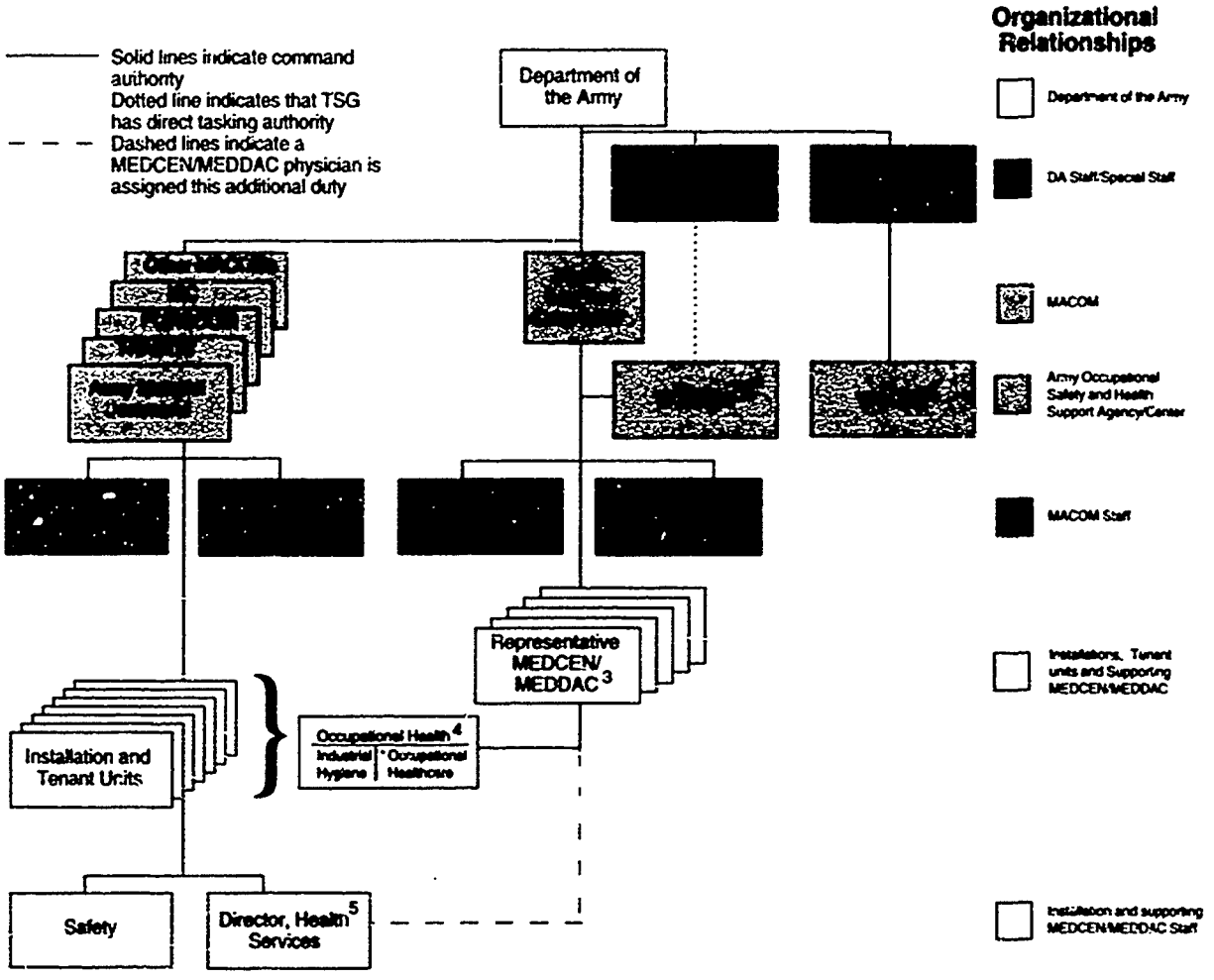
Ammunition loading plants—where open handling of very toxic explosives was commonplace—were the most hazardous facilities that the laboratory personnel evaluated. Workers routinely handled compounds such as trinitrotoluene; amatol; pentolite; tetryl; RDX (research department explosive, also called *cyclonite*: hexahydro-1,3,5-trinitro-1,3,5-triazine); lead oxide; mercury fulminate; and nitroglycerine. In high-explosives and chemical manufacturing plants, workers were also exposed to acids, nitrocellulose, diphenylamine, and ethyl alcohol; and at arsenals and ammunition depots, to solvents, paints, and chemicals related to the repair, maintenance, and renovation of ordnance materiel. Although these early army industrial hygienists were certainly concerned about the very hazardous explosives compounds, relatively few actual exposures to toxic explosives occurred. Much of the credit for this rests with the representatives from the Office of The Surgeon General assigned to the Safety and Security Division, Office of the Chief of Ordnance, who ensured that the public health aspects of worker protection were U.S. Army policy.[8]

When compared to World War I, fatalities caused by occupational diseases were extraordinarily low during World War II. The fact that industrial hygiene personnel identified hazards and recommended control requirements undoubtedly played a significant role in reducing the rates. In 968,000 man-years of operations in explosives manufacture, there were only 28 occupational disease fatalities: 22 from trinitrotoluene, 3 from oxides of nitrogen, 2 from carbon tetrachloride and 1 from ethyl ether. This was a rate of .03 fatalities per 1,000 man-years, or five deaths per billion pounds of explosives produced. In addition, there were 2.4 lost-time general illness and dermatitis cases per 1,000 man-years of operations. Dermatitis accounted for two-thirds of lost-time cases, and the more serious systemic illnesses had a rate of 0.8 cases per 1,000 man-years of production. However, these rates were "only a small fraction"[8(p167)] of the World War I experience. The 44 members of the Army Industrial Hygiene Laboratory helped achieve such low rates of occupational illness during World War II that they firmly established the utility of industrial hygiene and occupational medicine in the army.

The U.S. Army Environmental Hygiene Agency (USAEHA), which operates 31 diverse occupational and environmental health mission programs, evolved from this small World War II laboratory. As installation-level industrial hygiene operations became routine and shifted to the medical units—such as Medical Department Activities (MEDDACs) and Medical Centers (MEDCENs)—that provide installation medical support, the USAEHA concentrated more and more on highly specialized hazards such as chemical agent demilitarization, ammunition production, and healthcare-facility operations. The USAEHA also developed a consultant role in defining and responding to industrial hygiene issues having armywide impact, such as determining the medical requirements for respiratory protective equipment in militarily unique environments, and developing many technical and draft policy documents for new or changing hazards (eg, composite materials like Kevlar, asbestos use and disposal, lead hazards, and cumulative trauma disorders).

THE INDUSTRIAL HYGIENE PROGRAM

The U.S. Army Occupational Safety and Health Program is divided at the DA level into *occupational safety* and *occupational health*. The U.S. Army Occupational Safety Program, (defined in Army Regulation [AR] 385-10, *The Army Safety Program*[9]) is structured along Major Army Command (MACOM) lines and is executed by MACOM safety and operating personnel at the MACOM and installation levels. The U.S. Army Occupational Health Program is a medical program (defined in AR 40-5, *Preventive Medicine*[10]) that is struc-



1. U.S. Army Environmental Hygiene Agency HSC Subcommand; The Surgeon General has direct tasking authority
2. USAEHA and U.S. Army Safety Center provide army-level technical support worldwide
3. MEDCEN/MEDDAC support all installations and units in their geographical area
4. MEDCEN/MEDDAC occupational health support all units in the MEDDAC/MEDCEN area
5. Installation Director of Health Services support provided by MEDDAC/MEDCEN

* Although medical commands outside the continental United States (OCONUS) are in other organizational patterns, they have similar medical report responsibilities for their overseas areas

Fig. 4-1. Organizational relationships between the U.S. Army's Occupational Health Program and various installations. **Red:** The Department of the Army (DA) staffs develop army policy related to occupational safety and health. **Blue:** The Major Army Commands (MACOMs)are operating commands that follow DA policies, during their operations, that help ensure that their personnel follow safe and healthful work practices. Health Services Command (HSC) also has additional responsibility to provide medical support for the other MACOMs and their installations. That medical support includes providing industrial hygiene and occupational healthcare services. **Orange:** The U.S. Army Environmental Health Agency and U.S. Army Safety Center (USASC) act to develop depth and focus for the DA's occupational safety and health policies. They draft new policies for DA staff coordination; help the MACOM implement the approved policies through consultation and technical guidance; and perform oversight, investigation, survey, and study missions. **Green:** The various MACOM staffs work their respective safety and health issues for the MACOM. In addition, the HSC preventive medicine staffs also oversee the occupational health support provided to the other MACOMs by HSC's medical centers (MEDCENs)and medical activities (MEDDACs). **Yellow:** Installation and tenant unit commanders and supervisors are responsible for the occupational safety and health of their personnel. **Tan:** The installation unit safety staff and the MEDCEN–MEDDAC occupational health personnel assist commanders and supervisors to meet that responsibility. The installation Director, Health Services, is an installation staff position filled as an additional duty by a MEDCEN or MEDDAC physician. This medical officer provides advice to the installation commander regarding all medical issues affecting the post.

tured along medical command lines and executed primarily by MEDDAC and MEDCEN personnel, who support all MACOMs and their installations (Figure 4-1). The list of the primary documents that form the legal and regulatory basis of the army's Industrial Hygiene Program is shown in Table 4-1.

Within the Occupational Safety and Health Program, the assigned responsibilities for occupational safety, industrial hygiene, and occupational healthcare are not easy to separate; each area of responsibility has proponents and supporting participants, and there are interrelationships at several points (Figure 4-2). The occupational health portion of the program is divided into two main functional areas: *industrial hygiene* and *occupational healthcare* (which includes both medicine and nursing). Although the control of worksite health hazards is the primary mission of industrial hygiene, it also supports occupational healthcare personnel by

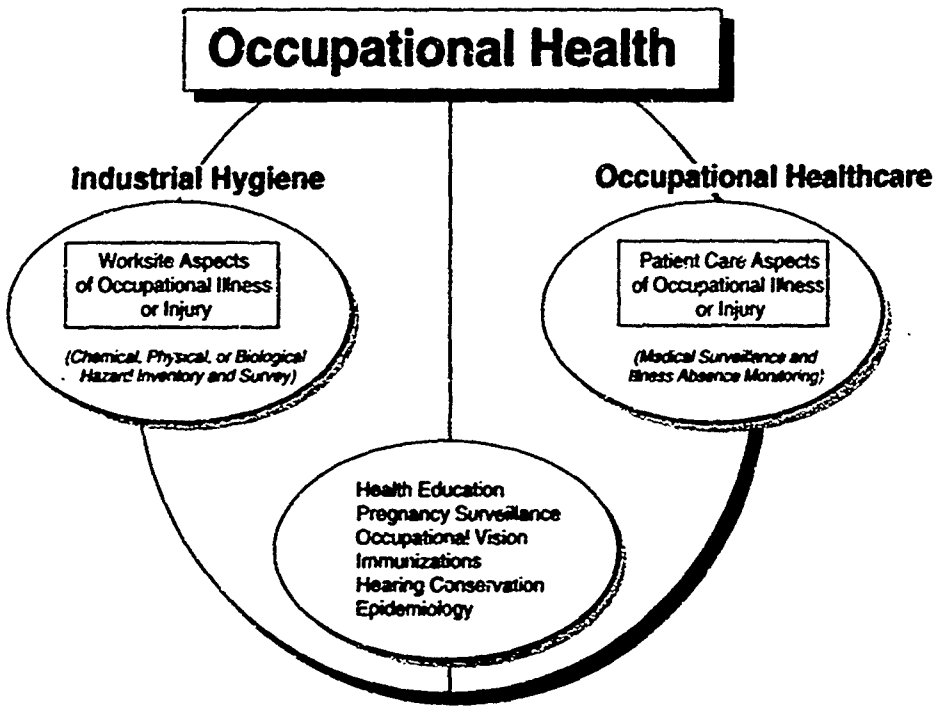
- quantitatively defining the level of worksite exposures to hazardous materials, allowing clinic personnel to (*a*) make informed patient-care decisions regarding medical surveillance and (*b*) target the hazards most likely to cause health effects on workers;
- recommending controls for existing hazards, which, when implemented, can eliminate or greatly reduce medical surveillance requirements; and
- operating the Health Hazard Information Module (HHIM) of the Occupational Health Management Information System (OHMIS), a comprehensive

TABLE 4-1

REGULATORY BASIS FOR AN INDUSTRIAL HYGIENE PROGRAM

Regulation	Description
The Occupational Safety and Health Act of 1970, Pub L No. 91-596	OSHAct, the basic law that requires safe and healthful working conditions for working men and women
Occupational Safety and Health (OSH) Program for Federal Employees, Exec Order No. 12196, 26 February 1980	Order that applies OSHAct standards to all agencies of the executive branch except military personnel and militarily unique situations and equipment
Basic Program Elements for Federal Employee OSH Programs and Related Matters, 29 CFR, Part 1960, rev. 1 July 1987	Regulation to provide OSH Programs for federal employees promulgated by the secretary of labor as required by Exec Order No. 12196
Safety and Occupational Health Policy for the DoD, DoDI 1000.3, 29 March 1979	DoD Instruction that requires adherence to OSHA regulations
DoD Hazard Communication Program, DoDI 6050.5, 29 March 1990	DoD Instruction that prescribes policy and practices for a comprehensive DoD Hazard Communication Program
DoD Occupational Safety and Health Program, DoDI 6055.1, 26 October 1984, rev. 11 April 1989 and 15 August 1989	DoD Instruction that provides policy, procedures, and responsibilities for administration of a comprehensive DoD OSH Program
Industrial Hygiene and Occupational Health Program, DoDI 6055.5, 10 January 1989	DoD Instruction that establishes uniform procedures for recognizing and evaluating health risks associated with chemical, physical, or biological stresses at DoD worksites
Army Safety Program, AR 385-10,* 23 June 1988	Army Regulation that implements safety requirements of federal and defense regulations
Preventive Medicine, AR 40-5,* 19 June 1985	Army Regulation that implements occupational health requirements of federal and defense regulations
Army Industrial Hygiene Program, TB MED 503,* 1 February 1985	Bulletin that explains the organization and responsibilities of the industrial hygiene portion of the Army Occupational Health Program

*Key documents for army installations



Responsibilities	OS&H		
	OS	OH	
		IH	OHc
Safety and health program coordination	P	P	P
Occupational safety functions of AR 385-10	P	R	R
29 CFR 1960 requirements (SAOSHI)	P	P	P
Personal protective equipment	P	P	P
Ergonomics	S	P	S
Epidemiology	R	P	P
Inventory of chemical, biological, and physical hazards	R	P	R
IH surveys	S	P	S

Responsibilities	OS&H		
	OS	OH	
		IH	OHc
Medical surveillance	R	S	P
Health education	R	P	P
Hearing conservation	R	P	P
Occupational vision	R	P	P
Medical treatment	R	S	P
Pregnancy surveillance	R	P	P
Immunizations	R	P	P
Illness absence monitoring	S	S	P
Hazard communication	P	S	S

OS&H: Occupational Safety and Health; OH: Occupational Health; OS: Occupational Safety; IH: Industrial Hygiene; OHc: Occupational Healthcare (includes medicine and nursing)
P: Has primary responsibilities; S: Provides support; R: Referral

Fig. 4-2. Occupational health's two major components—industrial hygiene and occupational healthcare—provide medical support to an installation and its tenant units through the worksite and patient-care aspects of the Occupational Health Program. The Standard Army Occupational Safety and Health Inspection (SAOSHI [CFR 1960]) specifies that both command and supervisory personnel (the Occupational Health Program–installation interface) be involved with issuing and assuring the correct use of personal protective equipment (PPE); communicating the hazards of a worksite to the operators (hazard communication); participating in the annual worksite inspections; and performing other related activities in coordination with supporting occupational safety, industrial hygiene, and healthcare personnel. Usually, commanders designate their own safety officers to work with the supporting medical unit to coordinate accomplishment and oversight of a comprehensive program with the participation of supervisors and supporting medical units. The various safety and occupational health program participants are responsible for performing their own primary missions, supporting other participants, and, at a minimum, ensuring that information regarding hazards is referred for action.

health database that provides exposure and other worksite data to occupational healthcare personnel in an easily accessible and usable form.

Furthermore, the missions of occupational safety and occupational health, especially the industrial hygiene portion, appear, on the surface, to be similar. Confusion frequently exists as to where their mission responsibilities and primacy lie. The primary differences between their missions are that

- occupational safety personnel are mainly concerned with the prevention and control of traumatic injury to personnel, and with accidents that result in loss of materiel; whereas
- industrial hygienists are mainly concerned with factors at the worksite that cause chronic or acute illness, disease, or injury to personnel.

In most instances, the distinction is clear. For example, an overhead crane that drops a load of lumber and injures several people, or a shorted electrical circuit that causes a building fire and electrical burns to personnel are both occupational safety issues. Occupational safety personnel would focus on the cause and prevention of these accidents. But a welder's increased body burden of lead from exposure to fumes from metal coated with lead-containing paint, or a painter's allergic sensitization and respiratory distress after exposure to epoxy resins and isocyanate compounds in chemical agent resistant coatings (CARCs) are industrial hygiene issues.

The two missions intersect where there are dual medical and safety responsibilities. Some situations have both traumatic injury and systemic components. For example, if acid bubbles out of a lead acid battery on high charge and burns a worker's unprotected hands, that is an occupational safety issue; however, the worker's inhaling the acid mist that forms, and the consequent respiratory illness, are industrial hygiene and occupational healthcare issues.

Personal protective equipment (PPE) also involves dual medical and safety responsibilities (see Figure 4-2). For example, the issue and use of respiratory protective equipment has traditionally been the domain of supervisors and occupational safety personnel. However, selecting the proper respirator requires a detailed industrial hygiene exposure evaluation, and the potential user must be medically evaluated before being required to wear a respirator.

RECOGNIZING HAZARDS

Effective industrial hygiene personnel know and follow all the potentially hazardous operations on an installation. They must begin to learn as much as possible about an installation and its industrial operations, processes, and possible hazards as soon as they arrive, and constantly track any changes. The development of this knowledge base will allow them to make valid comparisons and decisions about changes to any of the operations.

Sources of Information

Industrial hygienists can gain information about an installation from (*a*) the HHIM; (*b*) injury reports and complaint logs from clinics; (*c*) chemical inventories and chemical purchase requests; and, certainly, (*d*) referral from safety personnel, union representatives, supervisors, and individual workers.[11,12] The industrial hygienist maintains the HHIM, a database of information about operations collected from the local installation, the USAEHA, or contractor surveys of worksites. The database can be used to generate virtually any type of report required to define existing conditions. Industrial hygiene personnel develop and maintain an up-to-date HHIM using forms that contain the pertinent information regarding the operation, its personnel, and the potential hazards (Figure 4-3).

Injury and complaint logs from clinics, emergency rooms, and duty officers also provide industrial hygienists with records of potentially hazardous locations for surveys. Chemical inventories and chemical purchase requests, especially for newly introduced chemical compounds, are excellent and frequently overlooked sources of information concerning new or changed industrial processes or worksite operations. Hazard communication training and media reporting have increased the level of awareness of potential hazards from occupational exposures: installation safety personnel, union representatives, supervisors, and individual workers now often refer potential problem situations to industrial hygienists for survey.

Knowledge of the Installation

In addition to these sources of information, the industrial hygienist must become familiar with the particular installation's mission, the operations that support the mission, and the supervisors and production workers who compose the workforce.[11,12] The intimate knowledge required is gained only through

Figure 4-3a

HEALTH HAZARD INFORMATION MODULE; INDUSTRIAL HYGIENE SURVEY

*For use of this form, see HHIM User's Guide

SECTION 1. DEMOGRAPHIC DATA

ARLOC	INSTALLATION	BLDG/RM NO.
53456	Ft. Lewis	3516/Bay

LOCATION/CODE	OPERATION/CODE
Vehicle maintenance / GS	Brake Repair / BKR

SURVEY DATE	EVALUATOR (Initials)
910401	TW

MACOM/CODE	SUBMACOM/CODE	SUPERVISOR
FORSCOM / FC	N/A	CW3 Fxxxx

TELEPHONE/DSN NO.	UNIT/ORGANIZATION	RAC	FREQUENCY (hrs/day)
DSN 584-9113	DEH / Motor Pool	3	8

NO. CIV(S)	NO. MIL	NO. CONTRACTOR(S)	NO. LOC(S)	NO. OTHER
3	0	N/A	N/A	N/A

SECTION 2. FACILITY DATA

LAB HOODS	VAPOR DEGREASERS	SPRAY BOOTHS

MAINTENANCE BAYS	OPEN SURFACE TANKS	VENTILATION UNITS
6		1

SECTION 3. SURVEY DATA

CONTROLS PRESENT	EVALUATION	UNIT CODE	CONTROLS REQUIRED	STATUS
LEV	50	FPM	150 FPM	UNCON

PERSONAL PROTECTIVE EQUIPMENT (R = REQUIRED; U = UTILIZED)

GLOVES	R/U	RESPIRATOR	NIOSH TC NO.	MANUFACTURER	R/U
ACID	/	AIRLINE			/
COLD SURFACES	/	ABRASIVE BLASTING HOOD			/
HOT SURFACES	/	DISPOSABLE	None	3M	/ X
NBC AGENTS	/	FULL FACE AIR PURIFYING			/
OIL	X / X	1/2 FACE AIR PURIFYING			/
SOLVENTS	/	POWDERED AIR PURIFYING			/
SURGICAL GLOVES	/	1/4 FACE AIR PURIFYING			/
	/	SELF CONTAINED			/

EYES/FACE	R/U	HEARING	R/U	BODY	R/U	HEAD/FIT	R/U
CHEMICAL SPLASH	/	CANAL CAPS	/	APRONS	/	COLD WEATHER BOOTS/MATS	/
FULLFACE SHIELD	/	EARPLUGS	X / X	COLD WEATHER CLOTHING	/	HARD HATS	/
CHEMICAL/SAFETY	/	HELMETS	/	COVERALLS	/	IMPERMEABLE BOOTS	/
SAFETY/IMPACT	X / X	MUFFS	/	FULL BODY SUIT	/	SAFETY/CONDUCTIVE SHOES	X / X
WELDING HELMET	/	MUFF/EARPLUG COMBO	/	HEAT REFLECTIVE VEST/SUIT	/	SAFETY/NON-CONDUCTIVE SHOES	/
		MUFF/EARPLUG W/TIME LIMIT	/	SAFETY BELT/HARNESS	/		

AEHA FORM 271-R (TEST), 1 JAN 92 (HSHB-MI-I)

Fig. 4-3a–c. These five pages are facsimiles of the documents used to conduct a typical industrial hygiene survey using the Health Hazard Information Module (HHIM) database. The hypothetical data in Section 3, Survey Data, show that local exhaust rates are below standard, that noncertified respiratory protective equipment has been used, and that hearing protection is required and used.

Figure 4-3a (continued)

SECTION 4. HAZARD INVENTORY DATA

CAS CODE	HAZARD DESCRIPTION	PAC	EPC
P0 NOISECO	Noise, continuous	2	D
12172-73-5	Asbestos (Amosite)	1	A
12001-29-5	Asbestos (Chrysotile)	1	A

SECTION 5. PERSONNEL DATA

LAST NAME	FIRST NAME	MI	SEX	SSN	CATEGORY
Fxxx	John	A	M	003-04-0567	Civ
Fxxx	Mike	B	M	004-05-0678	Civ
Wxxx	Keith	C	M	005-06-0789	Civ

SECTION 6. COMMENTS

☐ No Comments ☐ See attached Sheet

PRIVACY ACT STATEMENT

Title 5 US Code, Section 301; Executive Order 9397 authorizes the use of your Social Security Number as an identification number. The purpose of this information is to identify and monitor data relating each DA civilian and military employee exposed to a hazardous workplace or operation. The use of this information is to provide histories of exposures for any given worker.

Disclosure of your Social Security Number is not mandatory; however, nondisclosures may result in untimely provision of proper medical monitoring.

2

Fig. 4-3a. This hypothetical survey identified three civilian workers—John F., Mike F., and Keith W. (Section 5, Personnel Data)—who were exposed to asbestos (Amosite and Chrysotile) and continuous noise from brake-repair operations (Section 4, Hazard Inventory Data). This operation has received a high-priority action code (PAC-1) for asbestos sampling and evaluation, and a moderate code (PAC-2) for noise survey. The exposure potential codes (EPCs) show occupational healthcare personnel that the asbestos exposure is controlled (EPC-A) but that surveillance audiometry is required for noise exposure over 85 dBA (EPC-D) even though hearing protection is worn.

Figure 4-3b

INDUSTRIAL HYGIENE AIR SAMPLE DATA — *8 HR TWA*

For use of this form see USAEHA TG 141; the proponent is HSHB-LO.

Return Address *(complete address including Zip Code)* USA MEDDAC ATTN: PM SVC-IH Ft. Lewis WA 99603	**Point of Contact** *(name/AUTOVON)* George Sxxx DSN 931-4763
	Associated Bulk Samples ☒ Yes ☐ No
Samples Collected By: Jay Jxxx	**Date Collected**: 910401 — **Date Shipped**: 910402
	Bulk Sample No(s):
Project Number	**Sampled Installation**: Ft. Lewis — **ARLOC**: 5 3 4 5 6
Location *(BLDG/AREA)*: 3516 / Bay / GS	**Description of Operation** *(details on reverse)*: BKR
3 Persons Exposed — **8** Hrs/Day	**Method of Collection**: CE Filter
Associated Complaints *(be specific) (state NONE if applicable)*: None	3
Analysis Desired: Amosite Asbestos, Chrysotile Asbestos	

Sampling Data

Sample No.	FTLW01	FTLW02						FTLW03
Pump No.	1234	5678						BLANK
Time On	0730	0731						
Time Off	1530	1531						
Total Time (min)	480	480						
Flow Rate (LPM)	2.0	2.0						
Volume (Liters)	960	960						
GA/3Z	BZ	BZ						
Employee Name/ID	004-05-067	005-06-07[illegible]						
Laboratory No.	AEHA 1	AEHA 2						AEHA 3

Results

Amosite (f/cc)	< 0.00[illegible]	< 0.1						0
Chrysotile (f/cc)	< 0.01[illegible]	< 0.2						0

Comments to Lab:

Lab Use Only

Analyst *(initials)*	Reviewed By *(initials)*	Date Received	Date Dispatched
KS	TL	910403	910404

AEHA Form 9-R, 1 Oct 84 (Replaces AEHA Form 9, 1 Oct 80 which is obsolete).

Fig. 4-3b. Because asbestos is a known carcinogen, the industrial hygienist sampled the air immediately; no exposures in excess of the health standards were found, although the local exhaust did not provide the generally recommended level of control.

Figure 4-3b (continued)

Calibration Information				
Pump No.	Calibration (L/min)		Rotorneter Setting	Date
	Pre-Use	Post-Use		
1234	2.0	1.9		910401
5678	2.0	2.0		910401

Name of Calibrator: Jay Jxxx

Operation

Source of Contaminant: Old brake lining

Operation Employee(s) Perform: Replace brake shoe lining

Ventilation: [X] Local Exhaust [ ] General Area [ ] None

Personal Protective Equipment *(check if worn)*

- [X] Respiratory Protective Equipment Type: Disposable
- [ ] Protective Clothing Type:
- [ ] Gloves Type:
- [ ] Goggles/Face Shield:
- [X] Ear Protection:
- [X] Other: Safety goggles, safety conductive shoes

Field Notes/Additional Comments

2

Figure 4-3c

BULK SAMPLE DATA

For use of this form see USAEHA TG 141; the proponent is HSHB-LO.

Return Address *(complete address including Zip Code)* USA MEDDAC ATTN: PM SVC-IH Ft. Lewis WA 99603	Point of Contact *(name/AUTOVON)* George Sxxx DSN 931-4763

Sampled Installation Ft. Lewis	Project Number	ARLOC 5 3 4 5 6
Samples Collected By Jay Jxxx	Date Collected 910401	Date Shipped 910402
Description of Operation Brake re-lining		Location *(BLDG/AREA)* 3516/BAY/GS

Associated Complaints *(be specific)* None

Associated Air Samples [X] Yes [ ] No — If yes, list sample numbers: FTLW01, FTLW02

Label Information

Trade Name None	NSN	Manufacturer
Address		MSDS Attached [ ] Yes [X] No

Analysis Desired

Amosite and chrysotile Asbestos=C

Lab Use Only	Sample No.	Constituents	Results	Remarks
	FTLW04	Amosite	50%	
	FTLW04	Chrysotile	< 5%	

Comments to Lab:

Lab Use Only

Analyst *(initials)* RS	Reviewed By *(initials)* KJ	Date Received 910403	Date Dispatched 910404

Procedures Performed TEM	Comments

AEHA Form 8-R, 1 Oct 84 (Replaces AEHA Form 8, 1 Oct 80 which is obsolete)

Fig. 4-3c. Bulk samples were also collected to characterize the types of asbestos present. The data elements tie together administrative, exposure, and control information for a particular date. The ability to query the HHIM database allows industrial hygienists to focus key program resources on hazards based on rational criteria such as exposure levels in excess of the standards, estimates of high exposures, the numbers of personnel affected, possible exposures to carcinogens, and so forth. The identification and subsequent quantification of hazards and exposure levels allow industrial hygiene and occupational healthcare managers to aim their limited resources at priority targets.

daily contact with the workforce at the worksite. Only through frequent observation can industrial hygienists see a true picture of potential hazards. Irregular or infrequent worksite visits are simple snapshots; they lead to false impressions of exposure potentials.

Sources of Occupational Illness

To immediately recognize potentially hazardous situations and substances, industrial hygienists must be familiar with a broad range of industrial operations and processes, and know the typical routes of entry, target organs, and actions of the chemical, physical, and biological agents of occupational illness.

Chemicals

Chemicals typically enter and act on the body through (*a*) direct action on the skin; (*b*) direct action on the respiratory system; (*c*) systemic illness via exposure through skin contact, inhalation, or ingestion; or (*d*) irritant or systemic action from the rare occurrence of physical injection of chemicals into the bloodstream. Experienced industrial hygienists realize that they must also be familiar with the relationship of the chemical's route of entry and mode of action to the operation and process involving the chemical, the engineering controls and PPE available, the short- and long-term exposure times, and the potential that an average worker will have an adverse reaction to the chemical.

Dermatitis is one of the leading indicators that workers are overexposed to chemical hazards. Industrial hygienists who know the typical classifications of dermatitides will be able to recognize the signs of chemical dermatitis and link the medical diagnosis to hazardous operations.[13]

- Primary skin irritants cause direct injury after sufficient contact. Strong organic and inorganic acids and bases are prime examples of this group. Sulfuric acid in automotive batteries or sodium hydroxide in strong inorganic cleaning solutions, for example, can cause serious dermal burns and ulcers.
- Allergic sensitizers do not cause visible effects on first contact. However, for some people, after continued exposure even very small amounts will cause dermatitis at the point of contact or even at other parts of the body. The epoxy resins found in CARCs are skin sensitizers commonly found on army installations.
- Drying agents, mainly organic solvents such as acetone, naphtha, xylene, and toluene, remove fats from skin, leaving it dry and susceptible to cracking and secondary infections.
- Occupational acne and other less frequently seen dermatitides such as photosensitivity, neoplasms, and changes in pigmentation can be associated with overexposure to petroleum, oil or grease, tar, and some chlorinated organic compounds such as the chlorinated phenols.

The exchange of information between the industrial hygienist and occupational healthcare personnel is useful when establishing other clinical diagnoses like occupational lung disease, and when identifying sources of exposure. Therefore, industrial hygienists must be knowledgeable about the direct effects of chemicals on the respiratory system (eg, asthma, pneumoconioses, and some cancers). These effects are caused by vapors, gases, and aerosols (ie, particulates suspended in a gas, usually air; smoke and dust are *solid* aerosols, whereas mist and fog are *liquid* aerosols). The relationship of the health hazard to the physical state of the chemical is discussed in the next section of this chapter.

Industrial hygienists must also be aware that chemical changes caused by human metabolic processes can either toxify or detoxify certain chemicals, and they must be able to make appropriate control recommendations (Table 4-2).

The least likely cause of overexposure to hazardous chemicals is the physical injection of chemicals into the body. Although rare, instances have occurred where high-pressure, compressed air from air guns or spray-paint apparatuses have injected pigments, solvents, and other chemicals through the skin (and potentially into the bloodstream) of the worker.

Physical Agents

The main physical agents of occupational concern for the typical army installation–industrial hygienist include noise, radiation, temperature extremes, and ergonomic stresses. Industrial hygienists measure potential noise-hazardous operations; noise and hearing conservation are covered in Chapter 7, Noise and the Impairment of Hearing. Army industrial hygienists need to identify and list radiation hazards in the HHIM so that occupational medicine and nursing personnel can schedule appropriate medical surveillance. However, health physics and the evaluation and control of nonionizing and ionizing radiation have become specialized fields in the army. Although industrial hygienists have radiation training and provide user support, many installations and medical units have specialty Radiation Protection Officers as-

TABLE 4-2

ENTRY AND ACTION OF TOXIC CHEMICALS (example exposures)

Exposure	Route of Exposure	Potential Health Effect	Recommended Control
Amalgam preparation	Inhalation of Hg vapor	Dementia	Enclosed amalgamation preparation Local exhaust Waste control
Firing-range cleaning	Inhalation/ingestion of Pb dust	Colic Palsy Encephalopathy Anemia	Respirators HEPA vacuum
Laboratory procedures	Ingestion/dermal contact with benzidine dye	Bladder cancer	Substitute reagent
Metal-parts cleaning	Inhalation/dermal contact with organic solvent	Cirrhosis	Local exhaust Protective gloves
Pest-control spraying	Inhalation/dermal contact with carbaryl	Depressed erythrocyte cholinesterase	Respirator Protective clothing

Carbaryl: (1-napthyl N-methyl carbamate)
HEPA: high-efficiency particulate air filters, which remove 99.97% of the aerosol particulates > 0.3 μ

signed to do day-to-day occupational safety and health work related to radiation (see Chapter 15, Nonionizing Radiation, and Chapter 16, Ionizing Radiation).

The adverse effects of heat and cold are concerns of industrial hygiene personnel on army installations. Soldiers' field exposures are the concerns of the supporting preventive medicine unit. The U.S. Army Research Institute of Environmental Medicine (USARIEM) publishes results of their investigations into the effects of and response to these exposures.[14-16] Where temperature extremes do occur, the principles of identification, evaluation, and control are applied using established occupational health standards.

The effects of heat and cold are associated with the *net heat balance* between the working environment and the worker's normal body temperature (98.6°F ±1°F). The body's heat balance *H*, which is usually measured as either BTU/hour or kcal/hour in any environment, can be expressed in the equation

$$H = (\pm R) + (\pm C) + M - E$$

where *R* represents the radiant heat gained or lost, *C* represents the heat gained or lost through convection (transferred between the skin and air), *M* represents the metabolic heat gained from varying work rates, and *E* represents the evaporative heat loss through vaporization of sweat.

Measurement of air temperature, air velocities, radiant loads and humidity, and estimates of work rates and clothing insulation will enable trained industrial hygiene personnel to evaluate potentially hazardous heat or cold conditions. Because these environmental conditions are interrelated, measurement tools were developed that integrate several of these factors for use in heat-stress and wind-chill indices (Figures 4-4 and 4-5). Industrial hygienists can use these measurements to determine hazard levels and, in conjunction with review of the operation, can recommend engineering, work practice, and personal protection controls.[17-21]

Ergonomic stresses are a recently expanding field of interest for army industrial hygienists and other professionals such as physical and occupational therapists, occupational health nurses, occupational medicine physicians, and safety officers. However, industrial hygienists evaluate worksite hazards and have the medical background to appreciate the physiology and anatomy required for ergonomic evaluation. Treating existing back and repetitive-motion illnesses or training personnel in proper lifting techniques is not enough; control of ergonomic hazards at their source

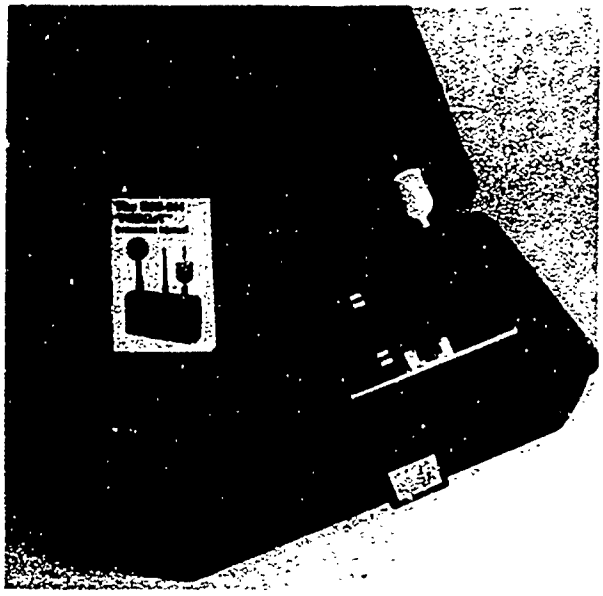


Fig. 4-4. This Reuter Stokes RSS-214 WBGeT Wet Bulb Globe Thermometer electronically records the wet bulb, dry bulb, and black globe temperatures, then calculates a heat-stress index that is used to determine the protection necessary for the stresses of excessive heat. Heat stress is the subject of US Army Research Institute of Environmental Medicine (USARIEM) Technical Note 91-1, *Sustaining Health and Performance in the Desert*,[15] which can be consulted for further information.

Wind Speed (mph)	Actual Temperature (°F)											
	50	40	30	20	10	0	−10	−20	−30	−40	−50	−60
	Equivalent Chill Temperature (°F)											
Calm	50	40	30	20	10	0	−10	−20	−30	−40	−50	−60
5	48	37	27	16	6	−5	−15	−26	−36	−47	−57	−68
10	40	28	16	3	−9	−21	−33	−46	−58	−70	−83	−95
15	36	22	9	−5	−18	−32	−45	−58	−72	−85	−99	−112
20	32	18	4	−10	−25	−39	−53	−67	−82	−96	−110	−124
25	30	15	0	−15	−29	−44	−59	−74	−89	−104	−118	−133
30	28	13	−2	−18	−33	−48	−63	−79	−94	−109	−125	−140
35	27	11	−4	−20	−35	−51	−67	−82	−988	−113	−129	−145
40	26	10	−6	−22	−37	−53	−69	−85	−101	−117	−132	−148
Wind speeds > 40 mph have little additional effect	**Little Danger** (in < 5 h with dry skin; greatest hazard is from false sense of security)				**Increasing Danger** (exposed flesh may freeze within 1 min)				**Great Danger** (exposed flesh may freeze within 30 sec)			

Fig. 4-5. Potential heat loss, skin cooling, and lower internal temperature can be increased by air movement. The wind-chill index integrates windspeed and air temperature to estimate associated risk of cold injury. The wind-chill temperature index is the equivalent still-air (no wind) temperature that would produce the same heat loss on bare skin. A full description of the medical aspects of military operations in the cold is the subject of US Army Research Institute of Environmental Medicine (USARIEM) Technical Note 92-2, *Sustaining Health and Performance in the Cold*. Source of chart: US Army Research Institute of Environmental Medicine Technical Note 92-2. *Sustaining Health and Performance in the Cold: Environmental Medicine Guidance for Cold-Weather Operations*. Natick, Mass: USARIEM; July 1992: 37.

is vital. For example, to eliminate lifting from floor level, industrial hygienists can recommend moving the storage of heavy parts to waist level; to ensure proper wrist position during equipment assembly, they can recommend tools designed to keep the wrist in a neutral position.

Biological Hazards

Biological hazards found on army installations are typically associated with the medical, dental, and veterinary facilities and their supporting agencies such as laboratories. For installations with personnel who spend time outdoors, other typical biological hazards can include such things as poison ivy, insect stings and bites, and arthropod-borne diseases (eg, Lyme disease). Protection is provided though training, avoidance where possible, protective clothing, repellents, and preparation both to identify these outdoor exposures and treat any personnel who report to a clinic. Biological hazards to healthcare workers is the subject of Chapter 5, Health Hazards to Healthcare Workers.

EVALUATING HAZARDS

Worksite exposures change as processes, personnel, and work rates change; as existing controls deteriorate through use; as buildings are modified; and even as seasons change. Therefore, a registry of worksite exposure levels must be maintained to (*a*) prevent hazard assessments based on single samples of potential hazards and (*b*) provide a usable record of increasing or declining exposure trends.

Monitoring Methods

Industrial hygienists use several monitoring methods at worksites to quantify exposure levels.[21-24] The main types of monitoring employ direct reading instruments, indirect measurement (ie, collection of samples for later laboratory analysis), or both. Portable, direct reading instruments are constantly being developed and improved; some in common use include combustion meters, flame ionization detectors, gas chromatographs, photometers, and certain gas-diffusion badges.

Direct Reading Instruments. Instruments that register direct readings allow measurements of worksite exposures to be made in real time. They use analog or digital meters; strip-chart recordings; tape printouts; and color changes in impregnated paper, liquid reagents, or colorimetric glass tubes filled with solid reagents.[22-24]

Direct reading instruments can be used as nonportable monitors to provide a continuous record of chemical concentrations over long periods. They can also be set to sound alarms if worksite concentrations exceed preset exposure level standards. Portable instruments are used to identify sources of potentially hazardous exposures at the worksite, to determine if exposure standards are exceeded, to check engineering controls, and to record exposure.

Chemical detector tubes are narrow glass tubes, sealed at each end, and filled with solid, finely granulated, reagent-impregnated materials (Figure 4-6). The industrial hygienist must first open both ends of the tubes and then pump known volumes of sample air through. Contaminants collect on the media and react to produce a color change. Exposure levels are determined by reading the length of the stain or the degree of color change. However, errors can occur due to chemical interferences, the operator's faulty estimate of the stain reaction, and the quality or age of the reagents.

Another frequently used monitor of exposure levels is the infrared spectrophotometer, which measures the attenuation of specific wavelengths of infrared light as they pass through a gas or vapor sample (Figure 4-7). Infrared spectrophotometers require frequent adjustment, must be calibrated with known concentrations of contaminants, and are subject to interference from chemicals with the same infrared light absorbance spectrum as the target chemical's.

Piezo electrical mass monitors measure aerosol mass by comparing frequency changes in an oscillating crystal exposed to the aerosol with another crystal—one not exposed to the aerosol—used as a blank to cancel out any changes due to temperature, pressure, or humidity (Figure 4-8).[21]

Direct reading instruments have limitations that must be considered before and during their use:

- Although the cost of the least expensive direct reading instrument, a detector tube, is relatively low ($5.00 each), more-accurate and -specific instruments (with electronics and electrochemical cells) can cost more than $5,000 each and can easily exceed $15,000 each.
- Many instruments react to classes or families of chemicals rather than to specific compounds; in some, even completely different chemicals can cause interference (eg, water vapor will interfere with infrared analysis of ethylene oxide on certain instruments).

Fig. 4-6. This GASTEC/Sensidyne pump, model 800 with formaldehyde low range (0.1–5 ppm) detector tubes, is used to rapidly screen areas for formaldehyde gas. Other types of tubes are available to screen for many common chemicals. Each carton contains specific instructions for sample volumes.

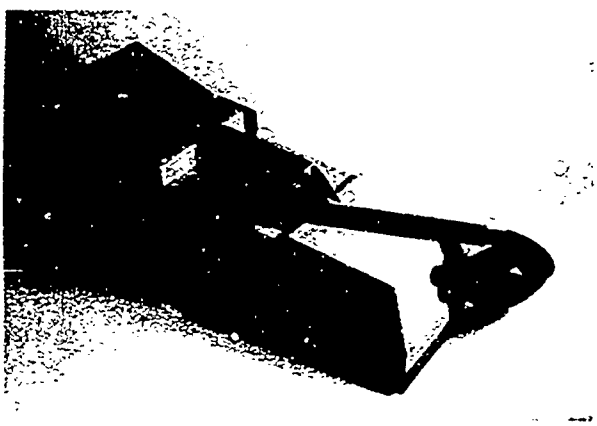


Fig. 4-7. This MIRAN 1B2 infrared gas analyzer provides sub-ppm measurement of a wide variety of gases and vapors. This instrument or a variant is frequently used to monitor for ethylene oxide and waste anesthetics in army medical treatment facilities.

Fig. 4-8. This TSI Respirable Aerosol Mass Monitor Model 3500 uses the frequency changes of piezo crystals to determine the mass of 0.01–10 μ particles in air.

- Separate direct reading instruments to measure all the chemicals that might be present at a worksite may be difficult to carry.
- Instruments that use colorimetric techniques, especially the detector tubes, can deviate ±50% from the true values (results within ±25% are acceptable, provided the error range is known and is included in the hazard analysis).
- Direct reading instruments require frequent calibration to meet published accuracy levels because electronic drift, vibration, pressure and temperature fluctuations, reagent batches, and other factors can adversely affect the instruments' accuracy.

The accepted accuracy of various instruments ranges between ±1% and ±25%. Before making recommendations based on a single reading, industrial hygienists must carefully assess an instrument's capability, the worksite's situation, and any new risks that could ensue from significantly changing an industrial process.

Indirect Measurements

Indirect measurement of airborne contaminants requires that industrial hygiene personnel collect the potentially hazardous material of interest and deliver it to the laboratory for analysis. Before a sample can be collected, the industrial hygienist must know (*a*) the physical state (eg, is it an aerosol or a gas or vapor?) of the contaminant and (*b*) the proper *sampling train* (the combination of equipment, connected in series) necessary to collect the specific contaminant in such a way that its volume or weight can be precisely determined in a laboratory.[22-24]

Aerosols contain liquid or solid material suspended in air. They include dusts, fumes, and smokes (solid aerosols) and mists and fogs (liquid aerosols). Aerosols are defined by their (*a*) aerometric diameters and (*b*) method of formation (Table 4-3). Although aerosols of interest to industrial hygienists have diameters ranging from 0.001 to 500 μ, the diameters of aerosols that significantly affect the body enter via the respiratory tract and generally range between 0.1 and no greater than 20 μ. Their size, density, shape, and other aerodynamic properties affect both the quantity of contaminant deposited and the respiratory site wherein the contaminant will accumulate:

- Aerosols with diameters larger than 10 μ tend to deposit in the nose and upper respiratory tract.
- Aerosols with diameters approximately 0.5 to 10.0 μ tend to be carried further and be deposited within the smaller respiratory passages.
- Aerosols with diameters of 0.1 to 0.5 μ are inhaled and exhaled, but tend not to be deposited.
- Extremely small particles (< 0.1 μ) are usually deposited in the smallest air passages after collision with gas molecules in breathing air. However, these particles are so small that their absolute quantity is minuscule, and they usually have no significant effect on human health.

Although gases and vapors are actually separate physical states, they are grouped together for purposes of this chapter because industrial hygienists use

TABLE 4-3

TYPES OF AEROSOLS

Type	Approximate Range of Diameters (μ)	Formation
Dusts	< 1 – > 500	Formed from solid materials by a mechanical action such as crushing or grinding
Fumes (colloids in air)	0.0001 – 1.0	Formed by vaporizing and condensing solids in air, such as when welding or cutting metal
Smokes	0.01 – 1.0	Produced by incomplete combustion of carbon-containing material
Mists	0.5 – > 100	Produced from liquids by mechanical action such as bubbling, splashing, or atomizing
Fogs	1 – 50	Formed from liquids that have vaporized and recondensed on microscopic particles of dust or fume, usually dense enough to obscure vision

Adapted from McKee SB, Fulwiler RD. Determination of particle size. In: Powel CH, Hosey AD, eds. *The Industrial Environment—Its Evaluation and Control*. Washington, DC: USDHEW, PHS, CDC, NIOSH; 1965: § B-7. PHS Publication 614.

similar sampling techniques to collect them. The common synonymous use of the terms *vapor* and *gas* sometimes causes minor confusion. A substance is considered to be a gas if it maintains that state at room temperature and normal atmospheric pressure; however, a vapor at room temperature is generally very close to changing in physical state from gas to liquid. Industrial hygienists take an interest in these differences because the entry and action of solid or liquid aerosolized chemicals differs from their entry and action as a gas or vapor. The industrial hygienist must consider the context of use. For example, methylene chloride in a paint-stripping preparation can cause dermal irritation if spilled on the skin; however, if inhaled in sufficient concentration, methylene chloride can quickly cause chemical anoxia. Failure of an industrial hygienist to consider these differences can cause a faulty evaluation of hazard potential.

In addition to understanding the physical state of the contaminant, industrial hygienists must also understand the components of a proper sampling train used to measure contaminant levels. Sampling trains for aerosols and gases and vapors are similar, yet have distinct differences in their collecting media (Figure 4-9). For aerosols, sampling trains are generally composed of (*a*) an air inlet device, which can be either a length of stiff or flexible tubing, or a part of the particulate collector; (*b*) a particulate collector; (*c*) a means of controlling flow; (*d*) an airflow metering device; and (*e*) an air pump. The most common particulate collectors use filters and cyclones (Figure 4-10).

Sampling trains for gases and vapors differ from those for aerosols in their collection devices—absorbers and adsorbers (Figure 4-11). Absorption is a chemical process in which the collected gas or vapor reacts with chemicals in the collection device. Commonly used absorption equipment consists of impingers and fritted bubblers. These devices use liquid collection media, each type of which provides different contact times, bubble size, and contact surface. These factors cause the collecting time or surface area or both to vary. In comparison, adsorption is a physical process in which the gas or vapor collected is trapped on the collection media, but with no chemical reaction. Adsorbers are used in packed tubes to collect insoluble or nonreactive gases and vapors. Tubes packed with activated charcoal and silica gel are the most common, but many other adsorbent materials are available for specific collection techniques (Figure 4-12).

Other methods are available for collecting samples

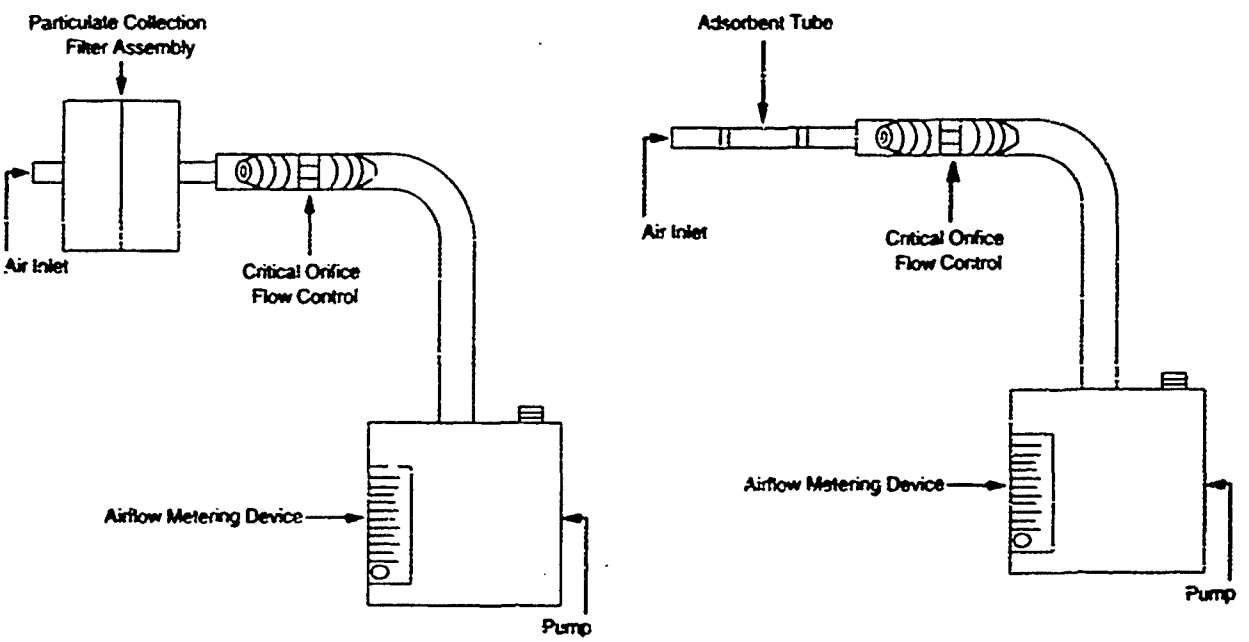


Fig. 4-9 Both these sampling trains for chemical collection use an air-sampling pump without constant flow capability. Flow control can be accomplished with valves or critical air-flow orifices. Control is required to ensure that the exact volume of air collected can be calculated. Without precise flow control, clogged collection devices or variable air pump speed caused by voltage fluctuations could cause a large measurement error. Air pumps are necessary to power the sampling train. Flow from the pump must be calibrated with the entire unit connected as if it were in actual use. This arrangement allows the system to be adjusted to overcome the resistance to air flow found in each separate component of the sampling train. A constant flow pump that uses electronic flow devices can be seen in Figure 4-11.

Fig. 4-10. Particulate filters, left to right: cellulose ester; glass fiber; polyvinyl chloride; a filter taken apart to show the body, support pad, and filter disk; and a filter mounted in a cyclone device that is used to separate out the respirable aerosols. These particulate collection filter assemblies differ according to the laboratory requirements for extracting the hazardous material collected from the filter media.

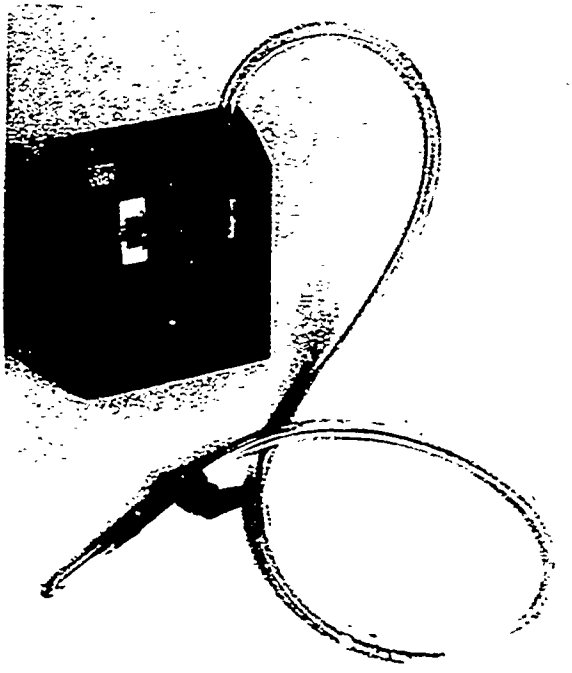


Fig. 4-11. This DuPont P4LC constant flow pump with its sampling tube attached is a sampling train used to collect many kinds of gas and vapor contaminants. A particulate sampling train would have a filter or filter/cyclone collection device. Constant flow pumps use microprocessors to sense airflow and alter pump speed to maintain a known collection rate.

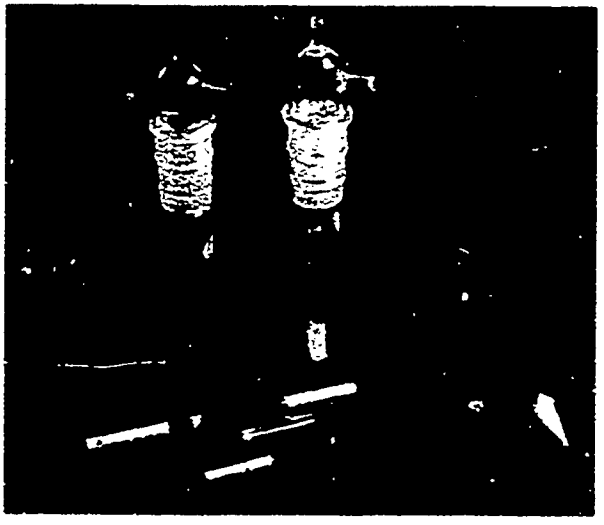


Fig. 4-12. The midget impinger shown in the left background is used to collect contaminants in a liquid medium; the midget *fritted* impinger shown on the right background breaks up contaminated gases into tiny bubbles, thereby increasing the collection efficiency. The impinger shown in the right foreground is designed to collect samples but not leak into the pump. Collection tubes containing Firebrick, activated charcoal, and silica gel, center foreground, top to bottom, are used to collect various gases and vapors for laboratory analysis.

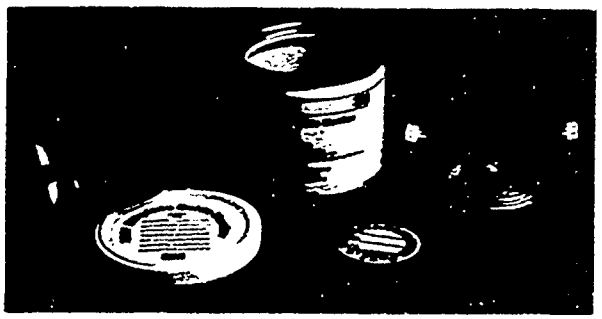


Fig. 4-13. This 3M Gas Badge, shown in front of its shipping container, is used to monitor for exposure to ethylene oxide; air pumps or other sampling-train components are unnecessary.

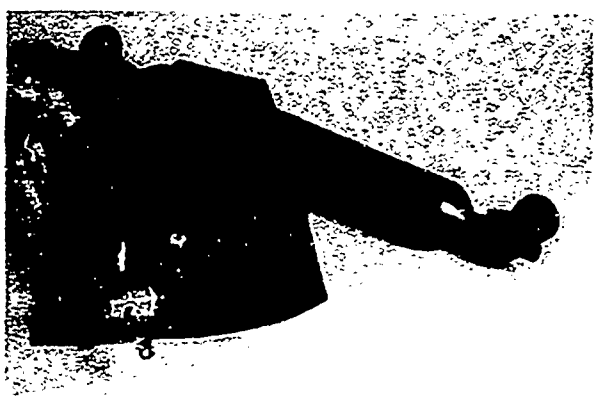


Fig. 4-14. Stainless steel, right, or glass, left, evacuation containers have valves to control the collection of grab samples of worksite air into rigid containers of known volume. The flexible collection bags, center, usually have fittings that connect to air pumps, which fill the bag with the air sample.

of contaminants that do not use elaborate mechanical sampling trains. In all methods, samples are collected at a known rate so the air volume collected can be related to the total amount of contaminant found by laboratory analysis. For example, gas-monitoring badges are available for many compounds and use diffusion through a membrane or into an orifice to collect samples at a known rate (Figure 4-13). After the collection period, the badge is sealed to prevent loss by diffusion and is sent to a laboratory for analysis.

Instantaneous or *grab* samples collect actual worksite air; the sample contains whatever contaminant exists at the instant of collection (Figure 4-14). Evacuated containers, displacement collectors, and flexible collection bags are used for collecting grab samples, which are then sent to a laboratory for analysis.

Assessing Measurements

Exposure standards have been developed for many physical and chemical hazards found in the work environment. The Occupational Safety and Health Administration's permissible exposure levels (OSHA's PELs) are regulatory standards that carry the force of law. The ACGIH's Threshold Limit Values (TLVs) are consensus standards that do not carry the force of law. Because both are applied by hygienists in their work, exposure sampling must quantify actual exposures for comparison with these standards to determine when corrective action or medical surveillance is indicated.

Identifying both the limitations inherent in the measurement process and the potential adverse impact of measurement variables are essential for meaningful exposure sampling. Industrial hygienists determine exposure levels by finding the amount of each particular chemical contaminant per unit volume of air; therefore, the mass of the chemical, the volume of the air sample, and the efficiency of the collection all subject this process to potential collection errors.

Reported exposure levels are actually surrounded by a range of possible values; the actual level lies within the range. For example, a laboratory might report that it analyzed an air sample and found 125 ppm benzene. Taking into account the statistical consideration of random and systematic errors found in sample collection, handling, and analysis, the level should have been reported as 125 ppm $\pm$10 ppm, with a confidence level of 95%. If numerous measurements have been taken, the mean and the standard deviations of the mean can be estimated very closely. Estimates are not nearly as good with fewer samples, and only broad confidence limits can be obtained (Exhibit 4-1).

In nonstatistical terms, error in calculating the mass of a chemical is usually a function of (a) collection

EXHIBIT 4-1

ERRORS IN MEASUREMENT

All exposure measurements can contain both random and systematic errors; therefore, they are only estimates of actual values. Random errors occur by chance, sometimes higher and sometimes lower than the true value. Systematic errors always skew a value either above or below the actual value. For example, a track coach repeatedly timing a runner with a highly accurate stopwatch will err randomly due to the reaction time required to stop the watch. If the coach uses an inferior watch that runs either fast or slow, however, then the elapsed time measured will always be too slow or fast, and the errors will be systematic. Because both random and systematic errors can occur concomitantly, our goals are to eliminate systematic error and to control for random error.

Systematic Error

The more complex the measurement, the more likely that systematic errors will occur. Typical errors that industrial hygienists see include malfunctioning or incorrectly calibrated equipment, untrained or inexperienced operators, and errors in recording data. For example, there is little chance for error when reading the numeric display on a digital carbon monoxide meter. However, other sources of systemic error could exist with this meter. Is the operator properly calibrating and operating the instrument? Are the correct scales used and are the results recorded in the correct units of measurement? If not, then several sources of systemic error have contaminated this simple, direct measurement of carbon monoxide–exposure levels.

Aggressive quality-control and quality-assurance measures can eliminate these errors. Credentialed operators maintaining, calibrating, and operating measurement equipment, and analytical laboratories participating in quality-control procedures such as external proficiency testing and internal quality control will produce accurate results.

Random Error

Instrument operators introduce random error when they read dials and meters, set flow rates, measure time, prepare solutions, and perform other tasks that require observation and reaction. The random error produced can neither be eliminated nor (for a single measurement) predicted.

Probability theory predicts that in a series of measurements the results will be evenly distributed around the true value. This *central tendency* is a fundamental principle of statistical analysis. It provides a powerful tool to develop measurement strategies that will recognize random error and accurately estimate true values.

Exhibit Figure 1

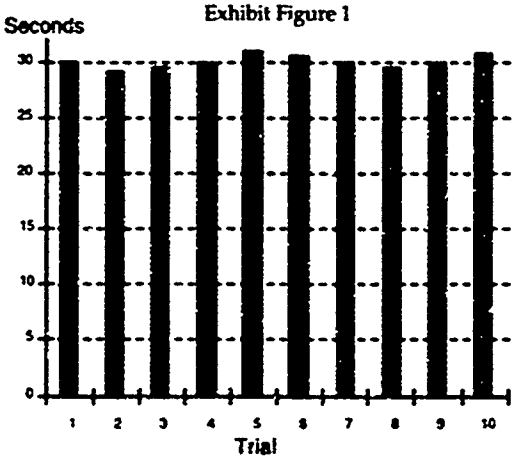
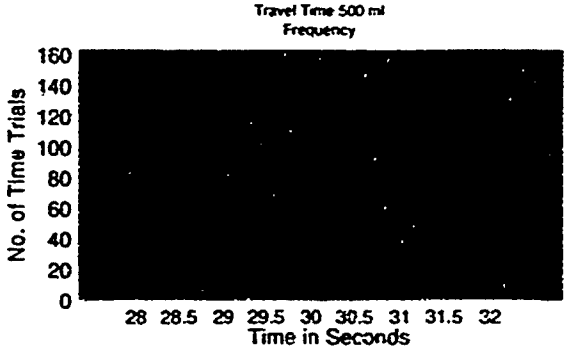


Exhibit Figure 2



For example, 10 timed measurements of air-volume flow (using a soap bubble in a Buret moving from the 0-mL to the 500-mL points) can be represented as a histogram (Exhibit Figure 1). We intuitively understand that the true flow time is close to 30 seconds. This means the air-volume flow is close to 1.0 L/min. And, indeed, the mean of all the measurements is 30 seconds.

However, more measurements produce a more-accurate estimate, and the more measurements taken, the more nearly correct the estimate will be. Eventually, further measurement is not worth the effort. If several hundred measurements of the time for a soap bubble to travel from the 0-mL to the 500-mL points on a Buret were plotted, a bell-shaped curve representing a normal probability distribution would develop (Exhibit Figure 2).

Truly random errors will be normally distributed around the mean. In a bell-shaped curve, the standard deviation (SD) measures this dispersion. In a normal distribution, approximately 68% of the values fall within the range of the mean, ±1 SD; 95% within ±2 SD; and 99% within ±3 SD. Generally, industrial hygienists will use the 95% confidence limits for their measurements.

Adapted from Johnson, DL, Bell ML. Sources and Control of Error in Industrial Hygiene Measurements. Presented at the First Annual Occupational Health Nurse Symposium; 18–22 June 1990; Xerox Training Center, Leesburg, Va.

efficiency, (*b*) sample stability, or (*c*) handling in the laboratory. A known collection efficiency is required for accurate determination of gas-and vapor-exposure levels. Chemicals in their gas or vapor phases are equally likely to be captured if temperature, pressure, and flowrate are kept constant. The collection of particulates, however, varies with their size, shape, and quantity. Various particulate samplers have different collection efficiencies for smaller and larger aerosolized particulates. Overloading the chemical onto filters or precipitators can cause variable collection efficiencies. In addition to error in chemical mass calculations as a function of collection efficiency, sample stability is also a factor. Losses or gains in chemical mass occur after formal collection has been completed. For example, chemicals having high vapor pressure can boil out of the collection media, and additional target chemicals can enter and contaminate samples that were improperly sealed at the worksite.

Other sampling errors can occur in the laboratory. The target chemical can react with the collection or storage container and be lost to laboratory analysis. Similarly, although laboratories generally have extremely accurate and highly sensitive analytical techniques and equipment, laboratories can lose chemical mass through a failure to fully extract the contaminant from the sampling media.

However, the greatest error in sample collection usually occurs in the field, when the sample volume is incorrectly determined. The instruments used to collect samples at the worksite are not designed to be as accurate as fixed laboratory bench equipment. Equipment used in the sampling train can also be affected by changes in temperature or pressure, physical damage during transportation, power-supply voltage changes, and operator error. Many flowrate and volume calibration devices are available, and sampling personnel must use them both before and after sampling to document the accuracy of the collection procedure (Exhibit 4-2 and Figure 4-15).

EXHIBIT 4-2

AIR-SAMPLING CALIBRATION PROCEDURES

- Use standard devices with care and attention to detail.
- Check all standard materials, instruments, and procedures periodically to determine their stability, operating condition, or both.
- Recalibrate a device whenever it has been changed, repaired, received from a manufacturer, subjected to use, mishandled, or damaged, and at any time when its accuracy is questioned.
- Understand how an instrument should be operated before attempting to calibrate it; use a procedure or setup that will not change the characteristics of the instrument or standard within the operating range required.
- When in doubt about procedures or data, assure their validity before proceeding to the next operation.
- Make all sampling- and calibration-train connections as short and constriction- and resistance-free as possible.
- Exercise extreme care when reading scales, timing, adjusting, and leveling, and during all other similar sample-collection operations.
- Allow sufficient time to stabilize conditions, overcome inertia, and establish equilibrium during calibration and sampling.
- Obtain enough points and different flow rates on a calibration curve to generate confidence in the plot obtained. Plot each point from more than one reading wherever practical.
- Maintain a complete permanent record of all procedures, data, and results. Include trial runs, known faulty data (with appropriate comments) instrument identification, connection sizes, and ambient barometric pressure and temperature.
- When a calibration differs from previous records, determine why the change occurred before accepting the new data or repeating the procedure.
- Properly identify the conditions of calibration, the device calibrated, the material it was calibrated against, the units involved, the range and precision of calibration, the date, and the name of the person who performed the actual procedure for all calibration curves and factors. If possible, indicate the location of the original data, and place appropriate calibration data on the instrument.

Adapted from Lippman, M. Instruments and techniques used in calibrating sampling equipment. In: *The Industrial Environment—Its Evaluation and Control*. Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 11.

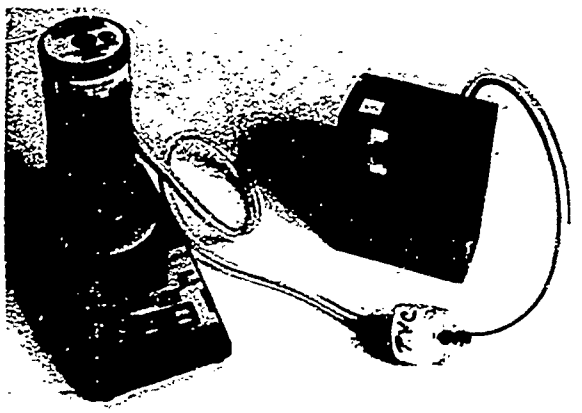


Fig. 4-15. The Gilian Instrument Corporation's Gilibrator Bubble Generator provides an efficient method of determining airflow rates before, during, and after sample collection.

Worksite Sampling Strategy

For each sampling situation, the industrial hygienist must use a logical sample collection strategy that will characterize the exposure of personnel at the worksite. The National Institute for Occupational Safety and Health (NIOSH) has published a recommended *decision logic* to help determine strategy (Figure 4-16). Whatever technique is used, industrial hygienists must consider five factors: the location, timing, and personnel to be sampled; the sampling period; and the number of samples.[12,25]

Location

Samples may be collected at the worker's breathing zone, at a specific worksite, or in the general area. The definition of a worker's exposure presupposes sample collection at the worker's breathing zone. However, it is sometimes impossible or dangerous to fit a worker

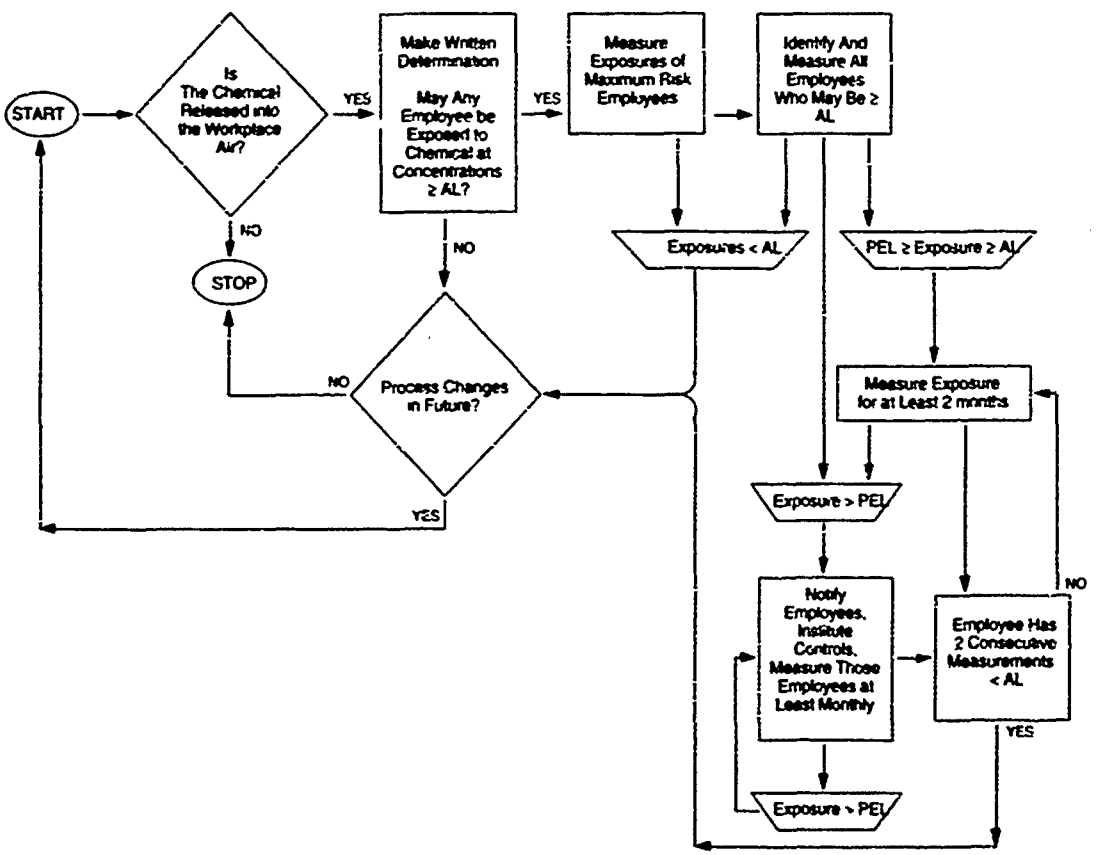


Fig. 4-16. Recommended employee exposure determinations and measurement strategy. This sampling logic uses the current permissible exposure levels (PELs) and the action level (AL, which is one-half the PEL) to set up a sampling strategy to determine exposures and sampling frequency. Source: Reprinted from National Institute for Occupational Safety and Health. *Occupational Exposure Sampling Strategy Manual.* Washington, DC: US GPO; 1977: 11.

with even the small air-sampling pumps or to place a direct reading instrument in the worker's breathing zone. In these instances, the industrial hygienist should collect samples close to the worker at the worksite. While breathing-zone samples are preferable, sampling at the worksite, or even in the general area of operation, can be used to define the effectiveness of engineering control measures, round out exposure data by defining the spread of contaminants, and support breathing-zone sampling results.

Timing

Worksite exposures change throughout the day. Times as short as a shift or as long as an entire season can alter the evolution, distribution, and dilution of hazardous chemicals. When developing the sampling logic, industrial hygienists must consider what time of day, week, month, or year will fully characterize exposure. This, of course, requires that the industrial hygienist be thoroughly familiar with the procedures used at the worksite and the differences in operations that are likely to depend on seasonal or weather conditions. For example, ventilation may be reduced to keep an area warm in winter, or increased to cool it in summer.

Personnel

Sampling the breathing zone of each individual at a worksite provides the most detailed information. However, this option would be impractical if 40 people were doing the same work. To collect samples that are as representative as possible, the industrial hygienist must make on-site determinations to designate the personnel with the highest probability of overexposure. NIOSH's *Occupational Exposure Sampling Strategy Manual* contains a method to determine the number of different samples that will ensure that at least one person from the top 10% exposure group is included in the sample, with 90% confidence (Table 4-4).[25(p35)]

For example, if 31 workers are all sanding paint off damaged trucks in a large maintenance bay, then N = 31. To be 90% confident that at least one of the three workers (10% of 31) with the highest of all exposures is included in a partial sample, at least 16 workers (n = 16) should be selected at random from the 31. Thus, we sample about 50% of the group to be 90% sure that at least one worker in the highest 10% of all exposures is included.

Sampling Period

The industrial hygienist has to analyze several variables to determine the volume and duration of sampling necessary to define the contaminant level at the worksite. Some variables that influence this determination include the appropriate exposure standard, the capability of the collection instruments, the estimated chemical concentration at the worksite, and the laboratory's capability. In most cases, the critical variable is the laboratory capability: their analytical equipment may need more volume of sample than can be collected during a short-term operation. The industrial hygienist and the analyst must come to agreement on the amount of sample required to satisfy both their needs.

NIOSH describes sampling periods as (*a*) full work period/single sample, (*b*) full work period/consecutive samples, (*c*) partial work period/consecutive samples, and (*d*) random grab samples (Figure 4-17).[25] Each of these sampling periods has a different purpose. For example, an 8-hour period single sample will provide only one number: the *average* exposure over

TABLE 4-4

SIZE OF SAMPLE THAT WILL INCLUDE TOP 10% EXPOSURES AND ACHIEVE 90% CONFIDENCE LIMITS

Size of Group (N)*	No. of Required Samples†
8	7
9	8
10	9
11–12	10
13–14	11
15–17	12
18–20	13
21–24	14
25–29	15
30–37	16
38–49	17
50	18

*N: Size of original group judged to have the same exposure potential

†n: Size of partial sample if N > 7 (the entire group must be sampled if N ≤ 7)

Reprinted from Keenan RG. Direct reading instruments for determining concentrations of aerosols, gases, and vapors. In: *The Industrial Environment—Its Evaluation and Control*. Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 16.

the entire 8-hour period. If a worker were exposed to 40 ppm for 1 hour, 100 ppm for 6 hour, and 0 ppm for 1 hour, and all exposures were collected on one sample medium, the laboratory would find only that the exposure over the 8-hour period averaged 80 ppm. Therefore, unless a direct reading instrument with a recorder is used as the collection device, industrial hygienists will not be able to determine if short-term overexposures occurred during this 8-hour period. These overexposures could be high enough to cause acute effects, yet not exceed the 8-hour standard when averaged. To escape this difficulty, consecutive, short-duration sampling over the 8-hour period provides both the full exposure and the short-term exposure levels (STELs). A time-weighted average (TWA) can be calculated from the series of sample results to determine the daily average and, because three different collection devices were used, we also see partial-period results:

$$\frac{(40\ \text{ppm} \bullet 1\ \text{h}) + (100\ \text{ppm} \bullet 6\ \text{h}) + (0\ \text{ppm} \bullet 1\ \text{h})}{8\ \text{h}}$$

$$= \frac{640\ \text{ppm/h}}{8\ \text{h}} = 80\ \text{ppm TWA}$$

Partial-period sampling can be used when the operation is uniform throughout the day, or when it is only done intermittently. One-time samples using detector tubes or evacuated containers can be useful as screening devices, but they provide only a single snapshot of possible exposure.

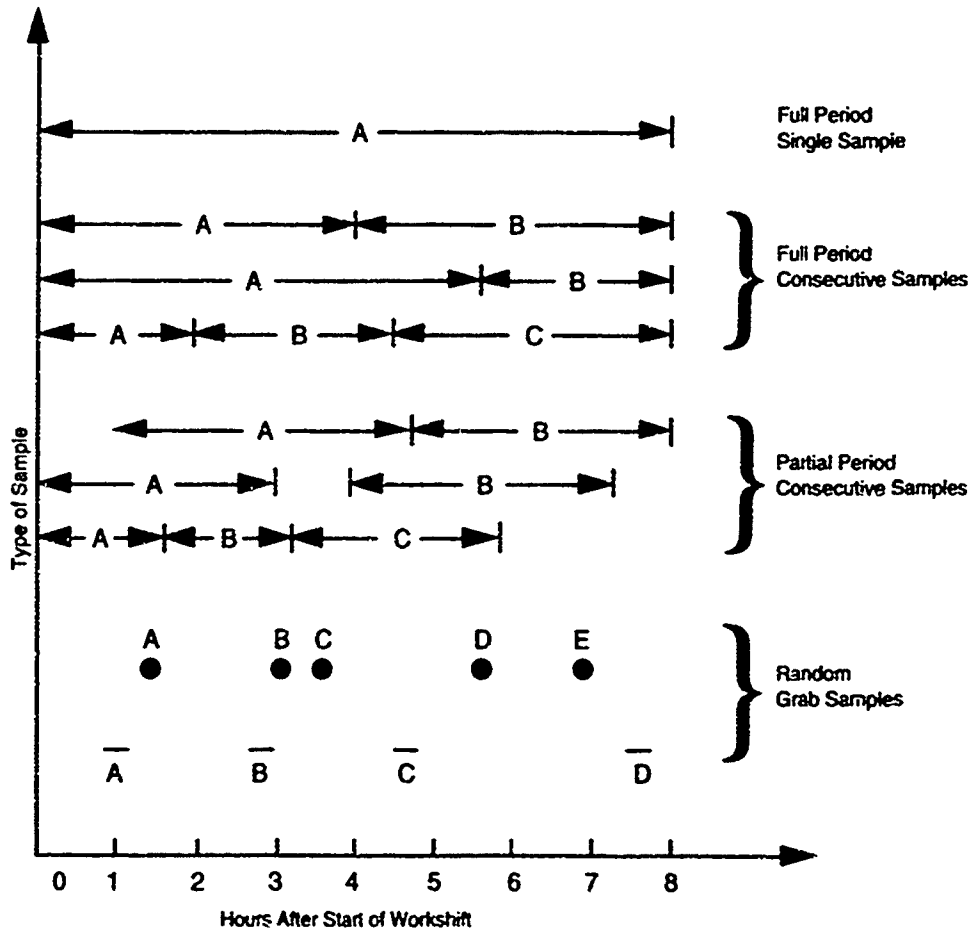


Fig. 4-17. Full period single samples provide only average exposures for the entire day; this technique cannot determine short-duration overexposures that might occur during a shift. Full period consecutive samples can be used to define exposures for different phases of an operation or to determine if exposure varies. As many as 16 and 32 samples (for 30- or 15-min intervals) are sometimes used to characterize exposures during an 8-hour workday. Partial period samples are useful for intermittent operations. Grab samples can be taken during expected peak exposures to determine if more sampling is required. Source: Reprinted from National Institute for Occupational Safety and Health. *Occupational Exposure Sampling Strategy Manual.* Washington, DC: US GPO; 1977: 38.

Number of Samples

The industrial hygienist also determines the number of samples required to accurately determine a worksite exposure. Single samples, even if they encompass a full shift, are not sufficient to characterize exposures. Many factors can alter exposures, such as interference from adjacent operations, the age of the chemicals used, and a change of operators. Only a series of samples taken over time and recorded in the HHIM can provide the record of exposure that is needed to show constant or fluctuating exposure levels. Industrial hygienists must adjust the sampling number over time if sample results prove to be all low, all high, or erratic.

Interpreting the Findings

The factors that industrial hygienists analyze to determine whether particular exposures are hazardous to health include (*a*) the reported exposure concentration (with appropriate consideration of the variance caused by sampling error), (*b*) the worksite (including the duration and type of exposure), (*c*) the nature and toxicity of the chemical, and (*d*) the existing standards. Various systemic sampling errors occur and the reported exposure concentration contains a positive or negative variance around the true exposure. This can have little impact if the reported exposure is far below or far above the health standards. Frequently, however, the reported result falls near the standard, and the statistical variance prevents making an accurate determination of whether the exposure has exceeded the standard. Then, the industrial hygienist must develop and execute a new, more defined sampling strategy, which could include more frequent consecutive samples or lower detection limit on the monitoring instrument.

Because most standards are based on the conventional 40-hour work week, unusual schedules (>8 h/d or 40 h/wk) require a special assessment of the hazard. Although the standards are generally proportionately reduced to incorporate increased exposure time and decreased recovery time, more complex models use pharmacokinetics to adjust exposure standards.[26,27]

To determine a hazard potential, industrial hygienists must know the rationale behind an exposure standard; they must correlate all exposure variables with the standard, while also considering that the standard was developed using data from animals, accidents, and laboratories. However, conditions at the actual worksite may not bear any relation to the data that were used to set the standard. The length of exposure, the physical state and purity of the chemical, and its toxicity will affect the industrial hygienist's determination.

The standards used by U.S. Army industrial hygienists are designed to conserve the fighting strength by controlling preventable disease and injury through command-oriented, occupational-, environmental-, and personal-protection programs. These standards are detailed in AR 40-5[5] and Technical Bulletin Medical (TB MED) 503[28] and include

- DoD and Department of the Army Occupational Safety and Health (DA OSH) standards for military (field and garrison) and nonmilitary worksites, for which regulatory agencies either have or have not issued OSH standards, and which are included in DoD and DA Pamphlets, circulars, TB MEDs, and messages;
- OSHA standards, including PELs, which are written into the regulations, and emergency temporary standards with minor adaptations as necessary, to conform with DA administrative practices;
- other regulatory worksite standards issued under statutory authority by other federal agencies such as the Department of Transportation and the Environmental Protection Agency;
- special DA OSH standards developed for militarily unique equipment, systems, and operations; and
- alternate worksite standards based on publications relating to worksite exposure criteria.

The army uses alternate standards in lieu of existing OSHA standards or when no OSHA standard exists. The current ACGIH TLVs[26] are used in DA military and civilian worksites if the OSHA PELs are less stringent or if no OSHA standard exists.

Outside the continental United States, DA OSH standards apply to Industrial Hygiene Program activities unless Status of Forces Agreements (SOFAs) require United States military forces overseas to comply with more stringent laws in host countries. In the absence of SOFAs, the most stringent applicable United States regulations apply.

The relationship of the current sample to the historical record of sample results that is kept in the HHIM must also be kept in mind. A significant difference from the historical record could be the result of an unreported change in the work routine or the chemical supply. It could also be nothing more than a human error in sample collection, transport, or analysis. In any case, when the record shows comparable results

that suddenly change, industrial hygienists must look closely at both industrial operations and industrial hygiene procedures. Another use of HHIM records is to display either increasing or decreasing trends in exposure levels. Gradually changing exposures could result from inappropriate maintenance of control equipment, progressive operational changes, or deteriorating chemical purity.

CONTROLLING HAZARDS

After industrial hygienists have characterized the hazards of a worksite, they provide recommendations to control or eliminate them.[11,29,30] Control measures are classified as *primary* and *secondary*. Not every type of control is necessary or appropriate in every situation: the willingness of the employees to accept and use the controls, the operating costs, and maintenance problems must all be considered.

Primary Controls

Primary controls—substitution, isolation, and local exhaust ventilation—prevent or eliminate worker exposure.

Substitution

Some hazards can be eliminated by substituting a less hazardous, yet effective chemical for the hazardous one (chemical substitution), or changing the process that produces the hazardous exposure (process substitution).

Although it is one of the best primary control measures, chemical substitution is not without its own risks if it is not fully researched before implementation, and carefully monitored thereafter. For example, an unsuccessful chemical substitution occurred in the dry cleaning industry: carbon tetrachloride was substituted for petroleum naphtha to eliminate a fire hazard. When carbon tetrachloride was later found to be associated with liver damage, chlorinated hydrocarbons such as trichloroethylene and perchloroethylene were substituted. Perchloroethylene is now listed as a suspected carcinogen.

Fluorinated hydrocarbons (Freons) have also been suggested for dry cleaning, and, because they have very low inhalation and fire hazard properties, they appear to be safe. However, these compounds are not without toxicity and also contribute to the deterioration of the earth's ozone layer (see Chapter 13, Solvents, Fluorocarbons, and Paints).

Like chemical substitution, process substitution can effectively control hazards. In many cases, the process itself increases exposure levels by spewing the chemical into the air or by transforming the chemical's physical state to one that more readily gains entry into (or onto) the worker. For example, instead of welding metals together with oxyacetylene or electric arc techniques, welders can join metals by bolting, riveting, or resistance spot welding. These processes generate virtually none of the metal and *flux* (an antioxidation compound) fumes associated with oxyacetylene or electric arc welding. Another example of process substitution can be seen in a painting operation. Instead of spray painting parts, workers could dip them or use electrostatic painting. Dipping reduces exposure to both paint solvents and paint pigments, and electrostatic spray painting controls exposure mainly to the pigments. Another example is the substitution of wet grinding for dry. This substitution reduces dust generation and therefore reduces possible exposures.

Isolation

Isolation is a control technique that imposes a barrier between the worker and the hazard. Barriers are generally distance or a physical structure. In some cases, merely increasing the distance between the worker and the hazard can reduce the hazard potential, especially for hazards such as heat, noise, or radiation, where intensity falls off rapidly with distance. Physical barriers can be as simple as a small operator's booth above the process or a reflective wall between the worker and a radiant heat source. However, complicated isolation systems (eg, enclosing the whole process or monitoring the work via television cameras) may sometimes be necessary. If hazards are completely isolated within process sites, consideration must be given to the hazard that will occur if a worker must enter the isolated machine or operation. In these cases, the exposure can suddenly increase from zero to extremely high levels. Industrial hygienists must prepare for such emergencies in advance.

Although PPE and work schedules serve as physical and temporal barriers to hazardous exposure, neither is considered to be an isolation technique. Both allow for more actual contact with the hazard than the other primary controls and therefore are classified as secondary controls.

Local Exhaust Ventilation

Properly designed, installed, and maintained local exhaust ventilation prevents exposure by capturing the contaminant at its source and removing it before it reaches the worker's breathing zone. However, the phrase "properly designed, installed, and maintained" does not fully convey the complicated nature of ventilation design nor the importance of adequate system maintenance (Table 4-5). A complete understanding of ventilation-system work requires significant training and experience. The ACGIH publishes a manual detailing the engineering of industrial ventilation, which is revised frequently.[31]

Secondary Controls

Secondary controls are used to reduce, but not entirely eliminate, exposure and include (*a*) general ventilation, (*b*) PPE, (*c*) worksite monitors, (*d*) medical surveillance, (*e*) administrative controls, and (*f*) training and education. Occasionally, several types of primary and secondary controls are employed together to control exposure.

General Ventilation

General ventilation dilutes a contaminant with clean air to concentrations below the accepted standards. However, industrial hygienists must consider the possible shortcomings of general ventilation as a method of exposure control. For example, the contaminant must not recirculate into the work area through adjacent air inlets and outlets. Buildings with designed air recirculation, intended to save money on air-temperature adjustment or filtration systems, can cause the same problem. General ventilation permits workers to be exposed to the contaminant; therefore it should not be used as a control for very toxic material, or when the contaminant cannot be diluted because workers are close to the source.[31]

Personal Protective Equipment

PPE must only be used as interim measures, or if engineering control absolutely is not feasible. These devices do not remove, reduce, or eliminate hazards from the worksite; they are merely insubstantial barriers between the worker and the hazard. Effective PPE is available for use as a temporary, emergency, or short-term control.[32]

No PPE is effective unless it is properly used. Any misuse or failure of the protective equipment will cause the worker to be exposed to the contaminant. Unfortunately, most PPE is uncomfortable and workers may misuse the devices. Respirators, hearing protection, face shields, gloves, and other PPE can cause physical and mental strain if they must

TABLE 4-5

LOCAL EXHAUST VENTILATION

Design Flaw	Resultant Problem
90° turns in ducting	Increased airflow resistance
Failure to provide make-up air to replace exhaust air	System resistance and drafts
Underestimating ventilation airflow system resistance	Undersized fans and motors
Use of blast gates	Inadequate control of airflow, system imbalance
Improper sizing of ducting	Inadequate control of airflow
Maintenance Requirement	**Problem Created by Omitting Procedure**
Lubrication of fan and motor bearings	Bearing seize-up, airflow stoppage, and equipment damage
Tightening/replacing fanbelts	Little or no air movement
Cleaning/replacing clogged filters	Increased airflow resistance and decreased contamination control
Cleaning of fan belts	Decreased fan efficiency
Confirmation of proper direction of fan-blade rotation	Little or no air movement

be worn all day. Therefore, industrial hygienists should strive to use primary controls so that PPE is unnecessary.

Respirators. The classifications of respirators include (*a*) air purifying respirators, (*b*) air supplying respirators, and (*c*) self-contained breathing apparatuses. The air purifying respirators remove contaminants by filtration, absorption, adsorption, or catalytic action. Air supplying respirators provide breathable air from compressors, blowers, or air cylinders. Self-contained breathing apparatuses supply air to the worker from a rebreathing device or an air tank that the worker carries.

The proper selection, use, escape requirements, and care of respirators is a complex subject; the currently accepted respirator selection decision logic must be fully considered before utilizing respirators (Figure 4-18).[33] Such concerns as the level of exposure, oxygen level, warning properties of contaminants, protection levels of each respirator class, carcinogenic properties of the contaminants, immediate danger to life or health,[34] levels of the contaminants, escape requirements, and approval restrictions must be fully considered before respirators are utilized. NIOSH, the accepted approval authority, and AIHA publish detailed materials on these subjects.[33-36] These must be read and understood before selecting respirators as protective devices.

Once qualified personnel have selected the proper respirator, workers and supervisors must receive training regarding its proper use and care. Workers and supervisors must understand the rationale behind the use of respirators instead of engineering controls. The user must be fully involved to understand the need for using such uncomfortable protective equipment. Existing OSHA and U.S. Army regulations also contain details concerning the full requirements for a complete respirator program.[33,37,38]

Eye and Face Protection. Eye and face protection provide a barrier against hazards ranging from liquid chemicals to solid projectiles to intensive light radiation. Individuals who select the protective devices must know the form of the hazard. For example, chemical splashes, mists, and streams require different levels of protection, ranging from chemical-splash goggles to full-face shields. Similarly, various levels and forms of intense visible, infrared, and ultraviolet light also require different protection levels in goggles and welders' face shields: oxyacetylene cutting, for example, does not require the level of eye protection against intense light that is needed for electric arc welding.

Gloves and Other Clothing. Gloves, leggings, boots, aprons, and other protective clothing provide a barrier to chemicals that either affect the skin itself, or gain entry to the body through the skin. Protective clothing is made with myriad materials, each with different permeation characteristics for different chemical groups. These characteristics range from easily penetrated to very protective.[39,40] When selecting protective clothing, industrial hygienists should consider not only an item's protective ability, but also its comfort and fit, and the likelihood that workers will wear it.

Worksite Monitors

Worksite monitors are warning devices that signal when a preset limit of exposure has been reached. These devices have some value, but they allow exposure lower than the monitor's alarm setting to occur. If worksite monitors are not calibrated or maintained, exposures can occur well above standards or settings. Additionally, if monitors are too sensitive or are set at too low a level, workers may either ignore or disable the frequent warning signal.

Medical Surveillance

Medical surveillance is an important secondary control because it alerts medical personnel that potential overexposures are occurring. This control can also identify those hypersusceptible individuals who might have adverse effects at exposures below the standards. Although medical surveillance allows early detection, exposure to the hazard has already occurred.

Administrative Controls

Exposure time limits and standing operating procedures (SOPs) are administrative controls. Exposure time limits ensure that, although short-term exposures over the exposure standard may occur, the 8-hour TWA remains below the standard. Operational SOPs direct the correct use of chemicals or personal protection. However, unless these controls are enforced, overexposure can certainly occur.

Training and Education

Workers, supervisors, engineers, and managers need to know and understand the hazards, their health effects, and the protective techniques recommended. The communication of worksite hazards to workers is now not only a basic, common-sense requirement, it is also a federal regulation.[34,41]

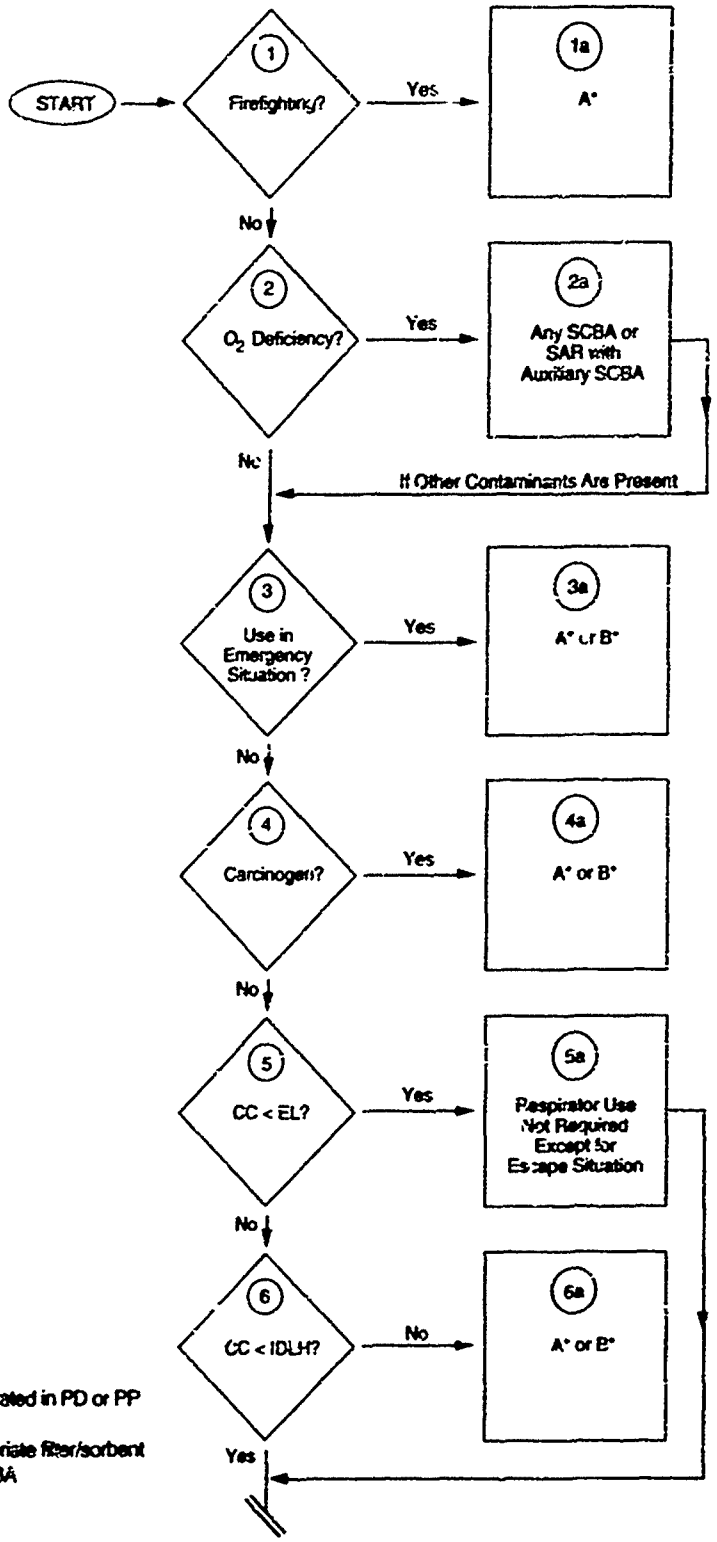
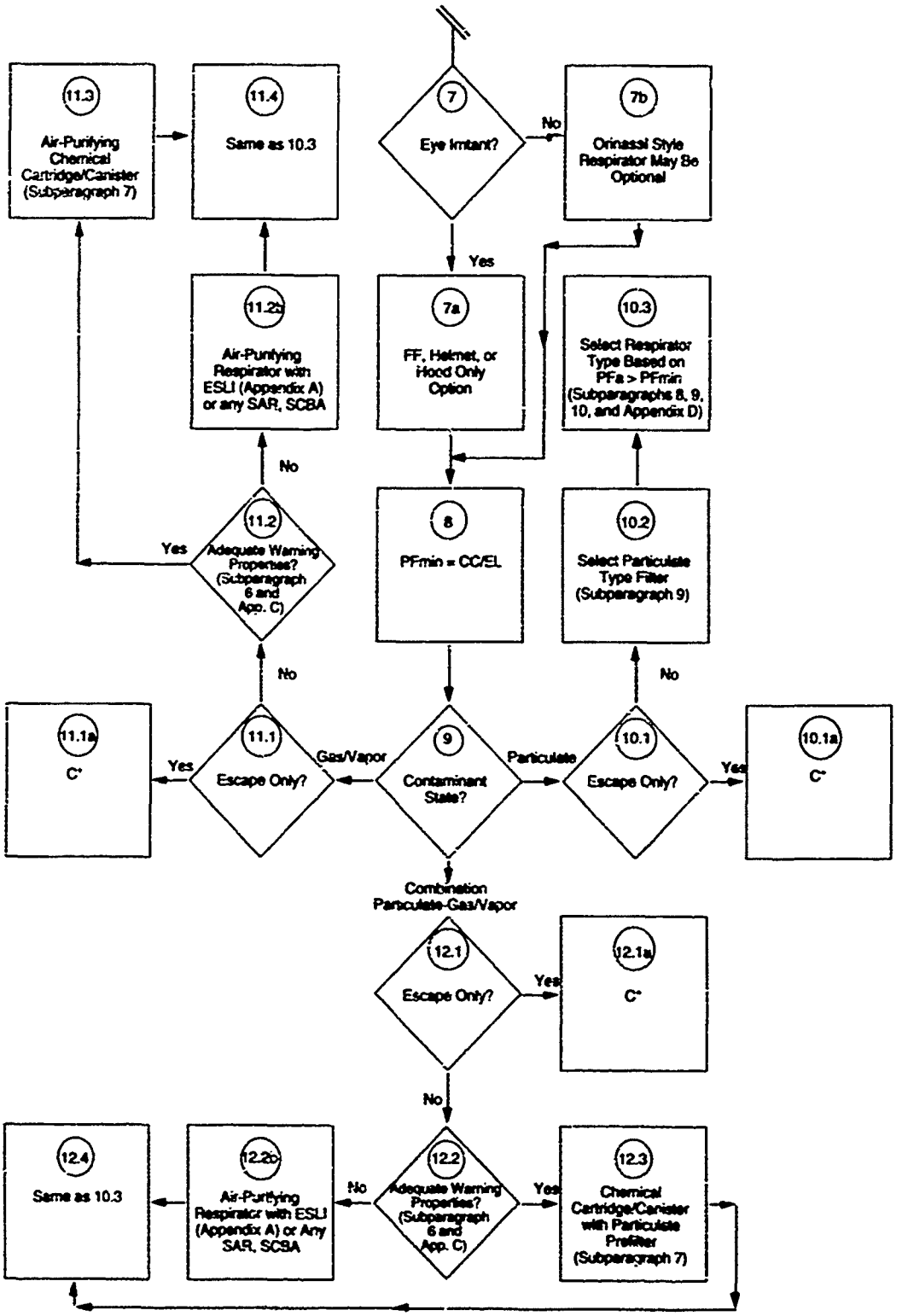


Fig. 4-18. The National Institute of Occupational Safety and Health's (NIOSH's) Respirator Decision Logic provides a basis for selecting appropriate respirators. Users must first determine if a primary control is required, and must fully understand the nuances of each decision point. A decision to use respiratory protection indicates that a respiratory hazard exists;



therefore, proper selection is a serious undertaking. Circled numbers refer to full-text descriptions of the respiratory decision logic in the source document. Reprinted from US Department of Health and Human Services. *NIOSH Respirator Decision Logic*. USDHHS, CDC, NIOSH; 1987: 19–20. DHHS (NIOSH) Publication 87-108.

SUMMARY

Industrial hygiene in the U.S. Army and the United States developed apace. The need to keep healthy, trained, productive personnel at materiel-production facilities operating at full capacity during our wartime mobilizations provided the initial impetus for the field of occupational health and the subdiscipline of industrial hygiene. The utility of hazard identification and control in the workplace has not faded. The expansion of industrial hygiene operations in the army and in the United States has significantly improved both quality of life and productivity.

Industrial hygiene, occupational healthcare, and occupational safety have separate but interrelated responsibilities. Their shared, broadly based concerns and interests make their close cooperation and coordination essential. Trained and experienced industrial hygienists are necessary to define work practices, understand and use appropriate monitoring equipment, analyze exposure data in relation to the route of entry and action, and determine the best control measures.

The basic goal of industrial hygiene is simple: identify, evaluate, and control worksite hazards. However, putting this into practice requires extensive education and experience. Industrial hygienists must be aware of the many sources of error in industrial hygiene measurements. Eliminating or controlling systematic and random error is a matter of aggressive quality control and quality assurance, and of the appropriate statistical treatment of data. Occupational health professionals should be aware of sources of error, be alert for flawed exposure estimates, and be prepared to ask the hard questions necessary to perform their true preventive medicine mission: eliminating and controlling occupational health hazards before they can do harm.

REFERENCES

1. American Industrial Hygiene Association. *1989–1990 Membership Directory.* Akron, Oh: AIHA; 1989.

2. American Conference of Governmental Industrial Hygienists. *By-laws of ACGIH. 1989 Directory.* Cincinnati, Oh: ACGIH; 1989.

3. Bayne-Jones S. *The Evolution of Preventive Medicine in the United States Army, 1607–1939.* Washington, DC: US Department of the Army, Office of The Surgeon General; 1968.

4. Anderson RS, ed. Special fields. In: *Preventive Medicine in World War II.* Vol 9. Washington, DC: US Department of the Army, Office of The Surgeon General; 1969.

5. Hatch TF. The Armored Force Medical Research Laboratory in WW II. *Medical Bulletin of the US Army, Europe.* 1985;42(1):22–26.

6. Gaydos, JC. A historical view of occupational health for the soldier. *Medical Bulletin of the US Army Medical Department.* 1988;2:4–6.PB 8-88.

7. Kneessy, AD. *Army Occupational Health and AEHA.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1981.

8. Cook WL Jr. Special fields. In: *Preventive Medicine in World War II.* Vol 9. Washington, DC: US Department of the Army, Office of The Surgeon General; 1969: Chap 3.

9. US Department of the Army. *The Army Safety Program.* Washington, DC: DA; 1988. Army Regulation 385-10.

10. US Department of the Army. *Preventive Medicine.* Washington, DC: DA; 1990. Army Regulation 40-5.

11. Hosey AD, Kusnetz HL. General principles in evaluating the work environment. In: Powel CH, Hosey AD, eds. *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1965: § B-1. PHS Publication 614.

12. Hosey AD. General principles in evaluating the occupational environment. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS. CDC, NIOSH; 1973: Chap 10.

13. Birmingham DJ. Occupational dermatoses: Their recognition, control and prevention. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 34.

14. US Army Research Institute of Environmental Medicine. *Heat Illness: A Handbook for Medical Officers.* Natick, Mass: USARIEM, USAMRDC; June 1991. USARIEM Technical Note 91-3.

15. US Army Research Institute of Environmental Medicine. *Sustaining Health and Performance in the Desert.* Natick, Mass. USARIEM, USAMRDC; July 1992. USARIEM Technical Note 91-1. Availability notice: Qualified requesters may obtain copies of this report from Commander, Defense Technical Information Center (DTIC) (formerly DDC), Cameron Station, Alexandria, VA 22314.

16. US Army Research Institute of Environmental Medicine. *Sustaining Health and Performance in the Cold: Environmental Medicine Guidance for Cold-Weather Operations.* Natick, Mass. USARIEM, USAMRDC; July 1992. USARIEM Technical Note 92-2. Availability notice: Qualified requesters may obtain copies of this report from Commander, Defense Technical Information Center (DTIC) (formerly DDC), Cameron Station, Alexandria, VA 22314.

17. Minard D. Physiology of heat stress. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW; PHS; CDC; NIOSH; 1973: Chap 30

18. Hertig BA. Thermal standards and measurement techniques. In: *Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 31.

19. Belding HS. Control of exposures to heat and cold. In: *Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW; PHS; CDC; NIOSH; 1973: Chap 38.

20. Alpaugh EL, Hogan TJ. Temperature extremes. In: Plog BA, ed. *Fundamentals of Industrial Hygiene.* Chicago, Ill: National Safety Council; 1988: Chap 12.

21. American Conference of Governmental Industrial Hygienists. *Air Sampling Instruments.* 7th ed. Cincinnati, Oh: ACGIH; 1989.

22. Keenan RG. Direct reading instruments for determining concentrations of aerosols, gases and vapors. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 16.

23 Roach SA. Sampling air for particulates. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 13.

24. Pagnotto LD, Keenan RG. Sampling and analysis of gases and vapors. In: *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 15.

25. National Institute for Occupational Safety and Health. *Occupational Exposure Sampling Strategy Manual.* Washington, DC: NIOSH; 1977.

26. American Conference of Governmental Industrial Hygienists. *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices.* Cincinnati, Oh: ACGIH; 1991.

27. Paustenbach DJ. Occupational exposure limits, pharmacokinetics and unusual work schedules. In: *Patty's Hygiene and Toxicology* 2nd ed, Vol 3a. New York: John Wiley; 1985.

28. US Department of the Army. *The Army Industrial Hygiene Program.* Washington, DC: DA; 1985. Technical Bulletin MED 503.

29. Hosey AD. Control of the occupational environment. In: Powel CH, Hosey AD, eds. *The Industrial Environment—Its Evaluation and Control.* Washington, DC: USDHEW, PHS, CDC, NIOSH; 1965; § C-1. PHS Publication 614.

30. Peterson, JE. Principles of controlling the occupational environment. In: *The Industrial Environment—Its Evaluation and Control*. Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 35.

31. American Conference of Governmental Industrial Hygienists. *Industrial Ventilation—A Manual of Recommended Practice*. 21st ed. Lansing, Mich: Committee on Ventilation, ACGIH; 1991.

32. Schulte HF. Personal protective devices. *The Industrial Environment—Its Evaluation and Control*. Washington, DC: USDHEW, PHS, CDC, NIOSH; 1973: Chap 36.

33. US Department of Health and Human Services, National Institute for Occupational Safety and Health. *Respirator Decision Logic*. Washington, DC: 1987. DHHS (NIOSH) Publication 87-108.

34. US Department of Health and Human Services, National Institute for Occupational Safety and Health. *Guide to Industrial Respiratory Protection*. Washington, DC: USDHEW, NIOSH; 1987. DHHS (NIOSH) Publication 87-116.

35. US Department of Health and Human Services, National Institute for Occupational Safety and Health. *NIOSH Pocket Guide to Chemical Hazards*. Washington, DC: USDHEW; NIOSH; 1990. DHHS (NIOSH) Publication 90-117.

36. American Industrial Hygiene Association. *Respiratory Protection: A Manual and Guideline*. Akron, Oh: AIHA; 1980.

37. 29 CFR, Part 1910 § 134.

38. US Department of the Army. *The Army Respiratory Protection Program*. Washington, DC: DA; 1990. Army Regulation 11-34.

39. US Department of Health and Human Services, National Institute for Occupational Safety and Health. *Personal Protective Equipment for Hazardous Materials Incidents—A Selection Guide*. Washington, DC: USDHEW, NIOSH; 1984.

40. American Conference of Governmental Industrial Hygienists. *Guidelines for Selection of Chemical Protective Clothing*. Cincinnati, Oh: ACGIH; 1987.

41. 29 CFR 1910, Part 1200.

Chapter 5

HEALTH HAZARDS TO HEALTHCARE WORKERS

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- Physical Hazards
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STRATEGIES FOR HAZARD ABATEMENT

THE MILITARILY UNIQUE ENVIRONMENT

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WALTER REED ARMY MEDICAL CENTER'S EXPOSURE CONTROL PLAN

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INTRODUCTION

Whether large or small, healthcare facilities are complex environments in which biological, chemical, and physical agents pose potential threats to the health of patients, staff, and visitors. Hospitals, medical clinics, and dental clinics that provide primary medical care are the most common healthcare facilities. Within the U.S. Army Medical Department (AMEDD), other research and service laboratories and veterinary clinics also contain many of the potential health threats that are found in the primary medical care facilities. AMEDD also uses deployable field medical treatment facilities (MTFs). These militarily unique MTFs pose greater challenges in the control of potential health threats than are found in fixed medical facilities. Effectively dealing with these potential health threats, regardless of the specific environment, requires knowledge about the hazards that might be present, the ability to define the nature and extent of exposure, and the expertise to develop and implement risk-reduction programs.

HISTORY

Ironically, patients themselves pose risks to healthcare workers. These not-insignificant risks range from contracting seemingly minor afflictions such as musculoskeletal discomfort to death from any number of infectious diseases such as tuberculosis and, in our own time, blood-borne pathogens. Bernardino Ramazzini (1633–1714), the Italian physician whom we acknowledge as the father of occupational medicine, recognized such hazards to healthcare workers when he described dermatitis and exhaustion as diseases of midwives in 1713.[1] The labor chair (which required the midwife to stand in an uncomfortable position) probably contributed to exhaustion; the constant bathing of the hands in lochia probably caused dermatitis. Ramazzini favored the new practice of having the patient labor and deliver in bed to ease the work of the midwives.[1,2]

The terrible mortality that characterized hospitals before the 20th century was at least partially iatrogenic in origin. Dr. Philipp Ignaz Semmelweis (1818–1865), as a result of his work on puerpueral fever, realized that by introducing a few simple maneuvers, he could reduce the mortality of this disease. He initiated routine handwashing by healthcare workers more than a century ago. During the second half of the 19th century, Florence Nightingale (1820–1910), who perceived that hospitals were hazardous not only to patients but also to those who took care of them, introduced open-window ventilation and worked to reduce patient overcrowding. Although most hospital hazards were considered to pose risks to patients rather than to the staff, attempts such as these to protect patients also benefited healthcare workers.[3]

The present emphasis on the hazards of blood-borne pathogens—both from patient to healthcare workers and vice versa—may be the most dramatic, but it should not obscure the numerous other, subtle hazards that also threaten healthcare workers. New hazards appeared during the early 1900s when physicians were exposed to radiation while experimenting with X rays, and operating room personnel faced possible explosions and other adverse health effects during surgery when flammable anesthetic gases were used.[3]

Until recently, healthcare facilities were traditionally considered safer than other work environments because employees were generally viewed as *providers*, not as *workers* exposed to a wide variety of hazards. The fact is, however, that hospitals are oriented toward reducing mortality and morbidity from disease, not prevention. As a result, few resources have been allocated for occupational exposures, and safety and health standards for healthcare facilities were promulgated only to protect patients.[3] The National Institute of Occupational Safety and Health (NIOSH) has identified several factors that have contributed to the lack of emphasis on the health of workers in the healthcare industry, including the beliefs that

- hospital workers were health professionals capable of maintaining their own health without assistance, and
- informal consultations with hospital physicians would replace medical-facility employee health services.[3]

To correct these misconceptions, safety and health standards have been, and continue to be, developed by various federal, national, and licensing organizations and agencies. Within the realm of their application, these standards are addressed later in this chapter.

TYPES OF HAZARDS

During the normal course of activities in healthcare facilities, exposures to (*a*) chemical; (*b*) biological; (*c*) physical, including ergonomic; and (*d*) psychosocial hazards occur routinely. Some exposures are similar to those in industrial environments, while others are unique to the healthcare setting; some exposures occur throughout a healthcare facility, while others are localized in a specific area (Exhibit 5-1). Variations in

EXHIBIT 5-1

OCCUPATIONAL HAZARDS IN AMEDD HEALTHCARE FACILITIES*

Maintenance and Engineering
- Adhesives
- Ammonia
- Asbestos
- Carbon monoxide
- Cold
- Ethylene oxide
- Fluorocarbons
- Fuels
- Heat
- Lubricants
- Mercury
- Noise
- Oils
- Paints
- Pesticides
- Sewage
- Solvents
- Welding fumes

Nuclear Medicine
- Biological agents
- Radionuclides

Pathology
- Biological agents
- Embedding media
- Fixatives
- Fluorocarbons
- Formaldehyde
- Glutaraldehyde
- Phenols
- Solvents
- Xylene

Patient Care
- Antineoplastic agents
- Biological agents
- Hazardous drugs
- Mercury
- Radiation
- Sharps

Pharmacy
- Antineoplastic agents
- Hazardous drugs

Radiology
- Developer chemicals
- Magnetic radiation
- X radiation

Operating and Delivery Rooms
- Anesthetic gases
- Antiseptics
- Biological agents
- Ethylene oxide
- Lasers
- Methyl methacrylate
- Sharps

Central Supply
- Alcohol
- Ammonia compounds
- Biological agents
- Detergents
- Dusts
- Ethylene oxide
- Fluorocarbons
- Formaldehyde
- Glutaraldehyde
- Mercury
- Noise
- Sharps
- Soaps
- Xylene

Dialysis Units
- Biological agents
- Disinfectants
- Formaldehyde

Dental Service
- Anesthetic gases
- Biological agents
- Compressed gases
- Ethylene oxide
- Formaldehyde
- Mercury
- Methyl methacrylate
- Noise
- Radiation
- Vibration

Housekeeping
- Biological agents
- Detergents
- Disinfectants
- Glutaraldehyde
- Sharps
- Soaps
- Solvents

Veterinary Clinic
- Anesthetic gases
- Biological agents
- Disinfectants
- Pesticides
- Sharps

Cast and Brace Shops
- Adhesives
- Dusts
- Noise
- Solvents
- UV radiation

*Musculoskeletal strain, psychological stress, and safety (such as electrical and explosive) hazards are not included

services, patient types, and staff make the listing of every specific exposure for all healthcare facilities impossible; therefore, this chapter addresses only the more typical exposure hazards. The comprehensive identification of health hazards and elimination or control of these hazards is a responsibility of each individual workplace—whether an industrial or a healthcare facility. In any occupational setting, the methods for hazard identification and control utilize good industrial hygiene practices (see Chapter 4, Industrial Hygiene).

Chemical Hazards

Exposures to chemicals—solids, liquids, or vapors—occur through dermal absorption, inhalation, or ingestion. The health effects, which can be acute or chronic, from exposure to chemicals range from mild (dermatitis) to severe (mutagenicity, teratogenicity, and carcinogenicity). The effects depend on the extent (concentration and duration) of exposure, the route of exposure, and the physical and chemical properties of the substance. The health effects that a chemical substance exerts may also be related to simultaneous exposure to other chemical or physical agents.[3] In most cases, exposures resulting from chemical accidents, spills, leaks, fires, and ventilation failures are more common than are problems from chronic exposure.[4]

The most common manifestation of toxicity from exposure to chemicals, and the most prevalent occupational illness among healthcare workers, is contact dermatitis. Nurses who administer drugs have the highest incidence. Housekeeping personnel, whose skin is frequently in contact with cleaners and disinfectants, are second. Dermatological reactions are also common among kitchen, radiography, pathology, surgical, and maintenance personnel from exposures to cleaners, disinfectants, solvents, and other chemical solutions.[4-6]

Exposures to aerosols and vapors are also potentially hazardous. Typical exposures include

- operating room personnel to anesthetic gases,
- pharmacy and nursing personnel to antineoplastic agents and hazardous drugs,
- central material supply workers to ethylene oxide,
- laboratory workers to aromatic solvents and formaldehyde, and
- dental personnel to mercury.

The multitude of chemicals found in healthcare settings prohibits their individual discussion, but several that have become notorious as a result of their mutagenic, teratogenic, carcinogenic, or acute toxicity warrant attention. These include (*a*) anesthetic gases, (*b*) antineoplastic agents and hazardous drugs, (*c*) ethylene oxide, (*d*) formaldehyde, (*e*) mercury, and (*f*) methylmethacrylate. Numerous other chemicals, including solvents, reagents, and disinfectants are also used in healthcare facilities and may be potentially hazardous to employees (Table 5-1). The scientific literature contains a wealth of information pertaining to the hazardous properties of chemicals.[7-12]

Anesthetic Gases

Anesthetic gases (such as nitrous oxide, halothane [Fluothane], enflurane [Ethrane], and isoflurane [Forane]) can be released into work areas of the healthcare facility: operating rooms, recovery rooms, labor and delivery rooms, dental operatories, and veterinary clinics.[13-16] The implications of occupational exposure to low concentrations of common anesthetic agents remains controversial.[4,17-19] The evidence for specific chronic effects and the exposure concentrations at which they occur are conflicting; however, the literature consistently indicates an association with various short-term, acute effects such as neurotoxicity. Workers exposed to excessive amounts of anesthetic gases complain about feeling as if they themselves are anesthetized. They experience drowsiness, irritability, depression, headache, nausea, fatigue, and impaired judgment and coordination.[13,20-23] These behavioral modifications are of great concern, particularly in the operating room, where they can compromise surgical success and the health of the operating-room personnel.

Assessing the long-term effects of exposure to anesthetic agents is more difficult. The chronic effects of anesthetic gas exposures are usually identified through retrospective epidemiological studies, followed by confirmational animal studies. The conclusions that could be drawn in some studies of chronic low-level exposures have been limited due to the lack of quantitative exposure data and heavy reliance on information from questionnaires.[4,17,18,24] However, chronic exposure to waste anesthetic gases has been associated with increased risk of spontaneous abortion in exposed women workers and the wives of exposed men. Other adverse reproductive effects among exposed females include involuntary infertility and infants with low birth weights and congenital abnormalities.[19,25] Most of these studies took place before scavenger systems for recovering of waste gas were installed, and the current opinion holds that, with proper functioning scavengers and ventilators, the risk of overexposure is greatly reduced.[26,27]

TABLE 5-1

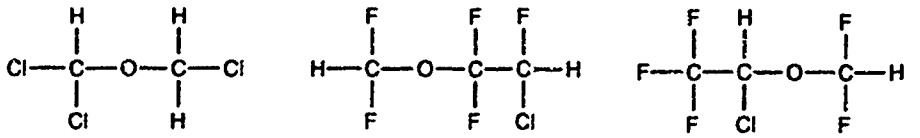
HAZARDS OF SELECTED SOLVENTS, REAGENTS, AND DISINFECTANTS*

Chemical	Main Biological Effects	Type of Work	Work Site	OSHA PEL in 29 CFR
Dioxane	Potential carcinogen Liver and kidney injury Neurotoxicity	Preparation of tissue sections Radioimmunoassay	Histology lab Serology lab	1910.1000
Benzene	Carcinogen (leukemia) Neurotoxicity	Chemistry procedures	Laboratory	1910.1028
Benzidine-based dyes	Carcinogen (bladder) Neurotoxicity	Biological stains Chemistry procedures Print dyes	Histology lab Chemistry lab Print shop	1910.1010
Xylene	Neurotoxicity Cardiovascular effects Reproductive effects Liver and kidney injury	Solvent Tissue processing	Histology lab Chemistry lab	1910.1000
Toluene	Neurotoxicity Cardiovascular effects Reproductive effects Liver and kidney injury	Solvent Tissue processing	Histology lab Chemistry lab	1910.1000
Chromic acid	Carcinogen (lungs) Irritant	Tissue processing	Histology lab	1910.1000
Phenol	Neurotoxicity Liver and kidney injury	Disinfection	Housekeeping Laboratory	1910.1000
Glutaraldehyde	Mutagenicity Respiratory effects Dermatitis	Tissue fixation Disinfection Dermal treatment X-ray file processing	Histology lab Central supply Dermatology Radiology	1910.1000
Picric acid (crystalline)	Liver and kidney injuries Dermatitis Gastrointestinal effects Hematological effects	Chemistry procedures	Chemistry lab	1910.1000
Azide	Neurotoxicity Cardiovascular effect Respiratory effects	Blood chemistries	Serology lab	None

*See 29 CFR, Part 1910 § 1000. *Occupational Exposures to Hazardous Chemicals in Laboratories.*

The possibility that a carcinogenic effect could result from exposure to anesthetic gases also has attracted attention.[3,20,28,29] The concern about this effect is partially due to the structural similarities between known human carcinogens (dibromoethane, dichloroethane, *bis*-chloromethyl ether, and chloromethyl methyl ether) and several of the halogenated inhalation anesthetics now in use (Figure 5-1). In addition, anesthetic compounds can be transformed into reactive metabolites, which can combine with tissue macromolecules and possibly initiate a carcinogenic event.[29,30] Several studies have noted elevated rates of specific cancers in hospital personnel who are chronically exposed to anesthetic gases: a higher incidence of death from reticuloendothelial and lymphoid malignancies was reported in anesthesiologists[31]; and a 3-fold increase in malignancies, which included unusual tumor types, was also noted in nurse anesthetists.[32]

Although in 1977 NIOSH recommended a standard to limit exposure to waste anesthetic gases, no federal regulatory standard currently exists.[13] The U.S. Army



bis-chloromethyl ether enflurane isoflurane

Fig. 5-1. Among the halogenated anesthetics currently in use, *bis*-chloromethyl ether is a recognized human carcinogen, with a Threshold Limit Value–time-weighted average (TLV-TWA) of 0.001 ppm. The structural similarities of other halogenated inhalation anesthetics give rise to the concern that they may also play a role in the development of cancer.

Office of The Surgeon General (OTSG) promulgated the U.S. Army exposure standards in 1982 in Technical Bulletin, Medical (TB MED) 510.[26] TB MED 510 has been revised and the 1993 draft revision is being staffed at OTSG. This draft contains the proposed army permissible exposure levels (PELs), which are the same time-weighted average (TWA) levels shown in Table 5-2. These particular TWAs are calculated from airborne concentrations that are measured over the time the anesthetic is administered. Therefore, they are *not* the 8-hour TWA exposures typically described by the Occupational Safety and Health Administration (OSHA) PELS or the American Conference of Governmental Industrial Hygienists' Threshold Limit Values (ACGIH's TLVs). This guidance applies to field hospitals during peacetime training but does not apply in combat zones.

When a halogenated anesthetic agent is used in combination with nitrous oxide, the TWA exposure limit becomes 25 ppm for nitrous oxide and 0.5 ppm for the halogenated agent. This reduction in the exposure limit is based on reported decrements in worker performance, which are believed to be caused by synergistic effects of exposure to both classes of anesthetic agents simultaneously, not to an increased health hazard.[20-23] There are no OSHA PELs, and the current TLVs are 50 ppm for nitrous oxide, 50 ppm for halothane, and 75 ppm for enflurane; therefore, the 0.5-ppm and 25-ppm exposure levels for nitrous oxide when used in conjunction with a halogenated anesthetic agent (which was recommended by NIOSH in 1977) is quite conservative. For this reason, the concept of an action level (one-half the PEL) that is customarily used in occupational health is not applicable to exposure limits for waste anesthetic gases.

Exposure to waste anesthetic gases can be controlled by following the guidelines set forth in TB MED 510 and by ensuring that employees are aware of the exposure sources. Employees should know that exposures usually result from careless work practices (such as an improper seal with the patient's mask, not eliminating anesthetics before removing the patient's mask or endotracheal tube, and not washing anesthetic gas from the patient's lungs with oxygen); leaking anesthetic equipment; inadequate waste-gas collection and containment (scavenging systems); and, to a lesser extent, poor general ventilation.

Antineoplastic Agents and Hazardous Drugs

Antineoplastic agents (cytotoxic drugs) are chemically unrelated but are capable of inhibiting tumor growth by disrupting cell division and killing actively growing cells.[33] They can be divided into structurally separate drug classes: (*a*) alkylating agents, (*b*) antibiotics, (*c*) antimetabolites, (*d*) mitotic inhibitors, and (*e*) a miscellaneous class (Exhibit 5-2).

Alkylating agents act by covalently binding to DNA, thus interfering with normal DNA replication. Antibiotics work as DNA intercalators, and interfere with

TABLE 5-2

PERMISSIBLE EXPOSURE LEVELS (PELS) FOR WASTE ANESTHETIC GASES

Anesthetic Gas	Concentrations (ppm)*
N_2O	50†
Halogenated agents used alone	2†
N_2O and halogenated agent used together	25 (for N_2O) + 0.5 (for halogenated agent)‡

*Time-weighted averages (TWAs)

†These values were adopted as PELS by the California state Occupational Safety and Health Standards Board on 24 February 1992 (General Industry Safety Orders § 5155)

‡Source: National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Waste Anesthetic Gases and Vapors.* Cincinnati, Oh: NIOSH; 1977. DHEW (NIOSH) Publication 77-140.

EXHIBIT 5-2

COMMON ANTINEOPLASTIC AGENTS*

Alkylating Agents
- Busulfan
- Carmustine (BCNU)
- CCNU (Lomustine)
- Chlorambucil
- Chloranphazin
- Cisplatin (Platinol)
- Cyclophosphamide (Cytoxan), (Neosar)
- Dacarbazine (DIC) (DTIC)
- Melphalan (Alkeran)
- Myleran
- Nitrogen mustard (Mustangen)
- Streptozocin (Zanosar)
- Triethylene thiophosphoramide (Thiotepa)
- Teosulfan
- Uracil mustard (Uramustine)

Antibiotics
- Bleomycin (Blenoxane)
- Dactinomycin (Actinomycin-D), (Cosmegen)
- Daunorubicin (Cerubidine)
- Doxorubicin (Adriamycin)
- Mithramycin (Mithracin)
- Mitomycin (Mutamycin)

Antimetabolites
- Azathioprine
- Cytosine arabinoside (Cytosar-U)
- Fluorouracil (Adrucil)
- Mercaptopurine
- Methotrexate (Mexate), (Folex)
- Procarbazine (Matulane)

Mitotic Inhibitors (Vinca alkaloids)
- Etoposide (VP-16-213), (VePesid)
- Vincristine (Oncovin)
- Vinblastine (Velban)

Miscellaneous
- L-Asparaginase (Elspar)

*List is not exhaustive

transcriptional processes in protein synthesis. Antimetabolites block the synthesis of essential cellular building blocks such as folate, purines, and pyrimidines, thereby inhibiting protein synthesis. Antimitotic agents act primarily as spindle poisons, and block mitosis and normal cell division. The miscellaneous category contains agents with various effect mechanisms. Several of these agents are mutagenic, carcinogenic, and toxic to the reproductive system and are discussed in greater detail later in this chapter (Table 5-3).[34,35]

Patients treated with these drugs have had significant adverse outcomes: hematopoietic effects and occurrences of second malignancies (usually hematological malignancies),[36,37] impaired reproductive function,[38] immunosuppression,[39,40] and case reports of malformed infants born to treated mothers.[41,42] These reports, together with laboratory evidence of the mutagenic activity of antineoplastic agents, have triggered concern about possible long-term health risks to healthcare personnel who handle these drugs.

Several investigations that attempted to assess this risk found increased measures of mutagenicity,[43-47] but contrarily, others found no excesses in workers who handle these agents.[48,49] Two epidemiological studies, both published in 1985, regarding reproductive outcomes of female workers exposed to antineoplastics are notable:

- Exposure to antineoplastic drugs during their first trimester of pregnancy was found to be significantly more common among nurses who gave birth to malformed infants than among those who delivered normal infants.[24]
- A statistically significant association was found between occupational exposure to antineoplastic drugs during the first trimester of pregnancy and fetal loss.[50]

These findings suggest that a significant reproductive risk may be incurred by workers who handle antineoplastic agents during pregnancy. Virtually all the reports that are discussed in these two epidemiological studies describe studies performed on oncology nursing and pharmacy personnel. However, several antineoplastic agents—cyclophosphamide, for example—are increasingly being employed for nonmalignant illnesses. Thus, the potential for exposing workers in other sectors of the healthcare setting will expand.[51-53]

While the primary focus of occupational exposure to these agents has been on measures of mutagenicity and potential chronic disease outcome (such as cancer), acute effects in exposed workers have also been reported among nurses and pharmacists who handle the drugs. These effects include dizziness, headaches, facial flushing, and nausea[54-56]; and bronchospasm, vomiting, and diarrhea.[57]

TABLE 5-3

TOXIC PROPERTIES OF REPRESENTATIVE ANTINEOPLASTIC AGENTS

Agent	Chromosomal Mutagenic*	Chromosomal Effects†	Carcinogenic‡	Teratogenic§
Actinomycin D	–	Chr ab	+ r, m; (+) hum	+ sev sp
Adriamycin	+	Chr ab, SCE	+ r	–
Azacytidine	+	–	(+) m	+ m
Azathioprine	+	–	(+) m, r; + hum	+ sev sp
Bleomycin	–	SCE	–	–
Busulfan	+	Chr ab, SCE	(+) m; + hum	–
Carmustine (BCNU)	+	Chr ab	+r	+ r
Chlorambucil	+	Chr ab	(+) m, r; (+) hum	–
Cisplatin	+	Chr ab	–	–
Cycloposphamide	+	Chr ab, SCE	+ m, r, hum	+ sev sp
Dacarbazine	+	–	+ m, r	+ sev sp
Danunorubicin	+	Chr ab	–	–
Fluorouracil	–	–	–	+ sev sp
Isophosphamide	+	Chr ab	(+) m, r	+ m
Lomustine (CCNU)	+	SCE	+ r	+ r
Melphalan	+	Chr ab, SCE	+ m, r, hum	-
Mercaptopurine	+	Chr ab	–	+ sev sp
Methotrexate	+	Chr ab	–	+ sev sp, + hum
Mitomycin C	+	Chr ab	–	
Prednisone	–	–	–	+ rod
Procarbazine	+	–	+ m, r	+ r
Streptozotocin	+	–	–	–
Thiotepa	+	Chr ab	+ m, r	+ m, r
Treosulfan	–	Chr ab	+ hum	–
Uracil mustard	+	–	+ m, r	+ r
Vinblastine sulfate	–	–	–	+ sev sp
Vincristine sulfate	–	–	–	+ sev sp

*+: mutagenic to bacterial or mammalian cells in culture; –: not mutagenic to bacterial or mammalian cells in culture

†: Chr ab, increased incidence of chromosomal aberrations; SCE, increased incidence of sister-chromatid exchange

‡+: sufficient evidence; (+): limited evidence for carcinogenicity to mice (m), rats (r), or humans (hum) according to the International Agency for Research on Cancer (IARC)

§+: teratogenic to mice (m); rats (r); rodents (rod); several animal species (sev sp); or humans (hum)

Reprinted with permission from Edling C. Anesthetic gases. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions.* Boca Raton, Fla: CRC Press, Inc; 1989: 133. © CRC Press, Inc, Boca Raton, Fla.

The genotoxic nature of many of the antineoplastics, together with evidence of second malignancies in addition to the occupational populations studied, prompted OSHA to issue guidelines for handling antineoplastic drugs in 1986.[58] The guidelines recommend the use of laminar airflow biological safety cabinets in drug preparations as well as personal protective equipment (PPE), worker education, standing operating procedures (SOPs) for handling, and medical surveillance for workers. Although not federal standards (and therefore not carrying the force of law), these guidelines may be enforced under the OSHA General Duty clause requiring employers to provide a safe and healthful workplace free of known hazards.[59] Although OSHA specified little detail in the surveillance examination content recommended for drug handlers, some guidance can be obtained in the literature.[60]

Antineoplastic agents should be prepared in a Class II biological safety cabinet (BSC) that conforms to the current National Sanitation Foundation Standard No. 49 (Figure 5-2). A Class II, Type A BSC is the minimum requirement for worker protection, but a Class II, Type B BSC is preferred.[61] Class I, Types A and B BSCs have vertical, laminar airflow. A horizontal-airflow cabinet must *never* be used for preparation of antineoplastic agents: it blows air that has been filtered through a

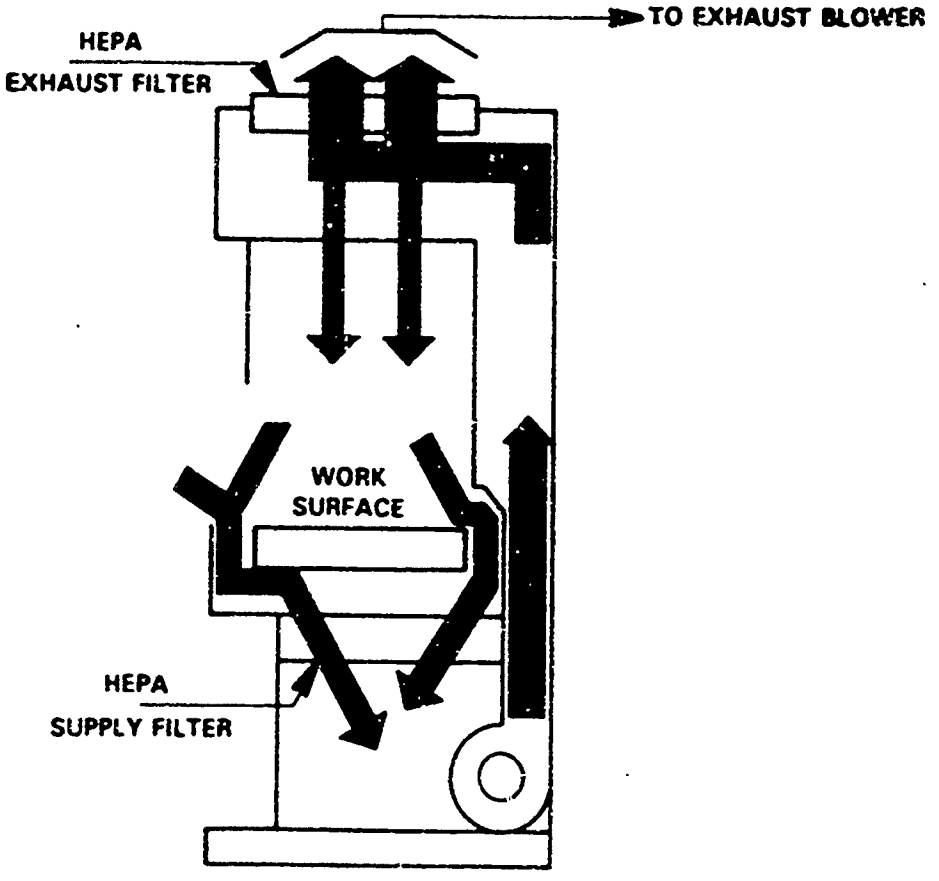


Fig. 5-2. Class II Laminar Flow Biological Safety Cabinet (BSC). The laminar airflow through the high-efficiency particulate air (HEPA) filter in the supply air provides sterile working conditions for drug preparation. The BSC also protects the worker by drawing air through the sash, thus preventing the antineoplastic agent from leaving the cabinet and entering the worker's breathing zone. The air passes through a second HEPA filter before it is exhausted. Source: Noll S, Caldwell DJ. *Guidelines for the Handling, Administration, and Disposal of Cytotoxic Drugs.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1987: 4. Technical Guide 149 (to be published as Technical Bulletin MED 515).

high-efficiency particulate air filter (HEPA-filtered air) over the work area to keep the drug sterile, but then exhausts the filtered air directly into the drug preparer's breathing zone.

The blower of either vertical-airflow BSC should be turned on at all times (24 h/d, 7 d/wk). Venting exhaust air to the outside is preferable where possible, and is required with a Class II, Type B BSC.[56] The exhaust air should be filtered, discharged at an appropriate height (1.3-fold greater than the height of the building), and directed away from air-intake units. Drugs should be prepared only when the movable sash is fixed at the required operating level to accommodate the drug-reconstitution procedure.

More recently, awareness that other pharmaceuticals in the hospital setting were also potentially hazardous but were not, strictly speaking, antineoplastics, prompted a committee of the American Society of Hospital Pharmacists (ASHP) to define a class of agents as *hazardous* drugs.[62] This report specified concerns about antineoplastic and nonantineoplastic hazardous drugs in use in most institutions throughout the country. The antiviral agent zidovudine (AZT) should be classified as a hazardous drug but is not thought of as antineoplastic. Recently, AZT was found to be carcinogenic in animals and thus is a potential human carcinogen.[63]

Unfortunately, the ASHP committee did not identify the specific drugs that should be classified as hazardous, leaving the compilation of such a list to individual institutions. The committee did, however, describe the following characteristics of drugs that

could be considered hazardous:

- genotoxicity,
- carcinogenicity in animal models, the patient population, or both, as reported by The International Agency on Research in Cancer,
- teratogenicity or fertility impairment in animal studies or treated patients, and
- evidence of serious organ or other toxicity at low doses in animal models or treated patients.

Guidelines for identifying potentially hazardous drugs in the hospital environment and clarifying their handling can be found in the literature.[64]

Handling antineoplastic and other hazardous drugs may expose healthcare workers to known carcinogens and reproductive toxicants. Implementing a comprehensive program of worker education, engineering and administrative controls, and medical surveillance will ensure the safest workplace possible, one where these useful therapeutic agents may be used without risking the workers' health.

Ethylene Oxide

Ethylene oxide is used routinely in healthcare facilities as a gaseous sterilant for heat- or moisture-sensitive equipment and instruments. In its pure form, ethylene oxide is highly flammable. Therefore, it is typically supplied in compressed-gas cylinders, which contain 88% Freon and 12% ethylene oxide, or in single-use cartridges of 100% ethylene oxide.[3]

In 1977, NIOSH recognized ethylene oxide as a hazard in healthcare facilities, and since then, attention has been focused on the hazard and its effects.[65] The acute toxic effects of exposure to ethylene oxide include respiratory and eye irritation, skin sensitization, vomiting, and diarrhea; the chronic effects include secondary respiratory infection, anemia, and neurotoxicity. In 1981, NIOSH published evidence of ethylene oxide's animal carcinogenicity and the recommended exposure limit was reduced from 50 to 1 ppm.[66] The report also noted adverse reproductive effects in mammals and possible chromosomal aberrations in workers.

Since 1981, NIOSH has completed a cytogenic study that shows an increase in sister-chromatid exchanges (a measure of point mutation) and chromosomal aberrations in monkeys that were exposed to ethylene oxide. Another NIOSH study performed during this period demonstrated statistically significant associations between ethylene oxide exposure and increased incidence of neoplasms in rats.[67] In addition, a literature review of studies of workers who were exposed to ethylene oxide indicates increased mutagenic activity in human cells, carcinogenesis, reproductive abnormalities, and neurological defects.[68] In 1984, OSHA issued a new standard, 29 CFR 1910.1047, to protect workers exposed to ethylene oxide: the PEL was reduced to 1 ppm.[69] The standard was revised in 1988 to include a 15-minute short-term exposure limit (STEL) of 5 ppm.

Exposures to ethylene oxide in a healthcare facility usually occur when sterilizers and aerators are operated or during maintenance and handling of preaerated packages.[70] During these operations, skin contact with ethylene oxide gas or liquid can cause skin irritation, but the primary route of exposure is inhalation (Figure 5-3). Because the odor threshold of ethylene oxide is 700 ppm and the mucous-membrane irritation threshold is 200 ppm, odor and irritation do not provide adequate warning to workers who may be exposed to levels higher than the PEL of 1 ppm.[3] Therefore, stringent control procedures are essential to meet the current standard (the federal law). Practices should include the following:

- routine environmental monitoring and medical surveillance,
- routine equipment maintenance and leak checks,
- effective sterilizer and aerator local exhaust ventilation, and
- the use of ambient ethylene oxide concentration alarms, general ventilation, and work procedures designed to reduce exposure.[67,69,71-73]

Formaldehyde

Formaldehyde is a common and hazardous chemical that is often controlled poorly in healthcare facilities.[4] The most extensive exposures occur while it is used in autopsy rooms and pathology laboratories as a tissue preservative[74,75]; in hemodialysis units as a disinfectant[76,77]; and in central material supply as a cold sterilant for various instruments.[2] (Although embalming facilities are not specifically addressed in this chapter, many hazards to healthcare workers are also hazards to embalmers, formaldehyde being an excellent example.)

As with other hazardous chemicals, the effects of exposure to formaldehyde depend on the duration and extent of the exposure. Low levels of exposure (< 1 ppm) may cause direct irritation of the skin, eyes, nose, throat, and lungs.[4,78] Higher concentrations (10–20 ppm) may cause coughing, chest tightness, increased heart rate, and a sensation of pressure in the

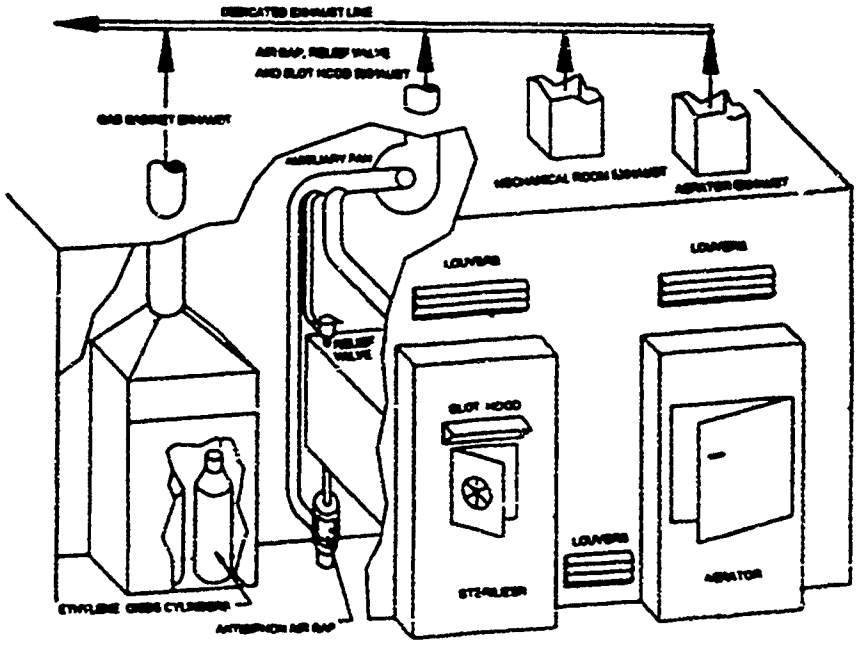


Fig. 5-3. Typical ethylene oxide sterilization equipment includes the ethylene oxide sterilizer, an aerator used to dissipate residual ethylene oxide after materials are sterilized, the ethylene oxide cylinder storage cabinet, and a local exhaust ventilation system to capture and remove ethylene oxide at the points of emission. To protect the health of hospital workers, the extent of ethylene oxide exposure must be characterized. Ethylene oxide concentrations in ambient air can be measured by a variety of sampling methods, and once the extent of exposure is known, engineering or administrative controls can be implemented to reduce the workers' exposure. A properly designed ventilation system can significantly reduce worker exposure to ethylene oxide. Sources: (1) Caldwell DJ. Evaluation of an add-on local exhaust ventilation system for an ethylene oxide (ETO) sterilizer. *Appl Ind Hyg.* 1989;4:88–91. (2) National Institute for Occupational Safety and Health. *Ethylene Oxide Sterilizers in Health Care Facilities, Engineering Controls and Work Practices.* Cincinnati, Oh: NIOSH; 1989: 4. Current Intelligence Bulletin 52.

head. Concentrations of 50 to 100 ppm are associated with pulmonary edema and death.[3,79]

Repeated exposure to formaldehyde vapors causes some healthcare workers to become sensitized. This may occur days, weeks, or months after the first exposure. Immunogenic responses include eye irritation, upper respiratory irritation, or an asthmatic reaction at levels of exposure too low to cause symptoms in most people. Reactions can be quite severe with swelling, itching, wheezing, and chest tightness.[3,6]

Direct contact with formaldehyde solutions can cause severe eye injury and corneal damage and dermatological signs. Primary irritation has been elicited when human skin has contacted solutions as dilute as 4%. Dermatitis (including red, sore, cracking, and blistered skin) is a common complaint; continuous contact may make fingernails soft and brown.[3,78]

As a reactive alkylating agent, formaldehyde is a biologically plausible potential human carcinogen because (*a*) other similar compounds are known or suspected to induce malignancies and (*b*) formaldehyde could be expected to react at the surface of the respiratory tract.[80] Several studies with animals have demonstrated experimentally that formaldehyde is both a mutagen and a carcinogen. In addition, inconclusive human epidemiological studies have associated formaldehyde exposure with cancers of the lung, nasopharynx, oropharynx, and nasal passages. This inevitably raises concern about chronic low-level exposures of humans.[3,4,75,76,79,80]

Standards and controls have been established to limit exposure to formaldehyde. NIOSH first proposed a recommended standard for formaldehyde in 1976 and published evidence of carcinogenicity in 1981.[78,79] OSHA currently regulates formaldehyde: the PEL for an 8-hour TWA is 1.0 ppm; the 15-minute STEL is 2.0 ppm; and the *action level* (the level at which workers must be enrolled in medical surveillance programs) is 0.5 ppm.[69] Occupational exposures are reduced by

- substituting safe products,
- using laboratory hoods,
- wearing appropriate PPE,
- instituting good work practices,
- installing and maintaining general ventilation, and
- training healthcare workers about the relevant hazards.[3,75,81,82]

Mercury

Mercury is used in many types of hospital equipment (manometers, thermometers, Coulter counters, Van Slyke apparatus, Miller-Abbot and Cantor tubes, and sphygmomanometers) and in tissue fixatives and dental amalgams.

Exposures to mercury in healthcare settings usually result from accidental spills, but they can also occur during routine work practices.[3] Additionally, droplets can become trapped in carpets and cracks in floors or counters. These droplets, which vaporize readily at room temperature, are not removed easily during routine cleaning and produce continuous exposure. Central material supply and maintenance personnel are exposed when biomedical equipment breaks or is repaired.[83] Technicians in histology laboratories are subjected to mercuric compounds during routine procedures.[84] However, the greatest potential for mercury exposure is found in dental clinics. Mercury-contaminated dust in dental laboratories is generated when mercury amalgam is cut, ground, and polished. In addition, vapors arise from mechanical amalgamators and ultrasonic amalgam condensers; when amalgam is mulled in the hand, or when excess mercury is squeezed from freshly mixed amalgam; when old fillings are removed; when amalgam-contaminated instruments are hot-air sterilized, and when mercury and amalgam scraps are stored.[85]

The adverse health effects associated with the absorption of mercury vapor through the lungs and skin prompted the establishment of occupational exposure standards and controls (Table 5-4). NIOSH recommended, and OSHA promulgated, a PEL of 0.05 mg/m^3.[69,86] Toxic mercury exposures can be minimized by installing impervious flooring and counters, instituting good work practices, effective handling of spills, good storage procedures, appropriate PPE, periodic air monitoring, good ventilation, and employee education.[87,88]

Methylmethacrylate

Methylmethacrylate is an acrylic cementlike substance derived from mixing a liquid containing methylmethacrylate monomer with polymethylmethacrylate powder immediately before using in orthopedic and other procedures.[89] Workers in healthcare facilities are subjected to exposures through inhalation of vapors, skin contact, or both. Those at risk include technicians who make and mend acrylic dentures and hearing aids, orthopedic surgical personnel who use the cement for fixation of metallic and plastic prostheses, and pathology personnel who work in areas where methylmethacrylate is used for imbedding histological preparations.[90]

Myriad health effects have been associated with exposure to methylmethacrylate. It is an eye, skin, and mucous-membrane irritant and is known to cause contact dermatitis and occupational asthma.[3,90] Surgical patients exposed to this compound have suffered acute episodes of hypotension and cardiac arrest.[91] In a 1976 study, NIOSH reported adverse health effects such as cutaneous, genitourinary, and respiratory complaints in workers exposed to methylmethacrylate in concentrations lower than 50 ppm.[92] Studies with

TABLE 5-4

HEALTH EFFECTS OF EXPOSURE TO MERCURY

Categories	Health Effects
Short-Term Exposures to High Levels	Severe respiratory irritation Chemical pneumonitis Digestive disturbances Marked renal damage
Chronic Low-Level Exposures	Tremor . ia Speech disturbance Psychic and emotional changes (irritability, combativeness and fatigue)
Associated Signs	Inflammation of the gums Excessive salivation Anorexia Weight loss Sensitization dermatitis

Sources: (1) US Department of Health and Human Services. *Guidelines for Protecting the Safety and Health of Health Care Workers.* Washington, DC: DHHS (NIOSH); 1988. Publication 88-119. (2) Patterson WB, Craven DE, Schwartz DA, Nardell EA, Kasmer J, Nobel J. Occupational hazards to hospital personnel. *Ann Intern Med.* 1985;102:658–680. (3) National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Inorganic Mercury.* Cincinnati, Oh: DHEW (NIOSH); 1973. Publication 73-1009.

animals have shown that methylmethacrylate is teratogenic[93] and mutagenic with the potential for carcinogenicity.[24] There have also been multiple findings of liver damage in rats exposed to various levels of methylmethacrylate.[89]

The OSHA PEL for methylmethacrylate is 100 ppm.[69] Exposure can be reduced by using portable or permanent local exhaust units when mixing the components; wearing appropriate PPE for the eyes, hands and body; practicing careful personal hygiene; and providing hazard-recognition training.[3,89]

Biological Hazards

The germ theory of disease made acceptable the fact that disease is spread by ill persons and fomites (contaminated objects). The germ theory also allowed for the recognition that patient care could, therefore, pose risks to healthcare facility workers. Medical history is replete with anecdotal reports of medical personnel who have succumbed to infectious diseases that were contracted during their work with patients or specimens from patients.[95] Recent attention has focused on the contribution of infectious diseases to the overall burden of work-related illnesses found among healthcare workers. Exposures to bacterial, viral, fungal, and parasitic organisms pose a constant threat to healthcare facility workers in essentially every work area. The healthcare professionals at greatest risk for exposure are medical practitioners,[4,95,96] dental practitioners,[97,98] and laboratory workers.[99] In addition, housekeeping, laundry, maintenance, and supply personnel within the healthcare environment also incur some degree of risk from contact with patient waste, soiled laundry, or contaminated equipment.

Within the healthcare setting, general infection control procedures have been developed to minimize the risk of nosocomial infection.[100-105] Such procedures are designed to prevent transmission of microbiological agents and to provide a margin of safety in the varied situations encountered in the healthcare environment. The modes of transmission found in the healthcare setting are also observed in the working environments of paramedics, emergency medical technicians, and public-safety employees. Therefore, the precautions developed for healthcare organizations are also applicable to these settings. Good infection and biosafety control measures include the following:

- eliminating infective organisms with systemic antimicrobial agents, disinfection, and sterilization;
- eliminating contact, airborne, or fomite transmission routes through personal hygiene (especially handwashing); judicious use of gloves, masks, and gowns; isolation techniques; and proper ventilation; and
- reducing worker susceptibility with immunization, medical surveillance, physical exams, and effective hazard training programs.

Many agents—including tuberculosis, varicella, and rubella—pose significant threats and deserve attention. However, the current interest of the medical community is strongly oriented toward exposure to blood-borne pathogens, especially the hepatitis B virus (HBV), the hepatitis C virus (HCV), and the human immunodeficiency virus (HIV). In December 1991, OSHA promulgated the final rule for occupational exposure to blood-borne pathogens.[106] This performance-oriented law states the required standards; however, it permits the employer to develop and implement individual programs that are protective and cost effective. The standard requires that the employer (*a*) produce a written exposure control plan, (*b*) identify those employees at risk for occupational exposure to blood and other infectious material, (*c*) provide appropriate PPE and enforce wearing compliance, and (*d*) provide hazard training for the employees. Housekeeping requirements and decontamination procedures, including a written schedule for cleaning and discarding sharps and regulated wastes, are also addressed in the standard. Limiting a worker's exposure to blood-borne diseases is achieved by implementing the following categories of controls:

- engineering;
- immunization programs;
- work practices, such as procedures for handling sharps;
- disposal and handling of contaminated waste;
- use of PPE such as gloves and gowns;
- use of mouth pieces, resuscitation bags, and other ventilation devices;
- use of disinfectants;
- labeling and signs; and
- training and education programs.

All healthcare facilities are required to comply with Title 29, Code of Federal Regulations (CFR) 1910.1030. As an example of the level of compliance that is required, an excerpt from Walter Reed Army Medical Center's *Exposure Control Plan*, adopted 4 May 1991, is included at the end of this chapter.

In 1982 and 1983, the Centers for Disease Control

(CDC) issued precautions against acquired immunodeficiency syndrome (AIDS) for healthcare facility workers and allied professionals.[107,108] In 1985, the CDC developed the strategy of *universal blood and body-fluid precautions* to address concerns regarding transmission of HIV in the healthcare setting.[107] This concept (now simply called the *universal precautions*) stresses that (*a*) all patients should be assumed to be infectious for HIV and other blood-borne pathogens and (*b*) health-care workers should perform their duties with prescribed work practices. Universal precautions apply in the healthcare environment when workers are exposed to blood and certain other body fluids (including amniotic, pericardial, peritoneal, pleural, synovial, and cerebrospinal fluids, and semen and vaginal secretions), or any body fluid visibly contaminated with blood.

Some body fluids are exempted from these universal precautions because the transmission of HBV and HIV via exposure to them has not been documented. For example, universal precautions do not apply to saliva when it is not visibly contaminated, or is unlikely to be contaminated, with blood. In the dental setting, however, where saliva is likely to be contaminated, universal precautions do apply. When differentiation between body-fluid types is difficult or impossible, the CDC recommends that medical professionals should treat all body fluids as potentially hazardous. Other body fluids to which universal precautions do not ordinarily apply include feces, nasal secretions, sputum, sweat, tears, urine, and vomitus.[110]

The CDC usually presents information concerning HBV and HIV together for several reasons:

- the modes of transmission for HBV are similar to those of HIV;
- the potential for HBV transmission in the occupational setting is greater than that for HIV;
- a larger body of experience has accumulated relating to controlling transmission of HBV in the work place; and
- because HIV is fragile in the environment, general practices to prevent the transmission of HBV will also minimize the risk of HIV transmission.[110,111]

Precautionary measures to prevent the spread of both HIV and HBV are found in various publications, which address general universal precautions, invasive procedures, autopsies, dialysis, blood or body-fluid spills, waste, emergency medical treatment, dentistry, laboratories, housekeeping, and laundry.[105,110-114]

Physical Hazards

Noise

Some workers in healthcare facilities encounter exposures exceeding the present OSHA standard of an 8-hour TWA of 90 dBA.[69] Most healthcare workers, however, are subjected principally to nuisance levels that are annoying and may interfere with work.[115-120] Noise can become a problem in food-service areas, laboratories (hospital and dental), maintenance and engineering areas, brace shops, incinerators, orthopedic cast rooms (from cast cutting), administrative areas (from printing and reproduction), and dental operatories (from high-speed hand pieces). Technical and physiological aspects of noise and noise control are addressed comprehensively in Chapter 7, Noise and the Impairment of Hearing.

Radiation

Sources of ionizing and nonionizing radiation are present in many areas of fixed medical, dental, and veterinary facilities. Most radiation sources are used for diagnostic and therapeutic purposes; other uses include food preparation with microwave ovens and germicidal treatment of room air with ultraviolet light. Additionally, diagnostic X-ray equipment can be found in field medical units. The health threats posed by these sources and appropriate control measures are discussed in Chapter 15, Nonionizing Radiation and Chapter 16, Ionizing Radiation.

Musculoskeletal Strain

Among the most common problems encountered by healthcare facility workers are back pain and musculoskeletal injury; these are the primary reasons for job-related lost time among these workers.[96,121,122] Most of these problems are associated with workers' attempting to lift or transfer patients. Those workers who are physically unfit, unaccustomed to the task being performed, suffering from postural stress, or doing work that approaches or exceeds the limits of their strength are at greatest risk. Other contributing factors include understaffing, lack of regular training programs regarding the proper procedures for lifting and other work motions, and inadequate general safety precautions.[3,123] The healthcare personnel associated with a high risk for sustaining back problems include surgeons, nurses, nurses aides, emergency medical technicians, dentists, dental assistants, physical and

occupational therapists and aides, radiology technicians, housekeeping and laundry workers, food service employees, maintenance and supply personnel, and, to a lesser extent, laboratory technicians and clerical staff.[3,96,124,125]

Primary and secondary approaches to preventing back pain and injury are the foundation of any back-injury-prevention program. In general, the primary approach to prevention involves reducing manual lifting and other load-handling tasks that are biomechanically stressful. The secondary approach relies on teaching workers how to perform stressful tasks while minimizing the biomechanical forces on their backs. The secondary approach also emphasizes maintaining flexibility and strengthening the back and abdominal muscles.[3] In addition to these approaches, several important techniques to prevent back injuries among hospital staff can be employed (Exhibit 5-3). Written guides and programs for preventing back pain and injury are available for all workers and specifically for healthcare personnel.[126-128]

Psychosocial Hazards

Workers in healthcare facilities face a variety of highly stressful work-related conditions in meeting the physical and psychological needs of patients (Exhibit 5-4). Supervisors and workers must be able to identify the many manifestations of psychological stress and be knowledgeable about stress-management techniques. Additionally, shift work, a major cause of stress, must be implemented properly.

Emotional Stress

Healthcare workers who are most subject to severe emotional stress while working include those in oncology units, burn units, emergency rooms, operating rooms, and intensive care units. Although most studies address the stress factors found among physicians and nurses, some have also identified laboratory and food-service work as high-stress occupations.[129-133]

EXHIBIT 5-3

TECHNIQUES TO PREVENT BACK PAIN AND INJURY TO HEALTHCARE WORKERS

Use mechanical devices for lifting patients and other heavy objects

Use wheels and other devices for transporting heavy, nonportable equipment

Provide adequate staffing to prevent workers from lifting heavy patients or equipment alone

Closely supervise newly trained workers to assure that proper lifting techniques have been learned

Use the preplacement evaluation of workers to identify those with existing back disorders and to tailor their job tasks to prevent additional injury

Educate and train both new and experienced staff on the proper measures for avoiding back pain, including: proper lifting techniques to prevent initial back pain (once back pain occurs, there is a higher probability for reoccurrence) and requesting help for tasks that may strain the back

Use proper patient-transfer techniques:

- Communicate the plan of action to the patient and other workers to ensure that the transfer will be smooth and without sudden, unexpected moves
- Position the equipment and furniture effectively (eg, move a wheelchair next to the bed) and remove obstacles
- Ensure good footing for the staff and patients (patients should wear slippers that provide good traction)
- Maintain eye contact and communication with the patient; be alert for trouble signs
- Request that a coworker stand by before attempting the transfer, if help is needed
- Record any problems on the patient's chart so that other shifts will know how to cope with difficult transfers; note the need for any special equipment, such as a lift

Post, remove, or repair accident hazards such as wet floors, stairway obstructions, and faulty ladders on step stools

Source: US Department of Health and Human Services. *Guidelines for Protecting the Safety and Health of Health Care Workers.* Washington, DC: DHHS; 1988. NIOSH Publication 88-119.

EXHIBIT 5-4

COMMON STRESS-ASSOCIATED FACTORS AMONG HEALTHCARE WORKERS

- Lack of essential support services and inadequate resources
- Strenuous work loads and prolonged work schedules
- Rotating shift work
- Sleep deprivation
- Working in unfamiliar areas
- Understaffing
- Discrimination
- Role conflict and ambiguity
- Underutilization of talents and abilities
- Lack of control and participation in planning and decision making
- Communication problems among aides, nurses, physicians, and administrators
- Lack of administrative rewards
- Keeping abreast of rapidly changing and increasingly complex technology
- Unrealistic self-expectations
- Guilt about negative feelings toward patients
- Participation in intense emergency situations
- Difficulty in dealing with deformity, terminal illness, and death
- Constant contact with ill and depressed patients
- Making rapid, complex, and critical decisions on the basis of inadequate data
- Constant interruptions that impair concentration
- Exposure to toxic substances and physical hazards
- Exposure to infectious patients
- Ergonomic factors

The manifestation of stress, which may ultimately lead to *burnout* (physical or emotional exhaustion from long-term stress), differs greatly among healthcare workers.[134,135] Stress can manifest as adaptive reactions such as delayed gratification, compulsiveness, and expressing the need for support. If continued for many years, some of these manifestations may lead to obvious physiological and psychological problems. Stress has also been associated with loss of appetite, ulcers, migraine headaches, fatigue, nausea, diarrhea, sleep disorders, oversleeping, increased smoking, disruption of social and family life, disorientation, disorganization, apathy, indecisiveness, reluctance to accept responsibility, and emotional instability. Stress also manifests as even more serious conditions: substance abuse, mental illness, suicide, and providing inadequate patient care (eg, careless examinations, poor treatment, abuse of patients, and gross sociopathic behavior).[3,4,6]

Methods of coping with stress have concerned educators, managers, and workers for many years (Exhibit 5-5). One study attempted to improve the work environment in a burn unit by providing feedback about the work setting and helping the staff use that information to formulate and implement changes[136]:

- The staff were encouraged to think about the elements of their work setting in terms of those elements that were stressful and those that were nonstressful.
- The staff began to focus on work-setting characteristics that are often overlooked, such as clarity of expectations.
- The staff attempted to effect change in only a few areas at a time, rather than in many.

As a result, improvements in morale and the quality of patient care were apparent:

- The staff's involvement in their work increased as they began to work together to affect change.
- The staff began to feel concern not only for their individual patients but also for all patients and staff members.

Shift Work

A major cause of stress in healthcare facilities is shift work, especially rotating work schedules. Shift-work–related stress results from three general problems: (1) disruption of the circadian rhythm (sleep-awake cycle), (2) disruption of social and family life, and (3) sleep deprivation. These factors may interact to produce deleterious effects on the general psychological and physical well-being of the shift worker. While there is insufficient evidence to demonstrate conclusively that shift work causes a specific illness, shift workers (especially those who rotate shifts) do have more health-related complaints such as digestive problems, chest pain, wheezing, nervousness, colds, and fatigue.[137]

As a rule, workers on rotating shifts dislike those aspects of their work schedules that violate circadian physiology. Worker satisfaction, subjective health estimates, personnel turnover, and productivity all seem to improve when schedules are designed to incorporate circadian principles.[136] Despite variations in current practice, most researchers advocate either a slow rotation of three or more weeks to permit circadian

EXHIBIT 5-5

METHODS FOR MANAGING STRESS

Institute educational sessions to improve skills and confidence
Institute stress-management and employee-assistance programs
Learn to identify the signs and sources of stress
Engage in activities to facilitate disengagement from work (such as hobbies)
Learn to reserve time and energy for oneself without feeling guilty
Emphasize the fun and reward of healthcare and intellectual achievement
Foster employee ability to recognize and respect each one's own limits
Provide readily available counseling from a nonjudgmental source
Provide group support systems, using a skilled neutral facilitator, for staff with particularly difficult professional problems
Facilitate effective teamwork and trust
Promote high-quality communication
Hold regular staff meetings and discussions to communicate feelings, gain support, and share innovative ideas
Encourage supervisory flexibility and innovation to create alternative job arrangements
Recognize and act on legitimate complaints regarding overbearing supervisors
Schedule rotation of assignments to allow adequate time for employee planning
Optimize shift-work schedules
Provide reasonable schedules for house staff to allow adequate time for sleep
Encourage organized and efficient work functions and environment
Provide adequate staff and resources

adaptation, or a rapid rotation of one to three consecutive nights followed by rest to prevent circadian disruption.[139] Shift changes should always be progressively later in rotation (ie, day to evening to night).[138]

STRATEGIES FOR HAZARD ABATEMENT

The initial step in eliminating or reducing hazards to human health in any healthcare setting is to develop a hazard inventory, which is usually the responsibility of the safety officer. This requires that the worksite hazards be observed, identified, and then compiled into an inventory. Support for this effort can be provided by the environmental science officer, industrial hygienist, occupational medicine physician, occupational health nurse, preventive medicine officer, and preventive medicine and industrial hygiene technicians. Hazard identification is only the first step; it is followed by the more difficult tasks of evaluation and control. Evaluation encompasses environmental sampling, surveillance, or both; detailed work-practice investigations; and medical surveillance. Hazard control comprises the following:

- diverse engineering interventions,
- proper ventilation,
- appropriate PPE,
- educational training for recognizing and avoiding hazards,
- safe work techniques or practices, and
- written safety or health procedures and programs that contain enforcement provisions.

Knowledge is the key to the prevention of hazardous exposures. To ensure that employees are knowledgeable about the hazards present and the proper use of safety equipment, personnel must be trained regarding

- the proper use of PPE;
- the potential hazards associated with toxic chemicals, equipment, and operations;
- safe work practices; and
- proper emergency procedures and abatement requirements.

THE MILITARILY UNIQUE ENVIRONMENT

U.S. Army healthcare operations in the field may be conducted using Table of Organization and Equipment (TOE) facilities (such as tents) and standard TOE material, supplies, and equipment. Additionally, field medical operations are sometimes carried out in existing facilities (such as buildings located in the training area or area of operations) using material, supplies, and equipment found in the fixed facility, listed in the TOE, or in any combination. The lack of an established, familiar fixed facility; the use of unfamiliar medical items; the absence of water, waste disposal, and other similar services in the field; and the physical and psychological stresses of training—or actual or threatened hostilities—can all greatly increase the potential that personnel in field healthcare facilities will face hazardous exposures and overexposures. Therefore, preventive medicine personnel assigned to division or corps preventive medicine sections or teams must be able to make (*a*) quick, thorough evaluations of the actual and potential health threats in field MTFs and (*b*) appropriate recommendations to reduce or eliminate the potential health threats. (There are no identified occupational medicine physician, occupational health nurse, or industrial hygienist positions in TOE units. All U.S. Army preventive medicine physicians, environmental science officers, sanitary engineers, and enlisted preventive medicine technicians receive training in occupational health and occupational medicine functions for TOE units.)

All actions taken to evaluate actual and potential hazards in a field MTF must be geared to the tactical situation. Circumstances may not allow the level and sophistication of hazard identification and evaluation normally found in a fixed medical facility. For example, state-of-the-art monitoring equipment and techniques may not be available, or may be impractical, and the time available for observing procedures within the facility may be limited. Additionally, each evaluation must be conducted with a clear understanding of the mission and priorities of the field facility and the means available to eliminate or control hazards. Therefore, preventive medicine personnel who conduct evaluations of field MTFs must be knowledgeable about field operations, able to quickly make qualitative assessments about hazards and their associated risks, and capable of clearly and concisely communicating to commanders (or their representatives) the significant hazards that require attention. Both a thorough base of knowledge about the hazards in medical facilities and common sense are absolute requirements. It is unrealistic to assume that an evaluation protocol used at an army medical center or other fixed medical facility could be directly applied to a field facility, particularly one that is engaged in receiving combat wounded. In the field, preventive medicine personnel must identify the hazards and threats and place them in perspective relative to the mission and task at hand.

The emphasis on hazard identification and control in an MTF may be dictated by the command surgeon, theater policy, or both.[140] Additionally, the nature of the diseases and injuries that occur may influence the emphasis placed on certain hazards. For example, if large numbers of patients present with enteric disease, then strict adherence to enteric-disease precautions would be warranted to ensure that spread of the disease-causing agent (patient-to-staff or patient-to-patient) would be minimized.

Depending on the type of unit and the nature of operations, the categories of hazards that are present in a field MTF will usually be similar to or the same as those found in a fixed facility. However, the harshness of the environment, disruption of the body's natural defenses through fatigue and other factors, and breakdowns in basic sanitation all require that considerable emphasis be placed on variables that are often taken for granted in fixed facilities, such as the availability of water and basic sanitary facilities.[140,141] Concerns about potable water, handwashing sources, and basic field sanitation may demand greater attention than environmental and occupational hazards such as ethylene oxide.

The first step in hazard abatement in a field MTF is to identify the hazards that are potential sources of danger. Identification of hazards in a field medical environment is a responsibility delegated by the commander to the staff. Execution of this task varies from unit to unit but usually requires someone with both access to all areas of the field medical unit and direct access to the commander for decision making. Preventive medicine personnel supporting field medical units should seek out the responsible staff member and work with him or her to evaluate and assess the hazards.

Appropriate and meaningful evaluations of field MTFs can prevent morbidity and mortality and preserve valuable human resources. Evaluations performed by preventive medicine personnel who are not knowledgeable or experienced in this area can waste the valuable time of healthcare providers and create confusion by issuing inappropriate recommendations.[142]

When conducting a survey, specific aspects of the field sanitation program should be reviewed, including:

- the water supply (water containers and trailers), to ensure that it is being monitored for potability, and that disinfection of the unit's water supply is being properly supervised;
- the unit's food operations, to confirm that basic food sanitation guidance is followed;
- unit waste-disposal operations, to ensure that acceptable policies are established and followed (in a field MTF, this element must include medical and chemical waste, in addition to wastewater and solid waste. The volume of solid waste and wastewater can be significant due to laundry, showers, bedpan washing, handwashing facilities for infection-control purposes, waste from X-ray units, and the use of disposable supplies);
- arthropod- and other animal-control measures, to ensure that they are appropriate and adequate;
- safety and health training programs, to evaluate their relevance to hazards found in the field medical environment;
- waste anesthetic gases, laboratory chemicals, and radiation, to ensure that the potential hazards are recognized and controlled; and
- autoclave operations, to ensure that sterilization procedures are adequate, and that explosive and burn hazards are controlled.

Simply talking with the personnel working in the field MTF can be extremely helpful. For example, these conversations may reveal valuable information about common health problems among the staff (eg, dermatitis or diarrhea); hazards that are not easily identified by short periods of observation (eg, malfunctioning switches on X-ray equipment); or supply shortages for critical items (eg, gloves or disinfectants).

After identifying the potential hazards, each must be analyzed to determine the probability that it will cause disease or injury, and the severity of the consequences should such a problem occur. Once the risks have been determined, the risk analysis must be presented to the decision maker (usually the commander), so that the risk is weighted against the benefits of performing a mission or task. It is the responsibility of the preventive medicine officer (who is a physician) or his or her representative to communicate to the decision maker (briefly and specifically) both the identified risk and appropriate recommendations on ways to reduce or eliminate the hazard.[141] The controls may be as substantial as substituting a less-toxic chemical and providing PPE or engineering controls. Or they may be as simple as implementing administrative controls such as writing an SOP, briefing personnel, and supervising adherence to the new procedures.

SUMMARY

Healthcare facilities are highly complex work environments with many varied occupational hazards. Employees are subjected to a surprising array of chemical, biological, physical, and psychosocial agents. The facility or unit commander, staff, supervisors, and workers themselves all have a responsibility to protect workers' health. Awareness of the hazards that are most likely to be encountered in the healthcare environment will, in most instances, enable the hazards to be identified and will generate the actions necessary to minimize, prevent, or eliminate the danger.

Although this chapter primarily addresses the hazards associated with fixed garrison healthcare facilities, many of the concepts presented also apply, in general, to field MTFs. Differences lie in the facts that (*a*) requirements for mobility may reduce the numbers of hazards, and (*b*) austere field conditions can lead to increased severity of exposures. Whether in a fixed or field healthcare facility, however, the effort to protect a worker's health reaps benefits beyond that afforded the individual. An ill or impaired healthcare worker can (directly or indirectly) adversely affect the morale and health of coworkers, patients, or both. Although such adverse effects are undesirable in any setting, in a field MTF supporting a combat operation, the impact could be catastrophic.

REFERENCES

1. Ramazzini _ *isease of Workers.* New York: Hafner Publishing; 1964. Originally published 1713.

2. Clever LH. The forgotten majority: Health hazards in hospital support service personnel. *Occup Health Nurs.* 1983;31:28–30.

3. US Department of Health and Human Services. *Guidelines for Protecting the Safety and Health of Health Care Workers.* Washington, DC: DHHS; 1988. DHHS (NIOSH) Publication 88-119.
4. Patterson WB, Craven DE, Schwartz DA, Nardell EA, Kasmer J, Nobel J. Occupational hazards to hospital personnel. *Ann Intern Med.* 1985;102:658–680.
5. Douglass BE. Health problems of hospital employees. *J Occup Med.* 1971;3:555–560.
6. DeRonde MM, Mason ME. Hospital employee health issues. *Occup Health Nurs.* 1984;32:44–49.
7. Sax NI, Lewis RJ. *Dangerous Properties of Industrial Materials.* 7th ed. New York: Van Nostrand Reinhold; 1989.
8. Sitting M. *Handbook of Toxic and Hazardous Chemicals and Carcinogens.* 2nd ed. Park Ridge, NJ: Noyes Publications; 1985.
9. Proctor NH, Hughes JP, Fischman ML. *Chemical Hazards of the Workplace.* 2nd ed. Philadelphia: Lippincott; 1988.
10. Plog BA, ed. *Fundamentals of Industrial Hygiene.* 3rd ed. Chicago: National Safety Council; 1988.
11. Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology.* 4th ed. New York: Pergamon Press; 1991.
12. Sax NI, Lewis RJ. *Hazardous Chemicals Desk Reference.* New York: Van Nostrand Reinhold; 1987.
13. US National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Waste Anesthetic Gases and Vapors.* Cincinnati, Oh: NIOSH; 1977. DHEW (NIOSH) Publication 77-140.
14. Dahlgren B, Olander, L, Ovrum P. Pollution of delivery ward air by nitrous oxide–methoxyflurane. *Am Ind Hyg Assoc J.* 1979;40:666–672.
15. National Institute for Occupational Safety and Health. *Control of Occupational Exposure to N_2O in the Dental Operatory.* Cincinnati, Oh: NIOSH; 1977. DHEW (NIOSH) Publication 77-171.
16. Manley SV, McDonell WN. Anesthetic pollution and disease. *J Am Vet Med Assoc.* 1980;176:519–524.
17. Ferstandig LL. Trace concentrations of anesthetic gases: A critical review of their disease potential. *Anesth Analg.* 1978;57:328–345.
18. Vessey MP. Epidemiological studies of the occupational hazards of anesthesia—A review. *Anesthesia.* 1978;33:430–438.
19. Cohen EN. *Anesthetic Exposure in the Workplace.* Littleton, Mass: PSG Publishing; 1980.
20. US National Institute for Occupational Safety and Health. *Effects of Trace Concentration of Anesthetic Gases on Behavioral Performance of Operating Room Personnel.* Cincinnati, Oh: NIOSH; 1976. DHEW (NIOSH) Publication 76-169.
21. Cook TL, Smith M, Starkweather JA, Winter PM, Eger EI. Behavioral effects of trace and subanesthetic halothane and nitrous oxide in man. *Anesthesiology.* 1978;49:419–424.
22. Bruce DL, Bach MJ. Psychological studies of human performance as affected by traces of enflurane and nitrous oxide. *Anesthesiology.* 1975;42:194–196.
23. Bruce DL, Bach MJ. Effects of trace anesthetic gases on behavioral performance of volunteers. *Br J Anesth.* 1976;48:871–875.
24. Hemminki K, Kyyrgronen P, Lindbohm M-L. Spontaneous abortions and malformations in the offspring of nurses [exposed] to anesthetic gases and other hazards. *J Epidemiol Comm Health.* 1985;39:141–147.
25. Babich H. Reproductive and carcinogenic health risks to hospital personnel from chemical exposures—A literature review. *J Environ Health.* 1985;48(2):52–56.

26. US Department of the Army. *Interim Guidelines for the Evaluation and Control of Occupational Exposure to Waste Anesthetic Gases.* Washington, DC: DA; 1982. Technical Bulletin MED 510.

27. Guirguis SS, Pelmear PL, Roy ML, Wong L. Health effects associated with exposure to anaesthetic gases in Ontario hospital personnel. *Br J of Industrial Med.* 1990;47:490–497.

28. Edling C. Anesthetic gases. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions.* Boca Raton, Fla: CRC Press; 1989:121–139.

29. Corbett TH. Cancer and congenital anomalies associated with anesthetics. *Ann NY Acad Sci.* 1976;271:58–66.

30. Cohen EN. Toxicity of inhalation anesthetic agents. *Br J Anesth.* 1978;50:665–670.

31. Bruce DL, Eide KA, Linde HW, Eckenhoff JE. Causes of death among anesthesiologists: A 20-year survey. *Anesthesiology.* 1968;29:565–569.

32. Corbett TH, Cornell RG, Lieding K, Endres JL. Incidence of cancer among Michigan nurse-anesthetists. *Anesthesiology.* 1973;38(3)260–263.

33. Harbison RD. Teratogens. In: Doull J, Klaassen CD, Amdur MO, eds. *Casarett & Duoll's Toxicology: The Basic Science of Poisons.* 2nd ed. New York: Macmillan; 1980:163–164.

34. Sorsa M, Hemminiki K, Vainio H. Occupational exposure to anticancer drugs potential and real hazards. *Mut Research.* 1985;154:135–149.

35. McDiarmid MA. Occupational exposure to pharmaceuticals; antineoplastics, anesthetic agents, sex steroid hormones. In: Paul M, ed. *Occupational and Environmental Reproductive Hazards: A Guide for Clinicians.* Baltimore, Md: Williams & Wilkins; 1993: 280–295.

36. Chabner BA. Second neoplasm—A complication of cancer chemotherapy. *N Engl J Med.* 1977;297:213–215.

37. Hoover R, Fraumeni JF Jr. Drug-induced cancer. *Cancer.* 1981;47:1071–1080.

38. Garrett MJ. Teratogenic effects of combination chemotherapy. *Ann Intern Med.* 1974;80:667.

39. Harris C. Immunosuppressive anticancer drugs in man—Their oncogenic potential. *Radiology.* 1975;114:163–167.

40. Vainio H. Inhalation anesthetics, anticancer drugs and sterilants as chemical hazards in hospitals. *Scand J Work Environ Health.* 1982;8:94–107.

41. Beeley L. Drugs in early pregnancy. *J Pharmacol Ther.* 1978;1:189–198.

42. Gililland J, Weinstein L. The effects of cancer chemotherapeutic agents on the developing fetus. *Obstet Gynecol Surg.* 1983;38:6–13.

43. Sieber SM, Adamson RH. Toxicity of antineoplastic agents in man: Chromosomal aberrations, antifertility effects, congenital malformations and carcinogenic potential. *Adv Cancer Res.* 1975;22:57–155.

44. Falck K, Gröhn P, Sorsa M, Vainio H, Heinonen E, Holsti LR. Mutagenicity in urine of nurses handling cytostatic drugs. *Lancet.* 1979;i:1250–1251.

45. Waksvik H, Kleep O, Brogger A. Chromosome analyses of nurses handling cytostatic agents. *Cancer Treat Rep.* 1981;65:607–610.

46. Nguyen TV, Theiss TC, Matney TS. Exposure of pharmacy personnel to mutagenic antineoplastic drugs. *Cancer Res.* 1982;42:4792–4796.

47. Hirst M, Tse S, Mills DG, Levin L, White DF. Occupational exposure to cyclophosphamide. *Lancet*. 1984;i:186–188

48. Venitt S, Crofton-Seligh C, Hunt J, et al. Monitoring exposure of nursing and pharmacy personnel to cytotoxic drugs: Urinary mutation assays and urinary platinum as markers of absorption. *Lancet*. 1984;i:74–77.

49. Gibson JF, Grampertz D, Hedworth-Whitty RB. Mutagenicity from nurses handling cytotoxic drugs. *Lancet*. 1984;i:100–101.

50. Selevan S, Lindbohm M-L, Hornung RW, et al. A study of occupational exposure to antineoplastic drugs and fetal loss in nurses. *N Engl J Med*. 1985;313:1173–1178.

51. Baker GL, Kahl LE, Zee BC, Stolzer BL, Agarwal AK, Medsger TA Jr. Malignancy following treatment of rheumatoid arthritis with cyclophosphamide. Long-term case-control followup study. *Am J Med*. 1987;83:1–9.

52. Hamza MH. Cancer complicating Behçet's disease treated with chlorambucil. *Am Rheum Dis*. 1986;45:789.

53. Moody DJ, Kagan J, Liao D, Ellison GW, Myers LW. Administration of monthly-pulse cyclophosphamide in multiple sclerosis patients. Effects of long-term treatment on immune parameters. *J Neuroimmunol*. 1987;14:161–173.

54. Reich SD, Bachur NR. Contact dermatitis associated with adriamycin and daunarubicin. *Can Chemo Rep*. 1975;59:677–678.

55. Ladik CF, Stoehr GP, Maurer MA. Precautionary measures in the preparation of antineoplastics. *Am J Hosp Pharm*. 1980;37:1184–1185.

56. Rogers B, Emmett EA. Handling antineoplastic agents: Urine mutagenicity in nurses. *Image J Nurs Sch*. 1987;19:108–113.

57. McDiarmid MA, Egan T. Acute occupational exposure to antineoplastic agents. *J Occup Med*. 1988;30:984–987.

58. US Occupational Safety and Health Administration. *Work Practice Guidelines for Personnel Dealing with Cytotoxic (Antineoplastic) Drugs*. Washington, DC: US Department of Labor, OSHA; 1986. OSHA Instruction Publication 8-1.1, App A.

59. *Occupational Safety and Health Act of 1970*. Pub L No. 91-596 § 5(a)(b).S.2193.

60. McDiarmid MA. Medical surveillance for antineoplastic drug handlers. *Am J Hosp Pharm*. 1990;47:1061–1066.

61. Noll S, Caldwell DJ. *Guidelines for the Handling, Administration, and Disposal of Cytotoxic Drugs*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1987. Technical Guide 149. To be published as US Army Technical Bulletin MED 515.

62. American Society of Hospital Pharmacy. Technical Assistance Bulletin on handling cytotoxic and hazardous drugs. *Am J Hosp Pharm*. 1990;47:1033–1049.

63. US Centers for Disease Control. Public Health Service statement on management of occupational exposure to human immunodeficiency virus, including consideration regarding zidovudine post exposure use. *MMWR*. 1990;39 (RR-1):1-14.

64. McDiarmid MA, Gurley HT, Arrington D. Pharmaceuticals as hospital hazards: Managing the risks. *J Occup Med*. 1991;33:155–158.

65. US National Institute for Occupational Safety and Health. *Use of Ethylene Oxide as a Sterilant in Medical Facilities*. Cincinnati, Oh: NIOSH; 1977. DHEW (NIOSH) Publication 77-200, Special Occupational Hazard Review with Control Recommendations.

66. US National Institute for Occupational Safety and Health. *Ethylene Oxide (EtO)*. Cincinnati, Oh: NIOSH; 1981. Current Intelligence Bulletin 35, DHHS (NIOSH) Publication 81-130.

67. US National Institute for Occupational Safety and Health. *Ethylene Oxide Sterilizers in Health Care Facilities, Engineering Controls and Work Practices.* Cincinnati, Oh: NIOSH; 1989. Current Intelligence Bulletin 52.

68. Landrigan PJ, Meinhardt TJ, Gordon J, et al. Ethylene oxide: An overview of toxicologic and epidemiological research. *Am J Ind Med.* 1984;6:103–115.

69. 29 CFR, Parts 1900–1910.

70. Caldwell DJ. *Evaluation and Control of Occupational Exposure to Ethylene Oxide in Health Care Facilities.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1985. Technical Guide 143. To be published as US Army Technical Bulletin MED 512.

71. US National Institute for Occupational Safety and Health. *Control Technology for Ethylene Oxide Sterilization in Hospitals.* Cincinnati, Oh: NIOSH; 1989. DHHS (NIOSH) Publication 89-120.

72. Association for the Advancement of Medical Instrumentation. *Good Hospital Practice: Ethylene Oxide Gas—Ventilation Recommendations and Safe Use.* Arlington, Va: AAMI; 1988.

73. Danielson NE. *Ethylene Oxide Use in Hospitals.* 2nd ed. Chicago: American Hospital Publishing; 1986.

74. Coldiron VR, Ward JB, Trieff NM, Janssen HE, Smith JH. Occupational exposure to formaldehyde in a medical center autopsy service. *J Occup Med.* 1983;25:544–548.

75. Clark RP. Formaldehyde in pathology departments. *J Clin Pathol.* 1983;36:839–846.

76. Breysse PA. Staff and patient exposure during hemodialysis treatment utilizing equipment disinfected with formaldehyde. In: American Conference of Governmental Industrial Hygienists. *Occupational Hazards to Health Care Workers.* Cincinnati, Oh: ACGIH; 1986; 160–167.

77. Smith KA, Williams PL, Middendorf PJ, Zabraysek L. Kidney dialysis: Ambient formaldehyde levels. *Am Ind Hyg Assoc J.* 1984;45:40–50.

78. US National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Formaldehyde.* Cincinnati, Oh: NIOSH; 1976. DHEW (NIOSH) Publication 77-126.

79. US National Institute for Occupational Safety and Health. *Formaldehyde: Evidence of Carcinogenicity.* Cincinnati, Oh: NIOSH; 1981. Current Intelligence Bulletin 34, DHHS (NIOSH) Publication 81-111.

80. Halperin WE, Goodman M, Stayner L, Elliott LJ, Keenlyside RA, Laudrigan PJ. Nasal cancer in workers exposed to formaldehyde. *JAMA.* 1983;249:510–512.

81. Jenkins MF. Reducing occupational exposure to formaldehyde in the dialysis laboratory. *Dialysis & Transplantation.* 1980;9(11):1049–1050.

82. Goldman M, Stein AA. Formaldehyde—Health effects, regulation and environmental control. *Journal of Histotechnology.* 1981;4(1):13–16.

83. Notani-Sharma P. Little known mercury hazards. *Hospitals.* 1980;54:76–78.

84. Porter DD. Mercury pollution by tissue fixatives. *Arch Pathol.* 1972;94:279.

85. Gehardsson L, Brune DK. Mercury in dentistry. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions.* Boca Raton, Fla: CRC Press; 1989: 307–332.

86. US National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard: Occupational Exposure to Inorganic Mercury.* Cincinnati, Oh: NIOSH; 1973. DHEW (NIOSH) Publication 73-1009.

87. Council on Dental Materials and Devices. Recommendations in mercury hygiene. *J Am Dent Assoc.* 1976;92:1217.

88. Wilson J. Reduction of mercury vapor in a dental surgery. *Lancet.* 1978;1:200–201.

89. Mattia MA. Hazards in the hospital environment—Anesthetic gases and methylmethacrylate. *Am J Nurs.* 1983;83:73–77.

90. Widstrom J. Methyl methacrylate. In: Brune DK, Edling C, eds, *Occupational Hazards in the Health Professions.* Boca Raton, Fla: CRC Press; 1989: 141–145.

91. Hyderally H, Miller F. Hypotension and cardiac arrest during prosthetic hip surgery with acrylic bone cement. *Orthop Rev.* 1976;5:55–61.

92. US National Institute for Occupational Safety and Health. *A Study of Methyl Methacrylate Exposure and Employee Health.* Cincinnati, Oh: NIOSH; 1976. DHEW (NIOSH) Publication 77-119.

93. Singh AR, Lawrence WH, Autian J. Embryonic-fetal toxicity and teratogenic effects of a group of methacrylate esters in rats. *J Dent Res.* 1972;51:1632–1638.

94. Poss R, Thilly WG, Kaden, DA. Methylmethacrylate is a mutagen for *Salmonella typhimurium. J Bone Joint Surg.* 1979;61:1203–1207.

95. Lingaas E, Kalager T. Microbiological hazards. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions.* Boca Raton, Fla: CRC Press; 1989: 71–119.

96. Lewy R, Christenson W. Committee Report: Guidelines for employee health services in health care institutions. *J Occup Med.* 1986;28:518–523.

97. Ellen RP. The dental practitioner and systemic infection of oral origin. *Int Dent J.* 1978;28:295–308.

98. Mosley JW, White E. Viral hepatitis as an occupational hazard of dentists. *J Am Dent Assoc.* 1975;90:992–997.

99. Pike RM. Laboratory-associated infections: Incidence, fatalities, causes, and prevention. *Ann Rev Microbiol.* 1979;33:41–66.

100. American Hospital Association. *Infection Control in the Hospital.* 4th ed. Chicago: AHA: 1979.

101. Dixon RE, Brachman PS, Bennett JV, eds. *Isolation Techniques for Use in Hospitals.* Atlanta, Ga: US Public Health Service, Centers for Disease Control; 1978. US DHEW (CDC) Publication 78-8314.

102. Garner JS, Favero MS. *Guideline for Handwashing and Hospital Environmental Control.* Atlanta, Ga: US Public Health Service, Centers for Disease Control; 1985. US DHHS (CDC) Publication 99-1117.

103. Richardson JH, Barkley WE. *Biosafety in Microbiological and Biomedical Laboratories.* Washington, DC: US Public Health Service, Centers for Disease Control, and National Institutes of Health; 1986. US DHHS (CDC) Publication 86-8395.

104. World Health Organization. *Laboratory Biosafety Manual.* Geneva, Switzerland: World Health Organization; 1983.

105. US Centers for Disease Control. Recommended infection control practices for dentistry. *MMWR.* 1986;35:237–242.

106. 29 CFR. Part 1910.

107. US Centers for Disease Control. Acquired Immunodeficiency Syndrome (AIDS): Precautions for clinical and laboratory staffs. *MMWR.* 1982;31:577–580.

108. US Centers for Disease Control. Acquired Immunodeficiency Syndrome (AIDS): Precautions for health-care workers and allied professionals. *MMWR.* 1983;32:450–451.

109. US Centers for Disease Control. Recommendations for preventing transmission of infection with Human T-Lymphotrophic Virus type III/lymphadenopathy-associated virus in the workplace. *MMWR*. 1985;34:681–686,691–695.

110. US National Institute for Occupational Safety and Health. *Guidelines for Prevention of Transmission of Human Immunodeficiency Virus and Hepatitis B Virus to Health-Care and Public-Safety Workers*. Cincinnati, Oh: NIOSH; 1989. DHHS (NIOSH) Publication 89-107.

111. US Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. *MMWR*. 1987;36(2S):1–48.

112. US Centers for Disease Control. Update: Universal precautions for prevention of transmission of human immunodeficiency virus, Hepatitis B Virus, and other bloodborne pathogens in health-care settings. *MMWR*. 1988;37:377–382, 387–388.

113. US Centers for Disease Control. Agent summary statement for human immunodeficiency virus and report on laboratory-acquired infection with human immunodeficiency virus. *MMWR*. 1988;37(4S):1–22.

114. World Health Organization. *AIDS Prevention and Control*. Oxford: Pergamon Press; 1988.

115. Van Wagoner R, Maguire N. A study of hearing loss among employees in a large urban hospital. *Can J Public Health*. 1977;68:511–512.

116. Falk SA, Woods NF. Hospital noise—Levels and potential health hazards. *N Engl J Med*. 1973;289:774–781.

117. Shapiro RA, Berland T. Noise in the operating room. *N Engl J Med*. 1982;287:1236–1238.

118. Lervik A. Hazards in the dental environment—Curing lights and high-speed hand pieces. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions*. Boca Raton, Fla: CRC Press; 1989: 291–297.

119. Seidlitz PR. Excessive noise levels detrimental to patients, staff. *Hospital Progress*. 1981;62:54–56,64.

120. Turner AG, King CH, Craddock JG. Measuring and reducing noise. *Hospitals*. 1975;49:85–86, 88–90.

121. Lewy R. Prevention strategies in hospital occupational medicine. *J Occup Med*. 1981;23(2):109–111.

122. World Health Organization. *Occupational Hazards in Hospitals*. Geneva, Switzerland: World Health Organization; 1981. EURO Reports and Studies 80.

123. Lloyd P, Tarling C, Wright B, Troup JDG. *The Handling of Patients: A Guide For Nurses*. 2nd ed. London: The Back Pain Association and the Royal College of Nursing; 1987.

124. Torma-Krajewski J. Analysis of lifting tasks in the health care industry. In: American Conference of Governmental Industrial Hygienists. *Occupational Hazards to Health Care Workers*. Cincinnati, Oh: ACGIH; 1986: 51–68.

125. Hagberg M, Hagberg C. Risks and prevention of musculoskeletal disorders among dentists. In: Brune DK, Edling C, eds. *Occupational Hazards in the Health Professions*. Boca Raton, Fla: CRC Press; 1989: 323–332.

126. US National Institute for Occupational Safety and Health. *Work Practices Guide for Manual Lifting*. Cincinnati, Oh: NIOSH; 1981. DHHS (NIOSH) Publication 81-122.

127. Barnes RJ. Prevention, principles, and techniques in lifting, transporting, and body mechanics. In: Stanley FE, ed. *CRC Handbook of Hospital Safety*. Boca Raton, Fla: CRC Press; 1981: 345–356.

128. Bigos SJ, Battie MC. Prevention of back disability. In: American Conference of Governmental Industrial Hygienists. *Occupational Hazards to Health Care Workers*. Cincinnati, Oh: ACGIH; 1986: 69–79.

129. McCue JD. The effects of stress on physicians and their medical practice. *N Engl J Med.* 1982;306:458–463.

130. Scully R. Stress in the nurse. *Am J Nurs.* 1980;8:912–914.

131. Huckabay L, Jagla B. Nurses' stress factors in the intensive care unit. *J Nurs Adm.* 1979;9:21–26.

132. Griffin P, Klun CL. Laboratory stress: What causes it? *Am J Med Technol.* 1980;46:490–494.

133. Hoiberg A. Occupational stress and illness incidence. *J Occup Med.* 1982;24:445–451.

134. Shubin S. Burnout: The professional hazard you face in nursing. *Nursing.* 1978;8:22–27.

135. Patrick PK. Burnout: Job hazard for health workers. *Hospitals.* 1979;53:87–90.

136. Koran LM, Moos B, Zasslow M. Changing hospital work environments: An example of a burn unit. *Gen Hosp Psychiatry.* 1983;5:7–13.

137. Scott AJ, Ladou J. Shift work: Effects on sleep and health with recommendations for medical surveillance and screening. In: Scott AJ, ed. *Occupational Medicine: State of the Art Reviews,* Vol 5, No 2. Philadelphia: Hanley and Belfus; 1990; 273–299.

138. Czisler CA, Moore-Ede MC, Coleman RM. Rotating shift work schedules that disrupt sleep are improved by applying circadian principles. *Science.* 1982;217:460–463.

139. Murphy TJ. Fixed vs. rapid rotation shift work. *J Occup Med.* 1979;21:319–322.

140. US Department of the Army. *Health Service Support in a Theater of Operations.* Washington, DC: DA; 1991. Field Manual 8-10.

141. US Department of the Army. *Field Hygiene and Sanitation.* Washington, DC: DA; 1988. Field Manual 21-10.

142. US Department of the Army. *Planning for Health Service Support.* Washington, DC: DA; 1985. Field Manual 8-55.

EXCERPT FROM WALTER REED ARMY MEDICAL CENTER'S EXPOSURE CONTROL PLAN

The following excerpt is from the *Exposure Control Plan* that was adopted at Walter Reed Army Medical Center on 4 May 1991. Students may find this helpful because it illustrates the level of compliance required by Title 29, Code of Federal Regulations (CFR) 1910.1030. The appendices mentioned, which are part of the original document, are not included in this excerpt.

10. METHODS OF COMPLIANCE.

a. Universal Precautions.

Universal precautions were implemented at WRAMC [Walter Reed Army Medical Center] in 1987. Universal precautions require all employees to treat blood, body fluids, and tissues of all patients as potentially infective with HBV [hepatitis B virus], HIV [human immunodeficiency virus], and other blood-borne pathogens. The precautions are intended to prevent parenteral, mucous membrane, and skin exposure to blood and body fluids. Universal precautions are outlined in Section 4, Isolation Procedures and Universal Precautions, of the Infection Control Policy and Procedure Guide (Appendix B).

b. Engineering and Work Practice Controls.

(1). Engineering and work practice controls will be implemented as the primary means of eliminating or minimizing employee exposure to blood and body fluids. When occupational exposure remains after institution of engineering and work practice controls, personal protective equipment (PPE) will be used.

(2). Engineering controls are controls that either isolate the employee from the hazard or remove the hazard from the workplace. Examples include sharps disposal containers, bio-safety cabinets, splash guards, and needleless IV systems.

(3). Work practice controls are those that reduce the likelihood of exposure by altering the manner in which a task is performed. An example of a required work practice control is prohibiting recapping of needles with a two-handed technique.

(4). All engineering and work practice controls in this section that are not currently in use will be implemented NLT [no later than] 6 July 1992.

(5). Handwashing.

(a). Handwashing facilities will be readily accessible to employees. Approved alcohol based waterless hand cleansers and paper towels must be used in all areas where sinks are not available. Handwashing technique is described in Section 5.1, Handwashing and Use of Gloves, in the Infection Control Policy and Procedure Guide (Appendix C).

(b). Employees will wash their hands immediately or as soon as feasible after removal of gloves or other personal protective equipment.

(c). Employees using a waterless hand cleaner must wash hands with soap and running water as soon as feasible.

(d). Employees will wash hands and any other skin with soap and water, or flush mucous membranes with water, immediately or as soon as feasible following contact of such body areas with blood or other potentially infectious materials.

(e). Hand cream application is permitted in a contaminated area if the hands are thoroughly washed immediately prior to application. Hand creams must be from small, individual, nonrefillable containers and not shared between individuals.

(6). Prevention of Sharps Injuries.

(a). Contaminated needles and other contaminated sharps will not be bent, sheared, or broken.

(b). Contaminated needles will not be recapped or removed from syringes unless it can be demonstrated that there is no feasible alternative or the action is required by specific medical procedure. The two exceptions where recapping the needle is permitted are: Performing a blood gas and administering incremental doses of a medication such as an anesthetic to the same patient. Removing the needle from a vacutainer sleeve is permitted. Recapping with the traditional two-handed method is prohibited in these situations. Recapping will be performed with the one-hand scoop method (the hand holding the sharp is used to scoop up the cap from a flat surface) or by using forceps to replace the cap. Removing the needle from a vacutainer sleeve will be done using the special area on the sharps container where the needle is inserted and the vacutainer is used to unscrew the needle and the needle drops into the sharps container. NOTE: Other exceptions must be submitted to the Infection Control Committee for approval. Applications must include a justification for the need to recap or remove a needle.

(c). Immediately, or as soon as feasible after use, contaminated needles or other sharps will be placed in leakproof, puncture resistant sharps containers which are located in patient rooms and in other areas as close to where sharps are used as feasible. Sharps containers in patient rooms are in a wall cabinet. This cabinet and the disposable sharps liners are to be labelled with a biohazard symbol IAW [in accordance with] the Labels and Signs section of this plan. Chemotherapy sharps containers will be labelled IAW the Labels and Signs section of this plan. All other sharps containers will be red in color.

(d). A red sharps container will be available in the laundry.

(e). Disposable sharps containers will be removed and replaced with a new one when ¾ full. They will be closed off by securely locking the closure mechanism, tagged with a burn label, and placed in the trash area for pick-up by housekeeping. If a sharps container is found to be leaking, it must be placed in a larger sharps container that is labelled and sealed. The OIC [officer in charge] in each work area is responsible for insuring that sharps containers are replaced when ¾ full and are not overfilled.

(f). Contaminated reusable sharps will be placed in containers until properly processed. The containers are puncture-resistant, leakproof on the sides and bottom, and labelled with a biohazard label IAW the Labels and Signs section of this plan. The containers need not be closable. Employees will not reach by hand into these containers. Employees will not reach into a water-filled sink or pan to retrieve contaminated instruments. Instead a perforated tray can be used or the instruments can be retrieved with forceps. A container for reusable sharps will also be available in the laundry.

(g). Reusable sharps containers will be cleaned with soap and water and then disinfected with a 1:10 solution of bleach after each use.

(h). In psychiatric units where there are no in-room sharps containers, needle users have two options: Carry a small sharps container to the room to immediately discard the sharp or use a self-sheathing needle-syringe unit.

(i). The Baxter needleless IV [intravenous] system will be used for access into IV lines. Stopcocks may also be used.

(7). Bio-safety cabinets and splash guards are used in laboratories to minimize splashing, spraying, splattering, and generation of droplets.

(8). Engineering controls will be examined and maintained or replaced on a regular schedule to insure their effectiveness, that they have not been removed or broken, that ventilation systems are functioning properly, and that filters are replaced frequently enough. The OIC in each work area will establish a written inspection and routine maintenance schedule for the engineering controls in that area.

(9). All specimens of blood, body fluids, and tissues will be handled using Universal Precautions and will be transported in sealed plastic bags. Specimen containers will be securely closed before placing in the bag. If outside contamination of the bag or primary container occurs, the bag or primary container shall be placed within a second container which prevents leakage during handling, processing, storage, transport, or shipping. If the specimen could puncture the primary container, the primary container will be placed within a second container which is puncture resistant. Containers used for transporting or shipping specimens outside the facility will be labelled with a biohazard label IAW the Labels and Signs section of this plan.

(10). Equipment which may be contaminated with blood or body fluids will be examined prior to servicing or shipping and will be decontaminated as necessary. If decontamination of the equipment is not possible (personnel do not have training to take apart technologically advanced equipment or equipment design prohibits cleaning), a readily observable label will be attached to the equipment stating which portion may be contaminated and this information will be conveyed to all affected employees, the servicing representative, and/or the manufacturer, as appropriate, prior to handling, servicing, or shipping so that appropriate precautions will be taken. See section on Labels and Signs for required label characteristics. See section on Housekeeping for instructions on decontamination. Biomedical maintenance personnel will be instructed in precautions to practice during decontamination of equipment.

(11). All procedures involving blood or other body fluids shall be performed in such a manner as to minimize splashing, spraying, spattering, and generation of droplets of these substances.

(12). Mouth pipetting/suctioning of blood or other body fluids is prohibited.

(13). Eating, drinking, smoking, applying cosmetics or lip balm, and handling contact lenses are prohibited in all work areas where there is a reasonable likelihood of occupational exposure. Eating or drinking are permitted only in designated areas separate from contaminated areas. Employees must remove any contaminated clothing or protective barriers prior to entering the clean area.

(14). Food and drink shall not be placed in refrigerators, freezers, shelves, cabinets, or on countertops or benchtops where blood or other potentially infectious materials are present or where specimens have been placed.

(15). All employees will be trained by their supervisor in the use of any engineering control before they are required to use it.

(16). Employees who have exudative lesions or weeping dermatitis will not perform or assist in invasive procedures or other direct patient care activities or handle equipment used for patient care.

(17). The Hospital Product Review Subcommittee will review the feasibility of testing engineering controls as new ones enter the market.

c. Personal Protective Equipment (PPE).

(1). Supervisors will insure that personal protective equipment in the appropriate sizes is readily available to employees in each work area that requires it. Supervisors will insure that employees are trained in its use and use it as required. PPE not currently in use must be implemented NLT 6 Jul 92.

(2). PPE is provided at no cost to the employee and includes, but is not limited to, gloves, gowns, laboratory coats, face shields, masks, eye protection, and mouthpieces, resuscitation bags, pocket masks, or other ventilation devices.

(3). PPE is considered appropriate only if it does not permit blood or other potentially infectious materials to pass through or reach the employee's work clothes, street clothes, undergarments, skin, eyes, mouth, or other mucous membranes under normal conditions of use and for the duration of time which the PPE will be used.

(4). Supervisors will insure that employees use appropriate personal protective equipment unless the supervisor can show that the employee temporarily and briefly declined to use PPE when, under rare and extraordinary circumstances, it was the employee's professional judgment that in the specific instance its use would have prevented the delivery of health care or public safety services or would have posed an increased hazard to the safety of the worker or a coworker. When an employee makes this judgment, the supervisor will investigate and document the circumstances. The documentation will be forwarded to the Safety Manager NLT the next duty day. The supervisor and the Safety Manager will determine whether changes need to be instituted to prevent such occurrences in the future. A decision not to use protective barriers will not be applied to a particular work area or a recurring task. Neither interference with ease of performance of a procedure nor improper fit of equipment are acceptable reasons to not use PPE.

(5). Supervisors will insure that PPE in the appropriate sizes is readily accessible at the worksite or is issued to employees. Hypoallergenic gloves, glove liners, powderless gloves, or other similar alternatives will be readily accessible to those employees who are allergic to gloves normally provided.

(6). PPE will be cleaned, laundered, or disposed of by WRAMC at no cost to personnel. Laboratory coats that are used as PPE will be laundered by the hospital and not taken home for laundering. Personal clothing contaminated by blood or body fluids will be laundered by the hospital laundry at no cost to the employee. Supervisors will contact Linen Services to make arrangements for laundering personal clothing when contaminated.

(7). Supervisors will insure repair or replacement of all reusable equipment as needed to maintain effectiveness.

(8). If PPE items are penetrated by blood or other potentially infectious materials, the item will be removed immediately or as soon as is feasible.

(9). All PPE will be removed prior to leaving the work area. PPE will not be worn into designated break areas.

(10). Gloves.

(a). Latex gloves will be worn when it can be reasonably anticipated that the employee may have hand contact with blood, other body fluids, mucous membranes, and non-intact skin; when performing vascular access procedures; and when handling or touching contaminated items or surfaces.

(b). Examples of tasks where gloves will be worn are: Phlebotomy, performing finger or heel sticks; during instrumental examination of the oropharynx, gastrointestinal tract, and genitourinary tract; during invasive procedures; during all cleaning of body fluids and decontaminating procedures; handling and processing blood and body fluid and tissue specimens; when examining abraded or non-intact skin or patients with active bleeding; when emptying drains and Foley catheter bags; and when rendering emergency medical assistance to individuals with traumatic injury.

(c). Single use disposable latex gloves shall be replaced as soon as practical when contaminated or as soon as feasible if they are torn, punctured, or when their ability to function as a barrier is compromised.

(d). Gloves will be changed and hands washed between patients or during the care of a single patient when moving from a contaminated to a clean body site or from one contaminated site to another contaminated site. Phlebotomists working in the outpatient phlebotomy room may wear gloves with several patients until they become visibly contaminated. This exception does not apply to phlebotomists drawing blood on inpatients or to any other personnel who draw blood.

(e). Hands will be washed as soon as possible after removal of gloves.

(f). Gloves should be discarded in the appropriate container.

(g). Disposable gloves such as surgical or examination gloves will not be washed or decontaminated for re-use.

(h). Sterile surgical gloves should be used for procedures involving contact with normally sterile areas of the body.

(i). Latex examination gloves should be used for procedures involving contact with mucous membranes, unless otherwise indicated, and for other patient care or diagnostic procedures that do not require the use of sterile gloves.

(j) Double gloving may be used for invasive surgical procedures where prolonged contact with blood may be expected.

(k). Used gloves will not be used to touch telephones, computers, keyboards, charts, elevator buttons, or other uncontaminated surfaces.

(l). Non-patient care services should use gloves appropriate to their type of work. Heavy duty utility gloves may be preferable for housekeeping personnel. These gloves may be washed and disinfected for reuse if the integrity of the glove is not compromised. If gloves are cracked, peeling, torn, or punctured, they are discarded.

(11). Masks, Eye Protection, and Face Shields.

(a). In general, whenever a mask is required, eye protection is required.

(b). Masks in combination with eye protection devices, such as goggles or glasses with solid side shields, or chin-length face shields shall be worn whenever splashes, spray, splatter, or droplets of blood or other body fluids may be generated and eye, nose, or mouth contamination can be reasonably anticipated.

(c). Prescription glasses may be used as protective eyewear as long as they are equipped with solid side shields that are permanently affixed or of the "add-on" type.

(d). Procedures requiring masks and eye protection include endotracheal intubation, bronchoscopy, GI endoscopy, dental procedures that splatter, autopsy, and certain surgical and other invasive procedures.

(e). During microsurgery, when it is not reasonably anticipated that there would be any splattering, it would not constitute a violation for the surgeon, while observing surgery through a microscope, not to wear other eye protection.

(f). Masks should be used once and discarded in the appropriate waste receptacle.

(g). Masks should not be worn around the neck or on top of the head.

(h). Masks must cover both the nose and mouth with no gaping at the sides.

(i). Reusable goggles and face shields will be washed with an approved detergent and water and disinfected with a 1:10 solution of bleach after each use.

(12). Gowns

(a). Gowns, aprons, laboratory coats, or clinic jackets must be worn where there is the potential for reasonably anticipated soiling of clothing with blood or other potentially infectious materials.

(b). A cover garment is appropriate only if it does not permit blood or other body fluids to pass through to or reach the employee's work clothes, street clothes, or undergarments.

(c). Gowns impervious to fluid will be worn for surgical procedures and autopsies.

(d). A long-sleeved cover will be worn when arms are likely to become contaminated.

(e). Scrubs are not considered PPE and will be covered by appropriate gowns, aprons, or laboratory coats when splashes to skin or clothing are anticipated.

(f). A gown which is frequently ripped or falls apart under normal use would not be considered appropriate PPE.

(g). A cloth gown or disposable cover gown will not generally prevent gross liquid contamination from soaking through to the skin, but they are adequate protection for common bedside patient care procedures in situations when gross liquid/blood contamination is not likely.

(h). Examples of activities requiring gowns or aprons are: changing the bed of an incontinent patient, lifting or moving a patient with draining wounds, diagnostic and therapeutic procedures that may cause splattering or aerosolization, and autopsy.

(i). Gowns and aprons should be worn only once and then removed and placed in the appropriate receptacle. These items will not be worn out of the work area.

(j). Cloth gowns and lab coats will be placed in the hospital laundry containers.

(k). Paper or plastic gowns/aprons will be discarded in the appropriate waste receptacle.

(13). Surgical caps or hoods and/or shoe covers or boots will be worn during surgical procedures, autopsies, or other situations when gross contamination can be reasonably anticipated. Shoe covers must be removed prior to leaving the work area to limit migration of contamination via shoes into other areas.

(14). Seal-easy masks are available in each patient room and in other areas of the hospital for use during mouth-to-mouth resuscitation to prevent direct contact between the employee and the patient. Ambu-bags are at each bedside in critical care areas and on each crash cart at the hospital. The seal-easy masks are disposable and will be discarded after each use. Ambu-bags that are reusable will be bagged and sent to CMS [central material supply] for high-level disinfection or sterilization.

d. Housekeeping.

(1). Supervisors will insure that the work area is maintained in a clean and sanitary condition. The provisions of this section not currently implemented will be in use NLT 6 Jul 92.

(2). All equipment and environmental and working surfaces will be properly cleaned and disinfected after contact with blood or other potentially infectious materials and on a regular schedule with an appropriate disinfectant.

(3). Contaminated work surfaces will be decontaminated with an appropriate disinfectant after completion of procedures; immediately or as soon as feasible when surfaces are overtly contaminated or after any spill of blood or other body fluids; and at the end of the work shift if the surface may have become contaminated since the last cleaning. A phenolic disinfectant approved by the Infection Control Committee and used according to the manufacturer's directions will be used in the laboratory, the Operating Room, and Delivery Room. The Dialysis Unit uses bleach.

(4). Blood spills will be cleaned up with an approved detergent and water and the area disinfected with a 1:10 solution of household bleach or an approved phenolic disinfectant.

(5). Protective coverings such as plastic wrap, aluminum foil, or imperviously-backed absorbent paper may be used to cover equipment and environmental surfaces. These shall be removed and replaced as soon as feasible when they become overtly contaminated and between patients.

(6). All bins, pails, cans, and similar receptacles intended for reuse which have a potential for becoming contaminated with blood or other body fluids shall be inspected, cleaned with an approved detergent and water, and disinfected with a phenolic disinfectant or a 1:10 solution of bleach immediately or as soon as possible after visible contamination. Routine cleaning of these items will be done monthly.

(7). Reusable items contaminated with blood or other body fluids shall be washed with an approved detergent and water. If an item is to be returned to the CMS, it will be placed into a plastic bag for transport (the bag must be labelled IAW the Labels and Signs section of this plan). If the item remains in the area, it will be wiped down with a phenolic disinfectant or a 1:10 solution of bleach.

(8). Broken glassware which may be contaminated will not be picked up directly with the hands. It will be cleaned up using mechanical means, such as a brush and dust pan, tongs, or forceps.

(9). Routine Cleaning Schedule:

LOCATION	FREQUENCY	CLEANERS AND DISINFECTANTS USED
Patient Room	Daily	Approved quaternary ammonium disinfectant
Patient Bathroom	Daily	Approved quaternary ammonium disinfectant
Exam Room	Daily	Approved quaternary ammonium disinfectant
Procedure Room	Between procedures	Approved quaternary ammonium disinfectant
Operating Room	Between cases	Approved phenolic disinfectant
Delivery Room	Between deliveries	Approved phenolic disinfectant
Dialysis	Between patients	Approved detergent and 1:10 bleach solution
Laboratory	When contaminated and/or daily	Approved phenolic disinfectant

e. Regulated Medical Waste.

(1). Regulated medical waste (RMW), including sharps, will be disposed of IAW WRAMC Reg 40-92 and section 5.11, Collection and Handling of Regulated Medical Waste, of the Infection Control Policy and Procedure Guide (Appendix D). Saliva-soaked gauze and cotton rolls in dental clinics and items caked with dried blood and capable of releasing the blood during normal handling procedures will be managed as RMW NLT 6 Jul 92.

(2). When moving containers of contaminated sharps from the area of use, the containers will be closed immediately prior to removal or replacement to prevent spillage or protrusion of contents during handling, storage, transport, or shipping. If leakage is possible, the container will be placed in a secondary container that is closable, constructed to contain all contents and prevent leakage during handling, storage, transport, or shipping and labelled or red in color IAW the Labels and Signs section of this plan.

(3). Other regulated medical waste is placed in plastic bags that line cardboard boxes which are labelled IAW the Labels and Signs section of this plan. When ¾ full, the bags are closed, the box is sealed, and labelled with a burn tag on the side of the box. If outside contamination of the box occurs, the waste is placed in a second bag inside another labelled box.

f. Laundry.

(1). All soiled linen will be handled using universal precautions. Personnel handling linen soiled with blood or other body fluids will use appropriate PPE as described in the Personal Protective Equipment Section. These practices are currently in place.

(2). Soiled linen will be collected in white or green laundry bags at the location where it was used. If linen is excessively wet, place it in a clear or black plastic bag before putting it in the laundry bag.

(3). Soiled linen will not be sorted or rinsed in patient care areas.

11. HEPATITIS B VACCINATION, POST-EXPOSURE EVALUATION AND FOLLOW UP.

a. Vaccination Program.

To protect employees as much as possible from the possibility of Hepatitis B infection, WRAMC implemented a vaccination program. This program is available, at no cost, to all employees who have occupational exposure to blood-borne pathogens. The vaccination program consists of a series of three inoculations over a 6-month period. As part of their blood-borne-pathogens training, our employees have received information regarding hepatitis vaccination, including its safety and effectiveness. Occupational Medicine in conjunction with Allergy/Immunology is responsible for setting up and operating our vaccination program. Vaccinations are performed under the supervision of a licensed physician or other healthcare professional. To ensure that all employees are aware of our vaccination program, it is thoroughly discussed in our blood-borne pathogens training. A record of the vaccination status of all employees will be maintained by the Occupational Medicine Program. Any exposed civilian declining to be vaccinated will sign the following declination statement (Appendix E). This statement will be maintained in the employee's medical record.

b. Post-exposure evaluation and follow up.

(1). Employees involved in an incident where exposure to blood-borne pathogens may have occurred will immediately report to the Emergency Room.

(2). The supervisor will immediately investigate the circumstances surrounding the exposure incident while making sure that our employees receive medical consultation and treatment (if required) as expeditiously as possible.

(3). Treatment will be in accordance with the Emergency Room's Needle Stick Protocol (Appendix F).

(4). The Safety Office will investigate every exposure incident that occurs in our facility. This investigation is initiated within 24 hours after the incident occurs and involves gathering the following information:

- When the incident occurred. Date and time.
- Where the incident occurred. Location within the facility.
- What potentially infectious materials were involved in the incident. Type of material (blood, amniotic fluid, etc.).
- Source of the material.
- Under what circumstances the incident occurred. Type of work being performed.
- How the incident was caused. Accident/Unusual circumstances (such as equipment malfunction, power outage, etc.).
- Personal protective equipment being used at the time of the incident.
- Employee decontamination/cleanup/notifications made.

After this information is gathered it is evaluated, a written summary of the incident and its causes is prepared, and recommendations are made for avoiding similar incidents in the future (Appendix G).

(5). In order to make sure that our employees receive the best and most timely treatment if an exposure to blood-borne pathogens should occur, our facility has set up a comprehensive post-exposure evaluation and follow-up process (Appendix H). We verify that all the steps in the process have been taken correctly. This process was implemented on or before July 6, 1992, and is overseen by the Occupational Safety and Health Committee.

c. Information provided to the healthcare professional.

Civilian employees have the right to choose a civilian physician for treatment. WRAMC, however, has the right to evaluate employees who are injured on the job. Therefore, personnel who suspect they have been exposed to a blood-borne pathogen are to report to the Emergency Room for evaluation. After the evaluation should the employee wish to be seen by their private physician they may do so. To assist the civilian employee's personal healthcare professional, we forward a number of documents to them, including the following (Appendix I):

(1). A copy of the Blood-borne Pathogens Standard.

(2). A description of the exposure incident.

(3). The exposed employee's relevant medical records.

(4). Other pertinent information.

d. Healthcare professionals written opinion.

Whether the employee is evaluated and treated within WRAMC or chooses to seek care in the private sector, the following information will be obtained by the treating physician and provided to WRAMC's Occupational Medicine Physician. After the consultation, the healthcare professional provides our facility with a written opinion evaluating the exposed employee's situation. The Occupational Medicine Physician, in turn, will furnish a copy of this opinion to the exposed employee. In keeping with this process' emphasis on confidentiality, the written opinion will contain only the following information:

(1). Whether Hepatitis B Vaccination is indicated for the employee.

(2). Whether the employee has received the Hepatitis B Vaccination.

(3). Confirmation that the employee has been informed of the results of the evaluation.

(4). Confirmation that the employee has been told about any medical conditions resulting from the exposure incident which require further evaluation or treatment.

(5). All other findings or diagnoses will remain confidential and will not be included in the written report. An employee fact sheet will be provided to the employee describing the symptoms of HIV and HBV infection (Appendix J).

e. Medical recordkeeping.

To make sure that we have as much medical information available to the participating healthcare professional as possible, our facility maintains comprehensive medical records on our employees. The Occupational Medicine Physician is responsible for setting up and maintaining these records, which include the following information:

(1). Name of the employee.

(2). Social security number of the employee.

(3). A copy of the employee's Hepatitis B Vaccination status. Dates of any vaccinations.

(4). Medical records relative to the employee's ability to receive vaccination.

(5). Copies of the results of the examinations, medical testing, and follow-up procedures which took place as a result of an employee's exposure to blood-borne pathogens.

(6). A copy of the information provided to the consulting healthcare professional as a result of any exposure to blood-borne pathogens.

(7). As with all information in these areas, we recognize that it is important to keep the information in these medical records confidential. We will not disclose or report this information to anyone without our employee's written consent (except as required by law).

. . . .

Source: Waxdahl, KA, LTC, AN, Chief, Infection Control Service; Phillips, KG, MAJ, MC, Chief, Occupational Medicine Program. *Exposure Control Plan.* Washington, DC: Walter Reed Army Medical Center. 4 May 1991: 16–31.

Chapter 6

HEALTH HAZARD ASSESSMENTS

ROBERT A. GROSS, M.S.* AND W. THOMAS BROADWATER†

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SUMMARY

THEATER HIGH ALTITUDE AREA DEFENSE SYSTEM: INITIAL HEALTH HAZARD ASSESSMENT REPORT

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INTRODUCTION

Although military duty is inherently hazardous, soldiers in combat should not be placed at a disadvantage or at unusual risk because their hardware is deficient or information is lacking regarding the health hazards associated with their equipment.[1] Neither should soldiers be exposed unnecessarily to health hazards during training, even though the training must be realistic to achieve a high degree of operational readiness. Therefore, the Army Medical Department's (AMEDD's) mission—to conserve the fighting strength—must include reducing the risks to soldiers' health that are posed by their own materiel (military equipment, weapons, clothing, and training devices). AMEDD must ensure that soldiers do not suffer serious adverse health effects as a result of operating their materiel systems, and that the equipment itself does not prevent them from performing at maximum efficiency.

By 1983, Department of Defense (DoD) Directive 5000.1, *Major Systems Acquisitions*, had instructed all uniformed services to consider health hazard assessment as an integral part of their materiel acquisition process, and Army Regulation (AR) 40-10, which formally established the U.S. Army Health Hazard Assessment (HHA) Program, was published in October of that year.[2,3] When properly executed and integrated into the army's Materiel Acquisition Decision Process (MADP), the HHA Program not only prevents injuries and job-related illnesses, but it also enhances the soldier's ability to accomplish his or her mission. For example, excess carbon monoxide reduces visual acuity (see Chapter 8, Conserving Vision, and Chapter 11, Carbon Monoxide). By eliminating or reducing carbon monoxide to its lowest acceptable level, the HHA Program prevents the soldier's performance from being degraded. There are no formalized, mandatory civilian programs for assessing the potential health hazards of equipment during the research and development stages.

Rationale

The HHA process applies biomedical knowledge and principles to document and quantitatively determine the risks that the materiel itself poses to the health and effectiveness of the personnel who test, use, or maintain U.S. Army equipment. The primary objective of the HHA Program is to identify, assess, and eliminate or control health hazards associated with weapons systems during the early stages of development and acquisition. Specifically, the program objectives are to identify and evaluate the health hazards caused by materiel to

- preserve and protect the soldier from such health hazards,
- reduce soldier performance decrement and enhance system effectiveness,
- reduce the need for retrofitting system designs,
- enhance readiness by reducing health hazards that cause training or operational restrictions (eg, reducing carbon monoxide to an acceptable level so that more rounds can be fired), and
- save money by eliminating or reducing occupational injury and illness compensations attributable to health hazards from the use of army materiel.[3]

This evaluation of hazard severity and hazard probability provides decision makers with (*a*) a formal estimate of the health risks associated with military hardware as it proceeds through the acquisition process, (*b*) a summary and discussion of potential and real health hazard issues, and (*c*) recommendations for methods of controlling, mitigating, reducing, or eliminating hazards.[3]

Militarily unique settings offer the preventive medicine team unusual and complex challenges. For example, tank or aviation crews can be exposed to simultaneous stresses—such as acoustical energy, chemical substances, temperature extremes, and whole-body vibration—each of which can produce several different adverse health effects.[4] All occupational health professionals within the military must deal effectively with the traditional hazards of an installation's industrial setting and the unique hazards of the military; and all military physicians must be competent to diagnose, manage, and report the adverse health effects associated with testing, using, and maintaining military equipment.

During the late 1970s and early 1980s, the army leadership as well as AMEDD and the materiel developers recognized the need for a medical review of new or improved equipment. This resulted from an increased awareness that *soldier performance decrements* (short-term, materiel-induced conditions that prevent a soldier from performing at maximum effi-

Fig. 1-1. The M198 155 mm Towed Howitzer. The M198 is a helicopter-transportable 155-mm medium towed howitzer. It has a conventional split trail carriage and uses a hydropneumatic recoil mechanism. The maximum rate of fire is 4 rounds per minute for the first 3 minutes and 2 rounds per minute sustained. It is capable of firing a 96 pound rocket-assisted projectile to a range of 30 km.

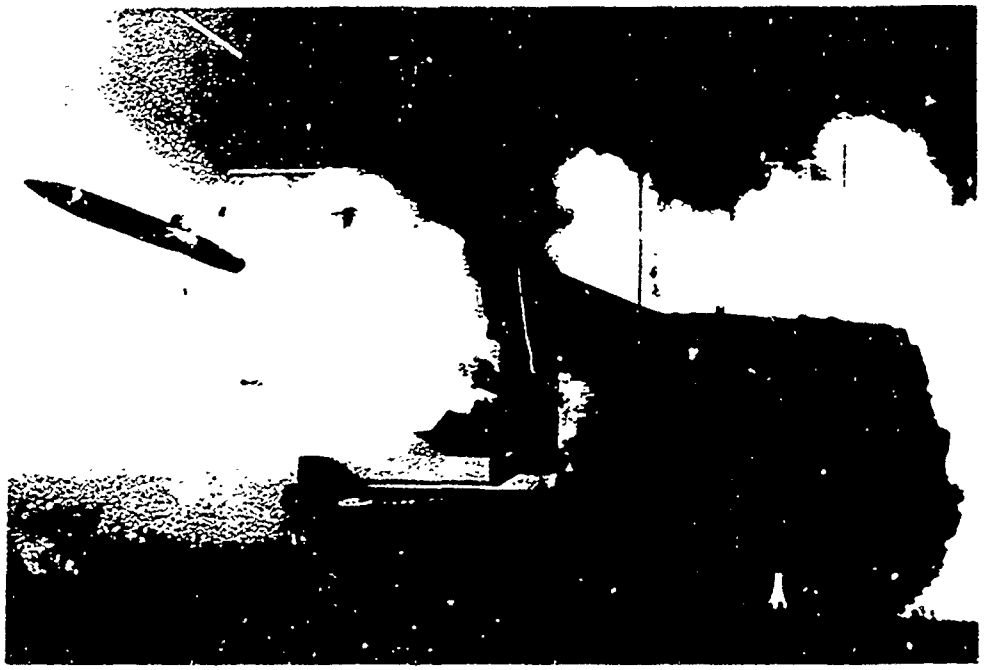

Fig. 6-2. The Multiple Launch Rocket System (MLRS). The MLRS is a free-flight artillery rocket system that improves the conventional, indirect-fire capability of the field army. It consists of a 12-round launcher mounted on a mobile, tracked vehicle. The MLRS is capable of launching rockets, with varying types of warheads, either one at a time or in rapid ripples, to ranges beyond 30 km.

Fig. 6-3. The Bradley Fighting Vehicle (BFV). The BFV is named for the late General of the Army Omar N. Bradley. Two versions, the M2 Infantry Fighting Vehicle (IFV) and the M3 Cavalry Fighting Vehicle (CFV), are externally indistinguishable and have the same armament and automotive performance. The major difference between the M2 and M3 is the arrangement of the crew compartment and internal storage. The armament includes the M242 25-mm chain gun, M240 7.62-mm coaxial machine gun, and a TOW (tube launched, optically tracked, wire-guided) antitank missile launcher.

Fig. 6-4. The Stinger Manportable Antiaircraft Missile. The Stinger was developed by the army to provide individual combat soldiers with effective air defense in forward combat areas. It is so popular that it is integrated into the active inventories of all four armed services. Three variants are in operational inventories: the basic Stinger, Stinger-POST (passive optical seeker technique), and Stinger-RMP (reprogrammable microprocessor).

ciency) and adverse health effects were associated with the use of field equipment and were defining the limits of technology for new systems. Systems that were developed during this time, which could have benefited from very early AMEDD HHA input during the MADP, include the M198 155-mm howitzer (Figure 6-1), the Multiple Launch Rocket System (MLRS, Figure 6-2), the Bradley Fighting Vehicle (BFV, Figure 6-3), and the Stinger Manportable Antiaircraft Missile (Figure 6-4).

Soldiers who fired the M198 experienced chest-wall pain and blood-tinged sputum. These are signs of *primary blast injury*, which occurred when the weapon was fired.

> *Blast injury* is a general term that refers to the biophysical and pathophysiological events and the clinical syndromes that occur when a living body is exposed to blast of any origin. Blast-wave physical properties, the complexity of the waveform, and the number of blast repetitions determine the potential for *primary blast injury*.[6:2:2]

In this instance, blast injury was controlled by restricting the number of rounds (blast repetitions) fired each day by each member of the howitzer crew.

Hydrogen chloride is a combustion product of ammonium perchlorate–based propellant used in the MLRS missile. The MLRS crew members experienced temporary eye and respiratory irritation when hydrogen chloride gas entered the crew compartment during missile launch. The hydrogen chloride levels outside the crew compartment are also high enough to incapacitate unprotected personnel. An early medical review would probably have recommended the use of an alternate, safe propellant. Initially, crew members inside the crew compartment were required to wear their protective masks until modifications were made to improve the compartment seals and overpressure system.

Several shortcomings were found in the design of the BFV. The high, steady-state (continuous) noise levels were similar to those found in its predecessor. Such high noise levels are typical of armored tracked vehicles, and are characteristic of the existing design of their suspension and drive system. Hearing loss among crew members and passengers is controlled by double hearing protection (the Combat Vehicle Crewmember [CVC] DH-132 helmet and army-approved ear plugs) and limiting the time each day that personnel can occupy the vehicle. However, double hearing protection adversely affects speech intelligibility among crew members talking on the vehicle's intercom system, and between the crew and external communications stations. Another shortcoming of the BFV is that the

heater in the crew compartment is unable to provide adequate heat while operating at the vehicle's minimum design temperature, and low temperatures within the crew compartment adversely affect the crew's ability to operate the vehicle. Testing found the heater to be unsatisfactory, especially at the driver's position. An early medical review would probably have recommended that more appropriate heater performance in the crew compartment be included in the vehicle's design specifications.

The rocket motor in the Stinger, like that in the MLRS, uses ammonium perchlorate–based propellant. Recommendations to wear the protective mask to prevent respiratory tract irritation from the high concentrations of hydrogen chloride gas following a Stinger firing proved to be inappropriate: a soldier must also hold his breath for 40 to 60 seconds after firing a Stinger to prevent any additional performance decrement. The protective equipment of choice (the mask) interferes with the soldier's ability to use the weapon: the facemask prevents the soldier from placing the weapon against his cheekbone. Using a different propellant would have solved this problem. However, the Stinger was one of the first weapons systems evaluated as part of the HHA Program; the weapon had already been fielded and its design could not be changed.

Unfortunately, because these shortcomings have not been properly addressed, they are perpetuated. Increased costs and additional health concerns inevitably result as combat and materiel developers attempt to improve or incorporate existing equipment into developing systems.

History

In 1866, the army replaced the union repeating gun with the Gatling gun—a six-barrel machine gun that employed a new, improved, steel-jacketed cartridge.[16] The redesign solved the sharp-trauma hazard: the misalignment of the gun parts had caused soft metal particles to be shaved off; the steel-jacketed cartridge

Fig. 6-5. White Armored Car (M1913-1914), shown at the U.S. Army Ordnance Museum, Aberdeen Proving Ground, Maryland. Built by the White Motor Company, this vehicle weighed about 2 tons and was a built-up armored truck. It had dual rear wheels with pneumatic tires and a caliber .30 Vickers-Maxim machine gun in the rounded turret. It was used by General Pershing's troops along the Mexican border in 1916 during their pursuit of Pancho Villa.

prevented this shaving. However, we have no idea whether the Gatling gun eliminated other health hazards such as toxic fumes, segmental vibration, or impulse noise, because *these were not recognized* as health hazards.

The cavalry's conversion of the horseless carriage into a moving armored fortress was probably one of the earliest steps in the development of the tank. At least as early as 1902, a heavy, low-powered, armored car with a periscope was developed (Figure 6-5).[7]

The U.S. Army used some versions of early armored vehicles along the Mexican border in 1916. Armored cars are lighter-armored, wheeled, and carry a machine gun. Tanks have heavier armor, heavier weapons, and are tracked vehicles. However, the development of tanks required the concomitant development of two major technologies: an internal combustion engine capable of providing sufficient power to move the heavy tanks, and tracks that would permit the vehicle to cross rough terrain.[7]

Tanks, like machine guns, were developed during an era when occupational health hazards were not a concern. Although specific health hazards associated with the earliest tanks were not documented, we can assume that the steady-state noise and whole-body vibration from the primitive track design would have caused severe health problems for soldiers in the tanks, and that the engines would have caused exposure to heat and toxic combustion products. Soldiers in tanks during World War I definitely experienced health hazards that were different from the impulse noise and blast overpressure traditionally associated with artillery and small arms:

> Inside the tanks, the crews worked manfully to steer and control their lumbering charges. There was very little room to move about in, most of the space being taken up by the large petrol engine in the centre. The interior was dimly lit by a naked electric light bulb, fed from the batteries. Vision to the outside was provided through narrow glass prisms, which had a habit of splintering into a driver's eyes when hit by a bullet.[7]

The first major tank battle occurred in northern France on 20 November 1917, when 378 tanks moved from behind British lines (see Figure 1-5, Chapter 1, Occupational Health in the U.S. Army). The crews inside these tanks experienced noise and vibration so intense that their wireless transmitters could not be used.[7] Semaphores were adopted as a less-than-ideal alternative. (Even now, the noise in tanks is so extreme that tank commanders often use hand and arm signals to communicate.)

The Armored Medical Research Laboratory was established at Fort Knox, Kentucky, in early 1942 as the U.S. Army's first organized attempt to evaluate the medical consequences of weapons systems designs. During World War II, the staff—physicians, medical and physical scientists, and engineers—compiled an impressive array of reports covering a wide range of human factors and health issues such as fatigue, heat stress, and toxic gases.[8,9] The laboratory made extremely valuable contributions regarding the identification, evaluation, and control of health hazards associated with the use of military equipment. The medical department, however, had yet to grasp the importance of systematically reviewing new military items for health hazards posed to the operators and maintainers.

THE HEALTH HAZARD ASSESSMENT PROGRAM

Weapons and equipment development continued after World War II, yet AMEDD was still not integrated into the MADP, through which new materiel items are developed and fielded. As a consequence of the questions that had been raised during the final stages of development of the M198 howitzer and the BFV system, the application of technology to new or improved systems was seen to be limited by soldier performance decrements and adverse health effects. Thus, army leadership—including the vice chief of staff, surgeon general, deputy chief of staff for personnel, and deputy chief of staff for operations—directed the formalization of a process to address, as early as possible in the MADP, health hazard issues associated with new materiel. The U.S. Army Health Hazard Assessment Program, which was formally established with the publication of AR 40-10, requires that a medical review of materiel items be performed at critical decision points during the MADP.[3]

In 1987, the Department of the Army undertook a large-scale effort to address the hazards of increasingly powerful and sophisticated weapons systems: greater noise and blast overpressure, more shock and vibration, and higher concentrations of toxic fumes and gases.[10] The army's deputy chief of staff for personnel initiated the Manpower and Personnel Integration (MANPRINT) Program.[11] MANPRINT integrates the full range of human factors engineering, manpower, personnel, training, HHA, and system safety considerations, with the goal of improving the performance of the individual soldier and the total system throughout the MADP. This ensures that the human aspects of the soldier–machine interface are considered early in the design and development of weapons systems.

The HHA Program is a primary domain within the overall MANPRINT Program. Careful coordination and interaction between HHA Program activities and other MANPRINT domains are essential for a cohesive, comprehensive, and efficient MADP. Thus, the MANPRINT joint working groups integrate the HHA report throughout all MANPRINT domains (such as human factors engineering; system safety engineering; and manpower, personnel, and training assessments). In addition, the U.S. Army Systems Acquisition Review Council verifies that the Office of The Surgeon General (OTSG) has completed the proper HHA report, and that appropriate action is taken by the materiel developer or the combat developer to resolve health hazard issues. AR 40-10 defines a *materiel developer* as "any organization responsible for developing or modifying materiel" and a *combat developer* as "any organization responsible for developing or modifying doctrine on how the Army will fight."[3(p15)]

Organizational Support

The AMEDD organizations that have major roles in supporting combat and materiel developers within the HHA Program, including technical expertise (which can include manpower), are the OTSG, the Health Services Command (HSC), and the Medical Research and Development Command (MRDC). Together, these organizations (*a*) coordinate the program and establish program policy; (*b*) review requirements documents, serve on MANPRINT joint working groups, and assist in preparing the System MANPRINT Management Plan (SMMP); and (*c*) conduct biomedical research.

Program Coordination and Policy Establishment

The OTSG is the proponent of the HHA Program, and thus establishes program policy and provides coordination for the program. The HHA coordinator, assigned to the Preventive and Military Medicine Consultants Division of the OTSG, provides this coordination. Normally, HSC's U.S. Army Environmental Hygiene Agency (USAEHA) prepares the HHA reports, but occasionally the MRDC prepares them.[12,13]

Requirements Documents

The army's requirements for a particular materiel system that is necessary to correct a battlefield deficiency, based on current army combat doctrine, are contained within a *requirements document*. The combat developer prepares the requirements document and staffs it worldwide for comments on the concept. This is known as the *concept-based requirements system.*

Several components within the HSC, including the AMEDD Center and School (formerly called the Academy of Health Sciences), review and comment on these documents. Preventive medicine personnel at Medical Department Activities (MEDDACs)—which support Training and Doctrine Command (TRADOC) schools and integrating centers—work with the Director of Combat Development to review requirements documents and provide relevant health hazard assessment guidance.

Biomedical Research

The OTSG and the MRDC play important roles in biomedical research. The OTSG is responsible for identifying the health hazard assessment–related biomedical research needs. The MRDC and the OTSG both establish and prioritize HHA research requirements; the MRDC then performs the HHA research as a part of its larger medical research and development programs. Such research may consist of laboratory investigations, the development of technology and methodology, mathematical modeling, field evaluations, or epidemiological surveys.

Biomedical research can be used to improve or develop new tools to advance HHA capabilities. For example, the HHA Program may be using tools (such as biomedical databases, prediction models, and methods for evaluating protection) that are unable to measure the specific health hazards of a developing system. The HHA Program has a mission to develop technology-based research efforts aimed at answering health hazard–related questions that are militarily unique and that have no direct correlates in the civilian occupational health community. Examples of militarily unique biomedical research issues include

- developing standards for
  - exposure to carbon monoxide;
  - short-term, high-level exposures to hydrogen chloride and ammonia from missile firing;
  - exposure to lead from the firing of self-propelled artillery;
  - short-term, high-level exposures to hydrogen fluoride and hydrogen bromide as combustion and decomposition byproducts of halon fire-extinguishing agents;
  - whole-body vibration from operating tactical vehicles; and
  - exposure to acoustical energy;
- and characterizing the toxicity of
  - military smokes and obscurants,

- propellant compounds, and
- materials that come in direct contact with the soldier.

The identification of health hazard research needs usually results from voids in basic data that are to be applied to the HHA of materiel. These research needs, in addition to the identified deficiencies and requirements, should be specified in key MADP planning documents (such as Mission Area Analysis and the Battlefield Development Plan). However, the incorporation of these research requirements into the HHA's research effort requires close coordination between planning agencies, especially TRADOC, the U.S. Army Materiel Command (AMC), and the OTSG. Therefore, it is essential that combat system and technology developers, test and evaluation personnel, and human factors and system safety personnel notify the OTSG when potential health hazard research requirements come to their attention. In addition, the MANPRINT joint working group should document health hazard research requirements in SMMPs.

Biomedical research is funded by the MRDC, the materiel developer, or the Program Executive Office budget, depending on whether the research is related to a specific materiel system. The MRDC provides funds for generic, armywide, HHA-based, research needs that are not system specific. However, funding for health hazard research that is relevant to specific materiel systems relies heavily on research, development, testing, and evaluation funds from the materiel developer or Program Executive Office budget. This research forms the basis for the materiel developer to provide customer funds to the MRDC.

Funds for health hazard research that is required to address specific health hazards associated with a particular materiel-acquisition program should be identified as early as possible in key acquisition program management documents, including the SMMP, to ensure that adequate resources are available in a timely manner.

The Health Hazard Assessment Report

The HHA report is a standardized, systematic, multidisciplinary evaluation of the health risks associated with a materiel system. The HHA report determines if materiel systems pose any potential health hazards, and presents recommendations for corrective or preventive measures or both. The report is designed to document the logical process for developing recommendations (Table 6-1). AR 40-10 defines the report's content and preparation.[3]

An initial HHA report is usually prepared early in the developmental cycle and identifies

- potential health hazard issues associated with a materiel solution for a projected battlefield deficiency (an item is either developed or purchased to solve a deficiency), and
- pertinent health standards based on both developmental and predecessor systems.

TABLE 6-1

FORMAT FOR THE HEALTH HAZARD ASSESSMENT REPORT

Paragraph Topics	Contents
References	Listing of source materials
Summary	Executive overview with a brief system description, potential health hazards, and a brief assessment of the system with major recommendations
Background	System description, use scenario, acquisition strategy, summary of previous assessments/text reports used to evaluate the system
Identification of Issues	Listing of potential/actual health hazards associated with the system
Assessment of Issues	Data analysis and conclusion compared to health standards
Recommendations	Recommended actions for hazard control/elimination with risk assessment codes
Identification of Preparer	Preparing organization, point of contact, date prepared

Adapted from US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process.* Washington, DC: DA; 1983. Army Regulation 40-10: 9.

The initial report's recommendations focus on both data that will be required and design specifications that address specific potential health hazards.

Required Data

Information and health standards are the key ingredients of an HHA report, but information is often difficult to obtain from materiel and combat developers. In general, both descriptive and quantitative information concerning the materiel system must be available to the independent medical assessor (IMA) who conducts the assessment. (The IMA—who can be either civilian or military—is not employed by the combat or materiel developer, but rather by the AMEDD organization that is completing the HHA report.) Health standards must also be made available, so that the IMA can compare or evaluate the severity of health hazards associated with the materiel system.

Definitive statements about levels of risk associated with potential health hazards are impossible to make without quantitative data. However, in the case of an initial HHA report, only data from a predecessor, or similar, system may be available. Quantitative information about materiel systems should include health hazard–related data (such as noise and vibration signatures and toxic-gas measurements) from technical testing, user testing, special hazard evaluations, previous HHAs, human factors engineering assessments, safety incident and system safety assessment reports, and modeling efforts.

Combat and materiel developers should also provide descriptive information, including a comprehensive account of components, subsystems, special materials, simulators and other training devices, special support and maintenance equipment, special salvage or disposal requirements, and system employment (such as operating and training doctrine; logistics support concepts; nuclear, biological, and chemical requirements; and expected environmental conditions).

Health standards (such as medical exposure limits, health conservation standards, and materiel design standards) are essential to gauge the severity of quantified hazards. Comprehensive biomedical databases are very helpful in gauging real levels of risk, especially when quantified hazards exceed established limits, although such databases are often unavailable.

Preparation Sequence

The IMA uses a systems approach in analyzing hardware and doctrine. This approach analyzes all components and subsystems, all phases of the system's "life cycle," how personnel interact with the system, the special operating conditions, and anticipated environmental conditions. Then, the IMA compiles a comprehensive inventory of potential health hazards and procedures from the analysis of hardware and doctrine. The inventory may include items such as materials, procedures, and design deficiencies. After compiling the inventory, the IMA analyzes the quantitative data available for each potential hazard, and requests further data to complete the analysis. Raw or intermediate data may need to be reduced, converted to a more useful form, or reorganized to a form more suitable for interpretation. When data are adequate for interpretation, the IMA compares them against pertinent health standards to ascertain whether the quantified levels are acceptable, given the frequency and duration of exposure expected.

The IMA then recommends means to eliminate, control, or reduce health hazards that pose an unacceptable degree of risk. These exposure controls can be tailored to the specific system and its operational requirements, and more than one type of control may be necessary for some hazards. Such control measures include engineering controls (such as redesign, system modifications, and retrofits), administrative controls (such as exclusion of high-risk personnel, and limiting duration or frequency of exposure), and requiring that personal protective equipment (PPE) be worn.

For each hazard, the IMA estimates the degree of risk that could result from noncompliance with recommended control measures. A scale of risk assessment codes (RACs) is used to classify the degree of each hazard (Table 6-2), which is useful in establishing priorities for control actions. The RACs relate hazard severity and hazard probability. The hazard severity (divided into categories) and the hazard probability (divided into levels) integrate to yield a number (1 to 5). The lower the number, the higher the risk assessed. For example, consider category II, level E in Table 6-2. The probability of occurrence is improbable (unlikely to occur, but possible), and the hazard posed may cause severe bodily injury (critical); therefore, the RAC is 4. The goal has been to make the RAC process as objective as possible, but the professional judgment of the IMA remains a subjective component.

Hazard severity assesses the worst potential consequence. Several factors define this assessment, including the degree of injury, occupational illness, health-related performance degradation, and possible bodily system damage. *Hazard probability* assesses the likelihood that a hazard will occur, based on factors such as location, exposure (in cycles or hours of operation), and population affected. The decision-making au-

TABLE 6-2

RISK ASSESSMENT CODES

Severity Category	Probability Level				
	A	B	C	D	E
I	1	1	1	2	3
II	1	1	2	3	4
III	2	3	3	4	5
IV	3	5	5	5	5

I (Catastrophic): hazard may cause death or total loss of bodily system

II (Critical): hazard may cause severe bodily injury, severe occupational illness, or major damage to a bodily system

III (Marginal): hazard may cause minor bodily injury, minor occupational illness, or minor damage to a bodily system

IV (Negligible): hazard would cause less than minor bodily injury, minor occupational illness, or minor bodily system damage

A (Frequent): likely to occur frequently, or continuously experienced

B (Probable): will occur several times in life of an item, or will occur frequently

C (Occasional): likely to occur sometime in life of an item, or will occur several times

D (Remote): unlikely, but possible to occur in life of an item, or, unlikely, but can reasonably be expected to occur

E (Improbable): so unlikely it can be assumed occurrence may not be experienced, or unlikely to occur, but possible

Adapted from US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process*. Washington, DC: DA, 1991. Army Regulation 40-10: 11–12.

thorities in the MADP use RACs to determine which health hazards must be either resolved or accepted before a materiel system can progress to the next level of development or production.

Health Hazard Assessments During the Materiel Acquisition Decision Process

Just as HHA concerns should be integrated throughout all MANPRINT domains, so they should also be integrated throughout all phases of the materiel system development and acquisition cycle. Initially, the materiel or combat developer must submit draft system requirements documents to the USAEHA, the MRDC, or HSC's AMEDD Center and School for a medical review. These organizations identify potential health hazards and applicable health standards and return their comments to the materiel or combat developer. HHAs should be used during (*a*) program initiation, (*b*) concept exploration, (*c*) demonstration and validation, (*d*) full-scale development, and (*e*) production and deployment.

Program Initiation

When the MADP is being initiated, the combat developer should assign responsibilities and formulate requirements documents. The combat developer should incorporate health hazard considerations and criteria into requirements documents based on predecessor or similar systems. AMEDD sources may provide such information. In addition, designated preventive medicine personnel assigned to TRADOC installations should identify responsibilities and tasks needed to control potential health hazards and include them in the SMMP.

Concept Exploration

During the concept exploration phase, the combat and materiel developers should ensure that requirements for an HHA are included in acquisition program management documents. The combat and materiel developers should also submit a request for an HHA report to the OTSG. This request should include any available health hazards–related test and evaluation data contained in other program documents.

Other required program acquisition documents may provide useful HHA-related information. These documents are the Human Factors Engineering Assessment, Safety Assessment Report, and safety and health data sheets. In addition, the OTSG, the USAEHA, and the MRDC also provide health hazard consultation as required.

Demonstration and Validation

During the demonstration and validation phase of the MADP, the combat and materiel developers and the IMA collect health hazard data, which will form the basis for an updated HHA report. AMEDD elements continue to furnish health hazard consultation to the materiel developer to control health hazards. When the developer is unable to address these issues,

AMEDD may assist in collecting data and in refining collection requirements and methods. In addition, formal requirements documents should specifically address health hazard considerations peculiar to a developing system.

Full-Scale Development

During the development phase of the MADP, test personnel collect data to address unresolved health hazard issues. The materiel developer should request an updated HHA report from the OTSG to determine the developing system's health-risk status. The results of this assessment should be included in the SMMP and other acquisition program safety and health documents such as safety and health data sheets, safety assessment reports, human factors engineering assessments, and MANPRINT assessments. The materiel developer corrects or controls remaining health risks, or documents management decisions that accept risks associated with major hazards. Contract specifications are developed and refined to ensure compliance with health hazard requirements.

Production and Deployment

Health hazard–control procedures adopted as a result of HHA report recommendations should be incorporated into acquisition program technical publications and training materials. *Production testing* documents the developing system's conformance with HHA-related contract specifications. Test personnel collect required data on unresolved health hazard issues during postproduction testing (such as Follow-on Operational Test and Evaluation) and submit it to AMEDD for review. The materiel developer ensures that (*a*) proposals for engineering change proposals receive proper review for health hazard implications and (*b*) decisions that resolve remaining health hazard issues are documented and implemented.[13]

FINDINGS OF HEALTH HAZARD ASSESSMENTS

About 100 HHAs per year have been done on army materiel systems since the program was formalized in 1983. (Approximately two-thirds of these assessments require a formal HHA report.) Nine general categories of health hazards have been identified (Table 6-3). An HHA will typically address army *developmental* and *nondevelopmental* items for each of these nine possible hazard categories. AR 70-1 defines a developmental item as one that is "under development or which was developed by the army."[14:p80] The same document defines a nondevelopmental item as

> those items available for procurement to satisfy an approved materiel requirement from existing sources (such as commercial items and items developed by other government agencies, U.S. military service, or countries) requiring little or no additional development.[14:p81]

Usually, the more complex and sophisticated the materiel system, the more categories of potential health hazards that will need to be addressed. The HHA report that is appended at the end of this chapter was selected for inclusion in this textbook because it addresses five categories of health hazards—an unusually high number—and deals with a system that is currently under development.

Acoustical Energy

Acoustical energy is defined as the potential energy that exists in a pressure wave, transmitted through air, which can interact with the body to cause hearing loss or damage to internal organs. It includes *steady-state* (also called *continuous*) noise from engines and helicopter rotors, *impulse* noise from firearms, and *blast overpressure* from mortars and towed artillery (free-field waves) and heavy weapons on crew-served vehicles (complex waves).[3]

Lighter, Air Cushion Vehicle, 30-Ton Capacity

The Lighter, Air Cushion Vehicle, 30-Ton Capacity (LACV-30) is an air-cushion cargo transport vehicle capable of operating over water, beaches, ice, and snow. This vehicle is powered by two sets of gas turbine engines, which drive lift fans, and two propulsion propellers (Figure 6-6).

An HHA of the LACV-30 identified the power train as a source of high levels of steady-state noise (see Chapter 7, Noise and the Impairment of Hearing, for a discussion of steady-state noise). Based on Military Standard 1474, the HHA report recommended that crew members and passengers use single hearing protection (such as the DH-132 CVC Helmet, the SPH-4 Aviator's Helmet, or approved ear plugs) to protect themselves from noise-induced hearing loss.[15]

Bradley Fighting Vehicle

The BFV is a tracked, light-armored vehicle. Both versions—the M2 Infantry Fighting Vehicle (IFV) and the M3 Cavalry Fighting Vehicle (CFV)—are equipped

TABLE 6-3

HEALTH HAZARD CATEGORIES

Category	Description	Examples	Related Publications
Acoustical Energy	Potential energy in a pressure wave, transmitted through air, which can cause hearing loss and damage internal organs	Steady-state noise: engines and helicopter rotors Impulse noise: small arms Blast overpressure: mortars, towed artillery (free-field wave) heavy weapons on crew-served vehicles (complex wave)	AR 40-5 MIL-STD-1474 MIL-STD-1294 DA PAM 40-501
Biological Substances	Pathogenic microorganisms, their toxins and enzymes	Sanitation concerns such as waste disposal, food handling, and personal hygiene	AR 40-5 FM 21-10 TB MED 530 TB MED 577
Chemical Substances	Excessive airborne concentrations of mists, gases, vapors, and particulate matter; also toxic liquids and solids	Combustion products from weapons or engines Exposures via inhalation, ingestion, dermal or eye contact	AR 40-5 MIL-STD-1472 MIL-HDBK-759 21 CFR 177 21 CFR 182 29 CFR 1910
Oxygen Deficiency	Sudden reduction of atmospheric O_2 to < 21% (by vol)	In confined spaces and high altitude: can cause shortness of breath; impaired vision, coordination, and judgment, progressing to unconsciousness and death	TB MED 288 DHEW(NIOSH) Pub. 80-106 29 CFR 1910 ANSI Z117.1
Radiation Energy	*Ionizing*: any form of radiation sufficiently energetic to ionize molecules in matter *Nonionizing*: emissions from the EM spectrum with insufficient energy to ionize molecules	Alpha and beta particles, gamma and X rays, neutrons UV, visible, IR, microwave, and RF radiation	AR 40-5 AR 40-14 AR 40-46 AR 40-583 AR 385-9 AR 385-11 MIL-STD-1425 TB MED 522 TB MED 523 TB MED 524 10 CFR 0-199 21 CFR 1040
Shock	Mechanical impulse or impact received by the body	Acceleration: recoil from weapon Deceleration: opening of parachute harness	MIL-STD-858 MIL-STD-1290 SAE-J* 855
Temperature Extremes	Injuries from excessive heat and cold, which can be exacerbated by humidity	Heat: heatstroke, hyperthermia Cold: frostbite, hypothermia	AR 40-5 MIL-STD-1472 TB MED 81 TB MED 288 TB MED 507
Physical Trauma	Injury to eyes or body from impact or strain	Penetrating Blunt: crush injury, bruise Musculoskeletal: lifting heavy equipment	AR 40-5 TB MED 506 29 CFR 1910 ANSI Z87.1
Vibration	Adverse health effects caused by contact of oscillating mechanical surfaces with the human body	Whole body: aircraft and vehicle operators and passengers Segmental: operators of hand-held power tools	MIL-STD-1472 ANSI S3.18 ISO 2631†

* Society of Automotive Engineers
† International Standards Organization
Adapted from US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process*. Washington, DC. DA; 1991. Army Regulation 40-10, App C: 12-13.

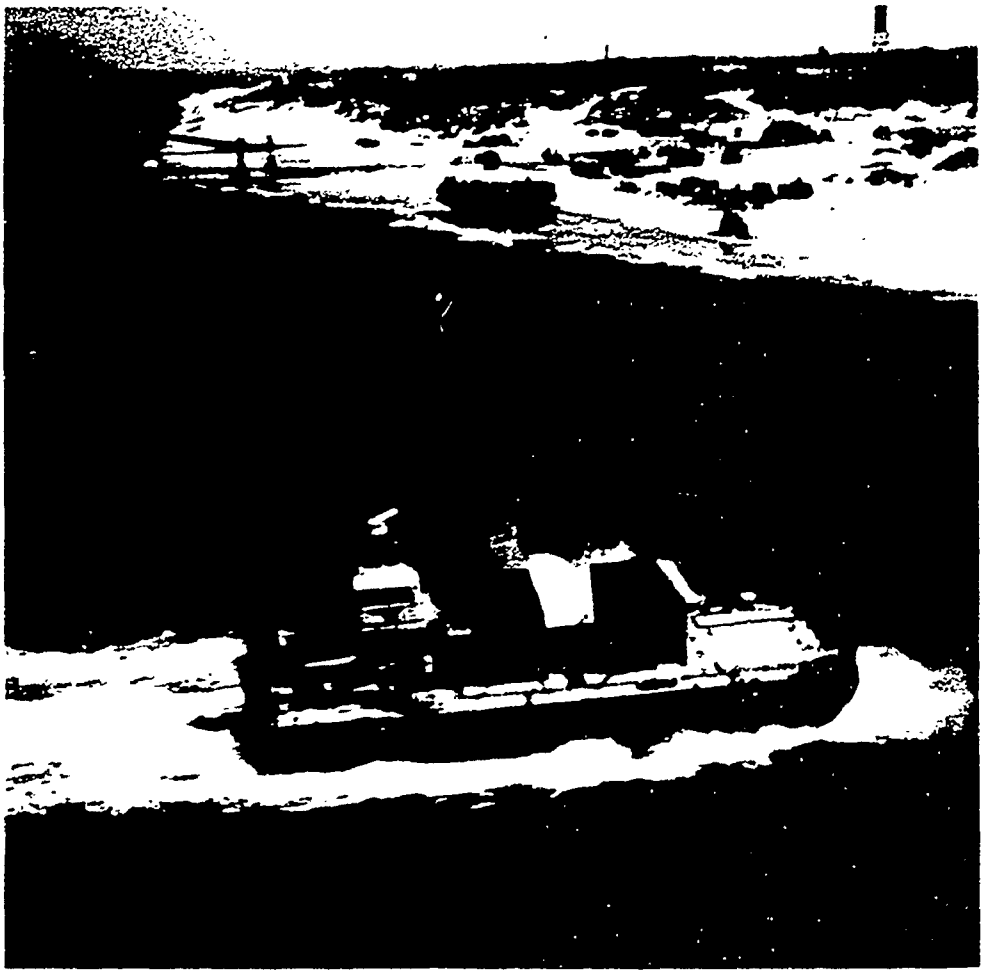

Fig. 6-6. The Lighter Air Cushion Vehicle, 30-Ton Capacity (LACV-30). The LACV-30 is capable of around-the-clock operations regardless of the weather. It easily transports wheeled and tracked vehicles, containers, and bulk cargo on its 1,660 ft² deck. Its maximum payload is 75 tons, cruise speed is 45 mph, and endurance is 8 to 10 hours. U.S. Army transportation units received their first production craft in 1982.

with a turret-mounted 25-mm gun, a 7.62-mm machine gun and a tube-launched, optically-tracked, wire-guided (TOW) antitank missile launcher (see Figure 6-3). The IFV carries a nine-man infantry squad and a commander, gunner, and driver. The CFV carries a crew of five: the commander, gunner, driver, and two reconnaissance crew members.

HHAs of the BFV identified high steady-state noise levels—due to the design of the suspension and drive system—and impulse noise when the guns were fired. The HHA report recommended that crew members wear double hearing protection, such as the DH-132 CVC Helmet worn with approved ear plugs. The HHA report also recommended that use of the vehicle be limited during training to prevent noise-induced hearing loss.[16]

M-120 Series 120-mm Battalion Mortar System

The M-120 Series 120-mm Battalion Mortar System (BMS-120) is a smoothbore, muzzle-loading, indirect fire system, which consists of the M-120 Towed Mortar, transported by a quarter-ton truck, and the M121 carrier configuration mounted in a modified M113 Armored Personnel Carrier (Figure 6-7).

The BMS-120 generates high-impulse noise levels, and a blast attenuating device (BAD) is used to reduce exposures at crew locations. An HHA report on the BMS-120 recommended that

- firing be limited when a BAD is not installed,
- all personnel within 200 m of the mortar wear ear plugs, and

Fig. 6-7. 120-mm Battalion Mortar System (BMS-120). The BMS-120 provides dismounted (walking alongside) and mechanized infantry units increased range and lethality with high-explosive, illumination, and smoke-screening rounds. Concurrently with the acquisition of the BMS-120, a new family of enhanced ammunition is being developed. This M121 carrier-mounted configuration is shown with a BAD (blast attenuating device) mounted on the muzzle of the mortar.

- the number of rounds fired per 24 hours be limited.[17]

M109 155-mm Howitzer Improvement Program Self-Propelled Howitzer

The M109 Howitzer Improvement Program (HIP) Self-Propelled Howitzer (SPH) is an aluminum-armored, self-propelled, air-transportable field artillery weapons system. It is designed to provide support to armored and mechanized infantry units. The HIP includes many survivability improvements and has a projectile range (increased over previous self-propelled 155-mm howitzers) of up to 30 km with rocket-assisted projectiles (Figure 6-8).

Several HHAs have been completed on the M109

Fig. 6-8. The M109 155-mm Howitzer Improvement Program (HIP) Self-Propelled Howitzer (SPH). The HIP includes a new cannon and mount, an on-board fire-control system, a navigation system, automotive improvements, additional ballistic protection, NBC (nuclear-biological-chemical) protection for the crew, a driver's night-vision device, built-in test equipment, and secure communications. HIP modifications will be applied to all M109 SPHs not converted to M109A4 or M109A5, under the designation M109A6 Paladin, shown above.

HIP SPH. This system generates high levels of steady-state noise. The particular blast overpressure experienced by the crew and resupply vehicle personnel is a function of a series of complex variables such as the type of charge, hatch and vehicle configuration, and quadrant elevation of the gun tube. Thus, the HHA report recommended that

- the crew use hearing protection,
- certain restrictions apply to vehicle and hatch configurations, and
- the number of rounds fired per day be limited, based on the type of projectile and the zone of the charge fired.[18]

Biological Substances

In the broadest sense, the term *biological substance* includes exposure to pathogenic microorganisms and their toxins and enzymes. In the specific sense used in HHAs, biological hazards include sanitation concerns such as waste disposal, food handling, and personal hygiene.[3]

Composting Toilet and Aerated Vault Toilet

The Composting Toilet and the Aerated Vault Toilet technologies are self-contained human waste–disposal systems designed for use at remote training and operational sites to replace chemical and pit latrines (Figure 6-9). The composting toilet is a large chamber into which wastes and organic bulking agents are placed. The Aerated Vault Toilet accomplishes natural aerobic decomposition of waste into humic material through aeration by a series of air channels, baffles, and a fan.

An early HHA of the two technologies recommended appropriate administrative controls and maintenance procedures to minimize harborage and breeding of

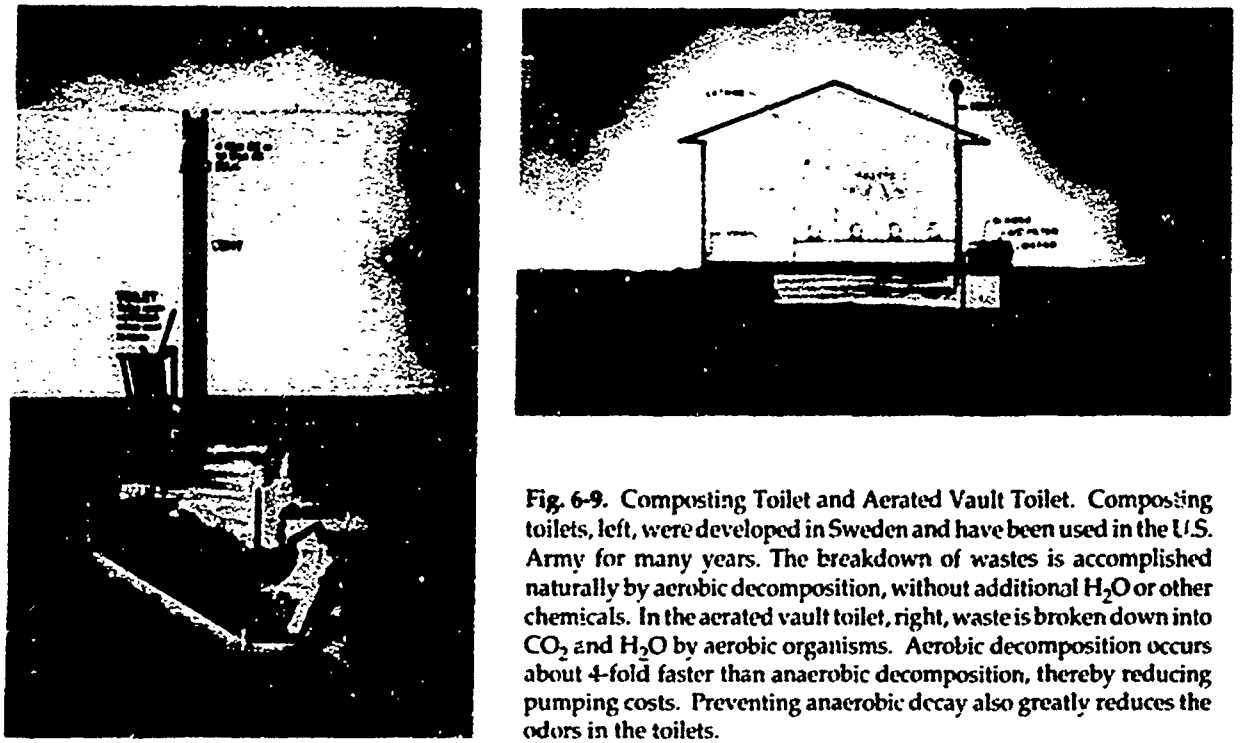

Fig. 6-9. Composting Toilet and Aerated Vault Toilet. Composting toilets, left, were developed in Sweden and have been used in the U.S. Army for many years. The breakdown of wastes is accomplished naturally by aerobic decomposition, without additional H_2O or other chemicals. In the aerated vault toilet, right, waste is broken down into CO_2 and H_2O by aerobic organisms. Aerobic decomposition occurs about 4-fold faster than anaerobic decomposition, thereby reducing pumping costs. Preventing anaerobic decay also greatly reduces the odors in the toilets.

insect and mammalian vectors of disease (primarily flies and rats), including

- enrollment of compost handlers in the Medical Surveillance Program for Wastewater Treatment Plant Operators, and
- daily cleaning (when in use) of the toilet seats and surrounding surfaces with soap and water.

Additional study of the dissemination of coliform bacteria via aerosols and direct contact was recommended to serve as a paradigm for the spread of these human pathogens.[19]

Resuscitation Fluids Production System

The Resuscitation Fluids Production System (REFLUPS) is a compact, self-contained unit designed to produce 75 L per hour of sterile, pyrogen-free water for injection, and to reduce transport and storage requirements in remote areas. The processed water is mixed with a fluid concentrate to reconstitute a variety of products for intravenous administration. The system will be used for on-site production of intravenous fluids at medical treatment facilities (MTFs) operating in combat zones and aboard naval vessels.

The HHA of the REFLUPS recommended a specific method to evaluate the system's ability to remove viral contamination. In addition, the HHA recommended that each lot of water for injection and reconstituted intravenous fluids be checked for compliance with U.S. Pharmacopeia standards for sterility and pyrogens.[20]

Chemical Substances

Hazards from chemical substances (not only the combustion products from weapons or engines but also other toxic materials) arise from excessive airborne concentrations of mists, gases, vapors, fumes, or particulate matter. Toxic effects may be caused by exposure via inhalation, ingestion, or eye or dermal contact. Hazards may also be caused by exposure to toxic liquids and solids by ingestion or eye or dermal contact.[3]

Avenger

The Avenger, originally called the Pedestal Mounted Stinger, is a component of the Forward Area Air Defense System (FAADS) and is employed in the rear battle area. The Avenger consists of a Stinger missile and a .50-caliber machine gun pedestal, which are turret-mounted on a High-Mobility, Multipurpose, Wheeled Vehicle (HMMWV), M998. The system is used against enemy fixed- and rotary-wing aircraft (Figure 6-10).

An HHA of the system during early developmental testing identified excessive levels of hydrogen chlo-

Fig. 6-10. Avenger Air Defense System. The Avenger enhances the Stinger missile with new capabilities such as shoot on the move, day/night operations, and multiple rapid sequential engagements. Avenger integrates the Stinger missile, a gyrostabilized turret, forward-looking infrared, laser rangefinder, identification of friend or foe, and a .50-caliber machine gun. It is operated either from the vehicle cab or remotely by a crew of two: the driver and the gunner.

ride gas in the HMMWV during certain Stinger missile firings, depending on the angle and direction of firing. The peak exposures of hydrogen chloride were in excess of 300 ppm (the militarily unique standard is now 100 ppm for 10 min). Thus, the materiel developer recommended and adopted engineering controls (such as exhaust deflectors, door and window seals, rigid doors, improved latches, and crew cab reinforcement) to eliminate the performance decrement resulting from acute exposures. The materiel developer also tested to verify that these engineering controls were effective.[21]

This system was redesigned to rectify HHA-identified hazards. It was used in Operation Desert Storm without report of adverse health effects to the soldiers who operated it.

Landing Craft Utility

The Landing Craft Utility-2000 (LCU-2000) class vessels are welded, steel-hulled marine vessels powered by two turbocharged diesel engines (Figure 6-11). The vessels are used for transporting rolling stock and general dry cargo on the ocean and coastal and inland waterways. They are also used for beaching and retraction on undeveloped and remote coastlines, assisting in discharging and backloading ships in a roll-on/roll-off or logistics-over-the-shore (LOTS) operation. The vessel is operated by a crew of 13: 11 enlisted personnel and 2 officers.

An HHA of the LCU-2000 identified a lack of ventilation for the control of toxic gases, fumes, organic vapor solvents, and particulate matter that were generated in the vessel's machine shop during degreasing, welding, soldering, sanding, and grinding operations. Thus, the HHA report provided detailed guidance for designing local exhaust ventilation. The materiel developer subsequently adopted these recommendations.[22]

M43A1 Protective Mask

The M43A1 Protective Mask is intended to be used by crew members operating rotary-winged aircraft and is designed to protect the face, eyes, and respira-

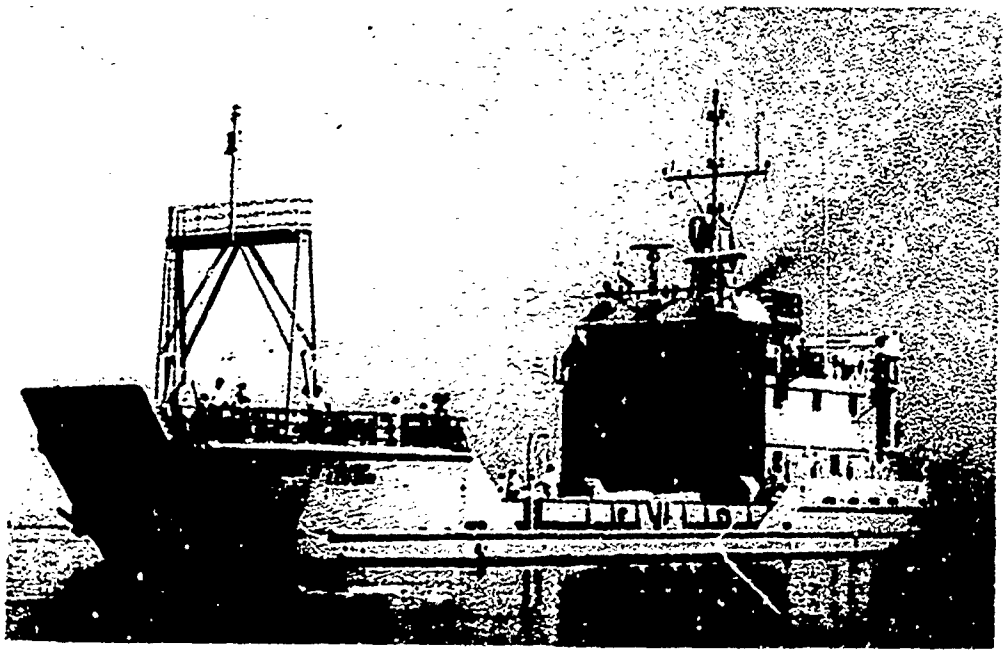

Fig. 6-11. Landing Craft Utility-2000 (LCU-2000). LCU-2000 ships are 174 ft long by 42 ft wide and displace 1,087 tons when fully loaded. Their speed is 12 knots and range is 5,000 miles. The main deck level houses the mess, sick bay, and recreation room. The crew quarters are on the second level; the pilot house is on top. A stern module contains the engine room and associated machinery.

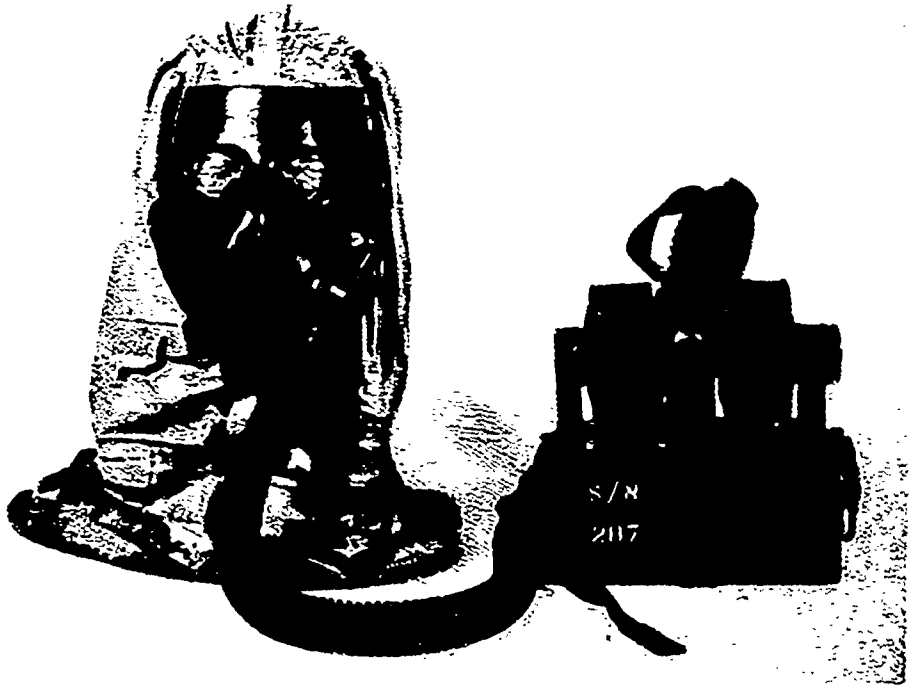

Fig. 6-12. M43A1 Aircraft Chemical-Biological (CB) Mask. The M43A1 mask consists of a form-fitting facepiece with spherical lenses fitted close to the eyes, an integrally attached CB hood and skull-type suspension system (fitted over and suspended from the head), an inhalation-air distribution assembly for regulating air flow to the mouth and nose, lenses and hood assembly, an exhalation valve assembly, an electronic microphone, and a portable motor-blower filter assembly for maintaining overpressure in the mask and hood.

tory system from field concentrations of chemical, biological and riot-control agents (Figure 6-12). The M43A1 was designed to improve the M43 Protective Mask by enhancing both minimum protection and nuclear-biological-chemical (NBC) survivability. In addition, replaceable prescription lenses were an added feature of the M43A1.

A planned product improvement for the mask is a change in the mask's formulation from bromobutyl/natural rubber to correct these deficiencies: (*a*) the faceblank cracks prematurely and (*b*) patch testing revealed a high percentage (1 in 200) of positive skin sensitization reactions among wearers. Civilian industry practice does not accept skin sensitization in excess of 1 in 10,000. Therefore, the HHA report recommended that none of the candidate formulations be used in fabricating rubber articles intended for use where repeated dermal contact is expected. The materiel developer accepted this recommendation and additional research is being done on the faceblank formulations.[23]

Oxygen Deficiency

When atmospheric oxygen is displaced from an enclosed or confined space, or when a system is operated at high altitudes, oxygen concentrations can be decreased below that which is commonly found in ambient air (21% by volume). Reduction of oxygen concentration to approximately 16% causes shortness of breath and impaired coordination and judgment. This condition, hypoxia, can cause visual, mental, and motor impairment and progress to unconsciousness and death.[3]

> When the oxygen level falls to 17 percent (129.2 mm Hg), the first sign of hypoxia, a deterioration of night vision, which is not noticeable until normal oxygen concentrations are restored, may occur. Physiological effects are increased breathing volume and accelerated heartbeat. At 14 percent to 16 percent (106.4–121.6 mm Hg) oxygen, physiological effects such as increased breathing volume, accelerated heartbeat, poor muscular coordination, rapid fatigue, and intermittent respiration may occur. Between 6 percent and

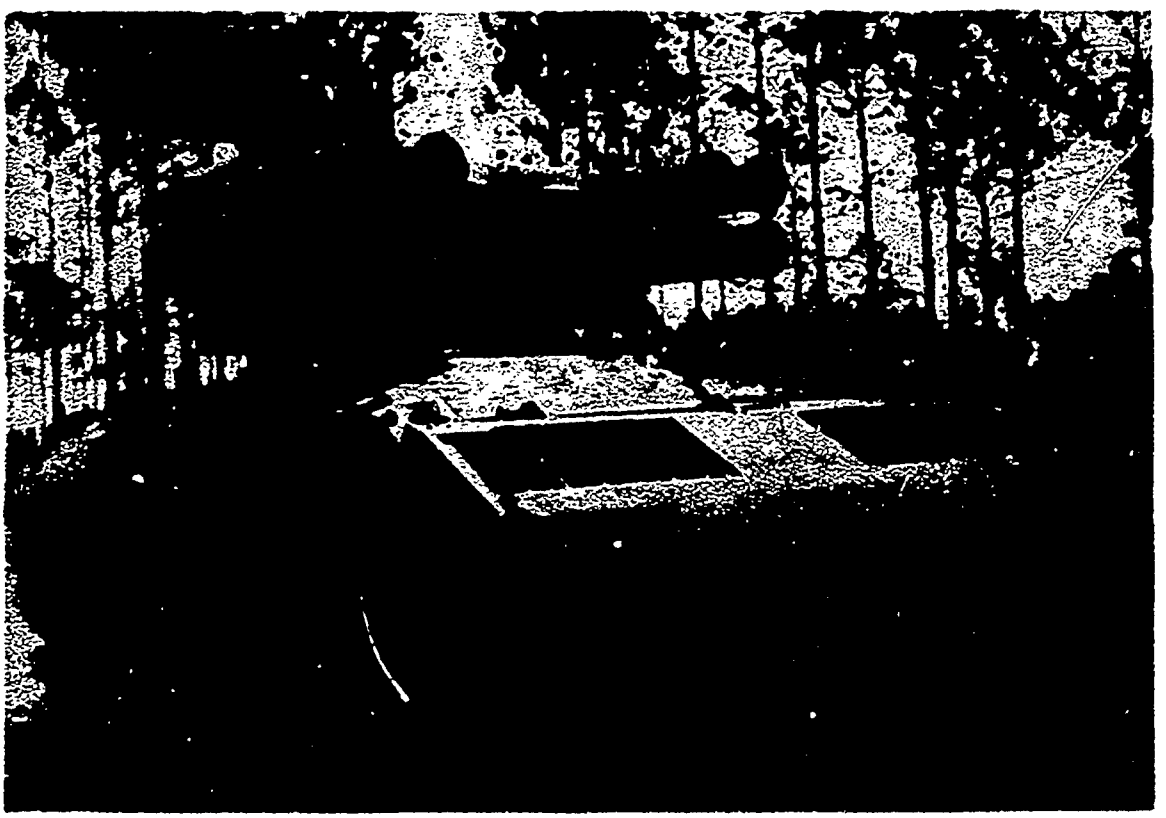

Fig. 6-13. Air Defense Antitank System. The ADATS is designed to operate autonomously or to use forward area air defense command, control, and intelligence data, during day or night, in obscurants, in adverse weather, and in battlefield environments where electronic and physical countermeasures are present. The system is operated by a crew of three: driver, gunner, and commander. This program was cancelled in the early 1990s both as a cost-saving measure and because the threat in Europe had changed.

10 percent (45.6–78 mm Hg), effects such as nausea, inability to perform, and loss of consciousness may occur. Less than 6 percent oxygen (45.6 mm Hg) results in spasmatic breathing, convulsive movements, and death in minutes.[24(p2)]

Air Defense Antitank System

The Air Defense Antitank System (ADATS) is the Line-of-Sight-Forward Heavy (LOS-F-H) component of the FAADS, designed to operate at or near the front lines (Figure 6-13). The system carries eight ready-to-fire laser beam-rider missiles designed specifically to counter low-level helicopters and fixed-wing aircraft. The crew of three (driver, gunner, and commander) use radar, forward-looking infrared (FLIR), and television sensors to detect, acquire, and identify targets. The missile fire unit is mounted on an armored tracked vehicle, the XM1069, which is a derivation of the M3A2 BFV chassis.

An initial HHA of the ADATS identified two shortcomings in the design of the vehicle's NBC air filtration system, one regarding the amount of filtered breathing air supplied to each crew member, and the other regarding the source of air during the backup mode of operation. These are typical deficiencies in tactical vehicles with ventilated facepieces; they are designed to deliver an average volume of 3 standard cubic feet (standardized for temperature and pressure) per minute of filtered air per person, which is less than the respiratory requirement for physically active crew members (eg, a loader or a gunner). The recycled air from within the vehicle was filtered, but no makeup air was introduced; typical carbon filtration does not filter or remove carbon monoxide. Thus, the HHA report provided the materiel developer with information concerning the minimum quantity of breathing air required, and proper design of the air source.[25]

Bradley Fighting Vehicle with Dual Shot Automatic Fire Extinguishing System

The BFV with Dual Shot Automatic Fire Extinguishing System (AFES) is a proposed product improvement to install two additional 5-pound halon fire extinguishers in the crew compartment. The system will provide the crew and squad compartments with the capability to detect and suppress slow-growth fires and two consecutive, explosive, hydrocarbon fires.

However, the HHA discovered two possible shortcomings of the system: oxygen deficiency and excessive levels of halon (the neat agent itself is toxic). The release of an excessive amount of Halon 1301 (which is the only halon fire-extinguishing agent allowed in crew compartments) into the BFV crew compartment will displace oxygen. Testing of the Dual Shot AFES determined that, under certain circumstances, the National Fire Protection Association's and the OTSG's recommended concentration for Halon 1301 was exceeded at several locations inside the vehicle (Table 6-4). Thus, the HHA report recommended that the combat and materiel developers adopt operating procedures to control exposures to both excessive Halon 1301 and reduced oxygen levels during AFES discharge. The report also recommended that warnings that such events are possible be included in the technical and training manuals for the BFV.[24]

Radiation Energy

Ionizing radiation—alpha and beta particles, gamma and X rays, and neutrons—is sufficiently energetic to strip electrons from molecules. This frees electrons and positive ions, which are then available to interact with other matter. *Nonionizing* radiation—emissions from the electromagnetic spectrum including ultraviolet, visible, infrared, and radio frequencies (including microwave)—has insufficient energy to ionize other molecules. Its biological effect is caused by exciting electrons to higher energy levels, thereby making molecules more chemically reactive. Lasers are a special category of nonionizing radiation technology; they amplify collimated electromagnetic radiation within the nonionizing spectrum.

Improved-Chemical Agent Monitor

The Improved-Chemical Agent Monitor (I-CAM) was originally developed for the United Kingdom's

TABLE 6-4

HALON 1301: RECOMMENDED CONCENTRATIONS AND PERSONNEL EXPOSURE TIMES

Concentration (% by vol)	Permitted Exposure Time (min)
≤7	15.0
7–10	1.0
10–15	0.5
>15	Prevent exposure

Adapted from US Army Environmental Hygiene Agency. *Health Hazard Assessment (HHA) for the A2 Bradley Fighting Vehicle System (BFVS) with Dual Shot Automatic Fire Extinguishing System (AFES).* Aberdeen Proving Ground, Md: USAEHA; 1990. Report 69-37-4776-90.

Fig. 6-14. Improved-Chemical Agent Monitor (I-CAM). The I-CAM detects vapors of chemical agents by sensing molecular ions of specific mobilities and uses timing and microprocessor techniques to reject common battlefield interferences. It consists of a drift tube, signal processor, molecular sieve, membrane, and expendable items such as batteries, confidence testers, and dust filters.

Ministry of Defense. It is a hand-held ion mobility spectrophotometer, used for chemical agent vapor detection (Figure 6-14). The I-CAM contains nickel 63, a beta-particle source, and is used to detect nerve and blister agents on personnel and equipment. The basic CAM has been improved, and maintenance procedures no longer require the removal of the assembly that contains the radioactive source.

The HHA report recommended that control procedures for radiation protection be developed and implemented for personnel who handle the nickel 63, and that specific radiation-safety instructions be incorporated in the I-CAM technical and training manuals.[26]

Enhanced M16A2 Rifle Optical Sight

A daylight optical sighting device is being considered for use on the Enhanced M16A2 Rifle and other weapons. The device will be used for battlefield observation up to 1,000 m, and will permit target engage-

ment by riflemen and gunners up to 600 m. The developer suggested that tritium be used in the sight as a light source and that the sight be optically hardened to protect the user from directed-energy weapons. The HHA of the optical sight recommended, however, that an alternate light source (such as promethium 147) be used because tritium emits a low-energy beta particle that can diffuse out of its encapsulating material and migrate to clean surfaces. It can then permeate the air of storage and use areas and be inhaled or percutaneously absorbed. Tritium also requires elaborate laboratory analytical detection and measurement techniques.[27]

Firefinder Mortar Locating Radar, Block II Program

The Firefinder Mortar Locating Radar (MLR) is a mobile phased-array radar system that is used to detect and locate high-angle-of-fire enemy weapons (mortars, short-range artillery, and rockets), to permit rapid engagement with counterfire. The system consists of an operations control group housed in an S-250 shelter, an antenna/transceiver group (ATG) and two MEP-112A 10-kW diesel generators (Figure 6-15). The Block II Program mounts all three of these subsystems on a pallet for placement on the cargo bed of a standard U.S. Army 5-ton truck to improve mobility, transportability, and emplacement and displacement time.

However, measurements of the radiofrequency (RF) radiation present during operation of the ATG demonstrated that the power density levels (see Chapter 15, Nonionizing Radiation, for a discussion of power density levels) may exceed the permissible exposure levels (PELs). Thus, an initial HHA recommended that personnel be prohibited from performing operations in front of the antenna while the system is radiating. In addition, the report recommended that RF radiation warning signs be placed so they are visible to personnel standing on the ground next to the antenna.[28] The materiel developer subsequently adopted these recommendations.

Fig. 6-15. Firefinder Mortar Locating Radar (MLR), Block II Program. The MLR is deployed close to the forward line of troops with direct support artillery battalions. In fiscal year 1990, the army approved the system's reconfiguration so it could be carried by 1¼-ton capacity HMMWVs (High-Mobility, Multipurpose, Wheeled Vehicle). Future improvements will also include eliminating the S-250 operations shelter, reduced emplacement time, faster access to data, increased program memory and digital map storage, improved throughput and processing, remote operation capability up to 100 m, and better probability of detecting the location of enemy weapons.

Fig. 6-16. Armed OH-58D Kiowa Warrior Scout Helicopter. The OH-58D uses a new drive train consisting of a four-bladed rotor, 650-hp engine and compatible transmission and tail rotor systems. Beginning in fiscal year 1991, the armed version was equipped with air-to-air Stinger missiles. An air-to-ground weapons suite will arm the aircraft with Hellfire missiles, Hydra 70 2.75-in. rockets, and/or a .50-caliber machine gun. The aircraft is operated by a crew of two, has a maximum gross weight of 5,500 pounds, and a maximum level speed of 118 knots.

Observation Helicopter-58D Kiowa Warrior Scout

The OH-58D Kiowa Warrior Scout is an improved, close-combat, aerial-reconnaissance, intelligence-gathering, target-acquisition and -designation surveillance system (Figure 6-16). It is assigned as an aeroscout helicopter for attack helicopter companies and air cavalry companies, and as an aerial observation helicopter for field artillery support sections. A mast-mounted sight (MMS) above the rotor contains a laser rangefinder and target designator (LRF/D).

An HHA of the optical radiation hazards associated with the LRF/D determined that it emits optical radiation in excess of current exposure limits for this specific laser (see Chapter 15). Thus, the HHA report recommended that the developer restrict unprotected personnel from entering the laser beam within 23 km of the laser, and require ground personnel (such as maintenance, test, and training personnel) to use laser eye protection.[5,6]

Shock

AR 40-10 defines shock as the "delivery of a mechanical impulse or impact to an individual transmitted from the acceleration or deceleration of a medium with which he has contact."[1] It is not to be confused with either physiological shock or electrical shock. The opening forces of a parachute harness and the forces delivered to the body as a result of weapon recoil are examples of this kind of shock.

Tactical Assault Personnel Parachute

The Tactical Assault Personnel Parachute (TAPP) is being developed for use in training and combat airborne operations. The design allows for a lower rate of descent to reduce the potential for landing injuries. With the TAPP, the combat jump altitude will be as low as 300 ft above ground level (AGL) and the training jump altitude will be as low as 800 ft AGL.

An initial HHA of the TAPP required the materiel developer to conduct tests to assess the potential health hazards of musculoskeletal trauma resulting from excessive opening forces and impact velocity, and set the criteria for data collection. The HHA recommended that both the current and the improved paratrooper helmets be included in the TAPP test program to evaluate the effect of helmet mass on neck loads during opening shock, and the effect of crushable foam on

reducing deceleration of the head during parachute landing falls.[31]

Temperature Extremes

The human health effects associated with high or low temperatures, possibly in conjunction with high humidity, can be exacerbated by a materiel system. Heat stress can cause heat disorders such as heatstroke and hyperthermia. Cold-induced disorders include frostbite and hypothermia.[3]

HHAs have addressed the hazards of temperature extremes and humidity associated with the use of several materiel systems. The potential for heat stress is inherent in the use of almost any protective overgarment, particularly a totally encapsulating ensemble such as the Self-Contained Toxic Environment Protective Outfit-Interim (STEPO-I). Similarly, the potential for cold injury is inherent in materiel systems that operate in cold ambient temperatures, such as the Landing Craft Mechanized-8 (LCM-8).

Self-Contained Toxic Environment Protective Outfit-Interim

The STEPO-I is used to provide respiratory and percutaneous protection for depot personnel working in highly toxic or oxygen-deficient environments while they process, handle, store, transport, dispose of, or decontaminate chemical agents. Two versions of the STEPO-I have been considered to replace the M-3 Toxicological Agent Protective (TAP) suit. Both consist of a fully encapsulating, impermeable, butyl-rubber–coated, nylon suit fitted with breathing and cooling systems.

An HHA report addressed the heat-stress concerns associated with the suit and recommended the preferential use of one suit and an ice vest when ambient temperatures exceed 80°F. The report also recommended that the materiel developer collect test data to support development of *safe stay-wear times* for both versions of the STEPO-I.[32] The concept of safe stay-wear time seeks to strike a balance between protecting the wearer from both exposure to chemical agents and heat stress. It is generally defined as the length of time that the suit can be worn to provide adequate protection from chemical contamination without compromising the wearer's health due to heat stress.

Landing Craft Mechanized-8 Mod 1, Service Life Extension Program

The Landing Craft Mechanized-8 Mod 1, Service Life Extension Program (LCM-8, SLEP) is a U.S. Navy-

Fig. 6-17. Landing Craft Mechanized-8 (LCM-8) Mod 1, Service Life Extension Program (SLEP). The LCM-8, Mod 1, SLEP is a product-improvement program intended to restore the mission capability and supportability characteristics of the existing fleet and extend its service life by 20 years. The primary modification is the replacement of the old twin Detroit Diesel 6-71 engines with new 12V-71 diesel engines and associated hardware. The army has a fleet of approximately 96 of these vessels assigned to Transportation Medium Boat Companies.

designed, welded-steel, twin-diesel–powered watercraft. It is approximately 73 ft long and capable of carrying 60 tons (Figure 6-17). The vessel is designed to provide water transport to cargo, troops, and vehicles during LOTS, fixed port, shore-to-shore, inland waterway, and amphibious operations. A pilothouse is located aft of the cargo well and the bow is fitted with a hydraulically controlled ramp. The LCM-8 is expected to operate in ambient temperatures as low as -25°F.

An HHA of the LCM-8 identified the potential for cold stress due to the lack of heating to occupied spaces on the vessel. The HHA report recommended that the materiel developer also use engine-cooling water as a source of heat in occupied spaces. The materiel developer modified the LCM-8s used in Alaska to use engine-cooling water as a source of heat in the pilothouse.[33]

Physical Trauma

Trauma to the eyes or body can occur on impact with sharp or blunt objects, and musculoskeletal trauma can occur when heavy objects such as boxes of ammunition are lifted. PPE such as chemical protective masks, eyewear, or helmets are often assessed for their ability to preclude traumatic injuries.[3]

M43A1 Protective Mask

The M43A1 Protective Mask (see Figure 6-12) is designed to protect the face, eyes, and respiratory system from field concentrations of chemical, biological, and riot-control agents. An HHA of the mask was completed during its development (as an improvement to the XM43 Protective Mask). One of the health concerns identified was the effectiveness of the lenses

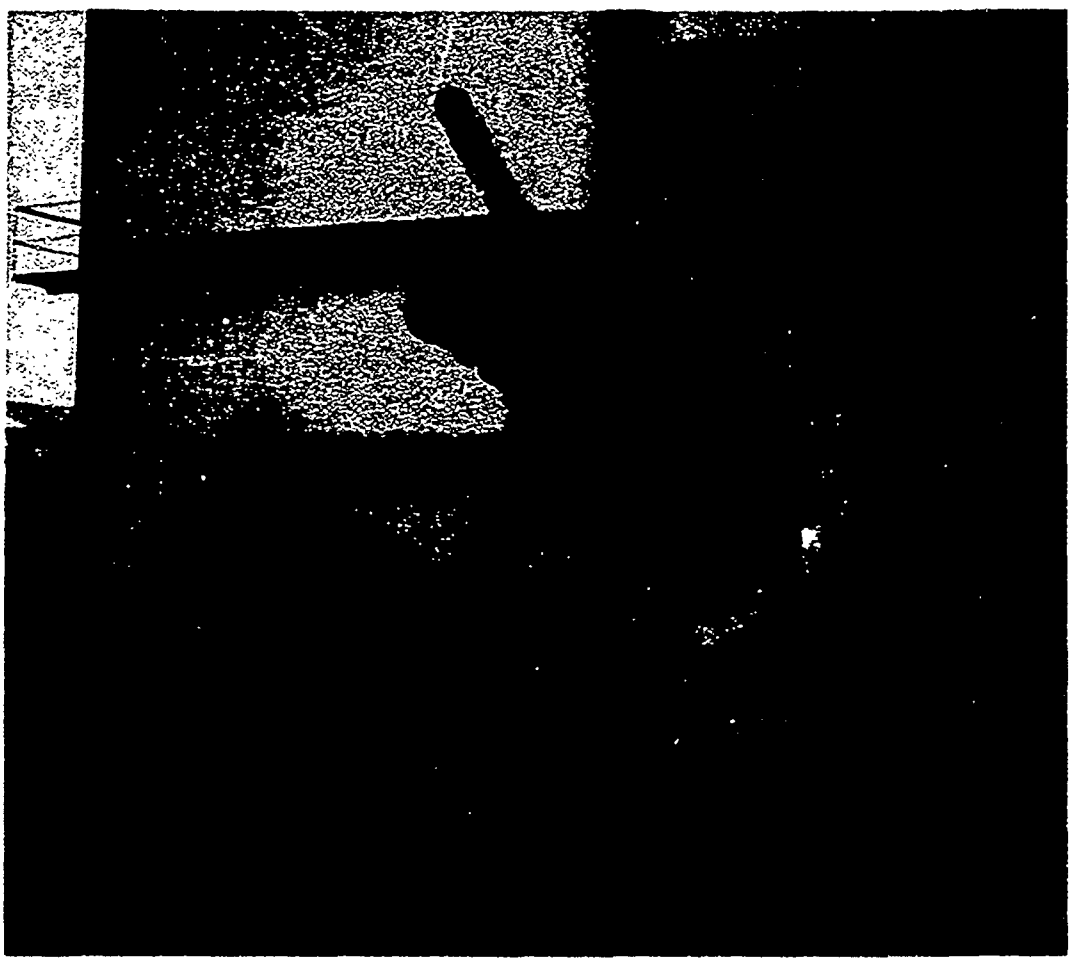

Fig. 6-18. M163A2 Self-Propelled 20-mm Vulcan Air Defense System (VADS). Major components of the M163A2 VADS include the M168 20-mm cannon, M61A1 director sight, AN/PVS-2 range-only radar, and the M741 chassis. It carries 1,000 ready 20-mm rounds in a linkless feed system.

in providing adequate eye protection. The HHA determined that the lenses afford the same degree of protection against eye injuries that industrial safety eyewear (which meets current national standards) provides from blunt- and sharp-object penetration.[34]

M163A2 Self-Propelled 20-mm Vulcan Air Defense System

The M163A2 Self-Propelled 20-mm Vulcan Air Defense System (VADS) is a lightweight, lightly armored gun system on a full-tracked vehicle, designed to provide air defense against low-altitude threats in forward combat areas (Figure 6-18). It may also be used against stationary or moving ground targets such as personnel, trucks, and lightly armored vehicles. The system is highly mobile and is capable of high-speed operation on improved roads, cross-country travel over rough terrain, and amphibious operation on streams and small lakes. The M168 20-mm cannon is capable of delivering selected rates of fire of 1,000 or 3,000 rounds per minute.

An HHA of the VADS identified a potential for musculoskeletal trauma when crew members lifted heavy boxes of spare ammunition onto the vehicular platform. The HHA report recommended specific ergonomic procedures for lowering the platform to minimize the likelihood of musculoskeletal trauma. The report also recommended that the materiel developer coordinate with the U.S. Army Human Engineering Laboratory for additional work practices and engineering-design modifications to mitigate the lifting hazard.[35]

Vibration

Segmental and whole-body vibration can occur "by contact of a mechanical oscillating surface with the human body."[3] Whole-body vibrations are transmitted through the feet of a standing person, the buttocks of a seated person, or the supported area of a reclining person, and are found in vehicles, vibrating buildings, and in the vicinity of vibrating machinery. Body segments including the head or limbs can also be

Fig. 6-19. Fast Attack Vehicle (FAV). The FAV is similar to a dune buggy and is used by the U.S. Army's Special Forces units. Although it has limited cargo-carrying and fuel capacity, it was used during Operation Desert Storm and reportedly carried the first coalition forces into Kuwait City.

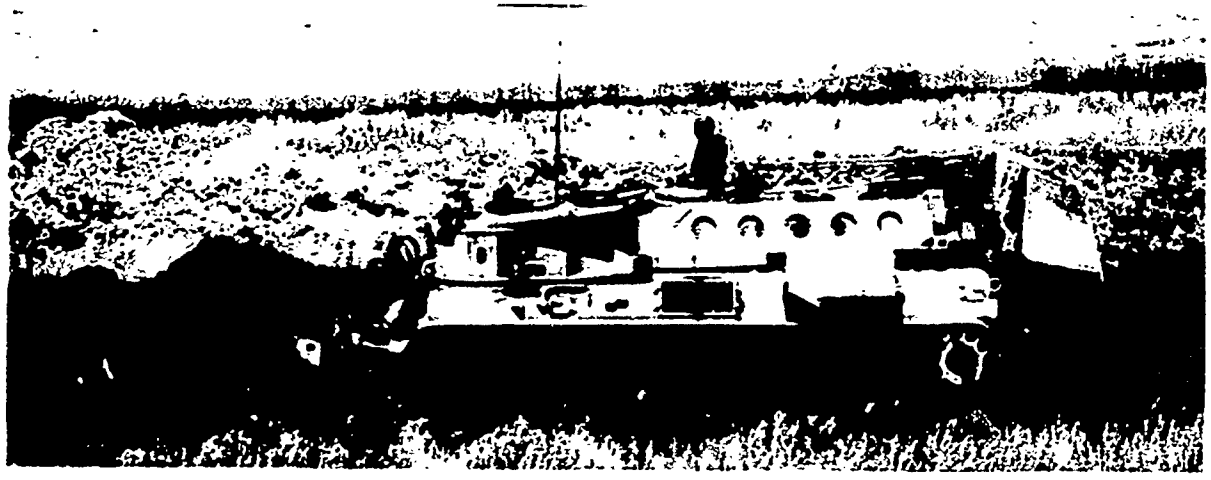

Fig. 6-20. Counterobstacle Vehicle (COV). The prototype COV pictured here is a highly mobile vehicle capable of clearing and creating major obstacles and emplacements. It is equipped with a combination bulldozer/mineplow and two telescopic arms. The arms are normally used with buckets, but can also accept a hammer, auger, lifting hook, grapple, and other attachments. These attachments enable it to move earth, breach minefields, knock down obstacles, dig defilade positions for armored vehicles, and excavate antitank ditches.

affected by vibrations from handles, pedals, headrests, or a variety of hand-held power tools and appliances.

Fast Attack Vehicle

The Fast Attack Vehicle (FAV) is a maneuverable, lightweight, all-terrain vehicle capable of high-speed, cross-country travel. The FAV serves as a weapons or communications platform for antiarmor, reconnaissance, deep attack, and other missions (Figure 6-19).

During development testing, 50% of the test personnel reported kidney and back injuries that were attributed to excessive levels of whole-body vibration. These injuries were apparently due to inadequate isolation of the vibration through the seats and inadequate shock absorbancy in the vehicles' suspension system. Thus, the HHA recommended that these deficiencies be corrected and that the FAV operators be placed in a medical surveillance program.[16]

Counterobstacle Vehicle

The Counterobstacle Vehicle (COV) is a highly mobile, armored vehicle equipped with a combination bulldozer and mine plow and telescopic arms that are capable of accepting several pieces of modified construction equipment. The original vehicle design was based on the hull and chassis of the M88A1 Recovery Vehicle and was considered as a replacement for the M728 Combat Engineer Vehicle and the M9 Armored Combat Earthmover. The COV will support heavy divisions in the performance of mobility, countermobility, and survivability tasks (Figure 6-20).

An assessment of whole-body test data resulted in recommendations to isolate the crew members' seats from the main vehicle frame by modifying the seats, seat cushions, or both. In lieu of accepting this recommendation, the HHA report advised that, for primary and secondary road surfaces, crew members be restricted to exposure to whole-body vibration for no more than 6.0 continuous hours in any 24-hour period.[17]

The International Standard Organization's (ISO) standards for whole-body vibration are specific to the high frequencies found in heavy equipment. The low frequencies found in wheeled vehicles traversing rough terrain are, however, not considered by this ISO standard. The only definitive evidence of physiological effects of whole-body vibration is the presence of microscopic hematuria. The OTSG has identified the need for a militarily unique whole-body vibration standard, and MRDC is currently conducting research to this end.

SUMMARY

The HHA Program is one of the most militarily relevant applications of occupational health within the preventive medicine arena. Since the formalization of the program in 1983, nearly every weapon and support system developed or procured by the army to assist the soldier in the field has been reviewed for health hazards by AMEDD.

Health hazards are identified, evaluated, and eliminated or controlled through a systematic review and analysis process as materiel progresses through the research, development, and acquisition process. Nine general categories of health hazard exposures have been defined: acoustical energy, biological substances, chemical substances, oxygen deficiency, radiation energy, shock (mechanical impulse or impact), temperature extremes, physical trauma, and vibration.

HHA is one of the principal domains within the army's MANPRINT Program. The health risk to the soldier (as an operator or maintainer of materiel) is evaluated and reduced by a multidisciplinary medical team including industrial hygienists, audiologists, physicists, toxicologists, engineers, biologists, chemists, and occupational medicine physicians.

The HHA Program has paid big dividends to the army's equipment modernization program, but these dividends are difficult to quantify—as they are with most successful preventive medicine programs. Suffice it to say that the recipient of the dividend is the soldier. The one who uses the equipment has every right to expect that the health risks from using military hardware will be reduced to the lowest feasible level. Recent conflicts such as Operation Desert Storm had few or no reported adverse health effects from the use of materiel, which testifies to the success of this AMEDD initiative.

REFERENCES

1. Gaydos JC. A historical view of occupational health for the soldier. *Medical Bulletin of the US Army Medical Department*. 1988;2:4–6. PB 8-88-2.

2. US Department of Defense. *Major and Non-Major Defense Acquisition Programs*. Washington, DC: DoD; 1983. DoD Directive 5000.1.

3. US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process*. Washington, DC: DA; 1983. Army Regulation 40-10.

4. Ranadive MV. Preface. *Medical Bulletin of the US Army Medical Department*. 1988;2:3. PB 8-88-2.

5. Stuhmiller JH, Phillips YY, Richmond DR. The physics and mechanisms of primary blast injury. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries*. Part 1, Vol 5. *Textbook of Military Medicine*. Washington, DC: DA Office of The Surgeon General and Center of Excellence in Military Medical Research and Education; 1991: 242.

6. Davis B. *The Civil War: Strange & Fascinating Facts*. New York: Fairfax Press; 1982.

7. Cooper B. *The Ironclads of Cambrai*. Toronto: Ryerson Press; 1967.

8. Hatch TF. The armored force medical research laboratory in WWII. *Medical Bulletin*. 1985;42(1):22–26.

9. Bean WB. The ecology of the soldier in World War II. *Medical Bulletin*. 1985;42:20–25.

10. Rowden SE, McIntosh RM. The health hazard assessment program: Occupational health for the soldier in the field. *Medical Bulletin of the US Army Medical Department*. 1988;2:7–10. PB 8-88-2.

11. US Department of the Army. *Manpower and Personnel Integration (MANPRINT) in Materiel Acquisition Process.* Washington, DC: DA; 1987. Army Regulation 602-2.

12. Leibrecht BC. Health hazard assessment: How the system works. *MANPRINT Bulletin.* 1987;2(4):1–3.

13. Leibrecht BC. Research supporting health hazard assessment. *MANPRINT Bulletin.* 1989;3(4):4–5.

14. US Department of the Army. *Systems Acquisition Policy and Procedures.* Washington, DC: DA; 1988. Army Regulation 70-1.

15. US Army Environmental Hygiene Agency. *Health Hazard Assessment (HHA) Report on the Lighter, Air Cushion Vehicle, 30 Ton Capacity (LACV-30).* Aberdeen Proving Ground, Md: USAEHA; 1983. Report 69-37-1109-83.

16. US Army Environmental Hygiene Agency. *Health Hazard Assessment (HHA), Noise Hazards Associated with Operation of the Bradley Fighting Vehicle (BFV), M2 and M3.* Aberdeen Proving Ground, Md: USAEHA; 1982. Report 69-37-1107-83.

17. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the 120-mm Battalion Mortar System.* Aberdeen Proving Ground, Md: USAEHA; 1991. Report 69-37-4735-91.

18. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Howitzer Improvement Program (HIP).* Aberdeen Proving Ground, Md: USAEHA; 1988. Report 69-37-4563-88.

19. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Composting Toilet and Aerated Vault Technology for Army Remote Sites and Mobilization Applications.* Aberdeen Proving Ground, Md: USAEHA; 1984. Report 69-37-1110-84.

20. US Army Environmental Hygiene Agency. *Health Hazard Analysis for the Resuscitation Fluids Production System (REFLUPS).* 2nd endorsement. Memorandum to US Army Medical Materiel Development Activity. SGRD-UMA-T, 3 November 1986.

21. US Army Environmental Hygiene Agency. *MANPRINT Assessment for the Pedestal Mounted Stinger (PMS).* 2nd endorsement. Memorandum to Office of the Deputy Chief of Staff for Personnel. DAPE-MRA, 24 August 1989.

22. US Army Environmental Hygiene Agency. *Updated Health Hazard Assessment Report (RCS MED 388) on the Landing Craft Utility (LCU-2000) Nondevelopmental Item.* Aberdeen Proving Ground, Md: USAEHA; 1989. Report 69-37-4772-89.

23. US Army Environmental Hygiene Agency. *Updated Health Hazard Assessment for the XM43 P3I Mask System.* 3rd endorsement. Memorandum to US Army Chemical Research, Development and Engineering Center. SMCCR-HV, 8 August 1990.

24. US Army Environmental Hygiene Agency, Health Hazard Assessment Office. *Health Hazard Assessment (HHA) for the A2 Bradley Fighting Vehicle System (BFVS) with Dual Shot Automatic Fire Extinguishing System (AFES).* Aberdeen Proving Ground, Md: USAEHA; 1990. Report 69-37-4776-90.

25. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Forward Area Air Defense Line of Sight-Forward-Heavy System.* Aberdeen Proving Ground, Md: USAEHA; 1990. Report 69-37-4731-90.

26. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Improved Chemical Agent Monitor (I-CAM).* Aberdeen Proving Ground, Md: USAEHA; 1990. Report 69-37-4525-90.

27. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Enhanced M16A2 Rifle Optical Sight.* Aberdeen Proving Ground, Md: USAEHA; 1988. Report 69-37-4629-91.

28. US Army Environmental Hygiene Agency. *Initial Health Hazard Assessment Report (RCS MED 388) on the AN/TPQ-36 Mortar Locating Radar, Block II Program.* Aberdeen Proving Ground, Md: USAEHA; 1988. Report 69-37-4693-88.

29. US Army Environmental Hygiene Agency, Laser Microwave Division. *OH-58D Helicopter Laser in the Day/Night Mast Mounted Sight*. Aberdeen Proving Ground,: USAEHA; 1987. Nonionizing Radiation Protection Study 25-42-0360-87. Classified, Confidential.

30. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Armed OH-58D Kiowa Warrior Scout Helicopter*. Aberdeen Proving Ground, Md: USAEHA; 1991. Report 69-37-4551-91.

31. US Army Environmental Hygiene Agency. *Request for Health Hazard Assessment on the Tactical Assault Personnel Parachute (TAPP)*. 3rd endorsement. Memorandum to US Army Troop Support Command, Natick Research, Development and Engineering Center. 27 September 1990.

32. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Self-Contained Toxic Environment Protective Outfit-Interin (STEPO-I)*. Aberdeen Proving Ground, Md: USAEHA; 1991. Report 69-37-4711-91.

33. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Landing Craft Mechanized-8 (LCM-8) Mod-1, Service Life Extension Program*. Aberdeen Proving Ground, Md: USAEHA; 1988. Report 69-37-4706-88.

34. US Army Environmental Hygiene Agency. *Health Hazard Assessment of the XM43 CB Protective Mask*. 1st endorsement. Memorandum to Department of the Army, Office of The Surgeon General. 15 March 1985.

35. US Army Environmental Hygiene Agency. *Health Hazard Assessment Report (RCS MED 388) on the Vulcan Wheeled Carrier*. Aberdeen Proving Ground, Md: USAEHA; 1986. Report 69-37-4582-86.

36. US Army Environmental Hygiene Agency. *Initial Health Hazard Assessment Report (RCS MED 388) on the Fast Attack Vehicle (FAV)*. Aberdeen Proving Ground, Md: USAEHA; 1985. Report 69-37-4532-85.

37. US Army Environmental Hygiene Agency. *Initial Health Hazard Assessment Report (RCS MED 388) on the Counterobstacle Vehicle (COV)*. Aberdeen Proving Ground, Md: USAEHA; 1988. Report 69-37-4558-88.

THEATER HIGH ALTITUDE AREA DEFENSE SYSTEM: INITIAL HEALTH HAZARD ASSESSMENT REPORT

The following HHA report is reproduced in its entirety so that interested readers can more fully appreciate the depth and scope of these investigations. This particular report was selected because (*a*) it demonstrates that one weapon system can expose personnel to several complex health hazards and (*b*) this emerging system could provide air defense support to U.S. military and civilian personnel well into the future.

INITIAL HEALTH HAZARD ASSESSMENT REPORT (RCS MED 388) ON THE THEATER HIGH ALTITUDE AREA DEFENSE SYSTEM 69-37-4847-91 JULY 1991

1. References. A list of references used in this initial health hazard assessment report (IHHAR) is contained in Appendix A. [The references were attached to the original document in the form of an appendix.—*Eds.*]

2. Summary. The Theater High Altitude Area Defense System (THAAD) is an area defense system designed to defeat tactical ballistic missile (TBM) threats directed against military forces and critical assets (e.g., air fields and command centers) and theater strategic targets (e.g., utilities and population, industrial, and government centers). The principal health concerns addressed in this IHHAR are:

a. Chemical substances.

b. Temperature extremes.

c. Oxygen deficiency.

d. Radiofrequency radiation.

e. Acoustical energy.

Assessments and recommendations concerning these issues are addressed in paragraphs 5 and 6, respectively. Additional health hazards may be identified in the future as more information and test data on the THAAD become available. Such information must be provided to support completion of a final health hazard assessment report (HHAR).

3. Background.

a. The THAAD will be a ground launched TBM defense missile system capable of endoatmospheric and exoatmospheric intercepts. The system will complement existing and future air defense systems by extending the TBM defense battle space and coverage beyond that of the PATRIOT Missile System. The THAAD will be fully interoperable with existing air defense forces and organizations. The system is currently in the demonstration/validation phase of a streamlined acquisition process. An Army Systems Acquisition Review Council (ASARC) Milestone Decision Review (MDR) I is scheduled for October 1991. The THAAD program includes plans for the fielding of prototype systems (i.e., a provisional THAAD battalion with two THAAD batteries) to support operational evaluation. The objective of this User Operational Evaluation System (UOES) is to have a deployable national asset with a limited capability to defeat a TBM threat by 4QFY95 (references 1 and 2).

b. The THAAD system design and configuration emphasizes worldwide deployability. The system will be transportable by land, sea, and air (C-130 aircraft) without disassembly of any major component. Assembly at the theater of operations will be limited to routine emplacement activities (e.g., stabilization, erection, cable connection and alignment) (reference 3). The major components of the UOES and objective THAAD systems are similar, but the proposed organization of each is somewhat different due to a limited number of launchers and missiles during the early stages of system development. A UOES THAAD Battery will have one Tactical Operations Center (TOC), one Theater Missile Defense-Ground Based Radar (TMD-GBR), and 12 missile launchers. The objective THAAD Battery will have two TOCs, two TMD-GBRs, and 18 missile launchers. Each type of battery will also have its associated maintenance/support equipment. Air defense artillery personnel, similar to those assigned to the PATRIOT Missile System, will operate and maintain the THAAD.

(1) Tactical Operations Center. The TOC will be housed in a Standardized Integrated Command Post System (SICPS) rigid walled shelter. The shelter is mounted on a modified M1097 High Mobility Multipurpose Wheeled Vehicle (HMMWV). Standard SICPS features include a 5KW generator, 9000 BTU/hr air conditioner, collective chemical/biological

protection, equipment racks, power and signal import/export panels, intercom, and operator seats. The TOC will use common hardware/software being developed by the Army Tactical Command and Control System (ATCCS) and be interoperable with other Army/DoD and allied Air Defense (AD)/C^3I systems (references 3 and 4).

(2) Theater Missile Defense-Ground Based Radar. The TMD-GBR is the primary THAAD sensor/radar system which performs target detection, acquisition, classification, identification, engagement and destruction, and kill assessment functions. It is a phased array radar system which will also provide cueing support to other AD systems (e.g., PATRIOT) and counterfire support by communicating estimated launch points to the command and control network to enhance the detection and destruction of enemy launch facilities (reference 5). The UOES THAAD TMD-GBR will be based upon travelling wave tube (TWT) technology and the objective TMD-GBR will use solid-state technology. Therefore, the radar support equipment used by each of the systems will differ slightly. The UOES system will consist of the radar antenna assembly, array cooling unit (650,000 BTU/hr), a prime power unit (three 500 KW generators), a radar control van/shelter, a high voltage power supply, radar electronics unit, and prime movers. The objective THAAD TMD-GBR will not require a high voltage power supply and radar electronics unit (references 5 and 6).

(3) Missile launchers. Missile launchers will be truck or trailer mounted using standard Army 5-ton trucks. Each hit-to-kill missile will be a certified round contained in a storage/shipping/launch canister (e.g., PATRIOT). The number of missiles/canisters per launcher will be determined by the capacity of the prime mover and C-130 transportability requirements. The missile will be liquid fueled (dinitrogen tetroxide oxidizer and hydrazine propellant) with pressurized/sealed tanks. The launcher will be operated remotely by data-link from the TOC.

c. No previous HHARs have been completed on the THAAD. However, HHARs have been completed on a similar missile system (i.e., Kinetic Energy Anti-Satellite System) and on items which will be used by the THAAD System. These HHARs were reviewed for lessons learned and possible application to the THAAD System (reference 7). Personnel from the U.S. Army Environmental Hygiene Agency's (USAEHA) Health Hazard Assessment Office attended a THAAD MANPRINT Joint Working Group meeting in order to provide health hazard assessment support to the THAAD Program and obtain information on the system (reference 6).

4. Identification of Health Hazard Issues. The following potential health hazards have been identified after reviewing the limited information currently available on the THAAD System. Additional health hazards may be identified as the development of the THAAD System continues and future HHARs are completed on the system.

a. Chemical substances.

(1) Diesel engine exhaust.

(2) Rocket motor propellant and oxidizer.

(3) Fire extinguishing agents.

(4) Nuclear, biological, and chemical (NBC) agents.

(5) Off gassing.

b. Temperature extremes.

(1) Heat stress.

(2) Cold stress.

c. Oxygen deficiency.

d. Radiofrequency radiation.

e. Acoustical energy.

(1) Steady-state noise.

(2) Impulse noise.

5. Assessment of Health Hazard Issues.

a. Chemical substances.

(1) Diesel engine exhaust.

(a) Combustion products from diesel engines include carbon monoxide (CO), oxides of nitrogen (NO_x), formaldehyde, acrolein, and sulfur dioxide (SO_2). Carbon monoxide is a chemical asphyxiant which decreases the ability of the blood to carry oxygen to body tissues. High concentrations of CO may be rapidly fatal without producing significant

warning properties (reference 8). Oxides of nitrogen are deep lung irritants which produce cough, shortness of breath, and pulmonary edema. Formaldehyde, acrolein, and sulfur dioxide cause skin, eye, and mucous membrane irritation (references 8 and 9). It is also important to note that the National Institute for Occupational Safety and Health (NIOSH) has proposed that diesel engine exhaust and formaldehyde are potentially carcinogenic materials. The NIOSH recommends that personnel exposed to diesel fuel exhaust be informed of this potential hazard and that exposures be reduced to the lowest level feasible (reference 10).

(b) Vehicle engine exhaust. The current host vehicle for the THAAD SICPS, the HMMWV, is powered by a 6.2 liter diesel engine and has a standard exhaust system. Vehicle exhaust concentrations inside the cab are not a problem if the exhaust system is visually inspected as part of the vehicle maintenance program and the integrity of the three piece exhaust system is not compromised. TM-9-2320-298-20 addresses exhaust system maintenance (reference 11). Personnel in the shelters should not be at risk from engine combustion products if vehicle engines are not running when the THAAD system is operational. Design requirements for exhaust systems are contained in MIL-HDBK-759A (reference 12).

(c) Generator engine exhaust. Diesel-fueled generators will provide power to occupied shelters and control vans for the electronics, heat, and air conditioning and other THAAD components. Combustion products from the generators should not be a concern because the generators will be positioned at a distance from the vehicle and the shelters (reference 6). However, a detailed system description and use scenario is required to fully assess this hazard.

(2) Rocket motor propellant and oxidizer.

(a) The THAAD missile is liquid fueled. The propellant is hydrazine, N_2H_4. The oxidizer is dinitrogen tetroxide, N_2O_4 (reference 6). The missiles will be preloaded with the propellant and oxidizer in pressurized sealed tanks (reference 1).

(b) There is a potential for serious exposure to THAAD missile maintenance and operational personnel, and any personnel involved in packaging, storage, handling, and transport of fueled missiles should a leak develop in the pressurized propellant or oxidizer tanks. Both hydrazine and dinitrogen tetroxide are extremely toxic substances (references 13, 14 and 15).

(c) Hydrazine is a suspected human carcinogen and a skin contact hazard. Hydrazine vapors are highly irritating to the eyes, upper respiratory tract, and skin. The liquid is corrosive, producing penetrating burns and severe dermatitis; permanent eye damage and blindness may occur if splashed in the eyes (reference 13).

(d) Dinitrogen tetroxide is a dimer of nitrogen dioxide. When nitrogen dioxide is under pressure, it is converted into dinitrogen tetroxide. When pressure is released the gas released is nitrogen dioxide (references 16 and 17). Nitrogen dioxide is highly toxic. Nitrogen dioxide is a pulmonary irritant. A relatively minor leak could expose personnel to debilitating or possibly fatal levels of NO. Exposure to 100 parts per million (ppm) for one hour can produce debilitating dyspnea or pulmonary edema (reference 17). Exposures of this level could occur if a minor leak developed in the system. However, under normal conditions the rocket propellant and oxidizer are in sealed tanks inside the missile which is stored in a sealed canister.

(e) The current Army adopted maximum exposure levels for hydrazine and nitrogen dioxide are:

Hydrazine		Nitrogen dioxide	
10 min	30 ppm	10 min	5 ppm
30 min	20 ppm	30 min	3 ppm
60 min	2 ppm	60 min	1 ppm
24 hr	0.08 ppm	2 hr	0.5 ppm
		4 hr	0.25 ppm
		8 hr	0.12 ppm
		24 hr	0.1 ppm

These are exposure levels at which Army personnel can continue to function in a military unique operation or emergency situation and be unlikely to suffer irreversible effects. However, a temporary performance decrement may result. Therefore, these levels must not be used as design standards by materiel developers. Exposures to chemical substances should be controlled to the lowest level feasible in accordance with MIL-STD-1472D (reference 18).

(3) Fire extinguishing agents.

(a) It is expected that an automatic fire extinguishing system (AFES) will be provided for the THAAD to protect both the safety and health of the soldier and the Army's investment in equipment. The documents reviewed for this

IHHAR do not address an AFES (references 3, 6, and 19). Therefore, an assessment of the health hazards associated with the AFES cannot be completed at this time.

(b) If an AFES is selected for use in the THAAD and a halon fire extinguishing agent is used, Halon 1301 is the only Office of The Surgeon General (OTSG)-approved halon fire extinguishing agent for use in occupied enclosed spaces (references 20 and 21). Halon 1301 was selected as the most satisfactory and least toxic among 97 agents tested for military vehicle applications (reference 21). Relatively small concentrations of Halon 1301 are required to extinguish fires by inhibiting the chemical reaction of fuel and oxygen (reference 22). The OTSG policy regarding Halon 1301 specifies that the average atmospheric concentration will not exceed 7 percent by volume, and the exposure time for personnel will not exceed 15 minutes. The policy assumes appropriate engineering design to sense the fire and deliver the agent, and to extinguish the fire promptly so that personnel exposures to the Halon 1301 and its toxic pyrolysis products are minimized. Halon 1301 total flooding system design standards may be found in reference 22.

(4) Nuclear, biological, and chemical agents. The SICPS, which houses the TOC, is normally equipped with modular collective protection equipment (MCPE) (reference 4). It is expected that MCPE will be provided for other THAADS shelters (e.g., radar control van) to protect the health of the soldier in an NBC environment. Test data which verify the effectiveness of MCPE are not available. Therefore, MCPE cannot be assessed at this time. Test data must be provided to support a final HHAR on the THAAD MCPE.

(5) Off gassing. The THAAD is required to be capable of storage and operation in extreme temperatures (reference 3). Prolonged storage or use at elevated temperatures could result in the release of gases and vapors from shelter construction materials (e.g., plastics and other synthetic materials). Soldiers occupying THAAD shelters may experience adverse health effects or performance decrement, depending upon the type and concentration of the gases and vapors to which they are exposed. The materiel developer should ensure that only safe construction materials are used in occupied THAAD shelters or vans. Detailed information on such materials is not available. Such information must be provided by the manufacturer to support completion of a final HHAR on the THAAD.

b. Temperature extremes. The THAAD will operate in hot, cold, and basic climatic categories (-50° to 120°F) and in severe cold using arctic kits (reference 3). Therefore, the potential for THAAD operators to experience injury or performance decrements due to exposures to temperature extremes is likely.

(1) Heat stress.

(a) A variety of heat illnesses may occur when personnel are exposed to hot, stressful environments for prolonged periods of time. According to TB MED 507, the commonly reported heat illnesses are heat cramps, heat exhaustion, and heat stroke (reference 23). Equally important is the performance decrement which may occur among THAAD personnel with elevations in core body temperature less than that required to cause heat illness.

(b) In hot/dry or hot/wet environments, the most important mechanism for lowering body core temperature is evaporative cooling. Adequate ventilation will aid in the evaporative cooling of THAAD personnel during hot weather operations. However, the potential for heat stress problems is significantly increased when personnel wear Mission Oriented Protective Posture (MOPP) gear for protection against NBC agents, due to its insulating effects between the wearer's body and the shelter environment (reference 24). Microclimatic cooling is an effective means of cooling personnel wearing MOPP gear.

(c) Reference 25 contains Permissible Heat Exposure Threshold Limit Values (TLVs). The TLVs are based upon the assumption that nearly all acclimatized, fully clothed workers with adequate water and salt intake should be able to function effectively under the given working conditions without exceeding a deep body temperature of 100.4°F. Since measurement of deep body temperature is impractical for monitoring workers' heat load, the measurement of environmental factors which most nearly correlate with deep body temperature and other physiological responses to heat is required. At the present time, the Wet Bulb Globe Temperature (WBGT) Index is the simplest and most suitable technique to measure the environmental factors.

(d) The SICPS shelter which houses the TOC for the THAAD system is equipped with a 9000 BTU heater/air conditioner. The cooling efficiency of the air conditioner has not been determined. Neither has a means of cooling the operator if MOPP gear is required to be worn inside the SICPS or other shelters in an NBC environment. Heating, ventilation, and air conditioning requirements in personnel enclosures are contained in MIL-STD-1472D, paragraph 5.8 (reference 18). Additionally, no heating/cooling information is available for the radar control van which is an occupied shelter. An assessment of potential heat injury to THAAD personnel cannot be completed at this time.

(2) Cold stress.

(a) In cold temperatures, physical and psychological handicaps will be presented to THAAD personnel. In cold weather, personnel efficiency and motivation may be impaired despite the best of cold weather clothing. Personal discomfort increases rapidly as the temperature drops below approximately 10°F. Below 0°F, performance decrement increases rapidly as temperature falls (reference 12).

(b) The type of cold injury produced depends upon the degree of cold to which the body is exposed, the duration of exposure, and certain concurrent environmental factors. Cold injuries may be type-divided into "freezing" (frostbite) and "non-freezing" (trench/immersion foot) (reference 26).

(c) Cold injuries are preventable and successful prevention requires prior planning, cold weather training, and the use of proper clothing and equipment. Specific preventive measures should be directed toward conservation of body heat, avoiding unnecessary or prolonged exposure to cold, moisture, and activities favoring cold injury. Detailed guidance regarding cold injury and its prevention, recognition, and treatment is contained in TB MED 81 (reference 26). The THAAD personnel will be exposed to limited periods of extreme cold since operation of the system will be done from inside shelters and vans. Information addressing the efficiency of shelter heaters is not available and the potential for cold stress injuries cannot be assessed at this time. Heating requirements for personnel shelters are contained in MIL-STD-1472D (reference 18).

c. Oxygen deficiency. Personnel will be working in the THAAD for undetermined lengths of time, and adequate ventilation, as described in MIL-STD-1472D (reference 18), must be provided. Shelter ventilation data are not available. Therefore, it cannot be assessed at this time.

d. Radiofrequency radiation.

(1) Theater Missile Defense-Ground Based Radar.

(a) The TMD-GBR candidate for the THAAD will use a transmitter-amplifier based either on TWT or solid state technology for generating radiofrequency radiation (RFR). The TMD-GBR antenna will be an electronically steerable phased array. This antenna will be able to instantly steer the very narrow main beam anywhere within the ±60° azimuth-elevation cone that is normal to the plane of the array (±60° about boresight) (this is the estimated capability of the system). That cone of instantaneous coverage can also be directed into any azimuth angle around a full circle (360°) by mechanically reorienting the antenna itself. It is unclear at this time what kind of transmission line (waveguide or coax) will be used to interconnect the antenna with the transmitter.

(b) Analysis shows the main beam of the TMD-GBR will be subject to radiation protection control to a range of about 3 km from the face of the antenna. Because of the full azimuth and elevation coverage that is possible for the radar, that range of control probably needs to be applied over the full upper hemisphere of coverage that surrounds the antenna. Analysis also indicates that an extremely high radiating power density level can be expected to a main beam range of about 0.5 km. An extremely high power density level can also be expected in the vicinity of any open or broken waveguide transmission line associated with the transmitter, if waveguide is used.

(c) Modern phased array radars will normally use sophisticated technology to control the direction, sequencing, and total RF power that is directed into any given narrow volume of space at any given time (reference 30). These controls also help to control unwanted irradiation of personnel who are working, or collocated, with the system. Based on the kinds of controls and the detailed mission requirements of the radar, such "built-in" radiation protection could be automatic with the TMD-GBR. If that is the case, the TMD-GBR antenna assembly could possibly be used without concern for control, within 1.4 km of personnel. Some tentative recommendations for control of the system have been made at this time. As the hardware design phase advances, and certainly as soon as an operating model is completed, a comprehensive RFR study and evaluation should be requested from USAEHA in accordance with AR 40-5 (reference 27).

(2) Single Channel Ground-to-Air Radio System (SINCGARS).

(a) Reference 3 specifies the use of the SINCGARS. This radio system utilizes a 50-W average power vehicular transmitter, operating over the 30–80 MHz frequency band, and a manpack radio that operates over the same frequency band at 4.0 W. The output power of the manpack radio is below the threshold for radiation protection control (7.0 W at frequencies less than 1.5 GHz). The antenna for the vehicular radio set is a 3-m whip. Analysis and measurement have shown that the 50-W system is able to produce power densities in excess of 1 mW/cm^2 within 0.7 m of the antenna. This is the PEL for the most restrictive portion of the SINCGARS frequency band. The SINCGARS is, therefore, subject to radiation protection control.

(b) To prevent possible RF shock and burn, personnel should be instructed to avoid contact with the antenna. This shock and burn avoidance procedure normally keeps a person's whole body well outside of the 0.7-m control range, and as such, constitutes adequate radiation protection for personnel using the SINCGARS.

(3) Joint Tactical Information Distribution System (JTIDS).

(a) Reference 3 specifies that THAAD will interoperate with the JTIDS. The JTIDS utilizes a 200 W peak power transmitter operating over the frequency band of 960-1215 MHz. There is a wide variation in available transmit power and frequency output options, and the option selected is primarily dependent upon the number of users and amount of data flow at a given time. A maximum transmitter duty cycle of 0.1 is normally estimated for the JTIDS, based upon all factors being at worst case. A 0.1 duty cycle results in a maximum average power output of 20 W.

(b) A 6- or 9-dBi gain omnidirectional antenna is used with the system. The selected antenna will be mounted either on the roof of the communication shelter or on top of a telescoping 10-m mast. The radiation element of either antenna is about 1-m long and 3 cm in diameter. The JTIDS will produce power density levels in excess of 10 mW/cm^2 to a range of 30 cm from either antenna. The only practical RFR threat comes from possible RF-shock or burn produced by touching the antenna during transmission.

(c) Personnel will not normally be within the control range of the antenna. However, where contact with the antenna is possible, RF shock and burn are a threat and personnel are instructed to avoid contact with the antenna. This shock and burn avoidance procedure normally keeps a person's whole body well outside of the 30 cm control range and, as such, constitutes adequate radiation protection for personnel using the JTIDS.

(4) Joint Surveillance and Target Acquisition Radar System (JSTARS).

(a) Reference 3 specifies that THAAD will interoperate with the JSTARS. This system serves as a data link transceiver which receives and processes near-real time radar target information about enemy follow-on forces. Two types of RFR sources are used with JSTARS: the Surveillance Control Data Link (SCDL) and the AN/VRC-46 radio.

(b) The SCDL is a wide-band data link operating in the Ku frequency band. The SCDL has the capability of transmitting in an uplink mode with an on-time duration of approximately 25 msec. Computer default settings limit the uplink transmission repetition-rate to once every 600 msec. This low on-time and repetition-rate constitute a very low duty cycle and resultant low average output power. The SCDL utilizes a radome-covered directional antenna which can be mounted either on a mast on top of the shelter or on a 1-m tripod on the ground. Actual transmitter power output and antenna gain values are classified.

(c) Power density levels can be emitted in the beam of the antenna for very short periods of time. However due to the extremely low duty cycle of the transmitter, the maximum average power density level is much lower than the 10 mW/cm2 PEL. The SCDL is not subject to radiation protection control.

(d) The AN/VRC-46 Radio is used for disseminating battlefield intelligence to appropriate users. These radios operate in the 30-88 MHz frequency band at a maximum power output of 35 W. The 3-m whip antennas used with these radios are mounted on the communications shelter roof. Power density levels exceeding the PEL of 1 mW/cm^2 (most conservative PEL in this band) can exist to a distance of 0.7 m from either antenna when the transmitter operates at maximum power output. The only practical RFR threat comes from possible RF-shock or burn produced by touching the antenna during transmission.

e. Acoustical energy.

(1) Steady-state noise.

(a) A steady-state noise level of 85 dBA or greater is considered hazardous (references 27 and 31). This limit assumes no more than 8 hours per day of high noise levels. For exposure exceeding 8 hours per day, noise levels below 85 dBA may be hazardous (reference 31). Prolonged unprotected exposure to hazardous noise levels will cause loss of hearing.

(b) The principal steady-state noise sources on the THAAD are expected to be generators, trucks, MCPE, and winch drive motors.

(c) Steady-state noise data at the operator's position and around the THAAD are not available; therefore, assessment of this issue is not possible. Steady-state noise associated with the THAAD must be collected in accordance with MIL-STD-1474C (reference 32) to support an assessment of this issue.

(d) The recommended design limit for steady-state noise is MIL-STD-1474C Category D (85 dBA). It should be noted that for clarity in communications (speech intelligibility), Category E or F may be appropriate (reference 32).

(2) Impulse noise.

(a) An impulse noise in excess of 140 dBP is considered hazardous (references 27 and 31). Repeated, unprotected exposure to hazardous impulse noise will cause permanent hearing loss. Exposure to impulse noise levels in excess of Curve Z, MIL-STD-1474C (reference 32), even when wearing hearing protective devices (HPDs), is considered hazardous for hearing conservation purposes.

(b) The potential sources of impulse noise on the THAAD are the rocket launcher and the AFES.

(c) MIL-STD-1474C, Figure 10 (reference 32) lists design limits for exposure of personnel wearing HPD's in terms of level, B-duration, and number of exposure per 24 hours.

(d) Certain AFES designs produce high impulse noise levels on activation. The noise source is the sudden release of the pressurized (750 psi) halon through the valve and nozzle. The noise level at any location in the shelter will vary according to the distance and angle from the nozzle.

(e) If the AFES currently used on Army tactical vehicles is employed on the THAAD, then existing data may

be adequate to evaluate this impulse noise hazard. This would entail the analysis of the present AFES data (references 33, 34, and 35) using the distances within the THAAD from nozzle locations to personnel positions.

(f) Impulse noise data at the THAAD operator's position and maintenance personnel locations are not available; therefore, assessment of this issue is not possible. Impulse noise associated with the THAAD must be collected in accordance with MIL-STD-1474C (reference 32) to support an assessment of this issue.

6. Recommendations.

a. Chemical substances.

(1) Diesel fuel exhaust.

(a) Ensure that the design and purchase specifications for the final configuration of THAAD require airborne concentrations of toxic substances, no matter what the source (e.g., vehicle and electric generator exhaust), inside occupied spaces, vehicle cabs, refrigeration and radar control areas and shelters to be controlled to the lowest level feasible and not to exceed current Army adopted exposure limits in accordance with MIL-STD-1472D (reference 18) and AR 40-5 (reference 27). Compliance with these requirements must be followed during all operational modes including extreme low temperature startup. Additionally, provide information as to the use of supplemental heaters for vehicle cabs, engine blocks and batteries. No risk assessment code (RAC) can be assigned at this time.

(b) Ensure that the final configuration of the THAAD positions sources of airborne contaminants (e.g., vehicles, generators and other engine exhaust) as far away from occupied shelter heater, air conditioning, ventilation and NBC filter system air inlets as possible in accordance with MIL-HDBK-759A (reference 12). No RAC can be assigned at this time.

(c) Provide a detailed system description and use/training scenarios for the final THAAD configuration to support completion of a final HHAR. No RAC can be assigned at this time.

(2) Rocket motor propellant and oxidizer. No recommendations are necessary.

(3) Fire extinguishing agents.

(a) Ensure that the design and purchase specifications for the final configuration of the THAAD AFES, if used, includes the current OTSG policy (i.e., the average concentration of Halon 1301 will not exceed 7 percent by volume, and the exposure time for personnel will not exceed 15 minutes). A RAC of 2 [Hazard Severity (HS) II, Hazard Probability (HP) C] is assigned for failure to comply.

(b) Provide detailed AFES design information, if used, for the final THAAD configuration to support completion of a final HHAR. The information should include the fire extinguishing agent concentration inside shelters following AFES discharge and the location of discharge nozzles. No RAC can be assigned at this time.

(4) Nuclear, biological, and chemical agents.

(a) Ensure that the MCPE incorporated into the THAAD is effective in providing an acceptable level of NBC protection to personnel inside occupied spaces/shelters. No RAC can be assigned at this time.

(b) Collect test data which measure the effectiveness of the THAAD MCPE to provide an acceptable level of NBC protection to personnel inside occupied spaces/shelters. Test data should include that data collected during challenges with ambient concentrations of chemical agent (or a suitable simulant) anticipated on a chemical battlefield during typical THAAD operations. Such data must be provided on the final configuration of THAAD to support completion of a final HHAR. No RAC can be assigned at this time.

(5) Off gassing.

(a) Ensure that THAAD design and purchase specifications require shelter and/or control van construction materials which will not release hazardous gases and vapors during prolonged storage or use at high temperatures. No RAC can be assigned at this time.

(b) Obtain a detailed list of THAAD shelter and/or control van construction materials and associated manufacturers material safety data sheets to support the completion of a final HHAR. No RAC can be assigned at this time.

b. Temperature extremes.

(1) Ensure that the design and purchase specifications for the final configuration of THAAD incorporates the heating and air conditioning requirements for occupied shelters contained in MIL-STD-1472D (reference 18). No RAC can be assigned at this time.

(2) Provide microclimatic cooling for THAAD personnel if MCPE is not included in the final configuration of the system or personnel are required to wear MOPP gear in shelters in an NBC environment. Comply with the cooling

requirements for such systems contained in MIL-STD-1472D (reference 18). No RAC can be assigned at this time.

(3) Collect test data which measures the effectiveness of the THAAD heating and air conditioning systems to meet the heating and cooling requirements contained in MIL-STD-1472D (reference 18). Test data should include that data collected during challenges at the upper and lower temperature extremes of the THAAD design operating temperature range. Such data and detailed heating and cooling system design information must be provided on the final configuration of THAAD to support completion of a final HHAR. No RAC can be assigned at this time.

c. Oxygen deficiency.

(1) Ensure that the design and purchase specifications for the final configuration of THAAD incorporates the ventilation requirements for occupied shelters contained in MIL-STD-1472D (reference 18). No RAC can be assigned at this time.

(2) Collect test data which measures the effectiveness of the THAAD ventilation system to meet or exceed the ventilation requirements contained in MIL-STD-1472D (reference 18). Such data and detailed ventilation system design information must be provided on the final configuration of THAAD to support completion of a final HHAR. No RAC can be assigned at this time.

d. Radiofrequency radiation.

(1) Theater Missile Defense-Ground Based Radar. Exclude personnel from the main-beam region of the antenna. Automatic control of the beam direction and intensity (with selectable or variable features) should be specified/designed into the system. A warning light should be used on the antenna assembly to warn personnel not to approach the antenna when the antenna is transmitting. To prevent personnel from entering the radiation control area, RFR warning signs should be placed along any routes into the area. The range of control should be specified as 0.5, 1.4, or 3.0 km, or more, depending upon the automatic controls, mission requirements, etc., that finally are specified for the system. A RAC of 2 (HS II, HP C) is assigned for failure to comply (this RAC applies only within the 1.4-km range). The hazard severity moves to III between 1.4 and 3.0 km, with higher RAC. A RAC of 3 (HS III, HP C) is assigned for failure to comply (between a range of 1.4 to 3.0 km).

(2) The following specific recommendations also apply to the TMD-GBR and affect the design and use of the system. A RAC of 2 (HS II, HP C) is assigned for failure to comply.

(a) Maintain maximum control of all areas that could result in exposure to greater than 5-times the PEL.

(b) The antenna assembly area should be located where operating personnel are not potentially exposed to the radiating field within a range of 1.4 km (required) and 3.0 km (ideally).

(c) All waveguide should be interlocked to prevent operation of the transmitter without all waveguide in place and in good operating condition.

(3) SINCGARS, JTIDS, JSTARS. Warn personnel to avoid physical contact with the vehicular antennas of these radio sets. A RAC of 5 (HS IV, HP C) is assigned for failure to comply.

e. Acoustical energy.

(1) Steady-state noise.

(a) Use Category D, MIL-STD-1474C, (<85 dBA) as the design goal for THAAD steady-state noise at normal operator positions. No RAC is required.

(b) Measure the steady-state noise levels associated with the THAAD at the operator's position, as outlined in MIL-STD-1474C (reference 32), and provide a training scenario. No RAC can be assigned at this time.

(2) Impulse noise.

(a) Use 140 dBP, Curve W, MIL-STD-1474C, as the initial design goal. If the 140 dBP level is unattainable, then use the applicable limits for personnel using HPD's. No RAC can be assigned at this time.

(b) If the AFES used in Army tactical vehicles is employed on the THAAD, measure the distances from the nozzle locations to the personnel positions, and provide a training scenario. No RAC can be assigned at this time.

(c) If the AFES currently in use on tactical vehicles is not used on the THAAD, then measure the noise levels associated with the THAAD AFES at the operator positions, as outlined in MIL-STD-1474C (reference 32), and provide a training scenario. No RAC can be assigned at this time.

7. Preparer Identification. This IHHAR was completed by the USAEHA, Aberdeen Proving Ground, MD 21010-5422, July 1991. The point of contact in the Directorate of Occupational and Environmental Health is the Health Hazard Assessment Office, DSN 584-2925.

APPENDIX A

REFERENCES

1. Integrated Logistic Support Plan (ILSP) (draft), subject: Theater High Altitude Area Defense System, June 1991.
2. Trade Study/Contractor Logistics Support (CLS), Rev. 1, subject: Theater High Altitude Area Defense System, June 1991.
3. Operational Requirements Document (ORD) for High Altitude Theater Missile Defense (HATMD) (draft), 17 June 1991, U.S. Army Air Defense Artillery School, ATSA-ATMD-H, Fort Bliss, TX 79916-0002.
4. Weapon Systems - 1991 United States Army, pp. 134–135, Department of the Army, ASA(RDA), Washington, DC, March 1, 1991.
5. Integrated Logistic Support Plan (ILSP) (draft), subject: Theater Missile Defense Ground Based Radar (TMD-GBR), July 8, 1991, U.S. Army Strategic Defense Command.
6. Memorandum for Record, USAEHA, HSHB-MO-A, 11 July 1991, subject: Trip Report, Health Hazard Assessment on the Theater High Altitude Area Defense (THAAD), Project No. 23-4847.
7. Initial Health Hazard Assessment Report (RCS MED 388) on the Kinetic Energy Anti-Satellite System, Project No. 69-37-4780-89, USAEHA, HSHB-MO-A, Aberdeen Proving Ground, MD, August 1989.
8. Occupational Health Guidelines for Chemical Hazards, U.S. Department of Health and Human Services (DHHS)-National Institute for Occupational Safety and Health (NIOSH), DHHS (NIOSH) Publication No. 81-123, January 1981.
9. NIOSH Pocket Guide to Chemical Hazards, DHHS-NIOSH, DHHS (NIOSH) Publication No. 90-117, June 1990.
10. Current Intelligence Bulletin No. 50: Carcinogenic Effects of Exposure to Diesel Exhaust, DHHS, NIOSH, DHHS (NIOSH) Publication No. 88-116, August 1988.
11. TM-9-2320-298-20, Organizational Maintenance Manual for Truck, Shelter Carrier, Tactical, 1-Ton, 4x4, M1028, April 1983.
12. MIL-HDBK-759A(MI), 30 June 1981, Human Factors Engineering Design for Army Materiel.
13. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants, Vol 4, National Academy Press, Washington, DC, 1985.
14. Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants, Vol 5, National Academy Press, Washington, DC, 1985.
15. Letter, National Research Council, Committee on Toxicology, August 8, 1989, subject: Short-Term Public Emergency Guidelines (SPEGLS) for Nitrogen Dioxide, Hydrazine, and Unsymmetrical Dimethyl Hydrazine.
16. The Condensed Chemical Dictionary, Tenth Edition, Gessner G. Hawlry.
17. Dangerous Properties of Industrial Materials, Seventh Edition. N. Irving Sax.
18. MIL-STD-1472D, 14 March 1989, Human Engineering Design Criteria for Military Systems, Equipment and Facilities.
19. Draft THAAD System Test and Evaluation Master Plan (TEMP), 12–14 June 1991.
20. 4th End, Office of The Surgeon General (OTSG), 14 May 1985, letter, to Army Materiel Command, AMSTE-CM-R, 14 February 1985, subject: Health Hazard Evaluation and Test Support of the Special Study of Halon Fire Extinguishing Agents, TECOM Project No. 1-VC-080-060-153.
21. U.S. Army Materiel Command, AMC TC No. 6704, 6 March 1969, subject: Policy Statement on the Use of Monobromotrifluoromethane (CBrF) Suppressant as a Fire Extinguishing Agent.
22. National Fire Protection Association (NFPA) Standard 12A; Halon 1301 Fire Extinguishing System, 1980.
23. TB MED 507, 25 July 1980, Prevention, Treatment, and Control of Heat Injury.
24. Performance Comparison of Three Microclimatic Systems for Cooling of Ground Vehicle Crewmen in Chemical Protective Clothing During Simulated Desert and Tropical Exposures, TR84/040L (IPL 267), U.S. Army Natick Research and Development Laboratories - Individual Protection Laboratory, July 1984.
25. Threshold Limit Values and Biological Exposure Indices for 1990–91, American Conference of Governmental Industrial Hygienists (ACGIH), Cincinnati OH.
26. TB MED 81, 30 September 1976, Cold injury.
27. AR 40-5, 15 October 1990, Preventive Medicine.
28. Letter, Office of The Surgeon General, 13 April 1987, subject: Technical Guide No. 153, Guidelines for Controlling Potential Health Hazards from Radiofrequency Radiation.

29. TB MED 523, 15 July 1980, Control of Hazards to Health from Microwave and Radio Frequency Radiation and Ultrasound.

30. TB 385-4, 25 March 1983, Safety Precautions for Maintenance of Electrical/Electronic Equipment.

31. TB MED 501, 15 March 1980, Hearing Conservation.

32. MIL-STD-1474C, 7 September 1990, Noise Limits for Military Materiel.

33. Technical Report 4936, FMC Corporation, Hearing Damage Assessment of the BFV Dual-Shot Fire Protection System Per MIL-STD-1474, 15 January 1990.

34. Memorandum, USAEHA, HSHB-MO-B, subject: Site Visit to CSTA, APG, MD, to View BFV-AFES, 19 April 1990.

35. FONECON between Mr. Glenn Rogers, STECS-EN-PH, CSTA, APG, and Mr. Felix Sachs, USAEHA, HSHB-MO-B, 16 February 1990, subject: AFES Impulse Noise Measurements.

Chapter 7

NOISE AND THE IMPAIRMENT OF HEARING

AMY M. DONAHUE, Ph.D.* AND DOUG W. OHLIN, Ph.D.†

INTRODUCTION

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- Cochlear Mechanics

HEARING LOSS AND AUDITORY DAMAGE
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- Health Education

SUMMARY

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INTRODUCTION

Occupational health professionals must understand both the auditory effects of noise and the proper administration and philosophy of the U.S. Army's Hearing Conservation Program to protect personnel from noise-induced hearing loss.

Casual observers of auditory physiology and hearing science assume that the effects of noise on the auditory system are well understood. Unfortunately, this is far from the truth because the auditory mechanism is startlingly complex. Advances in medical technology are only now allowing us to elucidate the morphology and the function of the structures of the normal ear. Theories on the etiology of noise-induced auditory damage are continually evolved and redefined as anatomical examination and electrophysiological-measurement techniques of auditory structures improve. For example, our understanding of the effects of noise on hair cell stereocilia, hair cell synapses, the cochlear vascular supply, and the central auditory pathways are still emerging.[1] Nuances regarding noise-induced hearing loss that are not yet understood are more numerous than the concepts that are universally accepted. Details of the unknowns will not influence a practicing physician's ability to identify or treat a clinical hearing loss. Yet, an understanding of the facts should greatly assist the physician in the evaluation of cochlear function following a soldier's or worker's exposure to high-intensity (ie, loud) sound. The physician's clinical tool is the audiogram, which depicts the hearing ability of the examinee in frequencies (measured in hertz [Hz]) and intensities (measured in decibel [dB] hearing threshold levels [HTL]).

Preserving a person's ability to hear low-intensity (ie, soft) sounds or speech on the battlefield is of utmost importance to the fighting efficiency and safety of soldiers:

> When you prepare to fight, you must prepare to talk. You must learn that speech will help save your situation. You must be alert at all times to let others know what is happening to you. You must use your brain and your voice any time that any word of yours will help you or others. You are a tactical unit and you must think of yourself that way. Don't try to win a war or capture a hill all by yourself. Your action alone means nothing, or at best, very little. It is when you talk to others and they join with you that your action becomes important.[2(p127)]

But, as is often the case, while the soldier's ability to speak on the battlefield is recognized, the second half of the communication equation—the ability to hear—is not. Sensitive hearing cannot be taken for granted in the army. Good hearing is particularly important when vision is limited—in sentry duty and night patrols—or during communication over noisy electronic systems.

Hazardous noise pervades the military environment; a soldier's ability to hear can be assaulted and damaged permanently even before basic training is completed. Most noise-induced hearing loss occurs during routine training exercises and therefore should be almost completely preventable.[3] The need to conserve hearing is especially important during practice and test firings for soldiers who soon afterwards must rely on their hearing to detect the enemy and to perform other communication requirements of the mission. The increasing demand for weapons systems with greater speed, range, and firepower confounds the problem with higher and more-hazardous noise intensities.

In addition, military-industrial operations (which may include the manufacturing, maintenance, and testing of military ordnance) can also include noise hazards for both military and civilian personnel. Except for large-caliber weapons testing, most military-industrial activities have counterparts in the private sector.

AUDITORY SENSATION

The transmission of sound through the ear (Figure 7-1) involves a series of energy conversions. When sound waves enter the ear canal, the tympanic membrane is set into vibration. At this point, acoustic energy is converted into mechanical energy. The vibrations of the tympanic membrane are then transferred through the ossicular chain of the middle ear to the inner ear. The process of articulation between the tympanic membrane, the malleus, the incus, and the stapes converts mechanical energy into hydraulic energy. The final conversion of energy occurs at the level of the receptor cells of hearing, the hair cells, with the release of the neurotransmitter substances that initiate a chem electrical electrical impulse.

A brief review of auditory anatomy, which emphasizes the inner ear and cochlear mechanics, will help to familiarize readers with the complex mechanisms of auditory injury.

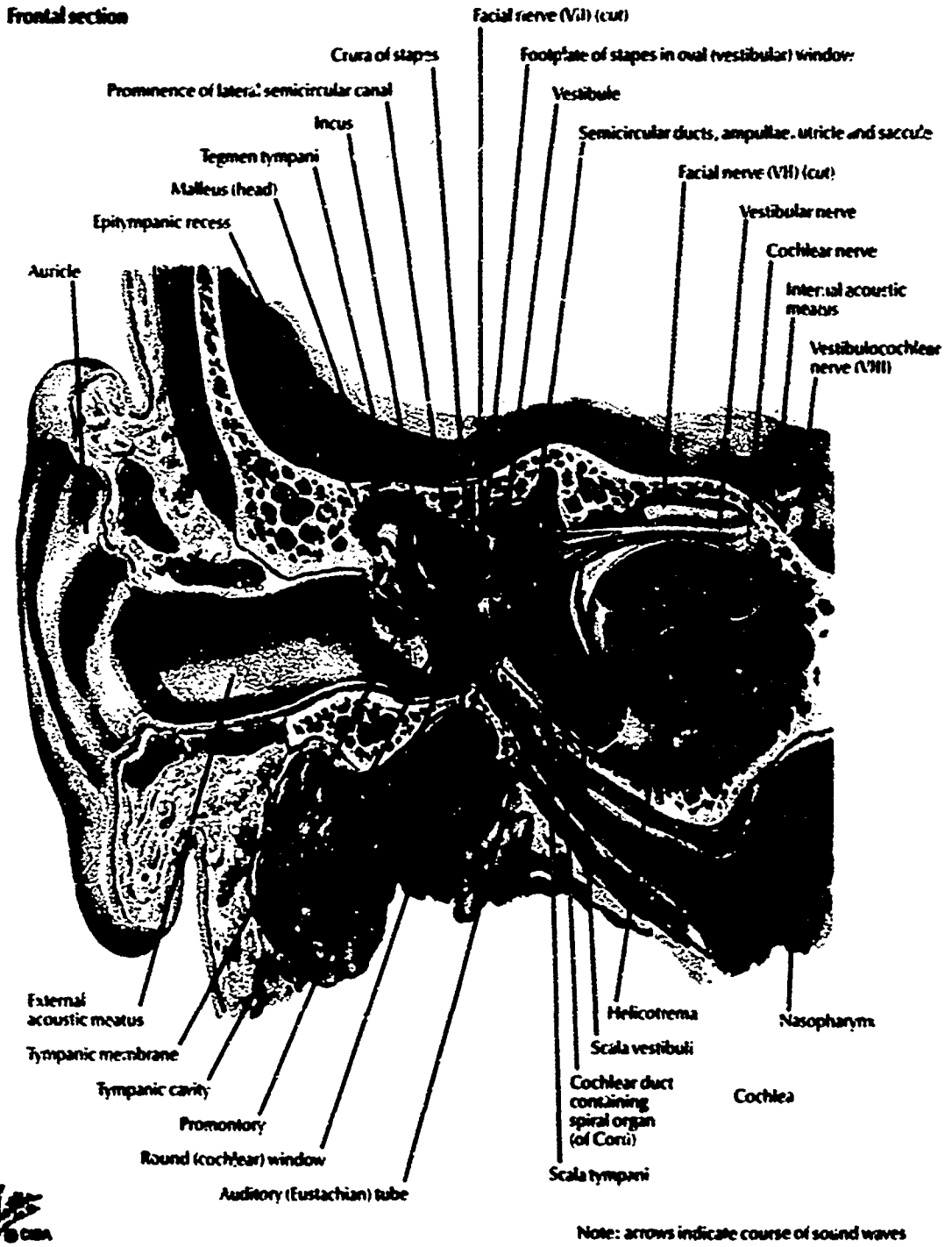

Fig. 7-1. The external, middle, and inner ears in man. Reprinted with permission from Otologic diagnosis and treatment of deafness. *Clinical Symposia*. 1970;22(2):38. Slide 1161. West Caldwell, NJ: CIBA-GEIGY.

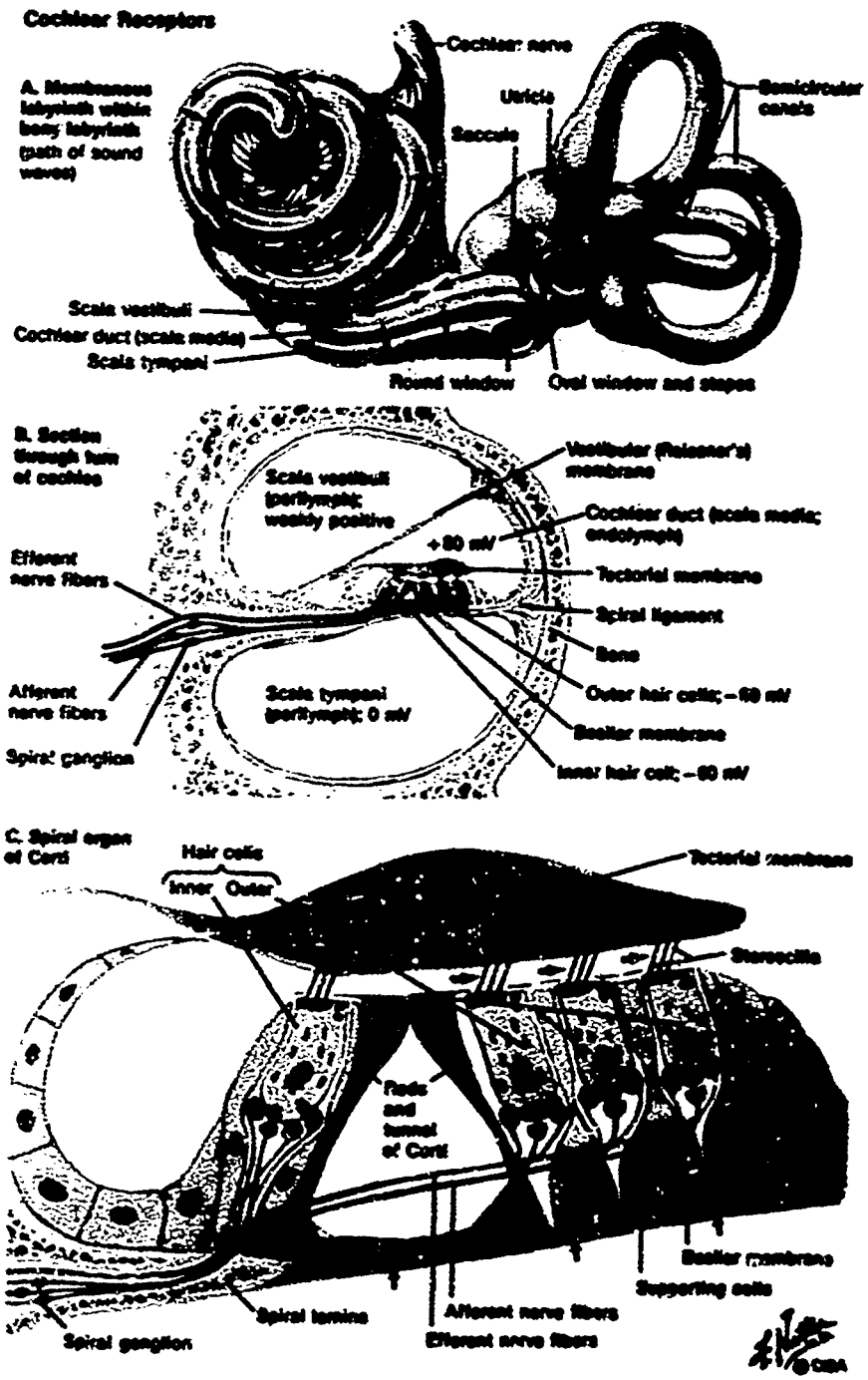

Fig. 7-2. Panel A is a low-power view of the osseous and membranous labyrinths. The cochlea is on the left. Panel B is a transverse section through the cochlea showing its three fluid-filled channels and the organ of Corti. The stria vascularis (not shown) is found on the spiral ligament at the outer circumference of the cochlear duct. Panel C is a high-power view that shows the constituent cells of the organ of Corti Reprinted with permission from Netter FH. The CIBA collection of medical illustrations. Vol 1. *Nervous System*. Part 1, *Anatomy and Physiology*. West Caldwell, NJ: CIBA-Geigy; 1987: 176. Slide 3132.

Auditory Anatomy

The cochlea, or inner ear (Figure 7-2), a fluid-filled medium that measures approximately 1 cm wide and 5 mm long in humans, has 2.75 turns. It is separated into three channels by the bony labyrinth, the basilar membrane, and the Reissner membrane. The uppermost channel—the scala vestibuli—and the lowermost channel—the scala tympani—are filled with perilymph, a fluid with a high concentration of sodium and a low concentration of potassium. Perilymph resembles normal extracellular fluid in composition and is near ground electrical potential. The medial channel—the scala media, also known as the cochlear duct—is located between the basilar and the Reissner membranes. This channel is filled with endolymph, a fluid with high potassium and low sodium concentrations. Endolymph resembles intracellular fluid and has a positive electrical potential. Within the scala media, the organ of Corti rests on the basilar membrane. The stria vascularis, a highly vascularized layer of tissue, lines the outer wall of the cochlea (on the surface of the spiral ligament) and has a significant function in the production of endolymph.

The organ of Corti, which contains the hair cells, supporting cells, and neural connections, is the key organ of hearing. It contains one row of approximately 3,400 inner hair cells, and three to five rows of outer hair cells totaling approximately 13,400 outer hair cells.[4] Cilia on the hair cells are arranged in visually distinct patterns (Figure 7-3): those on the inner hair cells form a nearly straight row (Figure 7-4), and those on the outer hair cells form a W-shaped pattern (Figure 7-5). The longer cilia on the outer hair cells are embedded firmly in the tectorial membrane, while the cilia on the inner hair cells are not embedded and may only attach loosely to the undersurface of the tectorial membrane.[5]

The cochlea is innervated by both afferent and efferent neural fibers. Humans have approximately 18,000 cochlear afferent fibers; 95% of them innervate the inner hair cells and only 5% innervate the more numerous outer hair cells. The inner hair cells have a divergent innervation pattern, in which each inner hair cell is innervated by many fibers. The outer hair cells have a convergent system, in which one nerve fiber innervates many outer hair cells. The cell morphology, neural innervation, and auditory functions of inner and outer hair cells are quite different. In

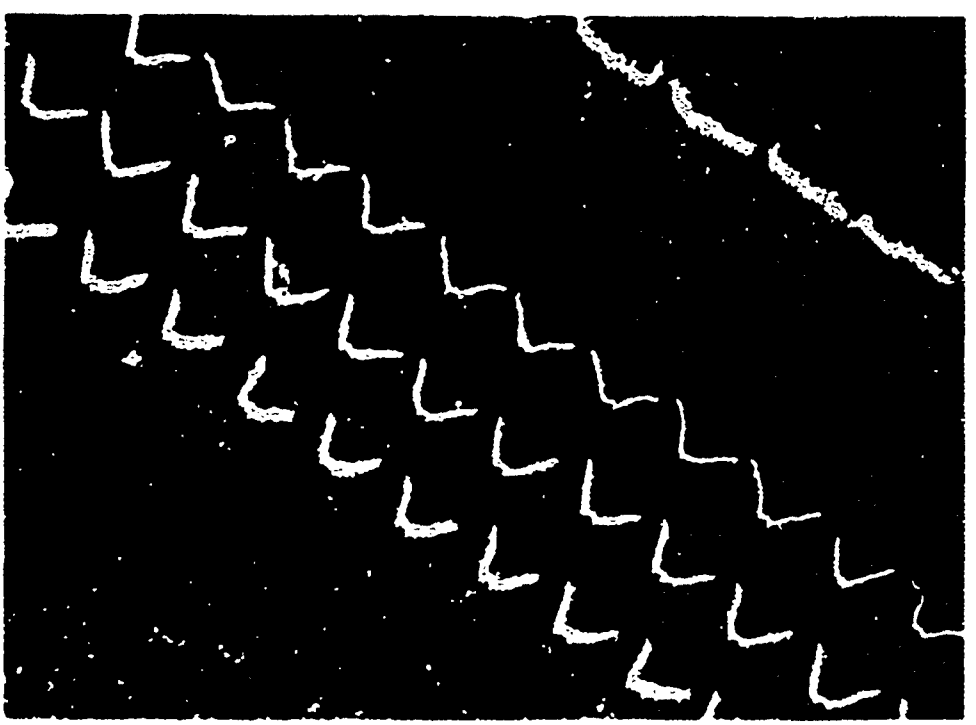

Fig. 7-3. A scanning-electron micrograph of the upper surface of the organ of Corti with the tectorial membrane removed. There are three rows of outer hair cells and one row of inner hair cells. Reprinted with permission from Pickles JO. *An Introduction to the Physiology of Hearing*. New York: Academic Press; 1982. © 1982, Academic Press, Orlando, Fla.

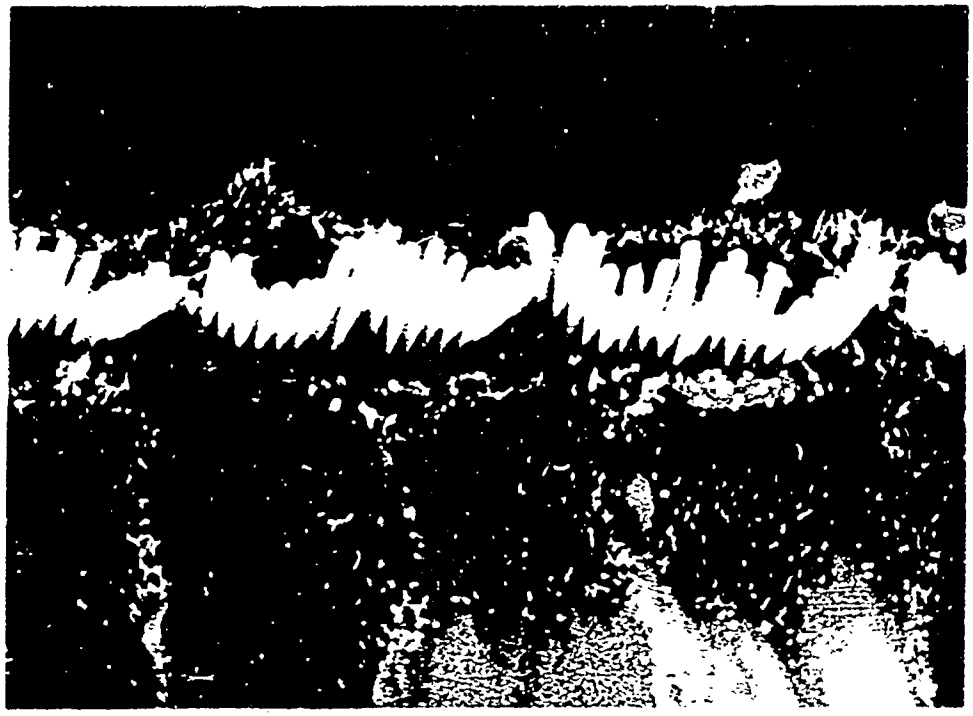

Fig. 7-4. The stereocilia on the inner hair cells form a visually distinct straight row. Reprinted with permission from Pickles JO. *An Introduction to the Physiology of Hearing*. New York: Academic Press; 1982. © 1982, Academic Press, Orlando, Fla.

Fig. 7-5. The stereocilia on outer hair cells are smaller and form a visually distinct "V" or "W" configuration. Photograph. Courtesy of Donald Robertson, Perth, Australia.

general, audiologists believe that the outer hair cells' activities are summed together to provide increased auditory sensitivity, while the inner hair cells' activities provide fine discrimination.

Cochlear Mechanics

Mechanical movement of the stapes at the oval window creates a fluid-pressure wave in the inner ear, and the round window of the scala tympani acts as the release outlet for the pressure wave. Because liquid is an incompressible medium, this dichotomy of the oval and round windows allows a pressure wave to displace the basilar membrane. The location and amplitude of this displacement depend on the spectral components (ie, the frequency and the intensity combinations) of the stimulus.

The pressure wave creates movement of the organ of Corti on the basilar membrane with respect to the tectorial membrane. Because the tectorial membrane is anchored differently than the basilar membrane, a shearing motion is created between the two structures (tectorial membrane and the organ of Corti), which results in the mechanical displacement of hair cell cilia (Figure 7-6). The movement of the cilia alters the electrical resistance of the hair cell membrane: the resulting ion current flow through the membrane changes the resting voltage of the hair cell. The movement of the cilia initiates the release of neurotransmitter substance at the base of the receptor cell and prompts the neuroelectrical transmission of the signal.

The auditory system analyzes low frequencies (those below 1500 Hz) and high frequencies (those above 1500 Hz) differently (Figure 7-7). During sound stimulation, the frequency of the stimulus determines the site of maximal displacement of the basilar membrane. High frequencies stimulate the basal portion of the basilar membrane nearest the oval window, with rapidly decaying displacement. The higher the frequency, the closer the hair cell receptors to the oval window. Low-frequency sounds cause wider areas of displacement and stimulate a much larger area of the cochlea; maximum displacement of the basilar membrane occurs at its apical (upper) end. Physical characteristics of the basilar membrane, most notably its increased width and reduced stiffness at the apical end, determine the resonant properties of the basilar membrane. The frequency-dependent points of maximum displacement along the basilar membrane are dictated by these physical characteristics.

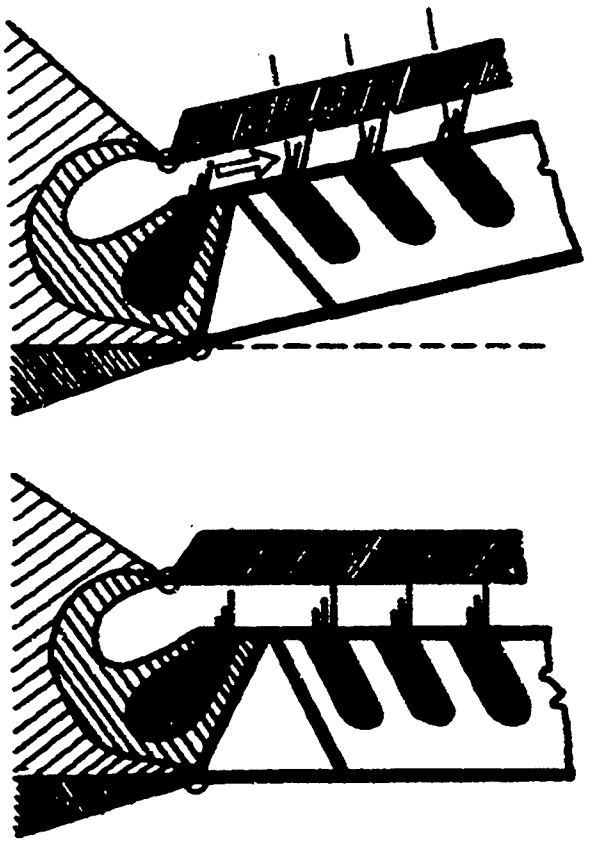

Fig. 7-6. Relationship between the tectorial membrane and cilia of outer hair cells. At rest, (lower illustration) the cilia stand perpendicular to the tectorial membrane surface of the cell. When pressure waves move the basilar membrane, a shearing force acts to alter the angle of the cilia with respect to the tectorial membrane. Note that cilia of the inner hair cells are shown to bend, not because of their tectorial membrane attachment, but because of fluid motion. Reprinted with permission from Dallos P, Ryan A. Physiology of the inner ear. In: Northern JL, ed. *Hearing Disorders.* Boston: Little, Brown: 1976: 95.

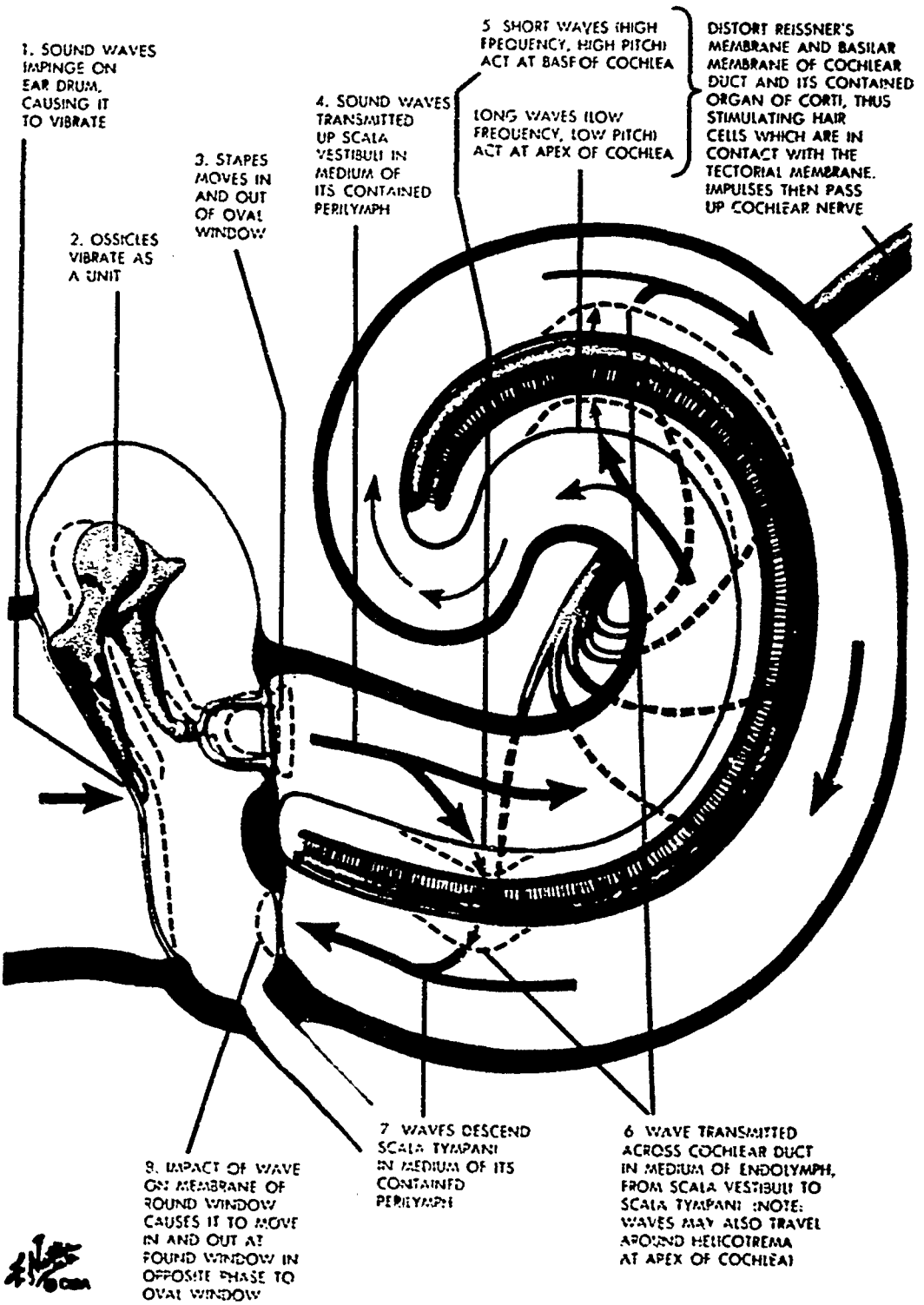

Fig. 7-7. Transmission of sound waves through the cochlea. Reprinted with permission from Otologic diagnosis and treatment of deafness. *Clinical Symposia.* 1870;22(2):42. Slide 1163. West Caldwell, NJ: CIBA-GEIGY.

HEARING LOSS AND AUDITORY DAMAGE

The auditory system, although somewhat sheltered anatomically under the temporal bone at the base of the skull, is not immune to environmental hazards. Prolonged or intense exposure to noise can be detrimental to the auditory system.

Audiometric Threshold Shifts

Noise alters auditory structure and function and causes a subsequent loss of hearing sensitivity known generally as a noise-induced hearing loss. The effects may be temporary or permanent. Typically, an audiometric threshold (ie, the intensity, measured in dB HTL, at which a human can just detect a specific frequency) is measured both before and after an exposure to noise, and any measured difference in hearing sensitivity is referred to as a threshold shift. If the threshold measured after noise exposure recovers to its preexposure sensitivity, the loss is referred to as a noise-induced temporary threshold shift (TTS). If the postexposure sensitivity of the threshold does not fully recover to its preexposure level, the loss is referred to as a noise-induced permanent threshold shift (PTS).

For years, audiologists have known that a relationship exists between the audiometric frequency of noise and the resulting frequency of the maximum threshold shift. For high-intensity, pure-tone exposures, the greatest threshold shift is most often demonstrated at a frequency one-half to one octave (ie, one-half to one doubling of the observed frequency) above the frequency of the noise. For example, a noise at 2500 Hz would produce an audiometric shift at 4000 Hz. For broad-band noise with equal energy in all bandwidths, however, the maximum threshold shift occurs between 3000 and 6000 Hz. In mammals, this phenomenon is explained by both cochlear mechanics and the location and maximum amplitude of the vibration of the partition (ie, the basilar membrane and the organ of Corti). Cochlear partition vibration patterns do not increase linearly with the amplitude of the sound wave at all frequencies. As the sound-wave amplitude (the intensity of the noise) becomes greater, the vibration becomes less localized and moves toward the basal portion of the cochlea.[6] This vibration damages a locus of the cochlear partition that is more basal than the stimulating frequency, and causes a subsequent audiometric loss at a frequency higher than that of the insulting noise.

Influence of the Outer and Middle Ears on Hearing Loss

Although most discussions of noise-induced hearing loss focus on the damage that occurs in the inner ear, the outer and middle ears also play roles in noise-induced hearing loss.[7] The characteristics of the outer ear create frequency enhancement and those of the middle ear create frequency selectivity. These two mechanisms help to explain why noise-induced hearing loss is so often found at 3000 to 4000 Hz, which audiologists describe as a classic "notch" on the audiogram.

The resonant characteristics of the ear canal, which are determined by its length and volume, enhance frequency in the following way: at 2500 to 3500 Hz, which is the ear canal's resonant frequency, the sound-pressure level (dB SPL) (ie, the variation of air pressure due to a disturbance in the acoustic range) is increased at the eardrum by 15 to 20 dB compared to the dB SPL at the ear canal's entrance (Figure 7-8).[8] Thus, the resonance of the ear canal provides a high-frequency boost of energy that effectively changes the spectral components of any sound that enters it. Due to cochlear mechanics, an audiometric threshold loss occurs one-half to one octave above the frequency of the insulting noise; therefore, an energy boost at 2500 to 3500 Hz and a resulting threshold loss at 3000 to 6000 Hz is consistent with this principle.

By comparison, the middle ear inherently discriminates against certain frequencies. The transfer functions of the middle ear allow the mid- to high-frequency sounds (defined here as those between 1500 and 4000 Hz) to pass through it more efficiently than the low-frequency sounds, with the low-frequency sounds reaching the inner ear at a reduced intensity (relative to their intensity when they entered the ear canal). This allows sounds at frequencies greater than 1000 Hz to be transferred to the inner ear more easily. The physical alteration of sound before it reaches the inner ear is only partially responsible for different noise spectra that yield identical audiometric configurations, with hearing losses first measured at 3000 to 4000 Hz.

The middle ear contains two muscles, the stapedius and the tensor tympani, which contract reflexively in response to sound. Early theories on the functions of these muscles purported that this acoustic reflex protects the ear from loud sounds, because when the reflex occurs, the middle ear is stiffened and becomes a less

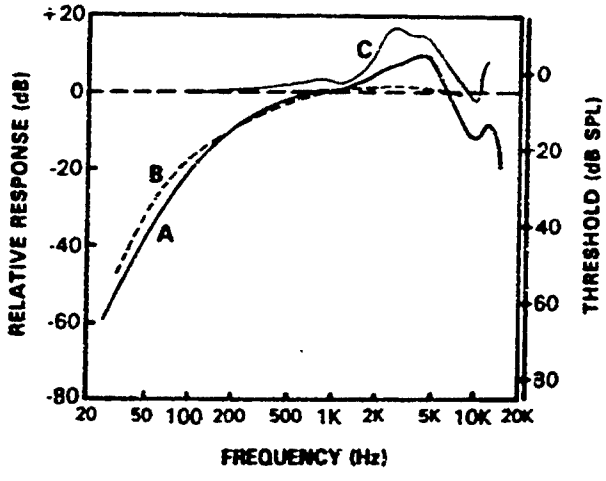

Fig. 7-8. Curve A: Response characteristics of the auditory system. A minimum audibility curve has been inverted as if a frequency-response curve had been "run" on the auditory system (actual thresholds, in dB SPL re 2×10^{-3} N/m^2, are shown on the right for reference). Curve B: A weighted response curve for sound-level measurement. Curve C: Sound pressure appearing at the eardrum as a function of the pressure in the sound field. Reprinted from Durrant JD. Anatomical and physiologic correlates of the effects of noise on hearing. In: Lipscomb DM, ed. *Noise and Audiology*. Baltimore: University Park Press; 1978 (out of print). Photograph: Courtesy of John D. Durrant, Philadelphia, Penn.

effective transmission system. Historically, this theory has been criticized for the following reasons:

- The reflex response fatigues rapidly, making it ineffective for continuous sounds.
- The muscle action is too slow (50–100 msec from latency to activation) for impulse sounds.
- The reflex provides protection only at frequencies below 1000 to 1500 Hz.
- The reduction of sound transmission is too small to have a protective effect.

The protective role of the muscles of the middle ear remains uncertain. Real-world stimuli, such as intermittent noise in a factory, may neutralize some of the above criticisms of reflex protection. For example, a noise may be of such short duration that the fatigue factor is rendered moot. Individual variability certainly exists in the degree of reflexive response. Numerous scientific papers discuss the relationship of the acoustic reflex to noise-induced hearing loss; promising new findings indicate that the role of the reflex in hearing may be more critical than was previously thought.[9-13]

Distinction Between Noise-Induced Hearing Loss and Acoustic Trauma

Current literature on damage to the cochlear structures is separated into distinct categories: noise-induced hearing loss and acoustic trauma. These categories may seem to be a contradiction in terms because acoustic trauma produces noise-induced hearing loss. However, an understanding of the anatomical consequences of the two shows that the types of injury are quite different.

Noise-induced hearing loss refers specifically to an injury that is caused by repeated exposures to moderate- or high-intensity noise. The noise may initially cause only a TTS, but at some point, the injury may become a PTS. This type of hearing loss, regardless of the frequency of the noise that caused it, usually begins audiometrically at 3000 to 6000 Hz and spreads to both higher and lower frequencies. The mode of destruction is more subtle, and the auditory effects evolve more slowly, than with acoustic trauma. Pathological changes may include (*a*) damage to intracellular structures and to the cilia of the receptor cells, (*b*) swelling of the nerve endings, (*c*) changes in vascular pathways, (*d*) biochemical changes in the cochlea, and (*e*) cell damage to the lateral wall of the cochlea in the stria vascularis and spiral ligament.

Damage to areas other than the receptor cells is usually found only when hair cell loss is almost complete. After the hair cells are injured, neural degeneration will appear. Some similar pathological injuries appear in both structures (Figures 7-9 through 7-12).

Much of the research on the auditory effects of noise has focused on the auditory periphery (ie, all the anatomical structures of the auditory system excluding the cerebral cortices and the brainstem). The central nervous system (CNS) response to noise insult—or to any type of damage in the auditory periphery—is still being investigated. Knowledge of the effects of hazardous noise on CNS function is expanding rapidly and new information on neural-feedback systems to the ear and how to protect it from acoustic overstimulation is forthcoming.

Acoustic trauma refers to injury that is caused by impulse or impact sounds of short duration and high intensity, which produce immediate, permanent hearing loss. The mode is mechanical. All structures of the ear are vulnerable to mechanical damage, but the most susceptible is the organ of Corti. Mechanical trauma to the auditory system usually consists of both PTS and TTS components, but some audiometric recovery (of the TTS component) may occur over a period of weeks. The audiometric frequency of the

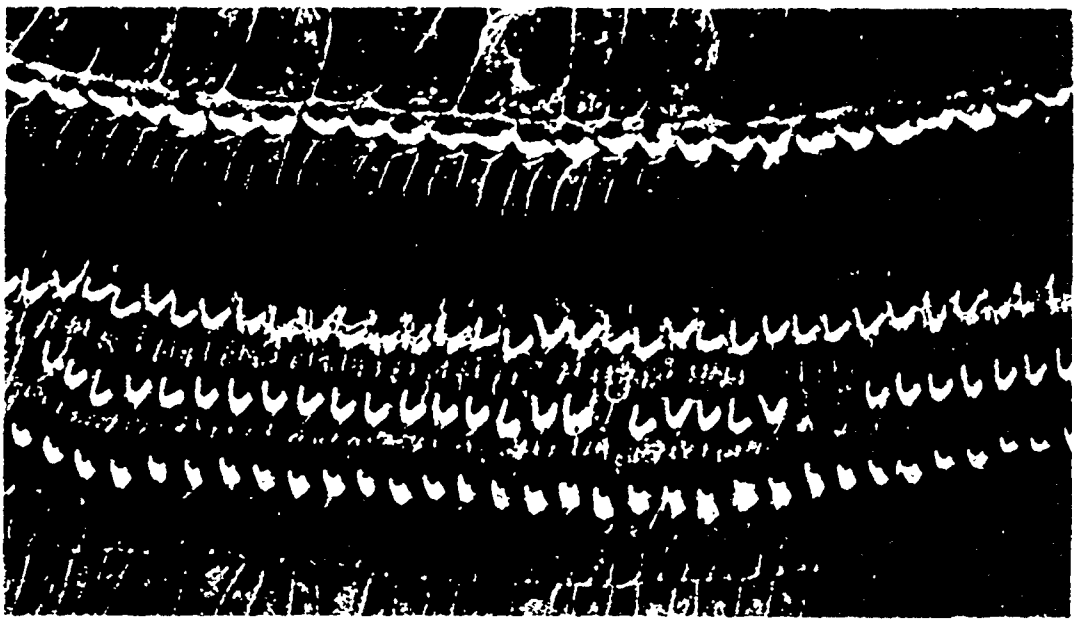

Fig. 7-9. This scanning-electron micrograph shows a pattern often seen in bent stereocilia of an outer hair cell (arrows) and in stereocilia after more severe damage from pure-tone stimulation. Patches of damage may extend over a full turn of the cochlea. Reprinted with permission from Hunter-Duvar IM, Suzuki M, Mount RJ. Anatomical changes in the organ of Corti after acoustic stimulation. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss*. New York: Raven Press; 1982.

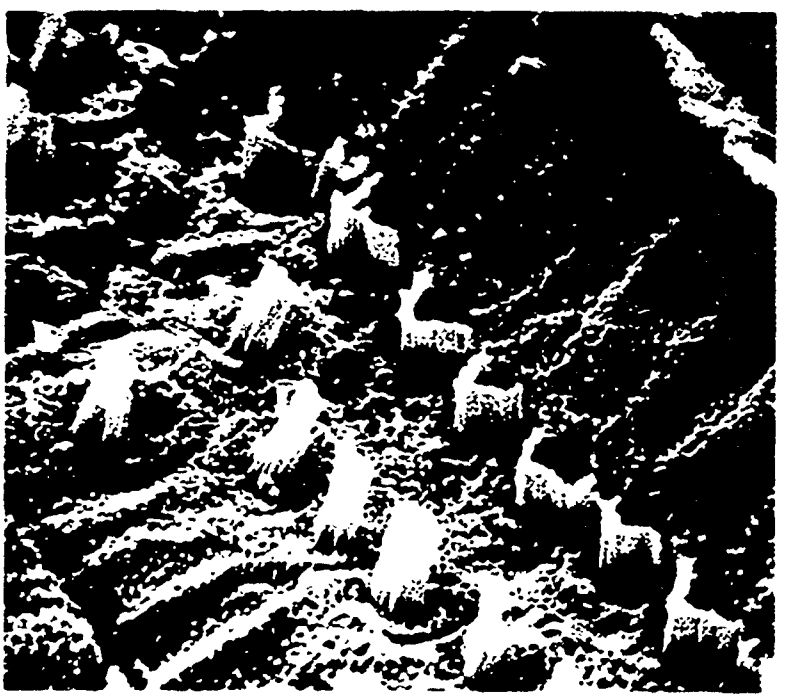

Fig. 7-10. Scanning-electron micrograph of the cochlea at a 3,000-fold magnification about 15 mm from its base in a guinea pig exposed to impulse noise. Several outer hair cells in the third row are missing. Two hair cells (arrows) have missing hair bundles. Reprinted with permission from: Nilsson P, Erlandson B, Hakanson H, Ivarsson A, Wersall J. Anatomical changes in the cochlea of the guinea pig following industrial exposure. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss*. New York: Raven Press; 1982.

Fig. 7-11. This scanning-electron micrograph shows an area of disrupted cilia of outer hair cells after acoustic stimulation. The proximity of the cells has allowed the cilia on adjacent cells to collide (arrows). Reprinted with permission from Hunter-Duvar IM, Suzuki M, Mount RJ. Anatomical changes in the organ of Corti after acoustic stimulation. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss*. New York: Raven Press; 1982.

Fig. 7-12. Scanning-electron micrograph of bent and fused stereocilia on inner hair cells. Reprinted with permission from Hunter-Duvar IM, Suzuki M, Mount RJ. Anatomical changes in the organ of Corti after acoustic stimulation. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise Induced Hearing Loss*. New York: Raven Press, 1982.

hearing loss may vary among individuals. Weapons fire may cause an asymmetrical high-frequency hearing loss that is due to the protective "head shadow effect" (ie, protection afforded the right ear by the shooter's own head and shoulder). For example, the hearing loss would be worse in the left ear for right-handed shooters. An asymmetrical hearing loss is also not unusual after exposure to the noise of an explosion. There is a greater chance that the audiometric configuration after this type of injury may be flatter; the pressure wave from an explosion may also damage the middle ear and add a low-frequency component to the hearing loss.

MECHANISMS OF AUDITORY INJURY

The major mechanisms of auditory injury are mechanical and metabolic. Vascular injury may also occur following noise exposure, but a specific injury mechanism has not yet been determined.

Mechanical damage, in which noise overstimulation directly injures cochlear structures, is caused by intense noise of rapid onset and short duration and its consequent acoustic trauma. Excessive force on the cochlear partition creates excessive stresses and displacement, which tear and disrupt the cochlear structure. Mechanical damage can include

- injuring hair cells in the organ of Corti;
- tearing the entire organ of Corti away from the basilar membrane so that it floats within the scala media[14,15];
- rupturing the basilar membrane[16];
- rupturing the Reissner membrane, which allows endolymph and perilymph to mix, creating a biochemical environment that is toxic to the receptor cells[16];
- tearing holes in the reticular lamina, allowing endolymph to flow into the organ of Corti[17];
- ripping apart tight cell junctions; and
- swelling and degeneration of hair cells, nerve fibers, and nerve endings in the organ of Corti at the apical and basal edges of the lesion.[18]

Data from researchers who exposed chinchillas to blasts demonstrate great variability in both sensory cell loss and in the formation of scar tissue.[14] This scar tissue prevents mixing of the cochlear fluids, and therefore prevents additional sensory cell loss.

Metabolic injury is manifested by disruption of internal cell processes. Rather than the intense mechanical force associated with impulse noise, this type of damage is more often associated with slow, insidious, noise-induced hearing loss. However, metabolic damage also occurs after direct mechanical damage from exposure to impulse noise.[19] Characteristics of metabolic damage include

- injury to hair cells and afferent dendrites;
- degeneration of scattered sensory cells, as a result of daily exposure, with damage more likely to affect outer rather than inner hair cells; and
- an increase in the number of damaged sensory cells with increasing noise exposure.

One theory attributed hair cell damage to exhaustion of cytochemical or enzymatic materials after or during exposure to noise.[20] This physiochemical theory is known as the metabolic exhaustion theory.[21] Numerous reports cite the apparent relationship between auditory damage and metabolic exhaustion. Morphological changes in hair cell structures (specifically, of mitochondria and of the endoplasmic reticular system) suggest that deficits occur in fuel utilization, in protein synthesis, and in energy production.[1] These metabolic or homeostatic disruptions, whether they occur independently or collectively, are considered to result from exposure to excessive noise: enzyme systems that are critical to these processes are found in noise-damaged cellular structures.[1] Cellular degeneration follows severe insults of this type.

The metabolic theory of damage has become even more pertinent because new information changed our view of the cochlea's role in auditory transduction.[22] Previously, the cochlea was thought to be a passive analyzer and transducer of sound. We now understand that the cochlea has its own motile properties and participates in an active transduction process that requires energy to change hair cell and stereocilia mechanical properties in response to sound. Because the hair cells are involved actively in sound transduction and analysis, the metabolic theory of damage may have greater responsibility for the injury process than was previously thought.

The auditory system has two independent vascular supplies: first, a dense capillary network, the stria vascularis, which lines the outer wall of the scala media, and second, radiating arterioles that serve the organ of Corti. The stria vascularis influences chemical and oxygen balances to maintain endolymphatic metabolism, while the radiating arterioles provide oxygen to the organ of Corti (Figure 7-13).

Regional vascular abnormalities have been found after exposure to excessive noise. The vascular theory

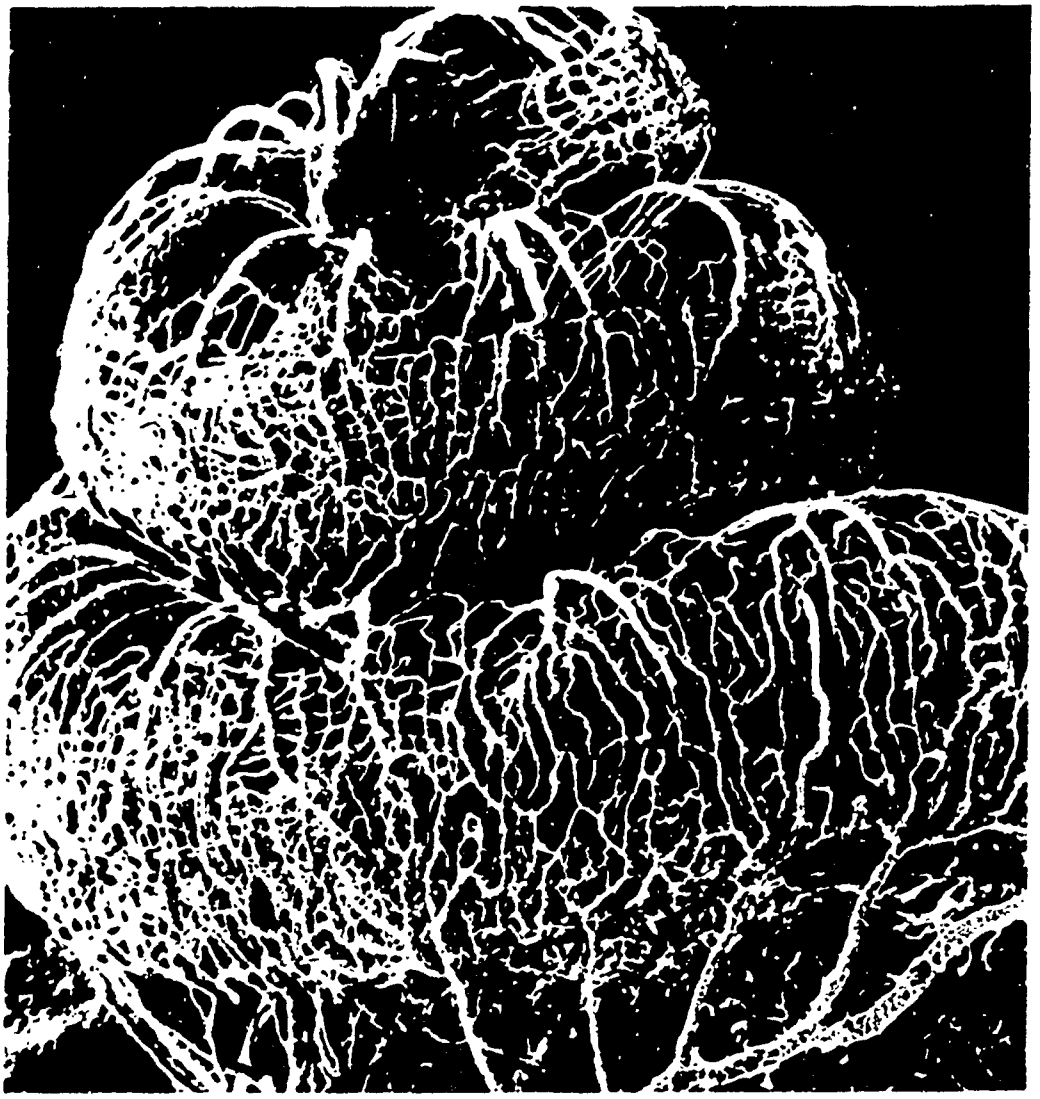

Fig. 7-13. Vascular network of the rat cochlea. This is a low-magnification, scanning electron photomicrograph of tissue prepared by injecting latex into the vascular system. After the latex solidified, all the cochlear tissue was dissolved in acid to reveal only latex-filled vascular channels. Photomicrograph. Courtesy of Jack A. Vernon, PhD, Kresge Hearing Research Laboratory, University of Oregon, Portland, Ore.

asserts that changes in the vascular system occur after exposure to noise and the resultant less-efficient delivery of nutrients to, and expulsion of waste products from, the cochlea make the auditory system more susceptible to injury. This may occur in conjunction with either metabolic or mechanical damage (Figures 7-14 and 7-15). Many vascular variables have been studied (Figure 7-16), including the number and the density of erythrocytes, the diameter of the blood-vessel lumens, the frequency and size of the perivascular cells, changes in oxygen tension, the thickness of the blood-vessel walls, and edema and atrophy of the stria.

The role of vascular factors in noise-induced hearing loss has been discussed for years. However, no consensus on the type and degree of vascular changes, the ultimate effect of the change on the cochlea, or the underlying mechanism responsible for the change has been reached. Conflicting results—due to the many different variables measured and the methodologies and species of test animals used—make conclusions difficult. Vascular abnormalities that occur in response to excessive auditory stimuli are probably one contributing variable of the metabolic theory.

One study utilized carbogen to investigate the relationship between noise and vascular deficits.

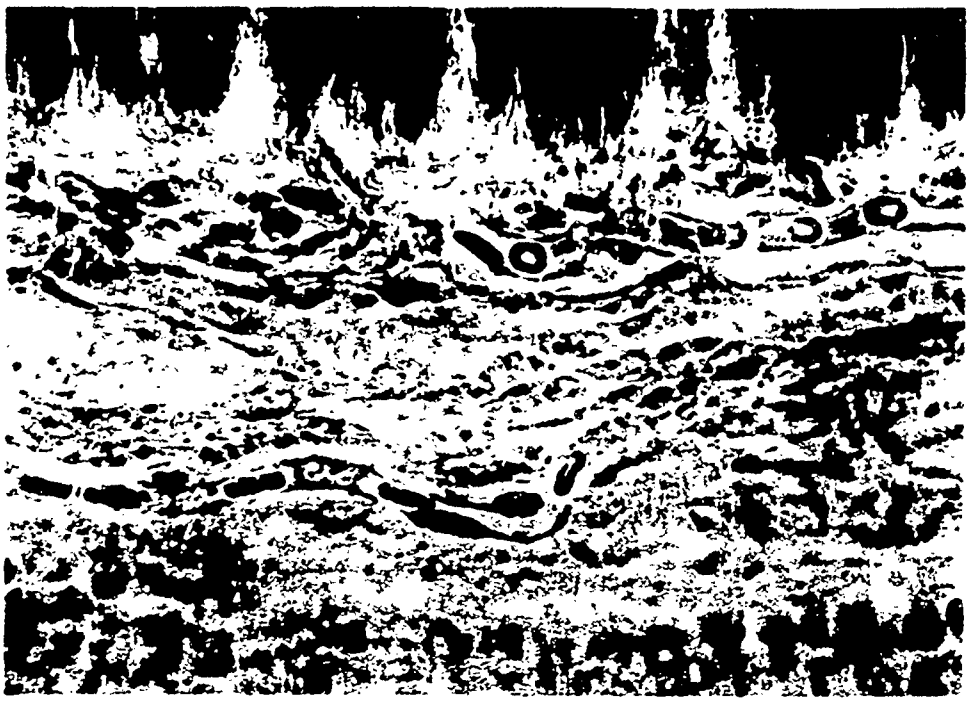

Fig. 7-14. Capillary vasoconstriction at the second turn of the cochlea with endothelial cell swelling and trapped erythrocytes (arrows) in inner and outer spiral vessels. Noise exposure was 118 to 120 dB for 30 hours continuously. Reprinted with permission from Hawkins JE Jr. The role of vasoconstriction in noise-induced hearing loss. *Ann Otol Rhinol Laryngol.* 1971;80:903-913.

Fig. 7-15. A transmission electron micrograph of a trapped erythrocyte in an outer spiral vessel at the third turn of the cochlea. The lumen is reduced to 1 micron (μ) or less. Noise exposure totaled 118 to 120 dB for 110 hours. Reprinted with permission from Hawkins JE Jr. The role of vasoconstriction in noise-induced hearing loss. *Ann Otol Rhinol Laryngol.* 1971;80:903-913.

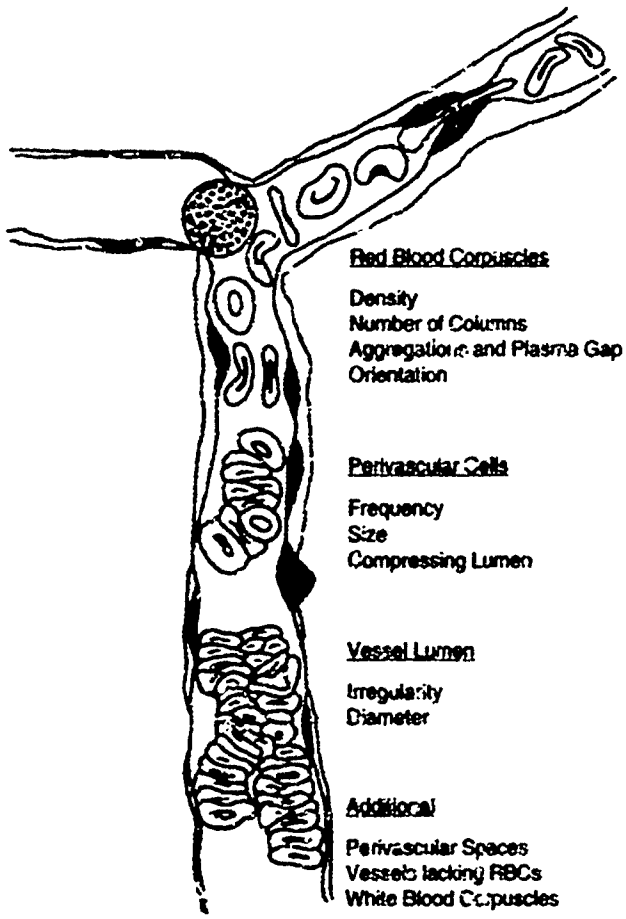

Fig. 7-16. Schematic representation of a cochlear vessel in which subjectively evaluated vascular parameters are depicted. Reprinted with permission from Axelsson A, Vertes D. Histological findings in cochlea vessels after noise. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss*. New York: Raven Press; 1982.

Carbogen (95% oxygen and 5% carbon dioxide), rich in oxygen compared to ambient air, was inhaled in different experimental noise-exposure paradigms that utilized both humans and chinchillas as subjects. The assumption was that increasing the oxygen available to the cochlea would (*a*) offset the detrimental effects of the vascular changes and (*b*) render the cochlear structures less susceptible to the hazards of noise. Both post- and prestimulatory carbogen inhalation decreased TTS, increased recovery rate, and, in chinchillas, decreased sensory cell damage. These results were considered preliminary, although apparently little follow-up research has been conducted in this area.

Another study tested the drug Dextran-40, but the evidence, although promising, was inconclusive.[31] During the 1970s, the West German army used Dextran-40 to treat military personnel who had sustained acoustic trauma. Dextran-40 supposedly increased circulation to the cochlea, and thus was assumed to facilitate the recovery process. While the reported results indicated that the treatment was effective, the experimental design—the selection of subjects and the lack of appropriate controls—limited the application of the results.[31]

Inner and Outer Hair Cell Loss

The inner and outer hair cells are morphologically and functionally different classes of cells. The two cell types differ in their size, shape, cochlear organization, cell-support system, stereocilia pattern, resting potential, and neural-innervation pattern. Their respective roles in audition are still being studied, refined, and changed.

Early cochlear dissection techniques provided information on hair cell counts, such as the number of inner hair cells compared to the number of outer hair cells that remained after exposure to noise. Today we are capable of more extensively analyzing cochlear hair cells for their qualitative morphological changes as well as for quantitative changes after exposure to noise. For example, simply counting the hair cells that remain after exposure to noise is not conclusive because myriad pathologies may exist in the remaining hair cells.

It is still generally true that, following exposure to high-intensity noise, outer hair cell loss occurs before inner hair cell loss. While outer hair cells are more susceptible to damage from noise, losing inner hair cells causes much greater hearing loss than does losing an equal number of outer hair cells. Data from experiments with chinchillas show that exposures to impulse noise may produce lesions with massive outer hair cell loss over 80% of the cochlea, but hearing sensitivity losses in the same animals seldom exceed 40 dB.[14] The data also show that, with lesions localized to the middle of the cochlea, hearing thresholds may be near normal if outer hair cell loss does not exceed 10% to 30%.[14] In both instances, researchers found that the inner hair cells appeared quite normal.

Much of the susceptibility of the outer hair cells can be explained by two mechanical factors: first, the outer hair cells undergo greater displacement due to their location on the basilar membrane, and second, the tips of the stereocilia of the outer hair cells are embedded in the undersurface of the tectorial membrane, which allows more direct, mechanical movement and consequent stress. In contrast, the cilia of the inner hair cells are not embedded in the tectorial membrane, and their

movement occurs not from mechanical linkage but from eddy current through the endolymph.

An additional factor that may play a role in this susceptibility is the pattern of neural innervation. The outer hair cells are innervated directly by efferent neural fibers. Although the role of the efferent fibers is still being studied, without them, the susceptibility of the outer hair cells may be even greater.

Outer hair cells are also known to be more susceptible to ototoxic drugs than are inner hair cells, so the outer hair cells' increased susceptibility to noise is probably due to more than just mechanical or neural factors. Specific metabolic processes of outer hair cells that differentiate them from inner hair cells appear to be partially, if not entirely, responsible, but these metabolic processes have yet to be delineated.

Stereocilia and Rootlet Damage

After the composition and structure of stereocilia were identified, researchers investigated morphological changes in stereocilia that followed exposure to noise.[32-35] Hair cell stereocilia are actin filaments, with crossbridges between filaments providing rigidity to the stereocilia. The movement of the stereocilia causes ion flow across the hair cell membrane and a subsequent voltage change: this is thought to initiate the release of the neurotransmitter substance. The stereocilia are very vulnerable to trauma from noise and may represent the weak link in the process of auditory transduction. Electron microscopic studies suggest that the rootlet structures, which anchor the cilia within the hair cell, are particularly susceptible to noise.

When PTS and stereocilia damage are correlated, most PTS can be directly linked to stereocilia damage. Following acoustic injury, subcellular stereocilia pathology can be found on hair cells that otherwise appear to be normal.[36] Permanent damage to stereocilia is documented in numerous studies and includes disarray, fusion, loss of the bundle, scarring of the bundles, and the appearance of floppy, giant, elongated stereocilia.

Temporary and Permanent Threshold Shifts

Noise can affect hearing either temporarily or permanently. Repeated TTS will presumably lead to PTS, although this presumption assumes that individual susceptibility to TTS and PTS are similar. Evidence from controlled laboratory experiments that demonstrate this relationship has not yet been acquired, although the hypothesis appears logical.

TTS studies are important in assessing noise hazards. Damage-risk criteria specify noise-exposure limits and their consequent associated risks. These criteria are based predominantly on animal experimentation in which damage, possibly TTS, occurred over a period of days to weeks. Real-world noise exposures experienced by humans in their workplaces, can last for 40 or more years and produce PTS. In the damage-risk criteria process, TTS measures are utilized and those results are extrapolated to PTS. Three postulates relating TTS and PTS were developed for the purposes of defining damage-risk criteria:

1. TTS 2 minutes after exposure is a consistent measure of the effects of a single day's noise exposure.
2. All exposures that produce a given TTS 2 minutes after exposure are equally hazardous.
3. TTS 2 minutes after 1 day's exposure is approximately equal to PTS after 10 or more years' exposure.[37]

The correlation of TTS and PTS continues to concern researchers in auditory science. The time constraints of following the course of human hearing over a 25- to 40-year history of occupational noise exposure seems to ensure that studies on animals and TTS measurements will continue as methods of auditory research into the next century.

New information is providing some insight into the physiological relationships and differences between PTS and TTS. The biological bases of PTS are relatively well defined—stereocilia damage with changes in rootlet structure and hair cell bodies—and pathological conditions of stereocilia probably account for most PTS.[38] Quite simply, if there is no transduction process at the level of the sensory cell itself, there will be no response to auditory stimuli.

However, the biological bases of reversible TTS are subtle indeed[36] and many factors may be responsible. Several studies suggest that rootlet damage to stereocilia may change cochlear micromechanics and be responsible for TTS. Subtle changes in the stereocilia that may be transient in nature, such as initial stiffness or disarray of the stereocilia, are also under investigation. Some potentially reversible factors include vascular changes, metabolic exhaustion, and chemical changes in the hair cells.

Correlation Between the Audiogram and Histological Damage

An audiogram does not accurately predict either cochlear pathology or the integrity of the inner ear. Health professionals who are responsible for identifying noise-induced hearing loss rely on the audiogram

for determining cochlear damage, but they generally do not understand that the audiometric configuration does not completely reflect the cellular condition of the cochlea. There may be considerable cochlear damage in the apical region, with normal hearing registered in the low-frequency portion of an audiogram.[14,39,40] In fact, it is probably impossible to predict the complex pattern of cochlear pathology from an audiogram.[41] The fact that a patient can hear does not mean that the cochlea is not damaged. An audiogram will provide the physician with a complete picture of a person's hearing as measured by an audiometer, but physicians should not make the intuitive leap and assume that there is no cochlear pathology.

SUSCEPTIBILITY TO NOISE-INDUCED HEARING LOSS

For years, researchers have been interested in the many individual differences in susceptibility to noise-induced hearing loss. Demographic studies show a 50-to-60 dB variability in hearing threshold shifts among individuals with an identical history of industrial noise exposure.[42,43] While demographic studies are limited in their reconstruction of individuals' complete auditory histories, the variation in susceptibility to noise-induced hearing loss is nonetheless astounding. A number of anatomical factors may contribute to this variability, such as qualities of tympanic membranes, individual differences in the contours of ear canals, middle ear characteristics, and the sensitivity of the muscles of the middle ear. However, these factors alone do not account for the magnitude of variability.

Until this variability is well understood, our ability to identify damage-risk criteria will remain inexact. A combination of several factors probably determines susceptibility to auditory damage, although each factor's relative influence is as yet undefined. A clinical battery of auditory tests or a mathematical or statistical model that incorporates the predictive effects of each factor may ultimately be developed.

The military would greatly benefit if those soldiers who are at risk for noise-induced hearing loss could be identified. Identifying and following the soldiers who are susceptible could save money by eliminating the need to retrain them for other jobs. Awareness of a predisposition to noise-induced hearing loss could also influence early counseling on career choices.[44] Soldiers with a high susceptibility require an emphasis on auditory management, a more frequent review of hearing acuity, and more intensive training in the care and use of hearing protectors.

Stimuli Variables

Noise-induced hearing loss may be affected by the following stimuli variables: (*a*) combinations of continuous and impulse noise, (*b*) intermittent noise (ie, noise that has a rest time), and (*c*) exposure frequency (in Hz).

Combinations of Continuous and Impulse Noise

Demographic studies indicate that the development of hearing loss may be accelerated when individuals are exposed to noise environments that contain both continuous and impulse noise, compared with exposure to continuous noise alone.[19] These demographic studies support data from controlled laboratory experiments. Workers exposed to impact and continuous noise also show extreme variability in the incidence of PTS.[19] The literature supports an interaction between continuous and impulse noise under specific conditions: when impulse levels are greater than 147 dB SPL, and when the two noise types overlap both spectrally and temporally.[45] This has particular importance to armored divisions, which are often exposed to continuous- and impulse-noise combinations.

Intermittent Noise

For the same total energy transmitted, intermittent noise is thought to produce less hearing loss than continuous noise. Researchers experimenting with chinchillas found that intermittent exposures to noise produce less temporary and permanent hearing loss and less cochlear damage than continuous exposure to noise of an equal energy. Two variables, which they found must be considered with intermittent noise, are (1) recovery during the noise off-time (quiet time) and (2) reduced adaptation of the acoustic reflex.[46] Recently, another investigator reported that intermittent exposures appear to make the ear more resistive to noise injury; exposure to low-intensity noise for several days may reduce the amount of PTS from exposure to a higher-intensity noise.[47] The scientific world awaits further developments regarding this new information.

Exposure Frequency

Generally, for noise exposure of a moderate intensity, high-frequency sound damages a restricted area

of the basal region of the cochlea, and low-frequency sound damages both basal and apical areas of the cochlea. In experiments with chinchillas, researchers have analyzed cochlear damage related to (*a*) continuous exposure to low-frequency noise, (*b*) continuous exposure to high-frequency noise, and (*c*) interrupted noise exposures.[17,48-50] High-frequency (in this instance, a range of frequencies with a center frequency of 4000 Hz), moderate-intensity noise caused damage in the region of the organ of Corti that is basal to the frequency location of the basilar membrane that was tuned to that exposure. As the intensity increased, damage spread both basally and apically. Low-frequency (a range of frequencies with a center frequency of 500 Hz), moderate-intensity noise caused damage predominantly to the outer hair cells in a broad area of the low-frequency region. As duration and intensity of the noise increased, the damage included more outer hair cells, with additional lesions in the high-frequency basal portion of the cochlea. The damage was more severe in the basal area than in the apex, and was also more severe than the damage caused by the high-frequency exposures. The loss of inner hair cells did not begin to occur until many outer hair cells were damaged. (From 30% to 50% of the outer hair cells may be missing in the apical region of the cochlea before the low-frequency thresholds are affected.) Interestingly, when interrupted low-frequency noise was presented to the chinchillas (6 h of noise with 18 h of rest), damage in the low-frequency region of the cochlea was reduced significantly. No such protective effect was found for loss in the high-frequency region. This relationship is consistent with the 4000-Hz notch that first appears on an audiogram from noise-induced hearing loss, regardless of the frequency of the insulting noise.

Variables That Affect Susceptibility

Factors that affect susceptibility to noise-induced hearing loss are (*a*) ototoxic drugs, (*b*) physical characteristics, (*c*) previous noise-induced hearing loss, (*d*) vibration, and (*e*) other variables.

Ototoxic Drugs

The aminoglycoside antibiotics streptomycin and neomycin produce more auditory sensory damage when combined with noise than they do when they are administered without noise.[41,45,51] Aminoglycoside therapy may destroy sensory hair cells and the stria vascularis of the cochlea, although the magnitude of the interaction between noise and the drugs appears to depend both on the intensity of the noise and the dosage of the drug. Thus, a patient receiving aminoglycosides should be considered to be at increased risk of a threshold shift when he or she is exposed to loud noise.

Cisplatin, a drug used in the treatment of some cancers, can also significantly increase auditory damage from noise. Again, the magnitude of the interaction depends on the intensity of the noise. Studies with animals show high concentrations of cisplatin in the stria vascularis and identify this area as the site of the pathophysiology.[41]

Salicylates, which are associated with temporary hearing loss and tinnitus, have also been implicated in causing an increase of TTS when taken in conjunction with noise exposure. However, salicylates have not shown an increase in PTS with noise exposure.[51] The debate over possible synergy between salicylates and noise continues.

Physical Characteristics

Physical characteristics that have been studied relative to noise-induced hearing damage are (*a*) melanin content, (*b*) age, and (*c*) serum magnesium levels. Melanin is present in the inner ear and is assumed to be involved in the normal function of the auditory system, although its exact role is undefined. Furthermore, the relationship of the melanin content in the iris or the skin to the melanin content in the ear has not yet been established. Several studies have investigated the relationship of melanin to noise-induced hearing loss, and assert that individuals with less melanin in their irises (those with blue or green eyes) exhibit more noise-induced hearing loss than those with brown eyes. Similarly, retrospective studies of black and white industrial coworkers have suggested that black workers experience less hearing loss than white workers. But the differences may not be industry related and there appears to be little evidence that eye color or skin pigmentation can accurately predict an individual's susceptibility to noise-induced hearing loss.[45]

Unlike the tenuous relationship of melanin to noise-induced hearing loss, however, strong support exists for age-dependent changes in susceptibility. Evidence from studies with animals indicates that once the auditory periphery is fully developed, the younger the animal, the greater the damage from noise exposure.[45] Studies on mice show that the greatest hearing loss for younger animals occurs only at the higher-intensity exposures.[52] We assume that the hearing losses from noise exposure and age (presbycusis) combine; this is

the basis for using age-corrected hearing thresholds in compensation cases.

Similarly, studies with animals suggest a relationship between serum magnesium levels and differences in the susceptibility to noise-induced hearing loss. Magnesium is present in perilymph, and a deficiency in magnesium has been linked to energy depletion and irreversible damage to the hair cells.[53,54]

Previous Noise-Induced Hearing Loss

People who have a history of previous noise-induced hearing loss appear to have unchanged susceptibility to additional noise-induced hearing loss. Generally, one can expect to find less TTS as preexposure hearing threshold level increases. Literature on this subject concludes that (*a*) when the region of the basilar membrane that was injured by prior noise exposure coincides with the region that is affected by current noise exposure, the threshold shift is less in the impaired ear, but the resultant shifted thresholds are identical, and (*b*) when the region of the basilar membrane that was injured by prior noise does not coincide with the region that is affected by the current noise exposure, the total region of damage is the simple sum of the two.[45]

Vibration and Other Variables

Vibration has a small, consistent, minor effect on the sensitivity of human hearing.[41] Although researchers have found evidence of a relationship between noise and whole-body vibration, the degree of interaction appears small.[45] A recent investigation of noise and vibration interaction in chinchillas demonstrated relatively small and inconsistent effects on hearing and sensory cell populations. The researchers concluded that "an increased risk of noise-induced hearing loss from vibrations in the industrial population is probably relatively small."[55]

The preceding variables are in no way inclusive of all affecting agents. For example, gender, hormonal cycles and oral contraceptive use,[56,57] levels of carbon monoxide,[51] air temperature, and cigarette smoking[58] have all been investigated regarding their interaction with noise-induced threshold shifts. The psychological role of noise as a stressor and the alteration of the physiological processes mediated by the autonomic, central nervous, and endocrine systems have been reviewed.[59] The general theme of research in this area is the interaction of noise with conditions that result in peripheral vasoconstriction, an elevated heart rate, and increased blood pressure.[59]

Future Research Objectives

Much of the literature on the physiological effects of noise consists of data obtained from animals during relatively short exposures (days or weeks). Studies paralleling the damage to hearing that accumulates over a worker's lifetime in the real world are needed and should address

- models to predict hearing loss based on cellular damage relative to exposure character-istics[60];
- greater study of exposure to low-intensity noise emphasizing metabolic damage because many studies report on high-intensity exposures and emphasize mechanical damage;
- systematic descriptions that trace the physiological pathways of cellular injury and cellular degeneration quantitatively, to improve our understanding of the mechanisms that cause the anatomical change[1];
- determination of critical levels for damage from various types of noise;
- further refinement of the relationship between TTS and PTS and their accompanying anatomical correlates; and
- further investigation of interactive agents and environmental stressors that can affect noise-induced hearing loss.

HEARING IMPAIRMENT IN THE U.S. ARMY

Noise-induced hearing loss is one of the most prevalent occupational health impairments in the army. The magnitude of the problem can be estimated from the following sources: (*a*) a hearing-loss prevalence study conducted in 1975, (*b*) hearing-loss data from the U.S. Army's Hearing Evaluation Automated Registry System (HEARS), and (*c*) compensation expenditures.

The Hearing-Loss Prevalence Study

In 1975, audiometric data were obtained from 3,000 enlisted men representing three combat branches (infantry, armor, and artillery) and five time-in-service categories.[3] In this prevalence study, significant hearing loss was defined as that decrement in hearing that

enabled a soldier to qualify for an H-2 profile or worse. A detailed discussion of the hearing-profile system is beyond the scope of this chapter; however, the severity of hearing loss that a soldier must exhibit to obtain an H-2 profile can be illustrated: the upper limit of an H-1 profile defines the hearing sensitivity of a 70-year-old man.

This prevalence study produced the following salient findings:

- Approximately 20% to 30% of all combat-arms personnel with more than 1.5 years of service had significant hearing losses.
- Over 50% of combat-arms personnel with more than 15 years of service (the army's senior noncommissioned officers) had significant hearing losses.
- The prevalence of hearing loss was roughly the same in all three combat-arms branches.
- A substantial difference existed between the prevalence of hearing loss according to time-in-service, and this difference could not be explained on the basis of age.
- Most soldiers did not carry the appropriate profile for hearing; for example, the calculated profile from their last hearing test was different from the profile assigned to them.[3]

An update of this prevalence study is long overdue. After validation studies for threshold determination and more extensive analysis of personnel databases have been done, HEARS data will be used to reexamine hearing-loss prevalence in the army.

The Hearing Evaluation Automated Registry System

Approximately 500,000 Department of the Army (DA) military and civilian personnel are reported to be exposed routinely to hazardous noise.[61] From 1980 to 1990, almost 2 million audiometric evaluations of these individuals were accumulated in a mainframe database at Fort Detrick, Maryland. However, poor participation in HEARS has limited the value of the data that demonstrate its effectiveness. Figure 7-17 compares participation in the registry as a function of rank for enlisted personnel, and Figure 7-18 compares the prevalence of hearing loss by rank. Similar patterns exist for commissioned officers and warrant officers. By 1989, only 41% of all active-duty personnel were enrolled in HEARS. Even with this limited participation, the data indicate a high prevalence of hearing loss among military personnel. For example, 10% of all active-duty warrant officers and 6% of all enlisted and officer personnel in HEARS have an H-3 or worse hearing profile. This H-3 profile signifies a substantial hearing loss, for which some soldiers may require both a hearing aid and reclassification from a noise-hazardous occupation. If all of these individuals were reassigned to jobs that were free of noise hazards, the disruptions in work schedules and increased training costs would be substantial. Although data on training costs are calculated on a case-by-case basis and are not readily available through medical channels, high costs would be incurred by training personnel for entry into the noise-hazardous specialty, retraining personnel for reassignment to a noise-free job specialty, and training the replacement personnel in the original noise-hazardous specialty.[61]

The prevalence of hearing loss among civilians is calculated in HEARS under the Department of Labor (DOL) hearing-loss formula in terms of the percentage of hearing loss and potential monetary compensation. Of the 82,716 civilians in HEARS, 13,449 (16%) have potentially compensable hearing levels, as calculated from the results of their last hearing test.[61] Currently, the army's potential compensation liability would total approximately $93 million if all 13,449 individuals filed and were found to be compensable.[61]

Compensation Expenditures

Although the figures do not reflect the more important factors—decreased quality of life and decreased job performance—that are associated with communication handicaps from hearing loss, the DoD's compensation expenditures have been staggering (Figure 7-19).[61] In 1990, the army was credited for 39,271 of the total 62,012 cases of primary hearing-loss disability (ie, when hearing loss is the greatest or only disability) and for 91,443 of the 171,192 secondary disabilities (ie, when hearing loss is one of several compensable disabilities) (Figure 7-20).[61] But two additional points must be noted: (1) there are undetermined expenditures for other disabilities computed into the primary hearing-loss figures, and (2) these expenditures are funded through a separate Veterans Administration (VA) budget to which DoD agencies are not accountable.

In February 1987, the DOL, which administers workers compensation for all civilian federal employees, adopted the hearing-impairment formula of the American Academy of Otolaryngology. The new formula added 500 Hz to the frequencies 1000 Hz, 2000 Hz, and 3000 Hz that were already in use. Since 500 Hz is a frequency that is less affected by noise, the use of this

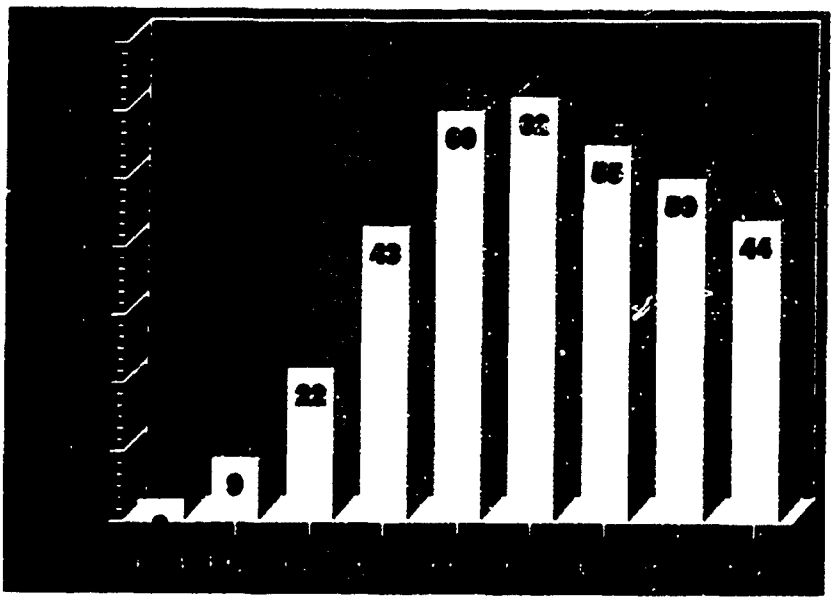

Fig. 7-17. Percentages of enlisted soldiers with a reference audiogram as a function of rank. An untimely reference audiogram and lack of subsequent audiometric monitoring precludes early detection of hearing loss. Source: Donahue AM. *Hearing Conservation Data Profile*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

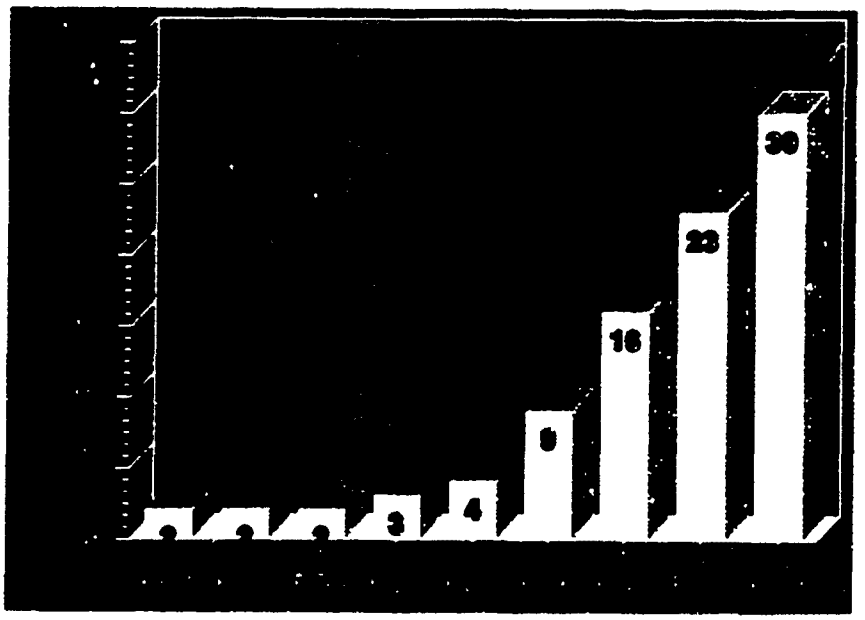

Fig. 7-18. Percentage of enlisted soldiers with a hearing profile worse than H-2 as a function of rank. The prevalence of hearing loss increases with rank and presumably with time in service. These increases are not attributable to aging. Source: Donahue AM. *Hearing Conservation Data Profile*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

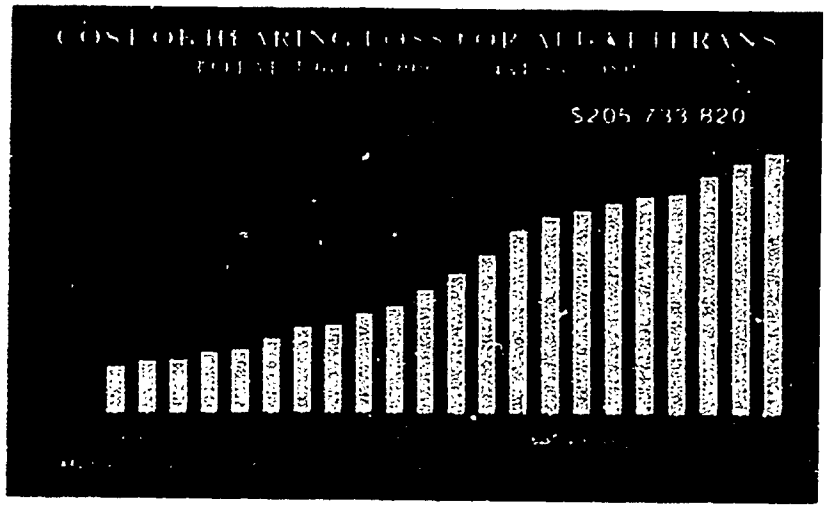

Fig. 7-19. Expenditures over the last 22 years for veterans in all services receiving hearing-loss compensation who had hearing loss as their primary disability. In the calendar year 1990, the army accounted for 67% ($138,138,804) of total primary-disability expenditures ($205,733,820). The total cost ($2,481,817,080) for 1969 to 1990 does not include expenditures for secondary-disability cases of hearing loss. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

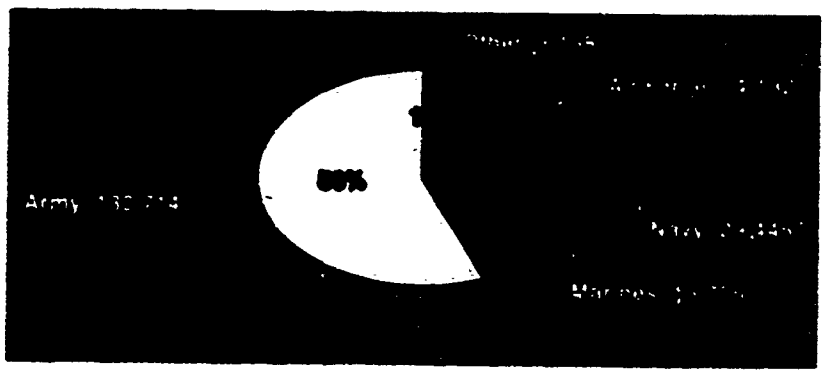

Fig. 7-20. The distribution of hearing-loss disability cases (primary and secondary) among the services. Veterans are included for calendar year 1990 only. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

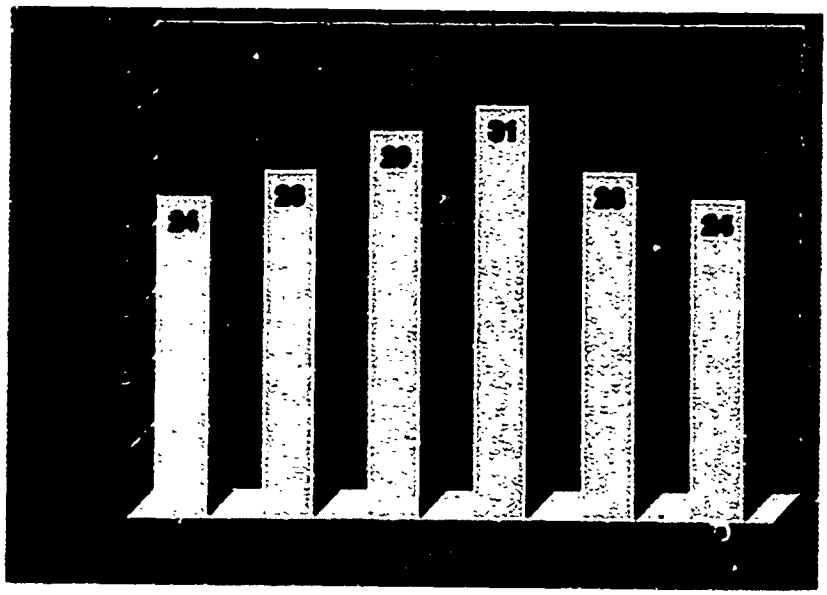

Fig. 7-21. Expenditures for civilian hearing-loss compensation for all federal agencies for fiscal years 1983 to 1988. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

formula reduced the number of awarded compensation claims for that year (Figure 7-21).

The latest data available from fiscal year 1990 show that the hearing-loss bill for all government agencies totaled $27,451,585, which constitutes 2% of the total monies paid for all forms of compensation ($1,440,980,764). The army was charged for 23% ($6,360,205) of the total hearing-loss bill, and accounted for 17% (908) of the 5,375 cases adjudicated. On average, the rate of hearing loss in the army was twice as high as the rate in the total federal government (4% versus 2%).[61] Moreover, since all civilian compensation is a charge back to the original agency, there is concern for reducing compensation at the DA level, where, in turn, charge backs are being directed down to the major command level and, eventually, to the installation. Current data portray a problem of sizable proportions; nevertheless, organizational accountability is linked to an accurate definition of this problem.

HEARING CONSERVATION IN THE U.S. ARMY

Noise-induced hearing loss is not a recent phenomenon in the army, nor are efforts to prevent it. Although it may seem to be contradictory, noise-induced hearing loss and measures to prevent it have coexisted for almost five decades. As the magnitude of this problem indicates, the army's efforts to prevent noise-induced hearing loss have not been entirely successful.

The development of military hearing conservation programs has been linked to the evolution of specialties in the fields of audition, speech science, psychoacoustics, and bioacoustics.[62] Milestones for army hearing conservation programs can also be linked to the establishment of facilities and the publication of key hearing conservation documents.

Facilities

Early army initiatives can be traced back to December 1941, when a research facility at Fort Knox, Kentucky, was established. Topics of investigation included the effects of noise on personnel efficiency, the nature of the temporary deafness that was caused by tank noise, and the physiological adaptation to tank noise. In August 1944, a project that addressed these topics recommended that gun crews, gunnery instructors, and other personnel who were exposed regularly to gunfire blasts be provided with hearing-protective devices.[63] The army procured a single-flange earplug, the V-51R, for general issue to those who required protection.

In 1942, the forerunner of the U.S. Army Environmental Hygiene Agency (USAEHA), the Industrial Hygiene Agency, was established at The Johns Hopkins University. For the next 27 years, hearing conservation was largely an industrial hygiene function, both within this agency and in the field, with an emphasis on the identification of noise hazards. In 1969, a military audiologist was assigned to the USAEHA, which was then located at Edgewood Arsenal, Maryland. In that same year, the Bio-Acoustics Division was created at the agency with the mission to provide consultation and advice in the medical, engineering, and administrative aspects of hearing conservation.

While the Bio-Acoustics Division was concerned with studying operational noise problems and monitoring the effectiveness of the Hearing Conservation Program, other laboratories were established, including the U.S. Army Audiology and Speech Center at Walter Reed Army Medical Center, Washington, D.C.; the U.S. Army Aeromedical Research Laboratory, Fort Rucker, Alabama; and the Human Engineering Laboratory, Aberdeen Proving Ground, Maryland. These laboratories were established to investigate noise-related problems involving protective equipment design, hearing loss, hearing protection, aural detectability, performance decrements caused by hearing loss, and aural rehabilitation.

Military Audiology and Other Disciplines

The specialty of audiology emerged from aural-rehabilitation centers that were established after World War II. By the late 1960s, there were 11 audiologists on active duty.[64,65] Today, more than 65 officers serve dual roles as clinical audiologists assigned to army hospitals or other medical installations or activities, and hearing-conservation officers who assist the local preventive medicine officer. They have the general responsibilities of monitoring and implementing the local Hearing Conservation Program. In this role, the audiologist is the responsible action officer for hearing conservation.

Because it is impossible for any one action officer to perform all hearing-conservation functions, the army employs a team approach. Although the disposition of resources is at the discretion of local commanders, program responsibilities for other related disciplines have evolved:

- Industrial hygienists have the primary responsibility for noise-hazard evaluation, and work

closely with facilities engineers to design and retrofit engineering noise controls.
- Occupational health nurses perform medical procedures such as fitting earplugs and providing audiometric testing for the civilian population, and manage programs in the absence of audiologists.
- Safety personnel perform a vital role in posting areas and equipment and enforcing the use of hearing protectors.
- Occupational health physicians have the final word in medical decisions and recommendations.
- Physician assistants, military corpsmen, civilian health technicians, and others also assist in accomplishing Hearing Conservation Program responsibilities.

Key Documents

Noise standards (ie, requirements for program implementation by the federal government) and documents that implement hearing conservation programs represent significant milestones in the development of these programs. The first document with standards was U.S. Air Force Regulation 160-3, which was issued in 1956.[66,67] For the next 25 years, the air force maintained the most well-established hearing conservation program in the military.

The army issued an implementing document in 1956 and revised it in 1965 and 1972.[68-70] However, the U.S. Army's Technical Bulletin, *Noise and Conservation of Hearing*, known as TB MED 251, did not include the Hearing Conservation Program requirements, but only recommendations for its implementation. The requirements for a program were outlined in a basic preventive medicine regulation, U.S. Army Regulation (AR) 40-5, which referred to the technical bulletin.[71] Unfortunately, because only a program outline was required by the regulation, only an outline existed. In 1977, the General Accounting Office (GAO) recommended that the DoD adopt a uniform policy on noise exposure.[72] A year later, a DoD Instruction (DoDI) was published to provide standards as well as uniformity to military hearing conservation programs.[73] The army's implementing document to the DoDI—TB MED 501, *Hearing Conservation*—was published in 1980.[74]

Federal noise standards evolved similarly, and the DoD implemented standards to parallel the federal regulations. The Walsh-Healy Public Contracts Act, *Noise Standard*, published in 1969, was incorporated into The Occupational Safety and Health Act of 1970 (OSHAct).[75] The noise section contained fewer than 350 words, with a key provision that required a "continuing, effective hearing conservation program" whenever a table of allowable levels and durations was exceeded.[75] After a protracted process of debate and comment on the specific requirements necessary for this program, a final noise standard was published in 1983.[76] The DoD implemented the directives of the 1983 Federal Noise Amendment in an update of the DoDI, which was published under a new designation, DoDI 6055.12.[77] Including policy issues such as assigning specific responsibilities has upgraded the army's implementing document to DA pamphlet status, designated as DA PAM 40-501.[78]

Noise-Hazard Criteria

Most aspects of noise-hazard evaluation in the army mirror those in the private sector, although some aspects are militarily unique. Both in private industry and in the army's program, industrial hygiene personnel evaluate potential hazards with noise-measuring equipment that is calibrated to the standards of the American National Standards Institute.[79] Unique features of the army program include more stringent noise-exposure criteria and the pervasiveness of high-intensity, impulse-noise sources.

The army has established noise-exposure criteria according to the specific type of noise: (*a*) continuous, (*b*) airborne high-frequency and ultrasonic, and (*c*) impulse.

For continuous noise, the army employs a modified version of the DoD criterion: as exposure time is doubled, a 4-dB decrease in intensity is enforced or suggested. For example, 85 dBA (ie, a weighting network for hearing—conservation—exposure criteria) is hazardous for 8 hours, so 89 dBA is hazardous for 4 hours. These criteria have also been extrapolated for noise exposure for longer than 8 hours. The establishment of representative time-weighted averages (TWAs) for civilian and military personnel working in industrial operations is in progress. For the purposes of administering the Hearing Conservation Program, levels of steady noise of 85 dBA or greater are presently considered hazardous, regardless of the duration of the exposure.[70] Practical guidance to preclude misuse or overzealous implementation of a single-number criterion were provided in implementing documents:

> This criterion affords the advantage of increasing the overall efficiency of the program by simplifying its administrative aspects....It will also better protect

those individuals who are more susceptible to the effects of noise. Although the requirements of the program demand the initiation of hearing conservation measures when levels are 85 dBA or greater, the implementation of all available measures may not be necessary in every case. For example, visitors to noise-hazardous areas are required to wear hearing protective devices, but the requirement for hearing evaluations does not apply to visitors. There may also be unique situations where noise levels rise infrequently and unpredictably to 85 dBA or greater for very short durations so that the wearing of hearing protective devices may be judged impractical or unnecessary. Decisions to waive the wearing of hearing protective devices or any other requirement of the program must not be made arbitrarily. Such judgments may be rendered by trained AMEDD [Army Medical Department] personnel who will perform a thorough evaluation using approved instrumentation and who will consider all factors relative to the potential for a given exposure to cause hearing impairment.[74(p3)]

Airborne High-Frequency and Ultrasonic Noise

Exposure to airborne high-frequency and ultrasonic noise occurs at army installations from various sources such as industrial cleaners and degreasers, dental drills and scalers, and aircraft compressors. The army has adopted the recommended Threshold Limit Values (TLV) of the American Conference of Governmental Industrial Hygienists (ACGIH) for potentially hazardous high-frequency and ultrasonic noise sources (Table 7-1).[80] Durations of permissible exposure are not included in these values, but only single-decibel levels in one-third octave bands are included. Decibel levels for one-third octaves above 20,000 Hz (ie, ultrasonic noise) were included to prevent possible hearing loss from the subharmonics of those frequencies that we do hear and that are generated within the ear. Equipment for measuring noise in the one-third octave bands is usually not available at local installations, but can be obtained from the USAEHA.

Impulse Noise

The impulse-noise exposures and the multiplicity of impulse-noise sources in the army environment dictate that the requirements of the hearing conservation program be mandatory. Where the Occupational Safety and Health Agency (OSHA) requires that exposures *should* not exceed 140 dBP (ie, criterion for exposure to impulse noise), the army dictates that exposures *must* not exceed this level.[74,75] Because the army uses small-arms ammunition (including blanks) that produce impulse-noise levels above 140 dBP, measures to conserve hearing must be instituted and enforced when weapons are fired during training.[74]

The single criterion of 140 dBP—notwithstanding several parameters—defines the hazard of impulse noise. These parameters include (*a*) peak decibel (or intensity) level, (*b*) frequency content, (*c*) number of impulses, (*d*) duration of each impulse, and (*e*) the angle of incidence of the incoming sound wave.

The higher the peak intensity, the more hazardous the noise.[81,82] Shoulder-fired, antitank rockets such as the Dragon can have peak intensities as loud as 185 dBP at the firer's ear. Artillery fire can exceed 180 dBP, depending on the charge, the length of the tube, the angle of fire, and the presence and type of muzzle brake. Mortars, depending on their charge and caliber, can produce intensities from 165 to 178 dBP. Rifle and pistol fire will measure 156 to 162 dBP at the firer's more exposed ear. Generally, the same peak intensity from artillery fire will be considerably less hazardous than that of rifle fire because artillery fire is of a lower frequency content.[83] The total noise hazard from shoulder-fired rockets was not great because they were expensive to test fire until simulators were developed that cost only pennies per shot.

The noise hazard also increases as the number of impulses increases over a given time.[81,82] As a general rule, the larger the caliber and the louder the weapons system, the fewer the impulses that are generated. In addition, the noise hazard increases with the duration of the impulse. Reverberations from reflected surfaces can lengthen the impulse.[81]

The angle of incidence also affects the severity of the noise hazard. The more the impulse impinges directly on the ear, the more hazardous it is. For example, the

TABLE 7-1

PERMISSIBLE NOISE LEVELS OF AIRBORNE HIGH-FREQUENCY AND ULTRASONIC RADIATION

One-Third Octave Band Center Frequency (kHz)	One-Third Octave Band Intensity Level (dB)
10	80
12.5	80
16	80
20	105
25	110
31.5	115
40	115

right ear (for right-handed shooters) is partially protected from the sound of the rifle fire by the "shadow" of the shooter's head.

All these factors are uniquely combined in mortar fire, which render it excessively hazardous to mortar crews. But the M16 rifle, because of its widespread use, potential rate of fire, and relative high-frequency content, has the dubious distinction of being the primary destroyer of hearing in the army.

Posting

The army emphasizes prominent posting of noise-hazardous areas and equipment with appropriate danger signs and decals.[74,84] For equipment that generates 85 dBA and 140 dBP noise-hazardous fields, signs must be posted to identify these contours.[74]

Although compliance is not guaranteed, a direct correlation has been observed between the presence of signs and the use of required hearing protectors.

Noise Controls

Engineering and administrative noise controls are essential components of a hearing protection program. Engineering controls are desirable; their use eliminates the noise hazard and renders other components of the Hearing Conservation Program unnecessary.[74] Administrative controls are generally employed when hearing protection cannot protect soldiers or civilian employees from a given exposure.

Noise reduction that employs engineering methods is based mainly on applying certain principles of the science of sound. Solving complex noise-control problems usually requires the services of acoustic engineers, who are available at the Bio-Acoustics Division of the USAEHA.[74] However, the industrial hygienist, audiologist, environmental scientist, or preventive medicine officer with a general understanding of acoustic principles can recommend measures that will often control many noise problems successfully.[74]

Engineering Controls

Engineering controls are used whenever feasible to reduce continuous noise to below 85 dBA and impulse-noise intensities to below 140 dBP (or to the extent possible).[74] Engineering noise control is generally feasible if implementation is practicable and cost effective, both technologically and operationally.[74] Engineering measures may involve significant expenditures, and thus

> installation planners must establish priorities so that available funds will yield the greatest benefits. Such priorities must be based on factors such as the number of personnel exposed to a particular noise source, future intended use of the facility, as well as the level and the duration of exposure.[74(p4)]

Two programs that complement engineering noise controls are the Health Hazard Assessment (HHA) process, which is discussed in detail in Chapter 6, Health Hazard Assessments, and the Quiet Tracked Vehicle Program (QTVP). The HHA process attempts to ensure that hardware design and procurements conform to both Military Standard (MIL STD) 1474 and medical policy for noise exposure, but all military materiel procured before the initiation of the HHA process were not subjected to any restrictions that may have been recommended through an HHA. Newly designed or purchased equipment, however, must exhibit the lowest possible noise-emission levels and conform to the acoustic noise limits prescribed in MIL STD 1474.[74,85]

Similarly, the QTVP has contributed to engineering noise controls. The high levels of noise produced by tracked vehicles have been a problem historically and are responsible not only for hearing loss, but also for both degraded communication and aural detection at great distances.[86–88] A 15-year effort has produced a compliant sprocket and an isolated roadarm and roundwheel to reduce the noise associated with tank movement. These innovations were incorporated into a demonstration vehicle; interior noise was reduced by 8 to 10 dBA and exterior noise by 3 to 4 dBA.[88] The durability of the reduced-noise suspension system is still under study.

Administrative Controls

Administrative controls to limit noise exposure are not always practical in army industrial operations. The characteristic understaffing of the federal civilian workforce can limit the use of administrative controls such as the rotation of workers through different job areas to limit noise exposures. These restrictions may be more practical in military training, however. For example, limits can be set on the number of rounds of ammunition fired or on the peacetime use of a particular weapons system.

In the design and procurement of equipment in the HHA process, administrative controls can limit the number of rounds fired by writing the appropriate guidance in the operator's manuals. These administrative controls are crucial; the nature of most noise

sources evaluated in the HHA process has defied reduction through engineering. However, an aggressive approach toward engineering controls at the Fort Belvoir Research and Development Center has produced some positive results. Researchers at the center have reduced noise on new military equipment such as generators and water purifiers, which have counterparts in the private sector

Personal Protective Equipment

Protecting hearing in the army is doubly challenging. First, many noise sources are not amenable to engineering controls, which increases the wearers' reliance on hearing protectors. And, second, use of the Kevlar helmet dictates the use of earplugs, which creates a dilemma: unless an expert inspects the seating of the plugs, only the user knows whether or not they are inserted properly. Consequently, the emphasis on promoting the proper use and care of hearing protectors that was initiated over 20 years ago continues today.

Only personal protective equipment (PPE) that has been approved by the Office of The Surgeon General (OTSG) is authorized for use. The nomenclature and National Stock Numbers of approved hearing protectors are included in DA Pamphlet 40-501.[78] These protectors have been tested thoroughly for their attenuation characteristics, durability, and freedom from toxic effects. Not only have all commercially available devices not been tested in this manner, but they also cost considerably more than those that are ordered through army supply channels. The army uses a carefully selected set of hearing protectors (Figure 7-22) including (*a*) preformed earplugs (triple- and single-flange), (*b*) hand-formed earplugs, (*c*) ear-canal caps, (*d*) noise muffs, and (*e*) noise-attenuating helmets. All hearing protectors are issued gratis, and a freedom of choice among these approved devices is required by the DoD unless the choice is medically or environmentally contraindicated.

Preformed earplugs include (*a*) the triple-flange earplug, which predominates because of its ease of fit and consequent popularity among soldiers, and (*b*) the single-flange earplug, the V-51R, which was developed over 45 years ago, and is used as a backup plug for difficult-to-fit cases, particularly those soldiers whose ear canals are excessively crooked. Although both of these preformed earplugs are available in the private sector, only the military color-codes them according to size and mandates that all sizes be available for fitting and issue.

Although the army sometimes uses hand-formed earplugs of foam or silicone, those installations that use them in large numbers often distribute them without proper instruction. This is reflected in data that show increased hearing threshold shifts among large numbers of personnel who are reported to be hand-formed earplug users. Hand-formed earplugs are best used for visitors or other transient personnel who do not have their fitted hearing protectors with them at the time.

Although noise muffs, ear-canal caps, and noise-attenuating helmets are also approved for use, they are used less frequently. Installations with an industrial base should use more noise muffs, because they more effectively protect against intermittent noise. Noise muffs are available as safety devices and are worn with suspension systems over the head, behind the head, or under the chin. Authorization has been granted to

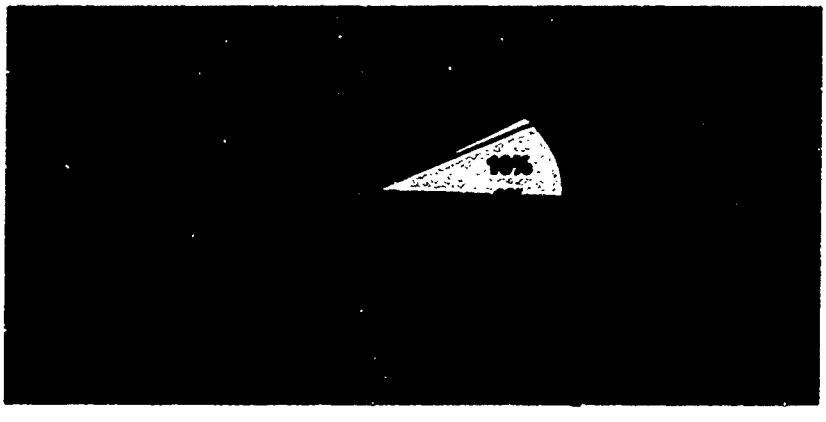

Fig. 7-22. The types of hearing protection used in the US Army. These data were obtained from 433,421 reference audiograms conducted from 1985 to 1989. Source: Donahue AM. *Hearing Conservation Data Profile*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

purchase noise muffs from commercial sources. Commercially available recreational muffs with built-in radios are not approved for hearing protection. Sound levels from radio earphones may pose a potential auditory hazard as well as a safety hazard because warning signals may not be heard. In comparison, ear-canal caps are a medical item and are restricted to noise environments under 95 dBA.[74] Significant differences exist in the issue and maintenance of the two types of noise-attenuating helmets: aviator helmets (SPH-4 and IHADDS) and the armored-vehicle crew-member helmets (DH-132). Aviator helmets are items of individual issue, are fitted individually, and are well maintained, but the armored-vehicle helmets are not. Every time these helmets are checked in the field, between 30% to 40% of them are unserviceable because of missing or hardened earcup seals or missing chin straps or both.

Earplugs are invasive medical devices that must be ordered through medical-supply channels and, in the case of sized preformed earplugs, must be fitted under medical supervision.[78] In contrast to the private sector, where less than 20% of the occupational health–hearing conservation programs maintain records on hearing protectors, the army closely monitors sizing distributions.[89] Although neither of the sizing distributions shown in Figures 7-23 and 7-24 is considered ideal,

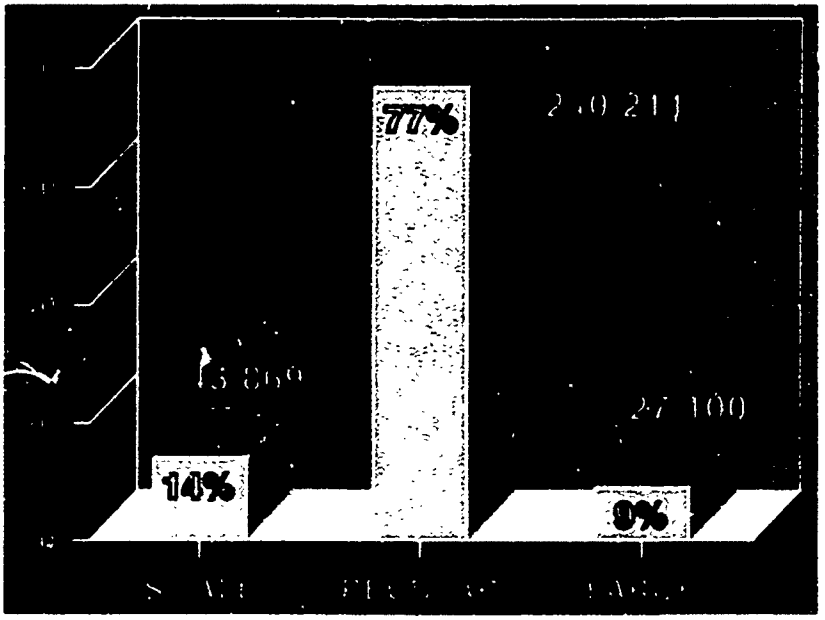

Fig. 7-23. The sizing distribution of 311,180 pairs of triple-flange earplugs fitted from 1985 to 1989. The Hearing Evaluation Automated Registry System (HEARS) program counts the size of the left earplug only. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

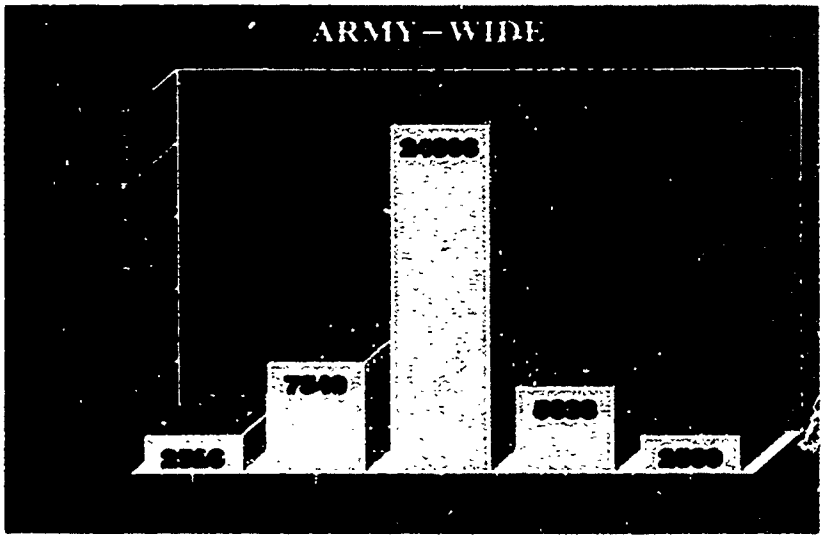

Fig. 7-24. The sizing distribution of 42,419 pairs of single-flange earplugs fitted from 1985 to 1989. Only the size of the left earplug was counted. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

data will be available in the next few years that will be used to issue standards on appropriate sizing distributions, depending on age and gender. For the present, trends are identified armywide and locally to monitor fitting procedures. For example, an increase in average age is expected with an increasing size of earplugs, and females are expected to be skewed toward the smaller sizes. The data available on the number of individuals who require a different-sized earplug in each ear are also suspect (Figure 7-25). Under carefully controlled fitting environments, at least 5% to 8% of personnel using the single-flange earplug have been found to require a different size in each ear. Only 1% to 2% of those who use triple-flange earplugs require different sizes.

Medical personnel are instructed in fitting techniques with an emphasis on comfort and proper seal. They are also taught to exploit and anticipate problems associated with the occlusion effect. For example, if earplugs (or other types of hearing protectors) are worn properly, the individual's own voice will sound lower in pitch to him or her. In addition, an individual's tinnitus will be more apparent when hearing protection is worn, particularly when the earplugs are fitted in a quiet clinic or classroom. Other issues such as excessive cerumen and the cough reflex are addressed in training materials.[90]

The army has developed an olive drab earplug carrying case that blends with the color of the battle dress uniform (BDU) and does not reflect light. Commanders should be encouraged to require that the case and earplugs be worn on the BDUs to ensure their availability.[74]

An earplug-insertion and -seating device is also included in the carrying case for the two preformed earplugs. The earplugs must be soft and compliant for the wearer's comfort and able to obtain a proper seal; however, individuals whose fingers are wide and blunt will have difficulty inserting their earplugs properly. Seating devices make insertion easier for these wearers (Figure 7-26) and improve noise reduction.[91]

Noise reduction ratings (NRR) that are obtained in laboratories with experimenter-supervised fittings have proven to be virtually worthless. Numerous studies have demonstrated the futility of attempting to predict protection in the workplace based on NRRs.[92-96] The army's approach uses (*a*) an approved set of high-quality hearing protective devices; (*b*) emphasis on proper fit and instruction; and (*c*) single-number, across-the-board limits for noise exposures. DA PAM 40-501 contains tables that detail these limits.

Theoretically, almost all noise-induced hearing loss that is incurred during routine training exercises is preventable if approved hearing protectors are properly used. However, an obvious gap exists between theory and reality: hearing conservation experts often say that the best hearing protector is the one that is *worn*. The expectation that protective devices will be worn only when the policy is enforced rather than when the devices are indicated may have been realistic for hearing conservation programs in their developmental stages. However, current army occupational health programs have reached a level of sophistication and, therefore, the expectation and provision of adequate hearing-protective measures should be raised.

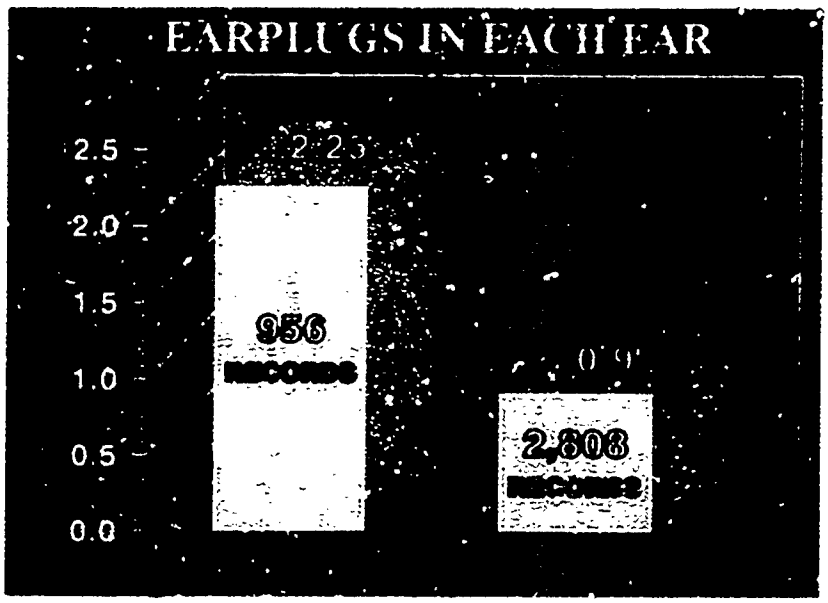

Fig. 7-25. These distributions pertain to the armywide data reported in Figs. 7-23 and 7-24. If they are fitted properly, the percentage of different sizes of single-flange earplugs should be at least 5% to 8%: 42,419 single-flange earplugs and 311,180 triple-flange earplugs from 1985 to 1989. Source: Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Armywide database 51-34-0251-91.

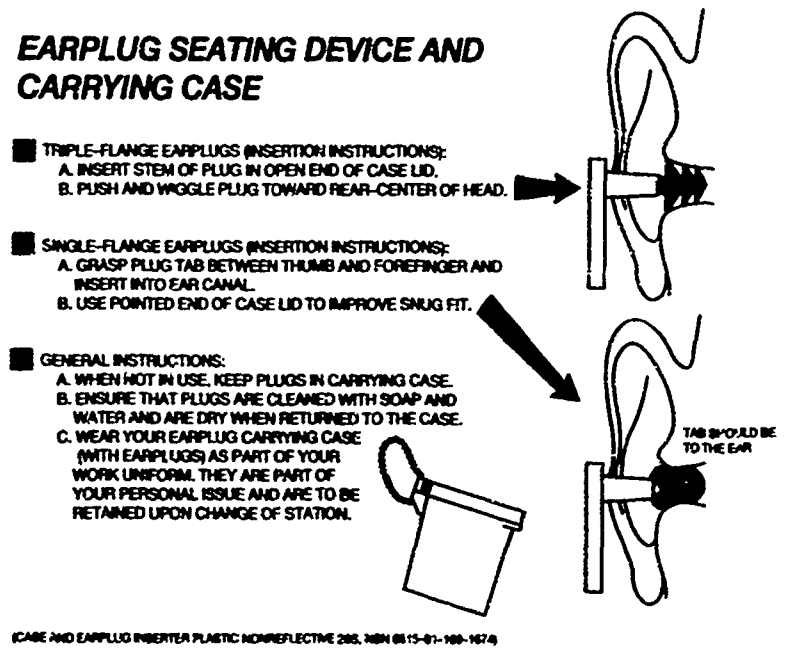

Fig. 7-26. This instructional poster provides insertion instructions and shows the seating device and carrying case for preformed earplugs. Source: DA Poster 40-501E, April 1991.

The Military Occupational Health Vehicle

Visits to fixed-site health facilities for medical surveillance can remove personnel from their jobs for up to one-half a workday. Military audiologists originally used military occupational health vehicles (MOHVs) with audiometric-testing capabilities to alleviate this problem at Forts Carson, Bragg, Knox, and Campbell. Multiphasic testing capability was developed in an MOHV at Fort Eustis. In the spring of 1988, the army fielded MOHVs with capabilities including audiometry, vision screening, pulmonary-function testing, blood-pressure screening, electrocardiography, and venipuncture to 16 major installations.

Hearing conservation activities occupy most of the space and the operation time of these MOHVs (Figure 7-27). An orientation room is used for fitting earplugs and for health-education activities that are facilitated by a television monitor, earphones, and a video cassette recorder.

Audiometric Monitoring

Audiometric monitoring detects changes in hearing sensitivity. Individuals who are susceptible to noise-induced hearing loss can be identified before their hearing sensitivity evolves into a communication handicap. In addition, statistical trends of hearing threshold shifts can be used to determine the effectiveness of hearing conservation programs.

The HEARS registry is a part of the Occupational Health Management Information System (OHMIS) and provides automated testing and data to a local manager's module. HEARS is also designed to transfer audiometric information from the installation to its armywide database. Quarterly, the HEARS database is compared to personnel tapes, and audiograms of former government employees are archived. The flow of information is circular, with the armywide database providing needed information to the installation manager's module and audiometer sites (Figure 7-28). Access to the database is limited to the OTSG, Health Services Command (HSC) Headquarters, and the functional proponent for the system, the Bio-Acoustics Division of USAEHA. Armywide and major army command comparative data are included in a user's guide. The operations of the HEARS audiometer and manager's module are detailed in USAEHA Technical Guides 167A and 167B.[97,98]

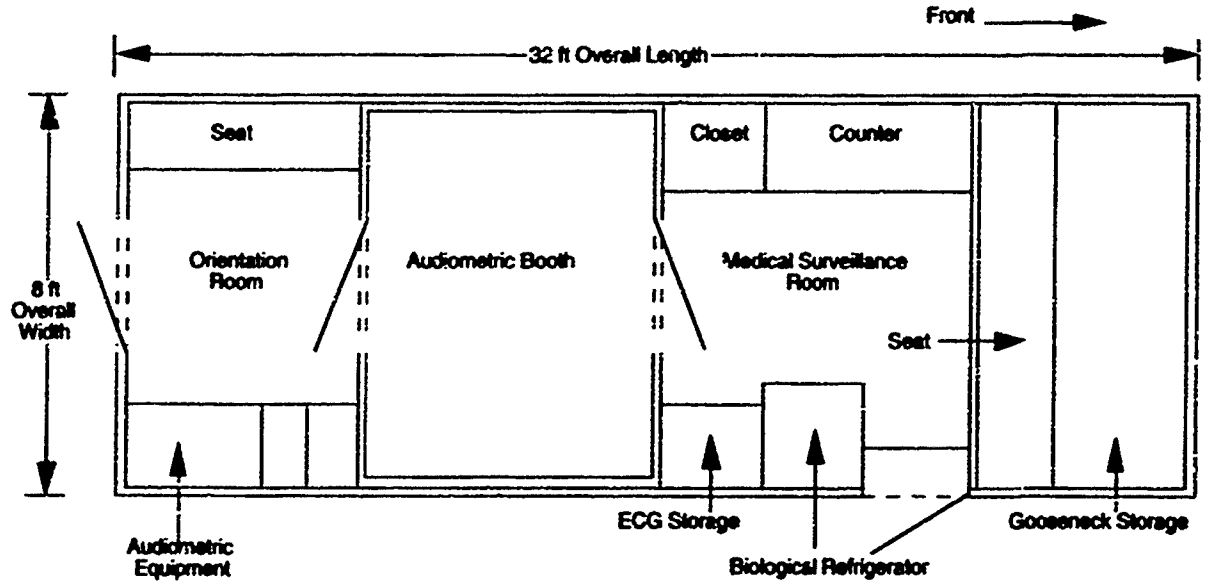

Fig. 7-27. An aerial view of the floor plan for the Military Occupational Health Vehicle (MOHV). When the MOHV is used exclusively for audiometric monitoring, six people can occupy the audiometric booth. Six others can be fitted with earplugs and briefed on hearing-conservation measures in the orientation room. Photograph. Courtesy of the US Army Environmental Hygiene Agency, Aberdeen Proving Ground, Md, 1991.

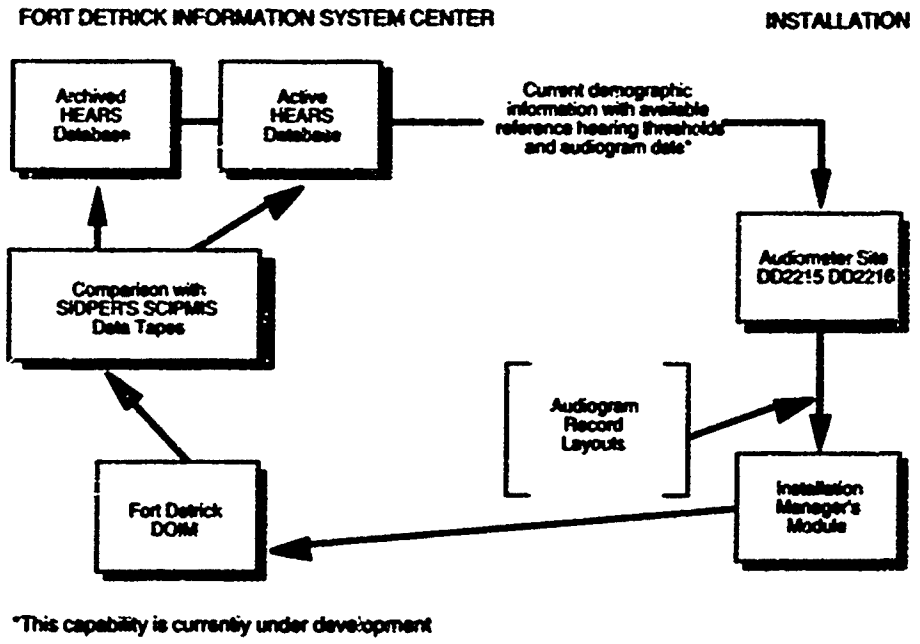

Fig. 7-28. HEARS data flow between Fort Detrick Information Center and the installation. Source: US Army Environmental Hygiene Agency. *Hearing Evaluation Automated Registry System (HEARS) Audiometer Operation Manual.* Aberdeen Proving Ground, Md: USAEHA; 1991. Technical Guide 167A. Available from the USAEHA, Bio-Acoustics Division, Aberdeen Proving Ground, MD 21010-5422.

Personnel Testing Requirements

All soldiers are required to receive reference and termination audiograms, and noise-exposed soldiers must also receive additional periodic testing (90 d after reference, annually, and any indicated interval for follow-up testing). Reference audiograms are used to

- monitor for hearing damage that is sustained during weapons qualification,
- serve as a baseline in the soldier's likely assignment to noise-hazardous duty at some point in his or her career, and
- provide a comparative population for soldiers who are routinely exposed to noise.

Since soldiers constitute a preselected population, comparisons to public health survey data or other databases in the private sector would not be epidemiologically valid.

Civilians who are exposed routinely to hazardous noise receive reference, periodic, and termination audiograms and are included in the HEARS database; however, all others must be excluded to avoid corrupting the database. Deaf civilians who work in hazardous noise environments must receive, at a minimum, reference and termination audiograms. Although the possibility that deaf personnel will incur additional hearing loss from noise is extremely remote, most have some residual hearing that should be documented for medical and legal purposes.

Audiogram forms designed specifically for DoD Hearing Conservation Programs are available for the clinical management of individuals in the program. The DD form 2215, *Reference Audiogram*, and the DD 2216, *Hearing Conservation Data*, were developed from U.S. Air Force forms in the late 1970s. Automation and recent changes in Hearing Conservation Program requirements have provided the impetus for forms revisions, which are imminent.

The HEARS audiometer prints completed audiograms for the health record and creates a record layout file of relevant fields for uploading into the manager's module and into the HEARS armywide database.

Variables in Audiometry

Accurate audiometric evaluation of military personnel has historically not been emphasized in policy directives. The army's documentation of widespread invalid test results in the past testifies to its lack of attention to, and limited appreciation of, the variables that must be controlled during a hearing evaluation.

Hearing is not tested directly with the conventional pure-tone threshold audiometry used for hearing conservation surveillance, but by interpreting a behavioral response to a pure-tone stimulus. Test results reflect the adequacy of the test environment, the instructions that are provided, the threshold technique that is used, the calibration of the audiometer, and the motivation of the examinee. The HEARS audiometer includes several features designed to control for, but not to eliminate, these variables.

Audiometric technicians are required to have successfully completed a minimum of 3 days of training specified by the Council for Accreditation in Occupational Hearing Conservation (CAOHC),[9] whether they are corpsmen, nurses, or health technicians. Although physicians and audiologists are excluded from such training requirements, they should be aware of the salient elements of an audiometric technician's training.

The HEARS audiometer is configured in one-, two-, four-, six-, and eight-station units. A talk-over mode permits supplemental instructions for individuals who experience difficulty during the test. Fault codes alert the technician if an examinee is not proceeding appropriately, and recommended instructions are available on the screen monitor.

The HEARS audiometer employs a threshold-determination technique, based on a psychophysical method of limits that is best understood as a bracketing procedure. For example, the threshold search begins at 0 dB HTL at 1000 Hz, and increases in 10-dB increments until the subject responds. Another tone presentation at that decibel level confirms the examinee's initial response and provides a reinforcing auditory image of the stimulus. The level then decreases 10 dB for every response and increases 5 dB for every nonresponse. Three responses at one dB level are accepted as threshold. This technique, called the modified Hughson-Westlake method, is the most accurate method in use and is recommended by the CAOHC.[9] In the automatic mode, this process is done for the operator. For difficult-to-test examinees, however, a manual mode is available, during which the examiner has control of the interstimulus interval, the order of the test frequencies, and the level of the initial tone presentation.

Currently, the HEARS audiometer cannot test hearing below 0 dB HTL, although some individuals may be able to hear below this level. Future considerations include a modification (ie, a firmware change) to a microprocessor chip in the audiometer, to add this capability.

The pulsed-tone mode is defaulted on the HEARS audiometer unless the continuous-tone option is selected. Pulsed tones can be followed more easily,

particularly for individuals with tinnitus: they can separate the pulsed tone from their perceived constant ringing sensation. Three tones, 200 msec each, with a 50% duty cycle (ie, 50% of the time the tone is on, and 50% of the time the tone is off) are presented. The examinee is allowed 1.8 seconds to respond from the onset of the stimulus, hence the need for a prompt response.

HEARS provides automatic retests to establish the validity of the data for a variety of conditions. The standard retest at 1000 Hz must be within 5 dB of the first threshold or thresholds obtained at other test frequencies will be suspect (eg, learning may have occurred or the examinee may have been inattentive). Other conditions that prompt an automatic retest include

- a 50-dB difference in threshold between adjacent test frequencies,
- a threshold greater than 30 dB at 500 Hz,
- any threshold greater than 90 dB HTL,
- test frequencies that time out (ie, take longer than 30 seconds for the examinee to determine the threshold), and
- any test frequency 1000 to 4000 Hz with a 20-dB or greater shift in either direction from the baseline.

HEARS uses automatic calculations to limit technician error and to save processing time. For example, hearing thresholds are determined from the appropriate number and sequencing of responses, stored in the computer's memory, and printed on demand. Similarly, the HEARS audiometer calculates military hearing profiles, as detailed in AR 40-501.[100] If a reference audiogram is performed before noise exposure and entered into HEARS, the profile system for an induction physical is activated. The percentage of hearing loss based on the DOL formula is calculated for civilian personnel and printed as a percentage of binaural impairment.

Periodic test results are compared to reference thresholds and significant threshold shift (STS) is calculated. Positive STS (ie, hearing loss) is confirmed using OSHA criteria, which state that shifts an average of 10 dB or greater at 2000, 3000, and 4000 Hz in either ear is an STS. The National Institute for Occ nal Safety and Health (NIOSH) age corrections ales and females are also incorporated for positive STS. Hearing loss attributable to the aging process is subtracted from the threshold shift. Age corrections are not applicable to a negative STS (hearing improvement).

Diagnosis and Patient Disposition

Army policy is to use positive TTS as a marker for individuals who are susceptible to hearing loss, for those who are not in compliance with the regulations regarding the use of hearing protection, or both. Since quiet periods are not required before a periodic test, some STS could be temporary. Follow-up testing confirms positive STS.

The first follow-up audiogram must be performed within 30 days of the identified STS, with a minimum of 15 hours before the test free of hazardous noise. If positive STS persists, the individual's supervisor is notified and a second follow-up examination is required, which must be preceded by at least 40 hours free of hazardous noise after the first follow-up audiogram.

After all required follow-up testing is performed, diagnostic testing helps the otolaryngologist determine the site of the lesion that is causing the hearing loss, but not necessarily to lower thresholds and defer the reporting of permanent STS. Obviously, clinical judgments must be made in cases of malingering or questionable audiometry on which the referral was based. Early detection serves the best interest of noise-exposed personnel, and in the long run, makes less work for medical personnel. Negative STS averaging 10 dB or greater at 2000, 3000, and 4000 Hz indicates, most probably, an invalid reference test. When there is a negative STS on the first follow-up, the audiometric results from that follow-up may be used to establish a new reference test. Audiological and otological referrals are optional in cases of negative STS, and referrals are only indicated if test results of the first follow-up test are questionable. Details on notification and reporting of STS, as well as procedures for reestablishing the reference audiogram are included in the HEARS Operations Manual.[97]

The practice of referring an individual for diagnosis before all follow-up testing is complete on the HEARS audiometer is not advisable. Audiologists and ear, nose, and throat (ENT) technicians may feel a responsibility to lower thresholds by performing follow-up testing on a diagnostic audiometer. However, such well-meaning intentions raise the following issues: (*a*) using the diagnostic audiometer defeats the purpose of monitoring audiometry and delays early detection of hearing loss, and (*b*) in a diagnostic setting, most testers can lower an individual's threshold at a particular test frequency by 5 dB. Although the hearing shift may no longer total enough for an STS, a 5-dB window is still within test-retest reliability. Moreover, the primary purpose of follow-up testing is to rule out TTS from noise exposure.

Physician involvement in STS follow-up testing procedures focuses primarily on the diagnosis and any recommendations regarding patient disposition. Only physicians can diagnose noise-induced hearing loss, and they should use all reasonable methods of differential diagnosis before establishing this diagnosis, including

- an investigation of the individual's auditory history and previous hearing tests;
- pure-tone, air-conduction measurements (which measure outer- and middle-ear conduction);
- pure-tone, bone-conduction measurements (which measure inner ear function, bypassing the outer and middle ears);
- speech-reception thresholds;
- speech-recognition measurements;
- oto-immittance testing (which measures the impedance of the middle ear and tympanic membrane); and
- masking, when indicated (which isolates and distracts the ear with normal hearing in order to examine the other ear).

The disposition and profiling process for hearing loss differs for military and civilian personnel. Military personnel should be issued a profile for hearing loss, if indicated. Profiling procedures are listed on DA Form 3349 (Block 1), *Medical Condition-Physical Profile Record*, and in AR 40-501.[100] Disposition of military personnel who have sustained hearing loss is defined within the profile system.[100] For the final disposition of DA civilian personnel, Standard Form 513, *Clinical Record Consultation Sheet*, is used. Guidance for civilians who have sustained progressive hearing loss is less well defined and involves case-by-case evaluations and close coordination with civilian personnel officers.

A civilian employee's removal from, or assignment to, a noise-hazardous job poses a dilemma for the occupational health physician, who must first consider the army's general philosophy for civilian employees: hearing loss is not in itself a contraindication to the assignment of these individuals to noise-hazardous work, provided the employees are protected against further hearing impairment. The army makes the job safe for the worker. However, the physician must consider whether the individual will be a hazard to him- or herself and others. The physician is also obliged to work within the guidelines that exist for protecting the handicapped from job discrimination and with a Civilian Personnel System that is obligated to find another job, which has equal opportunity for advancement, for an individual who is removed from a noise-hazardous occupation.

Unfortunately, an inability to meet the communication requirements of a job can only be inferred from clinical test results. Such a disability is not easily documented. Direct measurements of the relationship between hearing loss and job performance are virtually nonexistent.[101] A strong recommendation that a worker be removed from a job may be made if tests demonstrate that the individual cannot hear vital acoustic warning signals. But, obviously, modification of the warning system should be considered first.

During the diagnostic and referral processes, the issue of hearing loss compensation is sometimes raised. Although the physician is bound both ethically and legally to inform individuals of their hearing losses, only those offices and agencies charged with the administration of these programs are authorized to assess compensability. Regardless of the physician's intentions, estimating a patient's potential compensation could create credibility problems if the physician's predictions are not borne out by the actual proceedings.

Evaluation of Hearing Conservation Programs

The ability to generate numerous audiograms or to distribute thousands of hearing protectors does not accurately measure the effectiveness of the army's Hearing Conservation Programs. Only statistical trends of hearing loss can objectively measure whether hearing protectors are being fitted and worn properly and faithfully. The most useful way to evaluate a program is to focus on the Hearing Conservation Program results, not just on the testing procedures—on the goals of the program, not on the details of its operation.[102]

Statistical trends of hearing loss, participation in monitoring audiometry, and quality-assurance measures can help to identify and improve ineffective programs. Such measures can educate medical and command personnel and also be a source of satisfaction and reward for units or installations that promote effective programs. For over 10 years, HEARS has been providing comparative data to installations and major commands. Requirements for local program evaluation have recently been instituted.[78]

Major army installations and medical centers have one HEARS unit designated as a manager's module. The HEARS manager's module has additional computer storage capability. The software allows management to analyze data on participation in the Hearing Conservation Program, its effectiveness, and

quality-assurance information on demand. In addition to 71 standard reports, the manager's module also includes a nonprocedural language and a utility called TABLETALK that is used to produce ad hoc reports.

Program Compliance

The number of individuals who work in hazardous-noise environments (the denominator) can be compared to the number of individuals who are tested (the numerator) to measure compliance in monitoring audiometry. Twenty standard reports are available that can be used to identify the number and specific individuals tested. These reports include

- the number of tests administered,
- a list of names and social security numbers (SSN) by audiometric test date,
- record summaries by ZIP codes (counts of types of tests administered for military and civilian personnel),
- distribution of individuals by job code,
- serial hearing threshold data for individuals,
- retrieval of all DD 2215 or DD 2216 forms for an individual,
- a list of individuals who failed to take their annual tests,
- a list of individuals with STS on their periodic tests who require follow-up testing, and
- data regarding test counts by the audiometric technician's SSN and by the serial number of the audiometer to monitor work-load data by individual or test site.

Personnel turnover must be considered in assessing an installation's rate of participation. Also, the requirements for 90-day reestablished reference audiograms and termination audiograms should yield a 110% to 140% rate of participation if these additional tests are included in the numerator.

Quality Assurance

Checking for errors, automatic retests, automated calculations, and automated data-entries through HEARS have controlled quality-assurance measures significantly. Twenty-one standard reports are available on the HEARS manager's module to monitor potential problem areas. Although standards are still being developed for most of these measures, trends can be identified and armywide comparative data are available. The quality-assurance capability of HEARS allows it to generate reports concerning

- the same threshold at all frequencies that are tested,
- the absence of threshold entries at 0 dB,
- no reestablished reference audiogram,
- an elevated threshold at 500 Hz,
- a negative threshold shift,
- earplug sizes that vary by the type of protectors that are used,
- the need for different sizes of earplugs in each ear,
- types of hearing protectors in use, and
- multiple reference audiograms.

The quality-assurance reports are designed to check for a variety of potential problems:

- data fabrication,
- acoustic-calibration deviations,
- excessive background noise in the testing environment,
- invalid reference audiogram, and
- improperly fitted preformed earplugs.

Program Effectiveness

Unless otherwise specified, all of the 30 possible standard reports of program effectiveness can be run either separately for military and civilian personnel, or can be run according to ZIP code, job code, hearing protector, location (building number), or unit identification code (UIC). However, data from any report on program effectiveness will be of limited usefulness if there is poor participation (particularly among senior military personnel), poor quality control of the audiometric data, or a lack of follow-up testing to confirm whether an observed hearing threshold shift is permanent or temporary. Sources of program-effectiveness reports include (*a*) military profiles, (*b*) civilian hearing loss and potential compensation costs, (*c*) hearing threshold shifts, and (*d*) hearing threshold–level matrices. A hearing threshold matrix includes distributions and averages of hearing threshold levels.

When military profile reports are calculated, the most recent hearing test in the database is used. A discrepancy will exist between those profiles that are actually calculated and those that are assigned to individuals. Similarly, the most recent hearing test and pay grade are used to calculate hearing-loss percentages and potential compensation costs for civilians. The options to report the data in rank order either by cost or by the percentage of hearing loss are also included. These reports are limited to senior medical and command personnel only. Hearing threshold shift can also be calculated by the OSHA STS, or by

other measures of threshold shift that combine military and civilian personnel.

Health Education

The characteristics of noise-induced hearing loss make it a difficult subject to teach in health education. Noise-induced hearing loss is generally a slow, painless, and bloodless process. Hearing loss from noise is insidious and is not always recognizable to the individual until the magnitude of the loss has reached moderate-to-severe levels. The challenge to occupational health educators becomes apparent when placed in the context of the Accident Prevention Formula of the NSC: *See the hazard, Understand the defense, and Act in time.*

Nothing regarding noise-induced hearing loss is tangible. If an ear were to shed a drop of blood for every decibel of hearing that was lost, the task of identifying the hazard would be considerably easier. The usual progression of a noise-induced hearing loss from the high frequencies down into the low (or speech) frequencies can prevent an individual from quickly recognizing a problem and implementing timely action to prevent additional loss. But, as in the case of weapons fire, there may be no second chance. One afternoon on a firing range can wreak havoc on unprotected ears.

"Understanding the defense" against noise-induced hearing loss is not any more clear than "perceiving the hazard" to some of the individuals who are at risk for it. Most soldiers have been taught since childhood never to put anything into their ears; they now may simply be handed a pair of earplugs when noise becomes a hazard, without being instructed on their use. A complete and thorough military or civilian health-education program requires that (*a*) the students overcome behavioral obstacles or stereotypes, (*b*) the program's importance is emphasized by both command and management, (*c*) the importance of acute hearing for combat effectiveness or for the efficient performance of duties is stressed, and (*d*) the proper training aids and approaches are utilized.

Behavioral Obstacles

A lack of concern for hearing conservation can best be understood and addressed, both in the army and in the civilian-industrial sector, as a behavioral problem. Prevailing attitudes can frustrate the best-intended efforts to protect hearing. A preventable occupational injury that occurs on a large scale testifies not only to the pervasive nature of noise but also to the resistance against both hearing education and wearing hearing protection. Current challenges facing personnel who enforce hearing conservation include (*a*) auditory regression, (*b*) anatomical misinformation, (*c*) adaptation, (*d*) noise that is wrongly equated with power and efficiency, (*e*) denial of the hazard, (*f*) production of noise for social recognition, and (*g*) misplaced priorities.

Hearing has regressed in its importance to everyday life. Before artificial light was invented, during ages of nearly universal illiteracy, humans, like other mammals, relied heavily on their ears for information.[103] But with the advent of artificial light and the scientific revolution, learning became primarily visual. Today, the emphasis is on speed reading and visual scanning. How does this sensory shift affect our lives? Problems with sensory processing are generally tolerated until they become visual:[103] because aircraft noise does not assault the eyes, it may be tolerated until it interferes with television reception.

Among the medically unsophisticated, hearing deficits are associated with malfunction of the ear canal and the eardrum and the accompanying erroneous belief that noise-induced hearing loss is medically treatable. For example, some may believe that "noise only pokes little holes in your eardrums and old Doc can patch them up again," or that "noise can build up extra layers of skin on your eardrum and you can toughen your ears up to noise." Others have misinterpreted the limited benefits of cochlear implants, thinking that nerve cells can be restimulated back to life. Individuals may also believe that a hearing aid will be a perfect substitute for any hearing they have lost. Some in the medical community take the opposite and equally erroneous view that hearing aids cannot provide any benefit to those with high-frequency–noise-induced hearing loss. In addition, because the layman's knowledge of the ear does not usually extend beyond the outer ear, health-education activities should refer to *hearing* protectors, rather than *ear* protectors, when protective measures and equipment are discussed.[74]

Noise may damage physical health, but psychological adjustments are made to adapt to the noise. Adaptation is a two-edged sword; it can be a saving grace, but at the same time it may create a false sense of well-being.[103] The obvious danger of adaptation to noise is that our ability to recognize warnings of hazardous noise is lost, and we will no longer react to the hazard.[103] Similarly, TTS may give the individual a false sense of security when previous hearing acuity appears to have recovered after loud noise exposures.

The noise a machine makes may erroneously be equated with its power and operational efficiency.[103] Without a noise accompanying a function, consumers often believe that power and efficiency have been lost. For example, some consumers may believe that a whisper-quiet vacuum cleaner is not as powerful as an identical but noisier older model, and office workers

have complained that after their typewriters' clacking sounds were removed, they were noticeably slower than their noisier, but otherwise identical, typewriters.[103]

A reluctance to wear personal protective devices may be a mechanism for coping with the day-to-day hazards of an occupation: the worker is able to deny that the hazard exists if the protective equipment is not used. A form of denial may also be observed in those soldiers who exhibit "macho" behavior. Although denial may be more prevalent with life-threatening hazards, reactions like these to loud weapons fire are not uncommon. Denial among young soldiers may be transformed into feelings of indestructibility, which is a common trait of youth. In basic training and advanced individual training, intimidation is often used to break through attitudes to ensure hearing protection compliance. However, in duty assignments that have less supervision, compliance with proper and faithful use of hearing protection may become more lax.

Senior personnel offer greater challenges to health educators than do younger soldiers. Higher-ranking soldiers who have lost hearing may choose to ignore their deficits, hoping that others will as well. Their attitude seems to be that since they have already lost their hearing, there is no reason to wear hearing protectors. Obviously, they should be convinced both to protect the hearing that they still have and to set an example for their subordinates.

Making noise can be an attention-seeking behavior that results in at least temporary recognition by peers. For example, among youths whose unmuffled cars or motorcycles signal their arrival at and departure from a scene,

> such cacophony gives them a feeling of being part of the "in" or "hip" crowd.... It can also be interpreted as a protest against the establishments' highly organized, dull, quiet world.[103(p9)]

Young people with limited communication skills may prefer simple words and gestures through the raucous din. Noise also offers an opportunity to invade another person's space and get much closer while talking than might otherwise be acceptable.

Other priority issues in the world—drug abuse, crime, budget deficits, homelessness, hunger, and terrorism—overshadow noise-induced hearing problems. Understandably, a commander's priorities are similarly ordered. Encouraging and enforcing compliance with hearing conservation principles is difficult in a militarily unique or industrial environment that is fraught with life-threatening hazards and demanding training and production schedules. Commanders may use "realistic training" as their excuse for not enforcing the use of hearing protection. In addition, the requirement to report for an annual hearing test may be thought of as a detractor from training.

Command and Management Emphasis

Because most noise-induced hearing loss occurs during routine training exercises, it should be almost completely preventable. A concerned commander can have a dramatic effect on a Hearing Conservation Program. Health education must be provided for command personnel and for all levels of supervisory personnel in order to emphasize their responsibilities in the Hearing Conservation Program. Without their endorsement and support, the program will not succeed.

The value of the program can be emphasized in several contexts, but most importantly, the application and implementation of a Hearing Conservation Program is the *law*. In addition to existing OSHA, DoD, and DA regulations, the Federal Employee Reform and Tort Compensation Act holds federal supervisors liable if they are found negligent and not operating within the scope of their authority to provide protective equipment. Even supervisors who provide the required hearing protectors may be under the erroneous assumption that the soldier or employee can choose whether or not to use the protectors. Responsibilities to enforce the use of PPE and to ensure that subordinates report for scheduled hearing tests can be included in officer and enlisted evaluation reports and in civilian supervisors' performance standards.[104]

Command and supervisory personnel may recognize the value of a program for hearing conservation when it is explained in terms of reducing compensation expenditures or saving lost man-hours that are caused by accidents. One study found that the risks attributed to noise and hearing loss together accounted for 43% of injuries sustained in one shipyard.[105] This study identified factors that could interfere with the faculties that are needed for recognizing warning signals and imminent danger. The use of hearing protection was not identified as a factor.

Combat Effectiveness

Senior commanders usually recognize that a Hearing Conservation Program is valuable to soldiers and civilian workers. Focusing on hearing as our most precious social and learning sense may be of limited value to many command and supervisory personnel unless the necessity to preserve hearing is incorporated into the success of their mission. For example, the flight surgeon responsible for medical planning on the Son Tay prison camp raid in Vietnam insisted that all troops wear earplugs while being airlifted. As a result, when the troops arrived at the prison camp and

removed their earplugs, they found that their hearing was unimpaired from the noise of the helicopters.[90]

On today's high-technology battlefield, good hearing is a combat multiplier and an essential attribute of the effective soldier in both offensive and defensive operations.[78,106] Hearing is necessary in offensive operations to (*a*) locate snipers, (*b*) locate patrol members, (*c*) identify vehicles, and (*d*) determine types of booby traps. One Vietnam veteran reported that enemy snipers could be located by the reports of their weapons, even when muzzle flashes were not observed. In addition, patrol members often guide more by sound than by vision, especially when they are on night patrol under a new moon. Soldiers have also been able to hear the difference between hostile and friendly fire. The ability to determine the number and location of enemy vehicles may be crucial to the successful completion of a mission.

One Vietnam veteran could distinguish between the sounds generated by two types of trip wires. The sound generated by a trip wire that pulls the pin from a grenade is different from the sound made by a pressure-activated explosive. Quick movement away from the grenade is required, but the soldier must maintain pressure on the explosive until it is deactivated.[106]

In defensive positions, the soldier needs to hear both perimeter alarms that are activated by sensing devices that have been triggered by movement, and enemy movement through leaves, grass, and twigs. Experts have recognized the high-frequency nature of these sounds and the necessity for relatively normal hearing to detect them. Soldiers can determine the enemy's location by listening for sounds from wildlife, loading cartridges, safety locks, and the clipping of barbed wire. In Vietnam, soldiers could determine the proximity of the enemy by the cessation of bird calls in the upper canopy of the jungle. The presence of birds in the lower canopy meant that human refuse was nearby.

Soldiers must also be able to hear radio messages and verbal orders. Most military radios clip both the high- and low-frequency sounds. A soldier with a hearing loss will confuse similar-sounding verbal orders, such as the digits in a grid coordinate. Good hearing also aids in small-arms accuracy and weapon identification. Soldiers on pistol and rifle teams have been aware of the advantage of wearing hearing protection while firing their small arms. Wearing hearing protection increases their accuracy by reducing the tendency to flinch at the impact of the weapon and normal hearing can discriminate between M16 and Soviet-made AK47 rifle fire.

Performance Measures

Until recently, the relationship between the ability to communicate and the successful accomplishment of missions could only be suggested from anecdotes or inferred from vague clinical test results. A landmark study from the U.S. Army Human Engineering Laboratory has provided the first hard data of the effects of communication on performance.[101] Thirty experienced tank crews conducted gunnery exercises in the Conduct of Fire Trainer (COFT) tank simulator at Fort Knox under five communication conditions ranging from very good to extremely poor. Performance measures and results indicated that

- the mean time to identify a single target increased as communication conditions were degraded.
- the overall time to complete a firing mission varied from 40 seconds under good communication conditions to 90 seconds under the poorest conditions.
- target identification varied from a hit rate of 98% under the good conditions to 68% under the poorest conditions. The percentage of enemy targets killed also decreased as communication was degraded (Figure 7-29).
- commands communicated incorrectly varied from 1% to 37% over the range of communication conditions. As a result, the percentage of time the crew was killed by the enemy ranged from 7% under good communication conditions to 28% under the poorest conditions. Figure 7-30 shows the percentage of fire that hit the wrong target.[101]

Training Aids and Approaches

Effective health education will result in or reinforce the faithful and proper use of hearing protectors. Compliance with hearing conservation measures is individualized highly for both the military and the civilian sector.

Personal testimonials from peers or respected senior personnel on the dehabilitating effects of their hearing loss or the importance of hearing in combat may be effective as teaching tools.[107-111] For example, the film *Sounds of Combat* was introduced by a sergeant major and Medal of Honor winner.[107] Individuals like these have credibility with soldiers when they attempt to link good hearing to the success of a combat mission.

Individual counseling can be most effective, particularly when it is personalized. Demonstrating to a 30-year-old sergeant that he has the hearing of an 80-year-old man can be convincing. Training aids, unique to the military and designed for this purpose, are available through publication and audiovisual support centers.

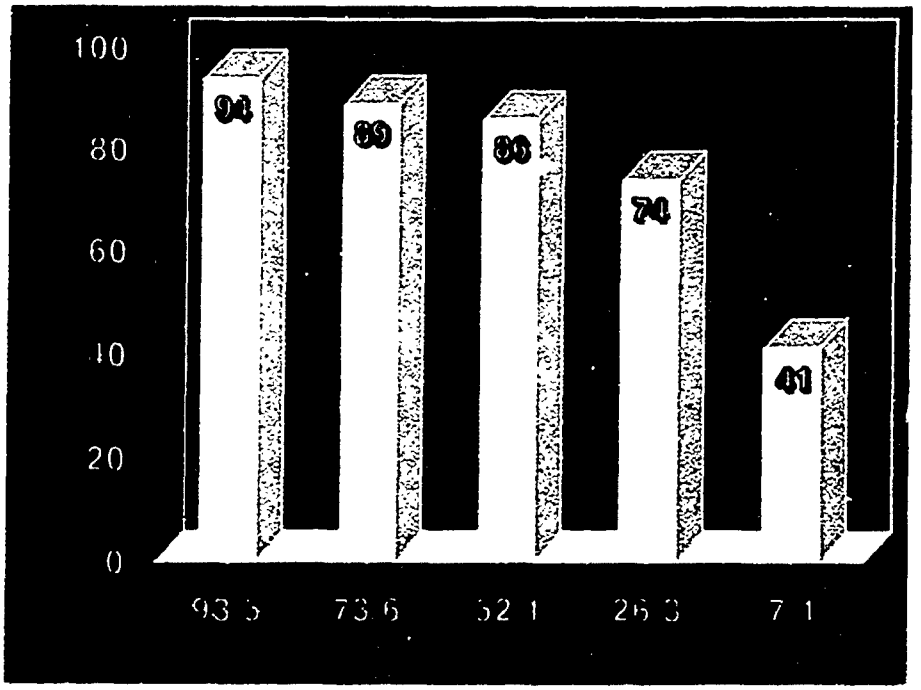

Fig. 7-29. Performance measures as a function of five conditions of speech intelligibility. For 30 experienced tank crews in a tank simulator, as their speech intelligibility was degraded their killed targets also decreased. Source: Garinther GR, Peters LJ. Impact of communications on armor crew performance. *Army Res, Development, & Acquisition Bull.* 1990;January-February:1–5.

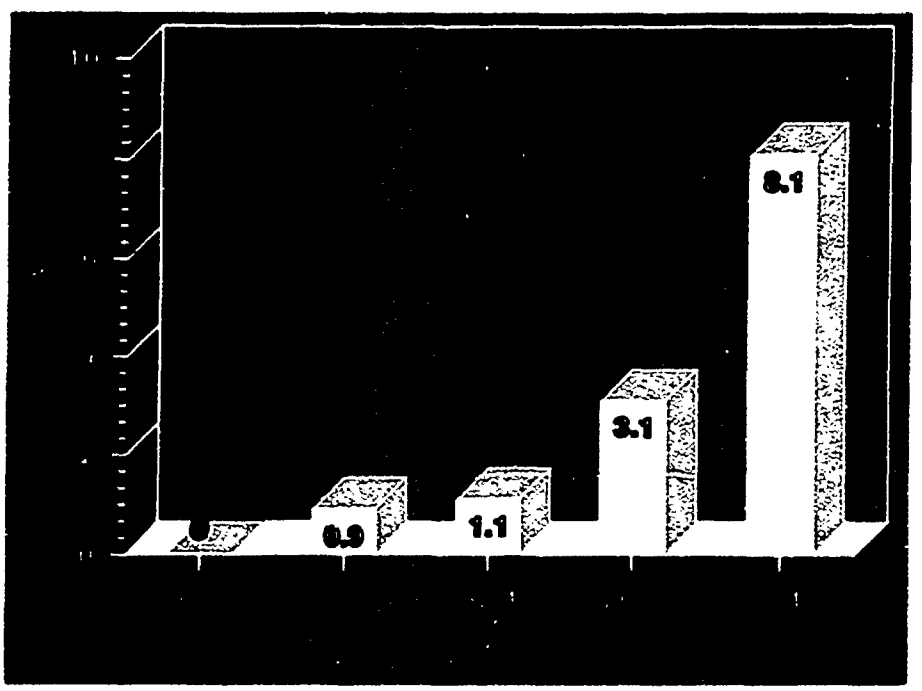

Fig. 7-30. Percentage of episodes in which the wrong target was shot as a function of the five conditions of speech intelligibility. Source: Garinther GR, Peters LJ. Impact of communications on armor crew performance. *Army Res. Development, & Acquisition Bull.* 1990;January-February:1–5.

SUMMARY

Noise-induced hearing losses can occur painlessly, are preventable, and will become permanent if effective hearing conservation programs are not enforced. The effects of noise on the complex auditory mechanism depend on the physical characteristics of the noise stimulus, the duration of exposure, the audiometric frequency, and the type of noise.

Factors that account for varying degrees of susceptibility to the hazardous effects of noise are under constant investigation. The dynamic processes and intricacies of the auditory mechanism make it impossible to formulate the exact relationship between the noise exposure, the receiver, and the amount of hearing loss that is sustained. Awareness of changes in the auditory mechanism following exposure to hazardous noise should assist the physician in evaluating cochlear function.

The army attempts to prevent hearing loss through the coordinated application of several program elements. No single program element can function effectively without all the other elements, which include

- noise-hazard evaluations,
- posting of noise-hazardous areas and equipment,
- engineering controls,
- use of PPE,
- audiometric monitoring,
- health education, and
- Hearing Conservation Program evaluation.

Effective hearing conservation programs are characterized by well-defined responsibilities among participants, and the presence of a single individual who functions as both a catalyst and a linchpin to ensure the implementation and the coordination of all program elements. The program is best implemented as a facet of combat-readiness medicine, with hearing preservation incorporated into the overall success of the mission.

REFERENCES

1. Saunders JC, Dear SP, Schneider ME. The anatomical consequences of acoustic injury: A review and tutorial. *J Acoust Soc Am.* 1985;80(2):569–584.

2. Marshall SLA. *Men Against Fire.* New York: William Morrow; 1947.

3. Walden BE, Prosek RA, Wortington DW. *The Prevalence of Hearing Loss Within Selected US Army Branches.* Washington, DC: US Army Medical Research and Development Command; 1975. Interagency IAO 4745.

4. Durrant JD, Lovrinic JH. *Bases of Hearing Science.* 2nd ed. Baltimore: Williams & Wilkins; 1984.

5. Pickles JO. *An Introduction to the Physiology of Hearing.* New York: Academic Press; 1982.

6. Patuzzi R. Mechanical correlates of noise trauma in the mammalian cochlea. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 122–136.

7. Durrant JD. Anatomical and physiologic correlates of the effects of noise on hearing. In: Lipscomb DM, ed. *Noise and Audiology.* Baltimore: University Park Press; 1978: 109–141.

8. Weiner FM, Ross DA. The pressure distribution in the auditory canal in a progressive sound field. *J Acoust Soc Am.* 1946;18:401–408.

9. Gerhardt KJ, Melnick W, Ferraro JA. Reflex threshold shift in chinchillas following a prolonged exposure to noise. *J Speech Hear Res.* 1979;22(1):63–72.

10. Gerhardt KJ, Hepler EL Jr. Acoustic-reflex activity and behavioral thresholds following exposure to noise. *J Acoust Soc Am.* 1983;74(1):109–114.

11. Nilsson R. The acoustic reflex in industrial impact noise. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 237–246.

12. Colletti V, Sittoni V. Noise history, audiometric profile, and acoustic reflex. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss*. New York: Plenum Press; 1986: 247–270.

13. Stevin GO. Stimulation of the middle ear acoustic reflex applied to damage-risk for hearing loss produced by burst fire. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss*. New York: Plenum Press; 1986: 271–280.

14. Hamernik R, Turrentine G, Roberto M. Mechanically-induced morphological changes in the organ of Corti. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss*. New York: Plenum Press; 1986: 69–84.

15. Bohne BA. Mechanisms of noise damage in the inner ear. In: Henderson D, Hamernik RP, Dosanjh DS, Mills JH. *Effects of Noise on Hearing*. New York: Raven Press; 1978: 41–68.

16. Spoendlin H. Anatomical changes following various noise exposures. In: Henderson D, Hamernik RP, Dosanjh DS, Mills JH. *Effects of Noise on Hearing*. New York: Raven Press; 1978: 69–90.

17. Bohne BA, Rabbitt KD. Holes in the reticular lamina after noise exposure: Implication for continuing damage in the organ of Corti. *Hear Res*. 1983;11:41–53.

18. Bohne BA. Patterns of cellular degeneration in the inner ear following excessive exposure to noise. Presented at the National Institutes of Health Consensus Development Conference on Noise and Hearing Loss; 22–24 January 1990; Washington, DC.

19. Henderson D, Hamernik RP. Impulse noise: Critical review. *J Acoust Soc Am*. 1986;80(2):569–584.

20. Stockwell CW, Ades HW, Engstrom H. Patterns of hair cell damage after intense auditory stimulation. *Ann Otol Rhinol Laryngol*. 1969;78:1144–1168.

21. Spoendlin H. Primary structural changes in the organ of Corti after acoustic overstimulation. *Acta Otolaryngol*. 1971;71:166–176.

22. Dallos P. Cochlear neurobiology: Revolutionary developments. *ASHA*. 1988;3C:50–56.

23. Lawrence M, Gonzalez G, Hawkins JE Jr. Some physiological factors in noise-induced hearing loss. *Am Ind Hyg Assoc*. September-October 1967:425–430.

24. Hawkins JE Jr. The role of vasoconstriction in noise-induced hearing loss. *Ann Otol Rhinol Laryngol*. 1971;80:903–913.

25. Hawkins JE Jr, Johnsson LG, Preston RE. Cochlear microvasculature in normal and damaged ears. *Laryngoscope*. 1972;7:1091–1104.

26. Lipscomb DM, Axelsso A, Vertes D, Roettger R, Carroll J. The effect of high level sound on hearing sensitivity, cochlear sensorineuroepithelium, and vasculature of the chinchilla. *Acta Otolaryngol*. 1977;84:44–56.

27. Axelsson A, Vertes D. Histological findings in cochlear vessels after noise. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss*. New York: Raven Press; 1982: 49–68.

28. Joglekar SS, Lipscomb DM, Shambaugh GE Jr. Effects of oxygen inhalation on noise-induced threshold shifts in humans and chinchillas. *Arch Otolaryngol*. 1977;102:574–578.

29. Witter HL, Deka RC, Lipscomb DM, Shambaugh GE Jr. Effects of prestimulatory carbogen inhalation on noise-induced temporary threshold shifts in humans and chinchilla. *Am J Otol*. 1980;1(4):227–232.

30. Dengerink HA, Axelsson A, Miller JM, Wright JW. The effect of noise and carbogen on cochlear vasculature. *Acta Otolaryngol*. 1984;98:81–88.

31. Melnick W. Medicinal therapy for hearing loss resulting from noise exposure. *Am J Otolaryngol.* 1984;5(6):426–431.

32. Saunders JC, Canlon B, Flock A. Mechanical changes in stereocilia following overstimulation: Observations and possible mechanisms. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 11–30.

33. Flock A, Cheung HC. Actin filaments in sensory hairs of inner ear receptor cells. *J Cell Biol.* 1977;75:339–343.

34. Engstrom B, Borg E, Canlon B. Morphology of stereocilia on cochlear cells after noise exposure. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 1–10.

35. Pickles JO, Comis SD, Osborne MP. The morphology of stereocilia and their cross-links in relation to noise damage in the guinea pig. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 31–42.

36. Liberman MC. Biological bases of acoustic injury. Presented at the National Institutes of Health Consensus Development Conference on Noise and Hearing Loss; 22–24 January 1990; Washington, DC.

37. Hodge DC, Price GR. Hearing damage risk criteria. In: Lipscomb DM, ed. *Noise and Audiology.* Baltimore: University Park Press; 1978: 167–191.

38. Liberman MC, Dodds LW, Learson DA. Structure-function correlation in noise-damaged ears: A light and electron-microscopic study. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 163–178.

39. Lipscomb DM. What is the audiogram really telling us? Part 1: Audiometrics vs cochlear damage. In: *Maico Audiological Library Series,* 13. Minneapolis, Minn: Maico Hearing Instruments; 1975:24–27.

40. Lipscomb, DM. Sensorineural hearing losses. In: *Maico Audiological Library Series,* 13. Minneapolis, Minn: Maico Hearing Instruments; 1975:28–31. Report 6.

41. Boettcher FA, Henderson D, Gratton MA, Danielson RW, Byrne CD. Synergistic interactions of noise and other ototraumatic agents. *Ear Hear.* 1987;8(4):192–212.

42. Taylor W, Pearson J, Mair A, Burns W. Study of noise and hearing in jute weaving. *J Acoust Soc Am.* 1965;38:113–120.

43. Burns W, Robinson DW. *Hearing and Noise in Industry.* London: Her Majesty's Stationery Office; 1970.

44. Hepler EL, Moul MJ, Gerhardt KJ. Susceptibility to noise-induced hearing loss: Review and future directions. *Milit Med.* 1984;149:154–158.

45. Humes LE. Noise-induced hearing loss as influenced by other agents and by some physical characteristics of the individual. *J Acoust Soc Am.* 1984;76(5):1318–1329.

46. Clark WW, Bohne BA, Boettcher FA. Effect of periodic rest on hearing loss and cochlear damage following exposure to noise. *J Acoust Soc Am.* 1987;82(4):1253–1273.

47. Henderson D. Acoustic parameters of hazardous noise exposure. Presented at the National Institutes of Health Consensus Development Conference on Noise and Hearing Loss; 22–24 January 1990; Washington, DC.

48. Bohne BA, Clark WW. Growth of hearing loss and cochlear lesion with increasing duration of noise exposure. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss.* New York: Raven Press; 1982: 283–302.

49. Bohne BA, Yohman L, Genner MM. Cochlear damage following interrupted exposure to high-frequency noise. *Hear Res.* 1987;29:251–264.

50. Bohne BA, Zahn SJ, Bozzay DG. Damage to the cochlea following interrupted exposure to low-frequency noise. *Ann Otol Rhinol Laryngol.* 1985;94:122–128.

51. Salvi R. Interaction between noise and other agents. Presented at the National Institutes of Health Consensus Development Conference on Noise and Hearing Loss; 22–24 January 1990; Washington, DC.

52. Yanz JL, Abbas PJ. Age effects in susceptibility to noise-induced hearing loss. *J Acoust Soc Am.* 1982;72(5):1450–1455.

53 Gunther T, Ising H, Joachims Z. Biochemical mechanisms affecting susceptibility to noise-induced hearing loss. *Am J Otol.* 1989;10:36–41.

54. Joachims Z, Babisch W, Ising H, Gunther T, Handreck M. Dependence of noise-induced hearing loss upon perilymph magnesium concentration. *J Acoust Soc Am.* 1983;74(1):104–108.

55. Hamernik RP, Ahroon WA, Davis RI, Axelsson A. Noise and vibration interactions: Effects on hearing. *J Acoust Soc Am.* 1989;86(6):2129–2137.

56. Dengerink JE, Dengerink HA, Swanson S, Thompson P, Chermak GD. Gender and oral contraceptive effects on temporary auditory effects of noise. *Audiology.* 1984;23:411–425.

57. Swanson SJ, Dengerink HA. Changes in pure-tone thresholds and temporary threshold shifts as a function of menstrual cycle and oral contraceptives. *J Speech Hear Res.* 1988;31:569–574.

58. Dengerink HA, Trueblood GW, Dengerink JE. The effects of smoking and environmental temperature on temporary threshold shifts. *Audiology.* 1984;23:401–410.

59. Dengerink HA, Wright JW, Dengerink JE, Miller JM. A pathway for the interaction of stress and noise influences on hearing. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 559–569.

60. Oftedal G. Noise-induced hearing damage caused by metabolic exhaustion: A mathematical model. *J Acoust Soc Am.* 1988;83(4):1499–1507.

61. Donahue AM. *Hearing Conservation Data Profile.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1991. Army-wide database 51-34-0251-91.

62. Gasaway DC. Occupational hearing conservation in the military. In: Lipscomb DM, ed. *Hearing Conservation in Industry, Schools and the Military.* Boston: Little, Brown; 1988: 243–262.

63 Walpole RH. *Investigation of Ear Plugs for Protection Against Gun Blast.* Fort Knox, Tenn: Armored Medical Research Laboratory; 1944. Project 26.

64. Northern JL. Military opportunities in speech pathology and audiology. *ASHA.* 1968;10:325–330.

65. Office of the Adjutant General. *Establishment and Utilization of Audiologist (MOS 3360) Positions in Hearing Conservation Programs.* Washington DC: DA; 1972. Extracted from letter.

66. US Department of the Air Force. *Hazardous Noise Exposure.* Washington, DC: DAF; 1956. Regulation 160-3, Medical Service.

67. Suter AH. The development of federal noise standards and damage risk criteria. In: Lipscomb DM, ed. *Hearing Conservation in Industry, Schools and the Military.* Boston: Little, Brown; 1988: 45–66.

68. US Department of the Army. *Noise and Conservation of Hearing.* Washington, DC: DA; 1956. Technical Bulletin MED 251.

69. US Department of the Army. *Noise and Conservation of Hearing.* Washington, DC: DA; 1965. Technical Bulletin MED 251.

70. US Department of the Army. *Noise and Conservation of Hearing.* Washington, DC: DA; 1972. Technical Bulletin MED 251.

71. US Department of the Army. *Preventive Medicine.* Washington, DC: DA; 1969. Army Regulation 40-5.

72. Comptroller General of the United States. *Hearing Protection: Problems in the Department of Defense.* Washington, DC: US General Accounting Office; 1977. Report to the Congress.

73. US Department of Defense. *Hearing Conservation.* Washington, DC: DoD; 1978. DoD Instruction 6055.3.

74. US Department of the Army. *Hearing Conservation.* Washington, DC: DA; 1980. Technical Bulletin MED 501.

75. The Occupational Safety and Health Act of 1970. Public Law 91-596.

76. US Occupational Safety and Health Administration. Occupational Noise Exposure: Hearing Conservation Amendment; Final Rule. *Federal Register,* March 8, 1983;48:9738–9785.

77. US Department of Defense. *Hearing Conservation.* Washington, DC: DoD; 1987. DoD Instruction 6055.12.

78. US Department of the Army. *Hearing Conservation.* Washington, DC: DA; 1991. DA Pamphlet 40-501.

79. American National Standards Institute. *Specifications for Sound Level Meters.* New York: American Institute of Physics. ANSI Standard S1.4-1983.

80. American Conference of Governmental Industrial Hygienists. *Thresholds Limit Values and Biological Exposure Indices for 1989–1990.* Cincinnati, Oh: ACGIH; 1990.

81. Coles RRA, Garinther GR, Hodge DC, Rice CG. Hazardous exposure to impulse noise. *J Acoust Soc Am.* 1968;43:336–343.

82. Ward WD, ed. *Proposed Damage-Risk Criterion for Impulse Noise (Gunfire)* (U). Washington, DC: Committee on Hearing, Bioacoustics and Biomechanics; NAS/NRC; 1968. Report of Working Group 57, National Academy of Science/National Research Council.

83. Price GR. Impulse noise hazard as a function of level and spectral distribution. In: Salvi RJ, Henderson D, Hamernik RP, Colletti V, eds. *Basic and Applied Aspects of Noise-Induced Hearing Loss.* New York: Plenum Press; 1986: 379–392.

84. US Department of the Army. *Safety Color Code Marking and Signs.* Washington, DC: DA; 1983. Army Regulation 385-30.

85. US Department of Defense. *Noise Limits for Army Materiel.* Washington, DC: DoD; 1979. Military Standard 1474B.

86. Norris TR. *Tracked Vehicles: Noise and Vibration Study Using a Reduced Scale Model.* Warren, Mich: US Army Tank Automotive Command; 1975. Technical Report 12099.

87. Hansen CH, Mathews M. *Noise Reduction Through Optimum Hull Design in Lightweight Tracked Vehicles.* 1983. Cambridge, Mass: Bolt, Beranek and Newman. Report 5074.

88. Garinther GR, Schmiedeberg JA, Turner KG. *Development of Technology for Reducing Noise of Light Armored Tracked Vehicles.* Aberdeen Proving Ground, Md: US Army Human Engineering Laboratory; 1989. Technical Memorandum 20-89.

89. Royster JD, Royster LH. Evaluating hearing conservation programs: Organization and effectiveness. In: *Proceedings of 1989 Industrial Hearing Conservation Conference.* Lexington, Ky: Office of Continuing Education and Extension College of Engineering, University of Kentucky and the National Institute for Occupational Safety and Health. US Government Printing Office; 1989.

90. Ohlin DW. *Personal Hearing Protective Devices, Fitting Care, and Use.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1975. USAEHA Technical Guide 041.

91. Ohlin DW, Michael PL Bienvenue GR, Rosenberg DM. An earplug insertion and seating device. *Sound Vib.* 1981;15(1):22–25.

92. Edwards RG, Hauser WP, Moiseev NA, Broderson AB, Green WW. Effectiveness of earplugs as worn in the workplace. *Sound Vib.* 1978;12(1):12–22.

93. Regan DE. Real ear attenuation of personal ear protective devices worn in industry. *Audiol Hear Education.* 1977;3:16–17.

94. Padilla M. Earplug performance in industrial field conditions. *Sound Vib.* 1976;10(5):33–36.

95. Royster H. Effectiveness of three different types of ear protectors in preventing TTS. *J Acoust Soc Am.* 1979;66(suppl 1):DD16.

96. Berger H. Using the NRR to estimate the real world performance of hearing protectors. *Sound & Vibration.* 1983;17(1):12–18.

97. US Army Environmental Hygiene Agency. *Hearing Evaluation Automated Registry System (HEARS) Audiometer Operation Manual.* Aberdeen Proving Ground, Md: USAEHA; 1991. Technical Guide 167A. Available from the USAEHA, Bio-Acoustics Division, Aberdeen Proving Ground, MD 21010-5422.

98. US Army Environmental Hygiene Agency. *Hearing Evaluation Automated Registry System (HEARS) Manager's Module Operation Manual.* Aberdeen Proving Ground, Md: USAEHA; 1991. Technical Guide 167B.

99. Council for Accreditation in Occupational Hearing Conservation. *Manual.* Cherry Hill, NJ: Fischler's Printing; 1978.

100. US Department of the Army. *Standards of Medical Fitness.* Washington, DC: DA; 1989. Army Regulation 40-501.

101. Garinther GR, Peters LJ. Impact of communications on armor crew performance. *Army Res, Development, & Acquisition Bull.* 1990;January-February:1–5.

102. Suter AH. The need for and benefits of audiometric data base analysis. *Sound Vib.* 1989;23:14–18.

103. Bragdon CR. *Noise Pollution: The Unquiet Crisis.* Philadelphia: University of Pennsylvania Press; 1970.

104. US Department of the Army. *Safety.* Washington, DC: DA; 1988. Army Regulation 385-10.

105. Moll von Charante AW, Mulder PGH. Perceptual acuity and the risk of industrial accidents. *Am J Epidemiol.* 1990;131(4):652–663.

106. Pierson LL. Hearing conservation for combat readiness. Presented at the Army Medical Department Audiology Seminar, Baltimore, Md, 7 May 1986.

107. *Sounds of Combat* (Film). Available from local Army Audiovisual Support Centers.

108. *Hearing Protection* (Film: TVT 20-878). Available from local Army Audiovisual Support Centers.

109. *Prevention of Hearing Loss* (Film: TF8-4602). Available from local Army Audiovisual Support Centers.

110. *The Sound of Sound* (Film: MF8-5810). Available from local Army Audiovisual Support Centers.

111. *Stick It In Your Ear* (Film: MF8-13077). Available from local Army Audiovisual Support Centers.

RECOMMENDED READING

Henderson D, Hamernik RP. Impulse noise: Critical review. *J Acoust Soc Am.* 1986;80(2):569–584.

Saunders JC, Dear SP, Schneider ME. The anatomical consequences of acoustic injury: A review and tutorial. *J Acoust Soc Am.* 1985;78(3):833–860.

Schmiedt RA. Acoustic injury and the physiology of hearing. *J Acoust Soc Am.* 1984;76(5):1293–1317.

Chapter 8

CONSERVING VISION

ALAN K. THOMPSON, O.D.*

INTRODUCTION

LEGISLATION AND GOVERNMENTAL AGENCIES
- Occupational Safety and Health Act
- Occupational Safety and Health Administration
- National Institute for Occupational Safety and Health
- Food and Drug Administration
- American National Standards Institute
- U.S. Army Environmental Hygiene Agency
- U.S. Army Regulations and Publications

THE VISION CONSERVATION PROGRAM

OCCUPATIONAL VISION
- Screenings
- Standards
- Examinations

EYE SAFETY
- Incidence of Eye Injuries
- Ballistic and Mechanical Hazards
- Chemical Hazards
- Radiant Energy Hazards

SELECTING EYE PROTECTION
- Ballistic, Mechanical, and Impact Protection
- Chemical Protection
- Radiant Energy Protection
- Militarily Unique Eye Protection
- Contact Lenses in Industry

ENVIRONMENTAL VISION
- Illumination
- Personal Computers and Video Display Terminals
- Solar Ultraviolet Radiation

SUMMARY

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INTRODUCTION

Ocular trauma is the sixth leading cause of blindness in the world following trachoma, xerophthalmia, onchocerciasis, cataract formation, and glaucoma.[1] Approximately 2.4 million individuals in the United States sustain ocular injuries annually; more than one-half of these injuries occur to individuals under 25 years of age.[2] Approximately 40,000 of them will suffer some degree of visual impairment and an estimated 1,500 will lose their sight permanently. Current estimates show that more than 900,000 individuals in the United States have permanent visual impairment caused by injury; 75% have monocular blindness.[3]

Ocular injuries are not without significant financial costs. Nationwide, ocular injuries account for an estimated direct cost of approximately $300 million in medical bills, compensation, and lost production time. For example, in 1980 the state of Ohio reported 6,457 work-related accidents that involved eye injuries, with direct medical and worker-compensation costs of nearly $20 million—approximately $3,067 per injury. These figures do not include either the associated costs of pain and anguish that eye injuries and blindness cause or the indirect costs (legal fees and judgments, time lost from work, costs associated with training replacement workers, or costs of repairing damaged equipment). Experts estimate that the indirect costs can be 5- to 10-fold higher than the direct costs.[4]

Statistics on industrial ocular injuries and principles used in industry to protect the eyes and vision have direct application to active-duty soldiers—in garrison or on the battlefield. While stationed in garrison, it is not unreasonable to assume that soldiers will suffer types and incidence of ocular injuries similar to their civilian counterparts. However, the battlefield, like the industrial workplace, may be both hazardous and lethal. In various worldwide military conflicts, eye injuries account for an estimated 4% to 9% of wartime injuries.[5-7] Soldiers with battlefield eye injuries must be evacuated to a fourth-echelon medical treatment facility (MTF) for definitive ophthalmic treatment and might not return to duty; even superficial foreign bodies in the eye will incapacitate a soldier at least 24 to 48 hours. To military commanders, losing 4% to 9% of their soldiers to eye injuries, even temporarily, may mean the difference between winning or losing a battle. As a result, efforts are currently underway to improve eye protection and reduce debilitating ocular injuries both in garrison and on the battlefield.

As compensation costs continue to rise, protecting and conserving vision has taken on increased importance. Initial efforts in occupational vision (sometimes referred to as industrial vision or eye safety) were directed toward reducing eye injuries by providing civilian employees working in eye-hazardous areas with industrial safety glasses. In 1992, occupational vision efforts within the army evolved to become the Vision Conservation Program, a more encompassing program composed of three program elements (occupational vision, eye safety, and environmental vision) and directed toward both soldiers and civilian workers.

LEGISLATION AND GOVERNMENTAL AGENCIES

The concept of occupational safety and health is relatively new, having evolved over the last 100 years. During the 19th century, on-the-job safety (including eye safety) was considered to be the responsibility of each individual employee, with employers assuming little or no liability in the event of an accident or death. The federal government did not become involved until the mid-1880s, and its focus was on job safety (the prevention of work-related accidents, injuries, and deaths) rather than occupational health (the prevention and control of work-related environmental disorders). Between 1890 and 1920, state governments became more active in job safety– and occupational health–legislation, while the federal government approached health problems through research and study programs. State laws, however, generally lacked uniformity (from state to state) and tended to be poorly enforced due to insufficient resources to hire inspection staffs.[8]

Occupational Safety and Health Act

Over the next 50 years, various federal initiatives to improve occupational safety and health were enacted, culminating with Public Law 91-596, the Occupational Safety and Health Act (OSHAct), which President Richard M. Nixon signed on December 29, 1970, and which went into effect in April 1971. The general-duty clause of the OSHAct emphasizes that each *employer*

shall furnish to each of his employees not only employment but also

> a place of employment free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees.[9(p4)]

In addition, employees are required to comply with the standards, rules, and regulations of the OSHAct and are responsible for their own actions and conduct. These provisions apply to safety in general as well as to eye safety.

Most of the OSHAct regulations that pertain to vision conservation are found in Title 29, Code of Federal Regulations, Section 133.[10] These regulations assert that protective eye and face equipment shall be required when there is a reasonable probability of the type of injury that this equipment can prevent. Eye protectors must provide adequate protection against certain hazards, be reasonably comfortable, fit snugly without interfering with the wearer's movements, be durable, tolerate disinfection, be easily cleaned, and be kept in good repair. Workers who require corrective lenses shall have the option of wearing prescription industrial safety eyeglasses, goggles over their ordinary dress safety glasses, or goggles that incorporate corrective lenses mounted behind the protective lenses. Eye protectors must be distinctly marked to facilitate their identification as approved industrial safety devices. The design, construction, testing, and use of ocular and facial protectors shall be in accordance with American National Standard Institute (ANSI) Standard Z87.1.[11]

Occupational Safety and Health Administration

The Occupational Safety and Health Administration (OSHA) has primary responsibility for

- developing mandatory job safety and health standards,
- enforcing the OSHAct through inspections of the workplace,
- maintaining a recordkeeping system to monitor job-related injuries and illnesses,
- implementing programs to reduce workplace hazards, and
- researching occupational safety and health issues.

OSHA requires that employers provide protective eyewear that meets ANSI standards at no cost to the employee.[10] Employees, management, and even visitors must wear approved eye protection when they enter or work in an eye-hazardous area. In some states, employers are required to provide any related technical services such as frame selection, lens design, ordering, verification, and fitting that are needed whenever corrective lenses are worn.[12] OSHA rules also require that employers provide safety training, including eye-safety training.

However, OSHA does not require employers to pay for their employees' vision examinations. Some federal agencies, including the Department of Defense (DoD), have taken the initiative to provide vision examinations for their employees who work in eye-hazardous areas. Most agencies have found that by providing eye examinations, they ensure that employees can see well enough to perform their jobs; worker productivity improves and the risk of costly eye injuries is reduced.

National Institute for Occupational Safety and Health

In conjunction with the passage of OSHAct, the National Institute for Occupational Safety and Health (NIOSH) was formed as a division of the Department of Health, Education, and Welfare (now the Department of Health and Human Services, DHHS) to assist in developing safety and health standards. Consequently, NIOSH is responsible for identifying occupational-safety and occupational-health hazards by gathering information through workplace surveys and laboratory research. Workplace surveys are accomplished through industrywide studies, which are specifically authorized by OSHAct. In addition to surveillance and data gathering, NIOSH conducts extensive research at its own laboratories and under contract at universities and private research institutes. Results of this research serve as the basis for recommending new health and safety regulations. These recommendations, known as *criteria documents*, are forwarded to OSHA, which has the ultimate responsibility for promulgating standards.

While NIOSH's first responsibility is to advise OSHA on standards, it also has other functions; for example, it publishes bulletins to inform health professionals about new health hazards and offers training programs on occupational safety and health. Numerous vision-related research projects, including studying the biological effects of ionizing and nonionizing radiation, have been performed at the institute's facility in Cincinnati, Ohio, and have resulted in several publications and articles concerning ultraviolet (UV) and infrared (IR) radiation.[13]

Food and Drug Administration

Before 1971, the eyeglass industry was largely unregulated. OSHAct was intended to protect industrial workers; there was no legislation like it to protect the vision of the general public. The lenses of dress safety glasses (street eyewear) were often ground extremely thin in order to improve their cosmetic appearance. Because these lenses shattered on minimal impact, many wearers' eyes were cut and irreparably damaged by broken glass.

To protect the vision of the general public, ANSI promulgated a voluntary set of standards for dress safety glasses in 1968. This standard, the *American National Standard Recommendations for Prescription Ophthalmic Lenses*, Z80.1-1968, recommended that all prescription lenses dispensed in the United States be impact resistant (the lenses should be able to withstand a specific impact: that from a ⅝-in steel ball dropped from a height of 50 in).[14] In December 1971, the Food and Drug Administration (FDA) issued a general policy statement on the use of impact-resistant lenses in eyeglasses and sunglasses. The general policy statement adopted an impact-resistance test similar to that stated in ANSI Z80.1-1968.

The Medical Devices Amendment of 1976 authorized the federal government to oversee the safety and effectiveness of all medical devices, including ophthalmic products such as eyeglass lenses, contact lenses, contact lens solutions, and ophthalmic medications.[15] Congress subsequently passed 21 CFR Part 801.410, *Use of impact-resistant lenses in eyeglasses and sunglasses*, which is also similar to ANSI Z80.1-1968.[16] Compliance with 21 CFR Part 801.410 by ophthalmic laboratories does not mean that the lenses are unbreakable or shatterproof; rather, it means that the lenses are impact resistant (as previously specified). Furthermore, the impact resistance of dress safety glasses should not be confused with that required for industrial safety glasses, which provide the industrial worker with greater eye protection.

American National Standards Institute

ANSI is a nongovernmental agency that has created more than 10,000 standards (with which compliance is voluntary). State and federal agencies often adopt ANSI standards as their own regulations, but ANSI's standards themselves have no statutory authority. Their purposes are to eliminate the duplication of standards and to develop a single, nationally accepted standard. The ANSI standards that apply to vision, and that have been (or will be) adopted by the FDA or by OSHA, include

- ANSI Z80.1-1987: American National Standard Recommendations for Prescription Ophthalmic Lenses;
- ANSI Z80.3-1986: American National Standard Requirements for Nonprescription Sunglasses and Fashion Eyewear;
- ANSI Z87.1-1989: American National Standard Practice for Occupational and Educational Eye and Face Protection;
- ANSI Z136.1-1986: American National Standard for the Safe Use of Lasers; and
- ANSI Z358.1-1990: American National Standard for Emergency Eyewash and Shower Equipment.

U.S. Army Environmental Hygiene Agency

In 1946, the Army Industrial Hygiene Laboratory, now the U.S. Army Environmental Hygiene Agency (USAEHA), initiated the Occupational Vision Program at some depots and arsenals within the army industrial base. This program was directed toward federal civilian employees rather than soldiers. By 1953, 19 army installations with 90,000 civilian employees had Occupational Vision Programs.[17] During the 1960s, the Occupational Vision Programs were expanded and directed toward civilian workers at all army installations. Presently, all army installations must have the Vision Conservation Program, which is directed toward both civilians and soldiers.

Currently, there are three optometrists (two military optometrists and one civilian industrial optometrist) who staff Program 63, *The Vision Conservation Program* within the Occupational and Environmental Medicine Division at USAEHA, located at Aberdeen Proving Ground (Edgewood Area), Maryland. Their mission is to write vision-conservation policies and doctrine for publication, to survey or assist the Vision Conservation Programs at various installations around the country, and to educate occupational health personnel on all aspects of vision conservation.

U.S. Army Regulations and Publications

U.S. Army Regulation (AR) 40-5, *Preventive Medicine*, Department of the Army Pamphlet (DA PAM) 40-506, *Vision Conservation Program*, and Technical Bulletin, Medical (TB MED) 506, *Vision Conservation*, constitute the basis for vision conservation in the army. Because army regulations are ever changing, readers should contact the USAEHA for the latest publications that pertain to vision conservation.[18]

THE VISION CONSERVATION PROGRAM

An effective vision conservation program requires a team of dedicated industrial and healthcare professionals: (*a*) an optometrist, an ophthalmologist, or both; (*b*) an occupational health physician, an occupational health nurse, or both; (*c*) an industrial safety specialist; and (*d*) an industrial hygienist. The installation medical authority (IMA) is responsible for appointing an optometrist as the installation vision conservation officer (VCO). In the absence of an optometrist, an occupational health nurse may be appointed as the acting VCO. The installation VCO is responsible for managing the installation's Vision Conservation Program, including assisting the industrial hygienist in identifying eye-hazardous areas and operations, advising the safety specialist on appropriate eye protection and vision-related safety issues, ensuring that employees working in eye-hazardous areas receive periodic vision screenings and vision examinations, and prescribing the appropriate corrective lenses for industrial safety glasses. The occupational health physician or nurse is responsible for monitoring the visual health of all employees, especially those who work in eye-hazardous areas, and for referring workers to an optometrist or ophthalmologist if they either fail the required vision screening or sustain an eye injury while on the job. The industrial safety specialist is responsible for approving all orders for *plano* (noncorrective) and prescription industrial safety glasses and for enforcing the wearing of safety eyewear throughout the workplace. The industrial hygienist is responsible for evaluating the workplace for eye-hazardous operations and taking action to reduce the risk of eye injuries.

An effective vision conservation program consists of the following essential elements, all of which will enhance vision, increase productivity, and reduce the risk of industrial eye injuries: (*a*) command and management commitment, (*b*) vision testing, (*c*) eye-hazard analysis, (*d*) accident prevention, (*e*) total participation, (*f*) education, (*g*) enforced use, (*h*) fitting and maintenance, and (*i*) emergency first-aid procedures.

Command and management commitment is absolutely essential for a dynamic vision conservation program. Commanders and supervisors bear the moral and legal responsibility for preventing eye injuries. Written policies and local standing operating procedures should be published to add emphasis and encourage compliance with the program.

Vision testing is utilized as part of preplacement and periodic physicals to find uncorrected vision problems that may decrease worker productivity, or, worse, lead to accidents. Workers who fail the vision screening should be referred for a complete vision evaluation to correct the visual deficiency.

Eye-hazard analysis requires the industrial hygienist or the installation safety specialist, or both, to evaluate every operation within the workplace, and to identify—and then mark with warning signs—all eye-hazardous operations. Once these hazards have been identified, the installation safety office should establish a job-title list that identifies those employees (by name) who work in eye-hazardous areas, the type of work that is done in each area, and the type of protection that is required for the job. At installations with access to the Occupational Health Medical Information System (OHMIS), the industrial hygienist should enter all data about eye-hazardous areas into the Health Hazard Information Management (HHIM) system, which is discussed in detail in Chapter 4, Industrial Hygiene.

Accident prevention can be a significant step in reducing or eliminating eye injuries. Safety training and motivation programs should also be utilized as a means for increasing safety awareness.

Total participation requires that eye protection be worn by all individuals (commanders, managers, soldiers, civilian employees, visitors, and contractors) when entering or working in eye-hazardous operations or areas. Plano safety glasses for visitors should be stocked at the installation safety office and at the entrances to buildings with eye-hazardous operations.

Education greatly enhances the effectiveness of a vision conservation program. All employees should be instructed in the proper use of eye-protective devices and should be reminded of the benefits of the program. A multidisciplinary team including plant supervisors, the occupational health nurse, the occupational health physician, the industrial hygienist, safety personnel, and the optometrist should develop the education program.

Enforced use is perhaps the most important, yet the most overlooked, element in an effective vision conservation program. Military and civilian supervisors should not hesitate to insist that soldiers and civilian employees wear their industrial safety glasses. Encouragement and rewards, rather than disciplinary procedures, should be used to encourage compliance, and whenever practicable, individuals who do wear appropriate eye protection should be praised or publicly rewarded. However, commanders and manage-

ment should publish the disciplinary procedures that will be implemented when civilian employees or soldiers fail to comply with the safety rules and regulations for wearing industrial safety glasses. Typically, these consist of

- a verbal or written warning for the first infraction;
- a 1-day suspension, invoked if the individual's noncompliant behavior persists (soldiers may be subject to disciplinary action under the Uniform Code of Military Justice); and
- initiation of steps to remove habitually noncompliant individuals from the job.

Fitting and maintenance of eye-protection devices helps to ensure wearing compliance. Shops should be discouraged from ordering inexpensive, nonadjustable plano safety glasses; likewise, workers should not wear eye protection that is ill fitting or does not work properly. An optician or qualified technician should be available to adjust safety eyewear, both prescription and plano. Lens-cleaning stations, stocked with lens-cleaning solutions, tissues, and antifogging products, should also be available throughout the plant.

Emergency first aid should be taught to all soldiers and civilians who work in eye-hazardous areas, especially where a chemical splash is possible. Those who work in areas where airborne foreign bodies (such as dust) make superficial injuries likely should be taught how to irrigate eyes with water. Those who work in areas where *ballistic* wounds (penetrating injuries caused by projectiles) are possible should be taught simple eye-patch and eye-immobilization techniques. Those who work in areas where chemical hazards are likely should be instructed in the proper use of eyewash fountains and in methods for retracting coworkers' eyelids.

OCCUPATIONAL VISION

The *occupational vision* element of the Vision Conservation Program consists of vision screenings and examinations. It is directed toward ensuring that soldiers' and civilian workers' vision is at least adequate—and preferably the best possible—to enable them to work productively, efficiently, safely, and comfortably. At a minimum, workers must be able to see well enough to perform their jobs safely, without risking injury to themselves or their fellow workers, or face job reclassification. If resources are available, workers should be provided a full range of vision services as a means for increasing productivity. Initiating and maintaining this element of the program presupposes that economic gains will follow: better retention rates; increased training efficiency; improved job performance; greater job safety; and, for civilian workers, better industrial relations.

Screenings

Vision screenings are designed to evaluate an employee's visual system, the results of which may be used to help select personnel for employment or to identify those employees whose vision might need further evaluation. Vision screenings, however, should not be confused with vision examinations; vision screenings superficially test a number of visual functions, while vision examinations are more thorough and are directed toward remediating a visual problem. Civilian workers generally receive their vision screenings at the occupational health clinic, while soldiers receive their vision screenings at the physical examination section (as part of a routine physical) or at the installation optometry clinic.

Binocular vision screening instruments (Figure 8-1) are more accurate than a simple Snellen eye chart. While a Snellen chart can only evaluate visual acuity, modern binocular testing devices check multiple visual functions, including:

- central visual acuity at both a distance of 20 ft or greater and at the nearpoint (13–16 in);
- muscle balance and eye coordination (the ability to keep the eyes pointed or directed toward an object);
- depth perception (the ability to judge the spatial relationships of objects); and
- color discrimination (the ability to differentiate colors correctly).

In addition, auxiliary lenses can be used to adapt these instruments for evaluating visual acuity at intermediate distances. Some testing instruments are even capable of determining rudimentary visual fields (the area of space visible to an eye).

Standards

During World War II, Joseph Tiffin and a team of researchers at Purdue University studied more than 4

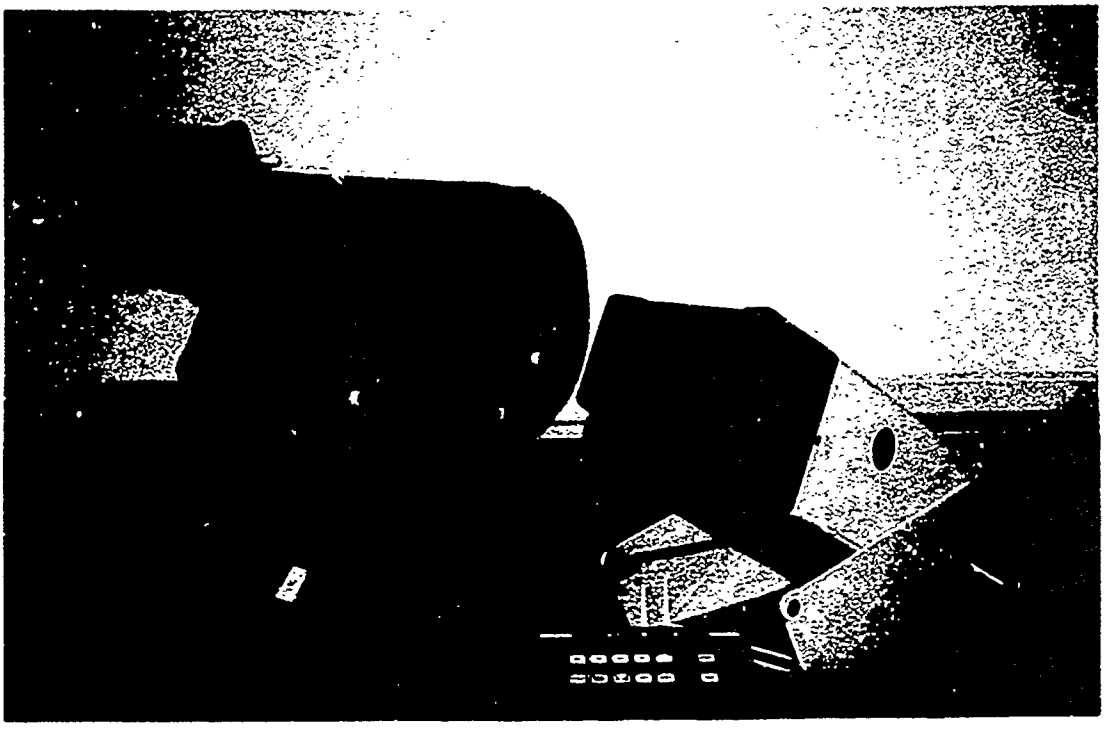

Fig. 8-1. The Armed Forces Vision Tester (AFVT), left, has been used for screening soldiers for decades and is still in use at most military entrance processing stations. The AFVT is mechanically operated, highly reliable, and is made of heavy-gauge steel. The AFVT is gradually being replaced by a new generation of lightweight, portable, and electronically operated vision screeners, such as the Titmus II Vision Screener, right. Other similar new vision screeners include the Optec 2000, manufactured by Stereo Optical, Inc., and the Sight Screener II, manufactured by American Optical Company.

million workers at thousands of different job sites. The purpose of this research was to improve industrial production during the war, a time when the healthiest males were unavailable, by maximizing worker efficiency and reducing industrial accidents. At the conclusion of their research, they developed minimum vision standards for six different categories of jobs.[19] Applying these standards to job applicants ensured that their visual acuity was adequate for the job and determined if and when they needed to be referred for vision correction. As a result of this research initiative, industry, including the DA, has universally adopted the six job vision standards. They were created before the advent of video display terminals (VDTs) and personal computers (PCs), however. To correct for this, The Surgeon General added a seventh vision standard for VDT operators serving in or working for the U.S. Army (Table 8-1).

Further research and industrial experience have shown that individuals who meet these standards should be able to perform their jobs safely and efficiently. However, these vision standards are often misinterpreted and should *not* be used as criteria for vision referrals. For example, while it may have been perfectly acceptable to allow a plumber with 20/30 vision to continue working at his job (vision standard no. 5) during World War II, today's standard of care mandates that this worker be referred for a complete vision examination to correct his visual acuity to 20/20. If, after a thorough vision examination, the plumber has a best (corrected) visual acuity (BVA) finding of 20/30, interpretation of vision standard no. 5 suggests that the worker can still perform his job safely and relatively efficiently. If the plumber's vision deteriorates (eg, as a result of cataract formation) to a BVA of 20/50, then his duties should be reduced or he should be reclassified into a different job until his cataract is removed and his vision improves.

There may be times when stricter vision standards should be adopted. In jobs with higher-than-usual risks to eyesight and personal safety, the usual job vision standards may be inadequate. For example, depth perception is critical for machinists, who may require stricter depth perception than 50 seconds of arc, as listed in vision standard no. 4. Color perception may not be a requirement for every job, but it might be

TABLE 8-1

RECOMMENDED JOB VISION STANDARDS

Vision Standard	Job Category	Acuity		Muscle Balance		Color Vision	Depth Perception
		Distance	Near	Distance	Near		
1	Administrative and clerical	20/30 20/25 OU	20/25 20/22 OU	4 eso 5 exo 0.5 vert	4 eso 5 exo 0.5 vert	Normal	NA
2	Inspector and assembler	20/35 20/30 OU	20/25 20/22 OU	4 eso 5 exo 0.5 vert	4 eso 5 exo 0.5 vert	Normal	50"
3	Vehicle driver, crane and forklift operator	20/25 20/22 OU	20/35 20/30 OU 0.5	4 eso 5 exo vert 0.5	4 eso 5 exo vert	Normal	40"
4	Machine operator	20/30 20/25 OU	20/30 20/25 OU	4 eso 5 exo 0.5 vert	4 eso 5 exo 0.5 vert	Normal	50"
5	Skilled trades: Plumber, millwright, and electrician	20/30 20/25 OU	20/25 20/22 OU 0.5	4 eso 5 exo vert 0.5	4 eso 5 exo vert	Normal	50"
6	Unskilled trades: Porter, warehouseman, and laborer	20/30 20/25 OU	20/35 20/30 OU	NA	NA	NA	NA
7	Video display terminal operator*	20/30 20/30†	20/25	NA 0.5 vert	ortho 8 exo	NA	NA

*Unlike the other, earlier standards, this one has an additional, intermediate distance visual acuity requirement
†Intermediate testing standard
NA: not applicable
OU: both eyes
eso: esophoria, the amount of inward turning of the two eyes, relative to each other
exo: exophoria, the amount of outward turning of the two eyes, relative to each other
ortho: orthophoria, the eyes directed toward infinity, the absence of eso- and exophoria
vert: vertical phoria, the amount of upward or downward turning of the two eyes, relative to each other

a necessity for certain workers such as electricians or painters. Because 8% of all males and 0.5% of all females are color blind, only those with superior color perception should be placed in jobs that involve colored wiring codes or colored dyes or paints. Because color discrimination declines with age (due to mild sclerosis or yellowing of the crystalline lens), these employees, when they reach 40 years of age, should have their color vision checked during their annual vision screening or physical.

Examinations

Civilian workers and soldiers should be referred for a complete vision examination when they (*a*) have significant vision complaints, (*b*) fail the vision screening (according to predetermined vision criteria), or (*c*) fail to meet the minimum vision standards for their jobs. Determining why an individual failed a vision screening is best left to the professional judgment of an optometrist or ophthalmologist. Military personnel are required to obtain their examinations at the nearest MTF. Civilians who work in eye-hazardous areas will either be provided vision examinations at government expense at the nearest MTF or be reimbursed for an examination at a private facility. Other federal civilian employees (those not working in eye-hazardous areas) who fail the vision screening must arrange and pay for their own vision examinations and eyeglasses.

EYE SAFETY

Eye safety is the element of the Vision Conservation Program that attempts to eliminate the incidence of eye injuries. Whether the setting is an industrial plant, a military training exercise, or a battlefield, civilian workers and soldiers can be exposed to a variety of eye hazards. Military commanders, industrial managers, and supervisors must understand that eye-hazardous areas and operations exist and must ensure that these hazards are periodically surveyed by the industrial hygienist. Initial efforts to reduce the risk to workers and their vision should be directed toward instituting engineering controls, administrative controls, or both. Additionally, installations must provide workers with personal eye protection that is commensurate with the potential risks to vision, meets or exceeds the legal standards, is comfortable to wear, and is cosmetically appealing.

Incidence of Eye Injuries

Two recent studies have evaluated the nature and degree of eye injuries. According to data from the National Health Interview Survey (the value for N is not available), most ocular injuries that cause severe visual impairment occur within the home (30%), while the workplace is the second most common location (27%).[20] Using data that were recorded in the Eye Injury Registry of Alabama (EIRA) from a study of 736 serious eye injuries that occurred August 1982 through May 1986, the most common sites of eye injuries were the workplace (28%) and the home (27%), followed closely by recreation sites (25%) (Figure 8-2). Other eye injuries occurred during criminal assaults (11%), while traveling (5%), and at school (1%). Plotting the data by age revealed that individuals 20 to 29 years of age had the highest rate for eye injuries (32%), followed by individuals 30 to 39 years of age (25%) (Figure 8-3).[21]

The EIRA study also found that blunt instruments were responsible for the most eye injuries (32%), followed by sharp instruments (23%), hammer-on-metal (chips that fragment off while metal is hammered) (11%), gunshots (8%), BB guns or pellet guns (7%), and fireworks (4%) (Figure 8-4). Blunt trauma was caused by objects such as fists, tree limbs, thrown projectiles (including balls), and objects propelled by lawn mowers. Penetrating trauma was caused by broken glass, fish hooks, tree branches, nails, screws, scissors, and thorns. Both extraocular and intraocular metallic foreign bodies were caused by hammer-on-metal injuries. Bottle rockets were the predominant source of fireworks injury. Alkali burns were the most common chemical injury. These burns can occur when lye or commercial drain cleaners come into contact with the eyes (eg, during domestic assaults or accidents).[21]

Industrial Eye Injuries

Almost 70% of all industrial eye injuries result from flying or falling objects that strike the eye. Nearly 60% of the objects that cause these eye injuries are smaller than the head of a pin and travel at high velocities. An additional 20% of industrial eye injuries are caused by chemicals, while the remaining 10% result from objects that swing from a fixed position (such as tree limbs, ropes, chains, or tools) and are unexpectedly pulled toward the worker.[22]

A Census Bureau study done in 1980 showed that 63% of all work-related eye injuries occurred within the construction industry. Of the eye injuries that occurred there, the most prevalent were to metal workers and welders (20%), followed by plumbers (8%), carpenters (7%), electricians (4%), and painters (4%). The other major group of eye injuries was sustained in the automotive-repair industry and accounted for 18% of work-related eye injuries.[23]

The relatively high number of eye injuries that occur in industry each year is surprising, considering that the surface area of the eyes is only approximately 0.54% of the entire frontal body surface area. Experts believe that at least 90% of workplace eye injuries could have been prevented had the worker simply used industrial protective eyewear.[22] According to the Bureau of Labor Statistics, almost 60% of workers in selected occupations who suffered impact eye injuries were not wearing eye protection at the time of the accident. Most of the workers who wore eye-protective devices and still sustained an injury were wearing the wrong kind of protective device for the particular hazard.[24]

Militarily Unique Eye Injuries

The number and incidence of ocular injuries has increased with each military conflict (Table 8-2). More accurate recordkeeping probably accounts in part for this trend, but a second and probably more important reason is that the weapons used in modern warfare increasingly depend on fragmentation as their mechanism of injury. Modern ballistic weapons are designed to break up into thousands of small-mass, high-velocity metallic fragments. These tiny fragments not only

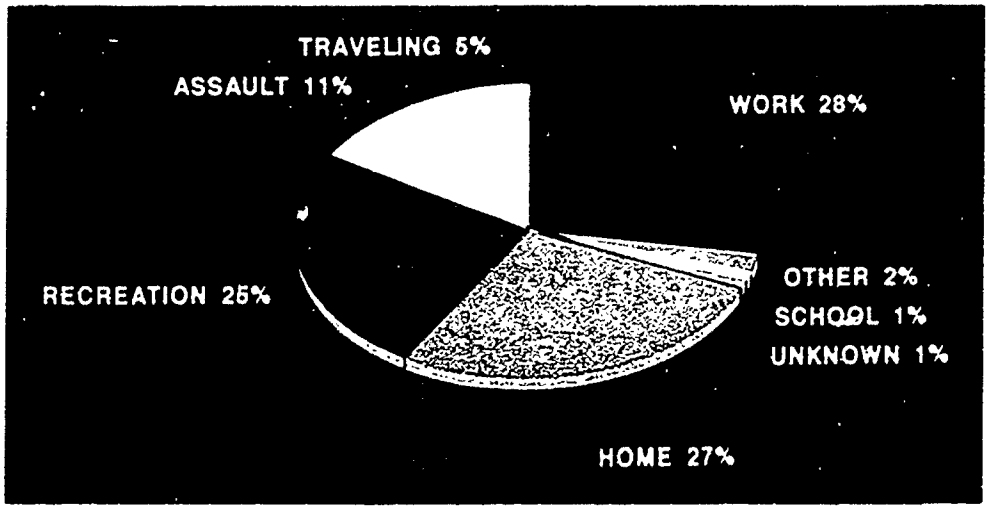

Fig. 8-2. The EIRA Study. Percentage of eye injuries by location of occurrence.

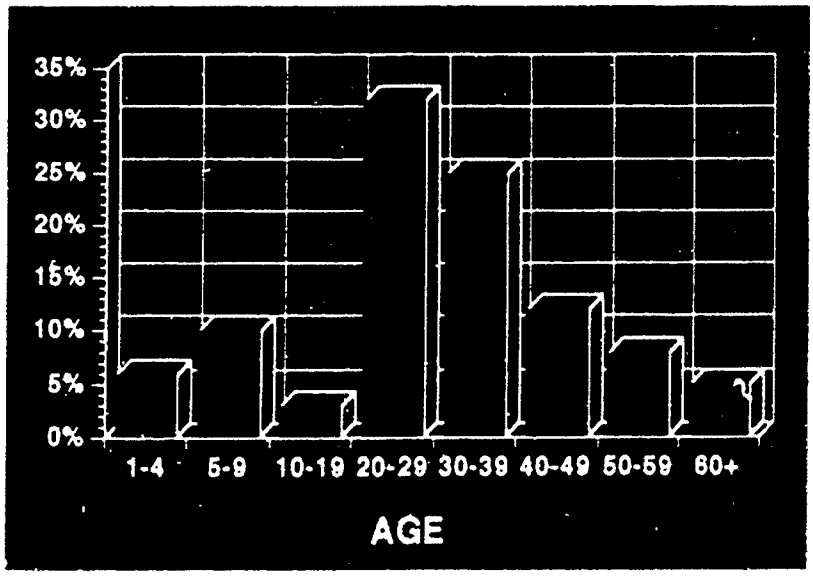

Fig. 8-3. The EIRA Study. Percentage of eye injuries by age.

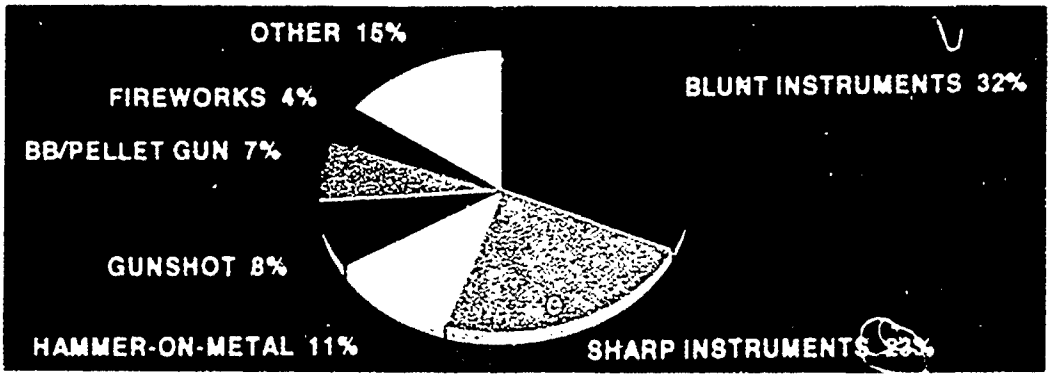

Fig. 8-4. The EIRA Study. Percentage of eye injuries by cause.

TABLE 8-2

INCIDENCE OF OCULAR INJURIES IN MILITARY ACTIONS

Military Conflict	Date	All Casualties (%)
American Civil War	1861–1865	0.57
Franco-Prussian War	1870–1871	0.81
Sino-Japanese War	1894	1.2
World War I	1914–1918	2.1
World War II	1939–1945	2.2
Korean War	1950–1953	4.1
Vietnam War	1965–1972	6.0–9.5
Arab-Israeli Six-Day War	1967	5.6
Arab-Israeli Yom Kippur War	1973	6.7
Arab-Israeli Lebanon War	1982	6.8

Sources: (1) Belkin M. Original, unpublished research. *Ophthalmological Lessons of the 1973 War: Prevention of Ocular War Injuries.* Jerusalem, Israel: Hadassan University Hospital, Dept of Ophthalmology. (2) Hornblass A. Eye injuries in the military. *Int Ophthal Clinics.* 1919;21:121–138.

cause vision-threatening injuries, but worse, they can also cause the loss of one or both eyes. (If these fragments were randomly to strike any other part of the body surface, the casualty might not even require evacuation from the battle zone.)

Each recent conflict has provided valuable information on the nature and extent of eye injuries that can occur when inadequate eye protection is worn. In the Six-Day War (1967), 25% of all eye injuries were perforations of the globe. In one battle for Jerusalem, there were only 100 casualties; 40 of them had perforating eye injuries. Ninety percent of these injuries were due to small-mass, high-velocity fragments, and nearly 25% of the injuries were bilateral perforations.[5,6] During the Vietnam War, an American soldier who was struck in the eye had a greater than 50% chance of losing it.[25] As a response to this rate, a primitive form of polycarbonate eye protection was tested on First Cavalry Division soldiers. This has led the U.S. Army Medical Department (AMEDD) and the U.S. Army Natick Research, Development, and Engineering Center to develop prototypes of eye protection: the Ballistic/Laser Protection Spectacle (BLPS) and the Special Protective Eyewear Cylindrical System (SPECS).

A change in the prevalent mechanism of injury in ocular casualties has occurred with technological advances in tactics and weaponry. Prior to the 1973 Yom Kippur War, the vast majority of eye injuries were due to the fragmentation of artillery projectiles. With the changes in tactics, only 14% of the eye injuries that were sustained during the 1973 Yom Kippur War were due to artillery projectiles. Instead, antitank weapons caused the highest number (72%). Sixty-five percent of the soldiers who sustained eye injuries were in tanks or armored personnel carriers, with tank commanders, tank crews, and armored infantry being the most vulnerable. Only a minority of eye injuries were inflicted on soldiers in open spaces.[5,6]

During peacetime, while troops are garrisoned, accurate eye-injury data have been difficult to obtain. To date, the army has neither an eye-injury data-collection form to collect such information nor a database from which to analyze it. However, it is not unreasonable to assume that statistics concerning civilian industrial workers might also apply to peacetime active-duty soldiers.[26,27] Data collected from army personnel during 1977 to 1981 revealed that 3,556 eye injuries, or approximately 710 eye injuries per year, had occurred. However, these figures are probably low because they include only soldiers who were hospitalized with eye injuries and exclude all soldiers who were examined in outpatient clinics. An analysis of the data reveals that the most prevalent causes of eye injuries were machinery or tool accidents (20%), land-transport vehicles (16%), athletics or sports (12%), falls or unspecified agents, (9%), and guns or explosives (7%).[28]

Ballistic and Mechanical Hazards

Ballistic and mechanical hazards are ubiquitous both in the industrial environment and on the battlefield. In industry, these hazards tend to be associated with metal shops (with equipment such as metal lathes, drill presses, and punch presses) and automotive shops (eg, rust particles can fall into the eyes of a mechanic who is working under a vehicle). During military training exercises and under battlefield conditions, many soldiers sustain ocular injuries when branches snap back into their faces, mortar or grenade fragments strike their faces, or rounds from their own weapons explode.

Foreign Bodies

Projectiles that impact and are retained on or around the eyes are called *foreign bodies*. They are generally classified as metallic or nonmetallic, toxic or nontoxic, and penetrating or nonpenetrating.

Civilian workers or military casualties with nonpenetrating foreign bodies of the cornea (Figures 8-5 and 8-6) require referral to an ophthalmologist or qualified optometrist for removal. Superficial foreign bodies (those that are located on or within the corneal epithelium) should be removed with irrigation, a needle, or a *spud* (a blunt, metal probe). Embedded foreign bodies or those with rust rings from iron-containing metals usually require removal with an Alger brush, dental burr, or large-gauge needle. Foreign bodies that penetrate into the corneal stroma will ultimately leave a scar. The resultant degree of visual impairment will depend on the scar's location: those that are closest to the center of the cornea will produce the greatest loss of visual acuity.

Penetrating foreign bodies (Figures 8-7, 8-8, 8-9, and 8-10) breach the cornea or sclera. These injuries usually occur during mechanical operations such as high-speed drilling, mechanical grinding, and pneumatic riveting. These perforating wounds of the cornea or sclera are often small and barely visible to the examiner and may have little or no associated pain (other than the initial insult to the eye). In many cases the entry wound is so small that diagnosis at the worksite is difficult; a hole in the iris or an irregular pupil may be the only evidence that the worker has sustained a penetrating injury. Fifteen percent of all intraocular foreign bodies are retained in the anterior chamber, 8% in the lens, 70% in the posterior chamber, and 7% in the orbit.[29] A worker who suspects that he or she has sustained a penetrating injury from a foreign body, and who was not wearing appropriate eye protection at the time, should be referred immediately to an ophthalmologist for evaluation, radiography, diagnosis, and possible surgery.

Blunt Trauma

A direct blow to the eye by a blunt missile (such as a clenched fist, a squash ball, or even a champagne cork) can produce one or more of the following signs: hyphema (a collection of blood in the anterior cham-

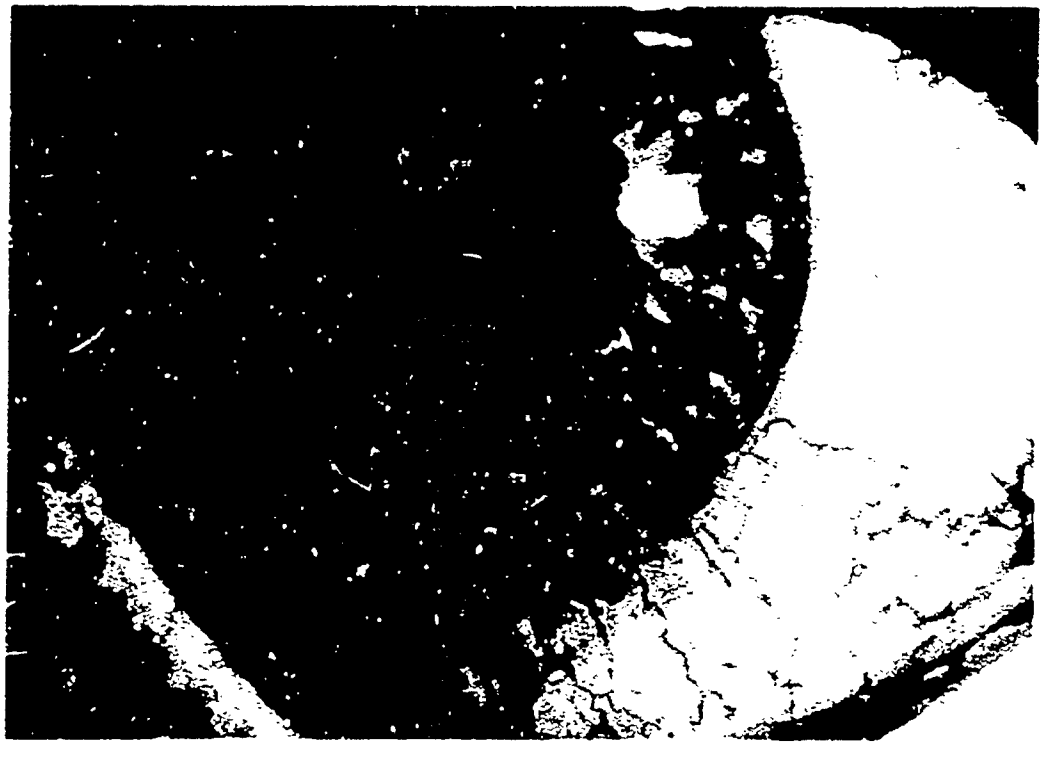

Fig. 8-5. Small, superficial, peripheral foreign bodies, such as that shown on the lower temporal portion of the cornea of the left eye, are often blown into the eye. These superficial foreign bodies can be easily removed (after the cornea has been anesthetized) with a needle or spud and leave little or no residual scarring. After the foreign body has been removed, treatment consists of topical antibiotics, analgesics, and a pressure patch (if the patient is uncomfortable); topical steroids should be avoided because they will slow healing. Photograph: Courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.

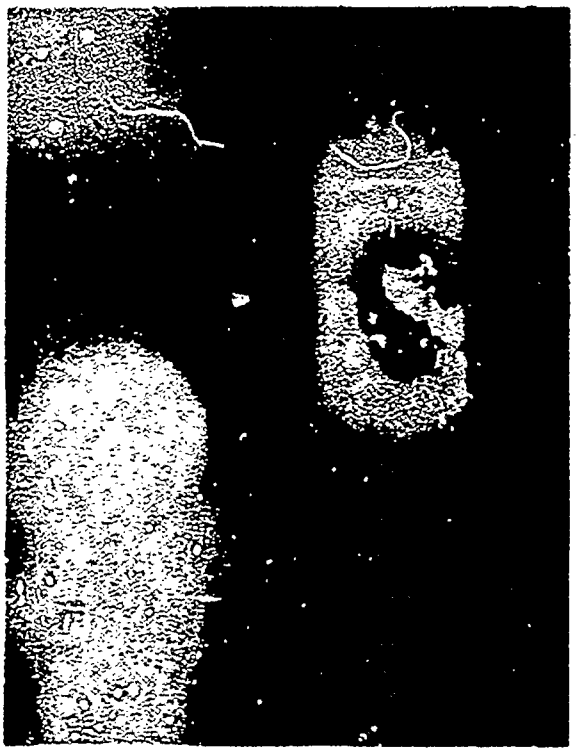

Fig. 8-6. Iron-containing metallic foreign bodies are typically projected onto the eye and leave a rust ring, which must eventually be removed with a spud, needle, or Alger brush if proper healing is to occur. The rust ring pictured here (the metallic foreign body has already been removed) shows that the original metallic foreign body struck the cornea with minimal force, with damage limited to the corneal epithelium. However, foreign bodies that penetrate beyond the corneal epithelium into the corneal stroma will ultimately leave a corneal scar; scarring at or near the visual axis (the line of sight) can ultimately degrade visual acuity, depending on the size and density of the resulting scar tissue. Photograph: Courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.

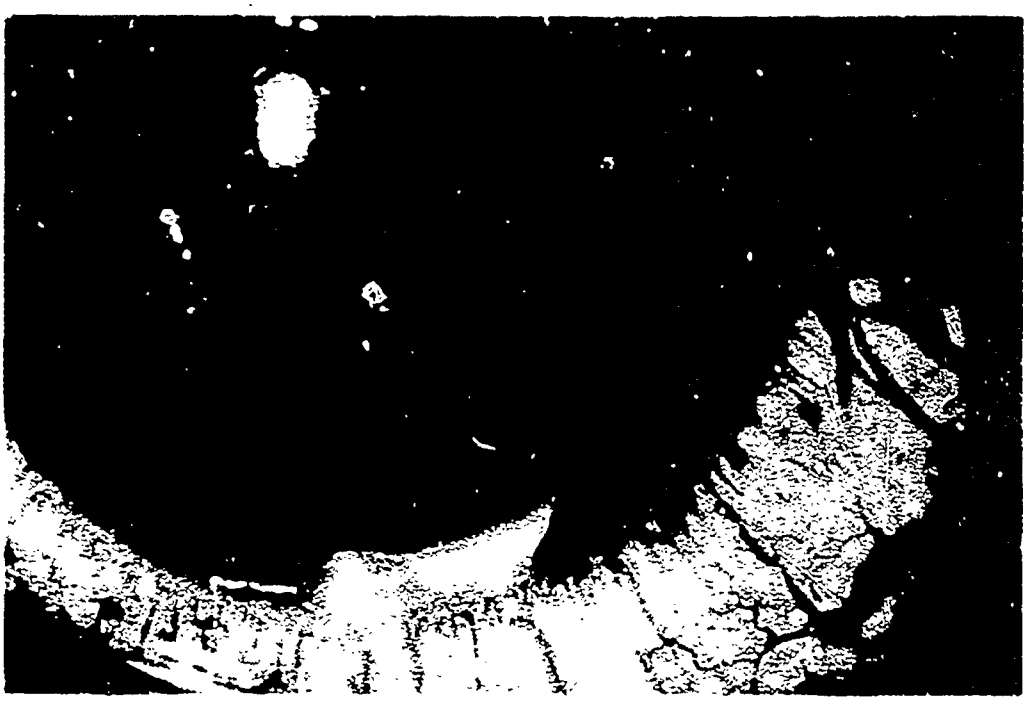

Fig. 8-7. A large, metal burr penetrated the lower temporal limbus of the left eye of a metal-lathe worker who was not wearing eye protection. The cornea and iris root were involved and ophthalmological surgery was required. Because the injury was peripheral to the visual axis, there was minimal effect on visual acuity; however, the worker missed several days of work. Photograph: Courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.

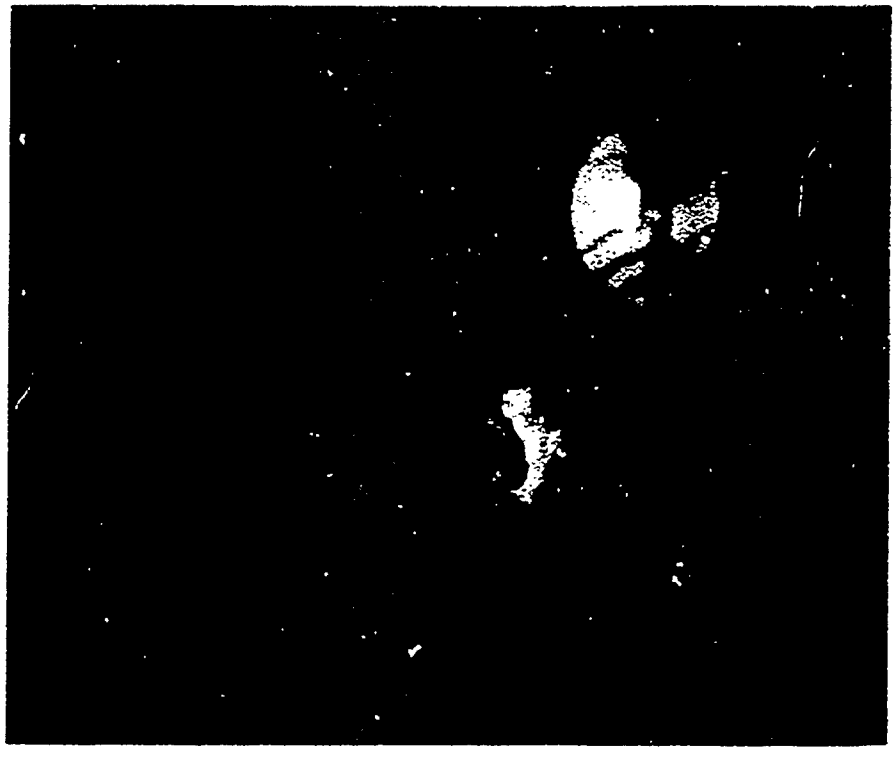

Fig. 8-8. Foreign bodies often have sufficient velocity to perforate the cornea or sclera—passing through internal structures such as the aqueous, iris, and vitreous—and can penetrate the lens or retina. Workers may not be fully aware that they have suffered an eye injury; the only visible signs may be some minor redness of the eye or an additional hole in the iris. These workers must be referred for evaluation and possible surgical treatment immediately. Industrial safety glasses might have precluded this injury.

Fig. 8-9. Whenever a worker suspects that a penetrating eye injury has occurred, he or she should be referred for radiography and further evaluation. The penetrating metallic foreign body in the right eye is easily seen in this radiographic (Water's) view. Photograph: Courtesy of David Talley; formerly, Redstone Arsenal, Huntsville, Alabama.

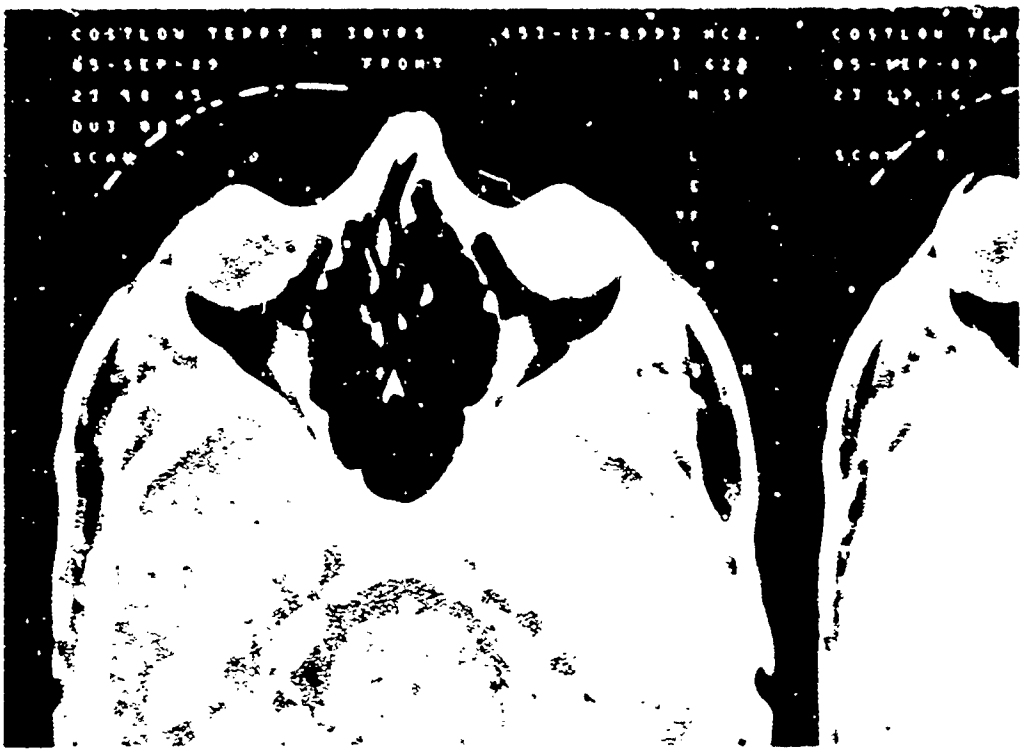

Fig. 8-10. A computed tomograph (CT) scan is often useful in determining the exact location of a penetrating foreign body, especially if surgical removal is required. This large foreign body is lodged on the nasal retina of the right eye. Photograph. Courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.

ber); subluxation (dislocation) of the crystalline lens; a blowout fracture of the orbital floor or nasal wall; iridodialysis (a rupture or tear of the iris from its base on the ciliary body); traumatic pupillary mydriasis (dilation caused by temporary or permanent paralysis of the sphincter muscle of the pupil); traumatic iritis; vitreous hemorrhage; and retinal hemorrhage, tears, or detachments. These signs all require that the patient be referred immediately to an ophthalmologist. Blunt trauma may also be characterized by ecchymosis (a black eye), subconjunctival hemorrhage, and occasionally, crepitus (air leaking under the skin) if a sinus has been injured.

Traumatic hyphemas (Figure 8-11) range from *mild*, wherein only a few erythrocytes are found floating in the anterior chamber during slitlamp examination, to *partial*, wherein blood pools in the lower portion of the anterior chamber; to *total*, wherein the anterior chamber virtually fills with blood. Partial hyphemas are usually resorbed through the trabecular meshwork of the anterior chamber within a few days. However, approximately 20% of hyphemas rebleed 3 to 5 days after the injury. Of the eyes that rebleed or have an initial total hyphema, 20% to 50% will be left with visual acuity of 20/40 or worse. Eight percent of the injured eyes that manifest hyphema will have a dislocated lens and approximately 7% will develop glaucoma in later years. Five to ten percent of traumatic hyphemas require surgical repair.[5] Complete bedrest is indicated, and an ophthalmologist should follow this condition to ensure that additional damage to the eye, which may not have been apparent initially, did not occur.

In a blowout fracture of the orbit, the energy of the impact forces the contents of the orbit (the eye, extraocular muscles, neurovasculature, and orbital fat) either downward, fracturing the orbital floor and forcing some of the orbital contents into the maxillary sinus, or nasalward, fracturing the wall of the ethmoid sinus. As a result, enophthalmus (the eye sinks into the orbit), diplopia (double vision), and entrapment of the extraocular muscles in the maxillary sinus or the ethmoid sinus can occur.

Chemical Hazards

While all eye injuries are considered to be potentially vision threatening and are emergency situations, chemical eye injuries must be treated immediately, even before the victim is transported to a medical facility for definitive care. *All chemical injuries, especially those that involve alkalis, pose a significant threat to*

Fig. 8-11. Hyphemas are usually caused by blunt trauma to an unprotected eye and vary from mild, in which there are a few erythrocytes in the anterior chamber, to total, where the entire anterior chamber fills with blood. This eye has a partial hyphema. It resorbed within a week with no loss of vision. All workers who suffer blunt trauma to the eye and adnexa should be referred for evaluation.

vision. The intact epithelium of the cornea resists damage from a rather wide pH range; however, a chemical with a pH less than 4 or greater than 10 increases cellular permeability of the corneal epithelium.[30] Immediate irrigation with water can help to prevent further loss of vision. Any delay in treatment can cause pain and irreversible loss of vision.

Any material that is labeled as an irritant or a corrosive can cause eye injury. Anterior segment burns from Mace or tear gas should be treated as chemical burns. Ocular injury from sparklers or flares that contain magnesium hydroxide should also be managed as chemical, rather than thermal, burns; if left untreated, the magnesium hydroxide will continue to cause damage to the eye long after the effects of the thermal damage have subsided.[29]

Acid Burns

Acid burns rapidly damage superficial tissues but are neutralized by protein barriers (which prevent deep penetration) within the first few minutes to hours (Figure 8-12). There are several exceptions, such as hydrofluoric acid or acids containing heavy metals, which can produce a penetrating injury because they resist the protein barrier. Automobile-battery explosions are probably the most common cause of acid burns to the eyes. These injuries tend to occur more frequently during the winter months when a lighted match or cigarette, or faulty jumper cables provoke an explosion.[28]

Alkali Burns

Alkali burns may initially appear innocuous, but they tend to progress rapidly and have a poorer prognosis than do acid burns (Figure 8-13). Alkalis such as lye, lime or plaster of Paris, or ammonia can penetrate to damage the deeper structures of the eye. Alkali burns tend to be more severe because alkalis combine rapidly with cell-membrane lipids this disrupts the microstructure of the cell and the stromal mucopolysaccharides, causing the eye tissue to soften. Thus, alkali burns of the eye require immediate but careful decontamination and treatment.

Evaluation and Treatment

Chemical burns to the eye are classified as mild, moderate, or severe. Mild burns cause corneal opacification, blurring of iris detail, and minimal ischemic necrosis of the conjunctiva and sclera. Reepithelization will be sluggish and a mild corneal haze will form, usually resulting in minimal loss of visual acuity. Moderate burns cause stromal opacification, with increased corneal thickness and considerable iritis. Superficial neovascularization of the cornea and conjunctiva may leave persistent epi-

Fig. 8-12. Acid burns to the eyes are most commonly caused by battery explosions, as was this one. The fact that the casualty's upper lid prevented damage to the superior portion of the cornea is of little consolation. After the initial chemical trauma to the corneal epithelium, protein barriers limit deep penetration by the acid. Workers in jobs with higher-than-normal risk, such as chemists and battery maintainers, should wear chemical goggles and a face shield.

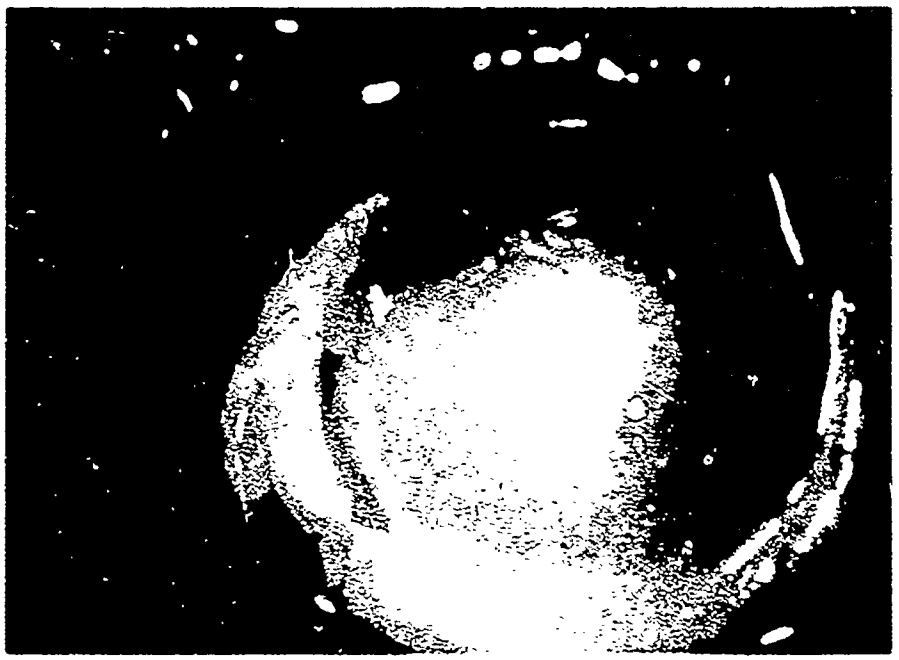

Fig. 8-13. Caustic alkali burns to the eye tend to be more serious and debilitating than acid burns. This cornea is extremely edematous, and blanching of the conjunctival and scleral vessels has occurred. To prevent further damage and deep-tissue penetration, alkali burns must be irrigated with water immediately; irrigation must continue while the casualty is transported to a medical treatment facility. Photograph: Courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.

Fig. 8-14. Severe acid or alkali burns that have not been promptly and thoroughly irrigated with water often result in permanent corneal opacification and neovascularization. A full-thickness corneal transplant to restore vision in this eye will be difficult to perform because the vessels have encroached on the central cornea. If a corneal transplant is not possible, then this worker could wear a cosmetic soft contact lens with a painted iris. These lenses restore only the appearance, and not the function, of the damaged eye.

thelial defects that ultimately lead to stromal thinning and perforation. Permanent visual impairment invariably results (Figure 8-14). The most severe burns (usually alkali burns) cause marked corneal edema and haze, along with blanching of the conjunctiva and sclera. Due to the blanching effect of the damaged cornea, any underlying iritis may go undiagnosed. Ulceration eventually occurs and perforation, due to collagenase-like enzymes that are released to heal the inflamed tissues, may ensue.[29]

This cannot be overemphasized: *chemical burns—especially alkali—must be treated immediately.* Emergency treatment involves copious irrigation using the most readily available water. *Do not wait* for a sterile physiological or chemical neutralizing solution. The rescuers should hold the victim's eyelids apart even though spasm of the orbicularis oculi muscle can make this extremely difficult. During the initial 15-minute lavage (as a minimum), the rescuers should telephone the nearest emergency room or ophthalmologist's office to inform the staff of the victim's pending arrival. After the casualty arrives at the MTF, irrigation should continue for at least 1 hour or longer, or until pH (litmus) paper demonstrates that the conjunctival pH is normal (the pH has returned to 7.3–7.7). Eyelid retractors may be necessary to keep the victim's eye or eyes open, and topical anesthetics may be needed to relieve the pain.

Eyewash Fountains

ANSI Standard Z358.1-1981 gives directions for the proper installation and maintenance of eyewash fountains and showers. Because the first 15 seconds following a chemical splash are the most critical, the standard recommends that eyewash fountains and showers be located as close to chemically hazardous sites as possible, preferably within 50 ft. The standard also recommends that all eyewash fountains be installed at the same height and in the same position and operate in the same manner throughout the workplace. Hand- or foot-operated valves must allow the eyewash fountain or shower to remain on after they have been activated. Furthermore, the standard recommends that eyewash fountains be identified with a sign and that the surrounding area be painted a bright color, such as high-visibility yellow and black.[31] If feasible, an alarm should be installed on the fountain and should sound when the fountain has been activated, to notify fellow workers that a chemical accident has occurred.

Eyewash fountains should be checked and maintained routinely. Plumbed eyewash fountains must be

Fig. 8-15. Eyewash fountains and showers should be (*a*) located within 50 ft of a potential chemical hazard, (*b*) accessible, and (*c*) painted high-visibility colors. The hand- or foot-operated valves must allow the eyewash fountain or shower to remain turned on after being activated. Because chemical trauma to the eyes can cause severe spasm of the lid musculature, victims may be unable to open their eyes by themselves. Workers in chemically hazardous areas should be instructed on assisting injured coworkers at eyewash fountains.

able to deliver 3 gallons of potable water per minute for 15 minutes (Figure 8-15). In remote sites where a plumbed water source is not available, self-contained, portable eyewash stations must be able to deliver 0.4 gallons of water per minute for at least 15 minutes.[31] However, the use of portable eyewash stations (especially gravity-flow eyewash stations) is generally discouraged because the holding tanks must be cleaned regularly and the bacteriostatic water, which is expensive, must be changed monthly. Squeeze-bottle eyewash stations, which are often poorly maintained and have a propensity to harbor microorganisms, are prohibited by army regulations.[32]

Regardless of how well eyewash fountains and showers are installed and maintained, employees must be properly instructed in their use. Training should emphasize that victims may be unable to open their eyes after a chemical splash; the lid musculature can react so quickly and powerfully that it may be impossible for victims to open their eyes without help.

Concerns have surfaced recently regarding the safety of eyewash fountains. *Acanthamoeba polyphaga, A. hatchetti, and A. castellanii* are small, free-living protozoa found in soil, air, and water, and have been cultured from water standing in the pipes of eyewash fountains.[33] Eyecare specialists are concerned that acanthamoeba organisms could be introduced when the eyes are lavaged at a contaminated fountain after a chemical splash. Acanthamoeba keratitis, a rare but serious infection of the cornea, has most often been associated with contact-lens wearers who use homemade saline solutions made with contaminated tap water. To date, there have been no reported cases of acanthamoeba keratitis following the use of emergency eyewash fountains[34]; however, to reduce the risk of acanthamoeba contamination, army policy recommends that eyewash fountains be flushed weekly.[32] Army policy does not specify the length of time for flushing, but scientific literature recommends 3 minutes.[34]

Simple methods of eye irrigation tend to leave chemical residua that can continue to destroy the remaining cornea, in part because chemical burn victims fight to keep their eyes closed in spite of the absolute necessity for flushing the eyes with water. A new method of eye irrigation is being used at some emergency rooms and

industrial facilities throughout the United States. The new irrigation method uses a Morgan lens: a large contact lens that can be slipped between eyelids that are open only 2 mm. A small polyethylene tube, which is connected to one side of the Morgan lens, pumps the irrigation solution into the eye. Once the lens is in place, the soothing bath of running fluid tends to calm most chemical-accident victims.[35]

Radiant Energy Hazards

Radiation hazards can be classified as industrial or environmental. Because many types of radiation are found in industrial settings, for purposes of this textbook they are classified within the realm of industrial eye safety. These hazards should be evaluated by the industrial hygienist and should be reduced or eliminated if they pose a threat to the workers' vision or ocular health.

Ultraviolet Radiation

UV radiation, the most common cause of light-induced ocular injury, is invisible to the human eye. It occupies the region of the electromagnetic (EM) radiation spectrum between the blue end of the visible-radiation region and the region of X radiation (Figure 8-16).

The categories of UV radiation are (*a*) UV-A (380–315 nanometers [nm] in wavelength), (*b*) UV-B (315–290 nm), and (*c*) UV-C (290–100 nm).[36–38] When individuals are exposed to sunlight, UV-A causes human skin to tan (the radiation stimulates the melanocytes to form pigment), and UV-B causes skin erythema or sunburn. (Large welding arcs can produce equally hazardous quantities of UV-B radiation.) UV-C is potentially the most dangerous to human health: it is used as a bactericidal and germicidal agent and is potent enough to kill humans. The ozone layer in the earth's upper atmosphere only partially absorbs UV-A and UV-B radiation; fortunately, however, it absorbs all solar radiation lower than 294 nm. Recent scientific literature reports that the protective ozone layer is thinning, and this could increase the amount of UV-C radiation to which we are exposed.[39] Currently, the only UV-C radiation sources that are detrimental to human health are manmade, such as germicidal lamps and some large welding arcs, and welders and employees who work in research laboratories are the most likely to be exposed to it.

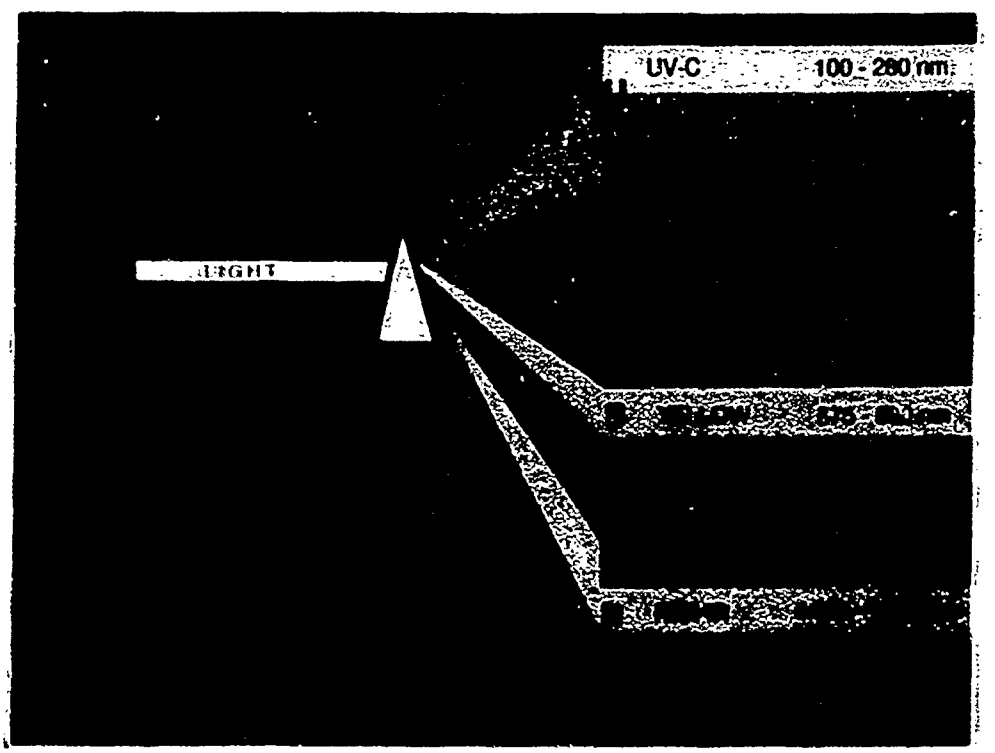

Fig. 8-16. Visible radiation, also known as light, occupies a very small region (400–780 nm) of the electromagnetic spectrum. Ultraviolet and infrared radiation, both invisible to humans, lie just above and below the visible spectrum.

Excessive exposure of the cornea to UV radiation causes photokeratitis, an acute ocular condition that is characterized by a massive sloughing of the central corneal epithelium. (Laymen call this condition "snow blindness" or "welder's flash.") The degree of corneal involvement depends on the victim's duration of exposure, the content of UV wavelengths that the source emits, and the energy level of the luminance. The latency for development of symptoms of UV photokeratitis varies from 30 minutes to 24 hours, depending on the radiation dose that was received. Symptoms can range from mild irritation and the sensation that an ocular foreign body is present to severe photophobia, pain, and spasm of the eyelids. Clinical signs include punctate lesions of the corneal epithelium that can be observed with a sodium fluorescein stain. Therapy for this condition includes short-acting cycloplegics, such as cyclopentolate (1%) or homatropine (2%), to relieve ciliary spasm. Topical antibiotics should be applied to prevent secondary infection. Pressure bandages, sedatives, and analgesics are not absolutely necessary but may make the patients more comfortable. Victims of UV overexposure are usually incapacitated 6 to 24 hours; complete reepithelization of the cornea usually occurs within 48 hours after the onset of symptoms.[29]

UV radiation was thought for many years to affect only the superficial structures of the eye (the cornea and conjunctiva). But recent studies suggest that the depth of tissue penetration is wavelength dependent. The cornea absorbs nearly all UV radiation of wavelengths shorter than 290 nm, but it allows longer UV wavelengths to be transmitted (to pass through it) to varying degrees: UV radiation with wavelengths of 250 nm to 200 nm (UV-C) primarily affects the corneal epithelium, while UV radiation with wavelengths of 315 nm to 295 nm (UV-B) tends to affect the corneal stroma and endothelium.[36]

While the cornea and the conjunctiva absorb most UV radiation, UV-A and UV-B (depending on their wavelength) can be transmitted to the lens and the retina. A recent study of 838 Chesapeake Bay watermen concluded that increased exposure to UV-B radiation increases the likelihood that *cortical cataracts* (opacification of the cortex or outer covering of the crystalline lens) will form.[40] However, the study failed to demonstrate any relationship between UV-A radiation and any type of cataract, or UV-B radiation and *nuclear cataracts* (opacification of the nuclear or innermost layers of the crystalline lens).

UV radiation can also cause retinal damage. In *phakic* eyes (eyes that have a crystalline lens), UV radiation with wavelengths greater than 320 nm is transmitted in varying degrees through the eye to the retina. In *aphakic* eyes (eyes that have neither a crystalline lens nor a plastic intraocular lens [IOL] implant), UV-A and UV-B radiation may cause retinitis. In cases of *pseudophakia* (eyes that have had the crystalline lens removed and replaced with a plastic IOL implant), ophthalmic surgeons are now using IOL implants that specifically block the transmission of UV radiation to the retina. Despite these medical advances, additional research on the retinal effects of UV radiation is needed.

Commercial and industrial sources that produce high-UV-radiant exposure levels (such as UV lasers, welding and carbon arcs, industrial sterilizers, spectrophotometers, and devices to photoharden dental resins) are more likely to produce harmful ocular and dermatological effects if UV protection is incomplete or inadequate. Engineering and administrative controls should be used to reduce the hazards from these sources before personal protective devices are required. Workers who are in close proximity to welding operations must be protected against accidental UV exposure with noncombustible or flame-resistant screens or shields. In addition, painted walls should have low reflectivity for UV radiation. Personal protective equipment (PPE) such as welding masks and goggles will be discussed later in this chapter.

Infrared Radiation and Heat

IR radiation occupies the portion of the EM spectrum just beyond visible red light and includes wavelengths 770 to 1,000,000 nm. IR radiation is used in industry to dry and bake paints and varnishes; heat metal parts for forging and thermal aging; and dehydrate textiles, paper, leather, meat, and pottery.

Although IR radiation can cause injuries to the cornea, iris, and retina, its damage to the lens is the most likely to degrade vision. Minor IR burns are usually of little consequence: they produce only temporary edema and erythema of the eyelids and little or no damage to the globe. However, continuous or excessive exposure, such as that from furnaces or similar hot bodies, has been known to produce heat cataracts. This type of lens opacity causes sloughing of the lens cortex and decreased visual acuity. Opacities of the posterior portion of the lens may also be observed.[41] These cataracts are becoming less common as large industrial blast furnaces become more automated.

Lasers

Lasers generate a beam of radiation that is *monochromatic* (of a single wavelength) and *coherent* (all of the EM waves are spatially in phase). The beam has a very small *angular divergence* (the light does not widen

significantly over the length of the beam). Depending on the lasing medium, the output beam may be in the visible radiation region (400–780 nm) and would therefore be seen as light, or it may be in the invisible (UV or IR) regions. The output beam may be a continuous wave, a pulse, or a train of pulses, depending on the manner in which the energy is pumped into the lasing medium.

Laser energy can be transmitted to the eye in three ways. The most hazardous transmission method is *direct laser exposure*, in which the individual looks directly into the laser beam. The second and almost equally hazardous method is *specular reflection*, which occurs when laser energy is reflected toward the eye from a shiny, highly polished surface such as a mirror, a piece of flat glass, or even the inside of a tin can. The method that is least hazardous to the eye is *diffuse reflection*, in which the laser energy is reflected toward the eye from a dull (nonshiny) object such as a wall or a tree.

Depending on both the wavelength and the energy of the laser emission, individuals who inadvertently look at a laser beam may suffer ocular injury and possible loss of vision. Far-IR laser radiation (1,400–1,000,000 nm) and UV laser radiation cannot pass beyond the anterior structures of the eye. Consequently, low-energy UV- and far-IR-laser radiation are absorbed by the anterior segment of the eye and can cause photokeratitis similar to welder's flash. High-energy, far-IR radiation will produce thermal burns to all layers of the cornea, which may lead to permanent corneal scarring.

Visible light and near-IR laser radiation (780–1400 nm) can pass through the eye to reach the retina. The degree of retinal damage is directly related to the amount of ambient energy and the length of exposure. Long exposures (many seconds) cause photochemical damage to the retina, while short exposures result in thermal injury. The heat from the laser emission causes thermal coagulation of the photoreceptors and other structures of the retina. Pulsed lasers (lasers that emit radiant energy in very short [nanoseconds] exposures) create intense energy that cannot quickly be dissipated; consequently, retinal cells explode and create shock waves that mechanically destroy surrounding tissues and cause a loss of retinal function (Figure 8-17). The shock waves can also rupture blood vessels in the choroid or retina and cause detachment of the retina. Blood that hemorrhages into the vitreous humor can resorb slowly and mechanically obstruct vision (Figure 8-18). In this event, the prognosis for regaining normal vision is usually poor, especially if the damage occurs in the central macular area.

Classification of Lasers. As a result of the hazards that lasers pose, an empirical classification system has been established to warn users and observers of the associated risks:

- *Class 1 lasers* are safe under virtually all viewing conditions because the output beam is considered to be incapable of causing radiation damage, and is therefore exempt from any control measures or other forms of surveillance.
- *Class 2 lasers* are low-power devices that emit only visible radiation. The maximum power of this class is limited to 1 milliwatt (mW), which is measured by a 7-mm pupil diameter in a *viewing box* (a black-box model, which is used to simulate a human eye). Because the duration of the normal blink reflex is 0.25 seconds, and 1 mW is not injurious at this duration, Class 2 lasers are considered to be eye safe unless a person makes a deliberate attempt to look into the beam for a period longer than 0.25 seconds.
- *Class 3 lasers* are medium-power lasers and are subdivided into two subclasses. Class 3A lasers produce visible radiation that, when viewed directly, is not hazardous to vision; however, the beam may be hazardous when collected and directed into the eye, as with binoculars. Class 3B lasers produce sufficient power to produce injuries when viewed directly or by specular reflection. Class 3 lasers usually do not present a combustion hazard.
- *Class 4 lasers* are high-power lasers. They are hazardous to the eyes and skin when there is direct or specular-reflection exposure, and some very high power Class 4 lasers can be hazardous even with diffuse reflecting exposures.[42–45] Class 4 lasers can present a combustion hazard if used improperly.

Soldiers use lasers for training and weapons-fire control (Table 8-3). The Multiple Integrated Laser Engagement System (MILES) is a Class 3B training laser that is used to simulate the firing of conventional weapons. It is considered to be eye hazardous to a distance of 7 m; beyond 7 m, the energy diminishes sufficiently to make it eye safe (unless it is viewed through an unfiltered telescopic sight to a distance of 300 m). On the battlefield, two types of fire-control lasers are currently being used with modern weapons systems: *laser rangefinders*, which measure the distance to the target, and *laser target designators*, which irradiate a target with an optical signature that can be used as a homing beacon for laser-guided munitions.[44]

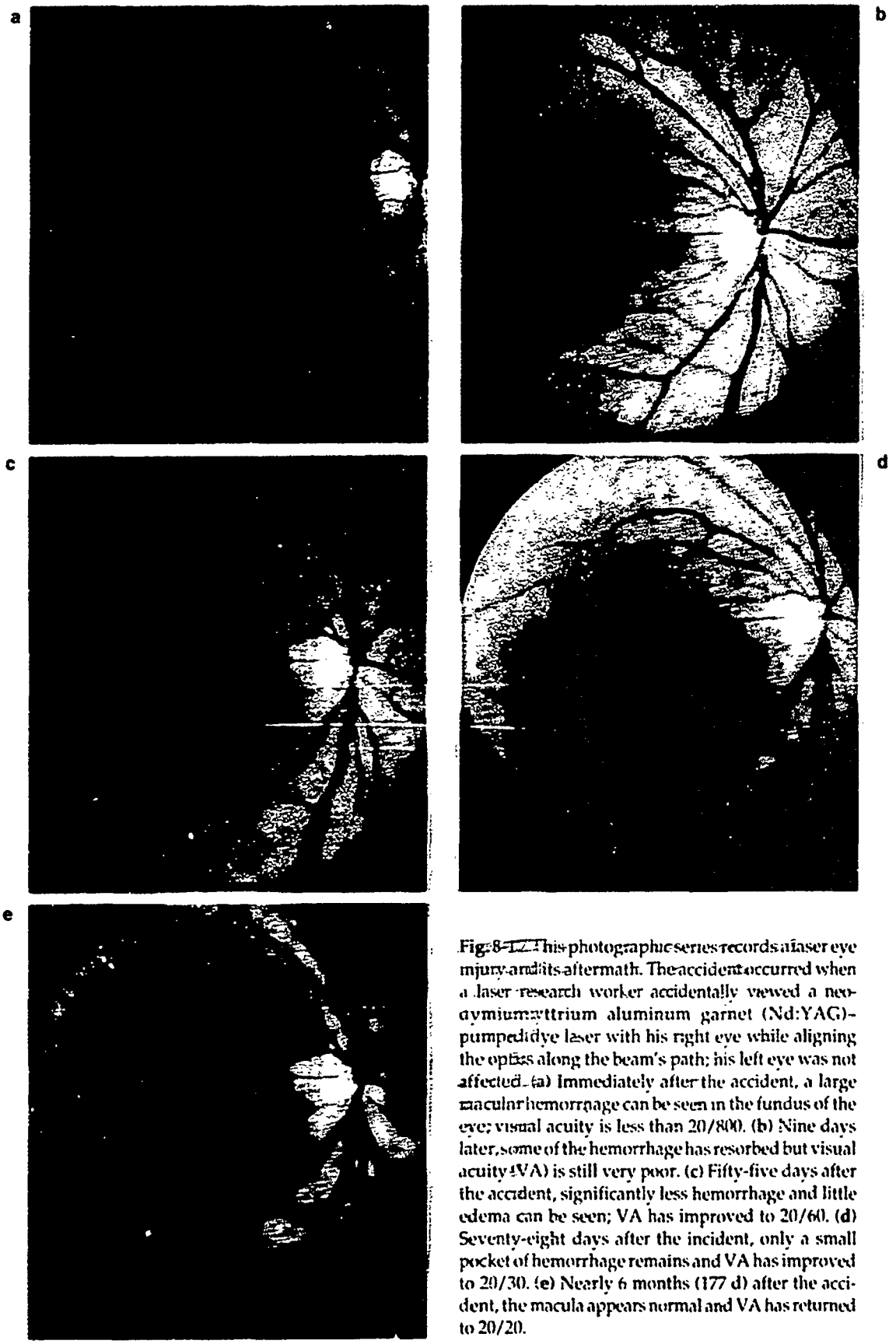

Fig. 8-12. This photographic series records a laser eye injury and its aftermath. The accident occurred when a laser research worker accidentally viewed a neodymium:yttrium aluminum garnet (Nd:YAG)-pumped dye laser with his right eye while aligning the optics along the beam's path; his left eye was not affected. (a) Immediately after the accident, a large macular hemorrhage can be seen in the fundus of the eye; visual acuity is less than 20/800. (b) Nine days later, some of the hemorrhage has resorbed but visual acuity (VA) is still very poor. (c) Fifty-five days after the accident, significantly less hemorrhage and little edema can be seen; VA has improved to 20/60. (d) Seventy-eight days after the incident, only a small pocket of hemorrhage remains and VA has improved to 20/30. (e) Nearly 6 months (177 d) after the accident, the macula appears normal and VA has returned to 20/20.

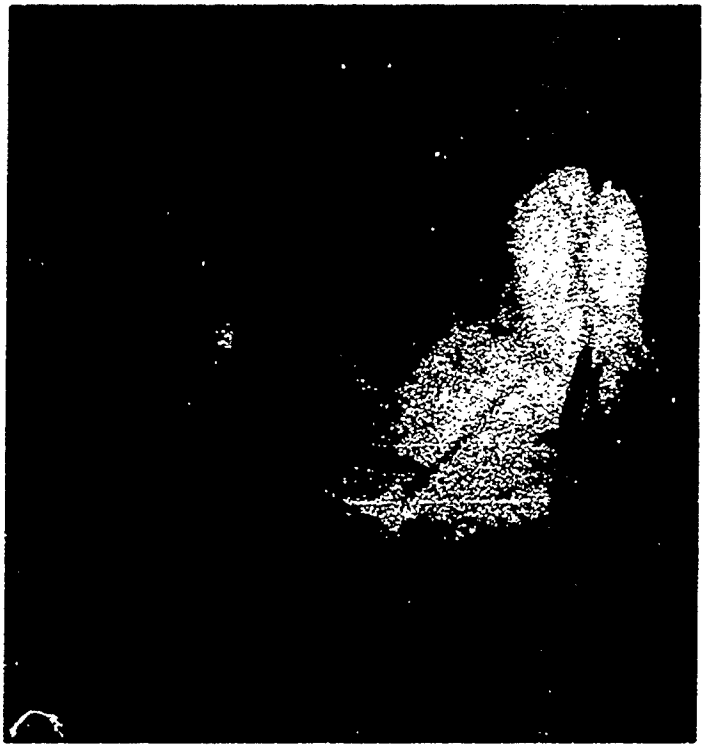

Fig. 8-18. Lasers, which destroy retinal tissue through to the vascular choroid, can cause hemorrhaging into the vitreous. Although the hemorrhage shown here will eventually be resorbed (albeit slowly), laser injuries involving the central macula usually do not have a good prognosis.

TABLE 8-3

TYPES AND CLASSES OF LASERS

Laser Type	Wavelength (nm)	Class*	Application
Gallium-Arsenide	905.0 (near IR)	1–3	Optical fiber communication, direct-fire simulators, and training devices Example: MILES
Helium-Neon	632.8 (red)	2	Distance measurements, bar code readers, patient alignment in radiology Example: M55 gunnery trainer
Ruby	694.3 (red)	4	Tank rangefinders Example: AN/VVG2, M40A3 Tank
Argon	510.0 (blue/green)	4	Entertainment, holography, printing plate manufacture, photocoagulation for diabetics
Nd:YAG†	1,064.0 (invisible, near IR)	4	Distance measurement and target marking Example: AN/TVQ2 ground laser designators and tank rangefinding
CO_2	10,600.0 (far IR)	4	Cutting welding, engraving, high-speed product labeling, and fire control Example: no CO_2 laser system is fielded currently

*Class designation depends on power output for any particular application

†Neodymium:yttrium, aluminum, garnet

Reprinted from Sliney DH, Kotulak JC. Hazards of fielded lasers. *Medical Bulletin of the US Army.* 1988;82:14–16.

Classification and Surveillance of Laser Workers. Appendix E of ANSI Standard 136.1-1986 provides guidance for the medical surveillance of the classifications of laser workers.[45] An *incidental worker* is a person whose work makes it possible, but unlikely, that he or she will be exposed to laser energy that is sufficient to damage the eye. Incidental workers include operators of fielded laser equipment, individuals who oversee laser use on approved laser ranges, and soldiers who participate in force-on-force laser-training exercises. A *laser worker* is a person who routinely works in a laser environment and therefore has a higher risk of accidental overexposure. Laser workers include those who regularly perform laser research, development, testing, and evaluation, and workers who perform routine laser maintenance.

The type of medical surveillance that is done on employees depends on the classification of laser work. According to the ANSI standard, incidental workers require only preplacement vision examinations using a screening protocol (distance and nearpoint visual acuity measurements). On the other hand, laser workers require a more extensive preplacement examination, which includes a medical history, visual acuity measurement, and selected examination protocols, depending on the type of laser that they will use. Periodic and termination examinations are advised but are not required.

Current U.S. Army policies concerning the medical surveillance of laser workers are similar to the ANSI standard. Incidental workers require preplacement and termination examinations utilizing a screening protocol (distance and nearpoint visual acuity measurements). Laser workers must also have preplacement and termination examinations, utilizing a different screening protocol (a medical history; distance and nearpoint visual acuity measurements; and an Amsler grid test, which tests macular function).

Laser Overexposure Incidents. Any DA employee—civilian or military—who is known or suspected to have been overexposed to laser radiation must be examined by an optometrist or ophthalmologist within 24 hours of being injured. In addition, the USAEHA must be notified by telephone as soon as possible after the incident, to initiate the investigatory process.[46] In most instances, patients suspected of having sustained a laser injury are evacuated to the Presidio of San Francisco, California, where they are evaluated at the Division of Ocular Hazards at Letterman Army Institute of Research (LAIR). Civilian employees who do not wish to be flown to LAIR can be followed by the nearest military ophthalmologist or their own civilian ophthalmologist at government expense. (With the Presidio scheduled to be closed, the Division of Ocular Hazards will be transferred to Armstrong Laboratory, Brooks Air Force Base, San Antonio, Texas, in 1993.) The laser equipment that was used during incident should be secured so the Laser Branch at USAEHA can do a full technical evaluation to determine if the injury was caused by equipment malfunction or operator error.

Clinicians who examine workers who may have been overexposed to a laser beam should avoid making a hasty diagnosis of laser injury until the alleged incident has been investigated and verified. Even if the ocular signs and symptoms are consistent with overexposure to a laser, the clinician should consider two additional factors before making a diagnosis: the circumstances of the exposure and any preexisting ocular lesions. The treatment for patients with confirmed laser injuries is usually limited to observation.

Thermal Radiation

Because the eyes are protected by the autonomic blink reflex, thermal injuries to the eyes tend to be limited to the eyelids, depending on the duration of the exposure. Most thermal burns are caused by boiling liquids, molten metal, flame, gasoline, explosions, steam, and hot tar. Glass and iron cause the most severe thermal injuries to the eyes and adnexa because their melting points are high: 1,200°C. Lead, tin, and zinc melt below 1,000°C and cause slightly less damage (Figure 8-19).[29]

Because lid edema and pain may make an objective examination difficult, applying topical anesthetic drops such as proparacaine or benoxinate may be necessary. Ocular burns should be treated with topical antibiotic ointment and sterile dressings; topical steroids may be necessary to decrease subsequent scarring between the eyelids and the globe.

Radio-Frequency Radiation

Radio-frequency (RF, 30 cm–1,000 m) and microwave (1 mm–30 cm) radiation have been implicated in the development of lens opacities. Cataractogenesis has been observed in rabbits when acute exposures of RF radiation exceeded 100 mW/cm^2 for more than 1 hour. Human exposure to 100 mW/cm^2 would immediately cause a *threshold response* (the individual would experience either segmental- or whole-body heating; he or she would immediately move away from the RF radiation beam and would know that a significant exposure had occurred). Currently, there is no evidence that chronic exposure to microwave fields of 10 mW/cm^2 or less can induce cataracts.[47] In 1977, a survey of 800 workers in the microwave industry at three army installations found no evidence of work-

Fig. 8-19. Thermal injury to an eye exposed to molten lead. This burn could have been prevented had the worker been wearing industrial safety glasses.

related lens opacities.[45]

From 1986 to 1991, army policy required that either a screening or diagnostic protocol be done on all workers who might be exposed to RF radiation. The army has since eliminated its ocular-surveillance program because (*a*) RF exposure is a threshold effect, (*b*) there is no method for measuring cumulative dose over time, (*c*) the program has not been cost effective, and (*d*) it will not prevent RF exposures from occurring.

Individuals who experience an acute overexposure to microwave radiation (including whole-, segmental-, or localized-body warming) should be examined by a qualified physician within 24 hours of the injury. The examination should include a slitlamp examination of the eyes by an optometrist or ophthalmologist. For all DA civilians or military personnel, the physician must report the incident to the USAEHA.[46]

Electrical Current

The electrical field associated with power transmission is considered to be part of the EM spectrum and has a wavelength of 1,000 m. Accidents involving electrical current have been known to produce cataracts of the lens cortex and anterior capsule. As with other radiation-induced anomalies, the latency for cataract development can vary from months to years. Following an electrical-shock injury, periodic slitlamp examinations through dilated pupils should be performed to identify early cataract formation.

Ionizing Radiation

The cornea, lens, uvea, retina, and optic nerve may suffer injury from excessive exposure to ionizing radiation from cyclotron exposure or during beta irradiation of the periorbital area to treat malignancy. Radiation keratitis ranges from a superficial punctate epithelial staining to sloughing of large areas of epithelium, stromal edema with interstitial keratitis, and aseptic necrosis. X radiation and other ionizing energy sources are well-established causes of posterior subcapsular lens opacities; at high radiation doses these opacities can occur in a matter of months, while years may elapse before doses that are closer to the threshold level for injury cause cataractogenesis. Four hundred to 2,000 cGy of exposure are required for cataractogenesis.[41] Younger patients are more vulnerable to cataract formation than are older patients who receive the same relative dose or exposure. Intraretinal hemorrhages, papilledema, and central retinal-vein thrombosis are also possible but rarely occur.

SELECTING EYE PROTECTION

Wearing PPE, including eye protection such as industrial safety glasses, goggles, face shields, and welding helmets, cannot completely eliminate the possibility of ocular injury under all circumstances. Employers are required first to evaluate all eye-hazardous operations and then to attempt to reduce or eliminate each hazard through engineering or administrative controls, or both. If the elimination of eye-hazardous operations is not feasible by these plant changes, then the employer is required by law to provide PPE for all workers in proximity to the hazard. Protective eyewear is also mandatory for supervisors and others who must enter the hazardous environment, even if they are not physically involved in the operation.[10]

In some instances, other types of PPE in addition to eye devices must be worn, and this may influence the selection of eye and face protectors. For example, if respiratory protective equipment, a hardhat, or both are worn, safety glasses must be compatible with them. When management or safety specialists select PPE, they often make the mistake of providing only one type of eye protector. This simplistic approach fails to meet the variety of eye hazards present throughout the workplace. For example, industrial safety spectacles would offer inadequate protection to a worker in a battery shop: in the event of a chemical splash, the impact-resistant safety glasses would provide insufficient protection against battery acid. Furthermore, workers should not be allowed to wear their dress safety glasses as a substitute for approved industrial safety glasses; dress safety glasses are inferior to industrial safety glasses in many respects (Table 8-4).

Industrial eye protection must conform to OSHA regulations, which originally adopted the provisions of ANSI Standard Z87.1-1968. The 1968 standard was a design-oriented standard that dictated how industrial-eyewear manufacturers must design safety glasses.

TABLE 8-4

COMPARISON OF ANSI Z80.1 AND Z87.1 STANDARDS

Criteria	Z80.1-1979 ("Dress" Safety)	Z87.1-1989 ("Industrial" Safety)
Removable lenses:		
Minimum thickness:		
Nonprescription lenses	2.00 mm*	3.00 mm†
Prescription lenses	2.00 mm center thickness*	3.00 mm center thickness
Plus lenses > 3.00 D	1.00 mm edge thickness*	2.50 mm edge thickness
Drop ball impact test:	0.63 in. (15.9 mm) diameter steel ball, NLT‡ 15 g, dropped from 50 in.	1.00 in. diameter steel ball, dropped from 50 in.
Penetration test (plastic only)	N/A	44.20 g projectile, dropped from 50 in.
Frames:		
High-mass impact test	N/A	500.00 g pointed projectile, dropped from 130 cm (51.2 in.)
High-velocity impact test	N/A	0.25 in. diameter steel ball, traveling 150 fps
Nonremovable lenses:		
Minimum thickness	N/A	3.00 mm§
High-mass impact test	N/A	500.00 g pointed projectile, dropped from 130 cm (51.2 in.)
High-velocity impact test	N/A	0.25 in. diameter steel ball, traveling 150 fps
Penetration test (plastic only)	N/A	44.20 g projectile, dropped from 50 in.
Markings	N/A	Frames: Manufacturer's trademark and Z87 logo Lenses: Manufacturer's trademark

*Applies to air-tempered glass lenses only; all other lens materials must meet impact testing
†May be thinner (but not < 2.0 mm) if high-velocity impact test (0.25 in. diameter steel ball, traveling 150 fps) is met
‡NLT: not less than
§Plastic lenses may be thinner (but not < 2.0 mm) if all impact testing requirements are met

With the advent of newer materials like polycarbonate plastic for ophthalmic lenses, the new ANSI Standard Z87.1-1989 has adopted a more performance-oriented standard that encourages innovation as long as the eyewear meets rigid industrial safety performance tests.

Wearing compliance is usually the most difficult aspect of any vision conservation program. Compliance is often poor among workers who do not wear prescription eyeglasses. Employers who purchase more-stylish, better-fitting frames will have better rates of compliance among their employees. They will find that better compliance will decrease the incidence of eye injuries, which, in turn, will lower injury-compensation claims. Adequate supervision is also essential to ensure that eye protection is not only worn, but also is worn correctly.

Ballistic, Mechanical, and Impact Protection

ANSI Standard Z87.1 describes two basic types of impact industrial eye protection: goggles and spectacles (eyeglasses) (Figures 8-20, 8-21, and 8-22). Both are considered to be *primary* eye protectors (they can be worn without additional protection). Goggles are subdivided into two types according to their use: impact (for mechanical and ballistic hazards) and splash (for chemical hazards). They are also subdivided into two types according to their wearers: the cup type is for workers who do not require prescription lenses, whereas the cover type is designed to fit over dress or industrial prescription eyewear. When goggles are selected, ventilation to prevent fogging of the lenses should be evaluated. Impact goggles have multiple holes across the top for direct ventilation of warm, moist air. Dust and splash goggles should have baffles (indirect venting), which permit air to circulate but exclude dust and liquids.

Industrial safety eyeglasses are available with plano or prescription lenses. Frames for both types must be marked with the Z87 logo, which identifies them as an approved industrial safety frame. In addition, industrial safety lenses must be identified with the manufacturer's monogram or logo.[11]

Cost

In many instances, wearing compliance is directly related to the cost of industrial eyewear. Workers are more apt to wear high-quality eyewear than inexpensive, ill-fitting eyewear. Emmetropic workers (those who do not wear prescription eyeglasses) often feel uncomfortable wearing plano industrial safety glasses or goggles for long periods of time. Supplying these workers with inexpensive, ill-fitting safety glasses or goggles will severely degrade their wearing compliance. Likewise, ametropic workers (those who must wear prescription eyeglasses) will function more efficiently with prescription industrial safety glasses than if they are required to wear goggles over their dress safety eyewear. The USAEHA actively discourages the wearing of goggles or plano spectacles over dress safety eyewear because visual acuity and job performance can be degraded by multiple optical surfaces; this in turn decreases wearing compliance. A supervisor should balance the financial costs of providing safety eyewear to employees against the benefits of improved vision conservation and worker performance.

Frames. Frame selection is another major consideration when choosing eye protection. To encourage maximum wearing compliance, employers should stock (or allow their employees to order) safety frames in a variety of styles, sizes, colors, and materials, including metal and plastic frames. Because many eye injuries are caused by particles that hit the eye from the side, the new ANSI Standard Z87.1-1989 strongly recommends that side shields be ordered with all safety frames unless there is a specific reason to preclude them (such as restricting peripheral vision).

Lenses. In the current legal climate, all industrial safety spectacles should be ordered with polycarbonate lenses. This recommendation stems from a lawsuit in which an autoworker (wearing ANSI Z87.1-approved industrial safety glasses) was struck by a foreign object that shattered the glass industrial safety lenses. The court initially ruled that, because polycarbonate lenses were available and would have provided a greater degree of eye protection, both the employer and the supplier of the industrial safety glasses would be liable for the worker's injuries. The case was subsequently overturned due to a legal technicality; however, the issue of providing state-of-the-art materials still applies.

Polycarbonate lenses are approximately 15-fold stronger than thermally-tempered glass lenses and 5- to 6-fold stronger than regular CR-39 (the 39th Columbia resin formula, an ophthalmic-grade allyl resin) plastic lenses (Table 8-5). Polycarbonate lenses have two disadvantages when compared with either glass or CR-39 lenses: they are more difficult to manufacture and are slightly more expensive. Despite the safety advantages that are associated with polycarbonate lenses, however, many workers still prefer glass lenses because they are more resistant to scratches. In addition, polycarbonate lenses should not be prescribed for

Fig. 8-29. Employees working in eye-hazardous areas must wear American National Standards Institute (ANSI) Z87.1-approved industrial safety glasses. (a) The young worker wears plano industrial safety glasses (with fixed side shields) while using a large industrial band saw; note the prominent yellow caution sign, reminding all employees to wear eye protection. (b) The older worker wears prescription industrial safety glasses, with removable side shields, in a fashionable metal frame. A choice of frame sizes and styles will usually improve wearing compliance.

Fig. 8-21. Automotive repair has the second-highest rate of eye injuries among workers. Rust often falls into the eyes of mechanics who work under vehicles, therefore, it is imperative that they wear industrial eye protection.

Fig. 8-22. The soldier on the left is wearing industrial safety goggles over the standard military-issue eyeglasses. Currently, the military optical fabrication laboratories do not manufacture American National Standards Institute (ANSI) Z87.1-approved industrial safety eyewear, however, soldiers can (and should) obtain civilian-style industrial safety glasses in the same manner as their civilian counterparts. The soldier on the right is wearing a pair of Visi-Specs (visitor's spectacles); these should be issued to temporary workers or visitors who must enter eye-hazardous areas. Caution should be exercised when issuing Visi-Specs because some are not ANSI Z87.1-approved.

TABLE 8-5

IMPACT DATA FOR OPHTHALMIC LENS MATERIALS

Lens Material (3-mm thickness)	Projectile Size					
	0.125 in		0.250 in		1.000 in	
	ft-lb	mph	ft-lb	mph	ft-lb	mph
Glass, heat treated	0.040	65	0.127	40	1.450	17
Plastic (CR-39)	0.370	196	0.950	109	1.09	15
Polycarbonate	4.24*	655*	12.6*	399*	> 9	> 42

*Impact data are for 1.9-mm polycarbonate

Source: Compiled by Davis JK, Gentex Optics, Inc., Dudley, Massachusetts, from research data from (1) Wigglesworth EC. A comparative assessment of eye protective devices and a proposed system of acceptance and grading. *Am J Optom Arch Am Acad Optom.* 1972;49:287–304; and (2) LeMarre DA. *Development of Criteria and Test Methods for Eye and Face Protective Devices.* Cincinnati, Oh: National Institute of Occupational Safety and Health, August 1977. NIOSH Research Project 210-75-0058. Reprinted with permission from Gentex Optics.

individuals who require corrections exceeding ±4.00 diopters; excessive chromatic aberration (colors that outline objects) may decrease visual acuity and patient acceptance.

The prescribing optometrist or ophthalmologist must decide whether the worker needs single-vision (monofocal), bifocal, trifocal, or some other type of occupation-specific lenses. Single-vision lenses are usually recommended for nonpresbyopic individuals or presbyopic individuals who work at a single working distance. Bifocal or trifocal lenses should be prescribed for presbyopic individuals whose job is performed at two or more distances. Some occupations, such as carpentry, require special double-segment lenses for working both overhead and at the normal reading position.

Photochromic and tinted lenses provoke controversy; some suggest that, in certain occupations, they may actually contribute to on-the-job injuries. One problem is that photochromic lenses are made of glass, which is less impact-resistant than plastic. Another problem is that tinted or photochromic lenses cannot adapt quickly enough to rapid changes in illumination. For example, if a forklift operator wearing photochromic lenses drives into a warehouse from the outside (from a bright to a dark environment), the lenses can increase the risk of serious injury: the time that is required for the photochromic lenses to change from dark to light may put the operator at risk of injuring himself or others if he cannot see properly as he enters the dark warehouse. However, while tints and photochromic lenses are controversial, there is little doubt that they improve wearing compliance: workers perceive sunglasses and photochromic lenses as a job benefit or perquisite.

Face Shields

Workers who require face and neck protection in addition to eye protection should use face shields (Figure 8-23). They are often worn by workers in metal manufacturing operations (such as grinding or machining of parts) where facial lacerations can be painful and disfiguring. They may also be worn in painting operations or areas where chemical splashes are likely. However, according to ANSI Standard Z87.1, face shields are considered *secondary* eye protection (they must be worn over a primary eye-protective device like industrial safety glasses or goggles). ANSI Standard Z87.1 requires that the manufacturer's trademark and the Z87 logo be visible, just as they must be on safety glasses.[11]

Chemical Protection

Chemical goggles (also known as splash goggles), face shields, or both should be worn wherever there is a risk of a chemical splash. Splash goggles are primary eye protectors, offering the same degree of impact protection as do impact goggles; however, splash goggles differ from impact goggles in that they have baffled or indirect ventilation that keeps liquids and chemicals out (Figure 8-24). Impact goggles, with their direct venting system, should *never* be used in chemically hazardous areas. The following guidelines have been established to protect the eyes from chemical splash:

Fig. 8-23. Face shields (secondary protectors) are designed to protect the entire face. They must be worn over industrial safety glasses, as pictured here, or chemical goggles (American National Standards Institute [ANSI] Z87.1–approved primary eye protectors).

Fig. 8-24. These soldiers are both wearing American National Standards Institute (ANSI) Z87.1–approved goggles. The soldier on the left is wearing chemical goggles with indirect venting (which prevents the direct transmission of fluids), the soldier on the right is wearing impact goggles. Impact goggles should never be worn if there is a risk of chemical splash, but chemical goggles do offer ballistic protection and they may be worn in ballistically and mechanically hazardous areas.

Fig. 8-25. For maximum eye, face, and neck protection against chemical splash and hazards, workers should wear face shields over their chemical goggles.

- If the risk of chemical splash is minimal to moderate, and if there is some risk of ballistic or mechanical injury, then chemical goggles may be worn alone.
- If the risk of chemical splash is moderate to high, and if there is increased risk of ballistic or mechanical injury, then the worker must wear both a face shield and chemical goggles for maximum protection (Figure 8-25).

Radiant Energy Protection

NIOSH and other agencies have studied the effects of UV and IR radiation and have issued guidelines on the maximum permissible exposure levels (PELs) and the use of filtering devices (Table 8-6). Filtering lenses are designed to reduce the intensity of specific wavelengths of optical radiation; the degree of reduction depends on the density of the filter. Filtering lenses, however, should not be confused with tinted lenses. Tinted lenses (such as those in sunglasses) reduce the overall intensity of the visible light and are usually not wavelength specific.

Ultraviolet Radiation

Welders tend to have a high degree of wearing compliance. One reason for this may be the image of specialized training that the welding helmet, like the hardhat, conveys. A second reason is that many (if not most) welders have had at least one overexposure to UV radiation and have experienced the painful effects of a photokeratitis. Despite this, however, some experienced welders continue to get overexposures because they often strike the welding arc before they bring the welding helmet into position.

In addition to protecting themselves against UV radiation exposure, welders must also wear ballistic eye protection to preclude any secondary injuries from stray foreign bodies (Figure 8-26). A 1985 study of the Workers' Compensation Board of Alberta, Canada, found that 21% of all reported eye injuries involved welders.

TABLE 8-6

GUIDE FOR WELDING SHADE NUMBER

Operation	Electrode Size (mm)	Arc Current (A)	Minimum Shade No.	Suggested Shade No.
Shielded metal arc welding	< 2.5	< 60	7	—
	2.5–4.0	60–160	8	10
	4.0–6.4	160–250	10	12
	> 6 4	250–550	11	14
Gas metal arc welding and flux cored arc welding		< 60	7	—
		60–160	10	11
		160–250	10	12
		250–500	10	14
Gas tungsten arc welding		< 50	8	10
		50–150	8	12
		150–500	10	14
Air carbon arc welding				
(Light)		< 500	10	12
(Heavy)		500–1,000	11	14
Plasma arc welding		< 20	6	6–8
		20–100	8	10
		100–400	10	12
		400–800	11	14
Plasma arc cutting				
(Light)		< 300	8	9
(Medium)		300–400	9	12
(Heavy		400–800	10	14
Torch brazing		—	—	3 or 4
Torch soldering		—	—	2
Carbon arc welding		—	—	14
	Plate thickness (in)	Plate thickness (mm)		
Gas welding				
Light	< ⅛	< 3.2		4 or 6
Medium	⅛–½	3.2–12.7		5 or 6
Heavy	> ½	> 12.7		6 or 8
Oxygen cutting				
Light	< 1	< 25		3 or 4
Medium	1–6	25–150		4 or 5
Heavy	> 6	> 150		5 or 6

Reprinted with permission of the American Welding Society. Miami, Fla, 1992.

Fig. 8-26. Statistically, welders suffer the greatest number of radiant-energy eye injuries. In addition to wearing leather gloves, a leather apron, and hearing protection, welders must also wear American National Standards Institute (ANSI) Z87.1–approved industrial safety glasses (to preclude ballistic or mechanical eye injuries) in addition to the standard welder's helmet. The density of the filtering lens in the welding helmet depends on the type of welding torch used.

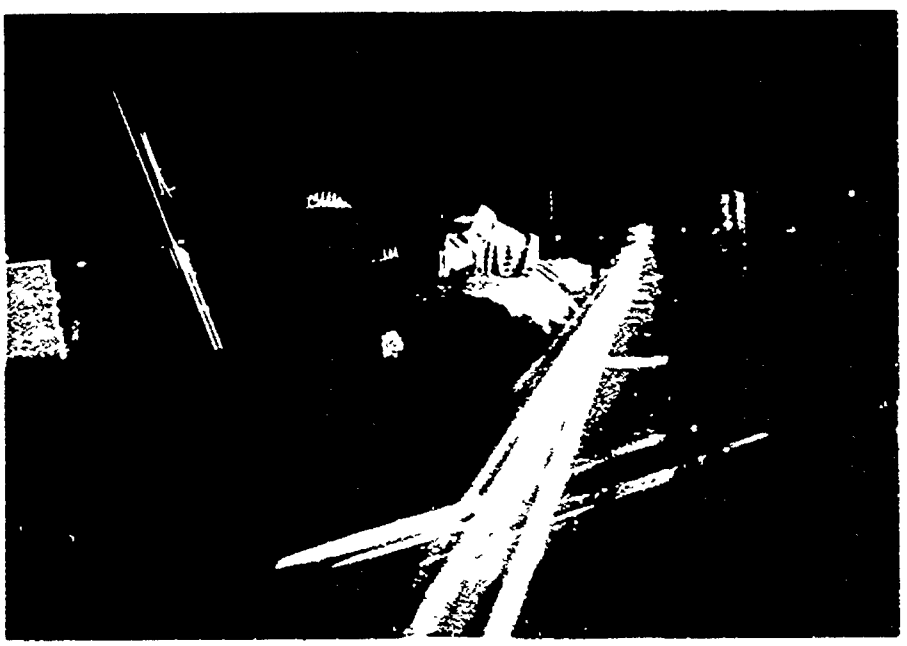

Fig. 8-27. Laser workers, such as laser maintenance personnel and laser researchers, must wear appropriate eye protection, consisting of wavelength-specific filtering lenses, whenever they work with or are near a laser. Laser goggles are preferred over laser spectacles; they prevent injuries to the retina from the side. Reprinted from the cover of *Occupational Health & Safety* July 1990. © Photograph by Joe Griffin.

Nearly 75% of these injuries were caused by cold metal foreign particles, and occurred during nonwelding tasks such as chipping, grinding, or buffing.[49]

Infrared Radiation

IR-absorbing lenses vary according to the degree of absorption required. Unfortunately, good IR-absorptive lenses also diminish the transmission of visible light. Cobalt-blue filters are issued to workers who determine the temperature of the melt in steel manufacturing. Didymium lenses eliminate much yellow sodium flare, which is a common hazard in the electronics and glass industries.

Lasers

No single type of light-filtering device offers protection against all laser wavelengths. Currently, there are two types of filtering technology used to protect soldiers and workers against lasers: *dye absorbers* and *reflectors*. Dye absorber devices must be of sufficient filtering density for a particular wavelength of laser emission to provide appropriate vision protection (Figure 8-27). For example, to reduce a Class 4 laser from a 10-W output to a safe level of 1 mW, the filtering goggles (or spectacles) must be optical density 4 (able to reduce the radiant-energy level by a factor of 10^4) for that particular wavelength.

Reflective technology includes *dielectric stacks* and *holograms*. While these two processes are both considered to be reflective technology, the application of the technology differs slightly. For example, dielectric stacks reflect a given wavelength by layering 6 to 12 layers of two different dielectric (insulator) materials that are only as thick as one-half the wavelength to be reflected. Examples of dielectric materials are silicone dioxide and magnesium fluoride. If one laser wavelength (eg, helium-neon [He-Ne] at 632.8 nm) were to be reflected, then 12 layers of alternating dielectric materials, each layer being 316.4 nm thick, would be applied to a lens. If a second laser wavelength (eg,

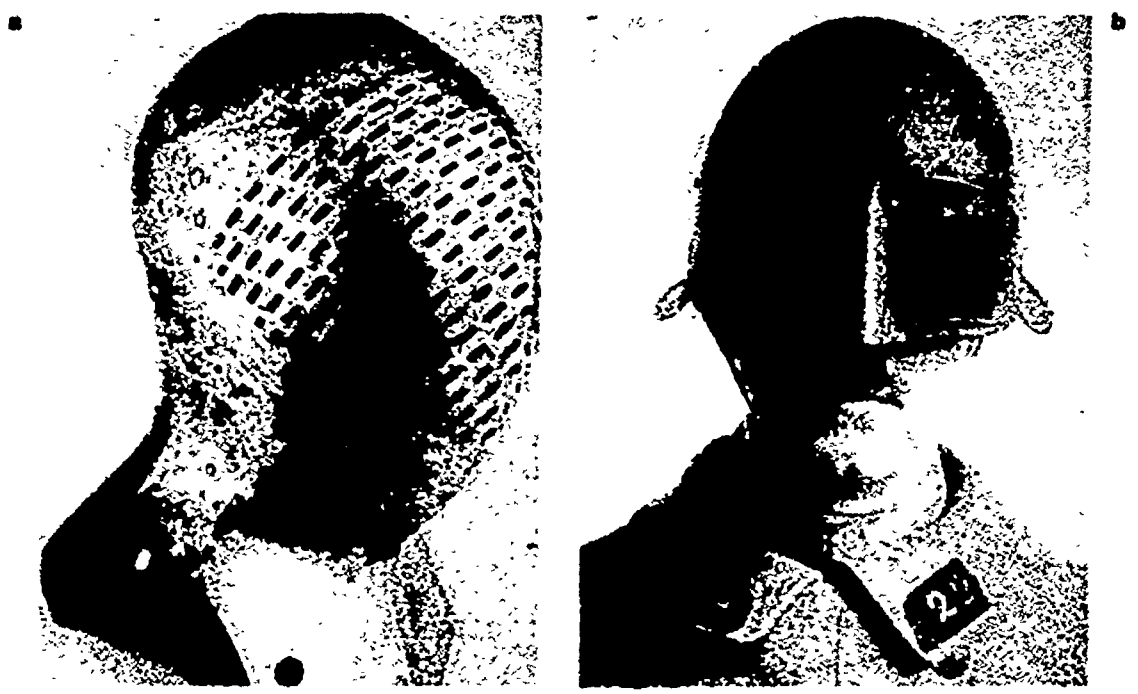

Fig. 8-28. (a) The Helm of 1514, never widely accepted, could be used only when the helmet rested on or was attached to the shoulders of the armored soldier (p100). (b) An experimental 1918 French helmet fitted with a Polack visor. The visor is shown dropped into place, but was designed to be worn up over the front of the helmet when not needed. The thin laminae and their vertical supports were mounted edgewise to interfere as little as possible with the soldiers' vision. However, as a defense against a missile or ball traveling at 600 fps, "the Polack visor is held to be worse than useless; it is penetrated, shattered, and an even more serious wound would be caused by the ragged ball and the inbent and broken ends of the visor's laminae" (p96).

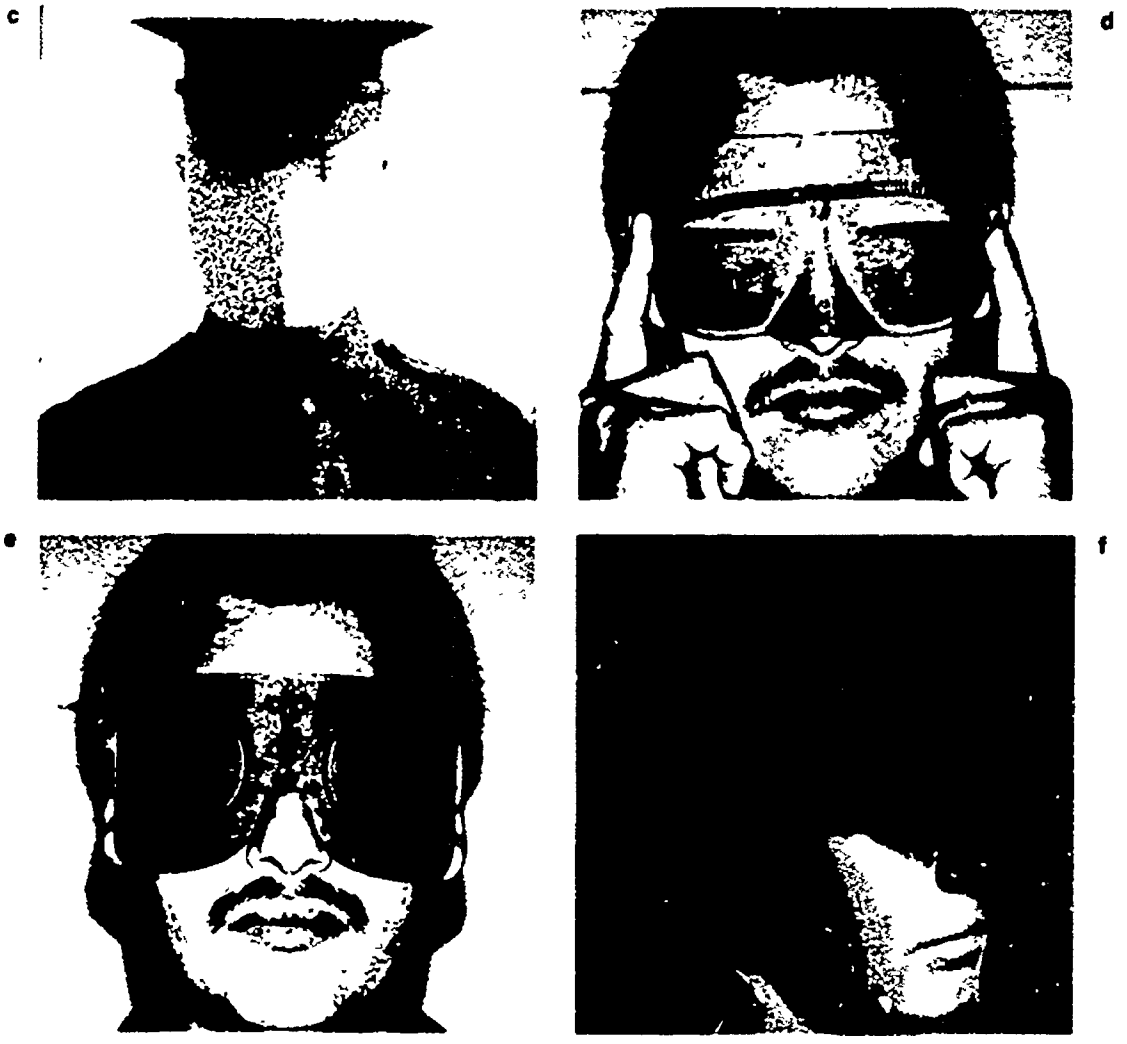

Fig. 8-28. (c) This British face shield (designed 1915–1916) consisted of a steel plate pierced with vertical and horizontal slits in front of each eye. It was designed to be worn fitted under the soldier's cap, but never progressed beyond the experimental stage (pp131–132). (d) U.S. Army Colonel W. Holland Wilmer suggested this 1918 visor design. It was based on the single-slot eye shield used by Native Americans in the northwest to protect against snow blindness. Made of soft steel, the visor fit snugly against the brow and cheeks by means of a sponge-rubber cushion, was attached to the soldier's helmet by a spring, and was designed to fit both British and American helmets. The visor permitted a wide range of vision. Note the apertures beneath the slits, they were positioned to allow the wearer a stereoscopic field of the ground immediately in front. These visors were disapproved because they were not readily kept in position (p236). (This photograph courtesy of Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.) (e) These 1917 British "splinter goggles," made of steel and weighing about 5.5 ounces, were said to allow surprisingly clear and extended vision through their narrow (0.2–0.5-in) slots. However, although these privately manufactured goggles were sold to allied soldiers, they never gained general acceptance (p233). (Photograph courtesy of Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC). (f) The chain-mail veil was the only eye defense produced by the British in large numbers (1916–1917). The visor, made of closely woven links, attached to a metal rod that passed under the brim of the helmet. Visors of this type were sent to the front during World War I, but the soldiers "found them to be annoying and soon cast them off....In actual use they produce dizziness, for the links of the visor change position in front of the wearer's eyes, following every movement of the helmet" (p133). The designers of this eye armor anticipated that it would prevent 50% of battlefield eye injuries. (This photograph courtesy of Colonel Francis G. La Piana, Walter Reed Army Medical Center, Washington, DC.) Source: (figure legends a–f, photographs a–c, e): Dean, B. *Helmets and Body Armor in Modern Warfare*. Tuckahoe, NY: Carl J. Pugliese, 1977.

neodymium:yttrium aluminum garnet [Nd:YAG] at 1,064.0 nm) were to be reflected, then an additional 12 layers of alternating dielectric materials, each layer being 532.0 nm thick, would be applied over the first 12 layers of dielectric materials. Dielectric stacks have this advantage over filtering technology: they do not diminish broad bands of usable light as dye absorbers do. The disadvantages of dielectric stacks are twofold: the manufacturing process is still very expensive, and the degree of eye protection diminishes rapidly if the laser beam were to hit the dielectric stack *off-axis* (not perpendicular to the dielectric stack).

The underlying principle for hologram reflectors is similar to that for dielectric stacks. The difference is that the hologram utilizes a photographic film 20 µm thick, which contains hundreds of layers of high- and low-reflective regions. The advantages and disadvantages of hologram reflectors are the same as those for dielectric stacks. Perhaps the most significant disadvantage of reflective technology is that the reflections create a large battlefield signature, easily seen by enemy snipers and gunners. Future laser protection may combine both filtering and reflective technologies to protect against multiple laser wavelengths.

Eye protection for carbon dioxide lasers, which radiate intense energy and can cause thermal burns to human tissue including the eyes and skin, consists of regular industrial safety glasses (or a face shield) made with CR-39 plastic or polycarbonate plastic. Consequently, no tint or filtering device is needed. Glass lenses are not recommended for use in eye protectors because the energy intensity from the carbon dioxide laser beam could cause them to shatter.

Militarily Unique Eye Protection

Armies have always sought eye armor that would protect the vision of their fighting forces. However, soldiers have always resisted wearing such PPE because it restricted their peripheral vision, or was too heavy or cumbersome (Figure 8-28). The U.S. Army entered World War II with no eye protection for its soldiers. The sun, wind, and dust (SWD) goggle, however, was developed in 1942 for use in the African desert.

Efforts to develop eye armor continued after World War II. In approximately 1953, John Fair, an ophthalmologist who served in the Korean War, advocated (to no avail) that both ametropes and emmetropes wear a spectacle made of case-hardened glass refractive material, with cable temples and side shields. Later, an ophthalmology consultant to The Surgeon General anticipated the threat from battlefield lasers and advocated that AMEDD and the Army Materiel Command (AMC) work together to develop laser eye protection.[50] Developing eye protection remains a challenge today, because for the emmetrope it is considered to be body armor (a nonmedical item), whereas for the ametrope it is a refractive device (a medical item).

Despite development efforts during the 1950s and early 1960s, soldiers went to Vietnam with essentially no eye protection. Soldiers who were issued the SWD goggle usually wore them on top of their helmets (otherwise known as the Rommel position). In 1962, a researcher at the U.S. Army Natick Research, Development, and Engineering Center developed the technique to injection-mold polycarbonate plastic, which in turn led to the 2-mm-thick polycarbonate face shield that was used in the army aviator helmet; many pilots' eyes were saved during the Vietnam War because they wore this helmet while flying. Attaching the polycarbonate face shield to the infantryman's helmet was another idea that was proposed. Because the army could barely enforce the requirement that infantrymen wear a helmet, however, enforcing a requirement to wear eye protection was thought to be next to impossible.[50]

Because standard military spectacles do not provide adequate protection against ballistic or laser hazards, AMEDD developed the BLPS to provide more effective eye protection for soldiers during training and combat. The BLPS is a wrap-around polycarbonate spectacle with *spherical* lenses (designed to curve in two meridians), and provides protection against both ballistic projectiles and laser energy. It can be worn in garrison, while playing sports, while working around the house, or even while mowing the lawn. The kit includes a clear pair for everyday use; a tinted pair for sunny environments; and a green-tinted frontsert, which clips to the front of the BLPS, to protect the wearer against low-energy lasers. Ametropic soldiers are provided with a prescription backsert, which mounts behind the polycarbonate eyewear (Figure 8-29).

The SPECS system is similar to the BLPS in that it is made of polycarbonate plastic. However, it differs from BLPS in that the lenses are *cylindrical* (designed to curve in one meridian) to facilitate the application of dye absorbers, holograms, or dielectric stacks for protecting the soldier against laser hazards. Currently, there is no means for providing prescription lenses for ametropic soldiers.

Contact Lenses in Industry

Wearing contact lenses in eye-hazardous occupations has always been very controversial. Approximately 12 to 15 million Americans wear contact lenses; an additional 2 million new wearers are fitted each year.[51] Some people are absolutely required to wear

Fig. 8-29. From the left, the three soldiers are wearing clear Ballistic/Laser Protective Spectacles (BLPS), clear with the two-wavelength laser-protective frontsert, and amber (for sunny days). In addition to providing ballistic and laser protection to emmetropic soldiers, BLPS can be fitted with a lens carrier behind the protective eyewrap for ametropic soldiers. The fourth soldier, right, is wearing the Special Protective Eyewear Cylindrical System (SPECS) without reflective laser technology. Currently, SPECS is being developed for emmetropic soldiers only.

contact lenses to correct visual problems such as aphakia, high degrees of myopia, keratoconus, or irregular corneal astigmatism from corneal scarring. These workers may actually function more efficiently and be less prone to on-the-job accidents when they wear contact lenses than if they wear eyeglasses.[50] However, most contact lens wearers wear them for cosmetic reasons.

There are significant safety concerns regarding the use of contact lenses in industrial settings. One concern is that contact lens wearers, like emmetropic individuals, may not wear the safety glasses that the employer provides. Another concern is that dusty, oily, or chemically toxic environments may not be appropriate for wearing contact lenses: dust or foreign bodies can become trapped under rigid contact lenses, causing corneal abrasions, and toxic chemicals can be absorbed into the matrix of soft lenses, leading to a possible toxic exposure to the cornea. In addition, coworkers might be unable to remove the contact lenses if a chemical were to splash into the eyes, thus prolonging the contact time.[51]

Given the fact that some workers will not always wear their eye protection, unprotected workers who are wearing contact lenses will actually fare better against ballistic and chemical hazards than others whose corneas are unprotected. Both rigid and soft contact lenses reduce, and can sometimes deflect the destructive forces associated with projectiles, foreign bodies, and chemical splashes. However, workers must be counseled regularly that contact lenses, by themselves, do not provide sufficient eye protection in an industrial environment against either ballistic or chemical hazards. Goggles or plano safety glasses must be worn over contact lenses, even though they defeat the cosmetic effect.

Wearing contact lenses in chemically hazardous environments provokes still more controversy. In 1978, NIOSH recommended that contact lenses not be worn when employees were working with any of several hundred listed chemicals. The rationale was that rigid contact lenses might trap chemical vapors beneath them, while soft contact lenses might absorb the vapors, which would prolong the contact time of the chemical. As a result, industrial safety specialists restricted the wearing of contact lenses in environments prone to chemical fumes and vapors, chemical splashes, dust, intense heat, or molten metals.[51] How-

ever, recent studies of rigid and soft contact lenses suggest that contact lenses act as a barrier, keeping chemical vapors away from the cornea and therefore minimizing injury.[54,55] However, persistent exposure to chemical vapors may have just the opposite effect: the vapors could be trapped or absorbed, leading to a toxic exposure of the cornea and conjunctiva.

Some workers will insist on wearing their contact lenses in eye-hazardous areas. Therefore, safety managers and supervisors should

- require the use of industrial safety glasses or goggles in conjunction with contact lenses,
- ensure that all workers always have a spare pair of eyeglasses or safety glasses readily available,
- provide workers with a clean area for removing their lenses, and
- instruct fellow workers in emergency contact-lens removal techniques.

Using contact lenses with commercial respiratory protective equipment and military protective masks has also been controversial. In May 1971, NIOSH and OSHA issued a regulation that prohibited the use of contact lenses with protective respiratory equipment in a contaminated atmosphere.[56] Similarly, the army prohibited the use of contact lenses with protective masks. However, a 1985 study of 13 firefighters reported that those who wore respiratory protective equipment were at greater risk of personal injury due to lost, bent, scratched, or fogged glasses than they were when they wore their contact lenses. In 1985, The Lawrence Livermore National Laboratory conducted a survey of 9,100 firefighters in the United States and Canada; of the 1,405 questionnaires that were returned, 403 firefighters reported that they wore contact lenses with respiratory protective equipment despite the regulation prohibiting it. Only six firefighters reported contact lens–related problems so severe that they needed to remove their masks.[57] In March 1987, OSHA announced that it would amend the contact lens prohibition and allow voluntary use of contact lenses.[58]

While the civilian sector is moving toward limited use of contact lenses with respiratory protective equipment, the army is still concerned that sweat will run into soldiers' eyes, causing excessive burning and stinging of the cornea. This could cause the soldier to unthinkingly unmask in a chemically contaminated environment. Currently, the army prohibits wearing contact lenses during gas-chamber exercises, field-training exercises, and combat.[59] It is anticipated, however, that army helicopter pilots will be allowed to request a waiver to this regulation because some aviation systems (such as the "Heads Up" display devices and the M43 protective mask) prohibit the use of spectacles.

ENVIRONMENTAL VISION

Environmental vision pertains to nonindustrial conditions such as illumination, VDTs, and UV radiation from sunlight that may have a detrimental effect on visual efficiency, ocular health, or both. Illumination and VDT problems often require that the surrounding environment be modified to improve workers' visual efficiency and productivity. Inadequate or insufficient illumination can significantly reduce productivity and simultaneously increase the number of work-related injuries.

Illumination

Illumination is an important element of vision conservation that is often overlooked. Industrial lighting should provide a safe working environment and improve visual efficiency, safety, and comfort. Fifty years ago, when wages were relatively low and incandescent lamps were inefficient, it was cheaper to hire additional workers to compensate for the inefficient work practices associated with poor lighting. However, with the passage of time, higher wages, and an ever-increasing variety of lamps being developed, it has become more cost effective to improve worker productivity by improving both the quantity and the quality of illumination in the workplace.

Illumination surveys are usually performed by the installation industrial hygienist. When an illumination survey is performed, three important factors should be evaluated: the quantity of illumination, the quality of illumination, and visual comfort. All three factors are interrelated and should complement one another.

Quantity of Light

The amount of light emitted by a light source that falls on a surface or work station (the quantity of illumination) should match the visual demands of the task. Detailed work (such as reading machinist's calipers) requires more illumination than gross tasks (such as driving a forklift). The Illuminating Engineering Society (IES) and many lighting-equipment compa-

nies publish tables of minimum recommended illumination by job categories (Table 8-7). However, minimum recommended levels of illumination should not be confused with the illumination levels that allow for maximum worker productivity. For example, the amount of light that is needed for maximum visual efficiency varies with the employee's age; an employee who is near retirement age often requires several times more illumination than a younger worker needs to see the same work.

The *illuminance*, or luminous intensity seen as visible light striking a surface of an object, is measured as the *candela* (candle), a unit based on luminous flux, the *lumen*, per unit area of surface. A source of light of 1 candela produces 4 steradians (π) lumens of luminous flux in all directions (Figure 8-30).

The intensity of illumination is expressed as the ratio of the illumination source and the radiated surface area, and is measured in units of *lux* (lumens/m^2), and the quantity of light, a parameter that includes the duration of light measured in seconds or hours. In practice, the luminous intensity from a source of light is measured in three different standard units:

- 1 lumen/cm^2
- 1 lumen/m^2 (1 lux, the modern metric term)
- 1 lumen/ft^2 (1 foot-candle [ft-c, the nearly obsolete term that is still used in the United States, especially in the lighting industry)]

TABLE 8-7

MINIMUM RECOMMENDED LEVELS OF ILLUMINATION

Task	Illumination (ft-c)	Examples
Casual	30	Warehouses
Rough	50	Reading large markings
Medium	100	Sewing, woodworking
Fine	500	Electronics

Adapted with permission from General Electric. Basic lighting considerations. In: *Industrial Lighting*. Cleveland, Oh: GE; 1969: 4.

The early photometric standard of light was actually a candle (made of sperm wax), hence the term *standard candle*. Later, the National Bureau of Standards retired the candle for carbon filament lamps and in 1948, a *new candle* standard was adopted. The new candle is based on the radiation of light emitting from a blackbody of platinum when the temperature is raised to the melting point of the noble metal, 2,047°K. At that temperature, 600,000 new candles/m^2 of luminous intensity is emitted (equivalent to 60 candles/cm^2).

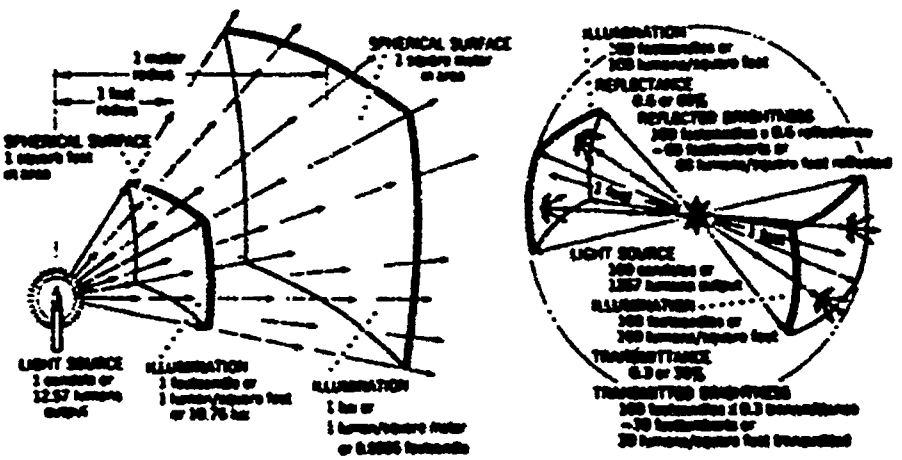

Fig. 8-30. The concept of illumination and its units of measurement, left. The amount of illumination falling on a 1 m^2 spherical surface, located 1 m from a light source that is emitting 1 candela (12.57 lumens) of output, is defined as 1 *lux* (1 lumen/m^2). The concept of luminance, right, which is defined as the amount of light transmitted through or reflected by a surface. Luminance is subjectively called "brightness." A surface that is illuminated with 100 lux and has a reflectance of 60% will measure 60 lamberts of luminance. Measurements of luminance portray true lighting conditions more accurately than do measurements of illumination. Reprinted with permission from General Electric. *Light Measurement and Control*. Cleveland, Oh: GE; 1971. Publication TP-118.

The historical confusion between the "brightness" of light as measured as intensity per unit area and "brightness" as judged subjectively through the human eye led to the use of the term: *luminance*, expressed as candles per m^2. The standard unit of *relative spectral sensitivity* of the eye (equal to light of an intensity of 1.0 new candle, which equals 1.0 π of light source) is equivalent to 685 lumens/W of luminous efficiency at the wavelength 555 nm. In the visible part of the EM spectrum, the spectral sensitivity of the human eye can be plotted as a bell-shaped curve. At 555 nm, spectral intensity is 1.0. Spectral sensitivity decreases from 1.0 in spectral intensity as wavelength decreases to approximately 390 nm (at 400 nm, spectral intensity equals 0.0004), and spectral sensitivity also decreases from 1.0 in intensity as wavelength increases from 555 nm, to approximately 760 nm (at 760 nm, spectral intensity equals 0.00006). The wavelength of light to which the human eye is most spectrally sensitive is 555 nm, in the green part of the visible spectrum.

Illumination is categorized as either *general illumination* (the ambient light, which illuminates a broad area) or *supplemental task lighting* (lighting that is added to increase the visibility of certain tasks). Many industries use fluorescent, mercury, metal halide, or high-pressure sodium lamps to achieve 30 to 50 ft-c of general illumination. While 30 to 50 ft-c of illumination may be sufficient as general illumination, this amount may not be sufficient to produce maximum worker productivity; supplemental task lighting may need to be added above specific work stations. For example, many machine shops have a general illumination level of 50 ft-c; to increase productivity and reduce the risk of on-the-job accidents, supplemental lighting will need to be added over each machine to provide at least 100 ft-c of illumination at each work station. Increasing the general illumination level to 100 ft-c is not recommended because it is usually more expensive than supplemental task lighting, and the resulting increase in *glare* (excessively bright light that causes visual discomfort) might negatively affect overall productivity.

Unfortunately, most lighting surveys stop at measuring the number of footcandles emitted by the lighting source, which may lead to erroneous conclusions as to the adequacy of illumination. In terms of visual comfort and job performance, measurements of luminance (the amount of light emitted or reflected from a work surface toward an observer or worker) are perhaps more accurate (and more important) than measurements of illumination. In the simplest terms, luminance (which is measured in footlamberts or lamberts) is the product of illumination (in footcandles) and the luminous reflectance of the surface. Tasks involving dark-colored (such as gray) objects may require as much as 10-fold more illumination than tasks involving lighter-colored (such as yellow) objects to achieve the same degree of luminance or brightness. Unfortunately, a standard light meter does not measure luminance. However, by holding the meter 2 to 4 in from the surface to be measured, it can be used to approximate the measurement of luminance.

Quality of Illumination

Proper lighting (ie, the proper distribution and control of lighting) allows the worker to focus on the task at hand, rather than causing his or her eyes to stray (which, in turn, increases ocular fatigue and reduces work efficiency). Unlike the quantity of illumination (which is measured without regard to the human observer), the quality of illumination deals with the degree of brightness (ie, a person's impression of the relative intensity of light). To improve task perfor-

TABLE 8-8

RECOMMENDED MAXIMUM BRIGHTNESS RATIOS

	Environmental Classification		
	A	B	C
1. Between tasks and *adjacent darker* surroundings	3–1	3–1	5–1
2. Between tasks and *adjacent lighter* surroundings	1–3	1–3	1–5
3. Between tasks and more *remote darker* surfaces	10–1	20–1	*
4. Between tasks and more *remote lighter* surfaces	1–10	1–20	*
5. Between luminaires (or windows, skylights, etc.) and *surfaces adjacent* to them	20–1	*	*
6. Anywhere within normal field of view	40–1	*	*

*Brightness ratio control not possible

A: Interior areas where reflectances of entire space can be controlled in line with recommendations for optimum seeing conditions

B: Areas where reflectances of immediate work area can be controlled, but control of remote surroundings is limited

C: Areas (indoor and outdoor) where it is completely impractical to control reflectances and difficult to alter environmental conditions

Reprinted with permission from General Electric. Basic lighting considerations. In: *Industrial Lighting*. Cleveland, Oh: GE; 1969: 6. Publication TP-108.

mance and productivity, recommended minimum brightness ratios, also called luminance ratios, have been established for use in industry (Table 8-8). Brightness ratios can be controlled by the proper selection and location of lamps; painting or cleaning reflective surfaces (walls, ceilings, equipment, or floors); and supplemental task lighting.

Direct and indirect lighting techniques are used throughout industry to improve the quality of illumination. *Direct lighting* (light that falls directly on the task) is the most efficient type of illumination; however, it tends to produce shadows and glare. *Indirect lighting* (lighting that is reflected off adjacent ceilings or walls) is more comfortable to work under than direct lighting because it produces significantly less glare. Unfortunately, indirect lighting is less efficient (and therefore more expensive) than direct: because indirect lighting is reflected off adjacent ceilings and walls, more initial illumination is required to achieve the same illumination as direct lighting.

Color contributes to the quality of illumination as well. *Object color* is defined as the color of light reflected or transmitted by an object when it is illuminated by a *standard light source* (standard source A [a tungsten filament lamp operated at a color temperature of 2,856°K], or standard source B [an approximation of sunlight at noon, having a correlated color temperature of approximately 4,874°K], or standard source C [an approximation of daylight provided by a combination of direct sunlight and clear sky, having a correlated color temperature of approximately 6,774°K]). *Color rendering* is a term applied to lighting sources; an object will *render* different colors depending on the spectrum composition of the lighting source (Figure 8-31). Poor color rendering by an illumination source can distort color perception, increase ocular

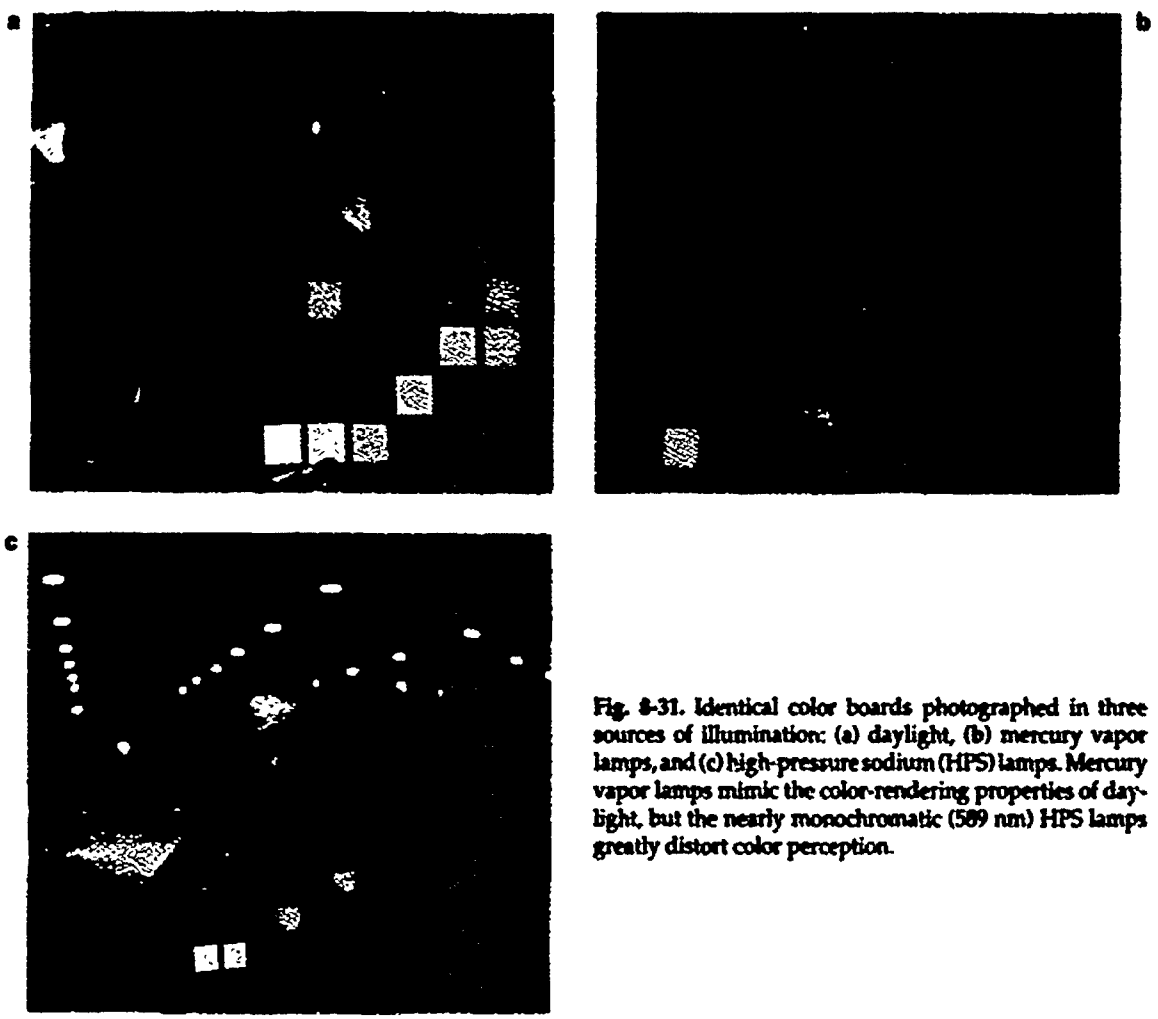

Fig. 8-31. Identical color boards photographed in three sources of illumination: (a) daylight, (b) mercury vapor lamps, and (c) high-pressure sodium (HPS) lamps. Mercury vapor lamps mimic the color-rendering properties of daylight, but the nearly monochromatic (589 nm) HPS lamps greatly distort color perception.

fatigue, and reduce worker productivity. Fluorescent lights, the lamps used most commonly in workplaces (due to their cost efficiency), emit a broad spectrum of light, giving them color-rendering properties second only to sunlight. Incandescent lamps also have excellent color-rendering properties but are less efficient and produce more heat than fluorescent lamps. Mercury vapor and metal halide lamps, often used in gymnasiums and large industrial bays, are even more efficient, but cause a mild distortion of color perception. High-pressure sodium-vapor (HPS) lamps are used in warehouses and many industrial manufacturing bays. They produce a golden white, broad-spectrum light with its maximum intensity centered around 589 nm. Low-pressure sodium-vapor lamps, which are the most efficient lamps made, are used to illuminate highways and parking lots. The golden orange light produced is almost monochromatic (consisting of a double wavelength at 589 and 589.6 nm) and significantly distorts color perception. As a general rule, lamps with good light efficiency tend to have poor color-rendering properties, and vice versa (Table 8-9).

TABLE 8-9

LAMP EFFICIENCIES

Luminaire Type	Lumens per Watt
Incandescent bulbs	17–23
Fluorescent tubes	70–80
Mercury vapor lamps	44–55
Multivapor lamps	80–90
High-pressure sodium lamps	115
Low-pressure sodium lamps	170

Sources: (1) General Electric. *Industrial Lighting*. Cleveland, Oh; GE; 1969, Pub TP-109. (2) General Electric. *Light Measurement and Control*. Cleveland, Oh: GE; 1971, Pub TP-118. (3) Kaufman JE, Christensen JF. *IES Lighting Handbook*. New York: Illuminating Engineering Society; 1972.

Visual Comfort

A worker's visual efficiency and comfort are maximized when glare from illumination sources or work surfaces or both are minimized. *Direct glare* comes from uncontrolled light sources (light sources without reflectors or diffusors). In order to reduce the uncomfortable and sometimes disabling effects that are associated with direct glare, lighting sources (such as lamps or windows) should be restricted by the use of appropriate reflectors, diffusors, blinds, or louvers. For example, light emitted from a bare light bulb is more harsh and uncomfortable than light emitted from a lamp equipped with a shade. *Reflected glare* comes from highly polished surfaces (such as desk tops, VDT screens, or glossy paper) and can cause significant visual discomfort. Reflected glare is controlled by moving the location of the light source or by changing the angle of the work plane so that the light does not reflect into the worker's eyes.

Illumination Dilemmas

In response to the world oil crisis, Congress passed the Energy Conservation Act in 1973 to reduce the federal government's use of energy and energy-related products such as natural gas and oil. The provisions of this act (Title 41, Code of Federal Regulation, Section 101-20.107, *Energy Conservation*) mandate that maximum illumination levels of work-station surfaces be no greater than 50 ft-c; of general work areas, no greater than 30 ft-c; and of nonwork areas such as halls, no greater than 10 ft-c. The purposes of reducing levels of illumination were twofold: to reduce electrical costs (which reduced the use of gas and oil) and to reduce air-conditioning costs (to offset the additional heat associated with increased lighting).

To maximize illumination and minimize electrical costs, many installations spent millions of dollars converting inefficient incandescent and fluorescent lamps to higher-efficiency HPS lamps. Most of these conversions were in poorly lit areas such as warehouses. Because warehouse lighting was successfully improved, similar conversions were made in industrial areas such as machine shops, and even in administrative offices. During routine USAEHA site visits, the staff received complaints from workers as a result of these conversions. Workers complained not only about the unnatural golden-orange color but also about their inability to discriminate colors properly in this light. For example, rust on metal parts, which is easy to see under fluorescent lighting or sunlight, cannot be seen under HPS lamps. Daytime shift workers had no complaints about the lighting (because the HPS lighting was supplemented with sunlight through the windows), but the evening and night shifts complained incessantly about the lighting.

Other problems associated with HPS lamps are excessive glare and headaches. Because HPS lamps are so efficient and cost effective, some installations have removed the supplemental task lighting from machine shops and have installed more HPS lamps than are usually necessary. This has resulted in ma-

chine shops having 75 ft-c to 100 ft-c of general illumination. While this meets the minimum IES-recommended levels of illumination for certain operations, the glare from so many HPS lamps can be tremendous; many workers wear hats indoors to reduce the glare and its consequent headaches.

To reduce worker complaints associated with HPS lighting and to improve productivity, the USAEHA has made several recommendations:

- No further conversions to HPS lamps should be made until the impact on workers has been assessed at the installation.
- HPS lamps should not be utilized in administrative offices, especially in buildings where sunlight cannot supplement the ambient lighting.
- General illumination levels should be reduced to 50 ft-c (or slightly less), but supplemental task lighting, which may have been removed, should be restored.
- Installations should mix mercury-vapor or multivapor lamps with HPS lamps to achieve a more natural spectrum of illumination.

Personal Computers and Video Display Terminals

In the workplace, typewriters have been supplanted by PCs and VDTs, not only in this country but also throughout much of the world. In 1975, there were fewer than 200,000 VDTs in use in the United States; 10 years later, after PCs were introduced, this number had grown to approximately 13 million units—with 100 million projected by the year 2000.[40] Concurrent with this phenomenal growth in VDT use came an increase in the clinical signs and medical symptoms of work-related health problems.

Problems with Vision

Approximately 50% to 75% of computer workers experience some form of visual discomfort, including fatigue, headaches, eyestrain, burning eyes, blurring of the monitor screen, intermittent double vision, distance blurring after using a PC, neckache, and backache.[40,41] This discomfort is most often attributed to visual problems of the worker, the surrounding environment, or a combination of both. Visual problems can stem from (*a*) uncorrected refractive errors, (*b*) accommodative problems or presbyopia, (*c*) binocular coordination problems, (*d*) glare, (*e*) contour sharpness, and (*f*) the flicker effect (which may cause fatigue, migraine headaches, and other nonvisual physical problems in certain flicker-sensitive people).[41,42] In addition, a dirty screen makes the information that is displayed more difficult to read, so screens should be cleaned daily.

The most common refractive condition associated with computer-related visual symptoms is latent or low *hyperopia* (farsightedness). Hyperopic individuals are usually able to compensate for the small degree of hyperopia by accommodating (or focusing) during short-term tasks; they usually do not require corrective spectacles until later in life. With the use of PCs, however, hyperopic individuals accommodate more extensively and eventually develop visual symptoms. Most of these individuals can be helped with *low-plus* (up to +1.25 diopters) lenses or bifocals.

Myopia (nearsightedness) does not usually produce visual symptoms in PC users. However, transient myopia (which is actually accommodative spasm) in otherwise emmetropic individuals can occur with prolonged use of PCs. Rather than wearing *low-minus* (up to –0.50 diopters) lenses, these individuals may benefit from visual training that is directed at relieving the accommodative spasm; in addition, they should wear either low-plus lenses or bifocals to relieve accommodative stress while working at their computers.

Astigmatism (a focusing anomaly that occurs at both distance and nearpoint viewing) can adversely affect PC workers. Individuals with moderate to large degrees of astigmatism usually wear glasses full time to correct the astigmatic error. Some individuals with small errors, however, function adequately without glasses until they are confronted by a visually intense task, such as looking at a computer screen for several hours. These individuals need to be referred for a visual examination.

Other vision-related problems include accommodative problems and presbyopia. PCs typically require an individual to accommodate for both the nearpoint (14–18 in) and intermediate (18–24 in) working distances. Some young individuals have dysfunctional accommodation systems and develop significant problems when working at a computer for prolonged periods. Everyone becomes presbyopic with age (an individual's amplitude of accommodation gradually diminishes). At approximately 40 years of age, prescription reading glasses or bifocals are usually required for any extensive reading or nearpoint task. Individuals with accommodative problems or presbyopia require a complete eye examination to assess their accommodative functions. Typically, these individuals will require either low-plus lenses or bifocals (to compensate for the lack of accommodation); in some cases, visual training (to improve the amplitude of accommodation) can be helpful.

Because working with PCs involves both nearpoint and intermediate distances, distance-specific spectacles should be prescribed: reading glasses, with sufficient depth of focus to encompass both distances; or bifocals, the upper section of which is used for the intermediate distance and the lower section for the standard reading distance. Conventional trifocals should be avoided; they tend to induce neck- and backaches due to improper positioning of the head (Figure 8-32). Special trifocals with an intermediate vertical-segment height of 10 to 14 mm (rather than the usual 7 mm) are available and may be prescribed in certain cases.

Some computer workers will have problems with their binocular vision. In many instances, visual symptoms stem from *esophoria* (overconvergence of the eyes) or *exophoria* (underconvergence or divergence of the eyes) at the intermediate or nearpoint working distances. Many individuals with esophoria or exophoria do not complain of visual discomfort until they are required to perform nearpoint tasks, such as using a PC, for extended periods. Like individuals with accommodative problems and presbyopia, these individuals also require a thorough eye examination.

In most esophoric patients, low-plus lenses should be sufficient to relieve the symptoms. In others, as well as in patients with exophoria, visual training is usually required to improve the eyes' abilities to converge and diverge. However, optometrists and ophthalmologists should avoid prescribing glasses with prism (to compensate for exophoria or esophoria); patients will accept prism initially, but will invariably require larger corrections of prism over time.

Glare causes considerable problems for PC users. Reflected glare (from overhead lights or nearby windows) makes the images on the screen difficult to see and causes eyestrain (Figure 8-33). Curtains or blinds can help control the glare from windows. Antireflection screens can significantly reduce the annoying effects of glare by reducing reflections.

Background or contrast glare is an even more significant problem at most PC work stations. If the general lighting is overly bright or if the screen is located in front of either a window or a white wall, the

Fig. 8-32. Workers who wear conventional bifocals or trifocals usually have few visual problems when doing routine desk work, but they often have problems when using a video display terminal. This office worker is trying to use a bifocal segment that is too weak for reading the monitor; at the end of an 8-hour day, she will undoubtedly have neck and back strain. To remedy this situation, this worker should have *occupation-specific* bifocals—with the intermediate prescription (for an 18–24-in. viewing distance) in the upper portion of the lens and the stronger reading prescription (for 14–18-in. reading distance) in the lower segment.

Fig. 8-33. Computer work stations should be positioned for maximum productivity. This worker might be distracted by glare from the window. Windows should be equipped with curtains, miniblinds, or both, to reduce direct glare. If this cannot be accomplished, the workstation should be moved so the source of glare is behind the worker.

iris will decrease the size of the pupil. This limits the amount of light that can reach the retina. The image on the screen will appear less bright, which can lead to eyestrain and headaches in operators who must view the screen for several hours. The solution is to decrease the ambient lighting to improve the brightness ratio between the background and the screen; however, if operators must work from hard copy, they should illuminate it with a spot lamp.

Contour sharpness is yet another problem for computer users. The sharpness of the images displayed on the monitor depends on the matrix (the number of horizontal and vertical dots per inch of screen). Older color graphics array (CGA) monitors (also known as red, green, blue [RGB] monitors) are being replaced with new-generation enhanced graphics array (EGA), video graphics array (VGA), and even higher-resolution monitors, which, because they have higher resolution, cause less eyestrain than the older models.

The McCollough Effect (an afterimage that causes white letters and objects to appear pink) is a startling visual phenomenon. It occurs in computer workers who spend long periods before a monochrome screen that displays green characters against a dark background. The theoretical explanation for this unusual phenomenon is that the green-stimulated retinal receptors become fatigued due to the constant stimulation; consequently, when the computer operator looks away from the screen, white objects are devoid of green, making them appear pink. Discovered in 1965, the McCollough Effect is seemingly harmless and relatively short lasting.

Radiation-Related Health Effects

A number of studies have associated cataract formation with PC use. In 1983, 10 anecdotal cases of cataracts were reported in VDT users; six of the patients had minor opacities that did not impair their vision, while four others had a history of exposure to other cataractogens. Many experts, however, discount the risk of cataracts. Testimony before a United States House of Representatives subcommittee suggests that 25% of the population of the United States has opacities of the lens without impaired vision, while approximately 4% of the population 35 to 45 years of age has age-related cataracts. In addition, radiation-induced cataractogenesis is thought to require exposures 10,000-fold greater than that expected from a PC.[80]

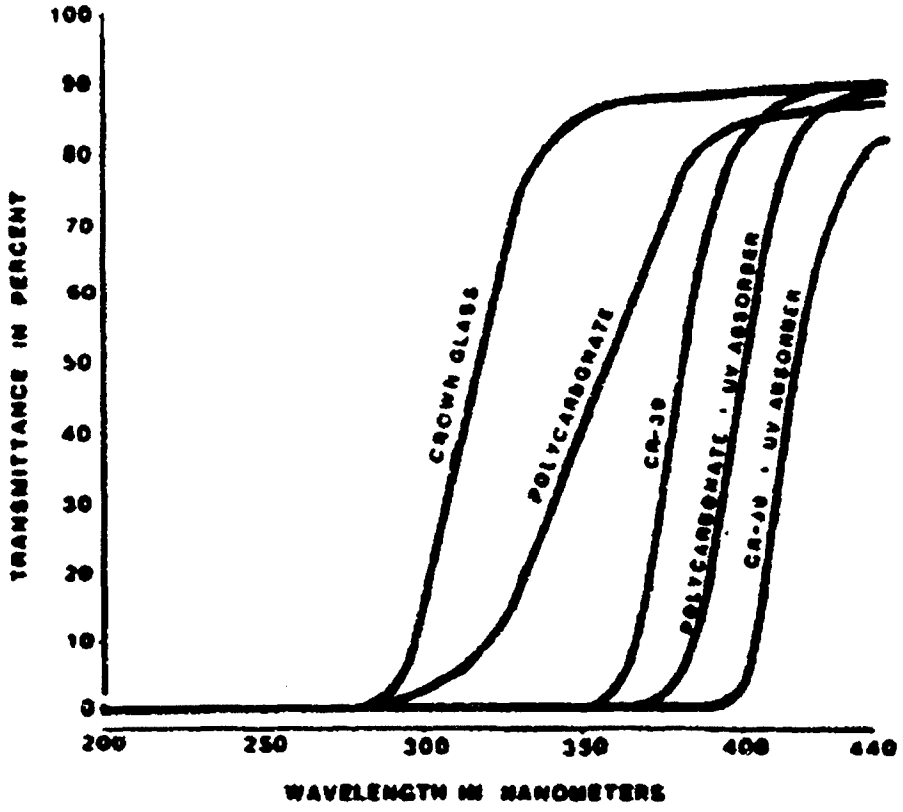

Fig. 8-34. Transmittances of different optical materials used to manufacture spectacle lenses. Source: Pitts DG. Ultraviolet protection, when and why? *Prob in Optom.* 1990;2(1):95–115.

Solar Ultraviolet Radiation

Although exposure to solar UV radiation is not considered to be an industrial hazard, it can be an occupational hazard. Many outdoor workers, including soldiers, can sustain excessive exposure and may be at risk for sunburn or premature cataract formation. Photokeratitis due to sunlight does not normally occur unless exposure levels are unusually excessive. Levels of solar UV radiation overexposure are difficult to estimate due to a number of variables including (*a*) the time of day, (*b*) the angle of the sun, (*c*) the latitude and altitude at which the person is working, (*d*) the degree of cloud cover, and (*e*) changes in the surrounding reflecting surfaces. For example, more than 60% of UV radiation exposure occurs between 1000 and 1400 hours, when the sun is highest in the sky. Furthermore, UV exposure increases by 15% for each kilometer of altitude (approximately 5% for each 1,000 feet). Clouds do not attenuate UV radiation; that is why sunburns occur even on overcast days. Green grass reflects only 3% to 5% of ambient UV radiation, while fresh and salt water reflect 3% to 8%, dry sand reflects 15% to 18%, and fresh snow reflects 85% to 95%. When all these factors are considered, snow skiing (due to the altitude and the reflectivity of snow) is the most UV-intensive environment, followed by sunbathing at the beach.[39]

Glasses with a filtering tint provide the best protection against low levels of solar UV radiation. However, it is dangerous to assume that all tinted glasses or sunglasses provide adequate or equal filtering protection. Data on lens materials reveal that regular glass lenses attenuate approximately 95% of UV radiation (the amount varies with the wavelength), followed by polycarbonate and then CR-39 (Figure 8-34). Manufacturers of CR-39 and polycarbonate lenses add UV inhibitors to prevent the virgin CR-39 polymer from yellowing as the lens absorbs UV radiation over time. While a clear CR-39 ophthalmic lens protects a wearer against low-level UV-C and UV-B radiation, it does not provide acceptable protection against UV-A radiation. Maximum protection against solar UV radiation occurs when CR-39 lenses are coated with an additional UV-absorbing dye called UV-400.[39]

SUMMARY

Soldiers require good, if not excellent, vision to be able to spot their enemies quickly and to fight to the best of their abilities. To this end, armies have sought eye protection: a soldier who has been blinded is both useless to the battlefield commander and at risk of being killed. The U.S. Army's interest in meeting the ever-increasing threats to vision and ocular health has expanded through the post–World War II inception of the Occupational Vision Program to the current Vision Conservation Program.

The Vision Conservation Program is an installation-based, dynamic program comprising three elements: occupational vision, eye safety, and environmental vision. The goal of the occupational vision element is to provide military personnel and DoD civilian workers with the best vision possible for them to work and recreate safely, productively, efficiently, and comfortably. Eye safety is directed toward eliminating injuries through training, administrative and engineering controls, and by providing individuals with appropriate PPE. The environmental vision element evaluates and provides solutions for environmental problems such as illumination and radiation (ionizing and nonionizing), which may negatively influence the worker's visual efficiency and health.

Perhaps the greatest challenge to the army and its Vision Conservation Program, after trying to field acceptable, standard-issue, eye-appropriate PPE, is wearing compliance—both on the job and off duty. Individuals who do not wear prescription eyeglasses are often uncomfortable wearing a device that does not obviously affect their performance. However, the use of eye protection can be expected to increase over time with safety leadership by supervisors and managers and continuous worker education and training.

REFERENCES

1. Kupfer C. Six main causes of blindness. In: Wilson J (ed); *World Blindness and Its Prevention*. New York: Oxford University Press, 1984: 4–13.

2. Parver LM. The national eye trauma system. *Int Opthalmol Clin*. 1988;28(3):203–205.

3. Vision problems in the US Facts & Figures. New York: National Society to Prevent Blindness, 1980. In: Dell WM. Epidemiology of ocular disease. *Prob in Optom*. 1989;1(1).1–17.

4. Thackray J. High cost paid by industry for preventable eye injuries. *Occup Health Saf*. 1984;53(2):31–33.

5. Belkin M. *Ophthalmological Lessons of the 1973 War: Prevention of Ocular War Injuries*. Jerusalem, Israel: Hadassan University Hospital, Department of Ophthalmology. Original, unpublished research.

6. Belkin M. Ocular trauma during Yom Kippur War. In: *Combat Ocular Problems*. Proceedings of Conference, Sponsored by the Division of Ocular Hazards and Letterman Army Institute of Research at Presidio of San Francisco, Calif; October 20–21, 1980.

7. Hornblass A. Eye injuries in the military. *Int Opthalmol Clin*. 1981;21:121–138.

8. Nothstein GZ. Inception and overview. In: *The Law of Occupational Safety and Health*. New York: The Free Press. 1981;1–23.

9. The Occupational Safety and Health Act of 1970, Pub L No. 91-596.

10. 29 CFR, Part 1910 § 133, Subpart I.

11. American National Standards Institute. *American National Standard Practice for Occupational and Educational Eye and Face Protection*. New York: ANSI;1968. ANSI 787.1.

12. Grover L. A written eye safety policy reduces risks and prevents injuries. *Occup Health Saf*. 1986;55(5):31–36.

13. Moss E, compiler. Notes of the *Symposium on Workers' Macular Degeneration*, 9–12 August 1982. Sponsored by the US Department of Health and Human Services, Public Health Service, Centers for Disease Control; and Division of Biological and Behavioral Science, National Institute for Occupational Safety and Health. Cincinnati, Oh: USDHHS,PHS, CDC, NIOSH; 1982.

14. American National Standards Institute. *American National Standard Recommendation for Prescription Ophthalmic Lenses*. New York: ANSI;1968. ANSI Z80.1.

15. The Medical Amendments Act of 1976, Pub L No. 94-295.

16. 21 CFR, Part 801 § 410, Subchap H.

17. Bailey RW. The Army's Occupational Vision Program. *The Sightsaving Rev*. 1956;26(3):144–146.

18 The address of the USAEHA Vision Team is: US Army Environmental Hygiene Agency, ATTN: HSHB-MO-O (Occupational Vision), Aberdeen Proving Ground, MD 21010-5422. The telephone number is: (410) 671-3534.

19. Fox SL. Visual requirements for the job classifications. In: *Industrial and Occupational Ophthalmology*. Springfield, Ill: Charles C Thomas; 1973: 44.

20. *Vision Problems in the United States*. Data Analysis. New York: National Society to Prevent Blindness; 1980.

21. White MF, Morris R, Feist RM, Witherspoon CD, Helms HA, John GR. Eye injury: Prevalence and prognosis by setting. *Southern Med J*. 1989;82(2):151–158.

22. US Department of Labor, Occupational Safety and Health Administration. *Eye Protection in the Workplace*. Washington, DC: US DOL; 1983. Fact Sheet OSHA-33-03.

23. Department of Commerce, Bureau of Census. *1980 Census of the Population. Classified Index of Industries and Occupations*. Washington, DC: GPO; 1982. Publication PHC80-R4.

24. Nakagawara VB. Functional model of an eye protection program: Guide for the clinical optometrist. *J Am Optom Assoc*. 1988;59(12):925–928.

25. La Piana FG. Fact sheet: Facts bearing upon the development of eye armor for military personnel. Washington, DC: Walter Reed Army Medical Center; 1984.

26. Ward DL, Gorie C. Occupational eye injuries in soldiers. *J Occup Med*. 1991;33(5):646–650.

27. Lott RJ. Occupational health for the soldier. *Medical Bulletin of the US Army*. 1988;2:28–30. PB 8-88.

28. National Society to Prevent Blindness. *Sports & Recreational Eye Injuries—1986*. Schaumburg, Ill: NSPB; 1987 (Updated)

29. Pavan-Langston D. Burns and trauma. In: *Manual of Ocular Diagnosis and Therapy*. Boston: Little, Brown and Co; 1980: 31–45.

30. Nyman JS. Chemical and thermal injuries. *Prob in Optom*. 1989;1(1):45–56.

31. American National Standards Institute. *American National Standard for Emergency Eyewash and Shower Equipment*. New York: ANSI; 1981. ANSI Z358.1.

32. Office of The Surgeon General. *Acanthamoeba keratitis and eyewash stations*. Washington, DC: OTSG; 1989. Memorandum.

33. Markell EK, Voge M, John DT. Lumen-dwelling protozoa. In. Ozmat S, ed. *Medical Parasitology*. 7th ed. Philadelphia: W. B. Saunders; 1992.

34. Tyndall RL, Lyle MM, Ironside KS. The presence of free-living amoebae in portable and stationary eye wash stations. *Am Ind Hyg Assoc J.* 1987;48:933–934.

35. Poe CA. Eye-irrigating lens more effective if applied seconds after accident. *Occup Health Saf.* 1990;59(1):43–44.

36. Nista FM. Spotlight on UVR. *Vision Care Assistant.* 1990;2(1):4–6.

37. McKinlay A, Harlen F. Ultraviolet radiation in the workplace. *Occup Health.* 1979;31(10):454–461.

38. Pitts DG. Threat of ultraviolet radiation to the eye—How to protect against it. *J Am Optom Assoc.* 1981;52(12):949–957.

39. Pitts DG. Ultraviolet protection, when and why? *Prob in Optom.* 1990;2(1):95–115.

40. Taylor HR, West SK, Rosenthal FS, et al. Effect of ultraviolet radiation on cataract formation. *N Engl J Med.* 1988;319(22):1429–1433.

41. Barker FE. Radiation-induced ocular injuries. *Prob in Opto.* 1989;1(1):169–177.

42. Cox E. An introduction to laser safety. *Occup Health.* 1986;38(1):10–15.

43. Large EJ, Olishifski J. Nonionizing radiation. In: Plog BA, ed. *Fundamentals of Industrial Hygiene.* 3rd ed. Chicago: National Safety Council; 1987: 227–257.

44. Sliney DH, Kotulak JC. Hazards of fielded lasers. *Medical Bulletin of the US Army.* 1988;2:14–16. PB 8-88.

45. American National Standards Institute. *American National Standard for the Safe Use of Lasers.* New York: ANSI; 1986. ANSI Z136.1.

46. US Department of the Army, Office of The Surgeon General. *Surveillance of Laser and Radiofrequency Radiation Personnel.* Washington, DC: DA OTSG; 1992.

47. National Research Council. *Effects of Microwave Radiation on the Lens of the Eye.* Washington, DC: National Academy Press; 1981.

48. Hathaway JA, Soles EM, Leighton E. Ocular medical surveillance on microwave laser workers. *J Occup Med.* 1977;19(10):683–688.

49. Reesal MR, Dufresne RM, Suggett D, Alleyne BC. Welder eye injuries. *J Occup Med.* 1989;31(12):1003–1006.

50. La Piana F. *Proceedings from Occupational Vision Consultants Symposium.* 17–19 September 1991. Towson, Md: US Army Environmental Health Agency; 1992.

51. Hirschfelder D. Contact lenses may pose new problems in the workplace. *Occup Health Saf.* 1984;53(3):54–58.

52. Schnider C. CLs in the workplace. *Contact Lens Spectrum.* 1990;5(8):24.

53. Dixon WS. Contact lenses: Do they belong in the workplace? *Occup Health Saf.* 1978;47:36–39.

54. American Optometric Association. *Guidelines for the Use of Contact Lenses in Industrial Environments.* Alexandria, Va: AOA; 1989.

55. Randolph SA, Zavon MR. Guidelines for contact lens use in industry. *J Occup Med.* 1987;29(3):237–242.

56. 29 CFR, Part 1910 § 134(e) (5) (ii), Subpart I.

57. American Optometric Association. *Contact Lenses and Respirators.* Alexandria, Va: AOA; 1989.

58. Assistant Secretary for Occupational Safety and Health, Department of Labor. Washington, DC. Written communication. 4 March 1987.

59. US Department of the Army. *Preventive Medicine*. Washington, DC: DA; 1990. Army Regulation 40-5.

60. Council on Scientific Affairs. Health effects of video display terminals. *JAMA*. 1987;257(11):1508–1512.

61. Professional News. *Optom Mgmt*. 1989;24(11):2.

62. Rosch WL. The big question: Is the PC environment a safe place to work? *PC Magazine*. 1989;8(21):275–297.

Chapter 9

MILITARY ENERGETIC MATERIALS: EXPLOSIVES AND PROPELLANTS

DONALD T. BODEAU, M.D., M.P.H*

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- Propellants
- Pyrotechnics

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- General Safety Practices
- Industrial Hygiene Principles

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- Acute Exposure Decontamination

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COMPOSITE PROPELLANTS AND EXPLOSIVES

LIQUID PROPELLANTS
- Rocket Propellants
- Gun Propellants

SUMMARY

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INTRODUCTION

The United States military is a major producer and consumer of explosives and propellants. Although we have recognized the toxic effects of some of these compounds for many years, most of the data on their effects on human health were published during World War I and World War II, and many voids remain in our knowledge of their human and ecological toxicity. The database on the effects on health (especially human) is uneven and we must remain alert for newly discovered or described effects, especially those that concern carcinogenic and reproductive effects. The lack of adequate data on exposure in human occupational epidemiology studies, and the lack of route-specific toxicity data (especially inhalation and dermal absorption) in animal studies, precludes our making dose-response estimates for most explosives. Therefore, it is our responsibility to err on the side of safety in making judgments about human exposures to these chemicals, and we must include data on structurally similar chemicals in our overall assessment of the probable health hazards of the explosives.

The production of most of these explosives parallels the military activity of the United States. Peacetime production is usually sufficient only for research and training needs. During wartime, the manufacture of these compounds increases; the workforce increases (thereby increasing the number of inexperienced workers who are unfamiliar with these compounds), and physicians who are inexperienced with the unique hazards posed by these chemicals are suddenly charged with the care of explosives workers. The rapid increase in production during wartime has tended to result in far higher exposures, with correspondingly more numerous and more severe adverse effects, than the few mild adverse reactions that occur during peacetime production.[1-3]

Furthermore, the evolving regulatory environment surrounds these and other industrial exposures to hazardous substances. For example, the Occupational Safety and Health Administration (OSHA) has revised the air-contaminant regulations for over 400 chemicals, including nitroglycerin. Whether these new standards can be enforced is still undecided; some questions have been raised regarding whether the proposed standards are feasible from an engineering perspective.

This chapter focuses exclusively on military explosives and propellants, and is structured according to the chemical family of the compounds: (*a*) aliphatic nitrate esters, (*b*) nitroaromatics, (*c*) nitramines, (*d*) initiating explosives, (*e*) composite propellants, and (*f*) liquid propellants. Most munitions, however, are mixtures of chemicals. Medical professionals new to propellants and explosives will need to break through the engineering and technical jargon and nomenclature to identify specific chemical exposures before they can deal with them. Occupational health personnel need to expect, in addition to exposures among plant workers, sporadic exposures among ammunition quality-assurance specialists, explosive ordnance-disposal specialists, and personnel who test or use explosives in enclosed spaces.

HISTORY

The Chinese are generally credited with inventing explosives—in the form of fireworks—before AD 1000. Black powder was not introduced to the western world until approximately 1225. Roger Bacon, an English monk, conducted and described some of the first scientific experiments with this explosive mixture of saltpeter, charcoal, and sulfur in 1249. The age of gunpowder began nearly simultaneously in Europe and China with the invention of cannons early in the 14th century, but until 1800, the development of explosives was limited mainly to improvements in the manufacture and application of black powder. Modern explosive technology was developed during the 19th century with increased research and development of propellants, high explosives, and weapons technology.[4]

Because of their ready natural availability, inorganic nitrate-based explosives were the first to gain importance. (Today, the most important inorganic nitrate explosive is ammonium nitrate, which is used in demolition and construction.) Inorganic nitrates formed the basis of black powder, which was the predominant explosive used in the United States before 1900.[5] Its last major military use was during the Spanish-American War of 1898. Black powder is an easily produced physical mixture of sulfur, charcoal, and potassium nitrate, but it is not well suited for most modern military uses: it produces excessive smoke and flash (which could alert the enemy to the position of the gun) and has a dangerous tendency to cake and misfire. However, it is still used in primers, safety

fuzes, flares, grenades, practice munitions, blanks, fireworks, signals, and specialized quarry work.

During the opening years of the 20th century, faster, cheaper, and higher-volume methods for producing explosives were developed. Numerous compounds were synthesized and used as detonators, boosters, and flash suppressors; dynamite almost completely supplanted black powder in commercial use, and trinitrotoluene (TNT) became the most commonly used military explosive.

With these various developments, attention focused on organic nitrate explosives. The aliphatic nitrates were the first group to achieve importance because cellulose, glycerol, sugars, and coal-tar derivatives were readily available for use as raw materials. Later, as cost-effective bulk synthesis of ammonia and formaldehyde became possible, the aromatic nitrates became important militarily. The most recent group to achieve prominence is the nitramines.

Throughout the early years of World War II, the low production capacity for most explosives and propellants plagued the United States, and numerous changes in the manufacturing processes were made in attempts to increase production. Adaptation to the shortages of raw materials, in addition to the unique requirements of each type of weapon, led to the increasing complexity of munitions design. This adaptive solution to inadequate resources was most prevalent in the search for rocket propellants. For example, the addition of nitroguanidine to nitrocellulose- and nitroglycerin-based propellants was found to both increase production capability and meet the unique and exacting requirements for newly developed weapons systems.

Additional weapons research after World War II has further expanded the uses of these compounds. The plethora of explosives and propellants currently in use and under development has resulted from continued research into the properties, cost, safety, stability, and predictable performance of explosives.

The British were the first to respond to the threat that the manufacture of explosive materials posed. In 1875, they passed the *Explosives Act* after an industrial explosion killed 53 people.[4] This law established "inspectors of explosives," who were authorized to inspect all magazines and factories to ensure that operations were accomplished safely.

At the beginning of World War I, TNT was generally believed to be nontoxic in all its stages of production, but this belief changed. During the course of the war, the major powers used approximately 5 billion pounds of high explosives, primarily TNT, resulting in an estimated 10 million battlefield casualties.[4] In the United States, at least 17,000 cases of TNT poisoning occurred during the war, resulting in more than 475 deaths.[6,7] Efforts to reduce the burden of disease included job rotation, medical examinations, and workplace ventilation and hygiene. These efforts were only marginally effective. Successful control of worker exposure was finally achieved through the automation of many operations during shell loading, and the application of strict standards of workplace hygiene.[8]

The World War I experience demonstrated that ammunition-loading plants were among the most dangerous industrial operations, due to the open handling of dusty and fuming compounds. Beginning in 1938, the Ordnance Department and the United States Public Health Service coordinated an intensive effort to forge an integrated health and hygiene program in ordnance plants to reduce this burden of death and disability of the workers.[3] This effort was

> the first large-scale demonstration of what can be accomplished in a large industry offering many serious health hazards by a vigorous medical and engineering program.[2,9,10]

Consequently, the successes of, and lessons learned from, this effort led to the establishment of the field of occupational medicine in the army, where it monitors the health of over 100,000 civilian employees at depots, arsenals, and ammunition plants.

ENERGETIC MATERIALS

An energetic material is a compound that can undergo rapid, self-sustaining, exothermic, reduction-oxidation reactions. Energetic materials may be categorized according to their intended uses: (*a*) explosives, (*b*) propellants, and (*c*) pyrotechnics. Explosives and propellants evolve large volumes of hot gas when burned; they differ primarily in their rates of reaction. *Pyrotechnics* (ie, a powder or ammunition used for igniting a rocket or producing an explosion; the term is also used in the military to designate flares and signals) evolve large amounts of heat but much less gas than explosives or propellants. Energetic materials may also be grouped according to their rate of reaction. Both propellants and pyrotechnics are considered to be *low explosives*, and the velocity at which the combustion proceeds through these materials is usually 400 m/sec or slower (Figure 9-1). In comparison, *high explosives* are *detonated*, a process in which the very rapid rate of the combustion reaction itself produces a shock wave, capable of shattering objects, in the surrounding me-

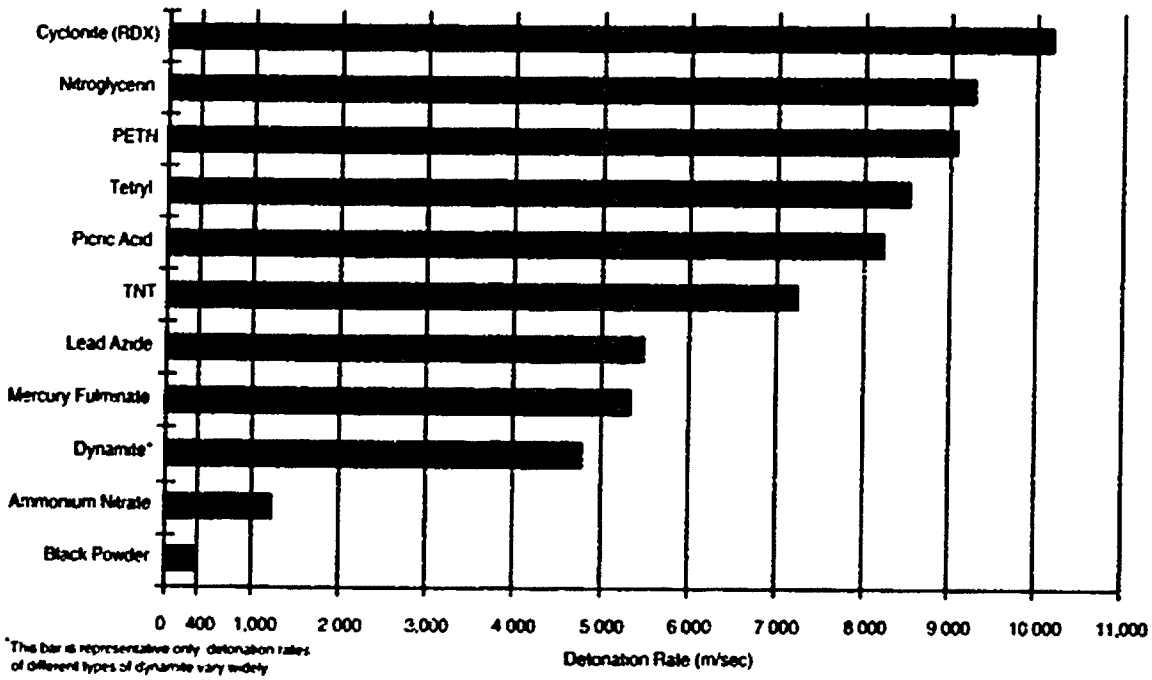


Fig. 9-1. Approximate detonation rates of common military and industrial explosives. Infinite variations on these rates are possible by mixing and by using additives. Both explosive and propellant formulations have taken advantage of these high detonation rates. Adapted with permission from Meidl JH. *Explosive and Toxic Hazardous Materials*. New York. Macmillan Publishing; 1970: 24.

dium. The shock wave moving through the explosive material causes further explosive decomposition of that material, and the reaction rate is determined by the speed of the shock wave. The range of velocities of the shock wave is 1,000 to 9,000 m/sec. In addition to being used as explosive charges, many high explosives are also used in propellant formulations. For purposes of this discussion, the term *explosive* is used generically to indicate any energetic material.

Explosives

Modern explosive devices employ an *explosive train* that takes advantage of the specific explosive properties of its components: the initiator, the detonator, the booster charge, and the main charge (Figure 9-2). The initiator, or primary explosive, consists of a small quantity of material that is very sensitive to heat, spark, impact, or friction. Primary explosives may intensify the energy up to 10 million times that of the initiating stimulus. Geometric arrangement of the explosive device directs either the flame or the detonation wave of the initiator toward the detonator charge. The detonator, a larger amount of less sensitive but more powerful explosive material, then detonates either the booster charge or the main charge. The booster charge is an optional component that further magnifies the explosive impulse. The main explosive (or bursting) charge contains the largest amount of an insensitive, but powerful, explosive. Explosives used as booster and main charges are usually not capable of being initiated by impact, friction, or the brief application of heat, and are known as secondary explosives. Some common primary and secondary explosives are listed in Exhibit 9-1.

The secondary explosives used currently in most military explosive devices are physical mixtures of one or more high explosives with various additives (Figure 9-3). (The use of mixtures, rather than single compounds, provides for greater flexibility in explosive design, and additives extend the range of performance even further [Table 9-1]). *Melt-loading*, commonly used with TNT mixtures, is a process in which a molten explosive mixture is introduced into an empty shell casing and allowed to cool and harden. Secondary explosive mixtures are used to facilitate the melt-loading process to optimize (*a*) the oxygen balance of the explosive, (*b*) explosive characteristics such as blast and fragmentation, and (*c*) engineering criteria such as malleability and strength.

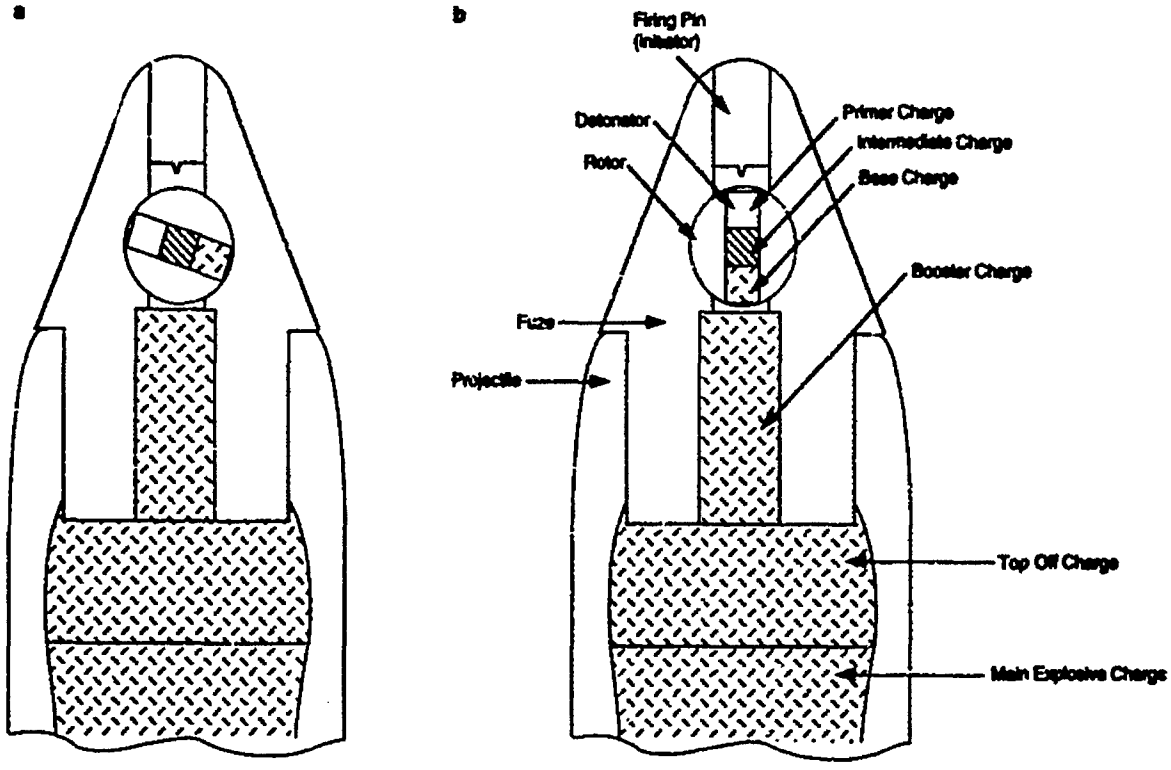


Fig. 9-2. These are diagrams of a typical high-explosive train. In (a) above, the rotor is turned in a safe position, interrupting the train. In (b), the unsafe, armed position, the rotor is turned, allowing geometric alignment of the various charges. On detonation, the stab firing pin strikes the input end of the detonator (initiation), piercing the thin metal disk and pushing it into the primer charge, which in turn initiates the intermediate charge. This initiation causes a reaction within the intermediate charge that is accelerated and converted to a small detonation. This small detonation is boosted within the booster charge and the train continues into the main explosive charge. Here the reaction is accelerated and converted to a detonation, which is successively transmitted through the remainder of the chain (boosting) to the main explosive charge. Reprinted from US Army Materiel Command. *Explosive Trains*. Washington, DC: USAMC; 1974: 1-3. AMC Pamphlet 706-179.

EXHIBIT 9-1

COMMON PRIMARY AND SECONDARY EXPLOSIVES

Primary Explosives		Secondary Explosives	
Lead azide	Diazodinitrophenol	Aliphatic nitrate esters	Ternary mixtures
Mercury fulminate	Potassium dinitrobenzofuroxane	Nitramines	Quaternary mixtures
Lead styphnate	Lead mononitroresorcinate	Nitroaromatics	Plastic-bonded explosives
Tetracene	Primary compositions	Binary mixtures	Industrial explosives

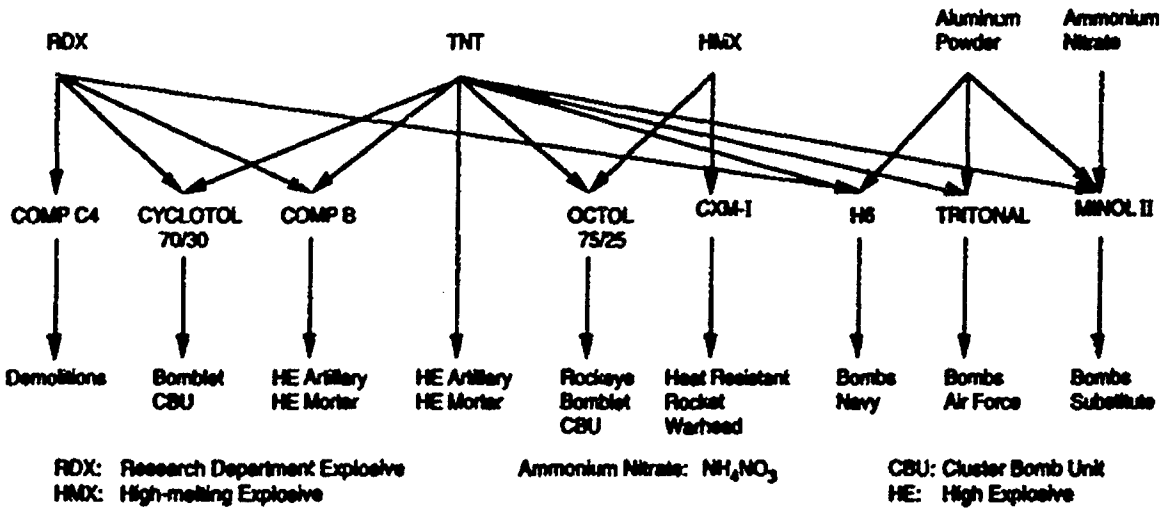


Fig. 9-3. Explosives family tree. Most explosives in military use are based on RDX, TNT, or both, in combination with other explosives. The occupational health practitioner needs to know the common names of the explosives being used at the installation to be able to ascertain the appropriate medical surveillance examinations. Adapted from US Army Environmental Hygiene Agency. *Water Pollution Aspects of Explosive Manufacturing*. Aberdeen Proving Ground, Md: USAEHA; 1985: 4. USAEHA Technical Guide 4.

Explosives and explosive-actuated devices are used widely in both industry and the military. Explosives are used in construction, mining, quarrying, demolition, metal forming, welding, and cladding. Explosive-actuated devices are used to drive turbines, move pistons, operate rocket vanes, start aircraft engines, eject pilots, and to provide heat. Between 1960 and 1975, domestic industrial explosive consumption increased from 500 million metric tons to over 1.4 billion metric tons. During this time, the use shifted from black powder, liquid oxygen, and dynamite to safer ammonium nitrate–based explosives. Today, over 100 million blasting caps are used annually in the United States.[4] Although the specific military uses of explosives are almost too numerous to count, they include the production of fragments, air blasts, and underwater shocks; the penetration of armor; demolition; the ejection of personnel from aircraft; and components of nuclear weapons.

Propellants

Propellants are explosive materials formulated and engineered to react at carefully controlled rates, producing a sustained pressure effect over a longer period of time than high explosives. In contrast to the detonation of high explosives, the process of propellant burning is referred to as *deflagration*, wherein the rate of heat transfer determines the rate of the reaction, which proceeds at subsonic speeds.

Like explosives, propellants utilize a series of materials in an ignition train (Figure 9-4). An electrical or mechanical impulse impinges on the sensitive primer material. This ignites the igniter, a pyrotechnic, which in turn ignites the main propellant grain.

Propellants may be formulated either as solids or as liquids. Solid propellants are used more frequently in guns, cannons, and smaller rockets, while liquid propellants are used in high-performance missile systems and other selected applications.

TABLE 9-1

TYPICAL EXPLOSIVE ADDITIVES

Additive	Purpose	Munitions or Other Use
Aluminum Pyropowder	Increase blast effect	Depth charges Cluster bombs Concrete fragmentation bombs
Wax Stearic Acid	Desensitize to explosion	Composition A Numerous others
Mononitrotoluene Dinitrotoluene	Explosive and plasticizer	Composition C
Polyisobutylene	Plasticizer	Composition CH6

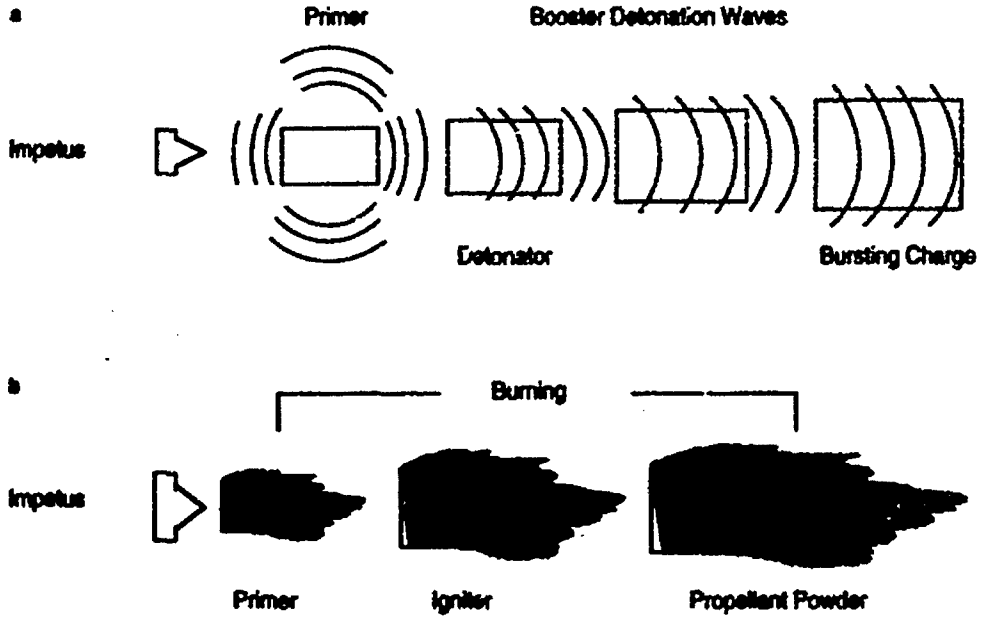


Fig. 9-4. Comparison of explosive train with ignition train. Although significant engineering differences exist between explosive trains (a) and ignition trains (b), in concept they are very similar. In both, a small electrical or mechanical stimulating impetus is magnified via a succession of intermediate charges to achieve optimum initiation of the main charge or propellant grain. The major difference between the two types of chains is in the component charges' rates of reaction. Adapted from US Department of the Army. *Military Explosives*. Washington, DC: DA; 1985. Technical Manual 9-1300-214, September 1984 with change 1 of 30 November 85.

Solid propellants may be classified by their chemical composition. Each class has unique properties that render it useful in certain applications. All solid propellants may contain additives similar to those used in explosive mixtures. The additives can be more toxic than the principal components of the propellant and must be considered in occupational-hazard analysis. Regardless of the composition class, the chief advantages of solid propellants include their compactness, safety, ease of storage, tolerance of temperature extremes, and ease of handling. In comparison, liquid propellant systems permit greater thrust control and deliver higher specific impulses. Liquid propellants have been limited to use in high-performance missile systems until recently, when research has focused on using liquid gun propellants for howitzers. Several candidate liquid gun propellants will be discussed later in this chapter.

Pyrotechnics

Pyrotechnic materials are relatively slow-burning, nonexplosive powders such as metals, alloys, and hydrocarbon mixtures. The only pyrotechnic compounds discussed in this chapter are those used in initiating compositions and propellants. However, pyrotechnics are also widely used in the military as flares, signals, relays, delays, and fuzes.

EXPOSURE

Ammunition plants, operated by the U.S. Army for the Department of Defense (DoD), are the primary sites of occupational exposure to military explosives. The types of ammunition plants include (*a*) propellant- and explosive-manufacturing plants, (*b*) metal-parts plants, (*c*) small-arms plants, and (*d*) shell loading, assembly, and packing (LAP) plants. Private companies have operated most of these ammunition plants under government contracts since the late 1940s.

In addition to those at ammunition plants, significant actions occur at other types of military facilities: munitions are manufactured (in limited quantities), tested, and stored at *arsenals*; tested at *proving grounds*; and maintained, stored, and demilitarized at *depots*.

Unique operations, with potentially hazardous exposures, are done at each type of facility. Workers at these facilities may perform duties that can potentially expose them not only to toxic hazards but also to other hazards of which occupational medicine physicians must always be cognizant.

Even though propellant and explosives manufacturing plants produce a limited number of specialized products, their workers can still be exposed to *feedstock, process chemicals,* and the finished explosives (Figure 9-5). Feedstock is a generic term for the raw materials that are used in chemical manufacturing. It can include chemicals such as toluene and nitric acid in the synthesis of TNT, and raw cellulose such as cotton or paper pulp for the synthesis of nitrocellulose. Process chemicals include all others in addition to the feedstock used in the synthesis (eg, salts or acids may be used in separations.) Exposures are usually controlled at the manufacturing plants by enclosing the *process streams,* which are flows (literal or figurative) of the partially processed feedstock through the additional chemical processes and reactions necessary to complete the synthesis.

LAP plants present the greatest exposure potential for employees, due to their use of a wide variety of explosive compounds and the labor-intensive nature of most loading operations. Comparatively few employees are exposed to explosives at small-arms plants, arsenals, or depots. Because ammunition is *proof-fired* there (ie, a batch of explosive is ignited to "prove" that it works and to assess its particular performance characteristics), small numbers of employees at proving grounds may be exposed to many and varied explosives and combustion products. Workers at all these facilities, especially *metal-parts plants,* can be exposed to common industrial chemicals such as carbon monoxide, lead, nitrogen oxides, solvents, paints, and cutting oils.[2] Metal-parts plants manufacture the hardware in which the explosives are loaded and used, such as rocket tubes, shell casings, bomb casings, and trigger assemblies. Cutting oils (usually mineral oil) are used to lubricate and cool the saws and machining tools that are used to shape the metal parts. During the past decade, cutting oils have been found to be contaminated with nitrosamines, a class of potent carcinogens. Machinists exposed to these oils via the dermal and inhalational routes may be at high risk for cancer.

Exposure Controls

Several types of workplace standards have been established to regulate employee exposure. U.S. Army

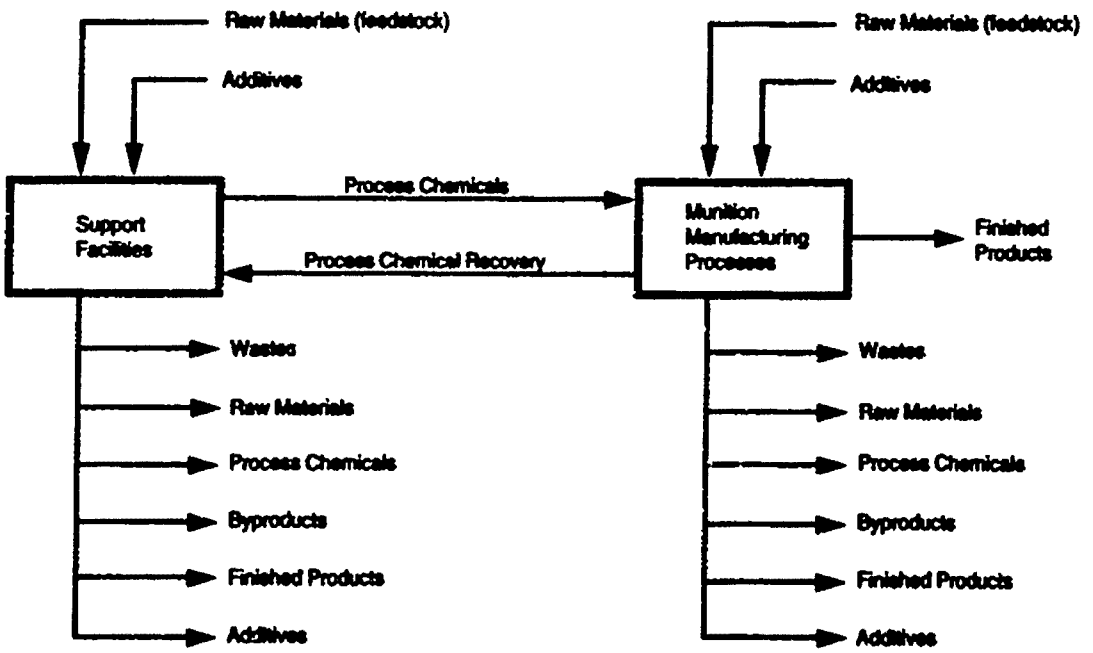


Fig. 9-5. Simplified flow for munition manufacturing processes. Adapted from US Army Environmental Hygiene Agency. *Water Pollution Aspects of Explosive Manufacturing.* Aberdeen Proving Ground, Md: USAEHA; 1985: 5. USAEHA Technical Guide 140.

policy follows the most stringent limit elaborated by OSHA, the permissible exposure limit (PEL); or the American Conference of Governmental Industrial Hygienists (ACGIH), the Threshold Limit Value (TLV). In addition, because dermal absorption is a significant route of exposure for many explosives, OSHA has given a skin designation to those chemicals, to aid in their identification. Therefore, skin exposure to chemicals with significant dermal absorption should be reduced to a minimum. However, where published limits from OSHA and the ACGIH[9] are either unavailable or inadequate to meet army requirements, the U.S. Army Medical Department establishes army-unique exposure limits. Table 9-2 shows the published limits for the more common explosive materials.

General Safety Practices

Safety is of paramount importance when personnel handle explosives and propellants. The accidental detonation or deflagration of these materials poses serious hazards to employees, other people nearby, and property, including blast overpressure, fragmentation, and burns. Creating a safe workplace around explosives demands that the cardinal principles of safety be followed:

- Separate each handling operation to prevent fires, blasts, or fragmentation.
- Use the minimum number of personnel for each operation.

TABLE 9-2

EXPOSURE LIMITS

Chemical Name	CAS Registry No.	Skin Designation	TWA (8 h)	STEL (15 min)
Nitroglycerin[1]	55-63-0	+	—	0.1 mg/m^3
Nitrocellulose[1]	9004-70-0	–	15 mg/m^3* 5 mg/m^3†	—
PETN[2]	78-11-5	+	—	0.1 mg/m^3
PGDN[1]	6423-43-4	–	0.3 mg/m^3	—
TNT[1]	118-96-7	+	0.5 mg/m^3	—
DNT (2,4-DNT)[1] (2,6-DNT)[1]	121-14-2 606-20-2	+	1.5 mg/m^3	—
Ammonium Picrate[2]	131-74-8	+	0.1 mg/m^3	0.3 mg/m^3
Picric Acid[1]	88-89-1	+	0.1 mg/m^3	—
RDX[1]	121-82-4	+	1.5 mg/m^3	—
HMX[1]	2691-41-0	+	1.5 mg/m^3	—
Nitroguanidine[3]	556-88-7	–	4 mg/m^3*	—
Tetryl[1]	479-45-8	+	1.5 mg/m^3	—
Lead Azide[1]	13424-46-9	–	50 µg/m^3‡	—
Lead Styphnate[1]	63918-97-8	–	50 µg/m^3‡	—
Mercury Fulminate[1]	628-86-4	+	—	0.1 mg/m^3 (as mercury)

[1]OSHA, 29 CFR 1910.1000; [2]US Army Environmental Hygiene Agency. *Medical Information Module of the Occupational Health Management Information System*. Aberdeen Proving Ground, Md: OHMIS, USAEHA; 1988; [3]Unpublished data. Aberdeen Proving Ground, Md: USAEHA.
*Total dust; †Respirable dust; ‡Measured as lead
CAS: Chemical Abstract Society; STEL: short-term exposure limit, defined as a 15-minute exposure level; TWA: the 8-hour time-weighted average exposure level; RDX: research department explosive, hexahydro-1,3,5-trinitro-1,3,5-triazine; HMX: high-melting explosive, octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine

Fig. 9-6. This photograph was made at New River Ordnance Plant, Dublin, Virginia, on 30 September 1941. The first step in the manufacture of powder bags is to lay the material on the table, mark for the size of the bag, and then cut with an electric knife. Numbers are then printed on the cloth to designate the charge that is to be put into the bag. The pieces of cloth are then sewn together to make the bag. The last step, shown here, is to weigh the charge of smokeless powder, fill the bag, and then seal it. The numbers on the bags are very large to prevent any error. Note the administrative safety control measures in place in this pre-World War II army public relations photograph: the number of personnel allowed in the area (8 operators, 2 transients) and the limits on the amount of explosives allowed to be used in the operation (140 lb of smokeless powder, 25 explosive charges in the chute). Photograph: Courtesy of the US Army Bureau of Public Relations.

- Stockpile only the minimum amount of explosive or hazardous material necessary for efficient operation.

The DoD has established uniform safety standards applicable to ammunition and explosives,[10] which the army implemented in Army Regulation 385-64.[11] Most of these are safety standards, which address factors including the sensitivity of explosive materials to accidental initiation; the quantity of material available to be detonated or deflagrated; the heat that would be generated; the rate of burning; the potential sources of accidental ignition and initiation; and the protection capabilities of shields, clothing, and fire-protection systems (Figure 9-6). Other health-focused standards address the potential toxicity of the explosive materials and the control measures that must be in place to ensure that worker exposure is within acceptable limits.

Industrial Hygiene Principles

Applying industrial hygiene principles such as (*a*) engineering controls, (*b*) administrative controls, and (*c*) personal protective equipment (PPE) in the workplace will further limit the potential for workers being exposed.

Engineering Controls

The ideal control of an industrial hazard is achieved through design changes such as substituting a safer or less-toxic process or material. Any such modification of the workplace should be closely coordinated between qualified industrial hygiene and safety personnel. But substitution is a long-term solution, and may not always be possible. For example, the U.S. Army Armament Research, Development, and Engineering

Fig. 9-7. This photograph is dated April 1942. A worker is preparing to dump a box of bulk TNT into the melt unit, through which steam is passed. The liquified TNT will then be poured into shells and bombs. The worker wears special nonsparking safety shoes made without nails. Photograph: Courtesy of the Ordnance Department, Signal Corps College; Ordnance Center, Picatinny Arsenal, New Jersey.

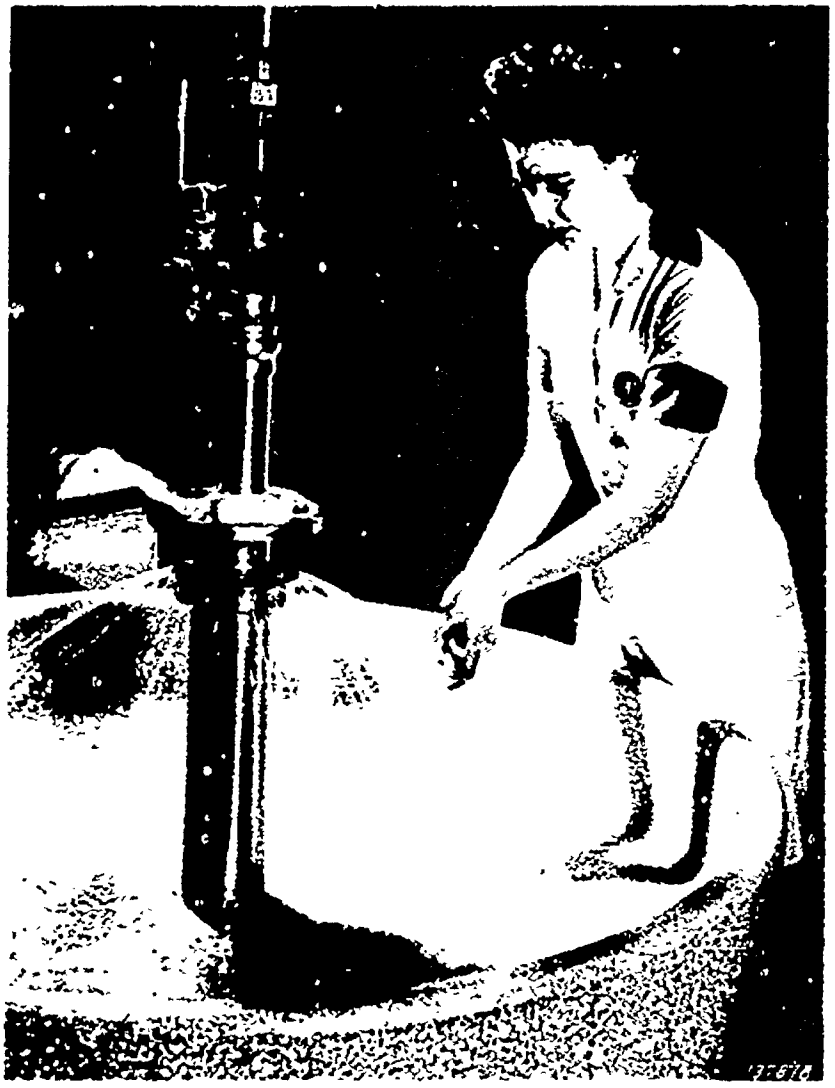


Fig. 9-8. This photograph was made at Wolf Creek Ordnance Plant, Milan, Tennessee, in June 1942. The ordnance worker is shown washing her hands after exposure to TNT, to prevent dermatosis. Photograph, Courtesy of the World War II Signal Corps College, *War Production* 7-12

Center at Picatinny Arsenal, New Jersey, is currently endeavoring to find a substitute for dinitrotoluene (DNT), which is both toxic to humans and mutagenic in animal systems. It has been classified as a suspect carcinogen.[12] Finding an appropriate substitute is expected to take about 5 years.

Some controls proved their worth during World War II and have endured the test of time. Some of those methods, still in use today, include

- enclosure of processes—the melt unit used in TNT melt-loading operations (Figure 9-7);
- general exhaust ventilation—the type used in rooms where poured TNT munitions are cooling;
- local exhaust ventilation—the type used in dusty operations such as sieving (known as *screening* in the chemical industry) flaked DNT or TNT;
- temperature control to reduce vapor generation—the type used in rolling operations with propellants containing nitroglycerin; and
- remote-controlled operations—the modern continuous-flow nitrators used to produce nitroglycerin.[13]

Administrative Controls

Since World War I, administrative controls have consistently emphasized work and sanitation practices (Figure 9-8). Today's administrative controls encompass more than just rotating employees in and out of areas with high exposure potentials; they include such essential measures as (*a*) educating workers about the toxic and safety hazards of the materials with which they work; (*b*) enforcing strict work-practice guidelines to minimize the generation of dusts and vapors, and to prevent dermal contact; (*c*) initiating appropriate sanitation practices, including paying strict attention to the waste-explosive contamination of workers' bodies and clothing; and (*d*) providing the workers' changing and shower rooms with separate locker facilities, to segregate their street from their work clothing. In addition, contaminated clothing should be removed immediately and placed in closed containers for storage, until it can be laundered or discarded properly. Contaminated skin should be washed promptly with soap and water. Furthermore, a worker who handles these toxic compounds should wash his or her face, hands, and forearms thoroughly with soap and water before eating, drinking, smoking, or using toilet facilities.[14] In keeping with these concepts, the following should also be prohibited in the work area: storing, preparing, dispensing, or consuming food or beverages; storing or applying cosmetics; storing or smoking tobacco products; and storing or using products for chewing, such as gum.

Personal Protective Equipment

PPE to control exposure should be used only when engineering and administrative controls are inadequate. During the world wars, the use of change houses and the wearing of coveralls became widespread. In addition, a great deal of attention was directed to the use of barrier creams to protect against the dermatitis and systemic toxicity associated with explosives such as trinitrophenylmethylnitramine (tetryl) and TNT. These creams have subsequently fallen into disfavor; their efficacy is limited and, increasingly, the emphasis is on exposure controls.

Appropriate respiratory PPE and gloves must be used where indicated; the National Institute of Occupational Safety and Health (NIOSH) has published guidance on the types needed.[12,15] The need for close coordination between safety and health personnel is emphasized by OSHA's position that the respiratory equipment supplied may itself create safety hazards in explosives manufacturing operations.[9] For example, some respirators, especially those with air supplied (by a tank, or a compressor and hose) can create sparks and therefore pose an unacceptable risk of igniting an explosion. In this instance, protecting the worker from a health hazard would compromise overall safety from explosion.

GENERAL MEDICAL CONSIDERATIONS

The greatest challenge facing any physician beginning to work in an industrial environment is to understand the hazards faced by employees in that industry. Military ammunition plants are no exception: each type of projectile and munition contains a unique combination of explosives. A carefully elicited occupational history might reveal, for example, that a worker is exposed to amatol or composition B. The physician must be able to interpret this information in terms of specific chemical exposures, just as he or she would interpret chemical trade names in the civilian sector. Those who work with composition C4 should be assessed for RDX (research department explosive, hexahydro-1,3,5-trinitro-1,3,5-triazine) toxicity, or given medical surveillance for RDX; cyclotol workers should be assessed for both TNT and RDX toxicity; amatol workers should be assessed for both TNT and ammonium nitrate toxicity; and double-base propellant workers should be assessed for both nitroglycerin and nitrocellulose toxicity. Sources of information include Material Safety Data Sheets, military specifications, and the model designations of specific ammunition items. Often the best information is available from the safety officer, industrial hygienist, or plant commander.

Preplacement Considerations

Preplacement examinations have been used by some industries as a measure to control costs rather than as a tool for maintaining worker health. As a consequence, professional organizations including the American Medical Association and the American College of Occupational and Environmental Medicine have made numerous, well-publicized comments on the appropriate, ethical use of these examinations. The Americans with Disability Act of 1990[16] precludes preemployment examinations from being applied as

discriminatory tools and requires that they be used only to assess critical aspects of job performance.

Despite this controversy, preplacement medical examinations remain part of the foundation of a medical surveillance program for workers exposed to hazardous agents. Medical surveillance should be performed primarily for the benefit of the individual employee and his or her immediate coworkers and should not be used for hiring and firing purposes.

Preplacement examinations are done to (*a*) identify preexisting conditions, (*b*) identify hypersusceptible individuals, and (*c*) establish preexposure baseline values. Preplacement examinations must identify preexisting conditions to ensure the worker's safe performance of critical job tasks. For example, blindness would preclude a worker's being assigned as a forklift operator. Similarly, certain neurobehavioral conditions such as epilepsy and severe psychiatric disorders may not be appropriate among explosives workers.[17] In addition, hypersusceptible individuals must be identified because they may be at higher risk for developing diseases related to specific occupational exposure. For example, individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency may have a hemolytic crisis when they are exposed to methemoglobin-inducing agents.

Preplacement examinations also establish preexposure baseline values for screening programs. For example, erythrocyte counts and liver-function tests may be appropriate for workers who are exposed to agents that are capable of inducing anemia or hepatotoxicity.

Acute Exposure Decontamination

The first-aid measures and treatment procedures for individuals who have been exposed to explosives and propellants are similar to those for exposure to other toxic substances. The rescue procedures follow those dictated for most emergencies, but the rescuers should try to prevent additional casualties among would-be rescuers. The main goals of initial treatment are to prevent further absorption and enhance excretion, which may be achieved by first removing the victim from exposure and then removing the contaminated clothing. Rescuers should thoroughly cleanse the skin with soap and copious quantities of water, paying attention to the hair and nails. Eyewash fountains should also be placed throughout the workplace to provide copious irrigation of the eyes in the event of a splash. Contaminated clothing should be either laundered carefully or discarded. The treatment of mild, asymptomatic cases may require nothing more than removal from exposure and decontamination.

COMMON MANIFESTATIONS OF EXPOSURE

Commonly, organic nitrates share these major toxic effects: dermal sensitization, methemoglobinemia, vasodilation, and carcinogenesis. Nitrates used in explosives are no exception. Each of these effects can occur separately or in combination; however, not every organic nitrate causes all four effects, at least according to current information. The prevalence of each of these effects varies with the specific chemical. For example, tetryl causes dermal sensitization almost exclusively, nitroglycerin causes vasodilation, and DNT is a mutagen and probable carcinogen. Many organic nitrates are potent vasodilators, and a few have found therapeutic uses in clinical medicine.

Dermatitis

Dermal sensitization refers to the induction of an allergic reaction via cutaneous exposure to a chemical. It manifests as allergic contact dermatitis. The other major occupational dermatitis is irritant contact dermatitis, which is a nonallergic reaction of skin exposed to a chemical. The immune system is not involved in irritant contact dermatitis, but is involved in the allergic form. Dermatitides caused by exposure to organonitrates have no pathognomonic characteristics to distinguish them from other irritant or allergic reactions.

Since World War I, both allergic and irritant contact dermatitides have been the most common toxic effects seen in explosives workers.[18] The agents most responsible are tetryl, TNT, amatol, ammonium picrate, picric acid, and mercury fulminate. However, the role of other ingredients and exposures must not be overlooked: industrial exposures to solvents, cutting oils, and degreasers all occur in the munitions industry and can also induce dermatitis.

Occupationally induced dermatitis (from all occupational exposures collectively) is considered to be the most prevalent occupational disease but it rarely causes any mortality. During World War I and World War II, however, morbidity from tetryl and TNT dermatitides was a major cause of time lost from work. Fortunately, these effects resolve after the worker has been removed from exposure, and they generally do not sensitize the individual to other chemicals.

Methemoglobinemia

Methemoglobinemia has been recognized as an adverse occupational effect since the 1800s, when coal-tar derivatives were introduced into the explosives and dye industries. Many drugs and chemicals exert an oxidant stress on hemoglobin, which oxidizes the iron in the heme portion of the molecule from the ferrous to the ferric form, thus rendering the hemoglobin molecule incapable of binding oxygen. The body spontaneously produces small amounts of methemoglobin, but enzymatic reducing systems within the erythrocyte normally maintain that concentration below 1% of the total hemoglobin. Clinical effects of methemoglobinemia may develop when more than 10% to 15% of the total hemoglobin is converted to methemoglobin. The acute signs and symptoms of methemoglobinemia include persistent, slate-gray cyanosis (the most readily apparent sign); fatigability; malaise; headache; and reddish-brown discoloration of the peripheral blood, which does not become bright red when exposed to oxygen. Massive exposure may cause 60% to 70% of the hemoglobin to convert to methemoglobin, which can produce collapse, coma, and death.

Chemicals that induce methemoglobinemia also tend to cause chronic anemia, which may develop insidiously even in the absence of cyanosis.[17] This anemia usually occurs when erythrocytes that contain methemoglobin hemolyze.

As they do with many toxic exposures, individuals have a wide range of sensitivity to methemoglobin-inducing chemicals. For example, individuals with G6PD deficiency and other hemoglobinopathies are uniquely sensitive to the hemolytic effects of exposure to these agents. Preemployment screening should identify individuals with G6PD deficiency and sickle-cell trait. Aggressive medical surveillance of workers at high risk has effectively reduced such exposures and health effects. Methemoglobin can be measured directly, but this must occur within just a few hours of sample collection because methemoglobin in erythrocytes reduces rapidly to hemoglobin. All cases of cyanosis and abnormal blood findings should trigger exposure-control action.[17]

Mild-to-moderate cases of methemoglobinemia will recover spontaneously within 2 to 3 days. In more severe symptomatic cases, methylene blue (administered intravenously as a 1% solution in saline at 1–2 mg/kg over 10 min) is an effective therapy. A second dose may be administered after 1 hour, if necessary.[19,20] Although hyperbaric oxygen has been advocated as a therapy, other authorities have not found it to be efficacious.[21,22]

Vasodilation and Carcinogenesis

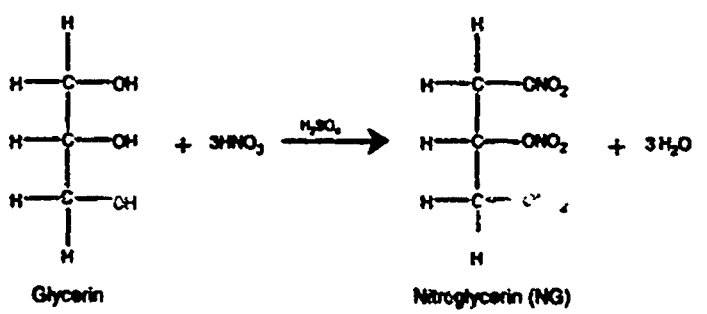
Although organic nitrates as a class cause both dermatological and hematological effects, specific explosives such as nitroglycerin and DNT are vasodilatory and mutagenic, respectively. These substance-unique effects are discussed in their specific chemistry sections, which follow.

THE ALIPHATIC NITRATE ESTERS

The aliphatic nitrate ester class of compounds includes many members with explosive properties, some of which are militarily significant (Figure 9-9). With the exception of nitrocellulose, members of this class are manufactured similarly and are similarly toxic. The physical properties and uses of the individual compounds vary, as does the amount of toxicological data available.

Nitroglycerin

Nitroglycerin was the first organic nitrate to be used



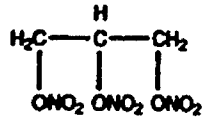
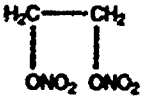
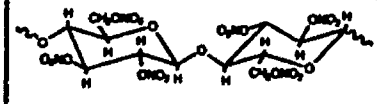
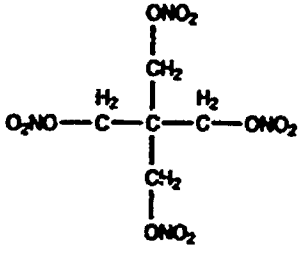
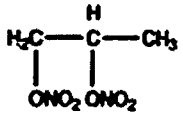
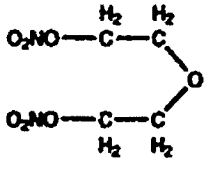
Common Name	CAS Registry Number	Synonyms	Formula	Structure
Nitroglycerin	55-63-0	1,2,3-Propanetriol trinitrate Glyceryl trinitrate Trinitroglycerin NG	$C_3H_5N_3O_9$	
Ethylene Glycol Dinitrate	628-96-6	Nitroglycol Glycol dinitrate EGDN	$C_2H_4N_2O_6$	
Nitrocellulose	9004-70-0	Cellulose nitrate Guncotton Collodion NC	$C_{12}H_{14}(ONO_2)_6O_4$	
Pentaerythritol Tetranitrate	78-11-5	2,2-Bis[(nitroxy)-methyl]-1,3-propanediol dinitrate Penthrite Nitropenta TEN PETN	$C_5H_8N_4O_{12}$	
Propylene Glycol Dinitrate	6423-43-4	1,2-Propylene glycol dinitrate 1,2-Propanediol dinitrate Propylene glycol 1,2-dinitrate PGDN	$C_3H_6N_2O_6$	
Diethyleneglycol Dinitrate	693-21-0	2,2'-Oxy-bis-ethanol dinitrate Dinitrodiglycol DEGDN DEGN	$C_4H_8N_2O_7$	

Fig. 9-9. The aliphatic nitrate explosives, together with their common names, Chemical Abstract Society numbers, synonyms, formulae, and structures. The reader can compare the similarities and subtle differences in formulae and structures among the compounds in this group. Nitrocellulose is a variable-length chain consisting of repeated $C_6H_7(ONO_2)_3O_2$ units.

as an explosive. Although Ascanio Sobrero, an Italian chemist, first synthesized nitroglycerin in 1847, it was not widely appreciated until 1863, when Alfred Nobel began to use it as a blasting compound.[8] To make nitroglycerin safer to work with, Nobel began using solid materials to adsorb liquid nitroglycerin, from which *dynamites* were formed. (There are many formulations for dynamite, using different liquid or gelatinous explosives on a matrix of various solid materials.)

In 1888, Nobel demonstrated that, by using nitroglycerin to gelatinize nitrocellulose, the explosive properties of nitroglycerin could be converted to propellant uses; as a result, he developed not only the earliest of the smokeless powders but also the double-base propellants. Until then, all propellants had nitrocellulose alone as their explosive component—now called single-base propellants. Double-base propellants are those with nitroglycerin in addition to nitrocellulose. Triple-base propellants have nitroguanidine included as the third explosive component.[9] Military

TABLE 9-3

MILITARY USES OF SOLID PROPELLANTS

Class	Ingredients	Uses
Single Base (Primary)	Nitrocellulose	Howitzers Small arms Grenades
Double Base (Secondary)	Nitrocellulose Liquid nitrate ester	Howitzers Small arms Mortars Rockets Jet-propulsion units
Triple Base (Ternary)	Nitroguanidine Nitrocellulose Liquid nitrate ester	Howitzers
Composites	Physical mixture of fuel, binder and inorganic oxidizer	Rocket assemblies Jet-propulsion units

use of nitroglycerin is almost exclusively in combination with nitrocellulose as double- and triple-base propellants (Table 9-3).

The freezing point of nitroglycerin (13°C) caused a major safety problem with the early dynamites.[24] Explosions were not uncommon when munitions or dynamite were accidentally frozen during winter. Nitroglycerin in the solid state is much less sensitive than in the liquid. But while thawing, nitroglycerin is much more sensitive to detonation than while either a solid or a liquid. Decomposed nitroglycerin is especially dangerous. Not only is it more sensitive to accidental detonation than when pure, but the formation of nitrogen oxides may also constitute a separate toxicity hazard.[5] However, because the military use of nitroglycerin is limited almost exclusively to the double- and triple-base propellants, which are stable colloidal mixtures with lower freezing points, the instability of nitroglycerin at its freezing point is not a problem.

Other aliphatic nitrate esters have limited, specialized uses. In 1905, ethylene glycol dinitrate (EGDN, freezing point -8°F) was introduced as an additive to lower the freezing point of nitroglycerin, and since 1920, EGDN has been a major component of most civilian dynamite formulations.[24] EGDN has little current military use. However, another of the aliphatic nitrate esters, propylene glycol dinitrate (PGDN), is used as a torpedo propellant.[4]

Manufacture and Exposure Hazards

Nitroglycerin is manufactured by one of three *closed, continuous-flow* processes known as the Biazzi, Schmid-Meissner, and the Nobel-Nitrator processes, in which glycerin is mixed with concentrated nitric acid (Figure 9-10).[4] A closed process is one in which liquid chemicals are piped from one closed container to another—from the beginning of the process where feedstock is introduced to the end where finished product is packed for shipping or storage. A continuous-flow process is one in which the reactions occur constantly, not in batches. The product is subjected to a series of purifying washes and then transported by gravity flow to storage tanks. The nitration and purification processes—controlled remotely via closed-circuit television—are conducted in small, heavily revetted buildings. The other liquid aliphatic nitrates may be prepared by similar methods using other aliphatic polyols instead of glycerin.

Liquid nitroglycerin, together with nitrocellulose and other ingredients, is manufactured into double- and triple-base propellants by two methods.[4] In general, the *solvent process* is used for propellants that contain less than 40% nitroglycerin, while the *solventless process* is used for compositions that contain more than 40% nitroglycerin.

The solvent process begins with the addition of a solvent such as ether or acetone to water-wet nitrocellulose in a dough-type mixer (Figure 9-11). Nitroglycerin and other ingredients are added and mixed until a dry colloid forms. The mixture is then subjected to a series of presses to remove the solvent and complete the colloiding process. The first type of press is a hydraulic *blocking* press, which simply squeezes the liquid from the nitrocellulose mixture; next is the *macaroni* press, which improves the colloiding of the nitrocellulose with the nitroglycerin. Finally, the mixture is extruded through a die, cut to length, and dried in an oven to form the finished propellant.

The solventless process begins with mixing a slurry of nitrocellulose and nitroglycerin in a tank of water (Figure 9-12). Other ingredients are added, and the excess water is removed by centrifugation. The resulting paste is dried further, and any remaining ingredients are added. Repeated rolling between heated rollers removes the remainder of the water and completes the mixture's colloiding. The process is completed by extruding the dried colloid through a die and drying in an oven.

Occupational exposure to nitroglycerin can occur during any of these operations. In the solventless process, dermal exposure is especially significant

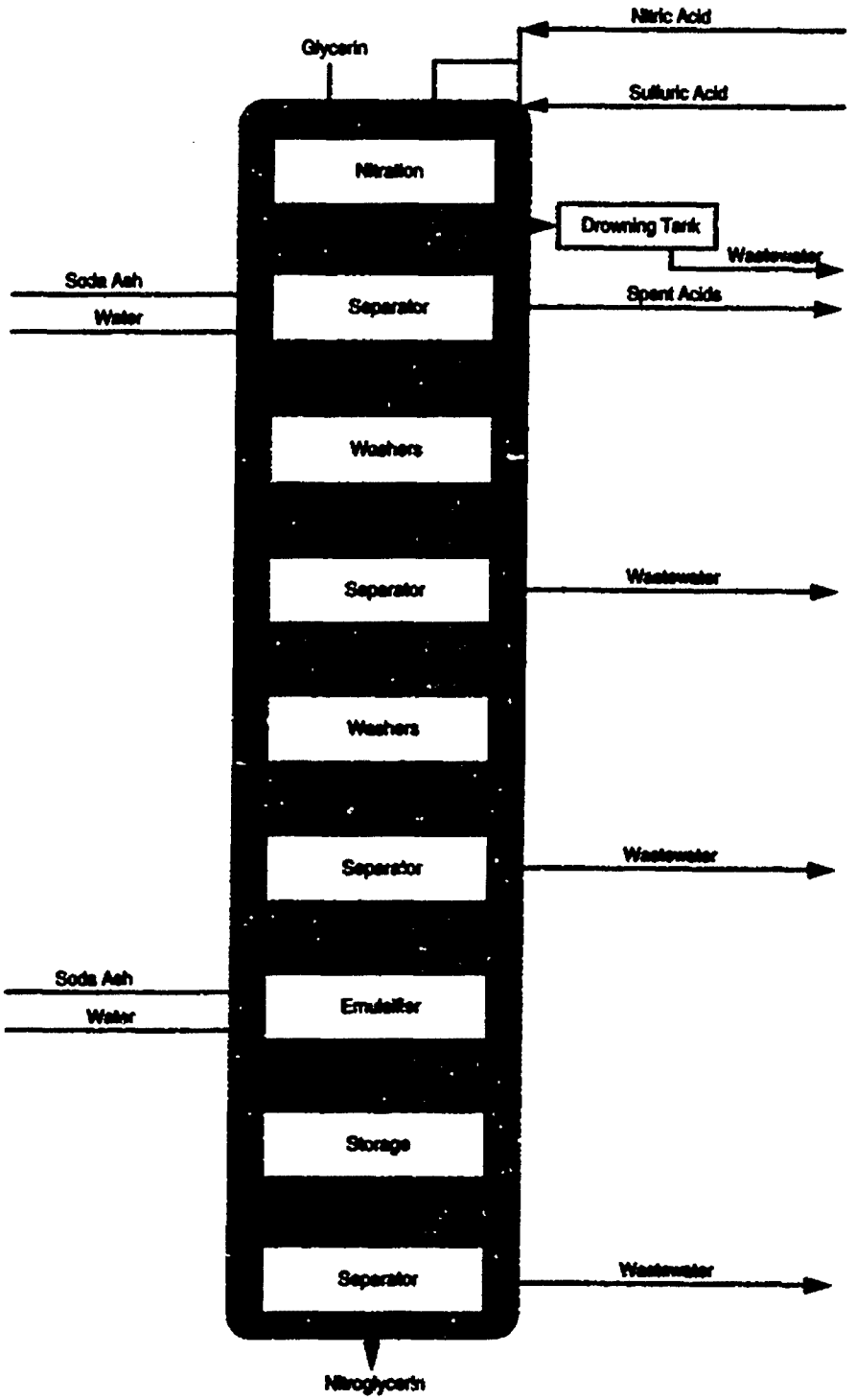


Fig. 9-18. In the Biazzi (continuous-flow) method of manufacturing nitroglycerin, all processes except the drowning tank are completely enclosed within stainless steel reaction vessels, which are connected by stainless steel pipes. Remote control is accomplished via gauges and closed-circuit television. Even the raw materials, wastewater, and nitroglycerin are piped into and out of the process, further limiting potential exposures of the workers. Adapted from US Army Environmental Hygiene Agency. *Water Pollution Aspects of Explosive Manufacturing*. Aberdeen Proving Ground, Md: USAEHA; 1985: 42. USAEHA Technical Guide 140.

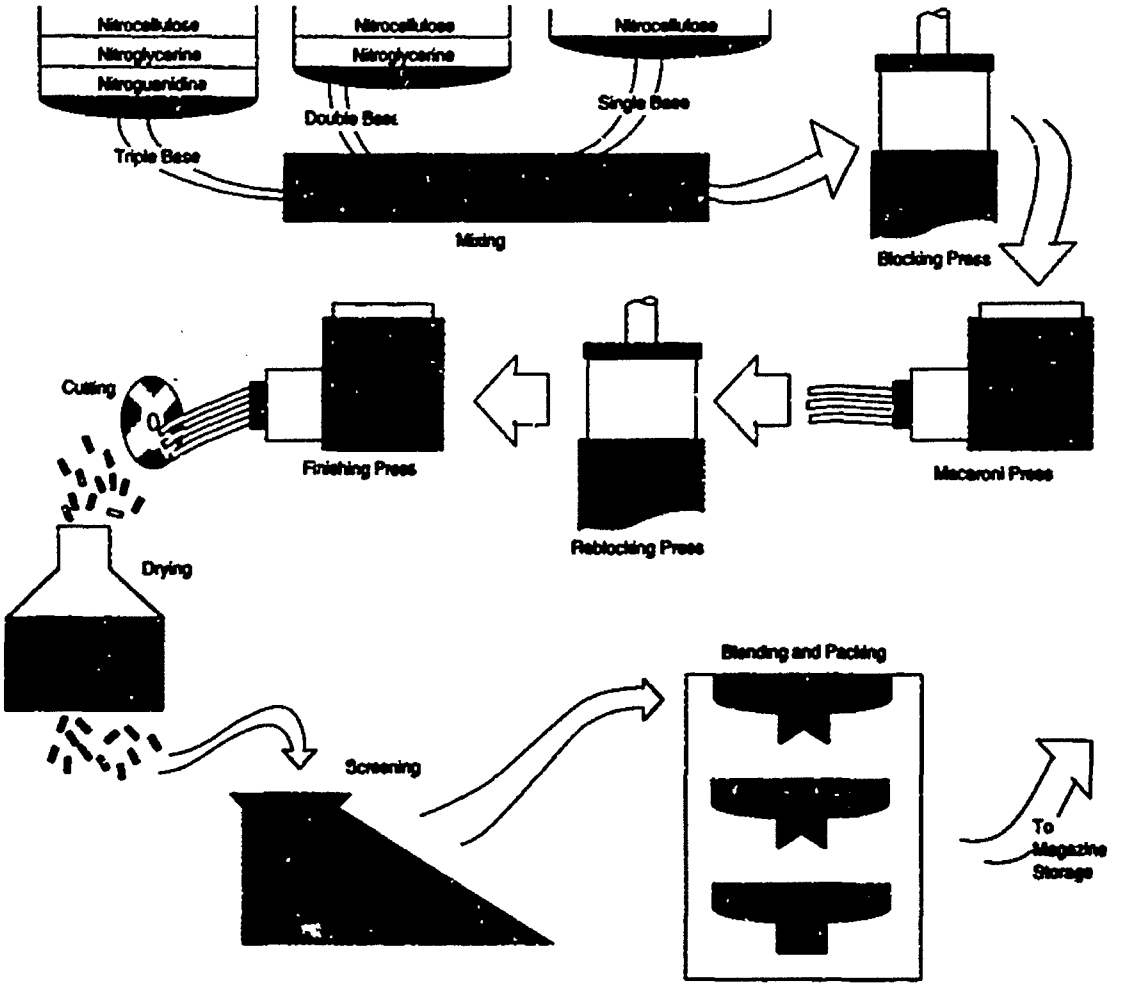


Fig. 9-11. In the solvent-based process of propellant production, the initial mixing is conducted in an alcohol-ether solution, which is then removed via the series of presses. Most production plants prepare these propellants in batches, and utilize manual labor at each step. Reprinted with permission from Chemical Propulsion Information Agency. Gun propellant process operations. In: *Hazards of Chemical Rockets and Propellants*. Vol 2, *Solid Propellants and Ingredients*. Laurel, Md: CPIA; 1985: 3-15. CPIA Publication 394.

among roller-press operators, and can be demonstrated: simultaneously collected samples will have higher concentrations of nitroglycerin in blood drawn from the cubital than the femoral veins. Due to the widespread use of engineering controls, exposure to vapors is minor during nitration, but inhalational exposure can be significant for press operators and drying-room attendants. During World War II, nitroglycerin toxicity caused at least 78 reported cases of lost time among propellant workers, several of whom required transfers to different work sites.[2] Almost certainly, other cases of nitroglycerin toxicity (including Monday morning angina, which is discussed below) occurred during World War II, but they either went unreported, were unrecognized, or did not result in time lost from work.

Human Exposure and Health Effects

The effects on human health from exposure to nitroglycerin have been observed since its discovery. Because of its vasodilating properties, nitroglycerin has been a mainstay of antianginal therapy since it was introduced to medicine in 1879. Reports of the effects that appeared in nitroglycerin workers and their families were described in the literature as early as 1890; yet consensus still has not been reached on all of the effects.[25-28] Contention still surrounds the chronic effects such as withdrawal syndrome and sudden death. Little epidemiological study has been done on these effects in humans, and consequently little proof exists of the chronic effects. The acute effects are so

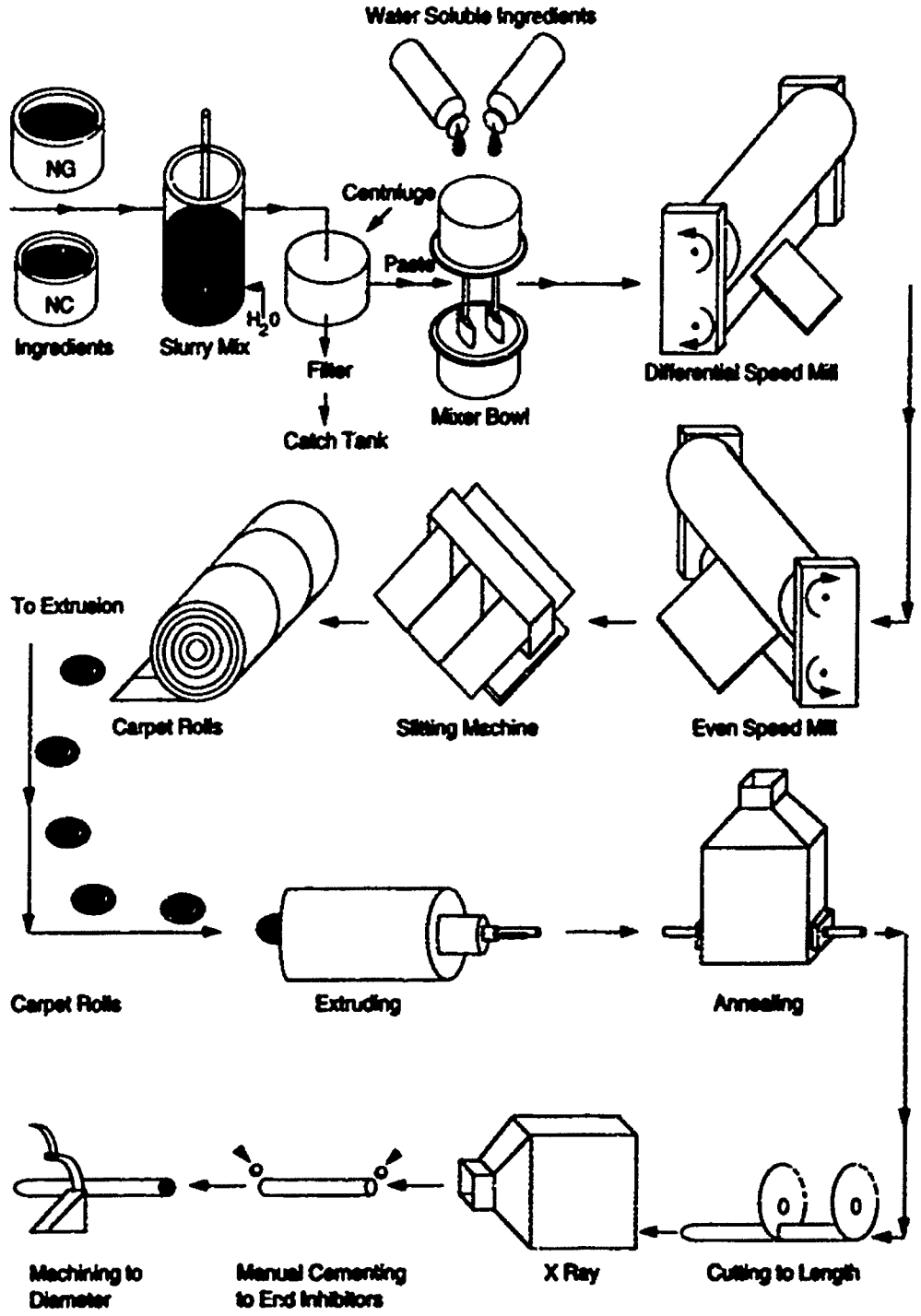


Fig. 9-12. In contrast to the solvent-based process, the solventless process uses no extraneous solvents during mixing. Nitroglycerin (or, less often, another liquid aliphatic nitrate such as EGDN or DEGN) is used to form a colloid with the nitrocellulose. Dissolution does not occur, but rather, the mixing and milling achieve a uniform colloid of the explosives and additives. Milling and extrusion are usually done at warm temperatures, and there is significant exposure potential for inhalation and dermal absorption of nitroglycerin. Reprinted with permission from Chemical Propulsion Information Agency. Gun propellant process operations. In: *Hazards of Chemical Rockets and Propellants*. Vol 2, *Solid Propellants and Ingredients*. Laurel, Md: CPIA; 1985: 3-16-3-17. CPIA Publication 394.

dramatic that our attention is frequently focused on the short term, to the neglect of the long.

Toxicokinetics. The toxicokinetics of nitroglycerin have been studied and reviewed intensively.[29-33] Nitroglycerin is readily absorbed through intact skin, as well as via the respiratory and gastrointestinal tracts. Vascular-tissue uptake and local metabolism are extensive, thus explaining the rapid systemic clearance of nitroglycerin. Once nitroglycerin is absorbed, it is rapidly metabolized by hydrolysis and glutathione-dependent organic nitrate ester reductase.

Because of their rapid hydrolyses, nitroglycerin and the other aliphatic nitrates have shorter biological half-lives than other classes of explosives.[24,33] The variations among individuals in their sensitivity, plasma levels, time of onset of symptoms, and duration of effects are extremely wide. Metabolites may alter the toxicokinetics of the parent compound during chronic dosing.[34]

Acute Effects. Acute or intermittent exposure to nitroglycerin may cause a constellation of symptoms in sensitive individuals. Vasodilatory effects can occur with inhalation of airborne concentrations as low as 0.1 mg/m^3. Most of these symptoms are due to direct vasodilation of the meningeal, cutaneous, and systemic blood vessels. Symptoms due to vasodilation include headache, dizziness, nausea, palpitations, hypotension, flushing, and abdominal pain. Other effects of acute exposure appear to be mediated by other mechanisms and include methemoglobinemia, reflex tachycardia, and increased respiratory rate. Hyperthyroidism has been reported to potentiate the acute toxicity of the organic nitrates.[27]

Inhalation exposure at levels as low as 14 mg/m^3 have led to more severe effects such as electrocardiogram (ECG) changes, chest pain, and palpitation. Massive acute exposure may cause cyanosis, coma, and death.

Other acute effects have been described, but they are less well documented. Central nervous system (CNS) symptoms such as confusion and hallucinations and psychotic episodes such as homicidal violence have been reported in patients after they have handled nitroglycerin. Peripheral nervous system effects such as paresthesias have also been reported.

Ingestion of nitroglycerin is not an industrial problem. It could theoretically occur via contamination of food or smoking materials in the workplace, but this has never posed a practical problem in manufacturing settings. Of course, the use of sublingual nitrates is a common form of therapy for coronary artery disease, taking advantage of the transdermal and transmucosal absorption and the vasodilatory effect of some nitrates.

Chronic Effects. Most workers become tolerant to the vasodilatory effects of nitroglycerin within 1 week after their exposure has begun and develop compensatory vasoconstriction. This effect has also been described in patients who receive therapeutic nitroglycerin.[34] The tolerance persists for approximately 1 week after the worker is removed from the exposure.

Workers who have become tolerant to the vasodilatory effects of nitroglycerin may experience a withdrawal syndrome if their exposure is terminated abruptly. This withdrawal may precipitate angina pectoris, myocardial infarction, and sudden death. The condition has been called Monday morning angina because the symptoms appear after a 48- to 72-hour absence from work. Anecdotal reports of these effects have appeared since the early 1900s, but the first medical case series was reported in 1952.[35]

The mechanism associated with angina and sudden death appears to be a series of events starting with habituation to the hypotensive effects of chronic nitrate exposure. When removed from exposure, the employee develops rebound hypertension, which may be followed by coronary insufficiency.[17] Coronary insufficiency is, therefore, a secondary effect due to rebound coronary vasoconstriction, making the heart less able to compensate for the additional strain caused by systemic hypertension. Studies done with animals have shown that nitroglycerin-tolerant subjects become more sensitive to vasoconstrictors after they are withdrawn from nitroglycerin. Some have shown electrocardiographic ST segment changes and ventricular arrhythmias suggestive of coronary artery spasm.[28] Evidence is accumulating that withdrawal from nitroglycerin increases the sensitivity of alpha 1 adrenergic receptors in the coronary arteries to endogenous and exogenous vasoconstrictive agents.[28]

The chronic cardiac effects of nitroglycerin withdrawal appear to be latent for 6 to 10 years before the onset of symptoms.[35] Several studies of Swedish dynamite workers have demonstrated excess mortality from cardiovascular and cerebrovascular disease. This excess mortality was only significant for workers with long-term employment and had a latency of 20 years.[32] A more recent, retrospective, cohort-mortality study of workers at a U.S. Army ammunition plant showed an excess of mortality from ischemic heart disease among workers younger than 35 years of age.[28] Pathological examinations of nitroglycerin workers who have experienced cardiac events have failed to reveal coronary artery disease, strengthening the conclusion that rebound vasospasm is responsible.[28,34]

A 1965 review of earlier case reports revealed complaints of digestive troubles, tremors, neuralgia, and, in rare cases, skin sensitization among nitroglycerin

workers.[37] Decreased alcohol tolerance is common and may be caused by nitroglycerin's interference with liver alcohol dehydrogenase. Simultaneous exposure to ethanol and nitroglycerin can cause manic behavior.[38]

Numerous other chronic effects of nitroglycerin exposure have been reported, but are poorly documented. Research has been conducted on other chronic effects in mammals, but the results have not been substantiated in humans. Chronic oral administration of nitroglycerin in rats has produced cancer of the liver. Other research with mammals has indicated the possibility of male reproductive, fetotoxic, and teratogenic effects.[29,39] It was previously believed that nitroglycerin could increase intraocular pressure and precipitate glaucoma, but further evidence has disproved this.[31]

Medical Surveillance

Early identification of cardiovascular disease is the primary goal of medical surveillance of nitroglycerin workers. A preplacement examination must be administered to all new employees, and should consist of both medical and occupational histories, a physical examination, and indicated laboratory tests (Table 9-4). When their employment begins, nitroglycerin workers should maintain a daily record of their pulse rates. Periodic examinations should be conducted semiannually, with the same focus as the preplacement examination. During the periodic examination, the physician should be aware that headaches that occur during workshifts can indicate skin absorption of nitroglycerin, even if air concentrations of nitroglycerin are below the PEL. Examinations with similar content are necessary when exposure to nitroglycerin has been terminated, although surveillance should perhaps extend beyond employment, due to the latency of the withdrawal effects.[14]

In addition to performing the medical surveillance examinations, the plant should follow this procedure to safeguard the health of its workers:

- First, the plant physician should alert the worker's private physician to the effects of exposure to and withdrawal from nitroglycerin.
- Second, workers who leave the plant due to any kind of illness should be cleared through the medical department.
- And third, workers should also be examined before they return to work after lengthy absences.

This procedure, common in all types of industries, is a management tool used as an administrative control measure. When workers leave the plant with any illness, a medical examination can help determine if that illness is due to an acute overexposure to nitroglycerin (or any other toxic agent). By early detection of a sentinel event, the plant management can intervene at the worksite and thus protect other workers in the area, as well as the ill individual on his or her return to work. An examination is necessary whenever a nitroglycerin worker returns from an illness to assure that the worker's health status has not changed in such a way that he or she will be placed at risk. Specifically, the occupational physician should look for changes in cardiovascular status, such as a recent myocardial infarction or new-onset hypertension.

A biological marker of exposure would be a useful aid to the occupational health physician (as is true for any chemical exposure). Blood methemoglobin levels increase after high exposures, but these are not sufficiently sensitive to monitor exposure to nitroglycerin.[40,41] Nitroglycerin can be detected in blood, but, because cubital venous blood samples reflect almost

TABLE 9-4

PREPLACEMENT EXAMINATION FOR NITROGLYCERIN WORKERS*

Component	Emphasis
Medical History	Alcohol use
	Tobacco use
	Hypertension
	Cardiovascular system
	Dermatitis
	Anemia
	Neurobehavioral disorders
	Medications
	Reproductive system
Occupational History	Prior respirator use
	Prior nitrate exposure
	Weekend/vacation chest pain
Physical Examination	Cardiovascular system
	Skin
	Nervous system
	Mental status
	Blood pressure
Tests	CBC
	Resting ECG
	Lipid profile
	Urinalysis

*Medical surveillance also applies to workers exposed to other aliphatic nitrate explosives.

exclusively the locally absorbed compound from the distal part of the arm, they are unreliable indicators of systemic exposure.[40–42]

Primary Prevention

The most efficacious method to control occupational nitroglycerin toxicity is to prevent exposure using engineering controls and hygienic work practices. This is especially true because adverse effects occur at exposure levels below the odor and eye-irritation thresholds that could warn workers of potentially hazardous environments.[26,43]

Several types of engineering controls have proven to be effective in reducing inhalational exposure, including automation, closed-circuit television, and ample work-area ventilation. Volatilization of the aliphatic nitrates can be minimized by processing at the lowest practicable temperatures. Operations that require heating should be controlled remotely.[17] Maintaining a water seal over liquid nitroglycerin will prevent its evaporation and reduce its concentration in air.

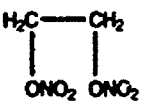
When necessary, PPE should be worn to prevent dermal contact and to reduce airborne levels to an acceptable range. Particular attention must be devoted to the type of gloves worn. Polyethylene gloves may be the best choice, because nitroglycerin easily penetrates neoprene, leather, and rubber. Cotton or canvas gloves, frequently changed, are also preferable to rubber gloves. A face shield or splash-proof safety goggles may also be necessary to protect the eyes. An organic vapor respirator may also be required to prevent headache, especially at concentrations higher than 0.02 ppm.[12]

The selection of a respirator is controversial at present because (*a*) the efficacy of half-mask, air-purifying respirators is unproven and (*b*) the respirators supplied to be used during explosives manufacture have potential safety hazards.[9,12] To date, the only respirators that have been demonstrated to provide a sufficiently high protection factor are the full-face, supplied-air respirators. However, even these are yet to be proven safe in the potentially explosive atmospheres that may exist in nitroglycerin manufacturing operations. Therefore, the only way to assure that workers are protected is to lower the airborne level through engineering controls. However, this is not feasible in all cases. Both the government and industry are aggressively pursuing a resolution to this problem, to comply with the lower OSHA PEL for nitroglycerin that was promulgated in 1989.[9]

Careful attention to personal hygiene is necessary to prevent workers from contaminating their street apparel, and, as a result, possibly poisoning their family members. At a minimum, manufacturing plants should provide change-house facilities that contain an adequate number of coveralls, and shower facilities for employees to use at the end of their shifts. Indicator soaps are available; they turn red in the presence of residual nitroglycerin not removed from the skin. Sodium sulfite in the soap reacts with nitrate groups in nitroglycerin to form sodium sulfonate.[43] (A similar reaction also occurs with asymmetrical TNT isomers and tetryl.)

The treatment for nitroglycerin poisoning consists of removing the patient from the source of exposure, thoroughly cleansing the skin and mucous membranes of nitroglycerin contamination, and providing cardiovascular support. Washing the skin with aqueous sodium thiosulfate will assist in neutralizing any nitroglycerin that remains. The use of oral nitrates and calcium channel–blocking agents has been somewhat efficacious in the treatment of nitroglycerin withdrawal. Both help in nitroglycerin withdrawal (but not in toxicity as such) by reducing reflex vasospasm; the oral nitrates work by drug replacement (analogous to using nicotine gum in tobacco cessation, or methadone in heroin withdrawal, to overcome the physiological effects of withdrawal); the calcium channel blockers work by a different pharmacological mechanism to induce vasodilation.

Ethylene Glycol Dinitrate

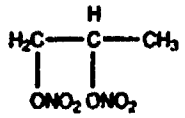


Ethylene glycol dinitrate (EGDN) is frequently used together with nitroglycerin in civilian formulations, but is rarely encountered in military propellants.

Due to its higher vapor pressure, EGDN vapors predominate at all concentrations when present with nitroglycerin. Liquid EGDN appears to be absorbed transdermally more rapidly than nitroglycerin, and EGDN vapors penetrate intact skin. Surgical gloves, used to protect the skin, absorb more of the vapor, but are less permeable to liquid EGDN through direct contact than are cotton gloves.[44]

The symptoms and effects of exposure to EGDN, as well as the medical surveillance and treatment, are the same as those for nitroglycerin. EGDN can be measured in blood and urine, but its concentration in urine is a more reliable indicator of exposure than blood levels. The clinical use of urinary EGDN levels to confirm exposure, however, has not yet been documented.[40,41]

Propylene Glycol Dinitrate



Propylene glycol dinitrate (PGDN) is the principal component (75% by volume) of Otto Fuel II, a torpedo propellant that was introduced in 1966; therefore exposure to PGDN can potentially occur during torpedo maintenance. This liquid has significant vapor pressure under ambient conditions, and is quite soluble in lipids. In the navy during torpedo defueling, refueling, repair, and maintenance, torpedo man's mates have been exposed to PGDN through dermal contact and inhalation. Airborne exposures of up to 0.22 ppm of PGDN can occur during defueling and refueling.

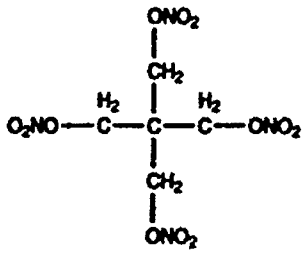
Acute exposure can have several effects similar to those caused by exposure to nitroglycerin, many of which are due to vasodilation. These effects include headaches, nasal congestion, dizziness, impairment of motor coordination and balance, eye irritation, disruption of visual evoked responses, and altered oculomotor function.

In humans, PGDN affects the cardiovascular system and the CNS. At concentrations higher than 0.2 ppm, acute exposure to PGDN produces headaches and CNS depression without evidence of biochemical, hematological, or spirometric changes.[45] Whether PGDN can cause significant cardiovascular effects remains controversial. Exposure to PGDN at levels twice the TLV did not cause cardiovascular or neurological effects in torpedo maintenance workers in one study.[46] Another cohort of torpedo man's mates who were potentially exposed to PGDN at levels up to 0.22 ppm showed an increased risk for myocardial infarction and angina pectoris.[47] The U.S. Navy is evaluating Otto Fuel II for teratogenicity, which is a suspected consequence of maternal methemoglobinemia and related blood dyscrasias.[48]

High doses of PGDN in animals have led to hypotension, methemoglobin formation, and hemosiderin deposits in the liver and kidneys, indicating that erythrocytes, the liver, and the kidneys are also targets for PGDN.[45]

The medical surveillance and exposure controls for PGDN exposure are the same as those for nitroglycerin exposure.[47]

Pentaerythritol Tetranitrate



Pentaerythritol tetranitrate (PETN) is prepared either in batches or by continuously nitrating pentaerythritol (tetramethylolmethane), which is manufactured from formaldehyde and acetaldehyde. PETN is used as a pentolite mixture with TNT in the manufacture of small-caliber projectiles, grenades, and booster charges and is also used alone in the manufacture of detonating fuzes and detonators. Additionally, PETN has been used therapeutically for its vasodilatory effects. Its trade name is Peritrate, manufactured by Parke-Davis.

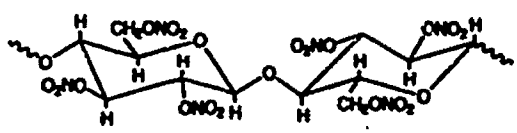
Under ordinary circumstances, PETN has very low potential for exposure. It is nearly insoluble in water, and is usually handled water-wet. PETN is absorbed slowly from the gastrointestinal tract, skin, and lungs. Compared to nitroglycerin, PETN is significantly less toxic and more stable.

The acute effects of exposure to PETN are similar to those of nitroglycerin.[49] Hypotension and increased respiratory rate may both occur, but to a lesser degree than is observed with nitroglycerin. In contrast to nitroglycerin, little reflex tachycardia is observed with PETN. Dyspnea and convulsions have also been reported.

Data on the human effects of chronic exposure to PETN are almost completely lacking. Chronic toxicity of PETN was studied in rats during the early 1940s. Although hemosiderin was found in the spleens of the PETN-treated rats, no pathological changes were observed in their vascular systems. The continued feeding of PETN in doses of 2 mg/kg daily over a period of 1 year did not produce significant effects in the rats.[49]

The medical surveillance for and treatment of exposure to PETN should be the same as that for nitroglycerin.

Nitrocellulose



Nitrocellulose is a nonvolatile, fibrous, white solid consisting of chains of glucoside units in which the hydroxyl groups have reacted to form nitrate esters. The molecular weight depends on the chain length and the degree of polymerization, which in turn depend on the source of the cellulose. Many sources of cellulose may be used, including paper rolls, cotton linters, wood pulp, and waste cotton.

Manufacture and Exposure Hazards

Nitrocellulose was first produced in 1838, but practical difficulties in manufacturing and using the material were not overcome until 1865. Since that time, it has become the basic component of single-base solid propellants. Nitrocellulose is the principal ingredient in gun and mortar propellants, smokeless powder, and ball powder. The military's production of nitrocellulose is second only to its production of TNT. Nitrocellulose is also a component of combustible cartridge cases, and in the civilian sector is used in manufacturing blasting fuzes and mining charges.

In explosive applications, nitrocellulose requires a higher degree of nitration than that produced for its nonexplosive uses such as lacquers, medical collodion, ink bases, or filter membranes. Military-grade nitrocellulose is produced at various U.S. Army ammunition plants in a process wherein cellulose is nitrated with concentrated nitric and sulfuric acids. The only significant byproducts of manufacture are the spent acids, which are concentrated and then reused.

Human Exposure and Health Effects

Insoluble in water and resistant to biological degradation, nitrocellulose per se has a very low potential as a hazard to human health. As an insoluble polymer, nitrocellulose is not absorbed in the gut, and in fact does not appear to be absorbed by any route. The only effects of ingestion are due to the bulk of fiber, which may occlude the intestinal lumen, and are no different than effects of nonnitrated cellulose. Nitrocellulose is not irritating to the skin, and no mutagenic activity has been detected.[50]

Other exposures during the manufacture of nitrocellulose are of greater significance to workers. These include exposures to acids and acid vapors during the initial nitration process, which may lead to dental erosions and chemical burns. Uncontrolled exposure to raw cotton dust from the linters before nitration can cause byssinosis, which is also known as cotton-mill or mill fever. It is a usually allergic, occupational, respiratory disease of cotton, flax, and hemp workers and is characterized by symptoms—especially wheezing—that are most severe at the beginning of each work week (because the lack of exposure over the weekend allows large quantities of the mediators of allergy, such as histamine, to accumulate).

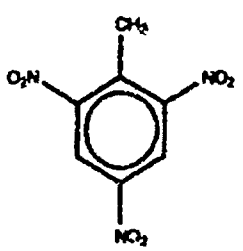
The potential hazards encountered during the manufacturing process necessitate that precautions be taken. Adequate ventilation during both preparation of the linters and nitration is essential. It is recommended that PPE be worn by employees who work near the acids.

No medical surveillance for exposure to nitrocellulose is necessary, and treatment for the sequelae of acid contact is not unique. No exposure limits have been established for nitrocellulose.

THE NITROAROMATICS

The nitroaromatics were the second class of organic nitrates to become important as explosive compounds, and they continue to be represented prominently in the world's arsenals (Figure 9-13). These chemicals are well absorbed by all routes and tend to rapidly penetrate the dermis. The major effects of these chemicals include methemoglobinemia, cancers of the urinary tract, anemia, and skin sensitization.[21,51]

Trinitrotoluene



Not the first to be synthesized but now the best known of the aromatic nitrate explosives, 2,4,6-trinitrotoluene (TNT) was first prepared in Germany in 1863. Although it was not manufactured industrially until 1891, TNT rapidly became the premier high explosive.[24] Major military powers adopted TNT as their major high explosive in 1901, when it replaced picric acid. The first significant military use of TNT was during the Russo-Japanese War of 1905.

Many factors, including its low cost, safety in handling, compatibility with other explosives, low melting point, moderate toxicity, and low sensitivity, have made TNT the most widely used military explosive of the 20th century. Before 1940, its manufacture was limited by the availability of toluene (then a byproduct of the coke industry; see Chapter 1, Occupational Health in the U.S. Army). Advances in petroleum chemistry during World War II permitted the synthesis of large quantities of inexpensive toluene, which greatly enhanced TNT production capacity in the United States.[24]

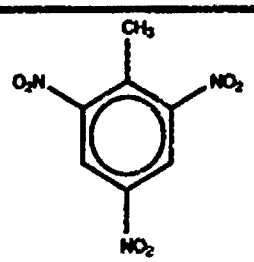
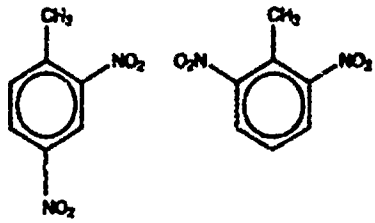
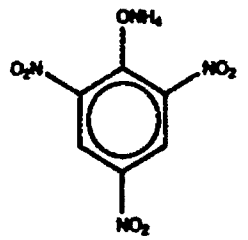
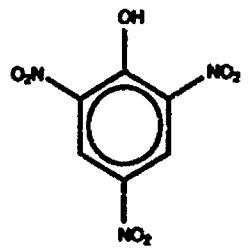
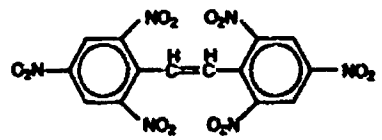
Common Name	CAS Registry Number	Synonym	Formula	Structure
Trinitrotoluene	118-96-7	2,4,6-Trinitrotoluene 1-Methyl-2,4,6-trinitrobenzene TNT	$C_7H_5N_3O_6$	
Dinitrotoluene	2,4-: 121-14-2 2,6-: 606-20-2	2,4-Dinitrotoluene 2,6-Dinitrotoluene DNT	$C_7H_6N_2O_4$	
Ammonium Picrate	131-74-8	Ammonium 2,4,6-trinitrophenolate Explosive D Dunnite	$C_6H_6N_4O_7$	
Picric Acid	88-89-1	2,4,6-Trinitrophenol Melinite Lyddite Shimose	$C_6H_3N_3O_7$	
HNS	20062-22-0	Hexanitrostilbene	$C_{14}H_6(NO_2)_6$	

Fig. 9-13. The nitroaromatic explosives, together with their common names, Chemical Abstract Society numbers, synonyms, formulae, and structures.

TNT can be found in virtually all military applications and is frequently mixed with aluminum and other high explosives to form binary or ternary explosives. Its easy availability during World War II made TNT a perfect suspension agent for more-powerful explosives such as PETN and RDX, and made melt-loading methods feasible.[21] In 1973, at the end of our mobilization for the Vietnam War, an estimated 200,000 tons of TNT were produced in the United States.[52] By 1986, however, domestic production had been curtailed due to the increasing pressure that had developed regarding the chemical's contamination of the environment. Civilian applications, less common than military, still include using TNT in commercial explosives and propellants, and as an intermediate in the production of dyes and photographic chemicals.[21]

Manufacture and Exposure Hazard

TNT was formerly prepared by batch nitration of toluene, but current manufacturing methods are based on continuous stepwise nitration of toluene, with a mixture of concentrated nitric and sulfuric acids flowing countercurrent to the toluene (Figure 9-14). Soda ash (anhydrous sodium carbonate) and sodium sulfite are used in the washing and crystallization processes to purify the crude TNT solution. The purified TNT is then dried in a steam-jacketed pan before being flaked and packed. Occupational exposure to acids, toluene, and impure TNT are minimal during the continuous-manufacture process, in contrast to the older batch processes.[53]

The most significant risk of exposure to TNT occurs during shell-loading operations. Exposure can occur during several of the steps, most of which involve the melt-loading process. In this process, dry flakes of TNT are poured into a steam-heated melting kettle and heated to approximately 100°C. Other high-melting, nonmetallic additives such as RDX or nitrostilbene are added at this point (usually water-wet to control their flammability). Continued heating drives off the water, and flaked aluminum may be added at this point. The mixture is then cooled until the established pouring consistency is reached. After the mixture is poured, the loaded shells are cooled under controlled conditions before the *risers* (funnellike devices that are fitted into the nose of each shell to facilitate filling the loads to the top) are removed and further processing occurs. Exposure to TNT dust, fume, and vapor can occur during any of these operations.

Exposure to TNT can also occur during numerous circumstances other than shell loading (Table 9-5). During World War II, some of the highest TNT dust levels occurred during screening operations (passing TNT flakes through a sieve), where concentrations up to 75 mg/m^3 were measured in breathing zones.[13] Workers can also be exposed to TNT fumes and vapors during demilitarization (removing TNT from shells), when munitions may be steam cleaned to melt and remove the high-explosive charge.

Humans can also be exposed to TNT that has contaminated the environment. Significant amounts of TNT and its manufacturing byproducts have been released into the environment as huge volumes of liquid wastes from factories and LAP plants. These liquid wastes (the *red* or *pink* water) contain TNT isomers, DNT isomers, and mononitrotoluenes. Due to the difficulty and expense of disposing of this waste, the United States currently relies on imported and stockpiled TNT. Two options may be for paper mills to use the red water as a process chemical in the manufacture of kraft products, or to concentrate and incinerate the effluent to yield crude sodium sulfate.[54]

Human Exposure and Health Effects

TNT's toxicity to humans has been recognized for at least 75 years. Most of our knowledge results directly from work performed during the two world wars. In the United States from 1914 to 1918, approximately 24,000 people were poisoned with TNT, fatally in 580 instances. Similar experiences were described in other combatant nations. In Great Britain from 1916 through 1941, 475 cases of TNT poisoning were reported, of which 125 were fatal.[55] In the United States during World War II, TNT poisoning was a factor at manufacturing and loading plants and arsenals, although the case rates at arsenals and manufacturing plants were less than one-half that at loading plants. Of the 21 deaths that occurred, 18 were at loading plants, 2 at arsenals, and 1 at a TNT-manufacturing facility.[2] Progressively more people were exposed to more chemical as the war continued, yet the morbidity was much lower. Case rates for all locations fell dramatically despite the marked increase in TNT production, thus demonstrating the effectiveness of occupational health interventions.

Researchers analyzed the 21 TNT fatalities of World War II, together with another late death of a former TNT worker. Of this series, 8 died of toxic hepatitis and 13 of aplastic anemia. The late death occurred in a worker who apparently had recovered from hepatitis but later succumbed to aplastic anemia. Only one-third of these fatalities had been exposed to average airborne concentrations higher than the maximum allowable concentration of 1.5 mg/m^3 that prevailed

a

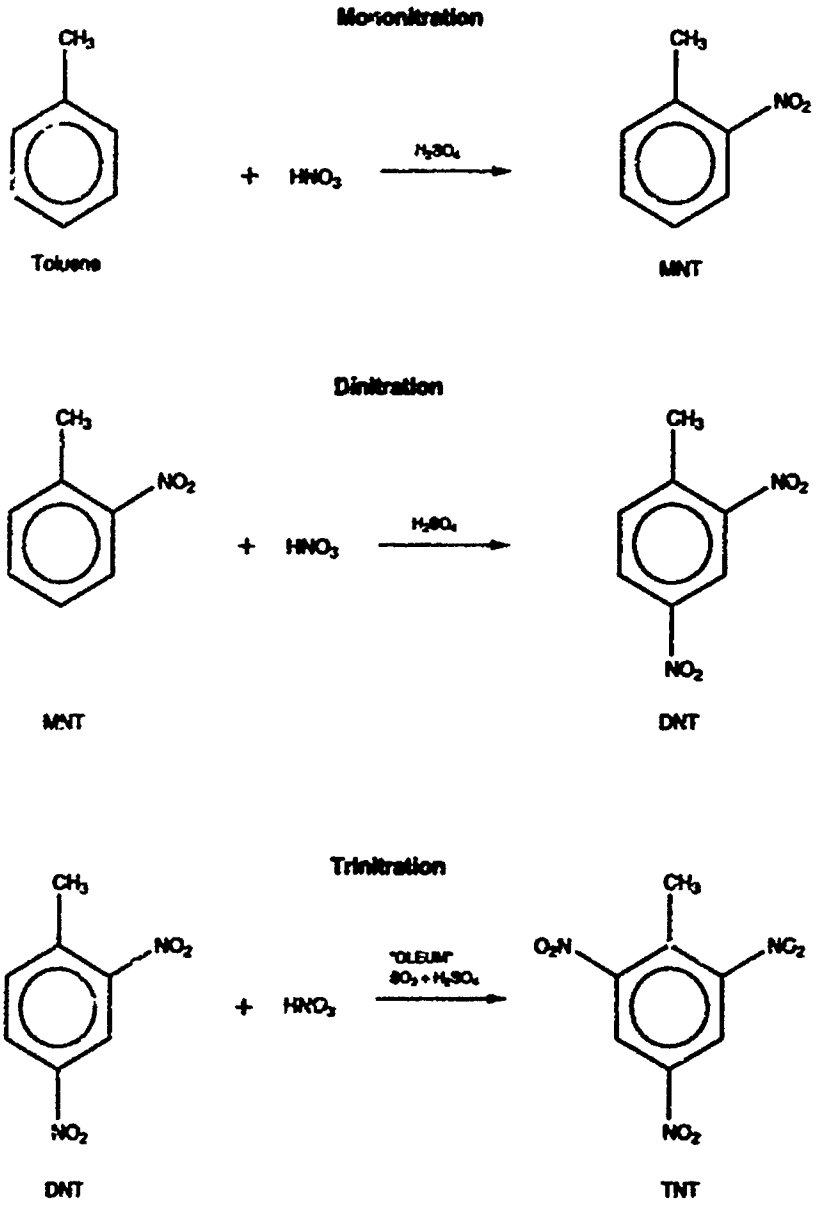
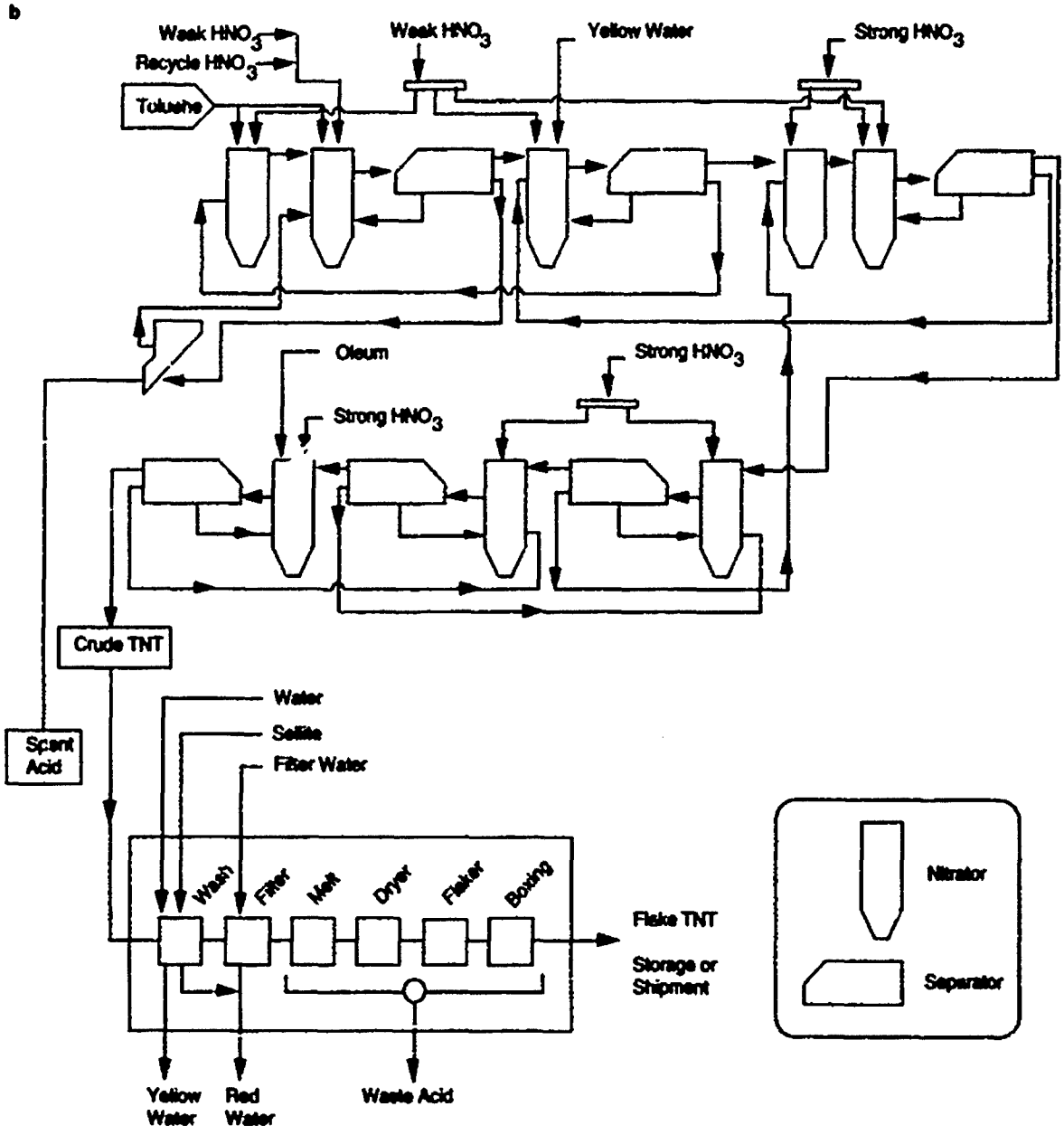


Fig. 9-14. The trinitration of toluene: (a) chemical synthesis and (b) industrial (enclosed) process stream. In the manufacture of TNT, toluene is first reacted with nitric acid to form mononitrotoluene (MNT), which is then pumped to another area, where it is reacted with nitric acid (under different reaction conditions) to form dinitrotoluene (DNT). The DNT is then pumped to yet another area where it is reacted with oleum (a solution of sulfur trioxide dissolved ——————→

b



in anhydrous sulfuric acid) to form crude TNT. The crude TNT then undergoes a series of washes to remove crude unreacted toluene, MNT, and DNT. Reprinted from US Army Environmental Hygiene Agency. *Water Pollution Aspects of Explosive Manufacturing*. Aberdeen Proving Ground, Md: USAEHA; 1985. USAEHA Technical Guide 140; (a) p 16, (b) pp 28, 30.

TABLE 9-5

HEALTH HAZARDS OF EXPLOSIVE OPERATIONS

Operation	Nature of Exposure	Degree of Exposure	Recommended Control Measures
Screening	TNT dust	Severe	Enclosed operations; local exhaust ventilation
Melting	TNT dust, fume, and vapor	Slight	Local exhaust ventilation
Draw off	TNT fume and vapor	Moderate	Local exhaust and room ventilation
TNT cooling	TNT fume and vapor	Slight to severe	Automated operations; local exhaust and room ventilation
Pouring	TNT dust, fume, and vapor	Slight to moderate	Room ventilation, careful work practices, worker rotation
Bomb filling	TNT fume and vapor	Slight to severe	Local exhaust and room ventilation, careful work practices
Bomb puddling, nose, and tail pouring	TNT dust, fume, and vapor	Slight to severe	Room ventilation, careful work practices, worker rotation
Adding TNT scrap	TNT dust, fume, and vapor	Moderate to severe	Room ventilation, careful work practices, gloves, worker rotation
Booster cavity drilling	TNT dust	Slight to severe	Elimination of deep drilling, local exhaust ventilation
Riser knockoff	TNT dust	Slight to moderate	Well-designed risers, local exhaust ventilation, careful work practices
Riser breakout	TNT dust	Moderate to severe	Enclosed operations, local exhaust ventilation
TNT scrap breakup	TNT dust	Moderate to severe	Enclosed operations, local exhaust ventilation
Flaking and graining	TNT fume and dust	Slight to moderate	Enclosed operations, local exhaust ventilation
Box filling	TNT dust	Slight to severe	Enclosed operations, local exhaust ventilation

Sources: (1) Buck CR, Wilson SE. *Adverse Health Effects of Selected Explosives (TNT, RDX).* Aberdeen Proving Ground, Md: USAEHA; 1975. Occupational Study 32-049-75/76. (2) Brandt AD. Engineering control of air contamination of the working environment. In: Gafafer WM, ed. *Manual of Industrial Hygiene and Medical Service in War Industries.* Philadelphia: WB Saunders; 1943: 198.

then, which reflects the contribution of dermal absorption. Workers who died of toxic hepatitis were younger than those who died of aplastic anemia (their median ages were 35 and 45 years, respectively). In both conditions, the median period of exposure was quite short: 63 days for hepatitis, and 216 days for anemia.[6]

Other cohort studies of TNT workers have shown that virtually all cases of toxic hepatitis have occurred within the first 3 months of exposure, while cross-sectional studies have not shown significant signs of hepatotoxicity.[56] This may indicate that a sensitive subgroup of individuals is at risk for this effect.

Another World War II-era study evaluated the effects of TNT intoxication in 250 male and 103 female workers in a bomb- and shell-loading facility.[57] No cases of severe TNT intoxication were seen; however, adverse effects of TNT exposure were found in 32 workers (30 of whom were males), of whom 21 had either gastritis or hepatitis; 14 had anemia; and 3 had systemic manifestations of intoxication.[57]

Toxicokinetics. TNT is readily absorbed by all routes of exposure. Approximately 60% to 70% of oral

doses are absorbed; inhaled TNT appears to be not only absorbed faster than oral doses but it also reaches higher concentrations in the blood. Dermal absorption is less efficient, but its significance must not be underestimated. TNT dissolved in water is particularly well absorbed through the dermis. As might be expected, this effect is greater in hot weather due to the combined effects of greater skin exposure and the dissolution of TNT dust in sweat. Workers' coincident exposure to hygroscopic chemicals such as ammonium nitrate further promotes dermal absorption by keeping the skin moist.[8] Consequently, measuring only airborne levels may significantly underestimate the workers' total systemic exposure.[56,58]

TNT is metabolized primarily by a two-step process: the reduction of the nitro group and its conjugation to glucuronide. Some enterohepatic recycling occurs, but urinary clearance of the glucuronides occurs fairly rapidly, preventing bioaccumulation. The urine of humans who have been exposed to (and of most experimental animals that have been given) TNT becomes discolored with a red metabolite.

Dermatitis and systemic effects do not correlate well.[56] Hematological effects appear to occur at lower doses than hepatic effects, but susceptible individuals will develop hepatotoxicity sooner after initiation of exposure.

Acute Effects. Acute exposure to airborne TNT can cause irritation of the upper respiratory tract and skin; symptoms include sneezing, coughing, rhinitis, and erythematous dermatitis. The onset of acute systemic toxicity is frequently heralded by gastrointestinal symptoms such as nausea, anorexia, and epigastric pain.[56] Systemic symptoms may progress to include headache, fatigue, malaise, palpitations, loss of memory, and cyanosis.[55]

Chronic Effects. The most serious chronic manifestations of TNT toxicity are (*a*) anemia and other hematological changes and (*b*) hepatitis; the chronic effects may also include (*c*) dermatitis, (*d*) ocular effects, (*e*) neurological effects, and (*f*) cancer.

Hematological effects result from the action of TNT on both the bone marrow and mature erythrocytes. Although virtually every cell series in the marrow is affected, the most significant hematological effects occur in the erythrocytic series, and may result in anemia with both aplastic and hemolytic components. TNT depresses erythropoiesis and induces aplastic anemia by suppressing two enzymes that catalyze heme synthesis: δ-aminolevulinic acid synthase (ALA synthase; see Chapter 12, Lead) and heme synthase. This suppression has been demonstrated even in the clinical absence of anemia. Some compensation with reticulocytosis may occur, and marrow hyperplasia has been noted as an early effect,[8] but this is followed by marrow hypoplasia and the compensation effect is lost.

Hemolysis in TNT toxicity occurs as a result of methemoglobinemia. This is a dose-related effect, with low-grade anemia and compensatory reticulocytosis noted at airborne TNT concentrations lower than 0.5 mg/m^3. Exposures of 0.2 to 0.5 mg/m^3 appear to have minimal and well-compensated effects on erythrocytes. Poikilocytosis may occur, as well as hepatic and splenic congestion related to hemolysis. Early signs and symptoms of fatal anemia—even in the absence of G6PD deficiency—include weakness, anorexia, weight loss, cough, epistaxis, elevated bilirubin, decreased hemoglobin, and decreased leukocyte counts. Survival in the case reports of fatal anemia varied from 6 to 185 days, but the median was only 40 days.[8] Hemolytic crisis has been seen in G6PD deficiency within the first few days after exposure.

Other hematological effects include both leukocytosis and leukopenia. Transitory leukocytosis and moderate eosinophilia have been described at airborne levels lower than 2.5 mg/m^3. Leukopenia develops late, well after the hemoglobin level and erythrocyte count fall, in contrast to other chemically induced aplastic anemias. Exposure to TNT causes the monocyte count to increase, regardless of the presence of symptoms, and neither the extent of dermal contact nor the length of inhalational exposure influences the intensity of the hematological response.[59]

TNT poisoning can induce both massive hepatic necrosis and cirrhosis. As with most hepatotoxic agents, the hepatitis manifests with increases in the concentrations of serum transaminases and lactate dehydrogenase (LDH). Researchers found no liver function abnormalities at a time-weighted average (TWA) lower than 0.5 mg/m^3, but they found elevated aspartate aminotransferase (AST) and LDH at airborne concentrations of 0.8 mg/m^3, which persisted even at 0.6 mg/m^3.[60] Early symptoms of TNT-induced hepatitis include nausea, vomiting, malaise, and hepatic tenderness. Jaundice, although a late symptom of TNT hepatitis, develops rapidly as the liver atrophies and has a poor prognosis. In a study of TNT-induced hepatitis fatalities from World War II, the average elapsed time from the first definite symptom to death was 34 days, with a range of 12 to 53 days.[8]

Dermatitis is the most common chronic effect of exposure to TNT. Yellow-orange staining of the skin, hair, and nails is a common sign, and irritant contact dermatitis may occur. Dermatitis requires at least 5 days of exposure to develop, and most patients become tolerant to mild cases.[56,61] Palmar lesions with deep vesicles are characteristic. Allergic contact der-

matitis with classic eczematous lesions has been reported, and may rarely appear as an erythema-multiforme-like eruption. The sensitization dermatitis usually affects the upper limb, but the skin at friction points such as the collar line, belt line, and ankles may also be involved.[14] Workers exposed to high levels of TNT dust are especially at risk for dermatitis, although it may occur in workers throughout the manufacturing process.

According to several studies performed in eastern Europe, exposure to TNT has also been associated with cataracts, but at undefined levels of exposure. TNT workers in Finland have developed equatorial cataracts at concentrations of airborne TNT of 0.14 to 0.5 mg/m^3. These characteristic cataracts are insidious in their development and are present only at the lens periphery; consequently, they do not affect vision. They may not be noted on a routine ophthalmological examination, although they are easily observed when the affected eyes are dilated and examined with a slitlamp.[62] Most affected subjects in the eastern-European studies had normal liver-function tests. These subjects' duration of exposure to TNT was 1.2 to 17.0 years, with a mean of nearly 7 years. Older workers were more commonly affected, and the lens changes appear to be irreversible. Cataract formation may result from direct action of TNT on the lens via lipid peroxidation and production of superoxide anions.[63]

Research into whether neurological signs develop from TNT exposure has yielded controversial results. European accounts of TNT exposure report neurasthenia and polyneuritis.[4] While some accounts of TNT exposure in the United States support these conditions, at least one investigator has concluded that symptoms of peripheral neuritis among workers were not solely due to TNT exposure.[64] This study found that, when present, symptoms were limited to mild sensory disturbances, with no objective evidence of the disease.

TNT has been implicated in carcinogenesis in studies done with laboratory animals, but this has not been noted in human epidemiological studies. The results of feeding studies performed on rodents have shown increased incidence of bladder papilloma and carcinoma, and statistically insignificant increases in leukemia and lymphoma. TNT might be genotoxic, as it given positive results in the Ames assay both with and without metabolic activation.[54] (The Ames assay, a basic toxicological tool, is an in vitro test for mutagenicity. It measures the occurrence of reverse mutagenesis in genetically modified *Salmonella typhimurium* strains. See also Chapter 14, Pesticides.)

Numerous other manifestations that have been attributed to exposure to TNT include myalgia, cardiac dysrhythmia, nephritis, increased vascular permeability, cardiotoxicity, pancreatic exocrine abnormalities, increased capillary fragility, menstrual disorders, and testicular atrophy and hyperplasia.[55]

Primary Prevention and Medical Surveillance

Historically, control of exposure to TNT has been accomplished through the general safety and hygiene measures discussed earlier in this chapter, yet additional, specific measures are necessary. The Hazard Communication Program, for example, should instruct workers about the need for strict personal and shop hygiene, and about the hazards of the particular operations that are conducted in that plant. In addition, soap that contains 5% to 10% potassium sulfite will not only help remove TNT dust from the skin, suds that turn red will also indicate any remaining contamination. Further-more, respiratory protective equipment should be selected according to NIOSH guidance, and should be worn during operations that release dust, vapor, or fumes.

Before World War II, research suggested that improving the nutritional status of TNT workers might help improve their resistance to toxic effects. However, in a World War II–era cohort study, multivitamin capsules were not shown to be efficacious in preventing TNT toxicity.[65]

Because TNT interacts with certain medications—including those that cause intrahepatic cholestasis, hepatocellular necrosis, and bone marrow depression—patients taking medications such as isoniazid, halothane, phenylbutazone, phenytoin, and methotrexate, and whose exposures to TNT cannot be precluded, should be closely followed by the occupational physician.

The U.S. Army currently recommends preplacement and periodic (semiannual) examinations of TNT workers. The recommended content of the preplacement examination is found in Table 9-6. The occupational physician should determine on a case-by-case basis which of the elements in Table 9-6 to use in periodic surveillance. However, to identify workers with higher-than-normal sensitivity to TNT toxicity, we recommend specifically that workers undergo monthly hemoglobin, LDH, and AST determinations during the first 3 months of exposure to TNT.[56] One study—which was cited both in the open and in U.S. Army literatures—demonstrated that assaying for AST, LDH, and hemoglobin in combination detected all abnormal cases, whereas if the assays were performed

TABLE 9-6

PREPLACEMENT EXAMINATION FOR TNT WORKERS

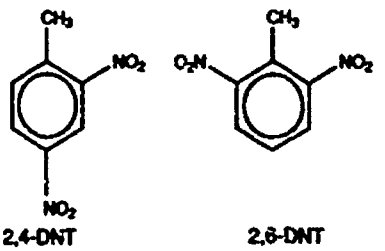
Component	Emphasis
Medical History	Alcohol use
	Tobacco use
	Allergies
	Cardiovascular system
	Skin
	EENT
	Hematologic system
	Liver disease
	Nervous system
	Respiratory system
	Renal System
	Medications
Occupational History	Prior TNT sensitivity
	Prior exposures to hepatotoxins
Physical Examination	Allergies
	Cardiovascular system
	Skin
	EENT, including slitlamp examination of eyes
	Liver
	Nervous system
	Mental status
Tests	CBC with differential and reticulocytes
	Methemoglobin
	Renal function
	Liver function
	Microscopic urinalysis

Adapted with permission from Hogstedt C, Davidsson B. Nitroglycol and nitroglycerine exposure in a dynamite industry 1958–1978. *Am Indust Hyg Assoc J.* 1980;41:373–375.

alone or in pairs, many cases were missed.[65,66] Periodic examinations provide inadequate warning of impending aplastic anemia.[67] Workers who have abnormal results should be removed from exposure and evaluated further.[66,68]

Bioassay for TNT exposure began during World War II with the use of Webster's test for urinary TNT.[69] This qualitative test was based on the reaction of alcoholic potassium hydroxide with an ether extract of acidified urine, wherein colors are produced when TNT and other metapolynitro compounds are present in urine.[70] (Anthraquinones and indole red may cause interference, however.[69]) In comparison to the qualitative Webster's test, a quantitative test for urinary aminodinitrotoluene (ADNT), a metabolite, can be related to TNT absorption within 24 hours of exposure. The urinary ADNT is measured via gas chromatography with electron-capture detection.[70] Most individuals excrete their highest concentrations of ADNT within a few hours after exposure, but some still excrete significant amounts many hours later. This prolonged excretion time may indicate that TNT or a metabolite has been retained, or may perhaps indicate delayed skin absorption. Prolonged dermal absorption has been indicated in a group of explosives workers whose urinary concentrations of ADNT indicated higher total exposures than were predicted from the concentrations in ambient air.[58,71]

Dinitrotoluene



2,4-DNT 2,6-DNT

In 1980, nearly 8% of the total toluene produced in the United States was converted to dinitrotoluene (DNT), which is widely used in military applications and in civilian applications such as dye manufacturing and organic chemical synthesis. Of the DNT produced currently, 99% is used in the synthesis of toluene diamine, an intermediate in the production of toluenediisocyanate. DNT may also comprise up to 10% of commercial dynamite formulations. Military uses of DNT are similarly broad, where it is usually used as an additive to modify the properties of other explosives. For example, DNT may function as a combustion modifier in propellants, as a gelatinizer in explosives, or as a waterproofing agent in explosives.[4]

Manufacture and Exposure Hazard

Due to the serious safety and health hazards inherent in the manufacture of DNT (it is regulated as a carcinogen and is even more hazardous than TNT), current practices for technical-grade DNT use continuous, closed systems that are highly automated and remotely controlled. Technical-grade DNT is a greasy liquid comprised of approximately 80% 2,4-DNT and 20% 2,6-DNT, but military-grade DNT requires highly purified 2,4-DNT flakes. Significant occupational exposure is possible during the purification and flaking

processes, as well as during the later mixing and shell-loading operations. Because DNT is also present in the waste water of TNT manufacturing and shell-loading plants, significant environmental contamination, and thus exposure, can also occur.

Human Exposure and Health Effects

DNT is readily absorbed via all routes of exposure, but absorption through the dermis is probably the most significant. In rats, both the 2,4- and 2,6- isomers are extensively metabolized by the liver and then excreted in bile.[72] Intestinal nitro-reductase-active bacteria further metabolize the product, which is resorbed and remetabolized to an as-yet-unidentified—but genotoxic—product.[73] The excretion of 2,4-DNT metabolites in humans is qualitatively similar to that in rats; however, humans do not excrete the reduced metabolite of 2,6-DNT. This qualitative difference in metabolism makes interspecies extrapolation of the carcinogenic risks difficult.[74,75]

Acute Effects. The most characteristic sign of acute DNT toxicity is methemoglobinemia. Associated symptoms include headache, fatigue, cyanosis, irritability, and nausea. Moderate exposures may cause ataxia, respiratory depression, and arthralgias, while severe exposure may lead to progressive CNS depression and death.[73,74]

Chronic Effects. Anemia and ischemic heart disease are the most commonly recognized chronic effects of exposure to DNT.[73] The anemia occurs when erythrocytes that contain methemoglobin hemolyze, and is typically low-grade and partially compensated.[74] Increased mortality from ischemic heart disease has been seen in munitions workers who were exposed to DNT during the 1940s and 1950s.[76,77] Unfortunately, the lack of adequate exposure data precludes our ability to make dose-response estimates for these effects.

Concerns about the carcinogenicity of DNT have been expressed for several years, and have recently focused on incompletely burned DNT in propellant residue at waste propellant disposal sites. Anyone exposed is at risk for carcinogenesis, including workers at the disposal sites, and all who are environmentally exposed via dust, groundwater, or direct contact with contaminated soil. DNT isomers exhibited only weak mutagenic activity in the Ames assay[74] and no activity in various mammalian cell culture genotoxicity assays. However, feeding studies in rats using both technical-grade DNT and 2,6-DNT showed a high incidence of hepatocellular carcinomas produced by 2,6-DNT, with a lower incidence in females compared to males. Enterohepatic recirculation with hepatic and intestinal microfloral metabolism are necessary for the production of an as-yet-unidentified ultimate carcinogen. Three major mammalian carcinogenicity studies have indicated that 2,6-DNT is both an initiator and a promoter, while 2,4-DNT is only a promoter.[72,74] Evidence of carcinogenicity in humans is lacking, however. Two occupational-cohort studies have been completed on workers exposed to DNT. Neither study showed any excessive incidence of cancer, but both demonstrated elevated cardiovascular and cerebrovascular mortality.[66,76]

Deleterious effects on the reproductive systems have been reported in rats that were given large doses of DNT (≥ 34.5 mg/kg/d), but such effects were not seen in a NIOSH study of workers at a DNT-toluenediamine (TDA) plant.[78] Testicular atrophy, decreased spermatogenesis, and nonfunctioning ovaries have been seen in rats, mice, and dogs in feeding studies performed to assess chronic exposures. Results of multigenerational reproductive studies in animals have been negative. Only one of three epidemiological studies has shown effects on the human reproductive system, and those were limited to decreased sperm counts, minor morphologic changes in sperm, and a small increase in spontaneous abortions among wives of exposed workers.[73,74] Studies done on animals and humans have failed to identify teratogenic effects.

Other chronic effects noted in studies on animals include neurotoxicity and hepatotoxicity, with histological changes in both organs noted at autopsy. Sensitization dermatitis may also occur, but not as frequently as with exposure to TNT. Friction sites are frequently affected by DNT dermatitis.

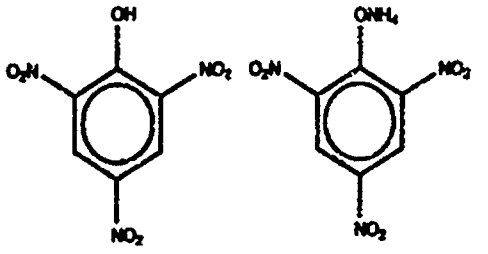
Primary Prevention and Medical Surveillance

As with all potential carcinogens, prevention of exposure is essential with DNT. Workers who could potentially be exposed to DNT should be informed of the deleterious health effects, including the possible reproductive system effects. In addition to the safety and hygiene measures previously mentioned, occupational health personnel should monitor for residual buildup of DNT on clothing, boot linings, and hardhat liners. Respiratory protection is usually unnecessary, because DNT has low vapor pressure.

Medical surveillance should consist of the same protocol as that for TNT, with the addition of a reproductive history, and the measurement of urinary DNT. The preplacement evaluation should include a baseline sperm count and morphology assessment for workers who intend to have children. Semen analysis should

not be necessary during routine periodic medical surveillance of exposed workers.

Picric Acid and Ammonium Picrate



Picric Acid Ammonium Picrate

Picric acid was first prepared in 1771 for use as a yellow dye, and in 1885 the French first utilized its explosive characteristics as a bursting charge for shells. However, the introduction of TNT during the 20th century eclipsed picric acid's use as a high explosive. For military purposes, some of picric acid's chemical characteristics are disadvantageous: its high melting point, which makes picric acid more difficult to shape into bombs and projectiles, and its tendency to form sensitive salts with materials such as calcium, lead, zinc, and other metals.[24] Sensitive salts are likely to explode with small shocks or bumps, and the rate and extent of these salts' formation is unpredictable.

The military currently uses picric acid only as a raw ingredient for ammonium picrate, which, due to its insensitivity to impact, is used in some armor-piercing shells. The civilian sector formerly used picric acid in burn ointments[5] and currently uses it in pharmaceutical manufacture,[21] leather tanning, dyes, copper and steel etching, forensic chemistry, and photographic emulsions.

Human Exposure and Health Effects

Systemic effects from inhaling or ingesting picric acid and ammonium picrate are rare; the main health effects that workers experience after exposure have been dermatological. Exposure to dust or fumes may cause skin and eye irritation, allergic dermatitis, and yellow staining of the skin and hair. Picric acid does not appear to penetrate intact skin, and dermal exposure probably contributes little to systemic absorption and toxicity. In contrast, inhaled and ingested picric acid is well absorbed, and in rare cases can cause headache, vertigo, loss of consciousness, myalgias, nausea, vomiting, gastroenteritis, hemorrhagic nephritis, and acute hepatitis. Evaluation of mutagenicity has yielded mixed results in prokaryotic and eukaryotic tests.[79]

Primary Prevention and Medical Surveillance

Preplacement examinations should be conducted according to the information contained in Table 9-7.[60,80] Once again, the contents of the periodic job-related examination should be determined by the occupational physician on a case-by-case basis.

Other Nitroaromatics

Several other nitroaromatics have unique properties that allow them to be used by the military in specialized but limited ways. The powerful explosives 1,3-diamino-2,4,6-trinitrobenzene (known as DATB or DATNB) and 1,3,5-triamino-2,4,6-trinitrobenzene (TATB) are used in plastic explosives.[24] Hexanitrostilbene (HNS) is a derivative of TNT that is very stable at high temperatures. It is used in plastic bonded explosives and detonation fuzes, and is being investigated as a nucleating agent for cast TNT and the TNT-RDX mixture that is known as composition B. As a nucleating agent, it reduces the crystal size in TNT casts and prevents formation of dangerous filling defects when the TNT contracts and cools.[81] HNS is used with 2,2',4,4',6,6'-hexa-nitroazobenzene (HNAB) in detonating fuzes.[4] Data regarding the toxicity of these explosives are virtually nonexistent, although they may be expected to show effects similar to the other nitroaromatic explosives.

TABLE 9-7

PREPLACEMENT EXAMINATION FOR PICRIC ACID AND AMMONIUM PICRATE WORKERS[a]

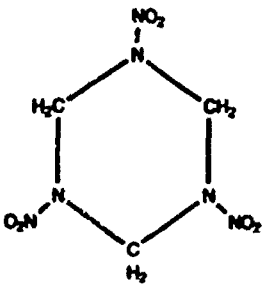
Component	Emphasis
Medical History	Asthma Allergies Sensitization to ammonium picrate and picric acid Sensitization to tetryl
Examination	Kidneys Liver Blood Skin
Laboratory Tests	Chemical and microscopic urinalysis Liver-function tests

[a]Periodic examinations should be administered annually.
Reprinted from Centers for Disease Control, National Institute for Occupational Safety and Health, Occupational Safety and Health Administration. *Occupational Health Guidelines for Chemical Hazards*. Washington, DC: US DHHS and US DOL; 1981. DHHS (NIOSH) Publication 87-116.

THE NITRAMINES

The nitramines are the most recently introduced class of organic nitrate explosives (Figure 9-15). The most prominent member of this class is RDX (research department explosive; hexahydro-1,3,5-trinitro-1,3,5-triazine, which is also known as cyclonite); HMX (high-melting explosive; octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine), nitroguanidine, and tetryl are also significant nitramines.

Hexahydro-1,3,5-trinitro-1,3,5-triazine



Although RDX (see Figure 9-15) was first prepared in 1899, its explosive properties were not appreciated until 1920. RDX was used widely during World War II because petroleum was not needed as a raw ingredient.[24] During and since World War II, RDX has become the second-most-widely used high explosive in the military, exceeded only by TNT. As with most military explosives, RDX is rarely used alone; it is widely used as a component of plastic explosives, detonators, high explosives in artillery rounds, Claymore mines, and demolition kits. RDX has limited civilian use as a rat poison.

Manufacture and Exposure Hazard

RDX is manufactured using the continuous Bachman process, in which hexamine is nitrated with ammonium nitrate and nitric acid in a solvent mixture of acetic acid and acetic anhydride. The byproducts of RDX manufacture include nitrogen oxides, sulfur oxides, acid mists, and unreacted ingredients (Figure 9-16). In 1964, during mobilization for the Vietnam War, the Holston Army Ammunition Plant alone produced approximately 750,000 pounds per day of RDX and HMX combined.[82]

Soldiers and other workers have been exposed to RDX during its manufacture, in the field, and through the contamination of the environment. The main occupational exposure to RDX during its manufacture is through the inhalation of fine dust particles. Ingestion may also be a possible route of exposure, but it is poorly absorbed through the dermis.[83]

The greatest potential for occupational exposure to RDX occurs at ammunition plants with LAP operations, where workers involved with melt-loading and maintenance operations have the greatest potential for exposures.[83] During World War II, there were no fatalities and little morbidity at RDX manufacturing plants. Small numbers of Italian and German workers, who handled powdered RDX in the drying, cooling, screening, and packing processes, were reported to have experienced RDX toxicity, but all recovered completely.[84]

In 1962, five cases of convulsions or unconsciousness or both occurred at an RDX manufacturing plant in the United States. In four of these instances, exposure was from inhaled dust during cleanup of a mixing area. The fifth employee screened and blended dried RDX from different batches; gross skin and air contamination occurred because no mechanical ventilation was used and the individual did not follow handwashing and hygiene precautions. All five employees had convulsions during their workshifts or within a few hours after their shifts were over. These patients exhibited little or no prodrome, and the postictal phase lasted up to 24 hours. No abnormal laboratory or physical findings were noted.[84]

Troops have also become intoxicated during field operations from exposure to composition C4 plastic explosive, which contains 91% RDX. These field exposures occurred because C4 was either chewed as an intoxicant or used as a fuel for cooking. Thus, the route of exposure was ingestion or inhalation. At least 40 American soldiers experienced convulsions due to RDX ingestion during the Vietnam War.[85,86]

RDX in the waste water from manufacturing and loading operations has also contaminated the environment. Although contamination has appeared in soil and groundwater near some ammunition plants, RDX's low solubility in water has limited its migration in most cases.

Human Exposure and Health Effects

The mainstay of treatment for RDX exposure is removal from exposure. Patients who are experiencing seizure activity should be given phenobarbital. Phenytoin is ineffective in controlling RDX-induced seizures.[85]

Toxicokinetics. Gastrointestinal absorption of RDX in humans is slow but complete; serum levels peak approximately 12 hours after ingestion. Clearance of RDX from the serum occurs in approximately 15 hours. The highest tissue levels of RDX occur in the kidneys,

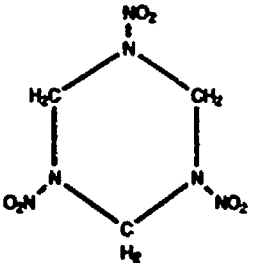
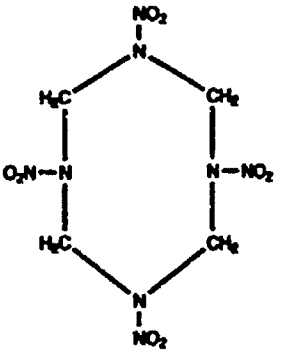
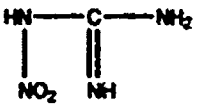
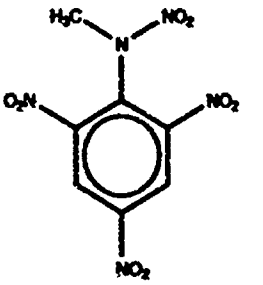
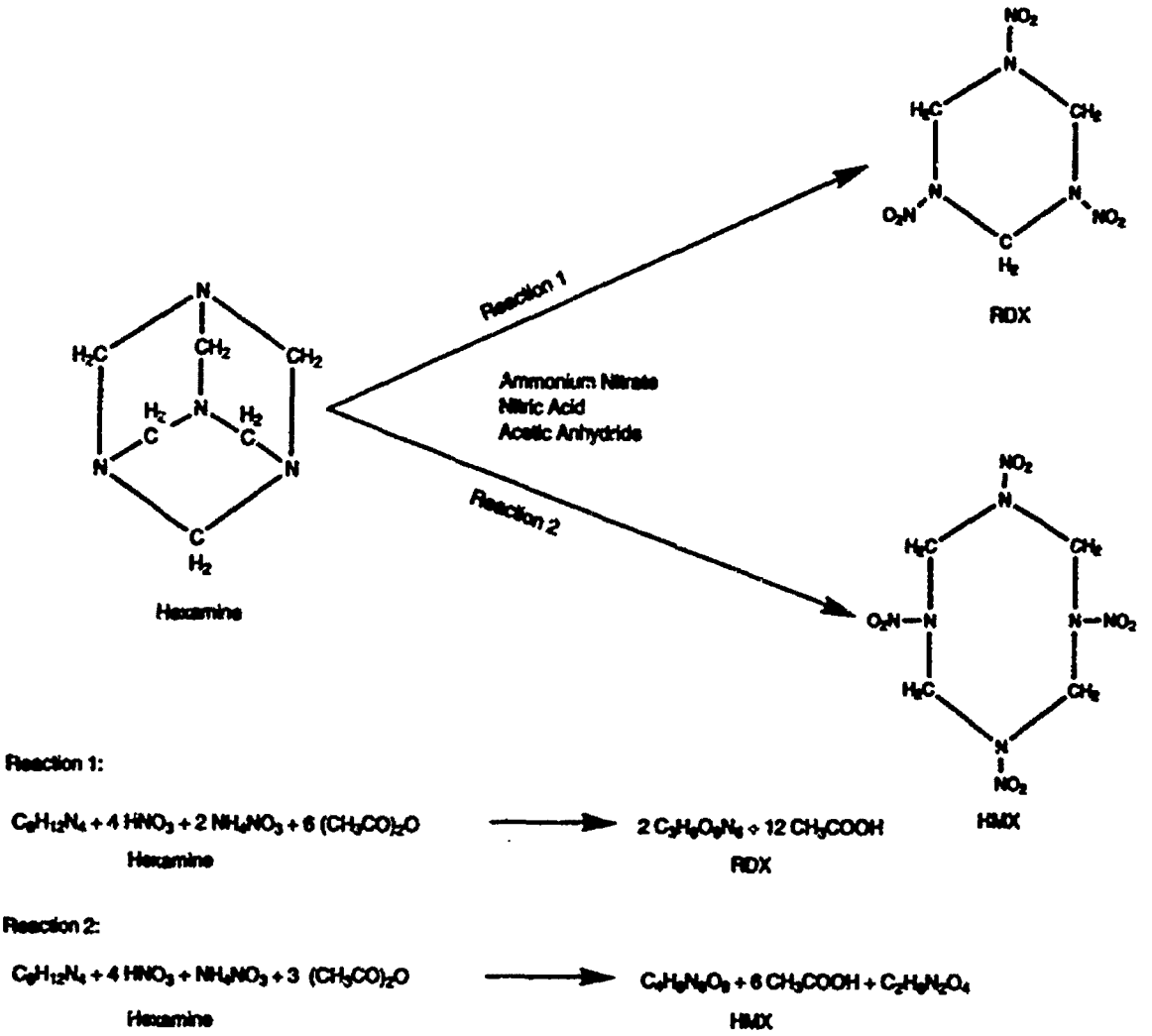
Common Name	CAS Registry Number	Synonyms	Formula	Structure
RDX (Research Dept. Explosive)	121-82-4	Cyclonite Hexahydro-1,3,5-trinitro-1,3,5-triazine	$C_3H_6N_6O_6$	
HMX (High-Melting Explosive)	2691-41-0	Cyclotetra-methylenetetra-nitramine Octahydro-1,3,5,7-tetra-nitro-1,3,5,7-tetrazocine Octogen	$C_4H_8N_8O_8$	
Nitroguanidine	556-88-7	Alpha-nitroguanidine Guanidine-1-nitro N(1)-nitroguanidine NQ	$CH_4N_4O_2$	
Tetryl	473-45-8	2,4,6-Trinitrophenylmethyl-nitramine N-methyl-N-2,4,6-tetranitro-aniline N-methyl-N-2,4,6-tetranitro-benzamine N-methyl-N,2,4,6-tetranitro-methylamuline Nitramine	$C_7H_5N_5O_8$	

Fig. 9-15. The nitramine explosives, together with their common names, Chemical Abstract Society numbers, synonyms, formulae, and structures.



Reaction 1:

$$C_6H_{12}N_4 + 4\,HNO_3 + 2\,NH_4NO_3 + 6\,(CH_3CO)_2O \longrightarrow 2\,C_3H_6O_6N_6 + 12\,CH_3COOH$$

Hexamine → RDX

Reaction 2:

$$C_6H_{12}N_4 + 4\,HNO_3 + NH_4NO_3 + 3\,(CH_3CO)_2O \longrightarrow C_4H_8N_8O_8 + 6\,CH_3COOH + C_2H_2N_2O_4$$

Hexamine → HMX

Fig. 9-16. The chemistry of RDX and HMX production. HMX is an unavoidable coproduct of the production of RDX from hexamine. Most RDX preparations contain at least 9% HMX. Reprinted from US Army Environmental Hygiene Agency. *Water Pollution Aspects of Explosive Manufacturing*. Aberdeen Proving Ground, Md: USAEHA; 1985: 21. USAEHA Technical Guide 140.

with slightly lower levels in the liver, brain, and heart. RDX is metabolized by the liver, and the unidentified metabolites are excreted primarily in the urine.[86] Unlike most other nitrated explosives, RDX does not metabolize to form nitrite in the blood.

Acute Effects. RDX has relatively low acute toxicity. After acute exposure by inhalation or ingestion, there is a latent period of a few hours, followed by a general sequence of intoxication that begins with a prodromal period of irritability. Neurological symptoms predominate and include restlessness and hyperirritability; headache; weakness; dizziness; hyperactive reflexes; nausea and vomiting; prolonged and recurrent generalized convulsions; muscle twitching and soreness; and stupor, delirium, and disorientation.[87]

Clinical findings in acute exposures may also include fever, tachycardia, hematuria, proteinuria, azotemia, mild anemia, neutrophilic leukocytosis, elevated AST, and electroencephalogram (EEG) abnormalities.[4] These abnormal effects, transient and unreliable for diagnosic purposes, last at most a few days. In fact, all physical and laboratory tests may remain normal, even in the presence of seizures.[4,8,86] EEGs made at the time of convulsions may show bilateral synchronous spike and wave complexes (2–3/sec) in the frontal areas with diffuse slow wave activity; nor-

malization occurs within 1 to 3 months.[85]

Patients will recover from acute RDX exposure within days to months, gradually but completely, and they may experience amnesia early in the process.

Several case reports of RDX ingestion have been documented. In one instance, a 3-year-old child ingested plasticized RDX that had adhered to the boots and clothing of its mother, who worked in a munitions plant. The child presented with status epilepticus, but recovered without sequelae. Laboratory tests were essentially normal, and the dose of RDX ingested by the child was estimated to be 84 mg/kg.[85] In the instances of convulsions that occurred among American soldiers in Vietnam, the signs and symptoms usually began 8 to 12 hours after ingestion. Renal toxicity was observed in 3 of 18 patients (16%) in one series.[85] The sequence of symptoms was similar to that which occurred after occupational exposures, proceeding from confusion and hyperirritability to myoclonic contractions, severe prolonged generalized seizures, prolonged postictal confusion, and amnesia.[4,85,86]

The effects of acute exposure to RDX have also been studied in animals. In rats, the median lethal dose of orally administered RDX was approximately 200 mg/kg. Groups of 20 rats at each dose level were administered 25 mg/kg, 50 mg/kg, or 100 mg/kg; all doses produced hyperirritability, convulsions, and mortality up to 86.6%.[87]

Chronic Effects. Although intensive research with animals has revealed some effects, few effects of chronic human exposure to RDX have been reported. One study reported that occupational exposure to TWAs of 0.28 mg/m^3 to 1.57 mg/m^3 did not cause hematological, hepatic, or renal abnormalities. This study also failed to substantiate a suspected association of RDX exposure with systemic lupus erythematosis. Moderate reductions of the erythrocyte count and hemoglobin occur during the first month of exposure, but these values return to normal by the end of the second month.[86]

Tests done on animals have supplemented the knowledge of the chronic effects of RDX on humans. Dogs fed 50 mg/kg of RDX daily for 90 days developed hyperirritability, convulsions, and weight loss, with no alterations of their blood chemistries or cytology. No histological lesions have been found in animals that have had RDX-induced seizures. In addition to those effects noted in humans, several others have been seen in animal tests: cancer, weight loss, anemia, hepatotoxicity, testicular degeneration, and suppurative inflammation of the prostate.[85]

Investigations into the mutagenicity and carcinogenicity of RDX have yielded conflicting results. RDX does not appear to be a mutagen, based on negative results in the Ames test, the dominant lethal test, and the unscheduled deoxyribonucleic acid synthesis assay. RDX has not been found to be carcinogenic in gavage studies performed on rats, but increased hepatocellular carcinoma and adenoma were noted in females of one strain of mice. Due to this finding, the U.S. Environmental Protection Agency has classified RDX as a possible human carcinogen.[85]

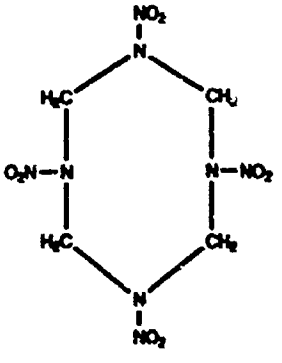
Reproductive effects have been noted in rabbits and rats. A study performed on rabbits showed teratogenic effects at 2 mg/kg/day (10% of the dose that caused maternal toxicity).[86] Similarly, a teratology study performed on pregnant rats exposed to RDX resulted in offspring with lower body weights and shorter body lengths than were found in the control group. These researchers therefore recommended that human females of childbearing age be protected from exposure to RDX.

Primary Prevention and Medical Surveillance

Despite the low toxicity of RDX, exposure should be maintained at the lowest levels possible due to its possible carcinogenicity. Sound industrial hygiene and preventive medicine measures, such as those used in the handling of TNT, should suffice to protect the worker.

General medical surveillance examinations can be conducted, but specific testing for the effects of low-level occupational exposure does not appear to be warranted, given the absence of abnormal results even in those patients with RDX-induced seizures. Surveillance for both males and females should also include a screening questionnaire for reproductive history.

Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine



HMX is the highest-energy solid explosive produced on a large scale in the United States. This explosive is used exclusively for military purposes to implode fissionable material in nuclear devices, as a component of plastic-bonded explosives, as a compo-

nent of rocket propellant, and as a high-explosive burster charge.[4]

Manufacture and Exposure Hazards

Exposure to HMX can occur during the manufacture and filling of munitions or through the environmental contamination of groundwater and soil. HMX, like RDX, is manufactured using the continuous Bachman process (see Figure 9-16). Although its solubility in water is very low, HMX can be present in particulate form in water effluent from manufacturing, LAP, and demilitarization operations.

Human Exposure and Health Effects

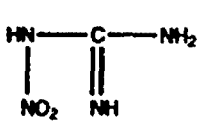
The data on the effects on human health of exposure to HMX are very limited. HMX causes CNS effects similar to those of RDX, but at considerably higher doses.[86] In one study, volunteers submitted to patch testing, which produced skin irritation. Another study of a cohort of 93 workers at an ammunition plant found no hematological, hepatic, autoimmune, or renal diseases. However, the study did not quantify the levels of exposure to HMX.

HMX exposure has been investigated in several studies on animals. Overall, the toxicity appears to be quite low. HMX is poorly absorbed by ingestion. When applied to the dermis, it induces mild skin irritation but not delayed contact sensitization. Various acute and subchronic neurobehavioral effects have been reported in rabbits and rodents, including ataxia, sedation, hyperkinesia, and convulsions. The chronic effects of HMX that have been documented through animal studies include decreased hemoglobin, increased serum alkaline phosphatase, and decreased albumin. Pathological changes were also observed in the animals' livers and kidneys.[89] No data are available concerning the possible reproductive, developmental, or carcinogenic effects of HMX.

Primary Prevention and Medical Surveillance

Both primary prevention and medical surveillance for HMX exposure should be conducted as they would be for exposure to RDX, discussed above.

Nitroguanidine



Nitroguanidine was first prepared in 1877, but was not used as an explosive until World War II. Today, it is a major component of triple-base solid propellants. The properties that give nitroguanidine an advantage over nitrocellulose or nitroglycerin include its cooler burning, greater production of gas, less flash, less smoke, and less corrosion in gun barrels.

Manufacture and Exposure Hazards

Nitroguanidine is produced using the British aqueous fusion process, which is not dependent on either coal or petroleum for raw ingredients. The ingredients and process chemicals used in the production of nitroguanidine include calcium carbide, nitrogen, calcium cyanamide, ammonium nitrate, guanidine nitrate, ammonia, and sulfuric acid.[4]

Workers can be exposed to nitroguanidine during the manufacturing process or during its incorporation into propellants. Nitroguanidine is moderately soluble in water and is rapidly absorbed in the gastrointestinal tract. It is only negligibly metabolized, however, and the body rapidly excretes unaltered nitroguanidine in the urine.

Human Exposure and Health Effects

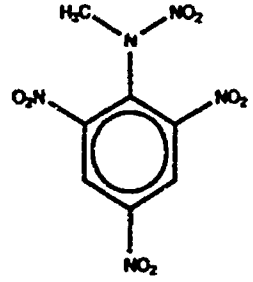
Although studies of the effects of nitroguanidine on humans have not been done, studies performed on animals have indicated generally low toxicity. The oral LD_{50} is 3.9 g/kg in mice and 10.2 g/kg in rats. Direct contact with nitroguanidine may burn the skin and eyes. Single sublethal doses of nitroguanidine in rodents have caused respiratory effects (epistaxis and dyspnea), gastrointestinal effects (diarrhea and hemorrhage), and CNS effects (depression, hyperactivity, ataxia, and tremors). Chronic exposure to nitroguanidine may result in osmotic diuresis and modest hematological and liver-function changes.[90] Results of studies of the reproductive and teratogenic effects of nitroguanidine appear to be negative and results of testing for mutagenicity, despite flaws in the study protocol, appear to be negative.

Primary Prevention and Medical Surveillance

Those who work with nitroguanidine should avoid exposing their skin, eyes, and respiratory tracts, and should wear PPE (safety glasses and respiratory protective equipment) when exposure to airborne nitroguanidine exceeds the exposure limit. Preplacement examinations should focus on the kidneys, the liver, and the blood and include renal and liver-func-

tion tests and complete blood counts. Because the toxic effects are subtle and the long-term implication of alterations in these clinical tests is unclear, the occupational physician should use an interim medical history to determine the contents of periodic examinations on a case-by-case basis. Altered test results may indicate the need for improved exposure control in the workplace and medical follow-up of the abnormal results.

2,4,6-Trinitrophenylmethylnitramine



Tetryl was first prepared in 1877, but was not used as an explosive until World War I, when it was found to be the most efficient booster charge for high explosives. From World War II until recently, tetryl was the booster charge most commonly used by the United States military. The armed forces have also used tetryl in blasting caps, primers, and shell bursters.[4] Although many of our allies continue to produce and use tetryl, production in the United States ceased in 1983 because tetryl tends to corrode steel and is unstable when stored at high temperatures. Thus, our current use of tetryl is limited to the remaining stock. Its only reported civilian use is as a pH indicator.[21]

Manufacture and Exposure Hazards

The manufacture of tetryl involves several steps during which workers can be exposed to nitrogen oxides and acid vapors. Sulfuric acid is first added to dimethylaniline; the acidified dimethylaniline is then nitrated by concentrated nitric acid, forming a granular tetryl precipitate. These grains of tetryl are then dried, screened, coated with graphite, and compressed into pellets for use in munitions.[24]

Occupational exposures to tetryl can occur during the various LAP operations that use tetryl pellets. These operations create tetryl dust, and thus expose the workers through dermal contact, inhalation, and incidental ingestion of the dust. In a retrospective cohort study of systemic toxicity, the chief site of exposure was a powder house in which workers loaded booster bags with tetryl.[97] Virtually all LAP operations with tetryl pellets significantly expose workers to tetryl dust.[13,16,61]

Human Exposure and Health Effects

The primary acute effects of exposure to tetryl are due to its irritation of the upper respiratory tract, the skin, and the eyes. Workers who have been exposed to tetryl dust complain of burning eyes, itching, sneezing, coryza, and epistaxis. These symptoms may begin at any time during the first 3 months of exposure. New workers may also experience nausea and vomiting, but they recover rapidly. Nasal symptoms abate within 48 hours after exposure has ceased, but bronchial symptoms may require up to 10 days to abate. Additionally, a yellow staining of the hands occurs during the first 3 days of exposure. Exposure to the sun deepens these stains to orange, and although they begin to fade soon after the exposure has ceased, some degree of staining persists for several months. Other acute effects include nervousness, headaches, insomnia, oligomenorrhea, weight loss, nausea, vomiting, anorexia, spasmodic coughing, and orthopnea.

Contact dermatitis and skin sensitization are the most common chronic effects of exposure to tetryl. During World War II, tetryl was the most common cause of dermatitis among ammunition workers. The incidence of workers who were sensitized was proportional to their degree of exposure: 2% of pellet workers, but 50% of workers in dusty operations.[92,93] Additionally, the risk for contact dermatitis was highest in those workers who were exposed to significant amounts of tetryl dust. The typical rash initially takes the form of erythema of the malar areas, neck, chest, back, and ventral surfaces of the forearms. Then the affected areas develop papules and desquamate after a few days. Tolerance may develop in workers who continue to be exposed. In severe cases, the dermatitis may develop vesicles and progress to massive generalized edema with airway obstruction and substantial exfoliation from edema of the oropharynx.[18,94] Removing the patient from exposure allows the cutaneous symptoms to resolve within 2 to 4 weeks. These dermal effects result from direct irritation from the tetryl crystals, as well as from the chemical properties of tetryl. Furthermore, because tetryl is metabolized to picric acid, cross-sensitization to picric acid and its derivatives may also occur.[1]

Virtually all workers with significant exposure to dust develop a deep cough that produces a thin, mucoid sputum. The cough appears to be a result of the irritant action of the tetryl particles on the upper airway. The large size (150 μ) of tetryl particles prevents

their entry into the lower respiratory tract; consequently, radiographs of exposed workers' chests have remained normal.[1]

Evidence of systemic toxicity has been overlooked because (*a*) these effects have a long latency, (*b*) exposed workers were scattered across the country after their demobilization, and (*c*) the large turnover of workers limited the number of workers with prolonged significant exposure to tetryl. The draft and other social effects during the war contributed to the very high turnover rate—the average time worked in these factories was only about 1 year. In fact, at least three cases of systemic toxicity have been reported.[91] Systemic symptoms were noted only among workers who did not develop the more common irritative symptoms of dermatitis, conjunctivitis, and nasopharyngeal irritation.

Clinical signs of chronic tetryl exposure include moderate leukocytosis with relative lymphocytosis, anemia, and epigastric tenderness.[91,95] Upper gastrointestinal symptoms (such as gastritis or peptic ulcer disease) were frequently so aggravated that affected workers were forced to leave work. In severe cases, toxic hepatitis can occur with resultant jaundice, edema, and ascites. In several cases, hepatorenal syndrome and death have resulted.[91] Substantiating findings, including irreversible liver necrosis, renal tubular degeneration, bone marrow–depression anemia, pulmonary edema, abdominal pain, insomnia, hyperreflexia, and mental excitation, have been described in exposure studies with dogs.[91] Tetryl is now considered to be a mutagen and probable carcinogen.

In addition, pure and military-grade tetryl were found to be mutagenic in three prokaryotic test systems. Metabolic action on tetryl in salmonella (the Ames assay) reduced the chemical's mutagenicity, suggesting that tetryl is a direct-acting mutagen.[96]

TABLE 9-8

PREPLACEMENT EXAMINATION FOR TETRYL WORKERS

Component	Emphasis
Medical History	Alcohol use Tobacco use Allergies Dermatitis EENT Hematologic system Liver disease Nervous system Respiratory system Renal system Medications
Occupational History	Prior sensitivity to tetryl or picric acid Prior exposure to hepatotoxins
Physical Examination	Allergies Skin EENT Liver Nervous system Respiratory system
Tests	CBC with differential Liver function tests Renal function tests Microscopic urinalysis

Primary Prevention and Medical Surveillance

The most effective exposure control is the isolation or enclosure of dusty operations.[81] Isolating operations that use equipment such as dryers, sieves, and conveyor transfer points minimizes the contamination of other work areas. Manufacturing processes that use granules are less dusty than processes that use crystals. Using sodium sulfite–indicator soap will detect traces of tetryl that remain on the skin. This soap also converts tetryl to a soluble form, which aids in removing it from the skin. However, one control that should be avoided is job rotation, because it may favor sensitization.

Preplacement, periodic, and termination examinations are necessary for workers who could potentially be exposed to tetryl. Table 9-8 shows the content of preplacement examinations. As before, the content of periodic examinations is determined by the occupational physician on a case-by-case basis.

THE INITIATING EXPLOSIVES

The initiating explosives are a heterogeneous group of chemicals, prepared and used in very small quantities, which thus limits their potential for exposure. The most frequently used initiating explosives include lead azide, lead styphnate, and mercury fulminate (Figure 9-17). Other, less common, initiators include

Common Name	CAS Registry Number	Synonyms	Formula
Lead Azide	13424-46-9		$Pb(N_3)_2$
Lead Styphnate	15245-44-01	Lead trinitroresorcinol Lead trinitroresorcinate	$C_6H_3N_3O_8Pb$
Mercury Fulminate	628-86-4		$Hg(ONC)_2$

Fig. 9-17. The initiating explosives, together with their common names, Chemical Abstract Society numbers, synonyms, and formulae.

tetracene, which is used in commercial priming compositions; diazodinitrophenol (DDNP), which is an ingredient in primers and commercial blasting caps; and lead mononitroresorcinate (LMNR), which is used in electric detonators among a variety of applications.

Lead Azide

Lead azide [$Pb(N_3)_2$], first prepared in 1890, is produced when lead nitrate is reacted with sodium azide; sodium nitrate is the byproduct.[26] Because it is quite stable, lead azide is considered to be one of the best initiators for sensitive explosives such as tetryl, PETN, and RDX. Lead azide is usually used in combination with lead styphnate, DDNP, or PETN. In the civilian sector, it is used in cartridge primers, primer cords, and blasting caps.

Lead azide is composed of 70% lead by weight, and it releases poisonous fumes of lead and nitrogen oxides when heated. However, due to safety constraints, there is little opportunity for exposure to lead azide itself. During its manufacture, lead azide is screened in barricaded rooms to avoid continuously exposing the workers. They could be exposed intermittently while entering the screening rooms, but the workers should be wearing their respiratory protective equipment at this time.

Some exposure can occur while primers are loaded, but local exhaust ventilation is helpful. The acute effects of exposure include vasodilation and headache, while the chronic effects are those of lead intoxication. Due to these health effects, silver azide has been investigated as a substitute for lead azide, with some promise. Regulations and medical surveillance associated with exposure to lead azide should be based on the lead content. These specific requirements are dictated by Title 29, Code of Federal Regulations, Part 1910.1025.[27]

Lead Styphnate

Lead styphnate was first prepared in 1914 by von Hertz in Germany, but it was first used as an explosive by Russia early in World War I. Although lead styphnate is ignited easily, it is a relatively poor initiator, and thus is often used in combination with other primary explosives. Lead styphnate, manufactured from 2,4,6-trinitroresorcinol, magnesium oxide, and lead nitrate, may be used as a *covering charge* (ie, the booster) for lead azide, as an ingredient of priming compositions, as a component in blasting caps, and as a component in small-arms primers (eg, the M16 primer uses 4 mg of lead styphnate).

The effects on human health have not been well studied, but acute effects appear to be limited to yellow staining of the hair and skin and dermatitis.[28] Chronic exposure may result in lead toxicity, and it is the lead content of this explosive that should form the basis of monitoring and medical surveillance for exposure.

Mercury Fulminate

Mercury fulminate [$Hg(ONC)_2$], also known as mercury cyanate, was first prepared in the late 17th century, but its explosive properties were not recognized until 1800. Until recently, mercury fulminate was used as a detonator and initiator for less-sensitive explosives such as TNT; however, the military no longer uses mercury fulminate because of its poor stability. Mercury fulminate must be stored water-wet because it is sensitive to accidental detonation when dry; small amounts are dried immediately before being used.

The acute health effects of mercury fulminate include mucosal irritation and the manifestations of mercury poisoning. The most common chronic effect is sensitization dermatitis, due to exposure to dust during the manufacture of detonators. The dermatitis usually affects the face and the anterior surface of the arms.[28]

Exposure limits and medical surveillance for lead styphnate and mercury fulminate are based on their respective metal components and can be found in OSHA 29 CFR 1910.1000[9] and in the ACGIH's *Documentation of Threshold Limit Values*, [illegible] edition.[10]

COMPOSITE PROPELLANTS AND EXPLOSIVES

Composite propellants are solid rocket fuels that are being used in an increasing number of applications. As with all explosives and propellants, they consist of an oxygen donor—the oxidizer—and a hydrocarbon fuel. The oxidizer is usually an inorganic salt, while the fuel is a polymeric binder (essentially a plastic). The composites have a wide range of performance characteristics, are tremendously stable, and are inexpensive. However, they are so reactive that they corrode the metal in gun barrels.

The vast number of alternatives available for use as oxidizers and binders preclude discussion of all of them (Exhibit 9-2). Information regarding the toxicity of the inorganic salts is widely available in the toxicology and occupational medicine literature. Therefore, this discussion focuses on ammonium perchlorate, which, due probably to its cost, stability, ease of manufacture, and versatility of use, is the most widely used oxidizer in composite propellants. Ammonium perchlorate is used in the Multiple Launch Rocket System and in rocket-assisted howitzer projectiles. Workers can be contaminated via the dermal and inhalational routes during all stages of propellant production.

Before it can be used in munitions, an oxidizer must be ground and screened by particle size to assure that it will burn uniformly. Both grinding and screening raise significant levels of dust, some of which is respirable and must be controlled. The mixing of the oxidizer with the binder can also be quite dusty.

Numerous polymeric binders are in use currently. After being mixed with the oxidizer, the resultant propellant can be either cast or pressed into a mold. Cast materials are melted, then poured as a liquid into a mold, while pressed materials are kept in their solid state and shaped by simply molding or extruding. A high-temperature curing process then effects polymerization, a process that releases toxic vapors, to which the workers can be exposed. The propellant core is then removed from the mold and machined or trimmed as needed. Workers can be exposed to dust during all of these last three operations.[9]

Plastic bonded explosives are similar in concept to the composite propellants, but are designed to function as high explosives rather than as propellants. Several major groups are the PBX, PBXN, and LX-10 series. They represent a variety of mixtures combining high mechanical strength, excellent stability, and a wide range of explosive properties. They contain a high percentage of basic explosive (RDX, HMX, HNS, or PETN), which is mixed with a polymeric binder (polyester, polyurethane, nylon, polystyrene, rubbers, nitrocellulose, or Teflon), plasticizer (dioctylphthalate or butyldinitrophenylamine), and metallic fuel (powdered aluminum or iron). A major advantage of using plastic bonded explosives is that the final product can be injection (or press) loaded at ambient temperatures, or even loaded in the field. The binders are thermally degradable, so that in demilita-

EXHIBIT 9-2

TYPICAL COMPONENTS OF COMPOSITE PROPELLANTS

Binders
- Polysulfides
- Polyurethanes
- Polybutadienes
- Carboxy-terminated polybutadienes
- Hydroxy-terminated polybutadienes

Oxidizers
- Ammonium perchlorate
- Ammonium nitrate
- RDX
- HMX

Fuels
- Aluminum
- Metal hydrides

Modifiers
- Metal oxides
- Ferrocene derivatives
- Plasticizers
- Bonding agents

Adapted with permission from Lindner V. Explosives and propellants. In: Grayson M, Kirk RE, Othmer D, eds. *Abridged Version of the Encyclopedia of Chemical Technology*. New York: John Wiley & Sons; 1985: 449.

rization operations the ingredients can be completely recovered.[4]

Specific medical information regarding composite propellants and explosives is difficult to provide. For all practical purposes, the polymers are medically inert. The other components, which are heterogeneous and from different chemical families, have vastly differ-ent effects, many of which are not yet characterized.

LIQUID PROPELLANTS

The two types of liquid propellants are liquid rocket propellants and liquid gun propellants. Both the National Aeronautics and Space Administration and the U.S. Air Force use liquid rocket propellants in high-performance missile systems. The armed services are currently developing liquid gun propellants for use in large-caliber weapons such as the 120-mm main tank cannon, 105-mm howitzer, 155-mm howitzer, and 8-in howitzer.

Rocket Propellants

Many chemicals have been used as components of liquid rocket propellants (Exhibit 9-3). Many are used extensively in industry and are well covered in standard toxicology and occupational medicine texts; most of them have only limited military use and therefore will not be discussed in this chapter. The liquid rocket propellants that do have military applications include (*a*) hydrazine, (*b*) nitrogen tetroxide, and (*c*) the boranes.

Hydrazine is widely used in the chemical industry, where most of the studies of the effects on human health have been conducted. Studies on humans and animals have demonstrated deleterious health effects. The effects on humans have been limited to irritations of the skin and mucosa and hepatic disorders, but the effects found in animal studies have been more serious. Mice have developed hepatomas after being fed hydrazine, rats exposed to hydrazine vapor have developed nasal tumors, and hamsters have developed lung adenomas. Urinary levels of hydrazine have shown some utility in monitoring exposure. At a minimum, medical surveillance should periodically assess erythrocyte indices, hypoglycemia, kidney and liver disease, hemorrhagic diathesis, and allergy to phenylhydrazine and isoniazid.[100]

Nitrogen tetroxide has also been used widely in the space program, but not without harmful health effects. The vapors can cause immediate or delayed swelling and blistering of the adnexa oculi and severely burn the dermis. When nitrogen tetroxide is inhaled, it can react with moisture to form nitric acid and cause delayed pulmonary edema.[5]

EXHIBIT 9-3

TYPICAL COMPONENTS OF LIQUID ROCKET PROPELLANTS

Fuel	Oxidizer
Methyl alcohol	Hydrogen peroxide
Ethyl alcohol	Liquid oxygen
Isopropyl alcohol	Red and white fuming nitric acid
Furfuryl alcohol	Nitrogen tetraoxide
Anhydrous ammonia	Liquid fluorine
Aniline	Oxygen difluoride
Butyl mercaptan	Ozone difluoride
Propyl mercaptan	Ethylene oxide
Amyl mercaptan	
Hydrazine	**Other**
Monomethyl hydrazine	Bromine pentafluoride
Unsymmetrical dimethyl hydrazine	Chloride trifluoride
Mixed amines	Pentaborane
Nitromethane	Triethyl boron
Tetranitromethane	
Aliphatic hydrocarbons	

During the past 30 years, the fuel boron hydride and its derivatives—also known as *boranes*—have become widely used within industry and rocketry as rubber vulcanizers, corrosion inhibitors, and in other chemical processes. The reactivity of the boranes has led to a proliferation of uses, but has also contributed to their significant toxicity. Regardless of their use, boranes are toxic to the respiratory system, cardiovascular system, CNS, skin, kidneys, and liver.

Carboranes—boranes that contain carbon in addition to boron and hydrogen—have recently been developed and investigated for use in solid-fuel systems. The carboranes are skin irritants, but they do not sensitize. They appear to have relatively low acute toxicity. Subchronic inhalation exposure in dogs resulted in interstitial pneumonitis and early emphysematous changes, but no developmental effects were noted.[101]

Gun Propellants

Liquid gun propellants have several advantages over conventional solid propellants for use in self-propelled howitzers and naval vessels: they are less expensive to produce and transport, less vulnerable to secondary ignition, easier to store in combat vehicles, and are demilitarized more safely and easily than solid propellants.[102] One disadvantage, however, is that more workers can be exposed to the chemical components during the manufacture, transport, and use of liquid than solid propellants.[103]

The liquid gun propellants of current interest consist of aqueous solutions of hydroxylammonium nitrate (HAN) mixed with either trimethanolammonium nitrate or triethanolammonium nitrate. No studies on the effects on human health have been reported on either of the mixtures or the individual components. However, the aqueous solutions and pure HAN have been evaluated for mammalian toxicity at the U.S. Army Environmental Health Agency (USAEHA) and at the U.S. Army Letterman Army Institute of Research.[103-109] The mixtures were found to be moderately toxic to both rats and rabbits: for male rats, the oral LD_{50} was 822 mg/kg, and for female rats, 520 mg/kg; for male rabbits, the oral LD_{50} was 101 mg/kg.[104] Oral exposure to the mixtures induces cyanosis, respiratory distress, and, at high doses, death.[104] A single intragastric dose of 400 mg/kg produced no ECG changes in dogs. Treatment with methylene blue rapidly reversed the acute toxic effects. The mixtures were also found to be ocular irritants, but were not corrosive to the cornea.[103] However, exposure to mixtures induced hematological changes: methemoglobinemia occurred; oxygen tension decreased; free nitrites, Heinz bodies, and crenated erythrocytes formed; and, at lower doses, serum potassium decreased.[104]

The armed forces have also used rabbits to test the health effects of HAN. When applied to the dermis, it caused chronic and ulcerative dermatitis, and at higher doses, caused hemolytic anemia in addition to the systemic effects described previously for exposure to the mixture; however, no blood chemistry changes were noted.[105] When administered orally to three groups of rabbits (1, 5, and 25 mg/kg/d) for 21 days, HAN induced splenic congestion and hyperplasia of the reticuloendothelial system at all doses.[106] At 25 mg/kg/day, HAN caused anemia and myeloid hyperplasia of the bone marrow.[106] Inhalation of aerosolized HAN has been found to induce Heinz-body formation and upper-respiratory irritation.[107] Several other liquid gun propellants have also been investigated as aerosols, and the effects they elicited were qualitatively similar to those of their principal ingredient, HAN.[108]

The Occupational and Environmental Medicine Division of USAEHA has established preliminary guidelines for medical surveillance and a provisional PEL of 3 mg/m^3 has been proposed for liquid gun propellants.[107,110] Testing for methemoglobinemia or examining the peripheral blood for Heinz bodies may comprise part of the appropriate medical monitoring for exposed employees. Reasonable occupational precautions include: restricting employees from eating, drinking, and smoking in areas where these chemicals are handled or stored; ensuring adequate ventilation; preventing spills and splashes; and using PPE such as splash goggles and gloves.

SUMMARY

In defending the United States, military and civilian personnel must necessarily produce, store, and handle a variety of munitions. In the army, these operations occur around the country at various arsenals, proving grounds, depots, and ammunition plants, which together employ more than 100,000 workers. Despite incomplete laboratory studies and imperfect data, information has been gathered during the last 50 years on the effects of workplace exposures to these chemicals, much of it recorded during wartime while large quantities were being produced.

The chemical families represented among energetic

materials include aliphatic nitrate esters (such as nitroglycerin), nitroaromatics (such as TNT), and nitramines (such as RDX). Considering the properties of the energetic materials—explosives, propellants, and pyrotechnics—it was inevitable that they would be utilized in military weapons. Explosives create a shock wave that progresses rapidly, while propellants release large amounts of hot gas in a more controlled manner. Pyrotechnics burn slowly, emitting tremendous heat or light. Most modern weapons utilize energetic compounds in combination, capitalizing on their individual properties.

As these energetic materials are synthesized and assembled into munitions, workers can be exposed to the raw materials, the finished product, or any number of chemical intermediates along the way. These chemicals are usually absorbed via the dermal, inhalational, and, less importantly, the ingestional routes; as a class they can produce dermatitis, methemoglobinemia, vasodilation, or cancer. The standard industrial hygiene principles of engineering and administrative controls and PPE can minimize these exposures. Obviously, the explosive properties of these chemicals necessitate strict compliance with safety guidelines. Preplacement screening and periodic surveillance must be tailored to the specific hazards in each industrial operation and at each site. Generalized medical guidance regarding these mixtures has little practical significance.

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REFERENCES

1. Bergman BB. Tetryl toxicity: A summary of ten years' experience. *Arch Indust Hyg Occup Med*. 1952;51:10–20.
2. McConnell WJ, Flinn RH, Brandt AD. Occupational diseases in government-owned ordnance explosives plants: Observations on their prevalence and control during World War II. *Occup Med*. 1946;1:551–618.
3. Cook WL. Occupational health and industrial medicine. In: *Preventive Medicine in World War II*. Vol 9, *Special Fields*. Washington, DC: DA, Office of The Surgeon General; 1969.
4. US Department of the Army. *Military Explosives*. Washington, DC: DA; 1985 . Technical Manual 9-1300-214.
5. Meidl JH. *Explosive and Toxic Hazardous Materials*. Beverly Hills, Calif: Glencoe Press; 1970.
6. McConnell WJ, Flinn RH. Summary of twenty-two trinitrotoluene fatalities in World War II. *J Indust Hyg and Tox*. 1946;28:76–83.
7. Kneessy AD. *Army Occupational Health and AEHA*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1981.
8. Hunter D. *The Diseases of Occupations*. 6th ed. London: Hodder and Stoughton; 1978.
9. 29 CFR, Part 1910 § 1000.
10. US Department of Defense. *Ammunition and Explosives Safety Standards*. Washington, DC: DoD; 1984. DoD Manual 6055.9-STD.
11. US Department of the Army. *Ammunition and Explosives Safety Standards*. Washington, DC: DA; 1987. Army Regulation 385-64.

12. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. *NIOSH Pocket Guide to Chemical Hazards*. Washington, DC: US. 1990. DHHS (NIOSH) Publication 90-117.

13. Seifert HE, Cralley LJ, Clayton GD. Industrial health hazards and their control in a bomb loading plant. *Indust Med*. 1945;14(1):75–80.

14. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health; US Department of Labor, Occupational Safety and Health Administration. *Occupational Health Guidelines for Chemical Hazards*. Washington, DC: USDHHS; USDOL; 1981. DHHS (NIOSH) Publication 81-123.

15. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. *NIOSH Guide to Industrial Respiratory Protection*. Washington, DC: USDHHS; 1987. DHHS (NIOSH) Publication 87-116.

16. Americans with Disabilities Act of 1990. Pub L No. 101-336. July 26, 1990.

17. Parmeggiani L, ed. *Encyclopedia of Occupational Health and Safety*. Vol 2, 3rd ed (rev). Geneva, Switzerland: International Labour Office; 1983.

18. Schwartz L, Tulipan L, Birmingham DJ. Dermatitis from explosives. In: *Occupational Disease of the Skin*, 3rd ed. Philadelphia: Lea & Febiger; 1957: 439–456.

19. Mansouri A. Review: Methemoglobinemia. *Am J of the Medical Sciences*. 1985;289(5):200–209.

20. Rieder RF. Methemoglobinemia and sulfhemoglobinemia. In: Wyngaarden JB, Smith LH, eds. *Cecil Textbook of Medicine*. 18th ed. Philadelphia: WB Saunders; 1988: 945–947.

21. Beard RR, Noe JT. Aromatic nitro and amino compounds. In: Clayton GD, Clayton FE, eds. *Patty's Industrial Hygiene and Toxicology*. 3rd ed. New York: John Wiley; 1981: 2413–2491.

22. Smith RP. Toxic responses of the blood. In: Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology*, 3rd ed. New York: Macmillan Publishing; 1986.

23. US Department of the Army. *Military Explosives*. Picatinny Arsenal, NJ: AMCCOM; 1984. Technical Manual 9-1300-214.

24. Lindner V. Explosives and propellants. In: Mark HF, Othmer DF, Overberger CG, Seaborg GT, eds. *Encyclopedia of Chemical Technology*. Vol 9, 3rd ed. New York: John Wiley & Sons; 1980.

25. Laws GC. The effects of nitroglycerin upon those who manufacture it. *JAMA*. 1898;31:793–794.

26. US Department of Health, Education, and Welfare; Public Health Service; Centers for Disease Control; National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard, Occupational Exposure to Nitroglycerin and Ethylene Glycol Dinitrate*. Washington, DC: USDHEW; 1978.

27. Shiotsuka RN. *Occupational Health Hazards of Nitroglycerin with Special Emphasis on Tolerance and Withdrawal Effects—A Literature Review*. Fort Detrick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1979. USAMBRDL Technical Report 7903.

28. Stayner LT. *Cardiovascular Mortality Among Munitions Workers Exposed to Nitroglycerin*. Chapel Hill, NC: University of North Carolina; 1989. Dissertation.

29. Gordon L, Hartley WR. *Trinitrotoluene Health Advisory*. Washington, DC: US Environmental Protection Agency, Office of Drinking Water; 1989.

30. Abrams J. A reappraisal of nitrate therapy. *JAMA*. 1986;259(3):396–401.

31. Needleman P, Johnson EM Jr. Vasodilators and the treatment of angina. In: Gilman AG, Goodman LS, Gilman A, eds. *Goodman and Gilman's The Pharmacological Basis of Therapeutics*. 6th ed. New York: Macmillan Publishing; 1980: 819–833.

32. Hogstedt C. Ethylene glycol dinitrate and nitroglycerin. In: Zenz C, ed. *Occupational Medicine, Principles and Practical Applications*. 2nd ed. Chicago: Year Book Medical Publishers; 1988: 730–734.

33. Posadas del Rio FA, Juarez FJ, Garcia RC. Biotransformation of organic nitrate esters in vitro by human liver, kidney, intestine, and blood serum. *Drug Metabolism and Disposition*. 1988;16(3):477–481.

34. Abrams J. Pharmacology of nitroglycerin and long-acting nitrates. *Am J Cardiol*. 1985;56:12A–18A.

35. Symanski H. Severe health injuries resulting from occupational exposure to nitroglycol. *Archiv fuer Hygiene und Bakteriologie*. 1952;136:139–158.

36. Ringenburg VL, Bloom TF. *Industrial Hygiene Survey Report No. IWS-113-10, Radford Army Ammunition Plant*. Cincinnati, Oh: USDHHS; NIOSH; Div, Surveillance, Hazard Evaluations and Field Studies; 1984.

37. Munch JC, Friedland B, Shepard M. Glyceryl trinitrate II, chronic toxicity. *Industrial Medicine and Surgery*. 1965;34:940–943.

38. Daum S. Nitroglycerin and alkyl nitrates. In: Rom WN, ed. *Environmental and Occupational Medicine*. Boston: Little, Brown; 1983: 639–648.

39. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer. *Occupational Safety and Health Guidelines for Chemical Hazards*. Washington, DC: USDHHS; 1988. DHHS (NIOSH) Publication 88-118, Supplement 1-OHG.

40. Lauwerys RR. *Industrial Chemical Exposure: Guidelines for Biological Monitoring*. Davis, Calif: Biomedical Publications; 1983.

41. Fukuchi Y. Nitroglycol concentrations in blood and urine of workers engaged in dynamite production. *Int Arch Occup Environ Health*. 1981;48:339–346.

42. Harrison RJ. Chemicals. In: LaDou J, ed. *Occupational Medicine*, Norwalk, Conn: Appleton & Lange; 1990.

43. US Army Environmental Hygiene Agency. *Medical and Industrial Hygiene Considerations for Nitroglycerin Operations*. Aberdeen Proving Ground, Md: USAEHA; 1976. AEHA Technical Guide 024.

44. Hogstedt C, Stahl R. Skin absorption and protective gloves in dynamite work. *Am Indust Hyg Assoc J*. 1980;41:367–372.

45. Horvath EP, Ilka RA, Boyd J, Markham T. Evaluation of the neurophysiologic effects of 1,2-propylene glycol dinitrate by quantitative ataxia and oculomotor function tests. *Am J Indust Med*. 1981;2:365–378.

46. Burrows EP, Rosenblatt DH, Mitchell WR, Parmer DL. *Organic Explosives and Related Compounds: Environmental and Health Considerations* Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1989. USAMBRDL Technical Report 8901.

47. Forman SA, Helmkamp JC, Bone CM. Cardiac morbidity and mortality associated with occupational exposure to 1,2 propylene glycol dinitrate. *J Occup Med*. 1987;29(5):445–450.

48. Kimmel EC, Houston WE, Kutzman RS, Carpenter RL, eds. *Toxic Hazards Research Unit Annual Report: 1986*. Dayton, Oh: Northrop Services, Environmental Sciences; 1987.

49. Von Oettingen WF, Donahue DD, Lawton AH, Monaco AR, Yagoda H, Valaer PJ. *Toxicity and Potential Dangers of Pentaerythritol-tetranitrate (PETN)*. Washington, DC: Federal Security Agency, US Public Health Service; 1944. PHS Bulletin 282.

50. US Environmental Protection Agency, Office of Drinking Water. *Nitrocellulose Health Advisory*. Washington, DC: USEPA; 1987.

51. Rosenstock L, Cullen MR. *Clinical Occupational Medicine*. Philadelphia: WB Saunders; 1986.

52. Small MJ, Rosenblatt DH. *Munitions Production Products of Potential Concern as Waterborne Pollutants—Phase II*. Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1974. USAMBRDL Technical Report 7404.

53. US Army Environmental Hygiene Agency. *Industrial Hygiene Evaluation Guide*. Aberdeen Proving Ground, Md: USAEHA; 1975. USAEHA Technical Guide 022.

54. US Environmental Protection Agency, Office of Drinking Water. *Trinitrotoluene Health Advisory*. Washington, DC: USEPA; 1989.

55. Zakhari S, Villaume JE, Craig PN. Occupational health and safety aspects of 2,4,6-trinitrotoluene (TNT). In: *A Literature Review—Problem Definition Studies on Selected Toxic Chemicals*. Vol 3. Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1978.

56. Hathaway JA. Subclinical effects of trinitrotoluene: A review of epidemiology studies in toxicity. In: Rickert DE, ed. *Toxicity of Nitroaromatic Compounds*. Washington, DC: Hemisphere Publishing; 1985: 255–274.

57. Sievers RF, Lawton AH, Skoog F, et al. *A Medical Study of the Effect of TNT on Workers in a Bomb and Shell Loading Plant: Report of a Fatal Case of Aplastic Anemia*. Washington, DC: Federal Security Agency, US Public Health Service; 1945. PHS Bulletin 291.

58. Ahlborg G, Einisto P, Sorsa M. Mutagenic activity and metabolites in the urine of workers exposed to trinitrotoluene (TNT). *Brit J Indust Med*. 1988;45:353–358.

59. Levine BS, Rust JH, Burns JM, Lish PM. *Determination of the Chronic Mammalian Toxicological Effects of TNT: Twenty-six Week Subchronic Oral Toxicity Study of Trinitrotoluene (TNT) in the Beagle Dog, Phase 2, Final Report*. Washington, DC: IIT Research Institute, US Army Medical Research and Development Command, 1983.

60. Morton AR, Ranadive MV. *Occupational Health Study 32-093-74/75, Newport Army Ammunition Plant*. Fort Sam Houston, Tex: US Army Health Services Command; 1974.

61. Schwartz L. Industrial dermatitis—In our war industries. *Indust Med*. 1942;11(10):457–462.

62. Harkonen H, Karki M, Lahti A, Savolainen H. Early equatorial cataracts in workers exposed to trinitrotoluene. *Am J Ophthalmol*. 1983;95:807–810.

63. Savolainen H, Tenhunen R, Harkonen H. Reticulocyte haem synthesis in occupational exposure to trinitrotoluene. *Brit J Indust Med*. 1985;42:354–355.

64. Sievers RF. *An Evaluation of Neurologic Symptoms and Findings Occurring Among TNT Workers*. Washington, DC: Division of Federal Security Agency, US Public Health Service; 1947. PHS Report 196.

65. Reed HL, McGee LC, McCausland A. Multivitamin capsules and the health of workers in trinitrotoluene. *Occup Med*. 1948;1:351–362.

66. Morton AR, Ranadive MV, Hathaway JA. Biological effects of trinitrotoluene from exposure below the Threshold Limit Value. *Am Indust Hyg Assoc J*. 1976;37:56–60.

67. Sievers RF, Stump RL; Monaco AR. Aplastic anemia following exposure to trinitrotoluene: Report of three cases. In: Sawyer WA, ed. *Occupational Medicine*. Vol 1. Chicago: American Medical Association; 1946: 351–362.

68. Buck CR, Wilson SE. *Adverse Health Effects of Selected Explosives (TNT, RDX)*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1975. USAEHA Occupational Health Special Study 32-049-75/76.

69. Snyder RK. Metabolites of 2,4,6-trinitrotoluene (TNT) excreted in the urine of dogs. *J Indust Hyg Toxicol*. 1946;28(3):59–75.

70. Woollen BH, Hall MG, Craig R, Steel GT. Trinitrotoluene: Assessment of occupational absorption during manufacture of explosives. *Brit J Indust Med.* 1986;43:465–473.

71. Ahlborg G, Bergstrom B, Hogstedt C, Einisto P, Sorsa M. Urinary screening for potentially genotoxic exposures in a chemical industry. *Brit J Indust Med.* 1985;42:691–699.

72. Decad GM, Graichen ME, Dent JG. Hepatic microsomal metabolism and covalent binding of 2,4-dinitrotoluene. *Toxicology and Applied Pharmacology* 1982;62(2):325–334.

73. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. *Dinitrotoluenes (DNT).* Washington, DC: USDHHS; 1985. NIOSH Current Intelligence Bulletin 44, DHHS (NIOSH) Publication 85-109.

74. Rickert DE, Butterworth BE, Popp JA. Dinitrotoluene: Acute toxicity, oncogenicity, genotoxicity, and metabolism. *CRC Critical Review in Toxicology.* 1984;13(3):217–234.

75. Kedderis GL, Rickert DE. Nitrotoluenes: Metabolism and bioactivation. *CIIT Activities.* 1984;4(2):1,4–6.

76. Levine RJ. Dinitrotoluene: Human atherogen carcinogen, neither or both? *CIIT Activities.* 1987;7:1–5.

77. Levine RJ, Andjelkovich DA, Kersteter SL, et al. Heart disease in workers exposed to dinitrotoluene. *J Occup Med.* 1986;28(9):811–816.

78. Hamill PVV, Steinberger E, Levine RJ, Rodriguez-Rigau LJ, Lemeshow S, Avrunin JS. The epidemiologic assessment of male reproductive hazard from occupational exposure to TDA and DNT. *J Occup Med.* 1982;24(12):985–993.

79. American Conference of Governmental Industrial Hygienists. Picric acid. In: *Documentation of the Threshold Limit Values and Biological Exposure Indices.* 5th ed. Cincinnati, Oh: ACGIH; 1986.

80. Brandt AD. Engineering control of air contamination of the working environment. In: Gafafer WM, ed. *Manual of Industrial Hygiene and Medical Service in War Industries.* Philadelphia: WB Saunders; 1943; 198–266.

81. Back S. Developments in high-explosives for artillery shells. *International Defense Review.* 1984;17:1748–1750.

82. Mura WR, Small MJ, Rosenblatt DH, Dacre JC. *Munitions-Related Substances of Potential Concern as Airborne Pollutants.* Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1974. USAMBRDL Technical Report 7409.

83. Small MJ. *The Hazard Ranking and Allocation Methodology: Evaluation of TNT Wastewaters for Continuing Research Efforts.* Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1978. USAMBRDL Technical Report 7808.

84. Kaplan AS, Berghout CF, Peczenik A. Human intoxication from RDX. *Arch Environ Health.* 1965;10:877–883.

85. Ketel WB, Hughes JR. Toxic encephalopathy with seizures secondary to ingestion of composition C-4. *Neurol.* 1972;22:871–876.

86. McLellan WL, Hartley WR, Brower ME. *Health Advisory for Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX).* Washington, DC: US Environmental Protection Agency, Office of Drinking Water; 1988.

87. Von Oettingen WF, Donahue DD, Yagoda H, Monaco AR, Harris MR. Toxicity and potential dangers of cyclotrimethylenetrinitramine (RDX). *J Indust Hyg Toxicol.* 1949;31:21–31.

88. Woody RC, Kearns GL, Brewster MA, et al. The neurotoxicity of cyclotrimethylenetrinitramine (RDX) in a child: A clinical and pharmacokinetic evaluation. *J Toxicol Clin Toxicol.* 1986;24(4):305–319.

89. McLellan, Hartley WR, Brower ME. *Health Advisory for Octahydro-1,3,5,7-tetranitro 1,3,5,7-tetrazocine (HMX).* Washington, DC: US Environmental Protection Agency, Office of Drinking Water; 1988.

90. Frost DG, Morgen EW, Letellier Y, et al. *Ninety-day Subchronic Oral Toxicity Study of Nitroguanidine in Mice*. San Francisco: Letterman Army Institute of Research; 1988. Final Report, Technical Report No. 319.

91. Hardy HL, Maloof CC. Evidence of systemic effect of tetryl with summary of available literature. In: *Ind Hyg Occupational Med*. 1950:545–555.

92. Foussereau J, Benezra C, Maibach HI, Hjorth N. Army arsenals. In: *Occupational Contact Dermatitis, Clinical and Chemical Aspects*. Philadelphia: WB Saunders; 1982.

93. Troup HB. Clinical effects of tetryl (CE powder). *Brit J Indust Med*. 1946;3(1):20–23.

94. Schwartz L. Occupational dermatoses. In: Gafafer WM, ed. *Manual of Industrial Hygiene and Medical Service in War Industries*. Philadelphia: WB Saunders; 1943; 161–197.

95. Witkowski LJ, Fischer CN, Murdock HD. Industrial illness due to tetryl. *JAMA*. 1942;119:1406–1409.

96. Whong W, Spencer ND, Edwards GS. Mutagenic activity of tetryl, a nitroaromatic explosive, in three microbial test systems. *Toxicology Letters*. 1980;5:11–17.

97. 29 CFR, Part 1910 § 1025.

98. American Conference of Government Industrial Hygienists. *Documentation of the Threshold Limit Values*. 6th ed. Cincinnati, Oh: ACGIH; 1991.

99. Hannum JAE, ed. Solid propellants and ingredients. In: *Hazards of Chemical Rockets and Propellants*. Vol 2. Laurel, Md: Chemical Propulsion Information Agency, The Johns Hopkins University Applied Physics Laboratory. 1984. CPIA Publication 394.

100. US Department of Health and Human Services. *Criteria for a Recommended Standard . . . Occupational Exposure to Hydrazines*. Washington, DC: USDHHS, NIOSH; 1978. DHHS (NIOSH) Publication 78-172.

101. McCreesh AH. Toxicological evaluation of n-hexyl carborane, carboranyl-methylethyl sulfide, and carboranyl-methylpropyl sulfide. In: *Proceedings of the 7th Annual Conference on Environmental Toxicology, October 1976*. Dayton, Oh: US Air Force Aerospace Medical Research Laboratory; 1977. AMRL Technical Report 076-125.

102. Pengelley R. Liquid propellant artillery proving begins in the US. *International Defense Review*. 1989;22:1082–1083.

103. Justus JD, Korte DW Jr. *Circulatory and Hematological Effects of Liquid Propellant 1846*. San Francisco: Letterman Army Institute of Research; 1988. LAIR Report 299.

104. Metker LW. *Physiological and Pharmacological Effects Following Oral Administration of Monopropellants, Phase I*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1979. USAEHA Report 75-51-0132-80.

105. Asaki AE. *Health Hazard Evaluation of Liquid Monopropellants: Effects of Dermal Administration of Hydroxylammonium Nitrate, Phase 2*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1982. USAEHA Report 75-51-0132-82.

106. Asaki AE, Harvey JG Jr, Haight EA, Metker LW. *Health Hazard Evaluation of Liquid Monopropellants: Range Finding Studies on the Effects of Hydroxylammonium Nitrate in Animals, Phase 3*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1984. USAEHA Report 75-51-0132-84.

107. Snodgrass HL, Asaki AE, Harvey JG Jr, Metker LW. *Health Hazard Evaluation of Liquid Monopropellants: Subchronic Inhalation of Hydroxylammonium Nitrate, Phase 4*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1985. USAEHA Report 75-51-0132-85.

108. Snodgrass HL. *Health Hazard Evaluation of Liquid Monopropellants: Comparative Inhalation Toxicities of LGP 1776, LGP 1845, and HAN, Phase 5*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1985. USAEHA Report 75-51-0132-85.

109. Orner GA, Korte DW Jr. *Circulatory and Hematological Effects of Liquid Propellant 1846 Following Oral Administration to Rats*. San Francisco: Letterman Army Institute of Research; 1988. LAIR Report 270.

110. Directorate of Occupational and Environmental Health. *Occupational Health Guide for Hydroxylammonium Nitrate (HAN) and Liquid Propellant Formulations Containing HAN*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency, Occupational and Environmental Medicine Division, Directorate of Occupational and Environmental Health; 1991.

Chapter 10

COMBUSTION PRODUCTS OF PROPELLANTS AND AMMUNITION

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INTRODUCTION

Soldiers and civilians whose work includes firing weapons, launching rockets, and demilitarizing ammunition, or who work around conflagrations that involve ammunition (or tactical vehicles that contain ammunition) encounter potentially hazardous exposures to combustion products. These exposures are related to the combustion of propellants and other chemicals that provide the energy for firing a projectile or rocket, the materials that are contained within the projectile, the interaction between the projectile and the firing tube or barrel, and numerous other chemical and physical events that occur during firing.

Propellants are chemicals that rapidly liberate large volumes of gases under high pressure and temperature. Military personnel probably have been exposed to the combustion products of propellants for more than 1,000 years.[1] These *energetic* gases (their reaction is exothermic) propel a projectile through and out of a gun tube or power the reaction motor when a rocket is launched. Propellants differ from other energetic materials such as fuels in their method of combustion: fuels require ambient oxygen to burn, whereas the oxidant is a chemical constituent of a propellant. The combustion of most propellants cannot be quenched once it has begun.

This chapter primarily considers the risks of exposure via inhalation, the skin, and the eyes to the major combustion products of commonly used solid propellants—carbon monoxide, ammonia, hydrogen chloride, oxides of nitrogen and sulfur, and lead—in army weapons systems. (Exposures to carbon monoxide and lead are such significant hazards to military personnel that they are considered in separate chapters of this textbook.) Potentially hazardous substances contained in projectiles (such as depleted uranium [DU], zinc, and lead) are also addressed. The U.S. Army has not used liquid propellants to any great extent, mainly because solid propellants have met operational requirements and were less expensive. Liquid propellants are considered to be potentially hazardous: they may contain chemicals that (*a*) are toxic (strong acids) and (*b*) could burn rapidly if the components come into contact with each other. The Lance missile system uses liquid propellants and has had an extremely good safety record despite both the theoretical hazards and considerable operational movement—including airdrops.[2] One system that currently uses liquid propellants has had operational problems: the Scud missiles that the Iraqi army used against Israel and the allied forces during Operation Desert Storm were charged with liquid propellants; the Scuds, fueled in the field, took longer than 1 hour to charge, and could only be charged one at a time.[2]

Conventional weapons of immense destructive power have been developed during the 20th century: effective rapid-fire weapons; recoilless weapons, rockets, and missiles, all of which liberate large amounts of combustion products per unit of time; weapons that are operated in confined spaces and in various urban locales; and tightly enclosed vehicles that can be used as weapons platforms for operations in chemically contaminated environments. A side effect of utilizing these weapons is that the soldiers who operate them are exposed to the combustion products of propellants.

Casualties of inhalational exposures to these combustion products have definitely occurred, but published case reports are not readily available in the medical literature of any language.[3,4] Several factors have minimized the number of reports documenting the adverse effects of these exposures. During weapons firing, the exposure settings are characterized by short-lived clouds of contaminants, with high peaks of pollution that rapidly dissipate. Exposures like these are very difficult to measure, particularly under field conditions. Sporadic, episodic weapons firing during training and battle operations deprives medical observers of a steady-state experience, such as would be found in an industrial setting where the same or similar operations are performed in the same location day after day. The scant information derived from field observations is often secluded in classified documents, or documents with limited accessibility, and often with little or no verification by the civilian or even the military medical communities.

One documented instance of carbon monoxide intoxication occurred at Fort Hood, Texas, in February 1984. It involved an M1E1 Abrams tank undergoing operational testing. This rare report of carbon monoxide intoxication is discussed in Chapter 11, Carbon Monoxide. Anecdotal evidence is not so rare; tank design was implicated as an important factor contributing to inhalational exposure, and poor crew conditions inside Soviet-made tanks that were captured by the Israeli Defense Forces during the Six-Day War (1967) have been described:

> One of the main drawbacks of the original Soviet [T-55] design was the total disregard of human engineering for crew members. The Soviets pick the smallest of their recruits to serve in tank units, but even these

midgets did not feel too happy in the confines of their tank turret and driver's compartment. Bad ventilation in the cramped interior caused fatigue and exhaustion reducing combat efficiency and endurance. In fact, Arab tank crews, overcome by deadly fumes and heat stress under severe climatic conditions, often abandoned their tanks, which were picked up perfectly intact.[5p18]

The U-STS (2A20) 115-mm smoothbore gun, at first, seems a very efficient tank gun, [but] its combat effectiveness leaves much to be desired. The firing sequences present most of the drawbacks. The gun's exhaust fumes are overwhelming and the fighting compartment soon fills with carbon monoxide, despite the bore evacuator designed to remove fumes. Poor ventilation causes combat fatigue, and crews have been known to abandon their mounts totally exhausted, choking from the poisonous fumes.[5p17]

By bringing the potential risks and problems related to the use of various weapons to the attention of medical personnel, those who care for soldiers will be prepared to recognize, treat, report, and, ideally, eventually minimize the adverse effects. Military physicians must take the time to describe and report the clinical details and exposure events surrounding workplace-related injuries and illnesses so that our ability to prevent morbidity and mortality can be improved. U.S. Army Medical Department (AMEDD) officers are required to report actual overexposures or any suspected clustering of symptoms in soldiers regardless of whether these occur in garrison or in the field. In these situations, the Special Telegraphic Report of Selected Condition (RCS MED-16[R4]—Requirement Control Symbol, Medical), which is required by army regulations, must be used.[6,7] This report is essential; it enables occupational health personnel to assess the effectiveness of previously established engineering, administrative, or personal protective controls and to identify areas where the need for controls was overlooked. Eventually, these reports will be used by design engineers to eliminate or reduce hazardous exposures in military ordnance, vehicles, and equipment that are being developed for future use. The ultimate objective is the primary prevention of morbidity and mortality and the elimination or reduction of exposures that diminish soldier performance.

HISTORY OF PROPELLANTS

Perhaps the first recorded uses of propellants were by the Byzantine defenders of Constantinople against the Saracen fleet in AD 673 and 717.[1] The Byzantines used *Greek Fire*, a flaming propellant that was itself a chemical weapon, generating acrid smoke. Greek Fire was probably made of a powdered mixture of sulfur, naphtha, and quicklime. By the 13th century, Mediterranean Muslims launched small stone balls that were propelled by *black powder*, a mixture of sulfur, charcoal, and saltpeter (potassium nitrate), through tubes.[1] The origin of black powder remains a mystery, but many believe the Chinese were the first to use it, probably in fireworks and signals.[8] By the 14th century, Europeans reportedly imported black powder from China. Black powder has been used for nearly two millennia, and is still used (in approximate proportions: potassium nitrate, 75%; charcoal, 15%; and sulfur, 10%) to a limited degree in modern munitions such as low-velocity guns, rockets, and as an igniter of larger propellants.[9]

Many renowned scientists contributed to the fields of propellants and the related discipline of ballistics. Vannoccio Biringuccio, a Florentine engineer, described a variety of weaponry—including grenades, flame throwers, and portable projectile hurlers (the forerunners of contemporary small-caliber arms)—in his 15th-century book, *The Pirotechnia of Vannoccio Biringuccio*.[10] Biringuccio analyzed the mechanical process of propellant combustion and also described the deleterious effects of inhaling combustion products.

By the early 17th century, King Gustavus Adolphus of Sweden established the first true field artillery, which consisted of heavy, iron, firing tubes on wheels; propellant in the form of black powder; and ball projectiles. He established artillery (and therefore propellants) as an important component of the battlefield armamentarium, and he laid the foundation for a flourishing Swedish arms industry (Figures 10-1 and 10-2).[11]

Dramatic improvements in propellants evolved during the 19th century:

Modern propellants date from 1845, when a German chemist, Christian Schoenbein, discovered nitrocellulose, which burns completely leaving no solid residues. Gunpowder [black powder] by comparison produces over half its weight as solid residue. A satisfactory propellant in the form of cakes was first produced in 1884 by a French physicist, Paul Vieille, by gelatinizing nitrocellulose with an ether-alcohol mixture. This was used by the French army under the name of Poudre B. Alfred Nobel produced a similar propellant by using nitroglycerine instead of ether alcohol. Abel in Britain gelatinized nitrocellulose ... using a mixture of nitroglycerine and vaseline [petro-

Fig. 18-1. Gustavus II Adolphus, called Gustavus Adolphus, was King of Sweden from 1611 to 1632. He reorganized tactical military units and effectively integrated artillery to achieve increased firepower and mobility. He also revolutionized the manufacture and use of artillery including standardizing components used in the manufacture of artillery pieces; increasing the mobility of artillery by shortening barrels; and increasing the rate of fire through the use of premeasured powder charges in bags. He established Swedish artillery as the best in the world. Source: Holmquist BM, Gripstad B. *Swedish Weaponry Since 1630*. The Defence Matériel Administration of Sweden. The Royal Army Museum. Arlöv, Sweden: Berlings; 1982.

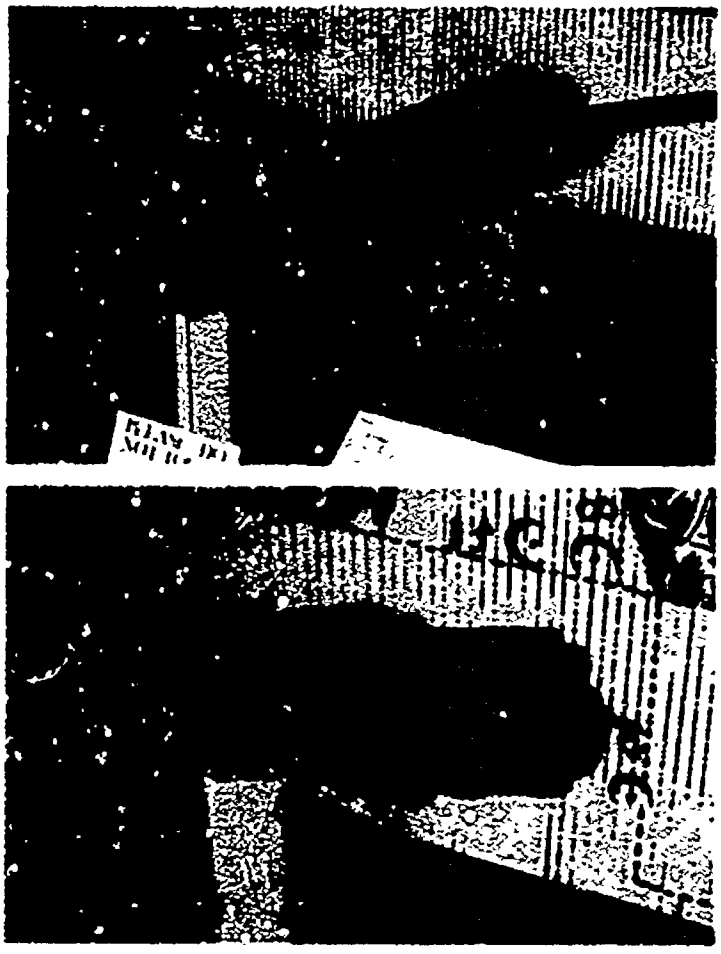

Fig. 18-2. By the middle of the 17th century, Sweden had developed a diversified and extremely productive arms industry that produced cannons, muskets, pistols, and many other items for its own army and for export. The two 17th-century weapons shown to the left were attempts during the black-powder era to develop rapid-fire guns. The *skottbelk* (organ-gun), (a), had twenty 20-mm barrels and was used as a defensive weapon. To fire the weapon, powder was placed in grooves in the crosspiece and butt and ignited with a match, thereby igniting the propellant charge in each barrel in rapid succession. This weapon fired bullets weighing 36.5 g each. The gun itself weighed 30 kg. The *pusslan* (fire-lance), (b), is an assault gun. It also has twenty barrels of 20-mm caliber. Powder and a fuse were put into the center core, which had a communicating opening into each barrel. This gun was mounted on the end of a lance, and after the fuse was ignited the lance would be pointed or thrown toward the enemy. Source: Holmquist BM, Gripstad B. *Swedish Weaponry Since 1630*. The Defence Matériel Administration of Sweden. The Royal Army Museum. Arlöv, Sweden: Berlings; 1982: 16.

leum jelly]. It was known as *cordite* due to its shape and was adopted by the British army in 1891 and is still used.[12p5]

Despite the European propellant discoveries, black powder was used almost exclusively as the propellant in military guns in the United States throughout the 1800s. At least one European innovation, a nitrated cotton called guncotton (a form of nitrocellulose) that was invented in Switzerland, was tried as a replacement propellant but was rejected because it was too costly to produce and its strong gases corroded gun barrels. Black powder was initially produced in small cottage industries. Later, powder mills were constructed but there were few production innovations. This situation changed in 1866 when Alfred Nobel perfected dynamite from nitroglycerin, black powder, charcoal, brick dust, and wood dust. Dynamite was a successful commercial explosive for mining and excavating but was not militarily significant. However, the flourishing dynamite industry of the latter 19th century demonstrated that a relatively safe energetic chemical mixture could be produced at reasonable cost and that the chemical constituents could be modified to achieve different explosive characteristics. These events signaled the development of a large, greatly improved military explosive and propellant industry.[8]

From the beginning of the American Civil War to the beginning of World War I (1861–1914), major improvements occurred in both the military propellant industry and the development of military ordnance such as cannon and guns (Figure 10-3). As a result, the Spanish-American War (1898) was the last major conflict in which black powder was used in large quantities. World War I was fought with new chemicals that propelled projectiles from modern artillery and with innovative smaller weapons like machine guns.[3,8,13]

During World War I, two new kinds of weapons systems significantly increased soldiers' exposure to the toxic products of propellant combustion: the battlefield tank with a breechloading gun, and the rapid-firing machine gun. The battlefield tank essentially

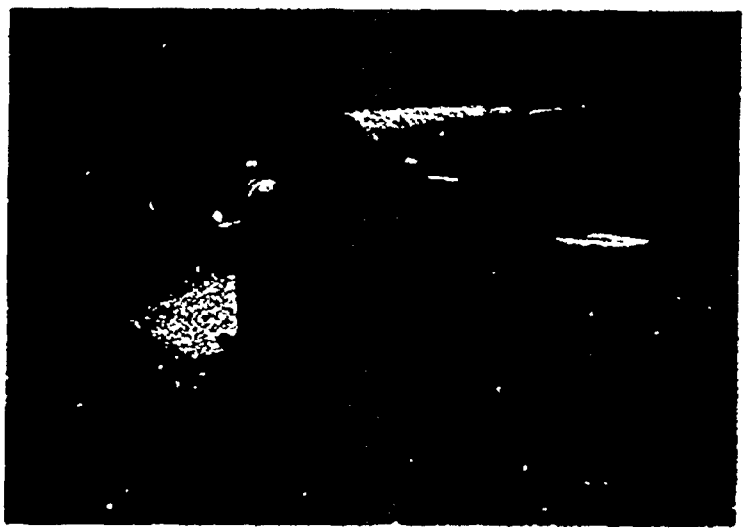

Fig. 10-3. An innovation that significantly increased the possibility that soldiers would be exposed to propellant combustion products was the rear- or breech-loaded cannon. The breech-loaded cannon was conceived at least as early as the Middle Ages but manufacturers could not produce a tight seal that would prevent leakage of propulsion pressure. A simple mechanism that was found to be effective was the "sliding breech block," which was used in this 19th-century Swedish cannon. After the breech door was closed, the piston on the right (the breech block) was moved to the left to complete the breech seal. Another 19th-century innovation was the brass cartridge case, which was forced tightly against the inside of the barrel. Other devices used to seal the breech include the interrupted screw breech block (sections of thread are cut away, allowing the breech block to be locked with only a turn of a few degrees) and the soft obturator ring. The obturator ring, located directly in front of the interrupted screw breech block, is forced against the breech block by the propellant explosion, forming a tight seal. Sources: (1) Holmquist BM, Gripstad B. *Swedish Weaponry Since 1630*. The Defence Matériel Administration of Sweden. The Royal Army Museum. Arlöv, Sweden: Berlings; 1982: 66. (2) Harding D, ed. *Weapons: An International Encyclopedia from 5000 BC to AD 2000*. Leicester, England: Galley Press (WH Smith and Son Limited); 1984.

Fig. 10–4. This German *Nebelwerfer* (smoke thrower) 42, or mobile rocket launcher, was towed; it fired a rocket that achieved a range of 4,976 yards. Each rocket contained seven tubular sticks of propellant composed of nitrocellulose and diglycol dinitrate. An electrical firing mechanism was used. These devices were originally used for chemical or smoke barrages, which gave rise to the name. However, high-explosive, antipersonnel rockets were added. Sources: (1) US Army Office of the Chief of Ordnance. *Catalogue of Enemy Ordnance Materiel*. Vol 1, German (R). Washington, DC: Office of the Chief of Ordnance, 1 May 1945. 350,354,375. (2) US Army Ordnance Museum, Aberdeen Proving Ground, Md. Accession File ORD-EMAR-1188. (3) US Army Ordnance Museum, Aberdeen Proving Ground, Md. Accession File ORD-EMAR-588.

enclosed the space around its gun breech. Because ventilation was often inadequate, air inside the tank quickly became oppressive, and it was painful for the crew to breathe.[14] When under enemy fire, the crew could close all the tank hatches and be relatively protected from the outside threat, but then they were at greater risk from their own gun's combustion products inside the tank. Chemical warfare forced soldiers to protect themselves in the field by sealing themselves inside their fortifications. Rapid-firing machine guns produced dangerous levels of carbon monoxide from incomplete propellant combustion inside these enclosed fortifications; for the first time, the gas produced by the combustion of propellants was itself reported to be the cause of casualties.[1]

The earliest reported use of rockets in warfare was during the siege of Kaifeng, China, in ad 1200; rocket experimentation, development, and use in warfare continued sporadically through World War I. (Rockets were fired by British troops at the siege of Fort McHenry in the Baltimore, Maryland, harbor during the War of 1812, an event that inspired Francis Scott Key to include the words "the rocket's red glare" in the song that became the United States' national anthem.) However, rifles and cannons were generally more popular than rockets, and rockets were unsafe and had to be hand made. As late as World War I, the only rocket propellant used, black powder, had to be tightly packed into the rocket by hand to achieve a high degree of compression when burned. The tightly packed propellant had a tendency to become brittle, crack, and explode. These rockets were considered unsafe and unsuitable for use in front lines.[15]

Between 1936 and 1940, Great Britain, Germany, and the United States initiated the development of improved rocket propellants. Safe and reliable charges that did not have to be compressed allowed for the mass production of rocket tubes and other rocket parts. The British propelling charge consisted of nitroglycerin (58%), nitrocellulose (41%), and carbamite

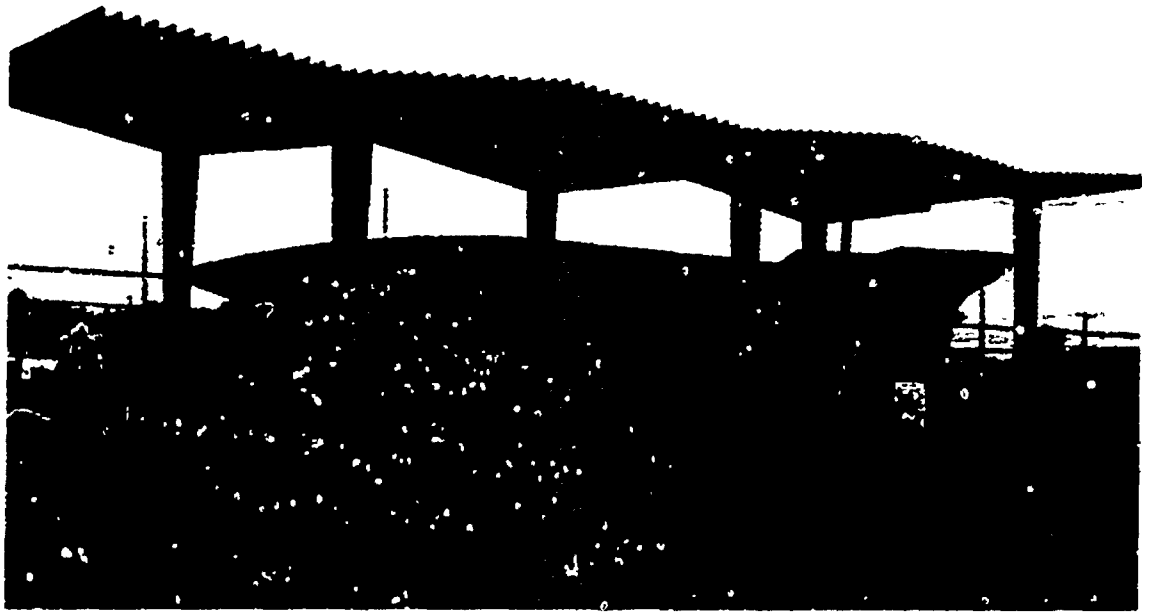

Fig. 10-5. The V-2 was the second "Vengeance" or "V" weapon developed by the Germans for use against England and Belgium in reprisal for the allied bombings of German cities. The V-1 or "buzz bomb" was a pilotless aircraft whereas the V-2 was the first inertially guided ballistic missile and traveled at 3,600 mph. From Peenemunde on the Baltic Sea, about 1,500 were fired at England; over 2,100 were directed at Antwerp; and about 650 malfunctioned after firing and never reached their targets. The V-2 was 45 ft 10 in. long and 5 ft 5 in. in diameter. It was powered by liquid propellant (4 tons of alcohol and 4 tons of liquid oxygen), which were pumped into the motor at a rate of 9 tons per minute. The rocket's total weight was 14 tons: 8 tons of fuel and 1 ton of explosive. It reached an altitude of 75 miles and had a range of 200 miles. After World War II, about 180 scientists from Peenemunde and 300 freight cars full of V-2 parts were captured by US Army Ordnance experts and taken to New Mexico, where the V-2 rockets were assembled and test-fired as the first step in the development of the United States's ballistic missile and space programs. Sources: (1) *German Explosive Ordnance (Bombs, Fuzes, Rockets, Land Mines, Grenades and Igniters)* TM 9-1985-2/TO 39B-1A-9. Washington, DC: US GPO; 1953: 209-215, 346. (2) US Army Ordnance Museum, Aberdeen Proving Ground, Md. Accession File ORD-AMMO-919

(9%). Carbamite (diethyldiphenyl urea) was a gelatinizer and stabilizer. The United States formulation was nitrocellulose (approximately 60%) and nitroglycerin (approximately 40%). Diphenylamine was used as a stabilizer, but apparently in very small quantities.[15]

During World War II, the antitank rocket and tube (bazooka), the 4.5-in rocket launcher, the German 300-mm Nebelwerfer 42 (which fired single rockets or multiple rocket barrages) and free-flight rockets (such as the German-made, liquid-propellant system V-2) were developed (Figures 10-4 and 10-5).[1] These three types of rockets generated larger amounts of combustion products per unit time, resulting in a greater potential for exposure to the soldiers who operated these weapons.

Since World War II, technological advances in rocketry and other weaponry and the need for sealed vehicles that are capable of surviving a chemically contaminated environment have markedly increased the risk that soldiers will be exposed to the toxic products of propellant combustion. Since the 1970s, increasing concern about chemical carcinogenicity and mutagenicity has created a broader perspective for examining the toxicity of propellant combustion products, extending beyond the acute effects.[16] As they did in World War I, gun breeches on armored vehicles still open into crew compartments; firing-tube exhaust and vehicle ventilation systems are still necessary to evacuate combustion gases. Additionally, the development of improved military armor that was very difficult to penetrate triggered a search for projectiles of greater density and strength. This led to the inclusion of DU in 25-, 105-, and 120-mm caliber projectiles and gave rise to a new potential exposure of concern for the soldiers who fired the projectiles or who would be exposed to projectile fragments.[17]

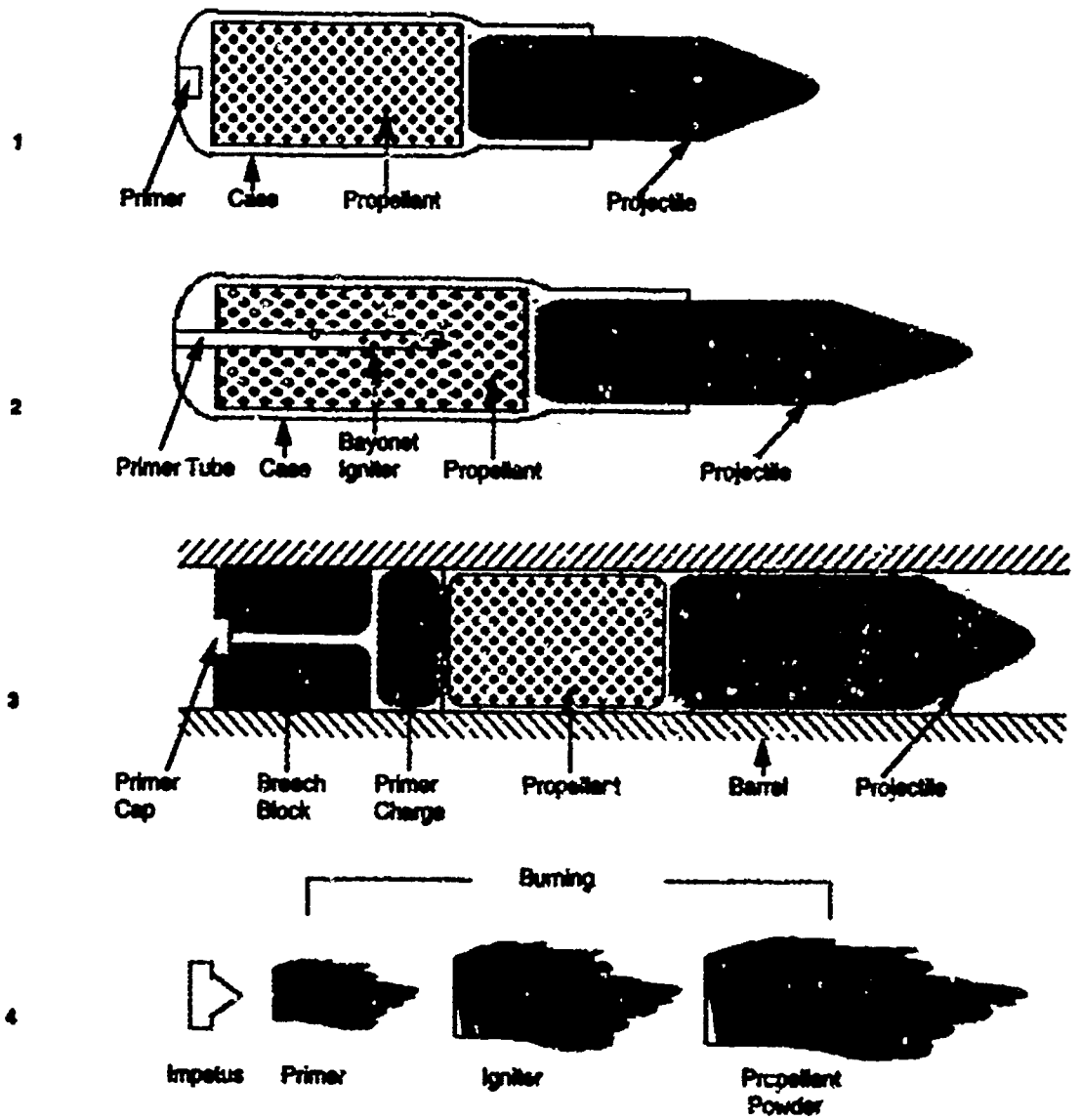

Fig. 18-5. Drawings 1, 2, and 3 show the general configurations of primer, igniter, and propellant mixes in typical ammunition. Drawing 4 is a schematic representation of the firing sequence in typical ammunition, usually referred to as the "explosive train" or the "ignition train." A small electrical and/or mechanical (percussion) stimulating impetus is intensified through a succession of intermediate charges to obtain optimum firing of the main charge, the propellant. The "ignition train" typically consists of four steps: stimulating impetus, primer burning, igniter burning (shown only in drawings 2 and 3) and propellant burning. Igniters are not generally placed in small-caliber ammunition (drawing 1) but are found in the bayonet igniter of tank gun rounds (drawing 2) and in close association with the propellant in artillery rounds (drawing 3). Igniter mixes are generally not used in rocket propulsion motors. Propellants are "low explosives" in that they burn relatively slowly (400 m/sec or slower) at a sustained rate. In contrast, "high explosives" like dynamite detonate at 1,000 to 10,000 m/sec producing a sudden, powerful shock wave. Sources: (1) Farrar CL, Leeming DW. Military ballistics—A basic manual. In: *Battlefield Weapons Systems and Technology Series*. Vol 10. Oxford, England: Brassey's Pub Ltd; 1983. (2) US Department of the Army. *Military Explosives*. Picatinny Arsenal, NJ: AMCCOM; 1984. Technical Manual 9-1300-214. (3) Ross RH, Pal DC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems*. Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6384/V1. (4) Ciccone FQ. Primers. In: Kaye SM, ed. *Encyclopedia of Explosives and Related Items*. Vol 8. Dover, NJ: US Armament Research and Development Command; 1978: 372-387.

COMPOSITION OF PROPELLANTS

In most military weapon propellant systems, the initiation of an impetus (usually a percussion force or an electrical stimulus) causes a number of different energetic chemicals to burn in series (Figure 10-6). The composition of these energetic materials varies among primers, igniters, and propellants; and the three different chemical mixes used vary with the weapon system. In small-caliber ammunition, as measured by the diameter of the projectile (generally considered to be less than 40 mm, like that used in the 5.56-mm M16 rifle and the 7.62-mm machine gun), the primer usually ignites the propellant charge. In larger-caliber ammunition (such as that used in 155-mm and 8-in howitzers and 105- and 120-mm tank guns), a booster charge, known as an igniter, acts between the primer and the propellant. These various mixes or charges (which are sometimes referred to collectively and incorrectly as "the propellant") are designed and developed by chemists and ballisticians to (*a*) achieve the desired projectile motion inside and outside the firing tube and (*b*) deliver the projectile payload accurately over a specified distance. Other considerations in the chemical design and development of charges include the potential toxicity to humans from component chemical ingredients and combustion products; the physical safety of manufacturing workers and soldiers who will use the propellants and ammunition; the storage life of the propellant; and the preservation and extended life of the weapon system used to fire the ammunition, especially the firing tube.

AMEDD personnel who evaluate potential or actual toxic exposures related to energetic chemicals in the ignition train often have difficulty locating anything more than imprecise qualitative and quantitative descriptions of the chemical mixes. Additionally, toxicity studies of combustion products are also difficult to obtain. There are at least two reasons for this: first, the compositions are militarily important and may be classified, or at least controlled in their release; and second, personnel who work in the research and development of energetic materials often focus on a limited area of interest and are not knowledgeable about the broad, general topic of human health effects. As a result, AMEDD physicians and scientists involved in toxicity assessment of a military propellant may not be able to easily locate toxicity data, or even the detailed chemical composition, of a propellant of interest. The toxicity data may not exist, and the propellant formulation may be classified for reasons of national security. Additionally, several propellant scientists may need to be interviewed before a reasonably complete database, one that will support a health hazard assessment (see Chapter 6, Health Hazard Assessments), can be assembled.

Primers and Igniters

Pyrotechnic devices (which we call fireworks or signal flares today) are alleged to have been used over 1,000 years ago. However, before the 19th century, little if anything was recorded about the manner in which the burning of the energetic chemicals in these devices was started. The first modern primer, which contained potassium chlorate, charcoal, and sulfur, was probably invented by Forsyth in 1807 (Table 10-1).[9,16] The first high explosive (HE) chemical (nitroglycerin [NG]), which was developed by Sobrero in

TABLE 10-1

COMPOSITION OF PRIMERS AND IGNITER

Primers	Ingredient	% by Weight
First modern primer	Potassium chlorate	70.6
	Charcoal	11.8
	Sulfur	17.6
FA70	Potassium chlorate	53
	Antimony sulfide	17
	Lead thiocyanate	25
TNT		5
FA956	Lead styphnate	37 ± 5
	Tetracene	4 ± 1
	Barium nitrate	32 ± 5
	Antimony sulfide	15 ± 2
	Aluminum powder	7 ± 1
	PETN	5 ± 1
	Gum arabic	< 1
Igniter*	Black powder:	
	Potassium nitrate	76 ± 2
	Charcoal	14 ± 2
	Sulfur	11 ± 1

*Also may contain nitrocellulose, mealed gunpowder, finely divided smokeless powders, and/or other substances

Sources: (1) Rose RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems*. Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1. (2) Ciccone TQ. Primers. In: Kaye SM, ed. *Encyclopedia of Explosives and Related Items*. Vol 8. Dover, NJ: US Armament Research and Development Command; 1978: 372-387.

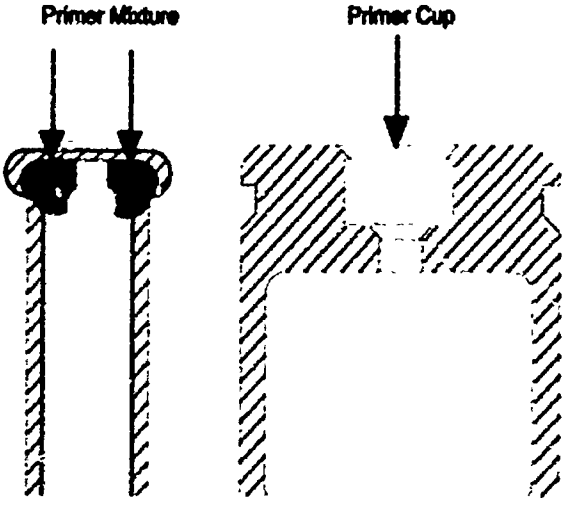

Fig. 19-7. Percussion primers in military ammunition may be either rimfire (left) or centerfire (right). In the rimfire model, the primer energetic material is placed around the rim of the base of the shell casing. The firing pin strikes the rim of the metal casing and the percussion force starts the burning of the primer. Rimfire primers are generally restricted to 0.22-mm caliber ammunition, which is extensively used in commercial sporting ammunition, and is also used by the military to a limited degree for training purposes. In centerfire ammunition, the box or cup containing the primer is located in the center of the base of the shell. The figure above is a representative centerfire box, but there are many varieties of centerfire percussion-box configurations. The metal used to make the cup may be a brass alloy (70% copper and 30% zinc) or some other metal or alloy that will provide the desired sensitivity to the applied percussion force. Adapted from Cocone TQ. Primers. In: Kaye SM, ed. *Encyclopedia of Explosives and Related Items*. Vol 8. Dover, NJ: US Armament Research and Development Command; 1978: P372–P387.

1846, and other significant improvements in energetic materials in the latter 19th century, stimulated interest and innovations in the physical and chemical means used to activate explosive and propellant charges. Within the scope of most of the discussion in this chapter, the impetus that begins the explosive or ignition train can be considered to be either a percussive or an electrical force. (Other means to initiate energetic materials, such as friction, have been used to a lesser degree.) In ammunition, the percussion force is applied to either the rim (rimfire) or the center (centerfire) of the end of the shell casing (Figure 10-7).[19]

During the early 1900s, a primer with a mercury fulminate base (which is believed to have originated in Austria) was widely used in the U.S. Army. Around 1917, the army switched to a nonmercuric formula, called FA70, which contained potassium chlorate, antimony sulfide, lead thiocyanate, and trinitrotoluene (TNT). A variant (FA90), which contained antimony sulfide and pentaerythritol tetranitrate (PETN, which replaced TNT), was used in caliber 0.50 ammunition. Potassium chlorate decomposed on firing to form potassium chloride. The potassium chloride was deposited in the gun barrel and quickly caused extensive rusting. In 1948, lead styphnate was chosen from among several candidates as the replacement compound and was incorporated into a standard primer mix called FA956.[18]

Priming mixtures are developed to (*a*) fire when the impetus of the ignition train is initiated, (*b*) propagate the ignition train once they are fired, and (*c*) reliably perform the first two functions under various conditions. For example, environmental temperatures may vary considerably and the pressures applied to shell casings that house percussion primers will differ with different weapon systems. (Potassium chlorate is still occasionally used in primers intended for use in very hot environments.) Additionally, the percussion force applied to the energetic primer mixture could be high or low, depending on the design requirements of the system. As the amount of applied energy is decreased, the sensitivity of the primer must be increased. Increased sensitivity is accomplished by using thinner metals and softer alloys to contain the primer mixture and by modifying the composition of the mixture itself.

The ignition train in rockets usually consists of an impetus, the primer, and the propellant. The impetus is an electrical impulse that travels along a bridging wire or squib to the primer, which usually contains $KBNO_4$. The current emphasis in the development of energetic materials for army rockets is to minimize the production of any substance produced during firing

that could reveal the firer's position (provide a battlefield "signature"). Substances of concern are those that could be detected by infrared sensors or that are visible to the naked eye (such as smoke or muzzle flash). While $KBNO_4$ is not entirely smokeless, it is considered an acceptable primer.[2]

Routinely used igniter mixtures are reported to include black powder (potassium nitrate, charcoal, and sulfur), nitrocellulose, mealed gunpowder, finely divided smokeless powders, and/or other substances.[9]

Gun Propellants

All solid propellants other than black powder are known as smokeless powders, although they are neither powders nor completely smokeless.[12] The basic types among these include the single-base, which are prepared by dissolving nitrocellulose in ether and alcohol; the double-base, which are prepared by dissolving nitrocellulose in nitroglycerin; and the triple-base, which are prepared by dissolving nitrocellulose in nitroglycerin with nitroguanidine added to reduce the temperature of the combustion-produced gas.[12]

Nitro-based propellant charges typically contain one to three nitro compounds and a wide variety of other added chemicals that are introduced to produce a particular, desired effect (Tables 10-2 and 10-3).

> Double-base propellants are more powerful than single-base propellants, however they suffer from high propellant-gas temperatures [that] can cause excessive barrel erosion and muzzle flash. Triple-base propellants are similarly powerful, but the addition of cool-burning nitroguanidine reduces the temperature of the gas to near that of the single-base propellants. Other ingredients added to smokeless propellants are used primarily to control [the] burning rate and [to] suppress decomposition during storage.[12p140]

Recently, compounds such as the nitramine propellant RDX (research department explosive, which is also called cyclonite; see Chapter 9, Explosives and Propellants), which have traditionally been considered explosives, have been used as propellants. An RDX-based propellant is now included in the M900 round for the 105-mm tank gun. The combustion products from this propellant are expected to be differ-

TABLE 10-2

TYPICAL COMPONENTS OF NITROCELLULOSE-BASED PROPELLANTS AND THEIR FUNCTIONS

Component	Application
Nitrocellulose	Energetic polymeric binder
Nitroglycerin, metriol trinitrate, diethylene glycol dinitrate, triethylene glycol dinitrate, dinitrotoluene	Plasticizers: energetic
Dimethyl, diethyl or dibutyl phthalates, triacetin	Plasticizers: fuels
Diphenylamine, diethyl centralite, 2-nitrodiphenylamine, magnesium oxide	Stabilizers
Organic and inorganic salts of lead (lead stannate, lead stearate, lead salicylate)	Ballistic modifiers
Carbon black	Opacifier
Lead stearate, graphite, wax	Lubricants
Potassium sulfate, potassium nitrate, cryolite (potassium aluminum fluoride)	Flash reducers
Ammonium perchlorate, ammonium nitrate	Oxidizers: inorganic
RDX, HMX, nitroguanidine, and other nitramines	Oxidizers: organic
Aluminum	Metallic fuels, cross-linking catalysts
Lead carbonate, tin	Defouling agents
Vaseline, centralite	Inert materials to decrease erosion
Graphite	Recarbonate steel barrels

RDX: research department explosive; HMX: high melting explosive

Adapted with permission from Lindner V. Explosives and propellants. In: Kirk RE, Othmer DF, eds. *Encyclopedia of Chemical Technology*. Vol 9, 3rd ed. New York: John Wiley; 1980: 622. Additional source: Ross RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems*. Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1.

TABLE 10-3

COMPOSITION AND SELECTED COMBUSTION PRODUCTS OF CHARACTERISTIC GUN PROPELLANTS

	Propellants					
	M1	M5	M8	M10	M15	M17
Composition, wt %[1,2]						
Nitrocellulose (nitrogen, %)	85.0 (13.15)	82.0 (13.25)	52.2 (13.25)	98.0 (13.15)	20.0 (13.5)	22.0 (13.15)
Nitroglycerin	—	15.0	43.0	—	19.0	21.5
Nitroguanidine	—	—	—	—	54.7	54.7
Ethyl centralite	—	0.6	0.6	—	6.0	1.5
Diphenylamine	1.0*	—	—	1.0	—	—
Dinitrotoluene	10.0	—	—	—	—	—
Dibutyl phthalate	5.0	—	3.0	—	—	—
Potassium nitrate	—	0.7	1.2	—	—	—
Barium nitrate	—	1.4	—	—	—	—
Potassium sulfate	1.0*	—	—	1.0	—	—
Lead carbonate	1.0*	—	—	—	—	—
Cryolite	—	—	—	—	—	0.3
Graphite	—	0.3	—	0.10*	—	0.15[4]
Combustion Products, mol/g x 10^{2}[†1,2]						
CO	2.33	1.61	1.28	1.81	1.45	1.15
CO_2	0.19	0.48	0.66	0.40	0.14	0.25
H_2	0.88	0.34	0.19	0.44	0.92	0.57
H_2O	0.64	1.08	0.11	0.99	0.83	1.07
N_2	0.44	0.48	0.54	0.46	1.29	1.30

*Added if required

†At loading density of 0.2 g/cm^3

Adapted with permission from Lindner V. Explosives and propellants. In: Kirk RE, Othmer PF, eds. *Encyclopedia of Chemical Technology*. Vol 9, 3rd ed. New York: John Wiley; 1980: 627.

[3] Rees RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems*. Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1.

[4] Roth J, Carpenter EL. Propellants, Solid. In: Kaye SM, ed. *Encyclopedia of Explosives and Related Items*. Vol 8. Dover, NJ: US Army Armament Research and Development Command; 1978: 402-473.

ent from nitrocellulose-based propellants in that much more hydrogen cyanide and nitrous oxide (which is relatively nontoxic compared to the other oxides of nitrogen) are produced.[19]

Rocket Propellants

In some respects, propellants used in rockets such as the soldier-held Stinger (shown in Figure 6-4, Chapter 6, Health Hazard Assessments), the Patriot air defense system, and the Multiple Launch Rocket System (MLRS), are different from those used in rifles, pistols, and artillery pieces. Successful rocket flight depends on the solid fuel's burning so that the gases produced forcefully exit rearward, thereby generating the pressure required to thrust the rocket forward.[18] One variable that is important in propulsion is related to the total surface area of the propellant mixture that

is being burned at any time. For example, consider a rocket propellant charge filling the entire circumference of the rocket (wall-to-wall) and running down the long axis of the rocket. If the burning is initiated in a simple longitudinal hole that runs the length of the propellant change, then the largest burning surface (and the concomitant greatest release of gas) would occur at the last instant of burning. To produce a more even burn, the center hole can be made in a star shape. In some rockets with wall-to-wall propellants, a center hole is not used; the propellant is ignited across the surface of one end and burns lengthwise to the other end (as a cigarette burns). Depending on the burn method desired, the primer compound is placed in a center hole or on the surface at one end of the rocket.[2,15] The double-base propellants of World War II–era rockets usually burned unrestricted. Since World War II, however, the burn of solid rocket propellants has been controlled by selecting both the chemical composition of the propellant and the method of burning.[15]

Double- and triple-base nitrocellulose propellants are used in rockets (Table 10-4). Based on their ballistic and thermochemical-thermodynamic properties, propellant A is classified as low energy and propellants B and C are classified as high energy.[9,20]

In 1942, work began on the development of composite rocket propellants, which usually do not contain nitrocellulose or an organic nitrate. However, they normally contain a fuel such as metallic aluminum; an organic polymer binder such as a synthetic rubber, which is also a fuel; and an inorganic oxidizing agent such as ammonium perchlorate (Table 10-5).[20,21]

The Stinger missile is a shoulder-fired rocket that provides air-defense protection. It has an infrared homing device that identifies the heat emitted by its target, such as aircraft. The Stinger has two rocket motors. When the soldier fires the missile from its launch tube, the small launch motor carries the missile a safe distance away; then the flight motor ignites the composite propellant, which propels the rocket to its target (Table 10-6). The combustion product of primary medical concern in the Stinger missile system is hydrogen chloride, which is equivalent to hydrochloric acid when in contact with water vapor.[22]

Research and development efforts are underway to improve the composition of currently fielded rocket propellants. These efforts are directed toward reducing cost, improving safety, decreasing the firer's battlefield signature, and/or reducing the possibility of adverse health effects from propellant combustion product toxicity.[2,22] For example, currently available composites containing ammonium perchlorate are unacceptable because (*a*) the hydrogen chloride produced as a combustion product of ammonium perchlorate forms a white cloud of gas that reveals the firer's position and (*b*) the gas can produce adverse health effects in humans. One consideration is to replace ammonium perchlorate with RDX (hexahydro-1,3,5-trinitro-1,3,5-triazine) or HMX (high-melting explosive; octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine). These two high-energy explosives would increase the sensitivity of the propellant (making the propellant more susceptible to explosion) and would require further modification of the chemical composition to reduce that sensitivity.[2]

TABLE 10-4

COMPOSITION AND MAJOR COMBUSTION PRODUCTS OF TYPICAL NITROCELLULOSE-BASED CAST ROCKET PROPELLANTS

	Low Energy	High Energy	
	Prop A	Prop B	Prop C
Composition of Propellants, wt %			
Nitrocellulose (12.6% N)	59.0	20.0	22.0
Nitroglycerin	24.0	30.0	30.0
Triacetin	9.0	6.0	5.0
Dioctyl phthalate	3.0	—	—
Aluminum	—	20.0	21.0
HMX	—	11.0	—
Stabilizer	2.0	2.0	2.0
Ammonium perchlorate	—	11.0	20.0
Lead stearate	3.0	—	—
Combustion Products Composition, mol/100 g			
C	2.12	—	—
CO_2	0.31	0.05	0.07
CO	2.12	1.30	1.15
H_2	1.06	0.75	0.66
H_2O	0.66	0.27	0.33
N_2	0.43	0.49	0.38
Pb	0.004	—	—
Al_2O_3	—	0.35	0.37
H	—	0.20	0.23
OH	—	0.05	—
Other	—	0.15	—
HCl	—	—	0.10

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TABLE 10-5

TYPICAL COMPONENTS AND CHARACTERISTICS OF COMPOSITE ROCKET PROPELLANTS*

Components	Characteristics
Binders	
Polysulfides	Reactive group, mercaptyl (–SH), is cured† by oxidation reactions; low solids loading capacity‡ and relatively low performance; now mostly replaced by other binders
Polyurethanes polyethers polyesters	Reactive group, hydroxyl (–OH); is cured with isocyanates; intermediate solids loading capacity and performance
Polybutadienes copolymer, butadiene and acrylic acid	Reactive group, carboxyl (–COOH) or hydroxyl (–OH), is cured with difunctional epoxides or aziridines
Terpolymers of butadiene, acrylic acid and acrylonitrile	Superior physical properties and storage stability
Carboxy-terminated polybutadiene	Cured with difunctional epoxides or aziridines; have very good solids loading capacity, high performance, and good physical properties
Hydroxy-terminated polybutadiene	Cured with diisocyanates; have very good solids loading and performance characteristics and good physical properties and storage stability
Oxidizers	
Ammonium perchlorate	Most commonly used oxidizer; has a high density, permits a range of burning rates, but produces smoke in cold or humid atmosphere
Ammonium nitrate	Used in special cases only; hygroscopic and undergoes phase changes, has a low burning rate, forms smokeless combustion products
High-energy explosives (RDX and HMX)	Have high energy and density, produce smokeless products, limited ranges of low burning rates
Fuels	
Aluminum	Most commonly used; high density; produces an increase in specific impulse and smoky and erosive products of combustion
Metal hydrides	Provide very high impulse but generally inadequate stability, smoky products, low density
Ballistic Modifiers	
Metal oxides	Iron oxide most commonly used
Ferrocene derivatives	Permit significant increase in burning rate
Others	Coolants for low burning rate and various special types of ballistic modifiers
Modifiers for Physical Characteristics	
Plasticizers	Improve physical properties at low temperatures and processability; may vaporize or migrate; can increase energy if nitrated
Bonding agents	Improve adhesion of binder to solids

*In 1942, at the Guggenheim Aeronautical Laboratory of the California Institute of Technology, work began on the development of cast composite propellants for rockets. The initial formulation contained approximately 25% asphalt and 75% potassium perchlorate.

†Cured: the conversion of a raw product to a finished and useful condition by application of heat and/or, as in this table, chemicals, which induce physicochemical changes. Source for this definition: *Condensed Chemical Dictionary*, 10th ed. New York: Van Nostrand Reinhold; 1981.

‡Solids loading capacity: the ability of a binder to allow more solids (eg, oxidizers) to be added to a propellant mixture. A binder with a higher solids loading capacity allows more oxidizer to be added and is therefore more energetic. Source for this definition: Chew B. Propulsions Directorate, Redstone Arsenal, Huntsville, Ala. Personal communication; 21 October 1992.

RDX: research department explosive

HMX: high melting explosive

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TABLE 10-6

COMPOSITION OF PROPELLANT AND COMBUSTION PRODUCTS OF STINGER ROCKET MOTORS

Propellant Ingredients*		Propellant Combustion Products (mol/100 g of propellant)		
Launch Motor	Flight Motor*	Combustion Product	Launch Motor	Flight Motor†
Ammonium perchlorate	Polymer, carboxy terminated	CO	0.551	0.944
		CO_2	0.539	0.045
Polyvinyl chloride	Resin	Cl	—	0.003
Di(2-ethylhexyl)adipate	Epoxy resin	H	0.869	0.009
Copper chromite	Chromium octoate	HCl	0.800	0.575
Aluminum powder	Di(2-ethylhexyl)adipate	H_2	—	1.297
Carbon black	Lecithin	H_2O	0.925	0.315
Stabilizer	Ferric oxide	N_2	0.308	0.290
Sodium dioctyl sulfosuccinate	Aluminum powder	Al_2O_3	0.018	0.333
		NaCl	< 0.001	—
Glycerol monooleate	Ammonium perchlorate	Cu	0.009	—
Pentaerythritol dioleate		CH_4	< 0.001	—
		H_2S	< 0.001	—
		CuCl	< 0.001	—
		Cr_2O_3	0.001	—

*Quantitative data are protected by security classification

†Only the "boost" grain was tested. Test results are suspect because of discrepancies in predicted and observed combustion product values

Sources: (1) Bourke KS, Redstone Arsenal, Huntsville, Ala to CDR, US Army Environmental Hygiene Agency. 9 June 1986. Written communication. (2) Gross R, US Army Environmental Hygiene Agency, Aberdeen Proving Ground, Md., and Cox R, Redstone Arsenal, Huntsville, Ala. Telephone communication; 22 June 1992.

Some rocket scientists are advocating a return to liquid propellants or some modification of them. Using propellant chemicals like those found in the Lance missile system could eliminate the need for perchlorates and could therefore produce less-detectable combustion products when fired. However, as discussed earlier, the use of liquid propellants is associated with concerns about safety issues and, as they were used by the Iraqi forces in Operation Desert Storm, can cause operational delays because of the time required to fuel them in the field. The Lance missile system uses two liquids (in addition to other energetic propellant chemicals): inhibited red fuming nitric acid and unsymmetrical dimethyl hydrazine. When these two liquids come in contact with each other, a hypergolic (self-igniting) mixture is formed. Scientists currently believe that containers can be made leakproof, even when the rocket sustains combat damage. Rockets with leakproof containers could be charged with fuel at the factory and arrive at their destination ready for firing. Furthermore, the chemicals that combine to form the hypergolic mixture do not necessarily need to be liquids; using them as gels may provide an additional safety factor.[2]

COMBUSTION PRODUCTS OF PROPELLANTS

The qualitative and quantitative exhaust emissions from the firing of a gun or rocket to which a soldier or civilian worker will be exposed depend on a multitude of variables including

- the chemical composition of the primer, igniter, and propellant;
- the pressure and temperature in the gun or rocket motor;
- the density of loading of the energetic chemicals (g/cm^3);
- thermal, chemical, and/or mechanical erosion of the combustion chamber, gun barrel, shell casing, and projectile; and

- the meteorological conditions during firing, including humidity, wind direction, and speed.[9]

The only sure way to determine the nature and degree of exposure is to collect samples for quantitative analyses from the person's breathing zone and/or to analyze solid residues and determine the extent of contact between the residues and the person's skin and mucous membranes.

Emissions from burning energetic chemicals—for individual and all components in the ignition train—are measured in laboratories. However, the laboratory setting requires that arbitrary decisions be made about the types and sizes of test chambers or devices that will

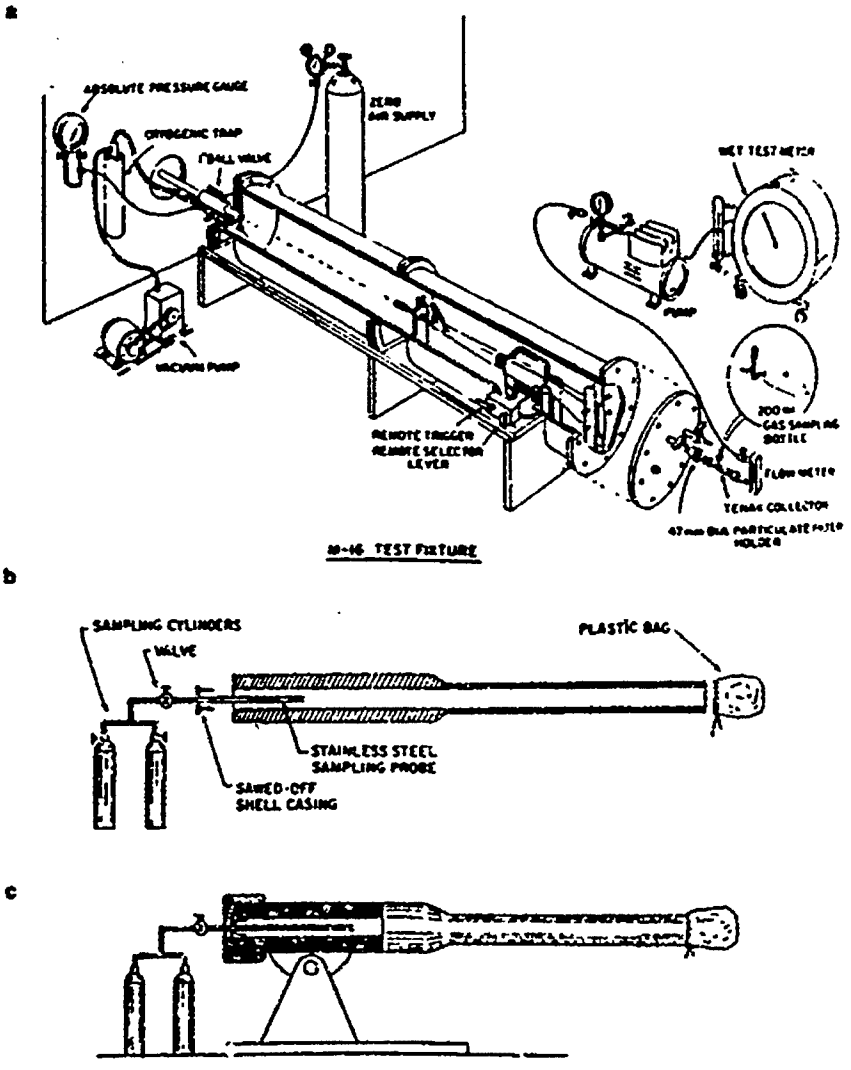

Fig. 10-8. Laboratory sampling systems for identifying propellant combustion products. The enclosure for studying emissions from the M16 rifle, shown in (a), was 6 ft x 11.75 in and contained 133 L. The chamber was Teflon coated to minimize gas absorption and was kept tight with O-ring seals and valves. After firing, chamber contents were evacuated and analyzed. The tank gun system was constructed in a bunker. A manifold for collecting specimens was connected to an adapter, which was a sawed-off shell casing. After firing, the end of the firing tube was covered, the spent shell casing was removed, and the sawed-off shell casing with manifold collection tube was inserted. In (b), the collection device is being inserted; in (c) it is fully inserted. Specimens were collected in the sampling cylinders. Source: Ase P, Eisenberg W, Gordon S, Taylor K, and Snelson A. Propellant combustion product analyses on an M16 rifle and 105-mm caliber gun. *J Environ Sci Health.* 1985;A20(3):337–368.

be used to collect emissions. Laboratory test conditions cannot and do not take into account all the potential variables that will influence the exposures received by personnel who operate the gun or rocket system. Two laboratory test systems have been used to evaluate propellant combustion products: one for rifle firing (5.56-mm M16 rifle) and one for 105-mm tank gun firing (Figure 10-8). Data collected from such laboratory test systems help in both assessing the potential for harmful exposures and investigating exposures that allegedly resulted in morbidity, mortality, or both. In drawing conclusions, however, the limitations of these data must be recognized. Additionally, in assessing reports of the qualitative and quantitative composition of energetic materials and their combustion products, it is usually difficult and often impossible for the AMEDD assessor to determine if the writer is addressing the primer, igniter, or propellant component, or a combination.

Primers and Igniters

Data are not readily available on the chemicals emitted exclusively by the combustion of primers. This lack of data is probably explained by the quantity of primer used: primers contribute a very small proportion to the total amount of energetic chemicals in an ignition train. When evaluating total emissions using systems like those shown in Figure 10-8, it is often difficult to determine the specific source chemical for a particular product of combustion. However, primers often contain antimony, barium, lead, and possibly other chemicals that may not be found in other ignition train compounds.[9,23]

Descriptive data on igniter combustion products also are not readily available in the scientific literature, probably for the same reasons that primer combustion data are scarce. However, black powder has been well studied and the approximate products of combustion are available (Table 10-7).

Gun Propellants

Carbon dioxide, water, carbon monoxide, hydrogen, nitrogen, and nitric oxide together typically constitute approximately 99% by volume of the total mixture of propellant combustion products.[24] The primary combustion products of most propellants are very similar and generally are considered to be predictable from the charge composition prior to combustion.[25] However, the total number of detectable, different chemical species formed during weapon firing is great. Ninety were reported from tests done on M16 rifle firing and 70 were associated with 105-mm tank gun exhausts.[23,24]

During the 1980s, AMEDD emphasized the major combustion products: carbon monoxide (see Chapter 11), lead (see Chapter 12), and, to a lesser degree, ammonia.[4] The irritant properties of ammonia could significantly detract from soldier performance on the battlefield. Other species (NOx, the generic expression for oxides of nitrogen) can also be formed. Under the correct conditions, ammonia can be formed as shown in the equation

$$N_2 + 3H_2 \longrightarrow 2NH_3 + 22.0 \text{ kcal}$$

Iron particles in the exhaust, which are formed as a result of erosion of the gun tube, catalyze this reaction.[9,25]

Weapons modernization occurred rapidly in the 1980s and with it grew concerns about the adverse health effects on soldiers who operated the new

TABLE 10-7

APPROXIMATE COMPOSITION OF COMBUSTION PRODUCTS OF BLACK POWDER

Component	Weight (%)
Gases (44% of total components)	
Carbon dioxide	49
Carbon monoxide	12
Nitrogen	33
Hydrogen sulfide	2.5
Methane	0.5
Water	1
Hydrogen	2
TOTAL	100.0
Solids (56% of total components)	
Potassium carbonate	61
Potassium sulfate	15
Potassium sulfide	14.3
Potassium thiocyanate	0.2
Potassium nitrate	0.3
Ammonium carbonate	0.1
Sulfur	9
Carbon	0.1
TOTAL	100.0

Adapted with permission from Lindner V. Explosives and propellants. In: Kirk RE, Othmer PF, eds. *Encyclopedia of Chemical Technology*. Vol 9, 3rd ed. New York: John Wiley; 1980: 660.

weapons systems.[3] As a result, propellant combustion products were evaluated more thoroughly. Other inorganic emissions (hydrogen fluoride, hydrogen sulfide, cyanide, and sulfur dioxide) have been described and a large number of volatile organic combustion products have been studied qualitatively and quantitatively.[9,23]

After they complete an artillery firing mission in the field, soldiers routinely burn any unused propellant. Large quantities of propellant and propellant-containing munitions have also been disposed of through a controlled process termed "open burning and open detonation."[26] The emissions produced by open burning and open detonation have been extensively studied, and current results indicate that combustion products of tested propellants have been far lower than the levels specified by the U.S. Environmental Protection Agency (EPA).[27]

Soot deposits generated from burning double-base propellants in laboratory ballistic ranges have demonstrated mutagenic activity as tested by the Ames *Salmonella* assay (see Chapter 9, Explosives and Propellants, and Chapter 14, Pesticides, for further discussion of mutagenic activity and the Ames assay).[16] Because mutagenic activity had been detected under laboratory conditions (using a gun specifically designed for laboratory use) in an enclosed, indoor environment, field tests (using operational weapons fired in the outside environment) were conducted at Picatinny Arsenal, New Jersey. These tests, now completed, were designed to determine if the mutagenic activity occurs in operational settings and, if so, what dangers, if any, are posed to soldiers. Preliminary assessment of the data indicates no mutagenic activity associated with the 155-mm, 5.56-mm, or 9-mm weapons systems evaluated. However, data from other tests indicate that further study is required to evaluate the potential health risks from exposure to combustion products that are formed during both the firing of other, small-caliber weapons systems and the burning of unused propellant bags after the artillery-firing missions have been completed.

This discussion has pertained to propellant combustion products that are generated during the firing of small-, medium-, and large-caliber weapons. However, combustion products are also formed and released when stored ammunition burns as a consequence of battle damage or training accidents. The modernization of weapons that occurred during the 1980s was accompanied by an effort to evaluate vehicle vulnerability and survivability and soldier survivability on the battlefield. One area identified for critical evaluation was the ammunition storage compartment (bustle) in the M1A1 tank. The major objectives of this evaluation were to

- ensure that the bustle was properly placed, to reduce its vulnerability;
- ensure that the bustle doors and seals provided adequate protection for the crew in case of fire; and
- design the bustle and crew compartment so that the crew would have time to escape from the tank if an explosion occurred.

TABLE 10-8

MAXIMUM RECOMMENDED EMERGENCY EXPOSURES FOR NITROGEN DIOXIDE

Time Limit (min)	Concentration (ppm)
30	10
10	25
05	50

Source: Davis DL, Executive Director, Board on Toxicology and Environmental Health Hazards, National Research Council Commission on Life Sciences, to Ranadive M. The Pentagon, Washington, DC. Written communication; 14 March 1985.

The OTSG identified carbon monoxide and oxides of nitrogen as the toxic gases of primary concern, and asked the Committee on Toxicology of the National Research Council to provide assistance. The committee used NO_2 as a surrogate for NOx in their discussions and proposed maximum recommended emergency exposure guidance levels (EEGLs) (Table 10-8).[28]

Rocket Propellants

The rocket propellant combustion product that has been of greatest concern for potential adverse health effects is hydrogen chloride (see Tables 10-4 and 10-6). Hydrogen chloride is released in significant quantities when propellants that include chlorine-containing compounds (such as ammonium perchlorate) are burned. In some cases, enormous quantities of hydrogen chloride are produced. For example, it has been estimated that 17 tons of the gas are released in the vicinity of a space-shuttle launch pad during a shuttle launch (Figure 10-9).[29] The amount of hydrogen chloride that soldiers or others could be exposed to depends not only on the amount generated in a given time but also on other variables including rocket speed, atmospheric conditions, and physical barriers that will

Fig. 10-9. The space shuttle Atlantis is shown being launched from the Kennedy Space Center, Florida, at 9:56 a.m. (EDT) on 31 July 1992. The large quantity of white smoke surrounding Launch Complex 39 is hydrogen chloride that was produced during combustion of the shuttle's perchlorate-based, solid rocket propellant. Photograph Courtesy of National Aeronautics and Space Administration, Houston, Tex. 31 July 1991. Photograph STS046-(S)-082.

contain the gas. For example, a soldier firing a shoulder-held rocket with a perchlorate-based propellant in a stagnant airspace will be at greater risk than a soldier firing the same weapon in an open field.

In most cases, concern about the production of hydrogen chloride from military weapon systems will be confined to exposing young, generally healthy soldiers in the immediate vicinity of the rocket firing. However, this is not always the case. When large quantities of perchlorate-based propellant are fired within a relatively short time, weather conditions may carry the gas far beyond the firing area (or the military installation boundaries). These quantities and concentrations may not be a matter of concern for healthy, young soldiers. However, they could be a threat to certain civilians such as infants, children, the elderly, and those with cardiopulmonary or other diseases. This situation occurred in the United States in 1988. The Intermediate-Range Nuclear Forces Treaty between the former USSR and the United States required that large Pershing missiles, which contained perchlorate-based solid propellants, be destroyed. The rocket motors were destroyed on the ground, by firing them while they were bolted to the ground horizontally. To protect those at risk off the military installation, prospective studies were conducted to determine the hydrogen chloride concentrations leaving the installation under defined firing conditions. Additionally, the Committee on Toxicology of the National Academy of Sciences (NAS) reviewed the data collected, procedures to be used, and exposure criteria. Specific restrictions were developed and followed during firing, and included permissible atmospheric conditions, times of firing, and frequency of firing.[30]

CONTAMINANTS FROM AMMUNITION

Lead or tin foil is placed in the propelling charge of some artillery ammunition to protect the gun barrel from accumulations of copper. When these decoppering ammunition rounds are fired, metallic lead or tin may be measured in the breathing zones of crew members.[31] Other elements included in shell casings, bullets, and projectiles can also vaporize or shear during firing and contaminate the breathing zones of soldiers or civilians working in the firing area. The potential for lead exposure has long been recognized as a significant risk to those who fire lead bullets in poorly ventilated areas (see Chapter 12, Lead). Bullets may also have a copper jacket, although the jackets are sometimes made of steel.[32] The full metal jacket that is required of all military bullets is something of a misnomer; the jacket on small-arms ammunition made in the

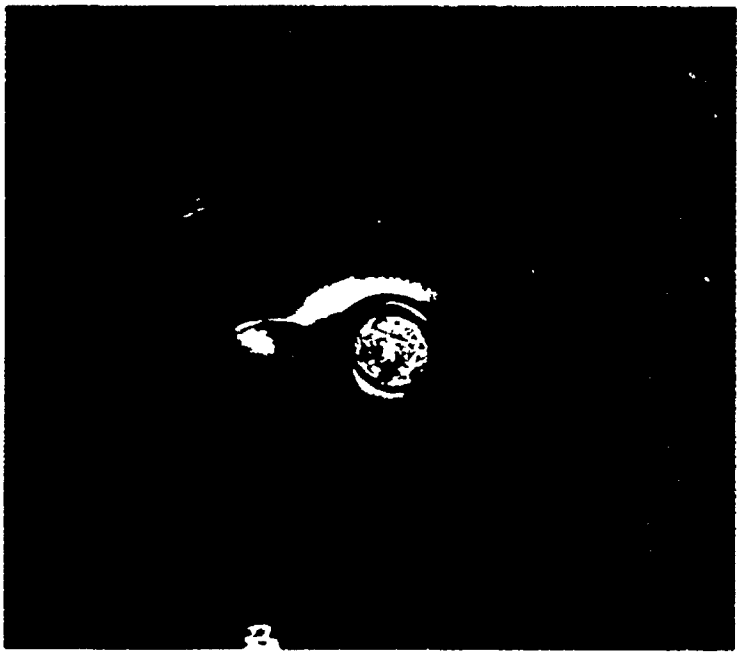

Fig. 10-10 The copper jacket does not completely surround this 5.56-mm bullet, the type fired by the M16 rifle; a portion of the lead core is exposed at the base. This exposed lead is subject to erosion from the hot gases that are produced during propellant combustion. However, this source of lead is minor; most of the lead in the environment surrounding the weapon comes from the combustion of the primer.

United States does not cover the base of the bullet's lead core (Figure 10-10). Zinc bullets have been used and studied, and they have been recommended as a replacement for lead bullets to reduce lead exposure.[11]

The only two components of bullets or projectiles that have raised concern about their potential for causing adverse health effects are lead and DU.[17] DU, which is a mixture of ^{234}U (0.0005%), ^{235}U (0.2500%), and ^{238}U (99.7500%), is a byproduct of the uranium-enrichment process and is of no use to the nuclear industry as a fuel for nuclear reactors. However, its high density and strength, ease and relatively low cost of fabrication, and availability make it desirable for use as the penetrator in antitank shells (the part of the projectile designed to actually penetrate the armor). DU emits alpha, beta, and gamma radiation, and may be contained in armor-piercing rounds of calibers 25 mm, 105 mm, 120 mm, and possibly other U.S. Army ammunition.[17,34]

Studies to detect the presence of DU and related compounds have been conducted (*a*) at the barrel muzzle and the weapon breech during firing; (*b*) in gun barrels, after DU rounds have been fired; and (*c*) inside the crew compartment of an armored vehicle that has been hit by a DU round. (Soldiers inside the spall zone of the penetrating round will be killed. If the ammunition or fuel cells do not explode, soldiers outside the spall zone will receive injuries to the tympanic membrane and ossicles, but will not be killed outright by the hit.) Detailed data from these studies are not available for public release. However, preliminary general information indicates that exposures are well below federal standards for soldiers conducting their military missions. This is not an unexpected finding, as DU-containing penetrators are completely enclosed within the round, and are not exposed at the muzzle or breach. Current assessments indicate that vehicle ventilation systems and personal protective equipment (PPE) available to soldiers will provide adequate protection in unusual situations where DU levels may exceed exposure standards.34 However, DU could accumulate in and around vehicles that are used frequently as targets.

Release of DU into the environment can also occur if vehicles that are carrying DU-containing ammunition catch fire. These and all other incidents involving DU ammunition must promptly be reported to ensure that the correct emergency response, rapid and proper evaluation of any radiation hazard, and appropriate cleanup of any contamination occur. Procedures to be followed in these situations are outlined in a Department of the Army Technical Bulletin (DA TB).[17]

Penetrators and other material (DU metal, oxides, and other DU compounds or mixtures) from DU rounds fired during testing and training can accumulate in the ground. Assessment of the risk to health and the environment from these situations and surveillance, if indicated, fall within the area of expertise of AMEDD health physicists and environmental health engineers.[34]

IDENTIFICATION OF HAZARDS

Risk assessors seek to prevent injury arising from the use of propellants and ammunition. Their assessments are predicated on defining (*a*) the hazardous components of the combustion products, (*b*) the extent of soldier exposure to these hazardous products, and (*c*) the controls available to reduce harmful exposure. Identifying whether the components of combustion products are hazardous can best be facilitated by actual reports of adverse health effects. However, as discussed earlier, relatively few accounts in the literature describe the actual adverse health effects from exposure to propellant combustion products in the United States military.[3,4,35]

In the absence of such data, the risks to soldiers can only be assessed by toxicological evaluation of the mixtures, or by assessing the risk based on knowledge of the individual components and combustion products of the mixtures. Obviously, the variability of the propellants' chemical composition and differences in firing conditions combine and cause differences in the types and quantities of combustion products formed—and thereby result in different risks to exposed soldiers.

Conditions of Exposure

The degree to which soldiers will be exposed depends on the amount and types of combustion products formed, the *dispersion volume* (the area contaminated by combustion products; for example, the inside of a tank or the vicinity of a rocket launching), and the relationship of the dispersion volume to the soldier's immediate environment. The amounts and types of combustion products formed depend on the chemical constitution of the propellant; the amount that is burned per firing (eg, rifle bullets contain only a few grams of propellant, whereas the propellant charge for howitzer rounds may be several kilograms); and other physical and chemical variables. High rates of weapons fire will produce higher concentrations of combustion products. These concentrations become hazardous when retained within the soldier's ambient environment, such as when guns or rockets are fired from enclosed bunkers, crew compartments, or rooms in buildings.

The conditions of exposure also depend on the measures initiated to limit the concentrations of combustion products, including ventilating enclosed spaces and wearing respiratory protective equipment. Administrative controls to limit the duration of exposure, which are incorporated into operational safety procedures, are also very important and are discussed at the end of this chapter.

In general, combat and many training scenarios are characterized by brief periods of high-intensity fire; thus, exposures are likely to be high in concentration, but of limited duration. Carbon monoxide, for example, has been measured in excess of 6,000 ppm in crew spaces under rapid tank-firing conditions, but this concentration decays rapidly (Figure 10-11).[4] The tank's vehicular ventilation system produces the rapid decay, returning carbon monoxide to concentrations at which the crew can continue to function without adverse effects on health or performance.[4] The breech scavenging system prevents gases from entering the crew compartment, but once gases are in the crew compartment, the vehicle ventilation system must exhaust the compartment.

Industrial workers may be exposed to much lower, but often increasing and cumulative, carbon monoxide exposures over a workday. The Occupational Safety and Health Administration (OSHA) has established a permissible exposure level (PEL) of 50 ppm for carbon monoxide. At an end-of-shift exposure of 50 to 100 ppm, symptoms of headache, giddiness, and tinnitus could occur among the workers. A worker exposed to persisting carbon monoxide concentrations of 6,000 ppm would quickly become affected to the point of developing unconsciousness and coma, and could die if not removed from exposure.[36]

Assessing Toxic Hazards

Before weapons enter full-scale production, the army requires that all systems being developed or undergoing major revision be evaluated for their potential deleterious effects on the health of the crew; this is known as the Health Hazard Assessment process (HHA) and is the subject of Chapter 6.[37] As part of this evaluation, the army has developed standardized testing procedures to determine potential toxic hazards of vehicles and other equipment.[38] Weapons mounted inside vehicles or on other weapons platforms are tested during sustained rates of fire and under various opera-

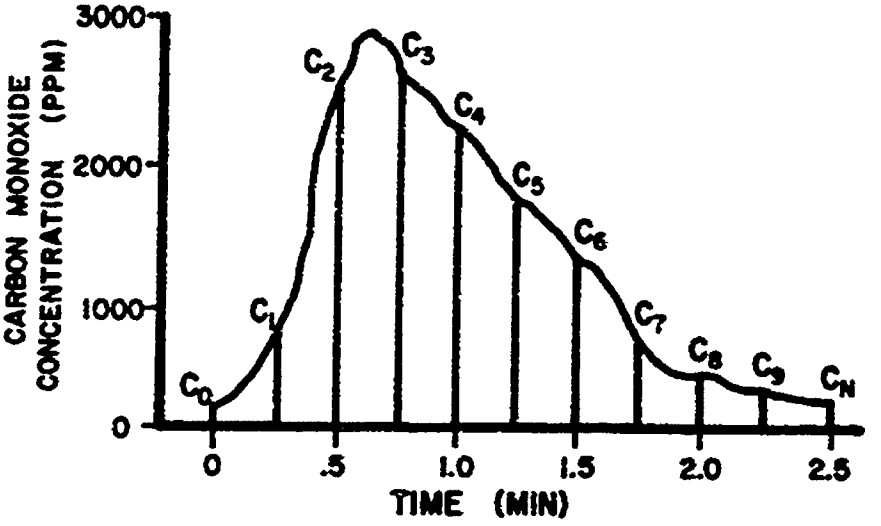

Fig. 10-11. This graphic example, based on actual measurements, shows typical carbon monoxide (CO) concentrations (in ppm) inside the crew compartment of an armored vehicle during a firing mission and the opening of the gun breech. Eleven air samples (C_0 through C_N) were obtained at 15-second intervals and analyzed for CO; the data were extrapolated to an expected, continuous concentration curve. The area under the curve may be represented by a definite integral and illustrates the total concentration of CO exposure for a given period of time. Increasing the number of measurement points improves the degree of accuracy in calculating the area. In this example, the firing and environmental conditions resulted in an estimated maximum CO concentration of almost 3,000 ppm at approximately 40 seconds after firing commenced. The ventilation system of the vehicle compartment, and perhaps a breech gas scavenging system, returned the CO concentration to near baseline level in about 2.5 minutes. This example also illustrates high baseline CO levels, exceeding 100 ppm, in the vehicle crew space. During actual field-firing situations, it is not unusual to find several armored vehicles in close proximity with their engines running, executing multiple firing missions. Therefore, high baseline levels may be obtained due to unexhausted CO from an earlier firing mission or CO from a gun muzzle or engine exhaust that has entered the vehicle being studied through an opened hatch or vent. In 1983, CO levels were monitored inside the crew shelters of a battery of M109, 155-mm self-propelled howitzers at Fort Sill, Okla. Firing was conducted with the hatches open; the environmental conditions, especially wind direction and speed, concentrated exhaust products around one particular vehicle in which the carboxyhemoglobin levels in the crew were higher than in crew members of the other vehicles. Sources: (1) Dalton BA, Deeter DP, Gaydos JC. Unpublished data from a field study performed at Fort Sill, Okla, 23–26 April 1983, by the Occupational Medicine Division, USAEHA. (2) US Army Combat Systems Test Activity. *US Army Test and Evaluation Command Test Operations Procedure: Toxic Hazards for Vehicles and Other Equipment*. Aberdeen Proving Ground, Md: DA; 1984. Report TOP 2-2-614. (3) Lucas TA. *Methodology Investigation of Toxic Gas Measurements During Weapons Firings from Vehicles While Moving: Final Report*. Aberdeen Proving Ground, Md: US Army Test & Evaluatic Command. TECOM Project 7-CO-PB9-AP1-002; 1984. Report APG-MT-5896. (4) Dalton BA. Carbon monoxide in US Arr..y tactical vehicles. *Medical Bulletin of the US Army Medical Department*. 1988;2:11–13. PB 8-88.

tional conditions, such as with their engines; ventilating systems; heaters; and nuclear, biological, and chemical (NBC) protection systems turned on and off.[28] The testing measures all sources of combustion products in the weapons system, including the weapon's propellant and the vehicle's fuel (combustion products of fuel could enter the crew compartment as exhaust gases). Vehicle crew compartments are sampled for carbon monoxide, ammonia, sulfur dioxide, nitric oxide, nitrogen dioxide, methane, hydrogen chloride, lead, and short-chain hydrocarbons such as acetylene or acetaldehyde. (Sampling decisions are based on the composition and conditions of combustion of the propellants and vehicle fuel, and depend on whether these chemicals are expected to occur.)

Studies That Predict Toxic Exposures

Relatively few studies have either assayed the actual propellant combustion products that form during firing in the field or assessed the risks experienced by soldiers. Within certain limitations, field studies can demonstrate the major toxic species in complex mixtures of combustion products. Field conditions such as

temperature, humidity, the maintenance of the weapons, and the chemical composition of the propellants that are employed all influence the mixture of combustion products. Instruments that are sophisticated enough to identify minor or trace species in these mixtures are not practicable in the field because they are too large, will easily be damaged under field conditions, or both.[9]

One study has highlighted the difficulties in sampling for combustion products in the crew compartment of the XM2 (the prototype for the Bradley Fighting Vehicle [BFV]) and the M198 howitzer.[24] Special portable collection equipment was designed to render reliable results given the time between field sampling and laboratory analysis. The sampling equipment failed during the XM2 field analysis, and accurate assessments of ammonia concentrations could not be made. Nevertheless, field analysis was able to demonstrate the major species produced following weapons firing (hydrogen, carbon monoxide, carbon dioxide, and methane) and the reduction of these concentrations after the vehicles' ventilation systems were activated (Table 10-9).

Due to the difficulties in field sampling, laboratory analysis of propellant firing has been conducted in laboratory containment fixtures such as those shown in Figure 10-8. These fixtures—specially designed chambers that allow weapons firing and sampling of combustion products—have permitted over 100 chemical species to be identified from the combustion of WC844 propellant, which is used in M16 ammunition, and over 60 chemical species from XXX, which is the name of the propellant used in the MLRS motor.

In an attempt to characterize the concentrations of major combustion products from successive rocket-motor firings in urban terrain (eg, if shoulder-held rocket systems were fired from an enclosed room), a test facility was constructed circa 1973 at the U.S. Army Missile Laboratory at Redstone Arsenal, Huntsville, Alabama.[39] Tests performed at this facility clearly demonstrated that weapons crews could be exposed to carbon monoxide, lead, and nuisance particles in excess of the short-term exposure limits (STELs) established by the American Conference of Governmental Industrial Hygienists (ACGIH)[40] and OSHA.[41]

Several computer models are available to predict combustion products. These computer programs, developed by American Cyanamid Corporation, Wayne, New Jersey; Stanford Research Institute, Menlo Park, California; and the National Aeronautics and Space Administration, Washington, D. C., are under continual modification. While the primary purpose of these programs is to solve ballistic problems, they can also produce estimates of contamination from combustion products. The programs are used to complement chemical analyses or to alert chemists to species that must be looked for. The predictions have been compared to actual field and laboratory sampling of the exhausts of various propellants; the concentrations of the predicted major chemical species agree fairly well with those that are actually measured through laboratory firing. However, there is poor agreement for the minor and trace (especially organic) species produced because the computers are coded to produce results at thermodynamic equilibrium conditions and do not consider nonequilibration processes such as incomplete combustion or condensation (soot-formation) processes.[9,19] Continued research through field studies, laboratory simulations, and computer predictive modeling is necessary to generate the information needed to assess and reduce the risk to soldiers from propellant combustion products.

TABLE 10-9

ANALYSIS OF GAS SAMPLES WITHIN THE CREW COMPARTMENT OF THE XM2 AFTER WEAPONS FIRING

Compound*	Concentrations (ppm)	
	Pre-Active Vehicle Ventilation System	Post-Active Vehicle Ventilation System
H_2	312 ± 2	39.9 ± 1.2
CO	942 ± 59	< 50
CO_2	995 ± 26	313 ± 5
CH_4	11.40 ± 0.25	3.41 ± 0.42

*Equipment failure prevented measurement of NH_3

Adapted from: Snelson A, Ase P, Bock W, Butler R. *Characterization of Combustion Products from Military Propellants.* Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory; 1983. IIT Research Institute, Contract DAMD 17-80-C-0019.

Toxicological Assessment of Combustion Products

Sophisticated chemical analyses have been developed for laboratory use that can identify hundreds of compounds found in weapons exhaust and analyze them for their toxicity. In most cases, the immediately toxic properties of the exhaust are determined by the predominant species in the mixture. Predictions of toxicological risks of exposure to the combustion products of propellants are made by comparing the measured concentrations of these individual components to the established health-based standards. The NAS

EXHIBIT 10-1

EXPOSURE ASSESSMENTS

Using the primer, igniter, and propellant compounds described in Exhibit Table 1, laboratory studies were done to qualitatively and quantitatively assess the combustion products generated during the firing of the M16 rifle and the 105-mm tank gun. Exhibit Table 2 lists the weight of selected combustion products per gram of propellant burned.

Let us say, as examples, that AMEDD personnel were asked to assess the following situations for their health risks:

- Firing 200 rounds/min of M16 ammunition over a period of 15 min in 100 m^3 of air space
- Firing 4 rounds/min of 105-mm tank gun ammunition over a period of 15 min in 100 m^3 of air space

These two situations are similar to many that are regularly presented to AMEDD health-risk assessors. These situations occur during the (*a*) design and testing of new or modified vehicles, guns and ammunition; (*b*) development of new training exercises; or (*c*) development or revision of test-firing laboratories where guns, gun parts, and/or ammunition are routinely tested. The standards or guidelines used in the evaluations may come from the Occupational Safety and Health Administration (OSHA), the American Council of Government Industrial Hygienists (ACGIH), the National Institute for Occupational Safety and Health (NIOSH), the Committee on Toxicology of the National Academy of Sciences, or may be developed by the U.S. Army Medical Department (AMEDD). In this example, let us say that AMEDD assessors, by agreement, will use the ACGIH's Threshold Limit Values (TLVs) as guidelines. The specific compounds of interest for the M16 will be limited in this example to carbon monoxide (CO) and hydrogen cyanide (HCN); for the 105-mm gun these will be CO and benzene (C_6H_6). However, assessments can be done for any emission product for which data or information is available.

The givens are (*a*) the propellant weights, (*b*) the number of rounds fired per minute, and (*c*) the number of minutes during which firing will occur. Therefore, using simple multiplication, the propellant mass required to generate combustion products can be calculated:

For the M16 Rifle:

Weight of Propellant: 1.65 g/cartridge (round)
Rounds per min: 200
Duration of fire: 15 min
Total mass of propellant burned: 4.95 kg

The total amounts of CO and HCN produced can be obtained by multiplying the total propellant burned times the amounts of CO and HCN produced per unit weight of propellant:

For CO:

3.37×10^{-1} g of CO are produced per g of propellant burned.
Therefore, 3.37×10^{-1} g x 4.95 kg = 1.668×10^{3} g of CO are produced.

For HCN:

8×10^{-4} g of HCN are produced per g of propellant burned.
Therefore, 8×10^{-4} g x 4.95 kg = 3.96 g of HCN are produced.

Assuming that the shots are fired in a space of 100 m^3 with no air dilution over the 15-min period, the amounts of CO and HCN produced by the M16 firings will accumulate.

The accumulated values are first divided by 100 m^3, then compared to the appropriate TLVs; these are found to greatly exceed the TLV values:

For CO: (TLV = 57 mg/m^3)

16.68 g/m^3 = 292.6-fold greater than the TLV

For HCN: (TLV = 11 mg/m^3)

39.6 mg/m^3 = 3.6-fold greater than the TLV

For the 105-mm Gun:

Weight of Propellant: 5.44 kg/shell (round)
Rounds per min: 4
Duration of fire: 15 min
Total mass of propellant burned: 326.4 kg

The amounts of CO and C_6H_6 formed when the 105-mm gun is fired are computed and compared to the appropriate TLVs:

For CO:

2.4×10^{-1} g of CO generated per g of propellant burned.
Therefore, 2.4×10^{-1} g x 326.4 kg = 78.34×10^{3} g of CO are produced.

For C_6H_6:

9.2×10^{-3} g of C_6H_6 are generated per g of propellant burned.
Therefore, 9.2×10^{-3} g x 326.4 kg = 30.03 g of C_6H_6.
Again, the TLVs are exceeded (assuming an undiluted air space of 100 m^3).

For CO: (TLV = 57 mg/m^3)

783 g/m^3 = 13,737-fold greater than the TLV

For C_6H_6: (TLV = 32 mg/m^3)

300 mg/m^3 = 9.4-fold greater than the TLV

Exhibit 10-1 *(continued)*

EXHIBIT TABLE 1

COMPOSITION OF ENERGETIC MATERIALS FOR TWO WEAPONS

M16 Rifle (Propellant in 5.56-mm Ammunition)				105-mm Caliber Gun			
Olin WC 844 Propellant (1.65 g)		Primer (0.032 g)		Propellant (5.44 kg)		M-83 Igniter (32 g)	
Component	Weight (%)	Component	Weight (%)	Component	Weight (%)	Component	Weight (%)
Nitrogen in nitrocellulose	13.05–13.20	Lead styphnate	35 ± 5	Nitrocellulose	28.55	Nitrocellulose	60.0
Graphite	0.4	Tetrazene	4 ± 1	Nitrogen in nitrocellulose	12.6	Unmixed black powder*	40.0
Sodium sulfate	0.5	Barium nitrate	32 ± 5	Nitroglycerin	22.23		
Calcium carbonate	0.2	Antimony sulfide	15 ± 2	Nitroguanidine	47.00		
Nitroglycerin	8.0–11.0	Aluminum powder	7 ± 1	Cryolite	0.31		
Diphenylamine	0.75–1.50	PETN	5 ± 1	Ethyl centralite	1.54		
Dibutylphthalate	3.0–6.0	Organic binder	small	Ethanol	0.24		
Total volatiles	< 2.0			Carbon	0.13		
Nitrocellulose	balance						

*The specific composition of the unmixed black powder was not defined. Black powder compositions typically lie in the following ranges: charcoal, 14%–18%; sulfur 10%–16%; and potassium or sodium nitrate, 10%–74%. The composition and amount of primer was not given. The primer mass would probably be < 0.1 g and would have a negligible impact on the overall combustion production generation.

Sources: (1) Ase P, Eisenberg W, Gordon S, Taylor K, and Snelson A. Propellant combustion product analyses on an M16 rifle and 105-mm caliber gun. *J Environ Sci Health.* 1985;A20(3):337–368. (2) Ross RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems.* Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1.

EXHIBIT TABLE 2

GASES DETECTED AND QUANTIFIED IN COMBUSTION PRODUCTS OF TWO WEAPONS

Number of Experiments	Compound	M16 Rifle		105-mm Caliber Gun	
		Mean*	± SD†	Mean*	± SD†
5	Carbon monoxide	3.37×10^{-1}	5	2.4×10^{-1}	11
7	Hydrogen cyanide	8.00×10^{-4}	52	—‡	—‡
6	Benzene	1.74×10^{-4}	18	9.2×10^{-5}	41
6	Acrylonitrile	2.36×10^{-5}	57	8.5×10^{-4}	59
6	Toluene	1.82×10^{-5}	9	2.2×10^{-5}	28
6	Cyanobenzene	9.91×10^{-6}	26	0–3.1×10^{-4}§	—‡
6	Crotonitrile	1.43×10^{-4}	24	0–1.3×10^{-4}§	—‡
6	Furan	1.10×10^{-6}	90	0–4.6×10^{-7}§	—‡
6	Dimethylnitrosamine	8.5×10^{-7}	39	0–3.0×10^{-6}§	—‡
6	Methacrylonitrile	7.6×10^{-7}	38	0–1.4×10^{-6}§	—‡
6	Quinoline	5.1×10^{-7}	50	0–1.6×10^{-6}§	—‡
6	Nitrobenzene	4.5×10^{-7}	44	0–2.3×10^{-6}§	—‡
6	2-Furfural	3.5×10^{-7}	52	0	—‡
6	Carbon disulfide	1.8×10^{-7}	56	5.2×10^{-6}	69
6	Hexane	1.7×10^{-7}	60	5.5×10^{-7}	50
6	Indane	3.0×10^{-6}	73	1.1×10^{-7}	69

*Grams of compound per gram of propellant burned

†SD is given as the percentage of the mean

‡No attempt to measure

§Where a range of values is indicated rather than the percent standard deviation of the mean, the range covers minimum and maximum values measured

Source: Ase P, Eisenberg W, Gordon S, Taylor K, and Snelson A. Propellant combustion product analyses on an M16 rifle and 105-mm caliber gun. *J Environ Sci Health.* 1985;A20(3):337–368.

has established standards—applicable to short, intensive periods of weapons firing—in the form of STELs that are especially relevant to the U.S. Army.[42]

The effects of each combustion product on different target organs determine the total potency of chemical mixtures. Interactions between these effects can be additive, synergistic, or antagonistic, and can produce acute and chronic medical problems. For example, in animal testing, carbon dioxide may have a protective effect in high concentrations in reducing carbon monoxide deaths (an antagonistic effect). Particulate matter may interact with sulfur dioxide to enhance bronchospasm over that expected from sulfur dioxide alone (a synergistic toxicological effect).[43]

Currently, the military's primary emphasis is on the identification of the major chemical species produced so that the acute effects on a soldier's performance in combat can be predicted. The level of exposure to these major species can be used to produce an assessment of risk with regard to incapacitation, morbidity, and mortality. The emphasis on immediate performance is necessary to ensure that soldiers can effectively perform their tasks and accomplish their missions; but exposure to minor or trace amounts of combustion products is also important and could serve as a risk factor for delayed health effects such as toxic effects on the reproductive system, the production of chronic disease, or carcinogenesis.

Risk assessments of combustion products should be and, eventually, will be based on the total mixture.[43] Nevertheless, until methods are established, validated, and standardized to toxicologically evaluate all propellant combustion products simultaneously, the current method of basing risk assessment on the major species produced will have to suffice.

An example of the types of calculations a risk-assessor might use in assessing health risks to soldiers is shown in Exhibit 10-1, in which the known weight of the propellant is integrated with data obtained from test firings of the M16 rifle and the 105-mm caliber gun. Often, the calculation of an exposure at any given time, or over an interval, is complicated by the episodic nature of weapons fire and by the dilution of the soldier's ambient air with clean air. In situations like these, it will probably be necessary to (*a*) plot the concentrations of the toxic chemical over the interval of interest and (*b*) determine the exposure over the entire time through a process of integration (define the area under the curve). Consultation with toxicological experts may be required when attempting to estimate the combined effect of multiple toxic chemicals. Such consultation has been obtained in the past from the Committee on Toxicology of the NAS[44] and the Armed Forces Epidemiological Board (AFEB).[45]

HEALTH EFFECTS OF EXPOSURE TO AIRBORNE PRODUCTS OF PROPELLANT COMBUSTION

Properly assessing the potential for adverse health effects associated with the operation of military weapons systems is often difficult or impossible because exposure data from operational settings are lacking. There is a pressing need for expanded, systematic study of field exposures to develop precise guidelines for both preventing adverse health effects and managing casualties of overexposures.

To diagnose and treat victims of overexposure in a timely fashion, field medical personnel should be alert for any medical sequelae arising in soldiers who may have been exposed to the products of propellant combustion. What may initially appear to be coincidental headaches or routine acute respiratory diseases in soldiers must take on new importance should an occupational history reveal problems with the ventilation system of the soldier's vehicle, that the soldier has been firing a new type of ammunition, or that the soldier has participated in missions with prolonged firing times. Reports of suspected health effects arising from exposure to combustion products are needed; these should be forwarded through medical command channels so that proper evaluations of the events can be made.[47]

As previously noted, exposure to the combustion products of propellants includes coming in contact with gases and particulate matter. The greatest hazard to soldiers is from the inhalation of these substances. In turn, the health effects that develop depend on whether and where deposition in the respiratory tract occurs. Deposition could result in direct damage to tissue or absorption of the material, which could result in a systemic health effect.

The major combustion products of most propellants are classified as (*a*) tissue asphyxiants (carbon monoxide, nitric oxide, and minor amounts of hydrogen cyanide), (*b*) irritant gases (ammonia, nitrogen dioxide, sulfur dioxide, nitric oxide, and hydrogen chloride), or (*c*) inhalable metal particulates (lead, copper, and others).

Tissue Asphyxiants

Because asphyxiants can quickly produce incapacitating central nervous system effects without warning signs, these compounds are of major immediate concern to the army. Asphyxiants act to interfere with the

delivery of oxygen to tissue cells or with the ability of the cell to use oxygen. Carbon monoxide and nitrites decrease hemoglobin's oxygen-carrying capacity. Carbon monoxide is a competitive inhibitor of the ferrous binding sites for oxygen in the hemoglobin molecule. Because carbon monoxide has a higher affinity for hemoglobin than does oxygen, the amount of oxygen available for transport to organs is reduced. Nitrites and nitric oxide also decrease the oxygen-carrying capacity of blood through the production of methemoglobin, which is less efficient than hemoglobin in releasing oxygen to the tissues. Hydrogen cyanide, which is produced by nitramine propellants such as RDX, acts to produce cellular asphyxia by interfering with the cells' cytochrome oxidase system, which disrupts oxidative metabolism.

Irritant Gases

When inhaled, the acute effects of irritant gases on the respiratory tract (eg, pulmonary edema, bronchoconstriction, and bronchorrhea) can cause an immediate decrement in a soldier's performance. Delayed effects include reactive airways dysfunction syndrome (RADS) and airway cellular destruction.[46]

RADS is characterized by continuing airway hyperresponsiveness, typically after an acute, high-level exposure to an irritant. This airway hyperresponsiveness is usually detected by physiological measurement of expiratory airflow or an increase in airway resistance after the administration of histamine or methacholine by inhalation. Although many such individuals will remain asymptomatic, some may develop overt asthmatic symptoms, often after exposure to other irritants or cold air. Airway hyperresponsiveness may persist for months or years. Case reports of RADS after exposures to chlorine, hydrochloric acid, sulfur dioxide, and ammonia have been described. The long-term health consequences of this condition are unknown. Healthcare personnel attending soldiers who may have been exposed to these irritants, especially if acute pulmonary conditions resulted, should remain cognizant of the potential late development of asthmatic symptoms and the implications of continuing exposure to irritant agents.[47]

The absorption of gases depends mainly on their solubility in the aqueous layer lining the mucosa in the upper and lower respiratory tracts. For example, both sulfur dioxide and ammonia are highly soluble and are normally absorbed in the upper airways; they are rarely deposited in the lower tract unless exposure to these gases has been overwhelming. On the other hand, gases like nitrogen dioxide are less soluble; they can bypass the upper airways and injure the lower airways. Because these relatively insoluble gases may not irritate the upper airway, they do not produce warning symptoms. However, in conditions of high humidity or moist mucous membranes, nitrogen dioxide may dissolve and form nitric and nitrous acids, which can irritate the upper passages. Additionally, certain cells in the airway may be more susceptible to these gases. An example is the Type I pneumocyte, found in the alveoli, which is thought to be particularly susceptible to damage because of its high surface area-to-volume ratio.

Particles, either solid or liquid, are deposited in the airway depending on their size. Particles greater than 10 μ are filtered out in the upper respiratory tract. Particles less than 0.5 μ are rapidly exhaled. Particles between 0.5 μ and 3.0 μ are deposited efficiently in distal airspaces, either through impact against the bronchiolar walls or through gravitational settling.[48]

Short-duration, high-intensity exposures to combustion products can lead to both immediate and delayed health problems. A fire in 1929 at the Cleveland Clinic in Cleveland, Ohio, burned 50,000 nitrocellulose films (then used for X-ray films, but now used in military propellants). The combustion of nitrocellulose liberated carbon monoxide, nitrogen oxides, and hydrogen cyanide. Ninety-seven people died immediately, probably due to asphyxiation from carbon monoxide and hydrogen cyanide. Two hours to 1 month after the fire, 26 more people died from pulmonary complications that were suspected to have been caused by exposure to nitrogen oxides.[49]

Ammonia

Ammonia is formed from the combustion of propellant compounds that contain nitrogen, especially those that contain nitroguanidine. All single-, double-, and triple-base propellants can produce ammonia. The nitrogen is converted first to molecular nitrogen during combustion, but it then quickly combines with hydrogen gas to form ammonia. Iron particles originating from heat-induced erosion of the gun tube catalyze this reaction.[9]

Due to its high solubility in water, ammonia is an upper respiratory tract, eye, and skin irritant (Table 10-10). Its *odor threshold* (ie, the lowest concentration of a substance at which its odor can be detected by humans) is below concentrations at which the eyes, nose, and throat become irritated. Once the eyes have been irritated by exposure to ammonia, lacrimation occurs. Lacrimation at ammonia concentrations of 134 to 150 ppm may be militarily significant when visual discrimination tasks are important. Acute, high-dose exposure causes coughing and severe irritation of the

TABLE 10-10

SIGNIFICANT, IMMEDIATE, REVERSIBLE EFFECTS OF AMMONIA

NH_3 (ppm)	Effects/Comments
1,000	Produces coughing
150–500	Produces changes in ventilation minute and tidal volumes and respiratory rate*
150	Produces lacrimation in subjects previously acclimated at 25–100 ppm for varying durations
140	Tolerated for 30 min by all unaccustomed subjects; for 2 h by highly motivated subjects
134	Produces lacrimation in 50% of unaccustomed subjects
110	Tolerated for 2 h by all unaccustomed subjects
100	EEGL†
50–72	Produces moderate eye, nose, and throat irritation in most subjects
20–30	Odor easily noticeable

*Tidal volume • respiratory rate = minute volume
†Emergency exposure guidance level
Source: Legters L, Morton JD, Nightengale TE, Normandy MJ. *Biological Effects of Short, High-Level Exposure to Gases: Ammonia, Carbon Monoxide, Sulfur Dioxide, and Nitrogen Oxides.* Rockville, Md: Enviro. Control, Inc. 1980. Final Summary Report AD A094505, DAMD 17-79-C9086.

throat. Early deaths can occur from laryngeal edema and severe airway obstruction. Patients who survive the initial exposure can develop tracheobronchitis, bronchoconstriction, and hypersecretion of mucus.[48] Tolerance to higher exposure levels may develop over time, and eye and upper respiratory tract irritation will lessen. For example, individuals exposed in an industrial setting to an average daily workday concentration of 100 ppm of ammonia have demonstrated a tolerance to irritation after 1 week of exposure.[50] Skin and mucous membranes are also sensitive to airborne ammonia. Due to ammonia's high solubility in water, it dissolves in the moisture of the eyes and skin,[51] and due to its alkalinity, it damages tissue by producing caustic burns.

The NAS has recommended an EEGL for ammonia of 100 ppm.[29] This value is based on limiting lacrimation and its consequent performance decrement. EEGLs represent levels at which reversible health effects may occur, but before judgment becomes impaired and responses to emergencies are inappropriate. No reliable information is available concerning inhaled ammonia's potential as a developmental, reproductive genotoxic, or carcinogenic agent.

The signs and symptoms associated with ammonia exposure partially depend on its concentration. Most soldiers can tolerate exposure to levels exceeding 140 ppm for short durations, but not for hours. However, asthmatics and those individuals who develop bronchospasm after exposure to other respiratory-tract irritants may be more sensitive to lower concentrations, shorter durations of exposure, or both. Exposure to levels exceeding 5,000 ppm have caused death due to airway obstruction in normal individuals.[52] Bronchiectasis has also been reported in previously nonasthmatic individuals after exposure to an ammonia cloud after a tank-car derailment.[53] Mild-to-moderate (50–100 ppm) exposure to ammonia can produce headache, burning of the throat, nausea, vomiting, and substernal pain.

The treatment recommended for exposure to ammonia consists of removing the casualty from the source of ammonia and flushing the eyes with water if severe irritation or lacrimation develops. Severely affected casualties who have visual difficulties (such as blurring of vision or difficulty focusing), laryngeal edema, signs of pulmonary compromise, or an abnormal chest film should be hospitalized and monitored.

Nitrogen Dioxide and Nitric Oxide

Nitric oxide and nitrogen dioxide often occur together. Nitric oxide and nitrogen gas are the first products formed from the combustion of nitrogen-containing compounds. At high concentrations (> 50 ppm), nitric oxide is rapidly converted to nitrogen dioxide in the presence of oxygen or ambient air. Both gases are considered to be pulmonary irritants, but nitrogen dioxide is generally considered to be the most toxic. Nitric oxide can cause methemoglobin formation, although its potential conversion to nitrogen dioxide should be considered the more important hazard for most exposures.[54,55]

Workers are exposed to nitrogen dioxide during welding, electroplating, and metal cleaning; nitrogen dioxide is also a combustion product of jet-engine fuels and a byproduct of blasting operations.[48] On the battlefield, nitrogen dioxide is formed by the oxidation of nitric oxide during high-temperature combustion of nitrogen-based propellants. Nitrogen dioxide can also be converted to nitric acid, which can damage and potentially cause fibrosis of lung tissue by denaturing tissue proteins. Tank crews can be exposed to nitrogen dioxide while they fire weapons, or, as a result of secondary fires and explosions, if tank rounds are ignited and burned inside the vehicles after battle damage or following vehicle accidents associated with fires. Nitrogen dioxide is heavier than air, reddish

brown in color, and has a pungent odor. It thus accumulates in the bottoms of enclosed spaces.[45] (This type of accumulation has occurred in the bottoms of poorly ventilated silos that contain grasses or hay, causing farmers to be exposed to nitrogen dioxide. The pulmonary damage that occurs is known as Silo-filler's disease.)[46]

During the World War I era, German naval personnel experienced problems with "nitrous fumes" filling gun turrets when the gun breech was opened.[13,57] These nitrous fumes were probably a mixture of nitrogen dioxide and nitric oxide. For protection, naval gunners wore respirator masks. These probably contained soda-lime and activated coconut-shell charcoal. Despite wearing the masks, some of the gunners were alleged to have developed methemoglobinemia, and death occurred. The masks probably did not remove nitric oxide and may have increased the nitric oxide in the inspired air through reduction of nitrogen dioxide. As a result of these incidents, German scientists conducted creative laboratory studies to differentiate the toxic effects of nitric oxide and nitrogen dioxide.[13,57-59] Studies of these two gases and their toxic effects in military settings have continued to the present in the U.S. Army.[60,61]

Because nitrogen dioxide has limited solubility, it causes negligible irritation in the upper airway tract. Acute exposure may cause little effect on the mucous membranes of the eye or throat, and thus there is little immediate warning of exposure. Exposed persons can apparently continue to breathe concentrations of up to 50 ppm for several minutes without cough, throat irritation, or chest discomfort (Table 10-11). The onset of respiratory symptoms may be delayed for 3 to 30 hours, at which time cough, dyspnea, acute pulmonary edema, fever, and peripheral blood leukocytosis may develop.[46]

Nitrogen dioxide reacts with lung tissue by oxidizing cellular lipids and inactivating surfactant. Type I pneumocytes lose their integrity and allow interstitial fluid to enter the alveoli. Type II pneumocytes are then activated, become hyperplastic, and may fill up the alveolar spaces, leaving no space for gas exchange to occur. Untreated pulmonary edema may progress to death.

Death from pulmonary edema can occur at nitrogen dioxide exposures of 150 ppm for less than 1 hour. Furthermore, even if the casualty apparently recovers from an acute exposure, he or she could present 2 to 3 weeks later with signs and symptoms of *bronchiolitis obliterans*. This condition is noted for its fibrotic destruction of the bronchioles, and its signs and symptoms include fever, cyanosis, and dyspnea.[52] No specific X-ray findings are associated with this condition.[46]

The deleterious respiratory changes induced by exposure to airborne nitrogen dioxide are partially dependent on its concentration. Mild, irreversible effects on lung function can be observed at exposures below 5.0 ppm. At 1.0 ppm, symptoms such as coughing, chest tightness, and laryngitis can appear, but these symptoms are reversible. Individuals with asthma or chronic bronchitis may experience mild, reversible symptoms at nitrogen dioxide levels of 0.5 ppm over a 2-hour period. This level is the odor threshold for nitrogen dioxide.[53]

Little evidence is available to assess concentration-time exposure parameters in relation to observed health effects for nitrogen dioxide. The army uses OSHA PELs or recommendations of the ACGIH, whichever are more conservative. However, the standards set by OSHA and the ACGIH are more applicable to industrial settings. In militarily unique equipment, systems, and operations, special DA standards are devised.[6] These may be based on guidance documents that the army has asked the AFEB or the NAS of the National Research Council (NRC) to draw up. If the question is

TABLE 10-11

TOXIC EFFECTS OF HUMAN EXPOSURE TO NITROGEN DIOXIDE

NO_2 (ppm)	Exposure Time (min)	Effects/Comments
1,000	15	Immediate incapacitation; respiratory and eye injury followed by death
100	60	Immediate respiratory and eye injury with progressive respiratory injury and death
50	60	Immediate respiratory and eye irritation with possible subacute and chronic pulmonary lesions
25	60	Immediate respiratory irritation with chest pain
5	60	Acute reversible respiratory function effects
5	15	ACGIH's STEL
~1	60	Equivocal respiratory function effects and impaired dark adaptation of vision

STEL: short-term exposure limit

Source: Davis DL. Executive Director, Board on Toxicology and Environmental Health Hazards, National Research Council Commission on Life Sciences, to Ranadive M. The Pentagon, Washington, DC. Written communication; 14 March 1985.

one of toxicology, the NAS Committee on Toxicology will consider the question or issue. If there are engineering implications, the multidisciplinary NRC may be asked to evaluate this situation. In 1985, at the army's request, the NAS Committee on Toxicology reviewed a situation involving tank-gun ammunition. The army recognized the need to develop blast-proof door seals on the bustle, to ensure that combustion products of burning propellants (a situation that could occur with battle damage) would not reach tank crews in significant concentrations. The NRC's committee studied the potential problem and recommended maximum exposure levels (see Table 10-8). These limits, which are essentially EEGLs and pertain to the ability to escape a contaminated environment, can be compared to those established by the ACGIH for civilian workplaces. The ACGIH standard is more conservative (their 15-min STEL is 5 ppm).[41] In a military training situation, exposures should be kept as low as possible, but at a minimum should be below the ACGIH criteria. For emergency escape purposes, the NRC guidelines should be used to assess the efficacy of hazard-control systems. However, the NRC notes that sensitive individuals may experience mild wheezing and chest tightness on exposure to 0.5 ppm of nitrogen dioxide for 2 hours.[28]

The treatment of casualties who are exposed to nitrogen dioxide consists of removing them from the offending source, keeping them at rest, and administering oxygen.[56] Because of the danger of pulmonary edema even in the absence of acute pulmonary irritation, close observation for 24 to 36 hours is also indicated after any significant exposure. Hospitalization is necessary for any person who has symptoms of pulmonary irritation manifested by increased pulse and respiratory rates. Supplemental oxygen may be indicated and corticosteroids may be given in life-threatening situations, but documentation of the beneficial effects of steroids is lacking.[48,53,62]

Blood methemoglobin should be measured as soon as possible after a casualty has been exposed to nitrogen oxides, *especially if the casualty is cyanotic.* Methemoglobin cannot transport oxygen efficiently; therefore, hypoxemia cannot easily be corrected with supplemental oxygen when methemoglobin is present. The patient may require intravenous treatment with methylene blue [3,7-*bis*(dimethylamino)phenazathionium chloride in a dosage of 1–2 mg/kg body weight],[53] which reduces methemoglobin's ferric iron back to the ferrous state found in normal hemoglobin:

Sulfur Dioxide

Sulfur dioxide may be formed when antimony sulfide (used in primers), sulfur (used in black powder igniter), and potassium sulfate (a flame retardant used in propellants) are oxidized.[9] Sulfur dioxide is a heavy, irritating gas with a characteristic pungent odor. It reacts with water to form sulfurous acid and therefore mucous membranes in the eyes, mouth, and upper respiratory tract are at risk for injury. Sulfurous acid will also burn the lungs.[46]

Sulfur dioxide induces bronchoconstriction; the stimulation probably occurs via an afferent nerve after direct stimulation of its sensory end organ in the airway wall. This stimulation leads to efferent vagal-induced contraction of smooth muscle in the airway. Asthmatic subjects exposed to sulfur dioxide at 5 ppm for 5 minutes while exercising have developed bronchospastic attacks.[63] However, research performed with normal adult subjects has shown that

- continuous exposure to sulfur dioxide at 3.0 ppm may occasionally produce a reversible decrease in small-airway compliance[53];
- exercise can also potentiate functional airway impairment in the presence of atmospheric sulfur dioxide[64];
- an estimated 10% to 20% of the adult population will exhibit hyperreactive airways after exposure to sulfur dioxide[64]; and
- exposure to less than 25 ppm can also cause other symptoms, including irritation of the mucous membranes, increased respiratory rate and depth, and coughing.[64]

Workers who are chronically exposed to 5 to 20 ppm can become acclimatized to these effects: their sense of smell becomes less acute, their reflex cough is lessened, and they are less aware of the irritation in their upper airways.[53] This acclimatization will not occur in soldiers who experience only intermittent high-level exposures. In general, 20 to 30 ppm of sulfur dioxide is very disagreeable.[65]

Exposure limits and an EEGL have been established for sulfur dioxide; the NAS recommends an EEGL of 10 ppm for a 1-hour exposure.[64] In addition, the ACGIH and OSHA have established time-weighted average (TWA, an average 8-hour exposure within a normal workday) exposure values of 2 ppm.[40,41]

The treatment of casualties who are exposed to sulfur dioxide is to remove the victim from the source and flush the eyes with water if they are irritated or lacrimating. However, severely symptomatic individuals should be hospitalized and monitored for the

signs of pulmonary edema. Bronchiolitis obliterans can occur within days or weeks after the patient has recovered from a moderate-to-severe exposure.

Hydrogen Chloride

Hydrogen chloride exists both as a gas and as an aqueous acid aerosol (microdroplet solutions of hydrogen chloride and water). Hydrogen chloride has an extremely high affinity for water, and its acidic properties make it a strong irritant of mucous membranes.

Although the odor threshold of hydrogen chloride is 1 to 5 ppm, 10 ppm is a noxious concentration. In fact, inhalation of hydrogen chloride causes an individual to promptly leave the offending environment. Inhalation of hydrogen chloride causes several respiratory tract signs and symptoms, including noticeable pain, coughing, inflammation, edema, and, at high concentrations, laryngeal or bronchial constriction (Table 10-12 and Figure 10-12).

Early test-firing of the MLRS, in which a perchlorate-based propellant is used, showed that hydrogen chloride was produced in large quantities, and was present at levels of 25 to 50 ppm in the crew compartment. This level irritates the eyes, and the resultant lacrimation would have prevented the crew from performing its mission effectively. During the late 1970s, the MLRS was reengineered: the crew compartment of the subsequent version could be overpressurized, and gas and particulate filters were included on its air intake.[40]

The shoulder-held Stinger, an antiaircraft missile, also produces high concentrations of hydrogen chloride when it is fired. Soldiers who fired the Stinger were required to wear chemical protective masks to reduce their exposures to hydrogen chloride, but the masks obstructed their views through the missiles' sighting devices. To sight their targets, soldiers took off their masks and held their breaths while they fired the weapons. Their intention was to improve their tracking capability while reducing the irritating effects of hydrogen chloride in the upper airways.

Several governmental regulatory agencies have established limits for exposure to hydrogen chloride. Both OSHA and the ACGIH recommend a ceiling of 5 ppm.[40,41] The NRC proposed a 10-minute EEGL of 100 ppm, but because this level is not likely to be tolerable for any sustained duration (up to an hour), a 1-hour EEGL of 20 ppm was adopted.[29]

The treatment of casualties who have been exposed to hydrogen chloride is mostly palliative: remove the casualty from the source and treat the symptoms. Little information is available concerning the long-term effects on health that may arise after acute exposure to hydrogen chloride.[40]

TABLE 10-12

TOXIC EFFECTS OF HUMAN EXPOSURE TO HYDROGEN CHLORIDE

HCl (ppm)	Exposure Time	Effects/Comments
1,000–2,000	Brief	Dangerous for even short exposures (pain; laryngeal and bronchial constriction)
50–100	1 h	Tolerable
100	10 min	EEGL*
91	—	Median concentration for odor detection
10–50	Few hours	Maximal tolerable concentration
35	—	Throat irritation after short exposure
10	Prolonged	No adverse effects
1–5	—	Odor threshold

*Emergency exposure guidance level

Source: National Research Council Committee on Toxicology. Ammonia, hydrogen chloride, lithium bromide, and toluene. In: *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants*. Vol 7. Washington, DC: National Academy Press; 1987.

Inhalable Metal Particulates

The inhalation of metal particulates can, over time, be expected to cause chronic health problems. Of the mass of inhalable metal particulates formed when an M16 round is fired, lead constitutes approximately one-half, and copper approximately one-third (the effects of inhaled lead particulates are described in Chapter 12, Lead). Inhalable copper comes from brass cartridges and copper jackets of bullets. Another source of copper is the rotating band on artillery shells.[9] Although most particles still consist of lead, the relative percentage of inhalable copper increases and that of lead decreases as more rounds are fired.[24] To reduce the amount of copper deposited in the barrel bore, lead foil has been added to artillery rounds, which, of course, also increases the amount of particulate lead produced. Tin foil is being considered as a replacement for lead foil, and no adverse health effects are expected from this substitution.[31]

The signs and symptoms of copper inhalation include nasal congestion, nasal septum perforation, nasal ulceration, and metal fume fever, which is characterized by a sudden onset of fever, chills, malaise, and headaches.[53] Therefore, OSHA has established an 8-hour TWA for copper dust of 1.0 mg/m^3 air.[40]

Fig. 10-12. This chest X-ray film was made 6 hours after a 19-year-old soldier was exposed to hexachlorethane smoke when a hexachlorethane grenade was thrown into her tent. The smoke is generated in the thermochemical reaction of zinc oxide, hexachlorethane, and aluminum metal, and is composed predominantly of zinc chloride with 1% to 2% aluminum chloride. Zinc chloride is a potent respiratory irritant with pulmonary effects similar to those caused by inhaled hydrogen chloride. This film shows bilateral, diffuse, air-space opacities, with an interstitial component consistent with pulmonary edema.

Antimony, barium, and zinc together constitute less than 5% of the metallic inhalable particulates that are produced when an M16 round is fired.[5] In normal firing conditions, these metallic particles are expected to occur in low concentrations, and their health effects are expected to be negligible.[5,6]

Antimony trioxide is a combustion byproduct of antimony sulfide, which is used in primers and antimonial-lead alloy bullets. (Because lead antimony has a higher melting point than lead alone, its use in the bullet's core allows the weapon to be fired at a faster rate.) Antimony trioxide is an irritant to mucous membranes, and acute inhalational exposure can cause coughing, loss of appetite, and gastrointestinal pain. Chronic exposure has caused lung fibrosis.[5]

Barium is a combustion byproduct of barium nitrate, which is also found in certain primers. Barium can also be a local irritant to mucous membranes and can cause coughing. Heavy industrial exposure to barium oxides or sulfates can produce a benign pneumoconiosis. Barium oxide is strongly alkaline and can cause eye and skin irritation. It is extremely toxic if ingested. No long-term health effects other than pneumoconiosis are known.[5]

Zinc, like copper, comes from brass bullet cases. Zinc can be a mild mucous-membrane irritant during exposure. Zinc oxide causes metal fume fever; it often occurs during periods away from work and can last 6 to 24 hours. No chronic health effects are described for zinc.[5]

Depleted uranium is a potential risk to personnel who may be exposed to the aerosolized products of a burning tank round (eg, during a fire in a tank bustle where the armor-piercing rounds are stored, or if a DU round has penetrated the armor into the tank's crew compartment). In these cases, particulate uranium oxide would form, some of which would be respirable (< 10 μ). Because DU contains both uranium ^{238}U and ^{234}U, both of which are alpha emitters, a theoretical risk of lung cancer exists for those who inhale these particulates. (Exposure to DU in this circumstance would be expected to be short-lived, however, and the far greater risk to the crew would be from the conflagration or the ballistic properties of the armor-piercing projectile.) Toxicity studies with animals have not borne out hypothesized carcinogenicity, although fibrotic lung changes have been noted with exposure to uranium oxides. These changes have not been noted in humans. The primary hazard of exposure to elemental uranium would be heavy metal toxicity in the form of nephrotoxicity, if ingested. However, feeding studies in rats have failed to demonstrate toxicity from the insoluble uranium oxide.[34,70,71]

Allied troops received wounds from fragments of DU during Operation Desert Storm. In some soldiers, the fragments were not removed. This raised a concern that the solubility of uranium in body fluids may exceed the solubility of uranium in water and therefore pose a greater health threat than was originally considered. In vitro solubility is being studied and soldiers with retained fragments of DU are being followed medically to detect any adverse effects.[34]

CURRENT MEASURES TO CONTROL EXPOSURE

The measures that the army uses to control soldiers' exposure to propellant combustion products are not unlike the measures that civilian industrial operations take to control their workers' exposures. Engineering controls (such as incorporating appropriate ventilation into the design of weapons systems) are the best for limiting exposure, followed by administrative controls (such as limiting firing or personnel exposure times), and finally, by providing PPE (such as respirators) if the other measures are inadequate. The health hazard assessment process identifies potential health hazards that arise from the accumulation of combustion products so that these controls can be implemented.

Currently fielded protective masks may efficiently remove chemical-warfare agents, but they may be unable to efficiently filter out nitrogen oxides, carbon monoxide, and low-molecular-weight aldehydes.[72,73] This caveat is important for medical officers to consider when they evaluate casualties who have been exposed to the products of propellant combustion. For example, during Operation Desert Storm, the protective masks that tank crews were issued to wear against the threat of chemical warfare may not have protected them against propellant combustion products inside their own tanks; these crews would have been particularly at risk had their tanks sustained battle damage and their own munitions caught fire.

Several engineering mechanisms are used in modern army fighting vehicles to limit the crew's exposure to propellant gases. The bore evacuator was an earlier engineering approach that was designed for large guns (Figure 10-13). Another system, the M81 closed-breech scavenger, used an inert gas to flush out the gun tube. The principal guarantor of the crew's safety is the vehicle's ventilation system, however, which can purge the pollutants by exhausting either the entire crew area, the area of the breech of the individual weapons, or both.[9]

Other possible means of limiting exposures include placing the gun breech outside the crew compartment, reformulating the propellant chemicals or other components of the shell (eg, adding more zinc to bullets to reduce the production of lead particulates), or redesigning military small-arms ammunition so that the metal jacket completely encloses the bullet.

The U.S. Army, primarily as a cost-saving measure, is considering using liquid propellants such as hydroxylammonium nitrate (HAN) in its artillery weapons. Computerized projections of the theoretical combustion products of HAN indicate that the major chemical species produced will be carbon dioxide and nitrogen in relatively low concentrations, water vapor, and minor amounts of carbon monoxide and mixed nitrogen oxide gases.[74] However, the reduced threat to soldiers' health from exposure to these combustion products may be offset by the potential for soldiers' direct exposure to the propellant liquid or its vapors. Not only can HAN cause methemoglobin formation, direct exposures to this propellant or its vapor have also produced respiratory distress, splenic enlargement, and anemia in laboratory animals.[75]

The air force and army are now evaluating gel propellants to replace the current, solid propellant, aircraft emergency escape systems (aircrew ejection systems). The system consists of a gas generator that applies pressure to separate gel fuel and gel oxidizer

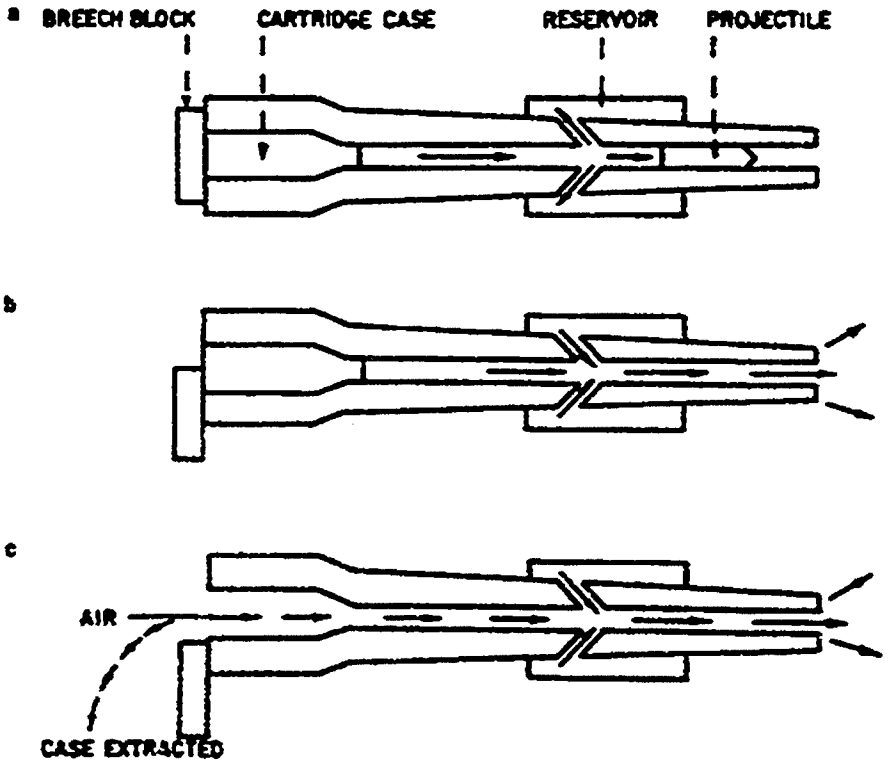

Fig. 10-13. (a) Following the firing of the weapon, gases are drawn into the reservoir as the projectile passes. (b) After the projectile is ejected from the muzzle, the gas previously drawn into the reservoir is expelled from the muzzle. (c) As the breech is opened to eject the cartridge case or reintroduce another round, air from the crew compartment is drawn into the bore and expelled, along with any combustion gas remaining in the reservoir, through the muzzle. Source: Ross RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems.* Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1.

tanks, which combine in combustion chambers to produce a significant thrust advantage over the current solid propellant systems. The gel fuel consists of aluminum powder, monomethyl hydrazine and hydroxy propyl cellulose as the gelling agent. The oxidizer gel consists of inhibited red fuming nitric acid, lithium nitrate, and fused silica. This gel propellant offers advantages over its liquid version in that it is insensitive to detonation if the fuel and oxidizer tanks are penetrated by projectiles during combat. In this situation, partial operability of the system would be maintained. Combustion products would not pose a threat to the aircrew who have ejected. However, some leakage of inhibited red fuming nitric acid could result in the leakage of nitrogen tetraoxide, which quickly dissociates to nitrogen dioxide. In this situation, aviators would probably switch from their respirator's regular air supply to 100% oxygen to avoid exposure.[76] Overall, gel propellants offer distinct safety advantages over both solid and liquid propellants and could be considered as components of a wide variety of weapons systems in the future.

SUMMARY

Although soldiers have been exposed to the deleterious effects from propellant combustion products for more than 1,000 years, innovations in weapons and tactics during the 1980s have increased the potential for exposure to these health hazards. The wide variety of gun and rocket propellants now in use have the potential to give rise to an even more complex array of combustion products during firing. In general, however, certain chemical species are thought to pose the greatest risk to soldiers, predominately through inha-

lation. These include asphyxiants (such as carbon monoxide and nitric oxide), pulmonary irritants (such as nitrogen dioxide, ammonia, and hydrogen chloride), and heavy metal particles (such as lead).

Evaluation of potential hazards has been assisted by computer modeling of predicted species; however, there have been few studies to validate the prediction capability. This is due to the difficulty of conducting appropriate chemical sampling under both laboratory and field conditions. Nevertheless, efforts are made during the health hazard assessment process to gather appropriate data, assess the health impact on soldiers, and design protection systems into military hardware while it is still being developed.

The potential for significant toxic effects is inherent in the firing of military weapons. Medical officers should thoroughly understand the occupational hazards that soldiers face, especially those from jobs that are performed within enclosed spaces. Combustion products of propellants encountered in training and battlefield conditions can have acute as well as long-term effects. Medical personnel must also recognize that engineering controls designed to reduce hazardous exposures (such as ventilation systems) may fail, administrative controls (such as limiting firing times) may be ignored, and PPE (such as respirators) may be discarded, worn improperly, or possess an inadequate protective factor.

REFERENCES

1. Winter FH. *Rockets into Space*. Cambridge, Mass: Harvard University Press; 1990.
2. Allan R. Propulsion Directorate Scientist, US Army Missile Command, Huntsville, Ala. Interview by JC Gaydos; 20 July 1992.
3. Gaydos JC. A historical view of occupational health for the soldier. *Medical Bulletin of the US Army Medical Department*. 1988;2:4–6. PB 8-88.
4. Dalton BA. Carbon monoxide in US Army tactical vehicles. *Medical Bulletin of the US Army Medical Department*. 1988;2:11–13. PB 8-88.
5. Eshel D. Soviet tanks: An Israeli view. *Armor*. 1988;(May–June):14–18.
6. US Department of the Army. *Preventive Medicine*. Washington, DC: DA; 1986. Army Regulation 40-5.
7. US Department of the Army. *Patient Administration*. Washington, DC: DA; 1983. Army Regulation 40-400.
8. Wilkinson NB. *Explosives in History, The Story of Black Powder*. Chicago: Rand, McNally; 1966.
9. Ross RH, Pal BC, Lock S, et al. *Problem Definition Study on Techniques and Methodologies for Evaluating the Chemical and Toxicological Properties of Combustion Products of Gun Systems*. Vol 1. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1988. Final Report AD-ORNL-6334/V1.
10. Biringuccio V. *The Pirotechnia of Vannoccio Biringuccio*. Cambridge, Mass: MIT Press; 1942: 410–411.
11. *The New Encyclopedia Britannica*. Vol 5, 15th ed. Chicago: Encyclopedia Britannica Inc; 1987.
12. Farrar CL, Leeming DW. Military ballistics—A basic manual. In: *Battlefield Weapons Systems and Technology Series*. Vol 10. Oxford, England: Brassey's Publishers; 1983.
13. Gaydos JC. A historical review of the need for military toxicology and the US Army's response. In: *Chemical Risk Assessment in the Department of Defense (DOD); Science, Policy and Practice*. Cincinnati, Oh: American Conference of Governmental Industrial Hygienists; 1992: 175–181.
14. Cooper B. *The Ironclads of Cambrai*. Toronto, Canada: Ryerson Press; 1967.
15. Ley W. Rockets. In: *Encyclopedia Americana International Edition*, Vol 23. New York: Americana Corp; 1966: 599a–599j.
16. Miller G, Felton J, Krause W, Lewis P. Mutagenic soot in ballistic ranges. *Annual Technology Review (1987)*. Livermore, Calif: Hazards Control Department, Lawrence Livermore National Laboratory, University of California; 1988: 15–24.

17. US Department of the Army. *Guidelines for Safe Response to Handling, Storage, and Transportation Accidents Involving Army Tank Munitions or Armor Which Contain Depleted Uranium.* Washington, DC: DA; 1990. Technical Bulletin 9-1300-278.

18. Ciccone TQ. *Primers.* In: Kaye SM, ed. *Encyclopedia of Explosives and Related Items.* Vol 8. Dover, NJ: US Armament Research and Development Command; 1978: P372–P387.

19. Fifer RA. Ballistics Research Laboratory, Aberdeen Proving Ground, Md. Written communication; 21 September 1992.

20. Lindner V. Propellants. In: Kirk RE, Othmer PF, eds. *Encyclopedia of Chemical Toxicology.* Vol 9, 3rd ed. New York: John Wiley; 1980: 620–671.

21. US Department of the Army. *Military Explosives.* Washington, DC: DA: 1984. Technical Manual 9-1300-214.

22. US Army Environmental Hygiene Agency. *Initial Health Hazard Assessment Report on the Stinger Alternate Flight Motor 69-37-XK06-92.* Aberdeen Proving Ground, Md: USAEHA; 1992. Requirement Control Symbol MED 388.

23. Ase P, Eisenberg W, Gordon S, Taylor K, Snelson A. Propellant combustion product analyses on an M16 rifle and a 105-mm caliber gun. *J Environ Sci Health.* 1985;A20(3):337–368.

24. Snelson A, Ase P, Bock W, Butler R. *Characterization of Combustion Products from Military Propellants.* Fort Detrick, Frederick, Md: US Army Medical Bioengineering Research and Development Laboratory. 1983. IIT Research Institute, Contract No. DAMD 17-80-C-0019.

25. Urbanski T. Smokeless powder. In: *Chemistry and Technology of Explosives.* Vol 3. New York: Pergamon Press; 1983; 528–569.

26. US Army Armament, Munitions and Chemical Command, Department of Methodology and Technology for Identifying and Quantifying Emission Products from Open Burning and Open Detonation Thermal Treatment Methods. *Final Report. Test Summary. Bangbox Test Series.* Vol 1. Rock Island, Ill: USAAMCC; 1992.

27. Stewart PC, Deputy Director for Logistics Readiness. *Open Burning/Open Detonation Study Final Report.* Rock Island, Ill: US Army Armament, Munitions and Chemical Command. Memorandum; 09 April 1992.

28. Davis DL. Executive Director, Board on Toxicology and Environmental Health Hazards, National Research Council Commission on Life Sciences. Written communication to Ranadive M. The Pentagon, Washington. DC; 14 March 1985.

29. National Research Council. Committee on Toxicology, Board on Environmental Studies and Toxicology, Commission on Life Sciences. Ammonia, hydrogen chloride, lithium bromide and toluene. In: *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants.* Vol 7. Washington, DC: National Academy Press; 1987.

30. US Department of the Army. *Supplemental Environmental Assessment for the Proposed Elimination of Intermediate-Range and Shorter-Range Missiles at Longhorn Army Ammunition Plant, Pursuant to the INF Treaty.* Washington, DC: DA; 1988.

31. US Army Environmental Hygiene Agency. *Updated Health Hazard Assessment (HHA) on the Substitution of Inorganic Tin Foil for the Lead Foil Decoppering Agent in the Propelling Charge of the M109A6 Paladin 105-mm Self-Propelled Howitzer Munition (Project No. 69-37-4563-91).* Memorandum for Headquarters, DA; 2 Aug 1991.

32. Kronoveter KJ. *A Study of the Effectiveness of Copper Jacketed Ammunition in Lowering Lead Exposures in Indoor Firing Ranges.* Cincinnati, Oh: National Institute of Occupational Safety and Health; 1978. Technical Assistance Report TA-78-22.

33. Lee S. *Health Hazard Evaluation Report on the Federal Reserve Bank.* Cincinnati, Oh: National Institute of Occupational Safety and Health; 1982 Report HETA-81-470-1040.

34. *DU Exposure with Field Ammunition.* Personal communication between Bower M, Chief, Medical Health Physics Branch, Health Physics Division, USAEHA, Aberdeen Proving Ground, Md.; and Gaydos J, Uniformed Services University of Health Sciences, Bethesda, Md; 24 July 1992.

35. Hatch TF. The armored force medical research laboratory in World War II. *Medical Bulletin of the US Army, Europe.* 1985;42:22–26.

36. US Department of Health and Human Services. *Occupational Health Guidelines for Carbon Monoxide.* Washington, DC: National Institute of Occupational Safety and Health; 1978: 1–4. NIOSH Publication 81-123.

37. US Department of the Army. *Health Hazard Assessment Program in Support of the Army Materiel Acquisition Decision Process*. Washington, DC: DA; 1983. Army Regulation 40-10.

38. US Army Test and Evaluation Command. *Test Operation Procedure 2-2-614*. Aberdeen Proving Ground, Md; Army Test and Evaluation Command: 1984. AMSTE-RP-702-101, AD-A 149164.

39. Thorn LB. A rocket exhaust toxicity evaluation facility. Presented at the Joint Army-Navy-NASA-Air Force Safety and Environmental Protection Subcommittee Meeting; Las Cruces, NM; May 1984.

40. American Conference of Governmental Industrial Hygienists. *Threshold Limit Values and Biological Exposure Indices for 1990–1991*. Cincinnati, Oh: ACGIH; 1990.

41. 29 CFR, Part 1910 § 1000.

42. National Research Council, Committee on Toxicology. *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants*. Vols 1–8. Washington, DC: National Academy Press; 1984.

43. National Research Council. *Complex Mixtures: Methods for In Vivo Toxicity Testing*. Washington, DC: National Academy Press; 1988.

44. Krewski D. Thomas R. Carcinogenic mixtures. *Risk Analysis*. 1992;12(1):105–1132.

45. Woodward TE. *The Armed Forces Epidemiology Board: Its First Fifty Years*. Washington, DC: Center of Excellence, Office of The Surgeon General, and Walter Reed Army Medical Center; 1990.

46. Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS). *Chest*. 1985;85(3):376–384.

47. Kennedy SM. Acquired airway hyperresponsiveness from nonimmunogenic irritant exposure. In: *Occupational Medicine: State of the Art Reviews*. Vol 7, No 2. Philadelphia: Hanley & Belfus; 1992: 287–300.

48. Murray JF, Nadel JA. Chemical agents. In: *Textbook of Respiratory Medicine*. Philadelphia: WB Saunders; 1988: Chap 71.

49. Gregory KL, Malinoski VF, Sharp CR. Cleveland clinic fire survivorship study, 1929–1965. *Arch Environ Health*. 1969;18:508.

50. Ferguson WS, Koch WC, Webster LB, Gould, JR. Human physiological response and adaptation to ammonia. *J Occup Med*. 1977;19(5):319–326.

51. Jarudi NI, Golden B. Ammonia eye injuries. *J Iowa Medical Soc*. 1973;63:151–158.

52. Henderson Y, Haggard HW. *Noxious Gases and the Principles of Respiration Influencing Their Action*. New York: Reinhold Publishing: 1943.

53. Rom, WN, ed. *Environmental and Occupational Medicine*. Boston: Little, Brown; 1983.

54. Doull J, Klaassen CD, Amdur MO, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 2nd ed. New York: Macmillan Publishing; 1980: 266.

55. US Department of Health and Human Services. *Occupational Health Guideline for Nitric Oxide*. Washington, DC: USDHHS; 1978: 1–3.

56. Fishman A, ed. *Pulmonary Diseases and Disorders*. Vol 1, 2nd ed. New York: McGraw-Hill; 1988: 851–852.

57. Utidjian, HMD, Corporate Medical Director, Central Medical Department; American Cyanamid Company. *Some Thoughts on the Toxicity of the Oxides of Nitrogen*. Wayne, NJ. Written communication; 20 June 1990.

58. Flury F. The role of nitric oxide in nitrous gas poisoning. *Naunyn-Schmiedebergs Arch Exp Pathol Pharmakol Ger*. 1930;157:104–106.

59. Pfleseer G. The significance of nitric oxide in poisoning by nitrous gases. *Naunyn-Schmiedebergs Arch Exp Pathol Pharmakol Ger*. 1935;179:545–557.

60. Stavert DM, Lehnert BE. Nitric oxide and nitrogen dioxide as inducers of acute pulmonary injury when inhaled at relatively high concentrations for brief periods. *Inhal Toxicol.* 1990;2:53–67.

61. Stavert DM, Lehnert BE. Potentiation of the expression of nitrogen dioxide-induced lung injury by postexposure exercise. *Exp Res.* 1989;48:87–99.

62. Schwartz DA. Acute inhalational injury. *Occupational Medicine, State of the Art Reviews.* 1987;2(2):297–318.

63. Sheppard D, Saisho A, Nadel JA. Exercise increases sulfur dioxide–induced bronchoconstriction in asthmatic subjects. *Am Rev Resp Dis.* 1981;123:486–491.

64. National Research Council, Committee on Toxicology. Chlorine; chlorine trifluoride; ethanolamine; fluorocarbon 11, 12, 21, 113, 114; isopropyl alcohol; phosgene; sodium hydroxide; sulfur dioxide; vinylidene chloride; xylene. In: *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants.* Vol 2. Washington, DC: National Academy Press; 1984.

65. Legters L, Morton JD, Nightengale TE, Normandy MJ. *Biological Effects of Short, High-Level Exposure to Gases: Ammonia, Carbon Monoxide, Sulfur Dioxide, and Nitrogen Oxides.* Rockville, Md: Enviro Control, Inc; 1980. Final Summary Report AD A094505, DAMD 17-79-C9086.

66. Cutting JW, Director of Occupational and Environmental Health, US Army Environmental Hygiene Agency. *Environmental Assessment of Training for the Multiple Launch Rocket System (MLRS).* Aberdeen Proving Ground, Md: USAEHA; Written communication to Commander, Fort Sill, Okla; 31 August 1982.

67. US Department of Health and Human Services. *Occupational Safety and Health Guidelines for Antimony and its Compounds (as Sb).* Washington, DC: USDHHS; 1988. NIOSH Document 81-123.

68. US Department of Health and Human Services. *Occupational Health Guidelines for Soluble Barium Compounds (as Barium).* Washington, DC: USDHHS; 1978. NIOSH Document 81-123.

69. US Department of Health and Human Services. *Occupational Health Guidelines for Zinc Oxide Fume.* Washington, DC: USDHHS; 1978. NIOSH Document 81-123.

70. Committee on the Biological Effects of Ionizing Radiation. *Health Risks of Radon and Other Internally Deposited Alpha Emitters: BEIR IV Report.* Washington, DC: National Academy Press; 1988.

71. US Department of Health and Human Services. *Occupational Health Guidelines for Uranium and Insoluble Compounds (as Uranium).* Washington, DC: USDHHS; 1978. NIOSH Document 81-123.

72. Gross RA Jr, Director, Physical Protection, Chemical Research, Development and Engineering Center. *Estimates of Gas Mask Performance in Removing Combustion Products.* Aberdeen Proving Ground, Md: US Army Chemical Research, Development and Engineering Center [now reorganized as US Army Chemical and Biological Defense Agency]. Written communication to Ripple JG, Staff researcher, Dept. of Respiratory Research; Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, DC; 13 February 1987.

73. US Army Test and Evaluation Command, Engineering Directorate, Physical Test Division. *Nitrogen Dioxide Removal by Special Charcoal, M1A1 Ammunition Compartment Fire Containment Test.* Aberdeen Proving Ground, Md: USA TECOM; 1985. Laboratory Report 85-CC-210, Project No. 1-EE-965-000-255.

74. Bagge K. *Proposed Concepts for Liquid Propellants and their Impact on Clothing and Industrial Equipment.* Natick, Mass: US Army Natick Research, Development, and Engineering Center; 1988. Technical Report 88-024L.

75. Snodgrass HL, Asaki AE, Harvey JG, Metker LW. *Health Hazard Evaluation of Liquid Monopropellants, Subchronic Inhalation of Hydroxylammonium Nitrate, Phase 4.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1985. Report 75-51-0132-85.

76. US Department of the Air Force. *Environmental, Safety, and Health Aspects of Gel Propulsion Systems on Aircraft Emergency Systems. Final Report.* Harry G. Armstrong Aerospace Medical Research Laboratory (AFSC). 25 February 1991.

Chapter 11

CARBON MONOXIDE

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INTRODUCTION

Carbon monoxide is a colorless, odorless, tasteless and nonirritating gas formed as a by-product in the incomplete combustion of carbonaceous materials. Frequently, but not reliably, the presence of carbon monoxide is accompanied by the odor of unburned organic matter or fuel.[1] Because of its high inherent toxicity and extensive exposure potential, carbon monoxide has historically been considered not only the most widespread poison known but also the most significant toxic gas in the workplace.[2] Human exposure to carbon monoxide has been estimated to account for approximately 2,300 suicides and 1,500 accidental deaths annually (ie, more than one-half the yearly poisoning deaths in the United States). In addition, an estimated 10,000 patients per year seek medical attention because of exposure to this chemical.[3]

Many of the effects of what we now recognize as carbon monoxide poisoning were discussed in ancient literature. During the Middle Ages, these effects were sometimes considered to be the work of demons and witches. Some of the worst workplace exposures occurred during the 19th and early 20th centuries, when industrial miners were frequently overcome by "white damp," a term applied to the "inodorous nature" of carbon monoxide and associated products of partial combustion.[2] Miners were often exposed, with frequent severe or fatal sequelae, after mine fires, explosions, and blasting operations.

Serious exposures have been reported in much less dramatic contemporary situations when individuals have used contaminated sources of compressed air for diving or positive-pressure respirators for industrial

Fig. 11-1. These workers are testing new 75-mm howitzer tubes for the U.S. Army at the General Electric Plant, Erie, Pennsylvania, in June 1941. Such indoor testing, without adequate ventilation, quite likely exposed these workers to hazardous levels of carbon monoxide. Under today's standards, a powerful, frequently monitored indoor ventilation system would be required. Photograph: Courtesy of the US Army.

operations.[1] Exposures in the home or workplace may occur in poorly ventilated areas when using improperly adjusted or inadequately vented heaters, kitchen stoves, water heaters, or charcoal braziers.

Army field exposures commonly occur in tanks, troop compartments of armored vehicles, enclosed communications vans, enclosed areas where portable, gasoline-powered generators are operating, and indoor firing ranges. Military industrial exposures are possible during peace and war and can occur during maintenance and repair of internal combustion engines, indoor operations with motorized equipment such as fork lifts, and indoor proof-testing or firing of weapons (Figure 11-1).[1]

TYPICAL SOURCES OF EXPOSURE

Natural sources of carbon monoxide include volcanic eruptions, lightning strikes, and beds of growing kelp. But atmospheric carbon monoxide primarily results from the incomplete combustion of carbonaceous materials. In nonindustrial, urban environments, the major man-made source of carbon monoxide is the incomplete combustion of motor-vehicle fuels, with exhaust gases from internal combustion engines accounting for approximately 60% of emissions.[4] Because fuels manufactured during the early 20th century (eg, water gas, coal gas, and producer gas) usually contained high concentrations of carbon monoxide, they caused innumerable instances of poisoning. In contrast, natural gas—obtained from wells in coal-bearing areas—contained only trace amounts of carbon monoxide prior to incomplete combustion.[1,2]

Because carbon monoxide is a combustion by-product, toxic exposures are especially likely in conflagrations of buildings, kilns, and grates; explosions or fires in mines; the detonation of explosives; and even tobacco smoke.[1,5] Fires associated with timbering in mines, reinforcement of deep dugouts, and interiors of burning buildings are recognized as sources of significant exposures to carbon monoxide.[6] During the early years of the 20th century, toxic exposures to people who attempted to remain warm using open coke braziers were not uncommon, especially among occupants of dugouts or other small, ill-ventilated structures. The most common cause of death in fires is smoke inhalation and therefore carbon monoxide is a major cause of death in individuals who succumb to smoke inhalation.[4]

Effects of toxic gas exposures associated with the workplace were described by Tanguerel des Planches in 1839 and Dr. Hermann Eulenberg in 1865. Claude Bernard first discovered the complex identified as carboxyhemoglobin in 1858.[7] In the 1880s, Professor K. B. Lehmann and his students expanded the scientific database related to toxic gas exposures based on animal experimentation, industrial observations, and human control experiments. One of the first instances of the involvement of academic medicine in occupational health occurred during 1906, when the Medical Department of the University of Pennsylvania emphasized the importance of carbon monoxide exposures in the felt hat industry.[8]

Early 20th-century scientific and medical literature documents not only the magnitude of the interest, frequency, and types of poisoning potentials, but also the medical importance of carbon monoxide poisonings that occurred in both domestic and industrial environments.[2] Before equipment to quantify carbon monoxide was readily available, it was considered to be a prudent safety measure to regard all flames from carbonaceous sources that impinged against metal as potential carbon monoxide exposure hazards: the metal would cool the fire and the resultant incomplete combustion would produce carbon monoxide.[2] Several excellent historical reviews of carbon monoxide exposure, such as J.S. Haldane's, which was based on his 1915 Silliman Lectures at Yale University, contributed to the developing database.[2,7,9]

Steel Manufacturing

Blast-furnace gas was a product of the steel industry. The gas was produced during the smelting of iron ore, as a result of partial combustion when coke and limestone mixes were injected into the blast furnace. In a collection process from the upper stack, particulate and gaseous contaminants were removed using filtration and water scrubbing. The resultant blast-furnace gas was carried by overhead pipes to be reintroduced into the furnace as a combustible source of heat for blast stoves, steam production, and gas-engine power. Although the composition of blast-furnace gas was variable, major components were, on average, nitrogen (57%), carbon monoxide (26%), carbon dioxide (11%), and hydrogen (3%–4%).[2,10] Because the steel industry in the United States, with its numerous facilities and industrial employees, was a prime source of carbon monoxide poisoning, the industry provided the impetus for developing effective engineering and administrative controls.

Combustion Engine Exhaust

Shortly after gasoline engines were introduced as a source of motive power, operators of gasoline launches and drivers in taxicab garages frequently experienced signs and symptoms of carbon monoxide exposure.[2] Production of carbon monoxide was demonstrated to be greater before the engine had warmed to normal operating temperatures. Other conditions that generated increased amounts of the gas included engines that were poorly tuned or operated at idling speed without a load. Exhaust from the gas engine contained 9.3% carbon monoxide, and an average concentration of carbon monoxide of 0.042% was measured in ambient air in five motor garages.[10] Current United States emissions standards are met with automobile exhausts that contain as much as 8% carbon monoxide,[11] although current automobile exhaust technology usually results in substantially lower emissions.

Methylene Chloride

The industrial use of methylene chloride is an unusual example of an industrial carbon monoxide hazard because there is no actual exposure to carbon monoxide itself. Workplace exposures to methylene chloride have been followed by increases in carboxyhemoglobin levels as a result of humans' metabolism of the methylene chloride to carbon monoxide.[12] Methylene chloride is a readily volatile chemical that is widely used in industry as a paint stripper, aerosol propellant, and degreaser. As a consequence of the volatility and lipid solubility, with uncontrolled use of methylene chloride, exposure potentials are high and the material is readily absorbed. Ineffective industrial ventilation of operations such as degreaser tanks or improper use of personal protective equipment may cause carboxyhemoglobin levels to rise.[2]

Smoking

Inhaling tobacco smoke (whether as an active or passive smoker) is another source of workplace exposure to carbon monoxide. The presence of carbon monoxide in tobacco smoke and the associated elevation of carboxyhemoglobin levels were recognized and reported in the 1920s.[2] Carboxyhemoglobin levels above 3% were seen in 85% of smoking workers and 47% of nonsmoking workers in New York.[4] Most smokers were found to have carboxyhemoglobin levels higher than 2% compared to 1% or less for nonsmokers. Carboxyhemoglobin levels were found to increase by 1% to 9% with smoking. Heavy smokers were reported to have levels of 15% to 17%. Taxi drivers who smoke were reported to have carboxyhemoglobin levels as high as 13% (the mean concentration was 6.9%).

MILITARY EXPOSURES

Although exposures to carbon monoxide have been recognized as long as work has been done in association with partially burned carbonaceous fuels, military personnel may also experience significant exposures in ways quite different from those in the civilian sector. Some documented historical circumstances and weapon systems have resulted in substantial exposures and sometimes the deaths of exposed service members.

The use of mining in military operations dates from remote antiquity. Inadequate ventilation was recognized as an early cause of the difficulty of maintaining burning torches, especially when the galleries (mine tunnels) were long. It was widely reported that men were frequently overcome by what we now recognize as both oxygen deficiency and carbon monoxide accumulation within the mines. The effectiveness of asphyxiating gases in mine warfare was well known, with the earliest recorded use at the siege of Ambracia in 189 BC. The Aetolians filled jars with feathers, which were then set on fire; the smoke was blown into the faces of the oncoming Roman soldiers.[6]

World War I: Combat Mining Operations

Only mining operations that were used in a defensive tactical posture were employed during the early part of World War I. Mining operations were simple, with a single mine gallery constructed to guard important trenches or sectors of the line. By the fall of 1915 and increasingly later, mining was used in a more offensive posture. Offensive operations required more extensive tunnels, tunnel galleries, and larger quantities of high-explosive munitions. And as mining operations increased in frequency and intensity, more soldiers became poisoned by the gases associated with the detonations. Although carbon dioxide, hydrogen, methane, and oxides of nitrogen were also generated by the detonation, carbon monoxide was by far the most important poisonous gas associated with military mining (Figure 11-2).[6,13]

Mining was also used to promote forward movement of the troops at the front lines. An explosive in a forward-directed mine gallery would be detonated, which would create a crater 60 to 90 ft in diameter.

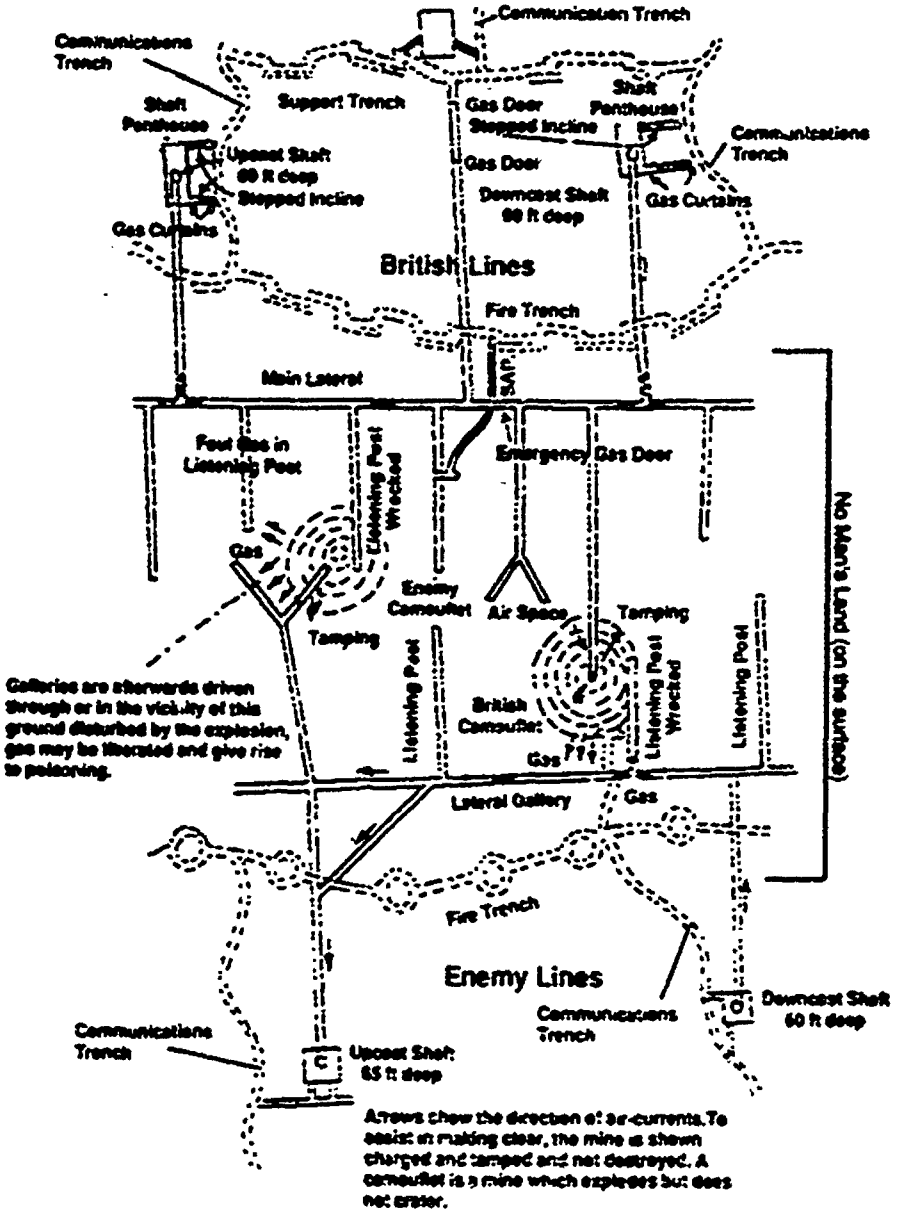

Fig. 11-2. Extensive military mine and trench operations were used during World War I. The above-ground area on the drawing between the British and the enemy lines is No Man's Land, but active warfare was being waged below. Offensive mining involved developing an extensive underground network of downshafts, tunnels directed toward the front line, main laterals (perpendicular to and crossing the forward-directed tunnels), and numerous forward-directed mine *galleries* (shafts). The galleries were used to listen for enemy operations, as conduits for additional mining operations, and as sites for placing offensive or defensive munitions. Tremendous quantities of tightly packed explosives were carefully placed in a procedure called *tamping*. This diagram shows the locations of two explosions, each of which wrecked a listening post. After the explosions, shafts might have been driven through the disturbed ground, potentially liberating carbon monoxide. The arrows show the direction of air currents. Reprinted from Macpherson W G, Herringham WP, Elliot TR, Balfour A. *History of the Great War Based on Official Documents: Medical Services Diseases of the War*. Vol 2. London: His Majesty's Stationery Office; 1923.

Within a few minutes, the infantry would move forward, occupy the crater, establish bombing posts, and consolidate their position. Although the loosened soil usually permitted rapid dissipation of the carbon monoxide, soldiers were frequently overcome as a result of gas entrapment, incomplete detonation with its subsequent gas collection, and absence of wind or "dull, heavy atmosphere."[6] Burning gas, which followed an incomplete detonation, could often be seen as a blue flame that could persist for 12 hours.[6]

Compressor engines were used to ventilate the mines and mine galleries. Carbon monoxide poisoning was often associated with the interruption of the engine power (in order to listen), the use of substitute lubricants containing mineral oil (such as castor oil) for the cylinder of the engine, and unexpected breakdowns from belt trouble. Small petrol engines, usually installed in dugouts, were often used as sources of energy for electric lights and power requirements within the mines. Carbon monoxide poisonings associated with petrol engines in mines were common, especially when the engines were new and care was not taken to ensure that the exhaust was discharged into the open air.[6]

Because carbon monoxide poisoning was a well recognized hazard, soldiers working in the mines often used mice or canaries as biological detectors. (Mice were sometimes preferred because they could be trained to be carried in a soldier's pocket.) However, the use of canaries was occasionally counterproductive, as the following anecdote illustrates:

> One of the canaries kept in a mine rescue station escaped from its cage and flew into the middle of "No Man's Land," where, alighting on a bush, it began to sing. Consternation was caused in the British lines, for the discovery of this bird by the enemy would indicate the presence of mining operations and would mean that the work of weeks would go for naught. The infantry in the trenches were immediately ordered to open fire on the canary, but it continued singing, heedless of the bullets which whistled round it. It was not until the trench mortars were called on to assist that a well-placed shell wiped out completely the bird, the bush, and the song.[6]

Although some medical historians of World War I identified carbon monoxide (along with dichloroethylsulphide, sulfuretted hydrogen, and nitrous fumes) as possible sources of poisonous substances of military significance, a medical treatise published in France in 1918 emphasized the risk of carbon monoxide.[13] This treatise, entitled (translated from the French) *The Clinical and Therapeutic Aspects of Gas Poisoning*, was directed toward diagnosing gas-intoxicated soldiers and providing appropriate therapy. Data for the treatise were gleaned from human exposures and laboratory animal experimentation with an extensive series of toxic gases.

The French writers identified several hazards associated with exposures in enclosed spaces:

- gun fire from closed shelters with inadequate ventilation,
- mine or camouflet [a mine that explodes but does not crater] explosions with subsequent contamination of communicating galleries,
- tunnel-construction operations in contaminated areas, and
- explosions of incoming enemy projectiles that secondarily contaminated dugouts, shelters, or produced entrapped pockets of the gas.[13]

The dangers of carbon monoxide exposures were enhanced by the absence of odor, color, and irritation. The gas was so insidious that an exposed individual could fail to grasp the exposure danger until he noticed that the use of his extremities was impaired. However, despite its advantages and possibly because of its limitations, carbon monoxide itself was never used as an offensive chemical warfare agent.[6]

World War II: Tank Warfare

The World War II experience with carbon monoxide was dominated by problems associated with large numbers of soldiers fighting in armored fighting vehicles (AFVs). The need for an accurate methodology applicable to quantitative detection of carbon monoxide led scientists at the Armored Medical Research Laboratory at Fort Knox, Kentucky, to develop an infrared gas analyzer that was not only reliable but was sufficiently transportable to be used for measurement within AFVs. Peak levels of carbon monoxide were detected shortly after firing bursts in the M3A4 tank.[14] After firing five rounds from the 75-mm gun, the level of carbon monoxide was found to increase rapidly to 0.718% within 1 minute. If no additional rounds were fired, ambient carbon monoxide decreased rapidly to baseline levels within 4 minutes. Carbon monoxide levels recorded with 37-mm firing were substantially lower than those found with the 75-mm gun (Figure 11-3).

The same problem had, of course, been recognized during World War I. In the early days of tank warfare, crews who spent prolonged times inside their tanks complained of headache and faintness. These problems became more severe as later tank models were introduced. Signs and symptoms of exposure were

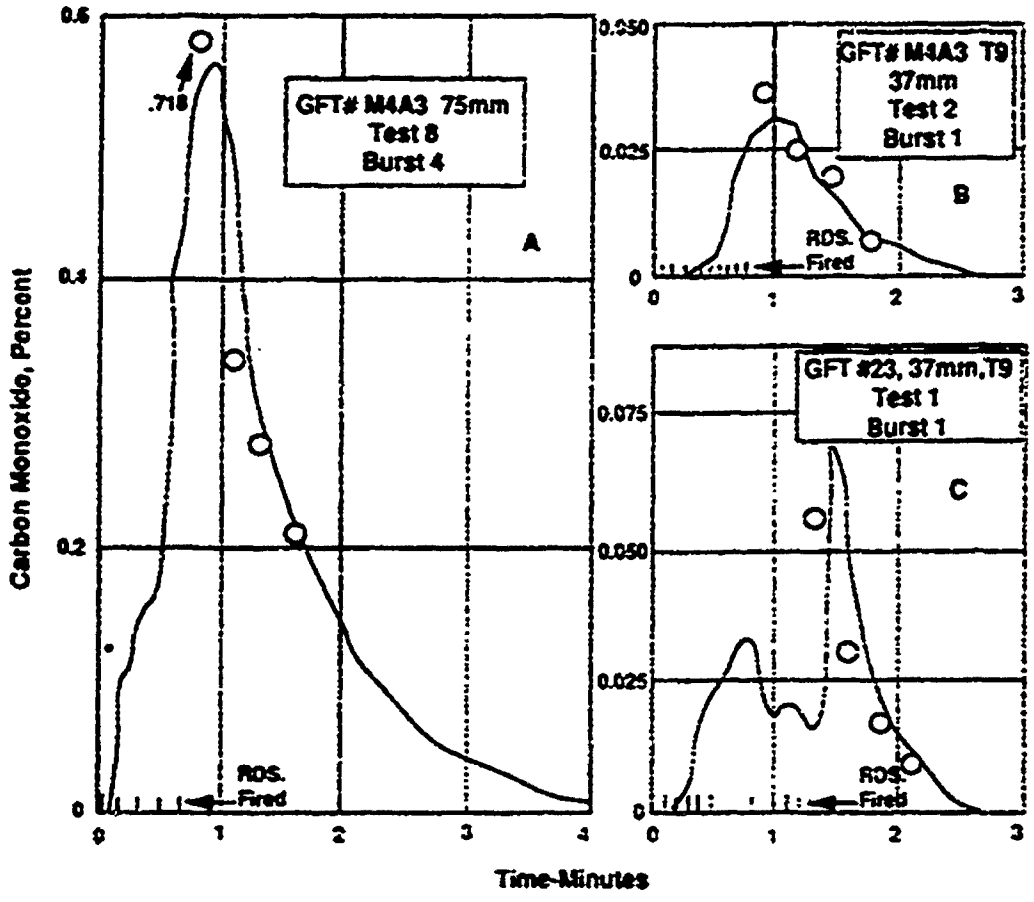

Fig. 11-3. Carbon monoxide concentrations are measured by means of a continuous recorder (——) and grab samples (o) after firing five rounds from the 75-mm gun, A, or ten rounds from the 37-mm gun, B, of the M3A4 tank. (Note that these graphs, which were originally published in 1945, contain an apparent misprint: the tank is identified as an M4A3. Because the data refer to both 37-mm and 75-mm guns, however, we know that the correct designation would be M3A4. The GFT seen in the test-identification boxes may have stood for *gas firing test.*) In both these graphs, the recorder did not show the true peak as measured by the grab samples. This is due to the relatively slow response time inherent in real-time monitors, which results in an "averaging" effect, and is to be expected. Source: Adapted from Nelson N. *NDRC Infra-Red Gas Analyzer for Carbon Monoxide.* Fort Knox, Ky: Armored Medical Research Laboratory; 1945. Provided by Alexandria, Va: Defense Technical Information Center. Report AD 655578.

clearly related to exposure to the toxic exhaust gases and ambient heat burden.[15] Although the soldiers sometimes became unconscious while in the tanks, they more frequently collapsed after reaching fresh air.[6] Their symptoms were aggravated when they fired the Hotchkiss[8,16] and 6-pounder guns.[6]

The interior design of the early tanks permitted heat accumulation and exposure to toxic combustion gases because the exhaust lines ran along the inside of the tank for some distance before they perforated the plating to the outside. As a consequence, carbon monoxide exposures from both internal leakage and backdraft from other tanks were common. The restricted crew space within the tank allowed significant accumulation of moisture, which further complicated the heat-exposure potential. In one incident, reported in 1918, the first and second tank drivers became unconscious. When the tank commander took control as the driver, he also succumbed and the tank was unable to get into action because of the condition of the crew. Clearly, improved tank ventilation was required. A supply of fresh, outside air was provided via infiltration around gun ports and other openings.[6]

Carbon monoxide exposures were measured in tanks towed behind each of two medium tanks (M4A1 and M3A4) and two recovery vehicles (M32B1 and M32B3).[17] Operational test variables included two courses (a flat surface and a 4% grade) and the use of

either a towing bar or towing cable. Carbon monoxide levels were measured in four crew positions within the towed vehicle: driver, assistant driver, loader, and commander.

Hazardous levels of carbon monoxide were not found in the towed vehicle when it was towed behind the M3A4 medium tank or the M32B3 recovery vehicle. In sharp contrast, however, the earlier M4A1 model tank and M32B1 model towing vehicle were designed with their engine exhausts directed rearward.[17] When either of these earlier models was the towing vehicle, riders in the towed tank were exposed to levels of carbon monoxide in excess of 0.2%. Exposures were affected by changes in wind direction, surface grade, monitoring position, and whether the towing cable or towing bar was used.

For the M4A1 tank and the M32B1 recovery vehicle, an exhaust deflector shield was developed and employed as a short-term remediation.[17] The deflector baffle design permitted retrofitting at the first-echelon maintenance unit. When it was attached to the towing M4A1 tank, the deflector directed the exhaust toward the ground and effectively limited the exposure of the soldiers who were required to ride in the towed vehicle (Figure 11-4).

The Modern Era: Armored Fighting Vehicles

The risks associated with carbon monoxide exposure and military equipment have been recognized for a long time. Why then does this remain an important, unresolved issue? During World War II, only about 3% of soldiers in the U.S. Army were deployed in AFVs. However, if a war were fought today the U.S. Army would deploy at least 30% of its soldiers in AFVs.[18] Not only are more soldiers likely to be exposed but the levels of exposure may also be significantly higher if control measures should fail. For example, as ammunition has become increasingly larger, the amount of propellant charge required to fire the larger round is even greater (round size is measured as a squared variable, charge volume as a cubic variable). Even the development and use of more effective ventilation systems and cleaner burning propellants have not completely eliminated the risk associated with carbon monoxide in today's AFVs.

M1E1 Tank

In 1984, an unusual exposure to carbon monoxide was reported during the operational test (OT II) of the M1E1 tank. The official memorandum filed after the event said, in part:

> At 1330 on 20 February, tank #120-5 began a silent watch and firing exercise as part of the [OT] II program. The exercise was run in accordance with conditions specified in the current Detailed Test Plan which called for it to be run with hatches closed, the primary NBC [nuclear, biological, and chemical] system off, and the backup system (M13A1) on. The silent watch portion of the exercise was run with the engine off and the breech open (no round chambered). The engine was started periodically to recharge the batteries, but the NBC system (which starts automatically when the engine is started) was selected "off" immediately upon actuation. The crew was dressed in MOPP IV [mission-oriented protective posture]. Breathing air was supplied to the protective masks through the M13A1 gas particulate filter unit. The driver stated that he used his mask only for the last 90 minutes of the exercise. The tank operated in silent watch mode until 2015, when it moved to the firing range. The firing portion of the exercise included firing thirteen main gun and approximately 100 coaxial machine gun rounds.
>
> Six main gun rounds and the coax rounds were fired between 2015 and 2040. The remaining seven main gun rounds were fired in the next seven minutes. At the end of the exercise, the loader slumped forward in his seat and appeared to be in physical distress. A short time later, the tank commander observed this condition, aroused the gunner, and assisted him out of the turret. After performing this action, the tank commander also experienced dizziness and [lay] down on the tank. The medics arrived at 2051 and the loader and tank commander were taken to —— Army Community Hospital, arriving a[t] 2121 hours. The hospital records show admission at 2145. . . .[19]

During the medical evaluation of the tank's crew members after this exposure, and shortly after their admission, carboxyhemoglobin levels were obtained. The loader had a level of 33% carboxyhemoglobin and the tank commander had a level of 27.8%. As a consequence of the two admissions and the results of the blood analyses, the remaining crew members, the gunner and driver, were brought and admitted to the hospital several hours later. Their admission carboxyhemoglobin levels were 16.5% (the gunner) and 12.7% (the driver). After treatment, all crew members were discharged without apparent sequelae of exposure.[19]

In March 1984, recommendations were offered for improving the M1E1 tank:

- Reroute the air intake for the M13A1 gas particulate filter unit from the turret area to the outside air (for intake)

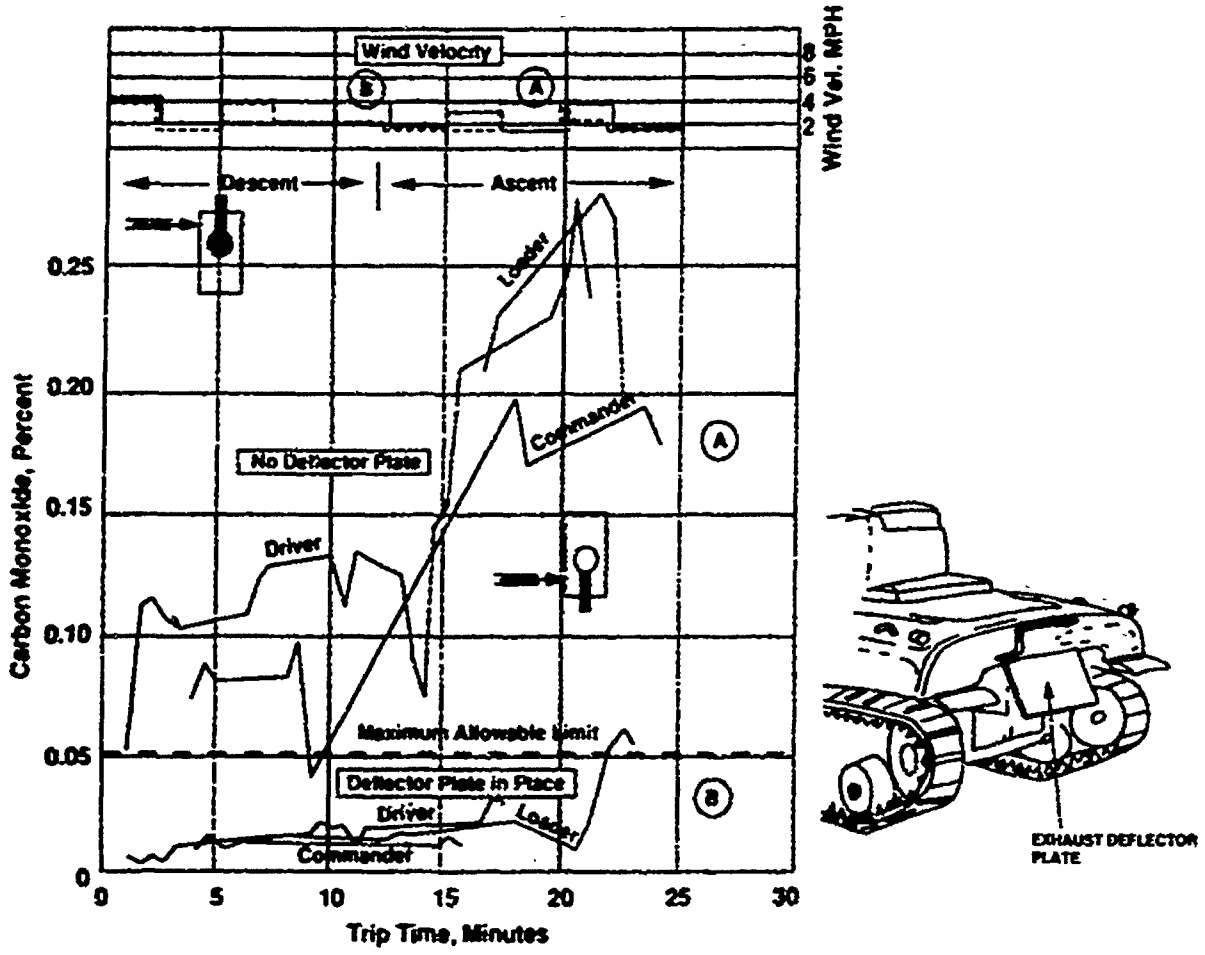

Fig. 11-4. Carbon monoxide was measured at three crew locations when a tank was towed by an M32B1 recovery vehicle with and without an exhaust deflector, and using a towing bar on a 4% grade. Placing an exhaust deflector (small drawing), as a product improvement on the towing vehicle, reduced the concentration of carbon monoxide within the tank. This graph conveys some of the complexity of making field determinations of carbon monoxide exposures. Many variables, if not controlled, must at least be accounted for (eg, wind direction and speed, engine speed, degree of surface grade, duration of the experiment, the location of the monitoring instruments within the tank, the reliability of the instruments). The increased concentration of carbon monoxide seen during observation A may be due to a combination of engine speed, the 4% grade, and the elapsed time. The missing tracing at the loader position in observation A may be due to equipment malfunction. What this graph clearly demonstrates, however, is that attaching an exhaust deflector plate to the rear of the towing vehicle—a seemingly minor modification made for observation B—significantly lowers the concentration of carbon monoxide at the crew positions. Source: adapted from Nelson N. *NDRC Infra-Red Gas Analyzer for Carbon Monoxide*. Fort Knox, Ky: Armored Medical Research Laboratory; 1945. Provided by Alexandria, Va: Defense Technical Information Center Report AD 655578.

- Advise individuals at all test sites that a hazard is associated with firing the main gun and/or coaxial machine gun with the hatches closed and NBC system off.
- Instruct test personnel at all test sites that the M13A1 gas particulate filter provides no carbon monoxide protection as configured.
- Revise the operator manuals to (*a*) include warnings of the carbon monoxide hazard associated with weapons fire and the inability of the M13A1 system to protect against carbon monoxide exposure and (*b*) describe the types of ventilation required for silent watch."

Infantry Fighting Vehicle

Toxic fumes testing was conducted at the U.S. Army Environmental Hygiene Agency (USAEHA) in the

infantry fighting vehicle (IFV) in 1980.[20] Real-time monitoring was done inside the IFV during a worst-case firing scenario to determine peak and total carbon monoxide exposure concentrations. The real-time samples were collected using direct reading instruments connected to appropriate recorders. Area samples were obtained from "the approximate breathing zones" of the crew compartment, driver's compartment, and the crew members. In one event, 200 rounds of 25-mm and 75 rounds of 7.62-mm ammunition were fired over approximately 6 minutes. In the turret, a peak exposure concentration of 1,920 ppm, average exposure concentration of 825 ppm, and total exposure concentration of 4,950 ppm-minutes were measured. (The term "ppm-minutes" used with the total exposure concentration is based on the concept that the cumulative personnel exposure dosage is a product of the average measured airborne concentration and total exposure time.)

It was decided that the initial exposure measurement period for the IFV firing event "should have been fired over a 20-minute period rather than the 6-minute firing."[20] Carbon monoxide measurements were obtained when the firing scenario was repeated over both 20- and 60-minute periods of weapons fire. Several peak exposures in the turret were above 600 ppm, with an average exposure concentration of 190 ppm and a total exposure concentration of approximately 11,400 ppm-minutes. The firing-exposure scenario was repeated; this time, peaks that were measured in the turret exceeded 800 ppm and total exposure concentrations measured approximately 24,730 ppm-minutes. In the driver's compartment, peaks in excess of 400 ppm and total exposure concentrations of about 10,600 were measured. In the crew compartment, peak exposures above 400 ppm and total exposure concentrations of approximately 8,200 ppm-minutes were measured.

In summary, total concentration exposures in the IFV were found to exceed the acceptable limit of 6,000 ppm-minutes established in Military Standard 800.[21] With respect to the measured peak levels, some peak exposures were above 800 ppm, which exceeds excursion values "considered acceptable by any standards-setting body."[20] For the IFV, under the conditions of the firing scenarios, the study concluded that exposures could result in carboxyhemoglobin levels of about 15% in 15 minutes. The potential exposures were considered to represent a health risk that could cause significant symptoms and signs consistent with impairment of combat effectiveness.[20]

In a study of the Bradley Fighting Vehicle (BFV) performed in 1984, carbon monoxide measurements were obtained using a dual channel, nondispersive, infrared, carbon monoxide analyzer for real-time carbon monoxide measurements.[22] Even though the detection methodology represented acceptable industrial hygiene practice, it was noted that the equipment was incapable of detecting rapid transient concentrations of carbon monoxide. Conditions in the BFV that were associated with firing and shown to affect carbon monoxide concentrations included the type of weapon fired, the position of the hatches (open or closed), the crew position, and the position of the turret with respect to the hull.

Firing conditions were limited by selecting wind-speed conditions. Closed-hatch firing was not permitted at wind speeds greater than 10 mph, and open-hatch firing was not permitted at wind speeds greater than 5 mph. Hull fans were turned off, gun bags were zippered, gas particulate filter units (GPFUs) were turned on, and both 7.62-mm and 25-mm rounds were fired.[22]

The results and conclusions of the 1984 study differed from those in a 1982 medical report, which indicated that no medical hazard was identified with firing the weapons in the infantry fighting vehicle.[23] With the single exception of one measurement (4.8% carboxyhemoglobin) in the 1984 study, all firing conditions were expected to generate carboxyhemoglobin levels above 5%, and in three of the conditions, carboxyhemoglobin levels ranged between 11.0% and 13.4%. The maximum peak concentration, 1,462 ppm, was measured at the driver position; levels of 1,087 and 1,200 ppm were identified in the troop compartment. Operation of the M13 GPFU, which has no capacity to remove carbon monoxide, resulted in uptake of carbon monoxide within the driver's compartment with subsequent distribution to each crew member and troop occupant.[22]

M109 Howitzer

The health hazard assessment on the M109 Howitzer Improvement Program (HIP) identified carbon monoxide exposure as an area of medical concern in the development of the howitzer. The HIP, designated as the M109A3E2, is an armored, full-tracked howitzer carrying a minimum of 34 complete, conventional-geometry rounds and two oversized projectiles on board.[24] The main armament is a modified, 155-mm, M185 cannon assembly (the M284), and M178 gun mount. The modified muzzle break deflects propellant gases back along the gun tube, rather than perpendicularly as the unmodified predecessor models did. The M109 howitzer is generally operated by a crew of four and is operationally supported by ammunition resupply vehicles.

Numerous environmental, sampling, and configuration variables influenced the concentrations of propellant combustion testing with the M109 howitzer evaluations.[24] Tube-firing elevation, wind speed, wind direction, hatch configuration, ventilator mode, propellant type, propellant quantity, system component failure, fire rate, and industrial hygiene sampling practices all appeared to influence the study results. The bore evacuator is a pressure-responsive, tube-evacuation system that is designed to promote the movement of postfire combustion gases from the breech toward the muzzle. Both compromised bore evacuator function and wind direction (ie, a head wind blows combustion gases out the breach despite a functional bore evacuator) are critical variables associated with exposure concentrations after firing.

Although the authors of the health hazard assessment, which was performed at USAEHA in 1988, conclude that the lack of data replication limits the general applicability of the findings, they consider several observations to be reliable. In general, they state that the worst-case firing scenario occurred when the vehicle's hatches were closed. In that operational mode, the crew compartment was maintained under a slight negative pressure, which drew combustion gases from the breech into the crew compartment when the cannon breech was opened. They concluded that a head wind significantly increased the exposure to combustion gases. Finally, they concluded that reconfiguring the muzzle break could actually increase the crew's exposure to the combustion gases.[24]

Projected exposure data for the M109 were developed for carbon monoxide based on the Operational Mode Summary/Mission Profile (OMS/MP). Carbon monoxide data have been used in a concurrent calculation of the maximum allowable consecutive episodes (MACE) to limit the M109-associated carboxyhemoglobin to 10%. The OMS/MP stipulates 5 rounds per mission, 51 missions per day, and a total of 254 rounds per day. Average carbon monoxide exposure levels during the HIP firings were identified in a broad range, from 0 to 2,300 ppm at the crew positions. The health hazard assessment team at USAEHA arbitrarily chose an average level of 120 ppm and calculated the 24-hour exposure risk of the development of a 2.09% carboxyhemoglobin level. Their estimate that a 5.3-minute exposure may be repeated 17 times (ie, the MACE) without exceeding a 10% carboxyhemoglobin level remains unconfirmed, but has been recommended for M109 training and testing missions. Similarly, their position that the risk of firing-associated health impairment is negligible is speculative and remains unconfirmed. As a consequence, medical monitoring for carbon monoxide exposure effects is currently required during operational testing of the M109 howitzer developmental series.[24]

Military Aviation

Unacceptable carbon monoxide exposures have been demonstrated in military aviation, where uncontrolled exposures in early piston-driven aircraft were responsible for the deaths of many pilots. One early investigator reported on a death that occurred in 1930:

> The source of carbon monoxide poisoning which forms the basis of this article is the exhaust gas of the gasoline motor of the airplane and airship and was brought to attention by the death of Capt. Arthur H. Page, United States Marine Corps, at the national air races, Chicago, September 1, 1930, and the subsequent report of the finding of carbon monoxide in his blood shortly after the crash.[5]

In a 1944 report, an aircraft pilot experienced carbon monoxide exposure as a result of a defective engine exhaust; his behavioral responses were recorded by his fellow aviators. They reported that the pilot's behavior appeared to be related to the simultaneous interaction of carbon monoxide and altitude (in other words, the decreased partial pressure of oxygen). At low altitude, the pilot's flight responses were normal. However, after ascending to 10,000 feet with his aviation section, he made only the first flight entry correctly. His subsequent flight patterns and behavioral responsiveness progressively deteriorated. As a result of the actions of another aircraft pilot, the availability of in-flight oxygen, and the decrease in altitude, the pilot improved enough to properly land his craft. The pilot recalled that he was aware that there were difficulties with his flight (his hand shook violently and he had difficulty grasping the throttle). He also remembered that he could not always see his section leader, the horizon, or the clouds, but had a feeling of "What's the difference?" Unfortunately, the pilot's carboxyhemoglobin levels were not determined.[25]

Although carbon monoxide exposures have been eliminated from the exhaust system of aircraft, high, transient exposures to the toxic fumes have been associated with gunfire when weapons are used aboard aircraft. In 1988, an evaluation was performed of the carbon monoxide emissions from the M134 minigun, mounted in the UH-60A Black Hawk helicopter. In the tests, a range of 1,400 to 2,700 rounds were fired in five replicates of five separate firing conditions. The highest average ambient carbon monoxide–exposure concentration was 79.4 ppm for a duration of 4 minutes. The calculated maximum predicted carboxyhemoglobin response was 4.90%, and no firing restrictions

were recommended.[26] Peak levels of carbon monoxide were not reported.

Household Heating

Exposure to carbon monoxide in the military is not limited to weapons systems: inadequate home ventilation is a common source of exposure. Large numbers of U.S. Army forces have been stationed in Germany, where coal has been used as a source of heat in family housing. Case reports of carbon monoxide poisonings involving family members living in military housing facilities have been reported.[27]

Charcoal block heating, the custom of heating homes with coal or charcoal fires inside or under the structures that is common in Korea, is a significant source of carbon monoxide during the winter months.[28] In one recent incident, a U.S. Army field commander and his family awakened in the middle of the night with severe headaches. Evaluation at the nearest medical facility confirmed carbon monoxide exposure and etiology.

PATHOPHYSIOLOGY OF EXPOSURE

The need to develop an effective treatment for carbon monoxide poisoning had a disproportionately large impact on medicine. Not only did it enable us to treat the poisoned patient, it also provided an insight into respiratory physiology. Carbon monoxide complexes with hemoglobin to form carboxyhemoglobin, which perverts hemoglobin's normal function: oxygen transport.

Carbon Monoxide's Hemoglobin-Binding Affinity

The complicated physiology of oxygen transport and carbon monoxide's deleterious effects on it are best understood when placed in their historical context. Controversy and difference of scientific opinion enveloped toxicity studies for carbon monoxide exposures during the early 20th century and were reflected in the early literature. Haldane believed that the effects of carbon monoxide exposure were caused by the preferential binding of carbon monoxide to hemoglobin, with resultant tissue hypoxia. Henderson, Karasek, and Apfelbach supported Haldane's view. In contrast, Poelchen had reported in 1888 that carbon monoxide toxicity resulted from a direct toxic effect on the tissues. The direct-effect theory was based on the rapid onset of narcosis, early muscular weakness in the lower limbs, occurrence of gangrene as a complication, and damage of the lenticular nucleus of the brain. Hill, Semerak, and Lanossier supported Poelchen's view. They noted that animal deaths occurred more rapidly, and were associated with different signs, when placed in an atmosphere containing carbon monoxide compared to simple asphyxiation with nitrogen.[2]

Studies that investigated carboxyhemoglobin stability and the recovery of hemoglobin's oxygen-binding capacities were performed in the early 1900s. Although some scientists reported finding carbon monoxide in blood 24 hours after the exposure to carbon monoxide had been terminated, one prominent researcher could identify no residual carbon monoxide 6 hours after exposure (using a spectrophotometric technique; the lower limit of sensitivity was 0.25%).[2] One researcher concluded, in 1920, that carboxyhemoglobin was actually metabolized in the liver, with metabolic components secreted into the bowel through the biliary tract.[29] The postulated method of animal and human excretion of carbon monoxide was thought to involve hepatic metabolism, with the carbon monoxide molecule probably remaining with the globulin end of the hemoglobin metabolite. This view held that the globulin moiety was presumed to be disposed of as a urea product and that carbon monoxide was "treated as foreign material and excreted."[29]

As a result of his interest in coal mining–related carbon monoxide poisonings, Haldane initiated studies in the mouse, which were first reported in 1905. He demonstrated that the mouse, when exposed to a partial pressure of 2 atm of oxygen and 1 atm of carbon monoxide, survived without difficulty. The mouse's only apparent limitation was a diminished exercise tolerance. Therefore, Haldane (*a*) discounted others' scientific claims concerning the potential inherent toxicity of carbon monoxide and (*b*) demonstrated that the adverse effect of carbon monoxide was related to its powerful, competitive, hemoglobin-binding affinity.[7]

Haldane and Douglas published their classic findings related to carboxyhemoglobin-dissociation curves in human blood in oxygen-deficient atmospheres in the early 1900s. They reported an apparent paradox: an individual with a normal hemoglobin level, of which 50% is carboxyhemoglobin, has more severe symptoms than does an individual with a 50% decrement in hemoglobin, none of which is carboxyhemoglobin. It appears that carbon monoxide has some effect on symptomatology that is unrelated to the amount of hemoglobin available for oxygen transport. Haldane and Douglas explained that the paradox arose

from the relative binding affinities of the differing oxygen-binding sites on the hemoglobin molecule. Hemoglobin molecules with one-half the available binding sites occupied by carbon monoxide released oxygen with great difficulty from the remaining two binding sites on each hemoglobin molecule.[7]

In Haldane and Douglas's early reports, comparisons of the relative oxygen- and carbon monoxide-binding affinities with the hemoglobin molecule had been performed using samples of their own blood. Interestingly, the relative carbon monoxide–oxygen binding affinity of Douglas's hemoglobin was reported as 246; Haldane's hemoglobin binding affinity was 299.[7]

In 1927, Haldane demonstrated that mechanisms in addition to the effective production of carboxyhemoglobin might account for the health effects from carbon monoxide exposure. Animals that he exposed to high concentrations of carbon monoxide under hyperbaric conditions developed a tissue toxicity despite adequate oxygenation. In these experiments, Haldane exposed animals to 3.1 atm of oxygen, then added an additional atmosphere of carbon monoxide. The animals' carboxyhemoglobin levels rose to 98%, but as long as oxygen was dissolved in the plasma, the rats suffered no ill effects. However, when a second atmosphere of carbon monoxide was added, the rats promptly died. This indicated to Haldane that carbon monoxide had a direct effect on tissues, probably at the cellular level. This explanation was supported in 1950 when a researcher demonstrated that microsomes in cells were inhibited by carbon monoxide, and that this inhibition involved enzymes associated with electron transport, specifically the cytochrome P-450 system.[12] The cytochrome P-450 system is now recognized as one of the most important metabolic biotransformation pathways in the human body. Carbon monoxide apparently affects oxidative and reductive reactions in these pathways.

Carboxyhemoglobin and Oxyhemoglobin Dissociation

The most commonly recognized primary action of carbon monoxide is its preferential binding with hemoglobin. This deprives hemoglobin of its normal oxygen-combining function and results in a condition that can be thought of as "anemic" hypoxia.[30] The chemical reaction is a reversible mass-action equilibrium and proceeds based on the mass or partial pressure (tension) of the gases in pulmonary air, an action that allows effective treatment.[9] In addition, carbon monoxide chemically binds to a number of heme-containing proteins including hemoglobin, myoglobin, cytochrome oxidase, cytochrome P-450, and hydroperoxidases. (However, these account for only 10%–15% of the extravascular carbon monoxide in a well individual.)[4]

The interaction between carbon monoxide and hemoglobin is graphically represented as the carboxyhemoglobin dissociation curve (Figure 11-5). At first glance the curve appears to be very similar in shape to the oxyhemoglobin dissociation curve (Figure 11-6). On closer inspection, however, it becomes apparent that the range used for the oxygen pressure scale ranges from 0 to 150 mm Hg while the range used for carbon monoxide is between 0 and 0.5 mm Hg. This difference represents the differential affinities of oxygen and carbon monoxide for hemoglobin; the difference is approximately 230-fold.[31] When oxyhemoglobin and carboxyhemoglobin dissociation curves are plotted along the same abscissa, the line representing saturation with carbon monoxide curves steeply upward to the left of the curve representing oxyhemoglobin. If the graph for oxyhemoglobin dissociation were to be superimposed on the graph of carboxyhemoglobin dissociation, complete saturation of the hemoglobin with carbon monoxide would occur prior to (to the left of) the steep upward slope of the oxyhemoglobin concentration. These differing curves demonstrate the difference between hemoglobin's oxygen- and carbon monoxide–binding affinities.

The National Institute for Occupational Safety and Health (NIOSH) defines the term *affinity constant* as "the number of moles of oxygen which must be present with each mole of carbon monoxide in order to maintain an equal saturation of hemoglobin."[32] Reported differences in the binding affinities appear to reflect individual researcher's preferences; for example, some accept 210, others 230, and others 250. When carbon monoxide binds with hemoglobin, the binding affinity of the carboxyhemoglobin complex for oxygen is increased, compared to that of normal hemoglobin. Not only does the hemoglobin combine preferentially with carbon monoxide rather than oxygen, the oxyhemoglobin dissociation curve is also affected so that oxygen is released less readily within the tissues (Figure 11-7).[5]

The physiological and clinical effects of carbon monoxide are primarily those of anoxemia (oxygen want) from a decrease in tissue oxygen (a combined effect of the reduced oxygen-carrying capacity and impaired oxyhemoglobin dissociation). As a result, the tissue hypoxia that is produced following carbon monoxide exposure is greater than the amount caused by an equivalent reduction of ambient oxygen (eg, altitude) or equivalent reduction in hemoglobin (eg, anemia).[4] After dissociating from carbon monoxide, the regenerated hemoglobin shows no impairment or

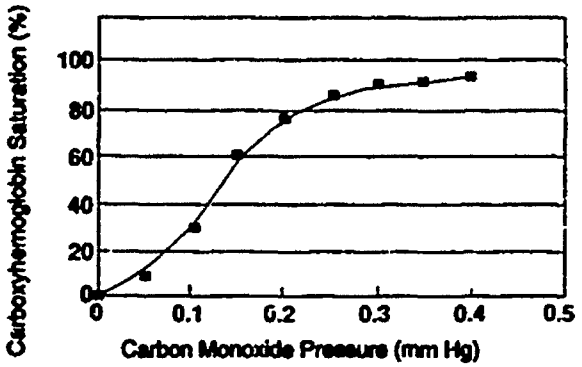

Fig. 11-5. The carboxyhemoglobin dissociation curve.

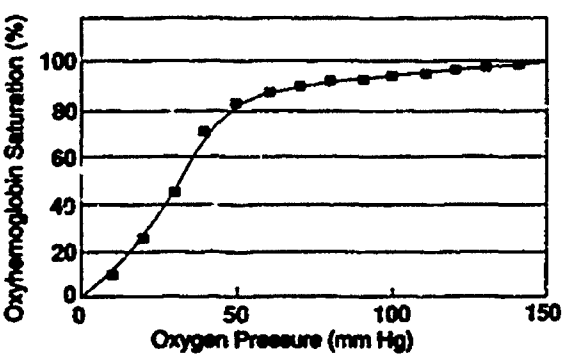

Fig. 11-6. In an oxyhemoglobin dissociation curve, decreased affinity of hemoglobin for oxygen shifts the dissociation curve to the right, whereas increased affinity for oxygen shifts the curve toward the left.

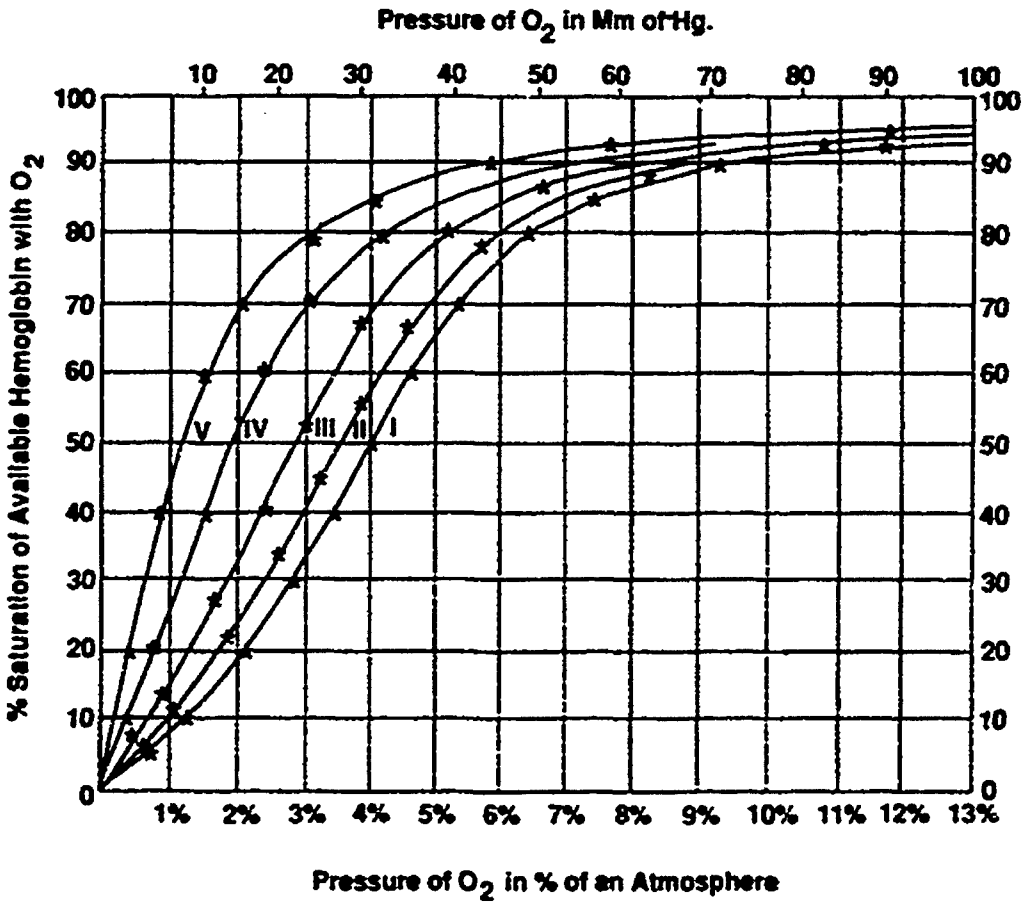

Fig. 11-7. The progressive left shift of the oxyhemoglobin dissociation curve is caused by increasing saturation of hemoglobin with carbon monoxide (I = 0%, II = 10%, III = 25%, IV = 50%, and V = 75%). This is Haldane's great contribution to our understanding of the pathophysiology of carbon monoxide exposure. Reprinted with permission from Haldane JS. *Respiration*. New Haven, Conn: Yale University Press; 1922. © 1922 Yale University Press.

residual decrement of oxygen-carrying capacity. The hemoglobin molecule resumes its normal configuration and function. The residual clinical effects following carbon monoxide poisoning appear to be secondary to tissue damage caused by oxygen deprivation at the level of the affected organ.[9]

However, because 10% to 15% of the body's total carbon monoxide is bound in the extravascular space,[33,34] it may be that some of carbon monoxide's toxicity is not directly related to defective oxygen transport. Cytochrome a_3 oxidase has been suggested as the (or perhaps *a*) major site of toxic action of carbon monoxide.[12,33] In addition to cytochrome oxidase, other extravascular proteins that bind with carbon monoxide include other heme proteins such as myoglobin, and cytochrome P-450. The interaction of carbon monoxide with the hyperperoxidases might cause oxygen radicals to be generated, which would result in cellular damage.[33]

More subtle modes of injury or dysfunction may also be possible. Humans normally produce small quantities of carbon monoxide through the action of the enzyme heme oxygenase. The fact that heme oxygenase is present in high concentration in several areas of the brain, together with carbon monoxide's known ability to activate intracellular enzymes that are important in the regulation of cellular function, suggests that carbon monoxide might serve as a neurotransmitter in the central nervous system (CNS).[35] It is easy to imagine that small amounts of exogenous carbon monoxide might cause undesirable effects that are not associated with carbon monoxide's effect on oxygen transport, not only in the CNS but perhaps elsewhere as well.

HEALTH EFFECTS

In experiments using himself as the study subject in 1895, Haldane reported that he noted the first apparent effects of carbon monoxide exposure at a concentration of approximately 20% carboxyhemoglobin.[7] At that level, he noted (in association with running up stairs) dizziness, palpitation, and hyperpnea. Haldane reported increased pulse rate and deeper breathing at 30% carboxyhemoglobin saturation; however, because he feared he would faint, he did not report on exertion at 40% saturation. His hearing, vision, and intellect seemed impaired at levels of 50%, with impaired motor function, diminished writing ability, and impaired perception. Associated signs included extreme exercise intolerance and collapse at 50% carboxyhemoglobin content.

Early reports indicated that death resulted at a level of approximately 80% carboxyhemoglobin. Most unconscious individuals did not immediately regain consciousness with treatment, and individuals who remained unconscious for periods longer than 24 hours after their removal from exposure were considered to have significant potential CNS or cardiac sequelae.[6]

Haldane initially postulated that a concentration of 0.05% carbon monoxide in ambient air would be required to produce an in vivo concentration of 30% carboxyhemoglobin in humans. The observations and conclusions derived from subsequent experiments resulted in the revision of the exposure level to 0.02% (200 ppm) of ambient carbon monoxide to produce 30% saturation.[7]

The relation between carbon monoxide's toxicity and its concentration in ambient air had been emphasized by the French authors of the 1918 medical treatise (discussed earlier in this chapter), who concluded that

- 1 part carbon monoxide per 10,000 parts ambient air may produce casualties;
- 2 parts per 10,000 may be fatal; and
- 3 parts per 10,000 is fatal within 20 minutes for a man at rest and much sooner during physical exertion.[13]

Symptomatology and collapse from exposures had been correlated with exposures early in the 20th century: a rough estimate of time to lethality for man was associated with breathing 0.2% carbon monoxide for 4 to 5 hours, or 0.4% for 1 hour. For air containing 2% to 5% carbon monoxide, such as is found in mine explosions, a few breaths were considered lethal, with death occurring as "quickly as in drowning" or "rapidly as if struck by lightning."[2] In cases of rapid collapse, no apprehension or other apparent prodromal warnings were reported. Poisonings associated with lower concentrations of carbon monoxide were reported to cause headache, tinnitus, epigastric distress, weakness, hallucinations, blurred vision, and convulsions.

Autopsy Findings

Early reports of death attributed to carbon monoxide poisoning indicated that anatomic autopsy findings were helpful in diagnosis of the cause of death. The skin color of the poisoned person differs from the skin color of persons dying of other causes.[9] The face may be bright red and rose-red spots may be present on the face, neck, breast, and limbs. The color of the

skin between the red areas is also likely to be abnormal and may be cyanotic. Blood color in poisoned individuals can range from bright red to very dark red to black. There is no change in blood coagulation. Ecchymoses, effusions, or hemorrhages occur with reddening of the digestive tract. There are no marked characteristic changes in the respiratory tract, although a thick, frothy mucus or digestive contents have been found in the upper respiratory passages. Hyperemia of the brain, with edema and blood-tinged intraventricular fluids are characteristic findings at autopsy. Local hemorrhagic lesions may range from microscopic to "the size of an apple."[9]

Other pathological abnormalities including spectroscopic evidence of carboxyhemoglobin, bronchopneumonia, fatty degeneration of blood vessels and heart, extensive tissue hemorrhage, necrosis of the lenticular nucleus, thrombosis, and encephalitis were commonly reported sequelae.[2]

Signs and Symptoms of Carbon Monoxide Poisoning

The progression of signs and symptoms of carbon monoxide poisoning and increasing levels of carboxyhemoglobin were described in 1923 and have been reproduced, with minimal modifications, in medical textbooks for more than half a century (Table 11-1).[9] Although more recent researchers have recognized that the carboxyhemoglobin level may not always correlate with the degree of clinical impairment associated with intoxication, the tabular information remains useful as a general guideline.[12]

The critical target organs of carbon monoxide exposure (*a*) are metabolically active and (*b*) require continuous supplies of oxygen-rich blood. The most critical organ systems appear to be the heart and the CNS.[4,11,32,36,37]

A carbon monoxide–exposed individual may appear to be grossly normal and the typical findings of carbon monoxide poisoning may not be identified during a routine exam. The findings are more apparent if the examination is directed toward abnormalities of function of the basal ganglia. Insults of basal ganglia function are characteristic of carbon monoxide intoxication and include tremor, slowed reaction time, impaired manual dexterity, impaired hand–eye coordination, and difficulty sequencing complex movements.[11]

With high-concentration exposures, carbon monoxide is readily absorbed and the carboxyhemoglobin content increases rapidly. In this circumstance, only transient weakness or dizziness may be noted before the individual becomes unconscious. When exposures are more prolonged and the carboxyhemoglobin content increases slowly, affected individuals may remain conscious but unable to escape because of weakness or impaired judgment.[1,2]

Based on lay accounts and clinical observations, the military circumstance in which exposure conditions

TABLE 11-1
SIGNS AND SYMPTOMS OF CARBON MONOXIDE POISONING

Carboxyhemoglobin Saturation (%)	Signs and Symptoms
0–10	None
0–20	Tightness across forehead, possibly slight headache, dilation of cutaneous blood vessels
20–30	Headache, throbbing in temples
30–40	Severe headache, weakness, dizziness, dimness of vision, nausea and vomiting, collapse
40–50	Same symptoms as at 30%–40%, but with greater possibility of collapse and syncope, and increased rates of respiration and pulse
50–60	Syncope, increased rates of respiration and pulse, coma with intermittent convulsions, Cheyne-Stokes's respiration
60–70	Coma with intermittent convulsions, depressed heart action and respiration, possibly leading to death
70–80	Weak pulse, slowed respiration, respiratory failure, and death

Source: Adapted from Sayers RR, Yant WP. Dangers of and treatment for carbon monoxide poisoning. *Reports of Investigations*. US Department of the Interior, Bureau of Mines; May 1923. RI 2476. Available from UPDATA Publications, Inc, Los Angeles, Calif.

were superimposed on combat conditions complicated the individual's awareness of the symptoms of carbon monoxide poisoning. With the moderate exposures experienced during World War I, the onset of symptoms of intoxication associated with uncomplicated anoxemia was often first noticed as a loss of power in the limbs. Giddiness, confusion, breathlessness, and palpitations were reported to follow increasing ambient carbon monoxide concentrations. Mental confusion caused individuals to appear drunk: shouting incoherently, laughing, swearing, or praying. The mental confusion appeared to suppress the desire or ability to seek escape despite apparent danger. As limiting factors in egress and avoidance, the mental impairments associated with carbon monoxide exposure appeared to be compounded by the loss of strength in the limbs. With progressive apathy and helplessness, many individuals passed gradually into coma and death.[6]

In contrast to the rapidly progressive signs of acute exposures to high ambient carbon monoxide concentrations, many cases of mild poisoning developed more slowly and were associated with the nonspecific clinical warning signs of headache and nausea, similar to those experienced with mountain sickness.[6] The degree of clinical response depends on the

- rate of absorption,
- final carboxyhemoglobin concentration in the blood,
- duration of hypoxia,
- preexisting health status, and
- concurrent whole-body oxygen requirement.[1]

The earliest and most constant ocular signs of carbon monoxide exposure are congestion of the retinal vessels and hyperemia of the optic disc. W. H. Wilmer noted in 1921 that findings of amblyopia and complete blindness were sequelae "not infrequently" observed.[10]

Staging of Signs and Symptoms

Since 1909, the signs and symptoms associated with exposures have been divided into two stages, depending on the responses of the nervous system.[9] Stage I, from normal to the onset of syncope, is often associated with neurological stimulation. Stage I signs and symptoms may include tightness across the forehead, cutaneous vasodilatation, frontal or basal headache, throbbing of the temporal regions, weakness, dizziness, nausea, vomiting, loss of strength or muscular control, increased pulse or respiration, and collapse. Signs and symptoms are more severe when exposure is associated with exercise:

> Men at rest have often been exposed to carbon monoxide all day without noticing any marked ill-effects, but on walking home or exercising have experienced severe symptoms, even to unconsciousness.[9]

In cases of high-dose exposures, the onset of syncope may be rapid and not associated with other signs or symptoms.[9]

Stage II, from syncope to coma and death, is characterized by nervous-system depression. These signs and symptoms include an increase in pulse and respiration, fall in blood pressure, loss of muscular control, loss of sphincter tone, loss of reflexes, convulsions, Cheyne-Stokes respiration, slowed pulse, decreased respiratory activity, apnea, and death.[9]

Exposure and Muscular Exertion

The character and degree of symptoms associated with carbon monoxide exposure depend on the duration and concentration of exposure and the amount of muscular activity. The symptoms and after-effects of exposure are more severe after prolonged than after short-term, high-level exposure. Muscular activity increases oxygen demand and accentuates exposure effects; resting individuals may experience no symptoms prior to the onset of unconsciousness.[9,38]

The relationship between carbon monoxide absorption and exercise is directly proportional to the amount of air breathed. At moderate exercise, a person breathes approximately twice as fast as at the resting rate; therefore, carbon monoxide absorption is approximately doubled. Heavy work increases the respiratory rate by approximately 3- to 4-fold, with a proportional increase in carbon monoxide absorption.[9] Firefighters are at extreme risk of carbon monoxide exposure while working at a heavy degree of exertion. For example, researchers have estimated that a firefighter has a 2.5% increase in carboxyhemoglobin after 2 minutes of exposure to a concentration of 1,000 ppm (0.1%). With heavy exercise, a 60% increase in carboxyhemoglobin content is predicted after a 2-minute exposure to 2% carbon monoxide and a 75% increase is predicted after a 1-minute exposure to 5% (50,000 ppm).[16] Other factors such as low oxygen pressures, high temperature, and high humidity also cause a relative increase in carbon monoxide absorption.[9]

Central Nervous System Effects

The plethora of signs and symptoms of carbon monoxide poisoning are characteristic of those seen with progressive hypoxia, but they also can mimic virtually any neurological or psychiatric illness. Signs

consistent with multiple sclerosis, parkinsonism, bipolar disorder, schizophrenia, and hysterical conversion reaction have been reported in association with acute carbon monoxide intoxication.[11] Numerous CNS sequelae following carbon monoxide poisoning have been reported. They may include headache, muscular pain, loss of strength, loss of memory, paralysis, temporary blindness, and mental derangement. In most cases, the sequelae clear within a few days, but may be seen for months or years following the acute event.[9] CNS sequelae have been reported to include choreiform movements and convulsions,[5,28] cortical blindness, peripheral neuropathy, and delayed neurological sequelae.[28] Neuropsychiatric sequelae may be more common than is generally appreciated, with findings of permanent sequelae such as personality deterioration or memory loss in 0.3% to 10% of patients.[39]

Acute neurobehavioral effects of carbon monoxide exposure such as compromised dark adaptation and impaired visual tracking have been postulated to impair performance in aircraft handling and target acquisition. Military medical concerns about the presumed impact of the neurobiological effects on performance resulted in the promulgation of carboxyhemoglobin levels for carbon monoxide exposure levels and equipment-design specifications.[40] Visual acuity appeared to be impaired by carboxyhemoglobin levels in the range of 3% to 5%.[36]

Scientific studies performed and published within the last several years have failed to replicate the findings of earlier neurobehavioral studies. For example, individuals who were exposed to carbon monoxide sufficient to produce carboxyhemoglobin levels of 16% to 23% failed to exhibit significant differences related to clinical symptoms, electroencephalographic recordings, and compensatory visual tracking from the control group.[41-44]

Circulatory Effects

The heart depends almost exclusively on aerobic metabolism and is a highly sensitive organ to the decrease of oxygen secondary to carbon monoxide exposures. Under normal conditions, the heart muscle extracts both pyruvate and lactate for use in metabolic oxidation. However, at carboxyhemoglobin levels above 8.7%, neither is extracted and both are produced by the myocardium.[12]

Individuals with compromised cardiac vascularity may have increased sensitivity to carbon monoxide exposures and could experience angina at low carboxyhemoglobin concentrations. A preliminary series of studies suggested an earlier onset of angina in individuals with carboxyhemoglobin concentrations as low as 2% to 3%. Although those studies failed to endure peer review, it has been postulated that angina could occur in working individuals following carbon monoxide exposure.[4] Other studies indicate that myocardial irritability may result in abnormalities of the electrocardiograph or arrhythmias with carboxyhemoglobin levels above approximately 9%.[36]

Chest pain and tachycardia, in response to tissue hypoxia, may be present with carbon monoxide poisoning. Carbon monoxide lowers the threshold for ventricular tachycardia and therefore death secondary to arrhythmia is commonly associated with poisoning.[11] Individuals with preexisting cardiac disease, coronary artery disease, anemia, and lung disease are more susceptible to the effects of carbon monoxide–induced tissue hypoxia.[11,31]

A study titled *Non Invasive Ambulatory Assessment of Cardiac Function and Myocardial Ischemia in Healthy Subjects Exposed to Carbon Monoxide (CO)* is in progress at the U.S. Army Biomedical Research and Development Laboratory (USABRDL), Fort Detrick, Frederick, Maryland. The proposed research attempts to explore the potential interaction between progressive levels of carbon monoxide exposures and myocardial responses in human subjects. It is hypothetically plausible that increasing levels of exposure may induce signs or symptoms or both of myocardial ischemia among crews of armored vehicles. In these vehicles, the crew is routinely exposed to the same levels of carbon monoxide that have been associated with ischemic responses in experimental animals and human subjects.[45,46] It is possible that a soldier with early cardiovascular disease could suffer an adverse myocardial event if he or she is exposed to the concentrations of carbon monoxide that can be generated when armored vehicles are operated.

Specific objectives of this study are to identify the potential adverse relationships between cardiopulmonary response and progressively increasing levels of carboxyhemoglobin. Dosage ranges for carbon monoxide exposures will be manipulated by monitoring control of carboxyhemoglobin levels in a range of 5% to 20%. In addition, the simultaneous performance of simulated work loads approximating moderate effort at the tank loader position will be superimposed.

The initial study will evaluate cardiac performance profiles in 20 apparently healthy research volunteers. Five experimental conditions will be imposed, with the subjects

1. at rest,
2. on a treadmill,
3. performing upper-body exercise,
4. on a treadmill and exposed to increasing lev-

els of carbon monoxide exposures (5%, 10%, 15%, and 20% carboxyhemoglobin), and
5. performing upper-body exercise and exposed to comparable increases in carbon monoxide.

In one report, the capacity of individuals with carboxyhemoglobin levels of 15% to 20% to perform short-duration, submaximal physical work was not compromised. Individuals with 10% to 13% carboxyhemoglobin levels who performed work at 35% of the maximal work rate demonstrated only minimal increases in heart rate after working periods of 3.5 hours. The maximum work capacity (as defined by the maximum amount of oxygen that can be transported by the cardiopulmonary systems, which is described as VO_2 *max*) is decreased following carbon monoxide exposures that generate carboxyhemoglobin levels less than 5%. The capacity to perform physical work is dramatically compromised at carboxyhemoglobin levels in excess of 40% to 45%.[4] Time to fatigue and time to angina are both shortened after carbon monoxide exposure.[37]

Chronic Effects

Effects of chronic exposures to carbon monoxide were reported in the early 1920s.[2] Two groups of workers were identified: those who become acclimated and those who do not. E. R. Hayhurst believed that the acclimation difference was probably associated with cardiac condition at the time of exposures. Those individuals who were able to become acclimatized developed compensating increases in their erythrocyte counts and hemoglobin content. Although Hayhurst's conclusion remains controversial, other investigators have concluded that long-term exposures to carbon monoxide may be associated with arteriosclerotic heart disease.[31,37]

After being removed from carbon monoxide exposures, patients usually progressively improve without sequelae to complete recovery. However, some patients may have a transient period of apparent normal physiological recovery for days to weeks after poisoning, then develop evidence of CNS or cardiovascular-system impairment.[1,11] In early reports, the prognosis of recovery following carbon monoxide exposure was considered to be associated with the degree of asphyxia related to the exposure. In many cases, men who were exposed to carbon monoxide in mines were thought to develop a permanent weakness of the heart muscle as a consequence.[2] Other severe exposures were reported to cause loss of vision, speech, or other CNS defects. However, more recent reports suggest that carboxyhemoglobin levels correlate with neither the severity of the acute poisoning nor the potential for delayed effects.[11]

The sequelae of nonlethal acute exposure to carbon monoxide reported in the literature of the early 1900s included pneumonia, psychoses, paralysis, bullous skin lesions, and gangrene.[2] The spectrum of medical opinion concerning potential sequelae of exposure reported in the literature was broad and controversial. For example, the chief surgeon of an Illinois steel company, who had extensive experience in caring for carbon monoxide–poisoned workers, reported that he had never seen a case of psychosis directly attributed to the carbon monoxide exposure. In contrast, a contemporary practitioner listed 105 neuropsychiatric conditions that were associated with carbon monoxide exposure sequelae. The most common acute-onset neurological sequelae are aggressiveness, moodiness, irritability, impulsiveness, and memory loss. Transient CNS disorders may include neurological deficits, memory loss, cognitive difficulty, and personality change.[3,11]

Bilateral, low-density lesions in the area of the globus pallidus are characteristic findings associated with carbon monoxide poisoning and may be identified using computed tomography or magnetic resonance imaging. The lesions are usually seen in about 50% of individuals with severe poisoning; however, neuroradiological studies may not be positive for 2 to 3 days following acute poisoning. Lesions in the gray matter of the basal ganglia may regress, but lesions in the white matter are likely to become permanent and a delayed neuropathy will ensue.[11]

The delayed neuropsychiatric syndrome may occur as long as 6 weeks after the patient has recovered from acute toxic exposure to carbon monoxide. The syndrome occurs as a complication in 2% to 30% of carbon monoxide–poisoned patients.[3] The first case was reported in 1926. A 58-year-old woman had attempted suicide with carbon monoxide inhalation. After acute recovery, the woman became mute and suffered progressive disorientation and parkinsonism, followed by death. The autopsy revealed bilateral necrosis of the globus pallidus and widespread demyelinization of the subcortical white matter.[11]

Delayed sequelae appear to be frequent in the young and old, but more commonly occur in elderly patients who have suffered coma. All individuals who suffer coma do not experience delayed neurological sequelae,[11] but coma has been identified as a risk factor in other reports.[28] Clinical signs of delayed sequelae may include urinary or fecal incontinence, weakness, gait disturbances, tremor, mutism, speech abnormalities, and mental deterioration. Complete recovery occurs in about 75% of individuals within a year.[3]

DIAGNOSIS AND TREATMENT

It cannot be emphasized too strongly that the signs and symptoms of carbon monoxide poisoning are pleomorphic: headaches of varying degrees of severity, dizziness, nausea and vomiting, blurred vision, impaired thinking, and numbness and seizures.[47,48] Therefore, physicians must maintain a high degree of suspicion when confronted with a patient who manifests some or all of these signs and symptoms. A diagnosis of carbon monoxide poisoning will *never* be made unless the physician thinks to order confirming laboratory tests.

Clinical Diagnosis

Laboratory evaluations for carbon monoxide–poisoned patients are usually deceptively normal, with the exceptions of the blood carboxyhemoglobin content[11] and elevated carbon monoxide concentrations in exhaled breath.[12] The arterial oxygen contents appear normal and fail to confirm the initial diagnostic impression of anoxemia. This is because conventional laboratory analysis depends on two determinations: (1) measurement of the partial pressure of oxygen dissolved in the plasma, which is used to estimate the oxygen saturation of hemoglobin from a standard oxyhemoglobin dissociation curve (see Figure 11-6). This value, in conjunction with the measured concentration of hemoglobin, is used (2) to estimate the arterial oxygen content. Unfortunately, neither the partial pressure of oxygen dissolved in plasma nor the concentration of hemoglobin is affected by the presence of carbon monoxide in the blood. Because the laboratory determination of arterial oxygen saturation depends on two parameters that are not directly affected by carbon monoxide, it is not surprising that significant hemoglobin desaturation may be missed.

Analyzing exhaled air has been advocated recently as a method of measurement of blood levels of carboxyhemoglobin following carbon monoxide exposure. Measuring the carbon monoxide concentration in end-alveolar breath samples after the subject has held his or her breath for 20 seconds has two advantages: the equipment is field transportable and takes minimal training for proper use. Some authorities indicate that the method is an acceptable technique for documenting workplace exposure controls.[16]

Commonly available laboratory modalities are rarely diagnostic but may be confirmatory. Metabolic acidosis may be reflected by an increased level of lactic acid. Blood glucose level and nonspecific enzyme levels indicative of tissue injury (eg, creatine kinase, lactate dehydrogenase, alanine transferase, and aspartate transferase) may be elevated.[11]

Electrocardiographic findings are not specific for carbon monoxide intoxication, but demonstrate changes consistent with hypoxemia. Individuals with coronary artery disease may complain of angina with carboxyhemoglobin levels as low as 10%.[11] At a minimum, an electrocardiograph should be obtained for all individuals with chest pain who have been exposed to carbon monoxide. At carboxyhemoglobin levels above 25%, ST segment depression may be seen in leads II, V_5, and V_6. Depression of the ST segment has been used as a criterion for instituting hyperbaric oxygen therapy.[12]

Treatment

The essential first step in treating an individual suspected of being poisoned by carbon monoxide is to remove the patient from the potentially contaminated environment. This action is necessary not only to prevent further poisoning of the patient but also—and of equal importance—to prevent poisoning the healthcare provider. The following measures should be instituted once the patient is in a safe environment:

- Insert an endotracheal airway if the patency of the patient's upper airway is compromised.
- Ventilate mechanically if the patient's respiratory gas exchange is inadequate.
- Infuse intravenous fluids and vasoactive drugs if circulatory shock is present.

The patient should receive supplemental oxygen at a concentration of 100% as soon as possible. This requirement will necessitate the use of a tight-fitting face mask if intubation of the upper airway has not been performed.

The treatment of carbon monoxide poisoning is, in theory, extremely simple: increase the partial pressure of oxygen in the lungs so as to displace carbon monoxide from the carboxyhemoglobin. Although delivering 100% oxygen at 1 atm is an effective modality, this displacement can be accelerated by using higher ambient pressures. The technology for creating ambient environments of super-atmospheric pressures has long existed (its first known use was in 1664) but its use until recently was confined to treating deep-sea divers

stricken with decompression sickness. Because delivering pure oxygen at hyperbaric pressures is now used to treat a number of conditions, it is not surprising that hyperbaric oxygen has also been applied to carbon monoxide-poisoned patients.[9]

Carboxyhemoglobin response curves using room air, 100% oxygen, and hyperbaric oxygen treatment modalities can be compared, using the half-life of carboxyhemoglobin concentrations for room air (5 h and 20 min), 100% oxygen delivered by tightly fitting mask (1 h and 20 min), and hyperbaric oxygen at 3 atm (23 min) (Figure 11-8). Delivery of hyperbaric oxygen at 3 atm results in the physical dissolving of 6.4 volumes percent (eg, 6.4 mL oxygen in 100 mL plasma), which is sufficient to displace carbon monoxide from cytochrome a_3 oxidase in tissues.[12] When the patient breathes oxygen pressurized to 3 atm, adequate oxygen—sufficient to support metabolism even in the complete absence of functioning hemoglobin—is forced to dissolve in the plasma. Because the arteriovenous difference in cerebral blood flow is only 6.1 volume percent, the patient's ability to adequately oxygenate the brain and other tissues improves immediately.

The immediate therapeutic goals of hyperbaric oxygen therapy are to reduce cerebral and myocardial hypoxia, reduce cerebral edema, and enhance carbon monoxide elimination.[28] Actual and suggested benefits of hyperbaric oxygen therapy are that it

- rapidly provides sufficient dissolved plasma oxygen to meet the metabolic oxygen requirement;
- significantly enhances carboxyhemoglobin dissociation;
- causes increased carbon monoxide clearance[11,12]; and
- is beneficial in managing cerebral edema, a complication of carbon monoxide poisoning, reducing secondary intracranial pressure by 50% within 1 minute of its administration.[12]

It is important to note that some authorities believe that no conclusive evidence yet demonstrates a relationship between a shortened duration of symptoms and the frequency of delayed sequelae of poisoning.[11] However, one researcher uses hyperbaric oxygen at carboxyhemoglobin levels above 25% even if the patient is not particularly ill. It has been his experience that patients treated with hyperbaric oxygen are spared the prolonged headache and nausea and might experience fewer delayed aftereffects of poisoning.[12]

Although some researchers conclude that the use of hyperbaric oxygen is controversial,[11] another states that failure to administer hyperbaric oxygen for severely poisoned patients has resulted in successful malpractice litigation.[12] Others stress the therapeutic efficacy of hyperbaric oxygen administration.[28] Authorities generally agree that, for individuals with no other risk factors, hyperbaric oxygen is beneficial if carboxyhemoglobin levels are in excess of 25%.[3,11] The most important reason for administering hyperbaric oxygen appears to be the presence of neurological deficits such as disorientation or focal signs, loss of consciousness, and seizures.[29] Other indications include cardiac ischemia, metabolic acidosis, and pregnancy.[3]

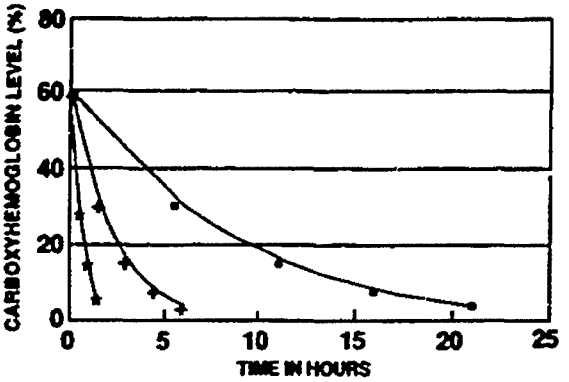

Fig. 11-8. Carboxyhemoglobin half-life. The comparative benefits of medical management for carbon monoxide-intoxicated patients using room air (■), 100% oxygen (+), and hyperbaric oxygen at 3 atm pressure (•). Reprinted with permission from Kindwall EP. Carbon monoxide poisoning. *Hyperbaric Oxygen Rev.* 1980;1(2):115–122.

Hyperbaric oxygen therapy has also been recommended for individuals who are considered to be more susceptible to carbon monoxide effects or sequelae (infants and children, and adults who have preexisting cardiac ischemia or seizure disorders,[11] arterial vasospasm, past myocardial infarct, anemia, and pregnant females).[28] The fetus is much more susceptible to carbon monoxide than the mother. The oxyhemoglobin dissociation curve for fetal hemoglobin is to the left of that for adult hemoglobin (oxygen does not dissociate as easily from fetal as from adult hemoglobin). As it does in adult hemoglobin, carbon monoxide reacts to form fetal carboxyhemoglobin, which accentuates the left shift. Carboxyhemoglobin levels in the fetus lag behind those in the mother; however, the final fetal hemoglobin level may be 10% to 15% higher than the maternal level.[30] In addition, the half-life of fetal

Fig. 11-9. Large walk-in clinical hyperbaric medicine facility, Travis Air Force Base, California. Photograph: Courtesy of the US Air Force.

carboxyhemoglobin is 15 hours; therefore, it takes 5-fold longer to regenerate oxyhemoglobin in the fetus than in the mother.[11]

Hyperbaric oxygen facilities are either a large walk-in chamber (Figure 11-9) or a small chamber that will accommodate only one person. The U.S. Air Force School of Aerospace Medicine at Brooks Air Force Base, Texas, maintains a 24-hour per day telephone system to help physicians locate the nearest hyperbaric oxygen treatment facility.[51] Although a specialist in hyperbaric medicine will probably manage the patient on arrival at the hyperbaric oxygen facility, it is important for occupational medicine physicians to understand these therapeutic procedures.

One clinician uses a walk-in hyperbaric chamber and routinely manages carbon monoxide–poisoned patients using 3 atm of oxygen pressure for 56 minutes (2 half-lives).[12] If the carboxyhemoglobin remains greater than 10% at that time, the pressure is decreased to 2 atm for the remainder of the treatment or until the carboxyhemoglobin is less than 10%. In very severe cases, the U.S. Navy Decompression Treatment Table 6 is used.[52] The table begins at 2.8 atm of oxygen for 4 hours, then decreases to 1 atm over 285 minutes, with 5- to 15-minute "air breaks" interspersed in the schedule. Occasionally, patients require therapy once or twice daily for several days. Patients may recover their memory after the second treatment, and patients treated with hyperbaric therapy do not appear to develop delayed cardiac or neurological sequelae of carbon monoxide poisoning.[12]

Hyperbaric oxygen administration has a number of therapeutic advantages, but is also associated with potential complications. Minor complications include tooth, ear, or sinus pain or discomfort. However, more serious complications—evidence of oxygen poisoning—can occur, including substernal pain, decreased vital capacity, or pulmonary microhemorrhage, although these conditions occur rarely. If individuals are treated for more than 6 hours with oxygen at 2 atm, pulmonary oxygen toxicity with microhemorrhage and fibrosis has been reported.[12] The only absolute contraindication against hyperbaric oxygen therapy is an untreated pneumothorax.[53]

HEALTH STANDARDS

Determining the exposure levels at which carbon monoxide causes health effects has been difficult; thus developing consistent, valid exposure standards has been problematic. This is due to both the large number of exposure variables that influence carbon monoxide poisoning and a high degree of fluctuation for many of the variables. For example, the respiratory rate, level of work effort, baseline carbon monoxide level, and individual tolerance for carbon monoxide must all be considered—in addition to carbon monoxide levels in air. Additionally, a *high rate of increase* in carboxyhemoglobin concentration may increase the probability that symptoms will develop. No direct, consistent relationship between estimates or measured carboxyhemoglobin levels and health effects has been demonstrated, at least for low-to-moderate levels of exposure.

The estimates of human toxicity were the subjects of early scientific controversy. Some scientists reported onset of symptoms at ambient carbon monoxide concentrations of 0.01%, while others reported onset of signs and symptoms at 0.05%. Another early investigator estimated the limit of toxicity as 0.2%. Such conflicts result from differing exposure scenarios and individual study differences.

Occupational Safety and Health Administration

The federal statutory exposure limits are promulgated by the Occupational Safety and Health Administration (OSHA). The current permissible exposure limit (PEL) for carbon monoxide is 35 ppm, with a ceiling concentration of 200 ppm. OSHA has defined *ceiling* as a concentration that should not be exceeded during any part of the workday. However, if instantaneous monitoring is not feasible, the ceiling must be assessed as a 15-minute time-weighted average (TWA).[34]

The PEL is intended to maintain the carboxyhemoglobin levels of exposed employees below 5%. The standard was developed to protect individuals with cardiovascular or pulmonary impairment and to protect healthy workers during conditions of heat stress, exertion, and strenuous conditions.[33] The immediately dangerous to life or health (IDLH) concentration promulgated by OSHA is 1,500 ppm. The IDLH for carbon monoxide is defined as the concentration that an unprotected worker could escape from within 30 minutes without experiencing irreversible health effects.[34] It has been estimated that for light activity (minute ventilation of 9–10 L/min), a carbon monoxide exposure concentration of 1% (10,000 ppm) could result in death in less than 10 minutes.[36]

American Conference of Governmental Industrial Hygienists

The term Threshold Limit Value (TLV) is published as an exposure recommendation for specific hazards by the American Conference of Governmental Industrial Hygienists (ACGIH). The TLV is defined as an 8-hour TWA concentration of a specific chemical to which nearly all workers may be exposed for 40 hours per week, day after day, without adverse effect.[33] The present recommended TLV for carbon monoxide is 50 ppm, with the indication that the ACGIH intends to reduce the recommended TLV to 25 ppm in 1993.

While the current TLV is intended to maintain carboxyhemoglobin concentrations below 10% in exposed workers, the intended 25 ppm level is expected to maintain carboxyhemoglobin levels below 3.5%. The decreased limit is intended to reduce exposure risks for susceptible workers such as pregnant women who must perform psychomotor tasks, or employees who have chronic cardiac or respiratory disease. Although the ACGIH currently recommends a 15-minute short-term exposure limit (STEL) of 400 ppm, the organization has expressed the intent to omit the STEL from future carbon monoxide exposure recommendations.[33]

Committee on Toxicology

The Committee on Toxicology of the National Research Council has developed and recommended emergency exposure guidance levels (EEGLs) for carbon monoxide (a ceiling limit for unpredicted exposure for a single, isolated, exposure time—usually less than 60 min, but never longer than 24 h) (Table 11-2). The EEGL has replaced an older term, the emergency exposure limit (EEL).[4]

In response to specific sponsor requests, the Committee on Toxicology will develop and provide recommendations for continuous exposure guidance levels (CEGLs). An older term, the continuous exposure level (CEL) was developed in response to a U.S. Navy request for guidance tailored to submarine environments. The CEGL is defined as a recommended exposure limit with the potential for continuous exposure for a duration up to 90 days. It is intended as a ceiling limit, and is designed to avoid both the degradation of

TABLE 11-2

CURRENT (1985) AND PREVIOUS (1965) EXPOSURE LEVELS FOR CARBON MONOXIDE

Exposure	1985 EEGL	1965 EEL	1985 CEGL	1965 CEL
10 min	1,500 ppm	1,500 ppm	—	—
30 min	750 ppm	800 ppm	—	—
60 min	400 ppm	400 ppm	—	—
24 h	50 ppm	200 ppm	—	—
90 d	—	—	20 ppm	25 ppm

EEGL: emergency exposure guidance level
EEL: emergency exposure limit
CEGL: continuous exposure guidance level
CEL: continuous exposure level
Source: Committee on Toxicology. *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants.* Vol 4. Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council. Washington, DC: National Academy Press; 1985.

military-mission performance and immediate or delayed adverse health effects among exposed military members. The current CEGL for carbon monoxide is 20 ppm, a decrease from the 1965 CEL of 25 ppm.[4]

In the past, both EELs and CELs have been used as design criteria for military equipment with enclosed environments such as submarines and spacecraft. The conditions and terms of applicability of the levels were related to narrowly defined occupational exposure groups and were not intended for general population exposure. They include no consideration of hypersusceptible individuals.[4] In 1985, the Committee on Toxicology provided the revised exposure levels (ie, EEGLs and CEGLs).

The Committee on Toxicology predicted that the recommended exposure levels should never result in carboxyhemoglobin levels above 10% as a result of exposure. The committee also predicted that an individual exposed to the 24-hour limit of 50 ppm should not demonstrate carboxyhemoglobin levels greater than 7.5%, or if exposed to 20 ppm for the 90-day limit, should not produce carboxyhemoglobin levels greater than 3.3%.[4]

The Committee on Toxicology stressed that individuals with compromised cardiovascular status (eg, as a result of atherosclerosis) may be at increased risk for angina or sudden cardiac death. The committee recommended that individuals exposed to these levels in militarily unique circumstances such as submarine service undergo careful physical examinations and refrain from smoking when exposed to these levels.[4]

The Committee on Toxicology reviews available scientific data and develops recommended exposure levels for use by its sponsoring organizations. The council was established by the National Academy of Sciences to assist the academy in preparing its advice to the federal government. The council has now become the principal operating agency of both the National Academy of Engineering and the National Academy of Sciences. Both organizations provide services to the government, the public, and the scientific and engineering communities. The Council is administered by both organizations and the Institute of Medicine.[4]

Military Standards

A militarily unique standard (Military Standard [MIL-STD]-1472C, Paragraph 5.13.7.4.1, *General*) has been promulgated for Department of Defense (DoD) use. The standard states that personnel will not be exposed to concentrations of toxic substances in excess of the DoD Occupational Safety and Health standards or specialized standards applicable to militarily unique equipment, systems, or operations.[60] With specific reference to carbon monoxide, MIL-STD-1472C, Paragraph 5.13.7.4.2, *Carbon Monoxide*, states

> that carbon monoxide in personnel areas shall be reduced to the lowest level feasible. Personnel shall not be exposed to carbon monoxide in excess of values which will result in carboxyhemoglobin levels in their

blood greater than the following percentages: 5 percent [carboxyhemoglobin] (all systems design objectives and aviation system performance limits); 10 percent carboxyhemoglobin (all other system performance limits).[40]

The pharmacodynamics of carbon monoxide intoxication have been discussed earlier in this chapter. Because many factors (in addition to absolute exposure level) influence the formation of carboxyhemoglobin, no one exposure level can directly be related to the 5% and 10% carboxyhemoglobin levels that are published in MIL-STD-1472C. Further complicating this dilemma is the large degree of variability of factors that relate to carbon monoxide exposure in military situations. Factors such as the duration of exposure, the rate of rise of carbon monoxide levels, and intermittent changes in levels are highly variable and, to some degree, influence carboxyhemoglobin formation.

Considerable time and effort have been spent attempting to resolve this dilemma. The goal has been to find and validate—over a wide range of exposure scenarios—a reliable predictive model for carboxyhemoglobin levels. In 1965, Coburn, Forster, and Kane evaluated major physiological variables that influence and determine the concentration of carboxyhemoglobin in humans.[34] In their experimental design, they developed equations using groups of normal subjects, male volunteers who breathed 100% oxygen for extended time periods, and patients with conditions known to contribute to elevated endogenous production of carbon monoxide. The Coburn-Forster-Kane equation resulted from these experiments.

Although it was first developed to calculate the endogenous production of carboxyhemoglobin, the Coburn-Forster-Kane equation is clinically useful when calculating carboxyhemoglobin levels following exogenous exposures.[4,34,35] The equation is based on parameters such as the

- ambient concentration of carbon monoxide,
- barometric pressure
- alveolar ventilation,
- comparative affinity of carbon monoxide for hemoglobin (a relative affinity of 218 is used in this formula),
- ambient concentration of oxygen,
- carbon monoxide diffusion across the alveolar membrane,
- rate of endogenous carbon monoxide production, and
- duration of exposure.[4,34]

Military Handbook 759A, paragraph 3.7.5, "Evaluation of CO Toxic Hazard," specifies the use of the Coburn-Forster-Kane equation:

> The prediction of carboxyhemoglobin blood content is determined by the following empirical equation:
>
> $$\text{carboxyhemoglobin}_t = \%\ (\text{carboxyhemoglobin}_0(e^{(-t/A)}) + 218(1 - e^{(-t/A)}) \bullet (1/B + \text{ppm carbon monoxide}/1316)$$
>
> where *carboxyhemoglobin*$_t$ is the predicted carboxyhemoglobin in the exposed individual; *carboxyhemoglobin*$_0$ is the amount of carboxyhemoglobin usually found in non-smoking adults; *t* is the exposure duration in minutes; and *ppm carbon monoxide* is the carbon monoxide exposure in parts per million of contaminated atmosphere; *A* and *B* are constants which are obtained from [Table 11-3] and depend on the estimated physical activity level [of] the individual during the exposure. This equation accounts for the minute respiratory volume of contaminated atmosphere actually respired by an exposed individual whose level of physical activity is either estimated or specified. For combat vehicle crewpersons, the specified work effort level scale required to be substituted in the equation is 4 for periods of weapons fire and 3 for intermediate periods. The equation also accounts for the elimination of carbon monoxide by the body.[36]

The equation is equally applicable to short-term, high-level exposures and low-level, long-term exposures.[36] The equation is considered to be a reliable tool and has demonstrated good correlation with experimental values under a variety of conditions.[37] With respect to potential exposure of soldiers and the devel-

TABLE 11-3

WORK-EFFORT CONSTANTS FOR THE COBURN-FORSTER-KANE CALCULATIONS

Work-Effort Scale	Work-Effort Description	A Value	B Value
1	Sedentary	365	939
2		211	1,623
3	Light work	155	2,211
4		119	2,874
5	Heavy work	97	3,536

Source: Military Handbook 759A. *Human Factors Engineering Design for Army Materiel (Metric).* Redstone Arsenal, Huntsville, Ala: US Army Missile Command, Standardization Division; 30 June 1981. Table 3-5.

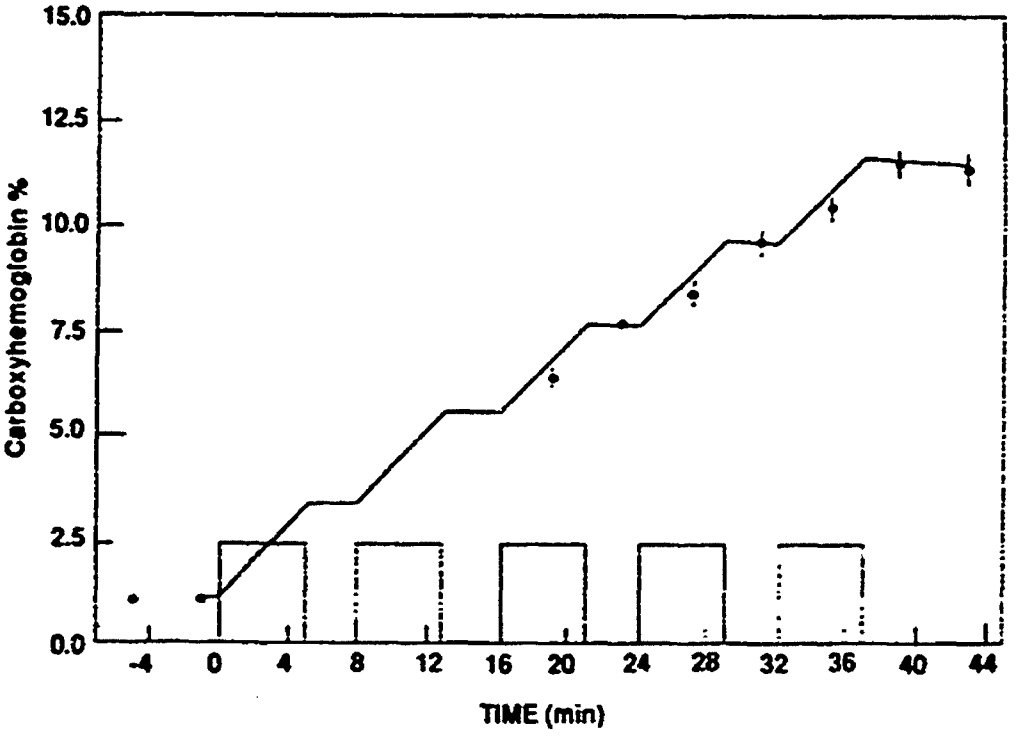

Fig. 11-10. Excellent correlation can be seen between calculated (—) and measured (•) levels for intermittent exposures (boxes along X axis). Subjects were exposed to 1,500 ppm of carbon monoxide for 5 minutes followed by a 3-minute rest. Source: adapted from Tikuisis P, Buick F, Kane DM. Percent carboxyhemoglobin in resting humans exposed repeatedly to 1,500 and 7,500 ppm CO. *Journal of Applied Physiology*. 1987;63:820–827.

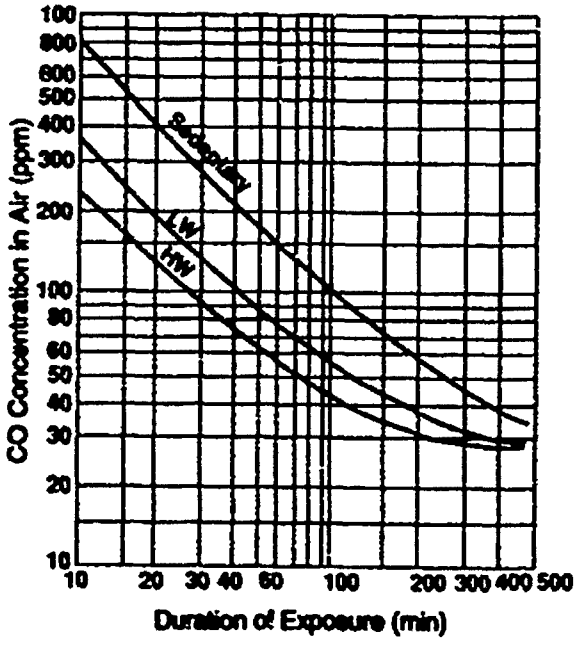

Fig. 11-11. The predictive relationship between the level of work effort, ambient carbon monoxide concentration, and duration of exposure to achieve a cumulative level of 5% carboxyhemoglobin. Source: US Department of Health, Education, and Welfare. *Criteria for a Recommended Standard .... Occupational Exposure to Carbon Monoxide*. Washington, DC: National Institute for Occupational Safety and Health and US Government Printing Office; 1972.

opment of military items, the exposure magnitude, frequency, and duration of exposure to carbon monoxide are not restricted as long as the criteria of MIL-STD-1472C, 2 May 1981, are met.[40,54]

The applicability of the Coburn-Forster-Kane equation to the intermittent, episodic, peak-and-valley carbon monoxide exposure profile of the exposed military individual has been questioned. However, through an intermittent series of questions and evaluations, the equation was determined to be both applicable and predictive of actual carboxyhemoglobin level following exposures.[24] The authors of an independent study calculated predicted cumulative carboxyhemoglobin levels following intermittent exposures.[55] Figure 11-10 is adapted from that study, which has been useful in clarifying this relationship, and reflects the cumulative amount of carboxyhemoglobin following repetitive, intermittent exposures to 1,500 ppm of carbon monoxide. The study also evaluated the predictive capability using shorter-term, higher-concentration exposures (7,500 ppm) and demonstrated similar predictive success.

Figure 11-11, which has been reproduced from the NIOSH criteria document for occupational exposure to carbon monoxide,[32] incorporates the Coburn-Forster-Kane variables to develop families of curves. Because these curves incorporate the level of exertion, healthcare workers can quickly assess the work-effort level of a job and estimate the permissible duration of exposure at a given ambient carbon monoxide exposure concentration to achieve a 5% carboxyhemoglobin level.

MEDICAL SURVEILLANCE

Employees who routinely are exposed to carbon monoxide at potentially hazardous levels should be carefully evaluated prior to exposure. In addition to being given a careful medical history, review of medical systems, and medical examination, the employee should be advised of the insidious hazard posed by carbon monoxide exposure. Proper engineering controls and necessary PPE should be provided and properly maintained for each employee. Exposures to hazardous levels should be prevented when possible and documented in the medical record if significant exposures occur.

Employees should have a preplacement examination to detect preexisting conditions that may increase the risk of carbon monoxide toxicity and to establish a baseline for future monitoring. Medical histories that could contribute to susceptibility include smoking, coronary artery disease, anemia, chronic obstructive pulmonary disease, cerebrovascular disease, and/or disorders of the CNS. Employees who are either pregnant or considering pregnancy should be advised of the increased fetal susceptibility (the fetus can be exposed in utero if external exposures are uncontrolled), encouraged to quit smoking, and informed concerning methods to minimize exposure potentials.

The physical examination provided for preplacement, periodic, and termination examinations should emphasize the cardiovascular system, the pulmonary system, and the CNS. A complete blood count baseline should be obtained, and subsequent analyses obtained if they are clinically indicated.

Employees whose occupations include the potential for daily exposures to carbon monoxide above the acceptable action level should be offered periodic medical examinations. In addition, a medical evaluation is indicated following an acutely hazardous carbon monoxide exposure (a situational exposure) or on termination of employment. In situational exposures, a venous blood sample should be obtained for carboxyhemoglobin measurement as soon as possible following the exposure. A determination of end-alveolar carbon monoxide, as an indirect measurement of carboxyhemoglobin, may be used when blood carboxyhemoglobin determinations are not available,[16] although this is less preferable. Workplace carbon monoxide concentrations should routinely be documented by the industrial hygienist; levels above the action level should be referred to the healthcare provider as a stimulus for surveillance evaluation.

The occupational medicine physician should perform and document evaluations of the patient's mental, baseline neurological, and visual or ophthalmological status. One useful tool is the carbon monoxide–neuropsychological screening battery. This test, which can be administered easily by healthcare providers, evaluates the following functions, which carbon monoxide disrupts most commonly:

- short-term memory (for events that occurred 1–24 h ago),
- concentration (ability to concentrate to perform simple tasks),
- visual spatial ability (ability to distinguish among several objects placed close to each other),
- agnosia (lack of sensory ability to recognize objects), and
- aphasia (a weakness in or loss of the ability to understand ideas by reading, writing, or speaking).[57]

SUMMARY

Exposure to carbon monoxide can cause acute clinical illness. Military exposures to the chemical occur in homes, vehicles, and weapons systems. The profile of exposure during the performance of military duties is typically episodic, with high peak exposures followed by prolonged periods of limited, minimal exposure.

Although the primary effect of carbon monoxide poisoning appears to be the consequence of hypoxia associated with the production of carboxyhemoglobin, cellular enzyme systems are also adversely affected by carbon monoxide poisoning. Individuals with coronary heart disease appear to be at excess risk of adverse effect, such as myocardial infarction, following acute exposures. Chronic ophthalmological and neurological sequelae have been reported following carbon monoxide exposure. Timely diagnoses and implementation of appropriate methods to improve tissue oxygenation, such as hyperbaric oxygen treatment, are beneficial in the reduction of mortality and morbidity following carbon monoxide intoxication.

REFERENCES

1. US Department of the Army. *Carbon Monoxide: Symptoms, Etiology, Treatment and Prevention of Overexposure.* Washington, DC: DA; 1968. Technical Bulletin MED 269.

2. Apfelbach GL. Carbon monoxide poisoning. (Revised) In: Kober GM, Hayhurst ER, eds. *Industrial Health.* Philadelphia, Penn: P. Blakiston's Son & Co; 1924.

3. Rubenstein E. Carbon monoxide poisoning and smoke inhalation. In: Rubenstein E, Federman DD, eds. *Scientific American Medicine.* New York: Scientific American; 1990: Chap 8.

4. Committee on Toxicology. *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants.* Vol 4. Board on Toxicology and Environmental Health Hazards, Commission on Life Sciences, National Research Council. Washington, DC: National Academy Press; 1985.

5. White JJ. Carbon monoxide and its relation to aircraft. *US Naval Medical Bulletin.* 1932;30:151–165.

6. Macpherson WG, Herringham WP, Elliot TR, Balfour A. *History of the Great War Based on Official Documents: Medical Services Diseases of the War.* Vol 2. London, England: His Majesty's Stationery Office; 1923.

7. Haldane JS. *Respiration.* New Haven, Conn: Yale University Press; 1922.

8. Kober GM. Prefatory: Historical review of industrial hygiene and its effects on public health. In: Kober GM, Hayhurst ER, eds. *Industrial Health.* Philadelphia, Penn: P. Blakiston's Son & Co; 1924.

9. Sayers RR, Yant WP. Dangers of and treatment for carbon monoxide poisoning. *Reports of Investigations.* US Department of the Interior, Bureau of Mines; May 1923. RI 2476. Available from UPDATA Publications, Los Angeles, Calif.

10. Wilmer WH. Effects of carbon monoxide upon the eye. *American Journal of Ophthalmology.* 1921;4:73–90.

11. Seger DL, Welch L. Carbon monoxide. In: Sullivan JB, Krieger GR, eds. *Hazardous Materials Toxicology: Clinical Principles of Environmental Health.* Baltimore, Md: Williams & Wilkins; 1992: Chap 114: 1160–1164.

12. Kindwall EP. Carbon monoxide. In: Zenz C. *Occupational Medicine: Principles and Practical Applications.* 2nd ed. Chicago: Year Book Medical Publishers; 1988: Chap 30: 503–508.

13. Sous-Secrétariat d'Etat du Service de Santé Militaire, ed. *The Clinical and Therapeutic Aspects of Gas Poisoning* [in French]. Office Chief of Gas Service, American Expeditionary Forces; 1918. Report Z-207. Provided by Alexandria, Va: Defense Technical Information Center Report. Defense Logistics Agency (trans). AD 498534.

14. Nelson N. *NDRC Infra-Red Gas Analyzer for Carbon Monoxide*. Fort Knox, Ky: Armored Medical Research Laboratory; 1945. Provided by Alexandria, Va: Defense Technical Information Center Report. Defense Logistics Agency. AD 655578.

15. Baetjer A. Professor emeritus, Department of Environmental Health Sciences, The Johns Hopkins School of Hygiene and Public Health, Baltimore, Md. Personal communication; 1984.

16. Stewart RD, Stewart RS, Stamm W, Seelen RP. Rapid estimation of carboxyhemoglobin level in fire fighters. *JAMA*. 1976;235:390–392.

17. Nelson N, Walpole RH, Swigert TC. *Carbon Monoxide Hazard from Exhaust Gases in Tanks That Are in Tow*. Fort Knox, Ky: Armored Medical Research Laboratory; 1944. Project 28.

18. Bellamy RF. The Borden Institute. Walter Reed Army Medical Center, Washington, DC. Personal communication, 1993.

19. Geddie JC. *M1E1 Carbon Monoxide Exposure Incident*. Fort Hood, Tex: US Army Human Engineering Laboratory, Liaison Office (TCATA); 1984. Memorandum for the Director, USAHEL.

20. Schmit A. *Toxic Fumes Testing of the Infantry Fighting Vehicle (IFV)*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency, HSE-OI-F/WP; 1980. Industrial Hygiene Special Study 55-35-0185-80.

21. US Department of Defense. *Procedure for Carbon Monoxide Detection and Control in Aircraft*. Washington, DC: DoD; 1958. Military Standard 800.

22. Brown L. *Initial Toxic Fumes Testing of the Bradley Fighting Vehicle M2E1 at Camp Roberts, Calif.* Aberdeen Proving Ground, Md: TECOM, STECS-MA-P, Measurements and Analysis Directorate, Physical Test Division; 1984. Laboratory Report 85-CC-23.

23. Cohen JP. *Toxic Gases Testing of the M2 Infantry Fighting Vehicle, FMC Corporation, Camp Roberts, Calif., 14–16 October 1981*. Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1982. Technical Report HSE-OI/WP; Industrial Hygiene Special Study 55-35-0109-82.

24. Shrum GL, Martin D, Breen D. Health Hazard Assessment Report (RCS MED 388) on the Howitzer Improvement Program (HIP). Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1988. Report 69-37-4563-88.

25. Carbon monoxide poisoning. *BUMED News Letter*. 1944;2(10, Aviation Suppl 12):4–6. Provided by Alexandria, Va: Defense Technical Information Center Report. Defense Logistics Agency. AD 895326.

26. Chemical warfare service develops a protection against carbon monoxide. *The Military Surgeon: Journal of the Association of Military Surgeons of the United States*. 1928;(July):115–116.

27. Gaydos JC. Department of Preventive Medicine, F. Edward Hébert School of Medicine, Uniformed Services University of the Health Sciences, Bethesda, Md. Formerly 7th Medical Command Consultant in Epidemiology (1979–1982). Personal communication; 1991.

28. Kim J-K, Coe C-J. Clinical study on carbon monoxide intoxication in children. *Yonsei Medical Journal* (Korea). 1987;28(4):266–273.

29. "WM." *Science*. 1920;51(April):437–438. Letter to the editor re: carbon monoxide.

30. Smith RP. Toxic responses of the blood. In: Amdur MO, Doull J, and Klaassen CD, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 4th ed. New York: Pergamon Press; 1991: Chap 8.

31. Guyton AC. *Textbook of Medical Physiology*. 7th ed. Philadelphia, Penn: W.B. Saunders; 1986; 481–500.

32. US Department of Health, Education, and Welfare. *Criteria for a Recommended Standard .... Occupational Exposure to Carbon Monoxide*. Washington, DC: National Institute for Occupational Safety and Health and US Government Printing Office; 1972.

33. American Conference of Governmental Industrial Hygienists. *Documentation of the Threshold Limit Values and Biological Exposure Indices*. 6th ed. Cincinnati, Oh: ACGIH, 1991.

34. Coburn RF, Forster RE, Kane PB. Considerations of the physiological variables that determine the blood carboxyhemoglobin concentration in man. *J Clin Invest*. 1965;44:1899–1910.

35. Verma A, Hirsch DJ, Glatt CE, Ronnett GV, Snyder S. Carbon monoxide: A putative neural messenger. *Science*. 1993;259:381–384.

36. Turino GM. Effect of carbon monoxide on the cardiorespiratory system. Carbon monoxide toxicity: Physiology and biochemistry. *Circulation*. 1981;63:253A–259A.

37. Nightingale TE. *Biological Effects of Short, High-level Exposure to Gases: Carbon Monoxide*. Rockville, Md: Enviro Control, Inc; 1980. US Army Biomedical Research and Development Laboratory Technical Report. Contract DAMD 17-79-C-9086.

38. Henderson Y. Carbon monoxide poisoning. *JAMA*. 1916;67:580–583.

39. Crocker PJ. Carbon monoxide poisoning, the clinical entity and its treatment: A review. *Military Medicine*. 1984;149:257–259.

40. US Department of Defense. *Human Engineering Design Criteria for Military Systems Equipment and Facilities*. Washington, DC: DoD; 1981. Military Standard 1472C.

41. Benignus VA, Muller KE, Kafer ER, Pieper KS, Smith MV. *Lack of Effects of High Level Carboxyhemoglobin on Compensatory Tracking and EEG: Protocol 3. Final Report* Fort Detrick, Frederick, Md: US Army Medical Research and Development Command; 1987. Technical Report, Project Order 1812.

42. Benignus VA, Muller KE, Barton CN, Prah JD. Effect of low level carbon monoxide on compensatory tracking and event monitoring. *Neurotoxicology and Teratology*. 1987;9:227–234.

43. Benignus VA, Kafer ER, Muller KE, Case MW. Absence of symptoms with carboxyhemoglobin levels of 16%–23%. *Neurotoxicology and Teratology*. 1987;9:345–348.

44. Hudnell HK, Benignus VA. Carbon monoxide exposure and visual detection thresholds. *Neurotoxicology and Teratology*. 1989;11:1–8.

45. Penney DG. A review: Hemodynamic response to carbon monoxide. *Environmental Perspectives*. 1988;77:121–130.

46. Davies DM, Smith DJ. Electro-cardiographic changes in healthy men during continuous low-level carbon monoxide exposure. *Environmental Research*. 1980;21:197–206.

47. Turnbull TL, Hart RG, Strange GR. Emergency department screening for unsuspected carbon monoxide exposure. *Annals of Emergency Medicine*. 1988;17(5):478–483.

48. Heckerling PS, Leikin JB, Maturen A, Perkins JT. Predictors of occult carbon monoxide poisoning in patients with headache and dizziness. *Annals of Internal Medicine*. 1987;107:174–176.

49. Heintish H. Hyperbaric oxygen therapy. *Hospital Physician*. 1990;(December):26–30.

50. Chance JF. Carbon monoxide poisoning. *Audio-Digest Emergency Medicine: Poison Particulars*. 1987;4(16). Case Report 1.

51. The hyperbaric oxygen treatment telephone system is maintained by the US Air Force School of Aerospace Medicine at Brooks Air Force Base, San Antonio, Tex. The telephone number is (commercial) (512) 536-3281 or (Defense Services Network) 240-3281 during the business hours 0730–1630 Central Time.

52. Naval Sea Systems Command. Air diving. In: *US Navy Diving Manual*. Vol 1. Washington. DC: Naval Sea Systems Command; 15 December 1988. NAVSEA 0994-LP-001-9010, Rev 2.

53. Sloan EP, Murphy DG, Hart R, et al. Complications and protocol considerations in carbon monoxide–poisoned patients who require hyperbaric oxygen therapy: Report from a ten-year experience. *Annals of Emergency Medicine*. 1989;18(6):629–634.

54. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. *NIOSH Pocket Guide to Chemical Hazards*. Washington, DC: US GPO; 1990.

55. Tikuisis P, Buick F, Kane DM. Percent carboxyhemoglobin in resting humans exposed repeatedly to 1,500 and 7,500 ppm CO. *Journal of Applied Physiology*. 1987;63:820–827.

56. Military Handbook 759A. *Human Factors Engineering Design for Army Materiel (Metric)*. Redstone Arsenal, Huntsville, Ala: US Army Missile Command, Standardardization Division; 1981.

57. Messier LD, Myers RAM. A neuropsychological screening battery for emergency assessment of carbon monoxide poisoned patients. *Journal of Clinical Psychology*. 1991;47(5):675–684.

Chapter 12

LEAD

RICHARD M. LACHIVER, M.D., M.P.H.*

INTRODUCTION

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- Handling and Firing Munitions
- Electrical Soldering and Ballast Handling

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- Water
- Ingested Food and Nonfood Material

PHARMACODYNAMICS
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SUMMARY

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INTRODUCTION

Lead is a member of the heavy metal series in the periodic table. Its chemical symbol, Pb, is derived from the Latin word for lead, *plumbum*. Metallic lead does not occur naturally. It is very dense, with a specific gravity of 11.34 and a molecular weight of 207.19, and is bluish white to silvery gray. It has no distinctive odor and at typical ambient temperatures is a soft, malleable solid. While lead's melting point (327.5°C) and boiling point (1,740°C) are fairly low, as a fine powder it is combustible when exposed to heat or flames. In general, lead compounds are insoluble in water, although many are readily soluble in acidic solutions, which is the chemical characteristic that most allows lead to cause physiological harm.

Many naturally occurring ores contain lead, but galena (lead sulfide, PbS) is the form most commonly found and mined. Inorganic lead, as used in occupational and environmental health contexts and for regulatory purposes, typically includes lead oxides, lead salts (exclusive of arsenate and organic salts), and pure metallic lead. Lead can also be alloyed with other metals. The most common alloys are bronze (with copper) and solder (with tin). Some modern solders are based on antimony or silver rather than lead, but these substitutes are costly and are necessary only in specialized applications. (No lead-based solders are used with consumable items such as canned foods, for example.) Although the number of common organic lead compounds is much more limited, and tetraethyl and tetramethyl lead account for by far the greatest amount of lead found in industrial processes, lead is still commonly used in industry (Table 12-1).

Lead's military utility was recognized early on, as were some of its potential adverse health effects. Most of the medical aspects of exposure to lead are not militarily unique, however, and therefore are beyond the scope of this chapter. More is known about lead than virtually all others metals; the full extent of its toxicity is the subject of many dedicated medical textbooks such as *Lead Toxicity*.[1]

HISTORY

Archaeological research on human skeletal remains suggests that the use of lead was minimal until about the second and third millennia BC.[2,3] Until then, exposure was limited to windblown metallic dust that was directly inhaled or ingested. Typical ambient-air lead concentrations were probably 100-fold lower than current levels.[3,4] However, lead's widespread availability and its ease of handling helped to make it an ideal raw material for early civilizations. Forming lead into useful products was possible largely because of the metal's low melting point, making sophisticated extraction and manufacturing techniques unnecessary. Egyptian civilization had a variety of uses for lead (eg, they discovered that it could be used as a pigment to add color to pottery and cosmetics). Subsequent Greek and Roman civilizations continued to use lead in pipes and water aqueducts, coins, vessels for water and food, roofing, writing tablets, cosmetics, and medicines.[5]

The archaeological and written evidence suggests that the Greeks and Romans were aware that lead was toxic. However, the degree of sophistication of their understanding and efforts to prevent intoxication remains largely unknown, as several historical records illustrate: while Hippocrates was probably the first to report *lead colic* (spasmodic and recurrent episodes of abdominal pain) in 370 BC, and Nicander described similar effects in the 2nd and 1st centuries BC, these healers gave no indication that they understood how exposure could have occurred or how the disease could have been averted. However, reports from the 1st century AD document that Pliny the Elder, who was not a healer but a historian, warned mariners to protect themselves when painting their ships: "Cover yourselves with...animal bladder...lest you inhale this pernicious dust."[5]

Pliny the Elder's warning indicates that while some people of the day may have understood that lead was a threat, many others probably did not. Because lead intoxication is often insidious and many of its effects arise only after long periods of exposure, only a few of those exposed may have suspected that they were affected. Relating exposure to effect was undoubtedly difficult. Social factors could also have played a role: the nobility may have surmised that, because "pernicious dust" was a problem encountered only by the working class, it did not concern them.

Although many causes probably led to the collapse of the Roman Empire, lead toxicity could have played a key role.[6] Household articles such as glazed pitchers and containers for foods and beverages often contained lead. Furthermore, lead containers were often used to store wine. Both the acidic character of the

TABLE 12-1

COMMON LEAD-BASED COMPOUNDS

Common Name	Formula	Use
Litharge	PbO	Red and yellow pigments, batteries, rubber manufacturing, glass, varnish
Red oxide	Pb_3O_4	Anticorrosives, red pigments, ceramic glaze
Black oxide	PbO_2	Batteries
White lead	$PbCO_3(OH)_2$	White pigments (once the most common source of industrial lead intoxication)
Lead chromate	$PbCrO_4$	Yellow pigment
Lead arsenate	$Pb(AsO_4)_2$	Insecticide
Lead nitrate	$Pb(NO_3)_2$	Explosive
Tetraethyl lead	$Pb(C_2H_5)_4$	Antiknock component for gasolines
Lead silica.	$PbSiO_3$	Ceramic glaze

wine and the practice of heating it (a social custom of that time) hastened the leaching of lead from the containers. This practice probably produced quite high levels of exposure to lead for all who consumed the wine. Further speculation suggests that the ruling or elite classes were preferentially exposed to lead, in that they had greater access to luxuries. For example, many high-quality ceramic pitchers were glazed with lead, and lead plumbing, lead-based ceramic tableware, and wine were generally unavailable to the lower classes. Thus, ironically, the poor were often spared exposure.

Some support of this theory of preferential exposure has come from archaeological examination of skeletal remains. These studies indicate that wealthy individuals in Roman society tended to have very high bone burdens of lead relative to the poor. The speculation that lead contributed to the fall of the Roman empire is based in part on an understanding of lead's toxic effects on the reproductive system: lead could have poisoned the noble class, and also their prospects for subsequent generations of leaders, by causing a wide variety of reproductive dysfunctions. The average number of offspring per reproductive nobleman or -woman was probably quite low and the few offspring born to them would have tended to have intellectual deficits. These offspring were rarely suited to carry on the intellectual and cultural responsibilities (and demands) needed to perpetuate an empire. Attempting to deal administratively with the low reproductive rate, the Roman Senate, under Caesar Augustus, enacted laws in 18 BC and AD 9 that penalized aristocratic bachelors and rewarded women who produced three or more offspring.[7] This illustrates why administrative controls often fail: the remedy did not address the root problem. As a result, because the administrators had no inkling that the real cause of the problem was exposure to lead, the elite class dwindled.

Lead-related disease was relatively forgotten from the collapse of the Roman Empire until the Middle Ages. Medieval Europeans may have continued to poison themselves with beverages contaminated with lead, but little detailed information exists. But rapidly expanding industrialization cultivated a growing appetite for lead. From this time forward, the use of lead flourished, and with it, the number of individuals exposed. By the 1700s, the father of occupational medicine, Bernardo Ramazzini, had related occupation to exposure, and, ultimately, exposure to effect. In particular, Ramazzini noted a high prevalence of lead exposure and lead poisoning among potters.[8]

History repeats itself. Many cases of militarily relevant industrial lead intoxication occurred after World War I and World War II. During the large-scale disarmament of naval vessels that followed those wars, personnel were exposed to metallic lead in the superstructures and also in lead-based paint. During our 20th-century prohibition of alcohol, an epidemic of lead poisoning occurred, brought on by the consumption of moonshine whiskey. The most convenient condenser for such stills was the coil of an automobile radiator—which was made of lead.

The use of lead-free gasoline and nonlead paints has dramatically reduced the potential for contamination

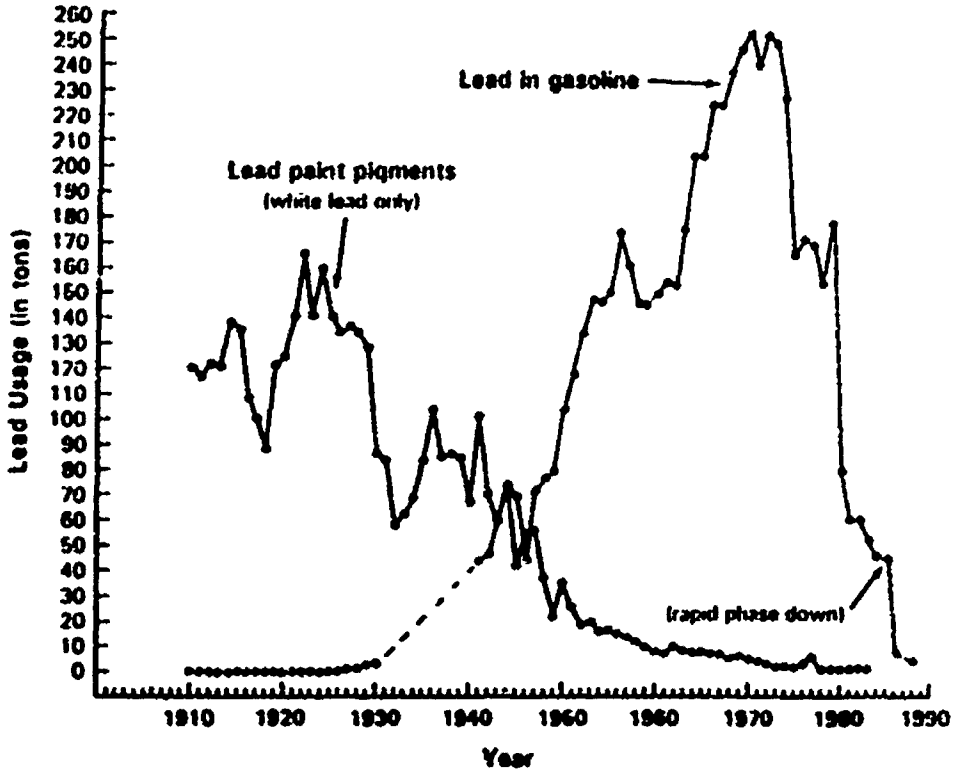

Fig. 12-1. The annual usage of leaded paint pigments and lead in gasoline has declined markedly during the 20th century. The advent of lead-free gasoline in 1978 resulted in dramatic reductions in lead exposure from gasoline emissions. Since 1910, gradual replacement of lead-based paints with nonlead pigments has lessened exposure from this source. Federal regulation of lead content in paint did not occur until 1977. Reprinted with permission from Adamson RH, et al, eds. A Digest special report: The Fourth National Environmental Health Conference. *Health & Environment Digest*. 1990;3(8):3. Adapted from Mielbe, National Environmental Health Conference Paper, 1989. Source: US Bureau of Mines.

from those sources (Figure 12-1). But lead exposure in the military—particularly in old military housing—has been sufficient to attract the attention of the Centers for Disease Control and the Department of Defense, and lead is sometimes touted as the asbestos of the 1990s.

OCCUPATIONAL EXPOSURES

The U.S. military has found many uses for lead, one of the earliest and most notable of which was as lead *shot* (musket balls and cannon shot). Numerous historical *shot towers* exist in the United States and elsewhere (Figure 12-2). Shot was produced at these towers by dropping molten lead from the top of a tall tower through a sievelike device. As it dropped, the molten lead solidified into small spheres: the shot. The modern uses of lead in the U.S. Army are more diverse: paints (particularly those with school bus–yellow and forest-green pigments), munitions components, electrical solders, and ballast (the army owns more boats than the navy and more aircraft than the air force).

Over 1 million workers in 100 different occupations in the United States are thought to be potentially exposed to lead as a result of their occupation.[10] The highest prevalence of civilian occupational lead intoxication in this country has been documented among lead-smelter and storage-battery workers.[11] Certain lead-related operations are not seen in the military (or are not as commonplace as they are in the civilian sector): lead smelting, primary fabrication (foundries), battery manufacture, and mining. However, at a typical army installation, 10% to 15% of the workers may be involved in potentially lead-hazardous operations. Based on industrial hygiene reviews of army

Fig. 12-2. Shot towers are one of the United States military's earliest sources of lead contamination. Soldiers working in shot towers were exposed to molten lead used in the production of musket balls and cannon shot. Source: US Army, Anniston Army Depot, Anniston, Ala.

worksites, the military occupations at highest risk for exposure to lead are, from highest to lowest, (*a*) abrasive blasters, (*b*) welders, (*c*) weapons firers, (*d*) painters, (*e*) electrical solderers, and (*f*) ballast handlers. As many as 5,000 to 10,000 workers in the army depot workforce may potentially be exposed to significant amounts of lead.

Applying and Removing Paint

Occupational exposure to lead in paint can occur during its application and removal. Spray painting can produce a respirable aerosol, and workers who fail to use proper respiratory personal protective equipment (PPE) or practices will be exposed to lead (Figure 12-3). Brush painting poses a much lower risk of exposure.

The use of lead-based paint has decreased significantly in recent years, but many of the paints used by the military still incorporate lead in small quantities. For example, lead is a component of some pigments, but usually constitutes less than 1% of the total. Lead is also used in *chemical agent resistant coating* (CARC) paints. Leaded non-CARC paints continue to be used because their resistance to corrosion and rust is far better than that of nonlead paints. This is of particular

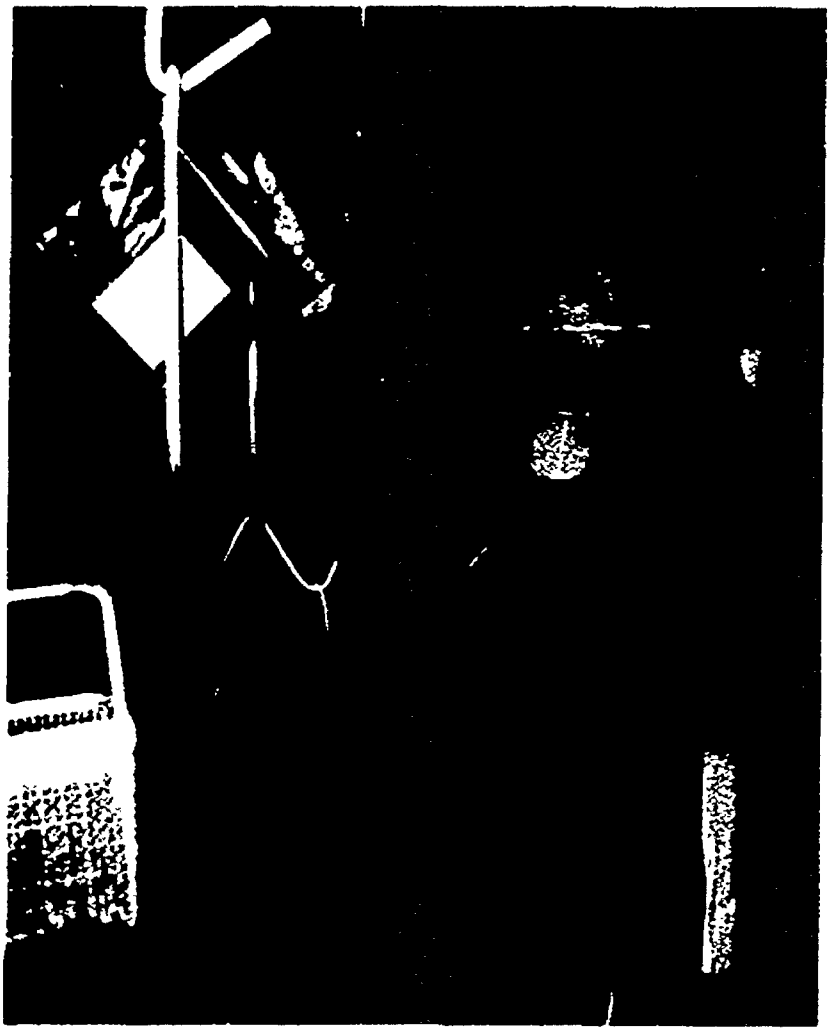

Fig. 12-3. Worker performs spray painting on drag-line component parts against a waterfall paint booth. During such operations, workers are at risk of inhaling aerosolized lead unless protective respirators are worn properly. Source: US Army, Anniston Army Depot, Anniston, Ala.

importance to the military; corrosion resistance under widely variable environmental conditions is a prime consideration in materiel specification and function. Probably the best example of this is the U.S. Navy's continued use of lead-based paints on its seagoing vessels. Therefore, resistance to corrosion, cost, and other factors such as formulation, application, and storage influence some procurement decisions toward the use of lead-based paints. In these instances, it is in the best interests of the military, or any other industrial employer, to control for exposure rather than substitute products, and leaded paints probably will continue to be used despite their potential adverse effects on health or their environmental impact.

Even if no new leaded paint were to be applied, many pieces of older military hardware still have coats of leaded paint on them. This equipment is, and will continue to be, maintained at depots. Thus, the potential for medically significant exposure to lead is likely to continue for as long as lead-painted equipment remains within the military inventory.

Paint is usually stripped by spraying sand (or some other abrasive material such as bits of steel, aluminum, or other hard substance) forced from a compressed air source toward a painted target. Despite the potential that the blaster will be exposed to lead, stripping may still be done because (*a*) it is safer to weld on clean, unpainted metal, (*b*) equipment sometimes needs a new, complete, and effective coat of paint, and (*c*) paint sometimes must be removed before equipment can be

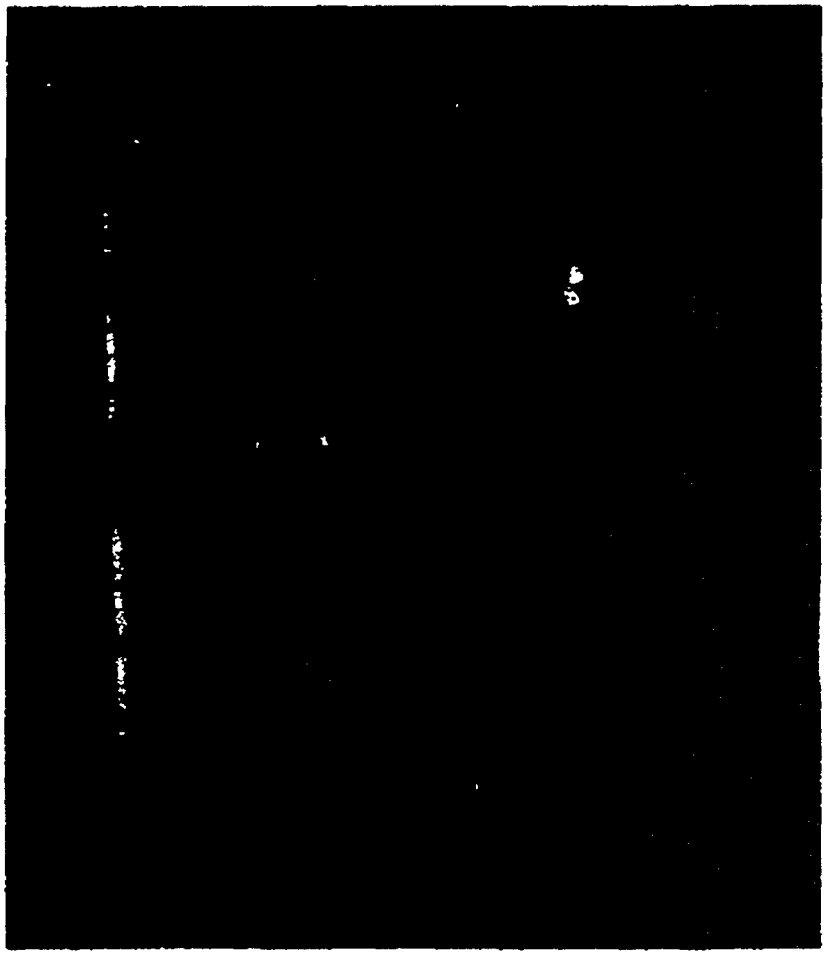

Fig. 12-4. A worker uses compressed air in the painting preparation process. This process is often called "blasting" or "sandblasting" if sand is the abrasive material. Exposure to lead can result from the dust from the residual paint. Source: US Army, Anniston Army Depot, Anniston, Ala

repaired. Stripping paint is not as easy to control as applying it, and therefore exposure to lead is more likely (Figure 12-4).

Stripping paint is quite labor intensive; it requires a significant amount of worker movement (bending, crouching, stretching) for prolonged periods of time in awkward positions to ensure that all paint is stripped off. PPE such as gloves, goggles, and respirators often does not work well with this kind of physical activity.

Stripping often generates highly respirable dust. Individuals who perform this kind of work must direct the flow of abrasive into the equipment's many nooks and crannies to remove paint that has sometimes been on for decades. Tanks or other vehicles can have hundreds of hard-to-reach places from which paint must be stripped. Sometimes this requires the worker—while lying under a vehicle—to spray the abrasive blast upwards; sometimes it requires the worker to direct the abrasive material into blind spaces, where it can only be reflected back at the blaster. Workers who operate the equipment may not be aware that the old paint they are stripping off actually contains lead.

The Occupational Safety and Health Administration, as part of the Hazard Communication Standard, requires that the Material Safety Data Sheet be included with every package or container of paint.[12] The availability of information relating to health hazards can play an important role in educating workers—and hence possibly reducing exposures.

Many depots use robots to perform repetitive and redundant painting operations, but no such robots have been developed for stripping paint. It remains a labor-intensive human task, and represents a major potential source for exposure. Workers who handle

Fig. 12-5. Any process that involves the burning of metallic lead is considered to be a high-risk operation. Steel or stick welding can generate high concentrations of lead fumes and place the worker at risk of lead poisoning. Source: US Army, Anniston Army Depot, Anniston, Ala.

waste material (putting the used, lead-contaminated abrasive into containers or cleaning the blasting booths) are also at risk for lead exposure. Furthermore, the spent blast material can pose an environmental hazard if not disposed of appropriately.

Welding

Welding, which creates 1,000°C–3,000°C temperatures, can effectively vaporize lead both at and near the point of welding.[11] The vapor is typically more respirable than the dust produced by abrasive blasting.[11] Thus, many welders who work with metallic lead or lead-coated materials may be at greater risk for lead intoxication than even abrasive blasters. At depots and shipyards, lead-based paint is stripped off material to allow for effective welding or to provide a clean, smooth surface for repainting or refurbishing (Figures 12-5 and 12-6).

Handling and Firing Munitions

Metallic and inorganic lead continue to be essential components of many modern munitions. For example, primers (the compounds that ignite the explosive sequence in a gun or mortar) often contain lead. Lead can also be a component of the shell, the bullet, and the propellant charge. Lead foil, which acts as a lubricant, is sometimes used to prevent copper deposits in large howitzers. As the weapon is fired, lead

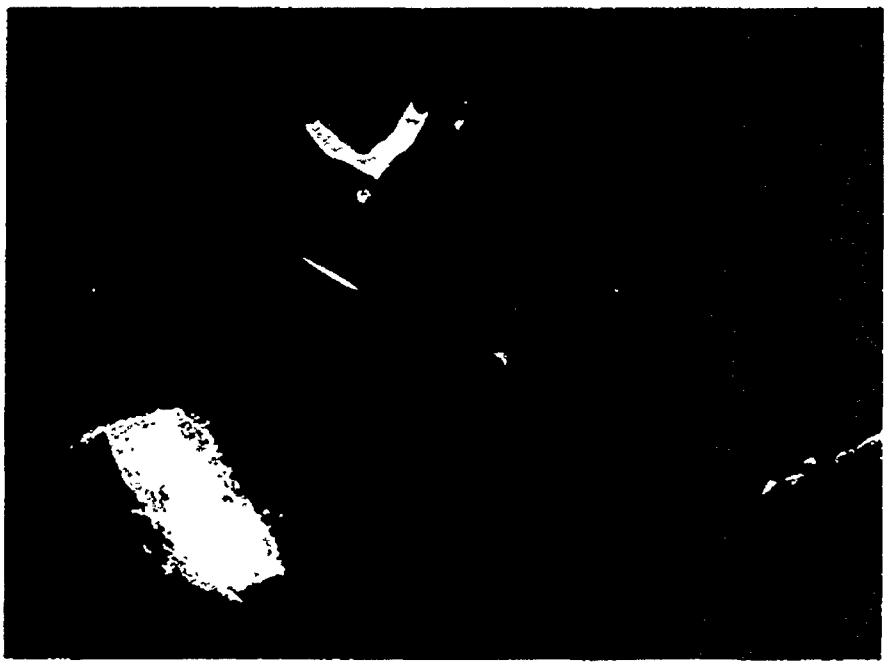

Fig. 12-6. Welding on lead-coated materials places the worker at risk of lead intoxication. Here, exposure may result from the vaporization of lead in paint that was incompletely removed from the surface before the welding was begun. Source: US Army, Anniston Army Depot, Anniston, Ala.

azide from the primer aerosolizes and forms a cloud of lead fume and dust near the breach of the weapon. Next, when the bullet hits a hard target, the bullet fragments and contaminates the area around the target with lead dust.[9] Outdoors, neither cloud poses a significant risk to the weapon firer; in most cases, natural ventilation will either blow away or dilute both clouds.

However, in indoor firing ranges and inside certain artillery and infantry vehicles with closed hatches, such ventilation and dilution often do not occur, and the potential for exposure to lead is significant.[15-17] Part of this problem has been due to the lack of planning in the design of indoor ranges; ventilation considerations are often afterthoughts.[17,18] Many ranges, located in buildings originally intended for other uses, have needed retrofitting of their ventilation systems, many of which had been improperly designed and have not always worked sufficiently well to reduce ambient lead levels.[19] The result is that not only the gunners but especially the cleaning crew are at risk for exposure to lead dust. Thus it is clear that, while at first glance indoor firing ranges may not appear to be sites of significant occupational exposure, they can be.

Soldiers involved in outdoor munitions training (such as tank or howitzer crews) and observers in close proximity to the firing operations may be significantly exposed to aerosolized lead (Figure 12-7).[17,18,20,21] Although these exposures may not be as constant or consistent as other occupational lead exposures, gunners may experience very high airborne levels for very brief periods of time. Gunners in training will not often be subjected to these levels for more than 30 days per year, and therefore do not require the lead surveillance mandated by the Occupational Safety and Health Administration (OSHA). Whether a military standard should be promulgated for these militarily unique exposures is a still-unresolved issue.

One further mechanism of exposure, lead poisoning from retained projectiles, has particular relevance for medical officers:

> Most retained projectiles are fragments made of iron. But given the large number of people who have retained projectiles that are partially or completely made of lead, the number of reported cases of lead poisoning caused by retained projectiles is surprisingly low. Nevertheless, lead poisoning does occur. Its clinical presentation can be quite pleomorphic and includes encephalopathy, anemia, neuropathy, and abdominal pain. Absorption of lead seems to be accelerated if the projectile is retained within a synovial space. Experimental studies indicate that lead concentration in the blood peaks within 4–6 months.[22,23,24]

Fig. 12-7. Soldiers involved in outdoor firing operations may be briefly exposed to high concentrations of aerosolized lead from the lead azide in the ammunition primer. Source: US Army, Anniston Army Depot, Anniston, Ala.

Electrical Soldering and Ballast Handling

Several U.S. Army depots employ electrical solderers. When lead-based solder is heated sufficiently to make electrical connections, small amounts of lead fume are produced. However, because the heat required to melt solder is relatively low, and the quantities of solder required to make electrical connections are small, the actual risk of lead exposure to solderers is relatively low—much lower than the risk to welders. Of course, the risk from electrical soldering is greatest where ventilation is limited, such as inside enclosed or confined spaces.

Ballast is typically bulk metallic lead. Ballast handlers, who place weight on ships and planes to improve their stability, can inhale lead dust that sloughs off. Lead dust can also be ingested. Others who work with bare metallic lead face similar hazards.

Although the risk of being exposed to lead dust from handling bulk metallic lead is not usually as high as the risk associated with inhaling lead fumes and vapors, the principles of occupational health must still be applied: first identify the risk; then control the exposure. The risk of potential exposure to lead can be defined by industrial health surveys. The controls in this instance are appropriate PPE and adequate ventilation. For workers known to be potentially exposed to lead, biological monitoring for blood lead, as part of a routine medical surveillance program, further reduces the probability for significant lead intoxication.

ENVIRONMENTAL EXPOSURES

Only since humans began to use lead has it become environmentally ubiquitous. But because lead is now found throughout the environment and in many manufactured goods in industrial societies, exposure is not confined to the occupational setting. Environmental lead can be found in contaminated air, water, food, soil, and other nonfood material (Figure 12-8).

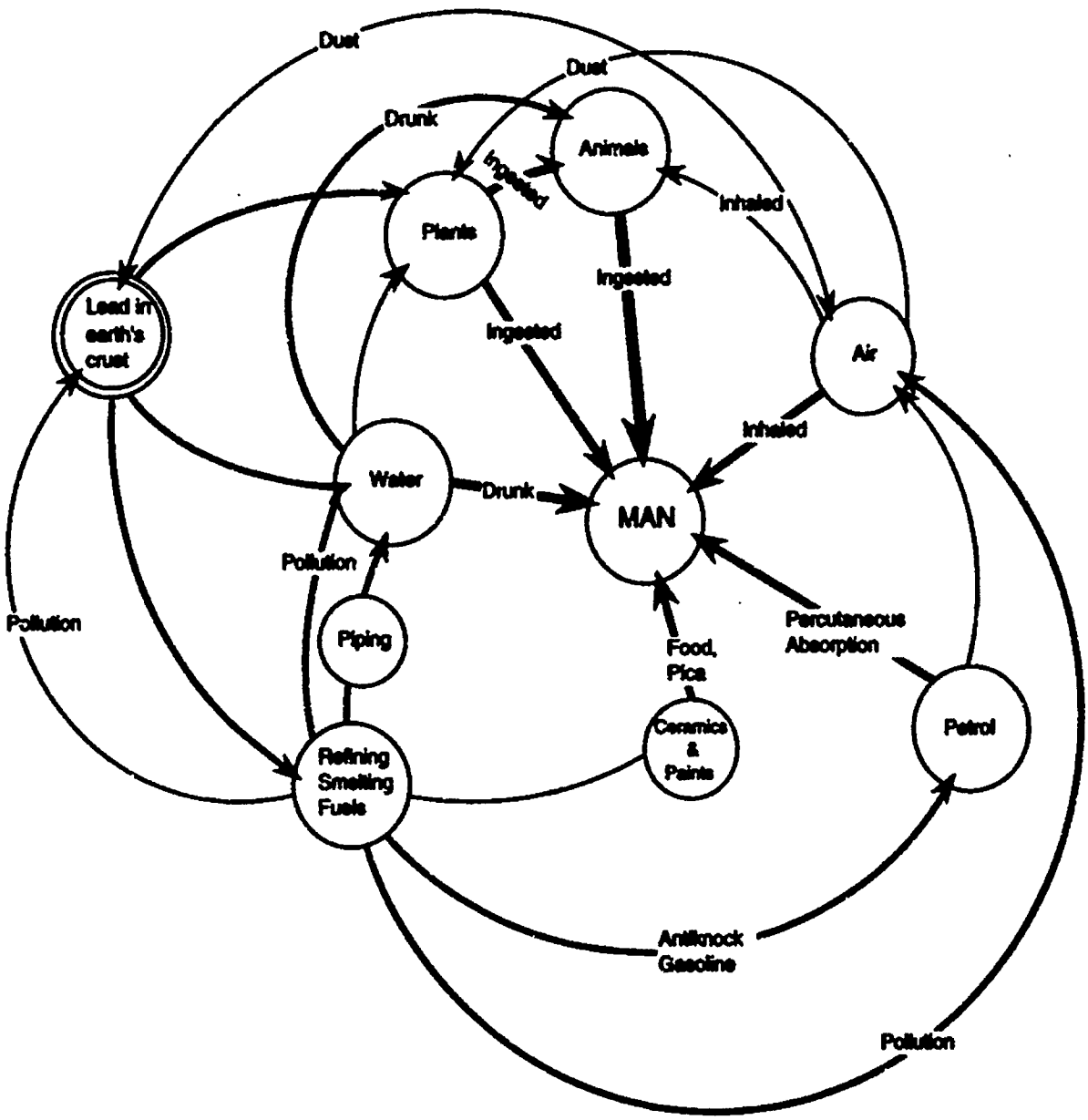

Fig. 12-8. Environmental routes of human lead exposure are indicated by arrows, with heavier arrows corresponding to more-significant exposure sources. For humans, a major route of lead exposure is ingestion of contaminated water and food. Other potential routes include exposure to lead-contaminated air, soil, and nonfood materials such as paint chips. Adapted with permission from Singhal RL, Thomas JA. *Lead Toxicity*. Baltimore: Urban and Schwartzberg; 1980; 87. © Williams & Wilkins.

Air

Urban dwellers, or those living or working near heavy traffic, face a greater risk of airborne lead exposure than rural dwellers.[23-25] Automobile exhaust from leaded gasoline is probably responsible for this finding. Fortunately, lead concentration in ambient air has decreased dramatically in recent years, primarily as a result of the reduction of lead in gasoline.[26] Smelting and mining operations can also produce substantial amounts of airborne lead, but generally only pose significant risks to nearby populations. In addition to automobile exhaust, smelting, and mining, ambient air can be contaminated by burning lead in welding or paint-stripping operations, and by grinding lead-based alloys.

Most inhabitants at military installations are at little or no risk from airborne lead pollution arising from on-post industrial activities. These activities generally do not produce large enough quantities of airborne lead to pose a significant hazard.

Water

Water can be contaminated with lead by (*a*) intentional or unintentional deposition or (*b*) being washed out of ambient air by rain. Water can unintentionally be contaminated when lead leaches from smelting, mining, and industrial wastes into groundwater and other bodies of water. Water can be polluted when industrial wastes are deliberately discharged into sewage systems as a convenient or inexpensive means of disposal. Lead can also be introduced into water as a contaminant by the very system that transports it: the plumbing. The likelihood of exposure is much greater in old plumbing systems, where lead-based solder was used. Although it is only slightly soluble in water under controlled conditions, factors in everyday life that tend to increase lead's solubility (and therefore increase its concentration) include electricity, heat, time, and acid pH. For example, lead concentrations in drinking water can be increased by using the plumbing system as an electrical ground; high ambient air or water temperatures; standing overnight (or longer, becoming stagnant) in pipes; and acidic pH of the water.[27]

Old plumbing systems were sometimes used as the electrical ground in indoor wiring. The electrical current pushing through will ionize lead from the pipes, which then dissolves in and contaminates the water. As a general rule, plumbing that contains lead should be replaced if increased lead is found in drinking water. If it is not feasible to replace the plumbing, then bottled water should be imported for human consumption.

Simply running water through the plumbing system to dilute the lead concentration can be an effective short-term solution. If high concentration of lead in drinking water is a community-wide problem, and if the water supply tends to be acidic, then neutralizing the pH of the water supply is useful. Despite the effectiveness of these temporary measures, the ultimate, long-term goal is to replace old, lead-contaminated plumbing with a new, safe system.

Ingested Food and Nonfood Material

Lead can contaminate food. The glaze on imported ceramic pottery sometimes contains lead; if the pottery is used to cook or serve food or drink, the glaze will be a source of ingested lead.[15] Food washed in lead-tainted water, or packaged in containers such as tin cans from which lead has leached, can also be contaminated. Before this problem was identified, the lead content of canned food could be as much as 10-fold greater than that of similar fresh food.[28] In the United States this problem has been avoided: food is no longer packaged in tin cans, but only in steel or aluminum cans, which are lead- and solder-free.

A common mechanism of environmental exposure, especially in children, is ingestion of contaminated soil. Adults who have frequent hand–mouth contact (eg, those who smoke or eat without first washing their hands) can also ingest lead in contaminated soil. The soil can be contaminated naturally (as rain washes the air), deliberately (as waste is dumped or discharged), or accidentally (through spills as lead-contaminated material is transported). Once in the soil, lead tends not to be easily mobilized; it remains near the surface, and usually near the source of contamination. Its physicochemical properties cause it to bind readily to anions (carbonates, sulfates, phosphates) and to complex with clay and other organic materials in the soil. If contaminated soil is disrupted, dust-borne lead can be aerosolized and inhaled. Whether the contamination was natural, intentional, or accidental, once contamination has occurred, elemental lead and all its compounds remain toxic. No nontoxic chemical modifications are available. The only way to eliminate the hazard is to physically remove the contaminated soil and dispose of it where human exposure is impossible or at least unlikely.

Children, in particular, are frequently exposed to lead via ingestion of paint chips:

> [A] single chip of paint of approximately 1 square centimeter surface area contains 1.5 to 3.0 mg lead (provided the chip initially contained one coat of paint which was 10% lead by weight). Since ingestion of 150 µg of lead in paint is already in excess of an

> individual's maximal permissible daily intake of metal, [and] .. children rather than adults exhibit a tendency to consume paper, paint chips, solder from cans and dirt.. it is not too surprising that lead tends to accumulate and induce toxicity.[p41]

Newly applied paint should be nonlead based, particularly in homes and child-care centers where children may come in contact with the painted surfaces. Specific areas of concern are surfaces within the children's easy reach, such as walls, windows, doors, and trim. These surfaces are subject to disrepair and may be significant loci of paint chipping, flaking, or dust-forming. Numerous reports of lead intoxication in children who have eaten paint chips stripped off older houses have been cited.[23,29-31] If lead abatement is not done with great care, exposure can be increased when inaccessible paint is ground into accessible dust. Even in houses where lead abatement has been carefully performed, the dust generated may still contain significant quantities of lead.

Pica, the physical craving for nonfood materials such as dirt and paint chips, is frequently found in children who have been exposed to lead. Ironically, the pica itself may be the underlying cause of lead intoxication. This is a chicken-and-egg cycle: pica induces the craving and the craving drives the pica.

PHARMACODYNAMICS

Paracelsus is reputed to have written that the dose makes the poison. This is most certainly true of lead. The quantity determines the ultimate toxicity. Environmental lead does not pose a human hazard per se. Only when a large enough concentration of lead in the blood is absorbed by the body and distributed to the appropriate organs can lead be considered toxic. A definite sequence of events occurs in lead intoxication (Figure 12-9). Lead toxicity presents as a broad spectrum of signs and symptoms, but not as a clear-cut syndrome. A list that purports to be comprehensive will probably mislead the student. Acute effects of inorganic lead poisoning may include colicky abdominal pain, constipation, encephalopathy, and renal failure. In chronic lead intoxication, clinical effects are very late events. Chronic effects may include fatigue, arthralgias, myalgias, peripheral neuropathy, anemia, renal failure, neurobehavioral disturbances, and encephalopathy. This chapter intends to describe lead intoxication not as a clinical entity but as the result of ineffective preventive and control measures.

To understand fully the significance of occupational and environmental exposures, we must also understand the routes of entry for the dose. Ingestion and inhalation are the common routes, especially in the occupational setting. Dermal exposures pose a substantial risk only when organic lead compounds are handled, as inorganic lead compounds tend not to penetrate the skin well. Therefore, this discussion is limited to ingestion and inhalation.

A general, although far from absolute, rule is that occupational exposures tend to be inhalational and environmental exposures tend to be ingestional, but there is considerable overlap. For example, a worker might contaminate his or her hands or clothing; then, by not washing the hands before smoking or eating, ingest the lead. By the same token, the general population inhales lead-contaminated automobile exhaust. The distinctions between occupational–inhalational and environmental–ingestional exposures to lead are arbitrary and physiologically indistinguishable. Lead dosing is additive regardless of exposure route or setting. Occupational medicine physicians must consider both routes of entry and both occupational and environmental sources. Individuals whose likelihood of environmental exposure is significant may require closer medical monitoring than would seem to be required if their only exposures were occupational.

Inhalation

Only a small portion of inhaled lead penetrates into the alveoli and becomes biologically available. The remainder is filtered out into the tracheobronchial tree and expectorated. The respirable fraction appears to depend on several variables, one being the aerodynamic diameter of the lead particulate. (Please see Chapter 4, Industrial Hygiene, for a discussion of particulates of vapors, mists, fumes, and aerosols.) The actual operational process that generates airborne lead plays a large role in determining its nature. Therefore, knowing the source of the airborne lead is essential to making an accurate assessment of risk. For example, welding done on surfaces that contain leaded paint will produce highly respirable vapors or fumes. Grinding produces an immense amount of dust but only a small portion of the total is actually respirable. Spray painting produces lead mists that can be variable in their respirability. Large particles normally remain in the nasopharynx, while smaller particles or fumes are most likely to enter the alveoli (particles < 0.5 µm are the most respirable). Many studies suggest that the aerodynamic diameter of the lead *particulate* in an aerosol plays a significant role in determining

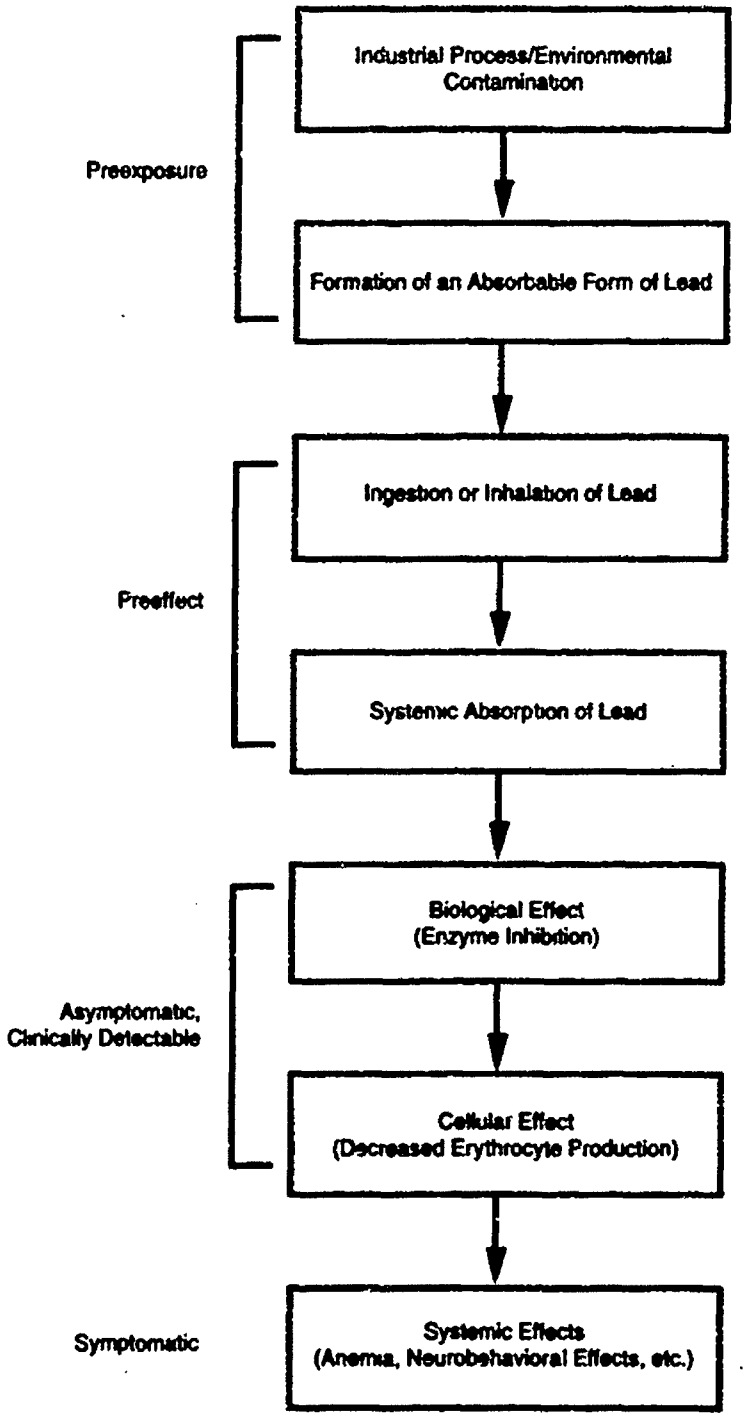

Fig. 12-9. Lead poisoning involves a definite sequence of events. In the preexposure period, absorbable lead is generated from environmental or industrial sources. After ingestion or inhalation, lead is systematically absorbed during the preeffect period. An asymptomatic period ensues, during which biological and cellular effects are clinically detectable. Lead toxicity culminates with a symptomatic phase in which systemic effects such as anemia or neurobehavioral changes predominate.

the resultant internal dose, but no study has yet provided a well-validated predictive model between aerosol particulate sizes and resultant blood lead levels.[32-34] Some studies using homogeneous aerosols of 0.5 μg aerodynamic size indicate that approximately 30% to 50% of the lead in an aerosol is ultimately absorbed (bioavailable).[33-36] The U.S. Army's own experimental data relating to the respirable fraction of howitzer-breech aerosol supports 30% as an estimate.[15]

Modeling for predicted lung deposition has not correlated well with experimental studies on humans. Establishing a predictive model for lung deposition and, ultimately, for resultant blood lead levels is difficult because so many factors can affect the outcome. For example, mucociliary clearance, depth of respiration, and variability in the particulate size of an aerosol can greatly influence respiratory deposition.[37] The direct correlation between airborne lead and blood lead may not exist. Attempts to develop predictive models have been made, with uncertain validity.[34] For example, one model predicts that for a homogeneous, respirable aerosol, approximately 1.0 to 2.0 μg of blood lead per mL of blood will result from a chronic exposure to 1.0 μg of lead in 1 m^3 of air.[34] However, we must be cautious when using experimental models relating the air concentration of respirable lead particulates to blood lead levels. Individual physiology and work activities, alternative routes of exposure, and other factors can produce great variation in blood lead for a given ambient-air lead concentration. This, among other reasons, is why lead intoxication is not a syndrome with a clear-cut list of signs and symptoms.

Ingestion

While no amount of lead ingestion is necessarily normal (for lead has no normal function in human physiology), the average daily intake of lead in the United States has been estimated at approximately 300 μg of lead per day. This value varies tremendously among individuals and groups, however, due to variations in

- the degree of plumbosolvency within the water distribution system,
- behavioral patterns of individuals (leading to greater likelihood of hand–mouth contact),
- the condition of lead-contaminated structures, and
- the total amount of lead in the environment.[35]

Some inhaled lead can be transported from the respiratory tract by ciliary action. This can then be swallowed, leading to gastrointestinal absorption.[38] Compared to respiratory absorption, however, gastrointestinal uptake is relatively poor. While the percentage of an inhaled dose of lead that is absorbed may be as high as 30% to 50% (depending on many variables including aerosol size), a typical adult will absorb only 10% of an oral dose.[36,39] However, there is an important exception to this general tendency: children seem to absorb a much higher proportion of ingested lead, perhaps as much as 50%. Thus, ingestion is often the most significant route of exposure in children.[10] This increased propensity for absorption is particularly ominous because children's developing nervous systems seem to be the most sensitive to lead's effects. Other groups also appear to absorb ingested lead more efficiently from ingestional sources: those who are pregnant, fasting, on a high-fat diet, and who have iron or calcium deficiencies.[10,40] The reasons why are not thoroughly understood, however.

A unique sign of lead exposure manifests as a bluish line on the gingiva. This manifestation, sometimes called the Burtonian Lead Line, results from precipitation of lead sulfide in the gingiva. Such a finding only indicates lead exposure and poor dental hygiene, and does not necessarily correlate with lead intoxication.

Absorption, Excretion, and Mobilization of Lead Stores

The quantity of lead and the period over which absorption has occurred play significant roles in toxicity. For example, a normal individual who ingests 2.5 mg of lead per day may take 4 years to reach a toxic blood level. But if the ingestion is just slightly larger, 3.5 mg per day, the human excretory mechanism is overwhelmed and a toxic blood lead level can be reached in a few months.[41] In acute exposures, where a large quantity of bioavailable lead enters the body in a short period of time, lead tends to be preferentially distributed to the soft tissues. Thus, the liver will often contain a large quantity of lead after an acute exposure. In more chronic and low-level exposures, lead has a proclivity to be deposited in the mineralized rather than the soft tissues. Thus, skeletal deposition takes on greater relative importance when absorption occurs over a period of time. In a *steady state* (ie, the amount absorbed equals the amount excreted), approximately 90% of the total body burden of lead is contained within the skeletal compartment.[13] Of the lead that remains in the blood pool, 99% is bound to the erythrocytes and the remaining 1% remains in the plasma.[10,42]

The skeletal pool can further be subdivided: one subgroup is relatively labile and passes readily into the blood circulation; the other subgroup appears to be more stabile (inert or slow to mobilize). Thus, the lead in these two subgroups is differentiated based on

accessibility for transport.[43] Another distinction is that lead within the labile pool is related to recent exposures, while lead within the stabile pool is related to prolonged exposures

The stabile pool can contain comparatively large amounts of lead, which can be mobilized during physiological stress (eg, changes in calcium balance, acid–base shifts, or bone trauma). Humans have a limited ability to excrete lead, however, and the rapid mobilization of lead stores can overwhelm the excretory capacity and cause lead intoxication. Lead is excreted primarily through urine and feces, although small amounts are also removed via nails and hair. Chronic absorption of more than 600 µg per day of lead will often result in a positive lead balance due to the inability to compensate via excretion.[41] Patients who have large stores may require months to years to mobilize and excrete sufficient lead before normal or relatively safe blood lead levels are achieved.

The body burden of potentially mobile lead can also be substantial: 200 mg or more.[43] Even in the absence of acute exposure, lead poisoning is possible if body stores are mobilized. For example, alcohol consumption probably mobilizes lead.[37] Anecdotal reports from the lead trades have often noted that workers experience symptoms of lead intoxication on Mondays, after a weekend of heavy drinking.[2] High metabolic states such as pregnancy and lactation can also accelerate lead mobilization. Lead can readily cross the placenta and be bioavailable to the fetus. It can also pass through the breast milk and be ingested by the nursing infant.[13,26]

PHYSIOLOGY

While metals such as copper and iron have physiological functions, and others such as magnesium and zinc act as catalysts, no normal physiological function or effect has been found for lead. Lead is toxic at a basic biochemical level; it can harm virtually every human organ system. Because lead is so active chemically, should it interact with an amino acid—in particular, with sulfhydryl groups (-SH), which are typically the active moieties on enzymes—the structure or function of an enzyme or other protein could be changed.

Through this type of action, lead blocks the synthesis of heme. Not only is heme an essential component of hemoglobin, it is also a component of cytochrome a_3, an intermediate in cellular metabolism. Therefore, inadequate heme production can alter cellular respiration and ultimately alter cellular function.

Hematological Effects

Anemia is a hallmark of lead intoxication. Lead-induced anemia is typically microcytic and hypochromic, but erythrocytes can also be normocytic and normochromic in the anemia's early stages.[13] Anemia is due to several factors, including (*a*) inhibition (restriction) of normal heme synthesis, (*b*) interference with the synthesis of globin, (*c*) interference with the incorporation of iron into erythrocyte precursors, and (*d*) shortened erythrocyte life span.

The biosynthesis of heme is catalyzed by enzymes, and lead probably interferes with normal enzymatic function. Lead probably affects at least two, and possibly four or more, enzymes in this pathway (Table 12-2 and Figure 12-10). The enzymes δ-aminolevulinic

TABLE 12-2

EFFECTS OF BLOOD LEAD LEVELS

Lowest Level at Which Effect Has Been Observed (µg/dL)	Physiological Effect	Population Affected
< 10	Erythrocyte ALAD inhibition	Adults, children
20–25	Elevation of FEP	Children
20–30	Elevation of FEP	Adult, female
25–35	Elevation of FEP	Adult, male
30–40	ATPase inhibited in erythrocytes	General
40	ALA excretion	Adults, children
40	Coporphyrinogen excretion	Adults

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acid dehydratase (ALAD), which catalyzes the synthesis of δ-aminolevulinic acid (ALA) into porphobilinogen, and ferrochelatase, which catalyzes the incorporation of iron into protoporphyrin IX, are the enzymes most affected by lead (the former being the more sensitive, but the latter being the rate-limiting step).[13]

ALAD inhibition may be evident in both adults and children at blood lead levels lower than 10 µg/dL. The result of this inhibition is an increase in the level of ALA and protoporphyrin IX. Although the level of ALA in both urine and blood can be measured, increased concentrations may not be measurable until blood lead has reached 40 µg/dl. Protoporphyrin IX also tends to accumulate in erythrocytes as a result of lead intoxication. One of the most commonly used laboratory diagnostic tests for assessing lead exposures is the measurement of free erythrocyte protoporphyrin (FEP, called "free" because the porphyrin is not bound to iron). Elevation of FEP is detectable when blood lead levels reach 20 to 35 µg/dL.

Because zinc is often present in the same cellular environment as the porphyrin, it will complex with protoporphyrin IX in erythrocytes, forming what is

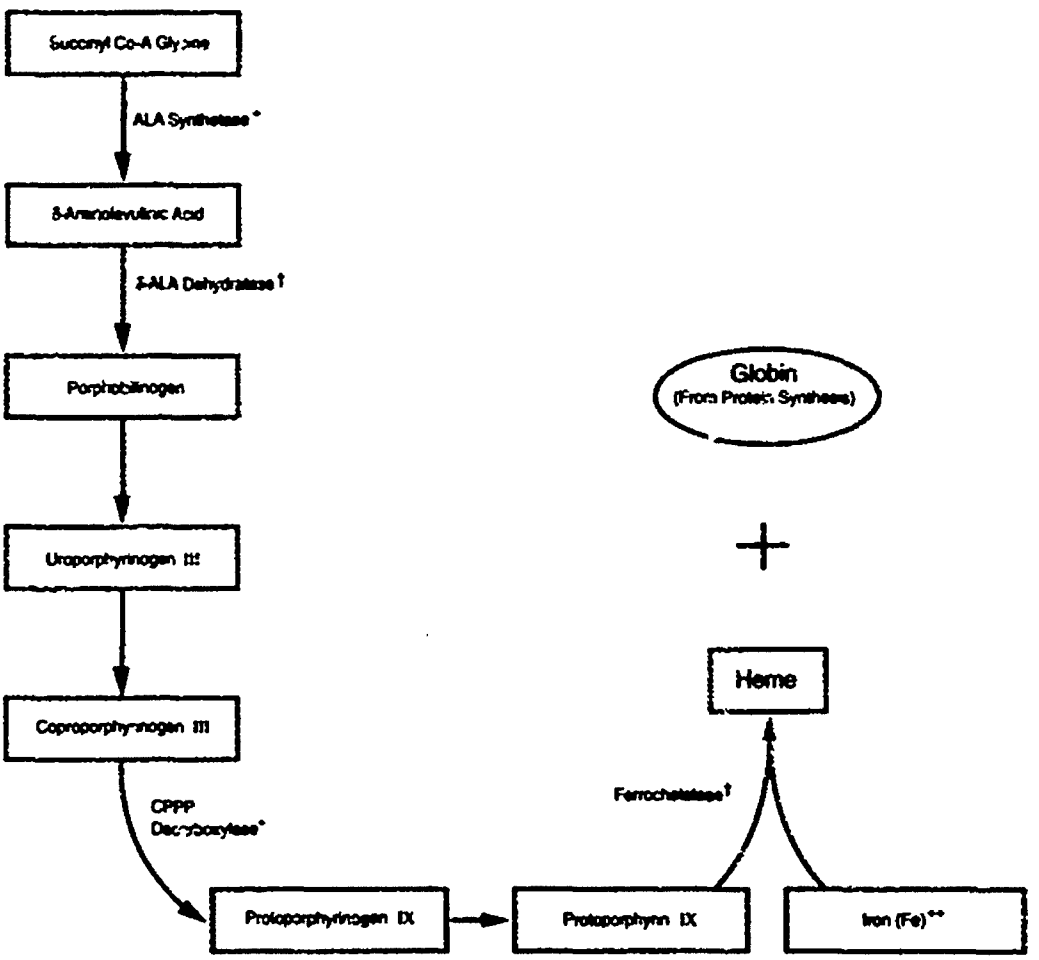

Fig. 12-10. The effects of lead on the biochemical pathway for the synthesis of hemoglobin. The synthesis of hemoglobin begins with the conversion of succinyl Co-A and glycine to aminolevulinic acid (ALA). This reaction is catalyzed by ALA synthetase, an enzyme that may be inhibited by lead. ALA dehydratase (ALAD), which converts ALA to porphobilinogen, is extremely sensitive to the effects of lead and is markedly inhibited in its presence. Activity of coproporphyrinogen (CPPP) decarboxylase, which catalyzes the conversion of coproporphyrinogen III to protoporphyrinogen IX, may be diminished. Finally, lead inhibits ferrochelatase, which catalyzes the incorporation of iron into protoporphyrin IX. This is the rate-limiting step in hemoglobin synthesis.

known as zinc protoporphyrin (ZPP). Some laboratories will assay for zinc protoporphyrin rather than FEP, but the tests measure the same phenomenon. Both the FEP and ZPP assays measure the *effect* lead has on heme synthesis rather than directly measuring the content of lead in the blood. Since the buildup of FEP is gradual, the FEP or ZPP assay is only useful as an index of a long-term (3–4 mo) response to lead.

Lead may also inhibit coproporphrinogen decarboxylase, which converts coproporphyrinogen III to protoporphyrinogen IX. This results in increased coproporphyrinogen excretion, which is evident when blood levels reach 40 µg/dL. Activity of ALA synthetase, an enzyme responsible for the conversion of succinyl Co-A and glycine to ALA, may also be reduced in the presence of lead.

Lead also interferes at two other points in hematopoiesis: with the protein synthesis of the globin moiety, and with the incorporation of iron into erythrocyte precursors. Formerly, basophilic stippling—a characteristic sign of lead exposure—was thought to be caused by small intracellular inclusions of iron, remnants of lead's interference with intracellular iron. Now, however, the basophilic stippling is thought to be the remnants of lead-induced intracellular organelle destruction during erythrocyte formation.[30] In erythrocyte precursors, lead interferes with the incorporation of iron into the hemoglobin molecule by causing ferrous iron to precipitate out of hemoglobin.

The anemia of lead poisoning results not only from interference with heme synthesis, but also from shortened erythrocyte life span. Although increased fragility of the cell membrane and inhibition of ATPase have been associated with a reduction in erythrocyte life span, the actual biochemical basis for this effect remains unknown.[33]

Neurological Effects

Lead's effects on the central and peripheral nervous systems (CNS and PNS) are at once profound and subtle (Exhibit 12-1). Although the profound effects have been known for years, only recently have we begun to appreciate the subtle effects and the level at which they start to appear. The fundamental reasons for the neurotoxicity of lead are not entirely known. What is clear is that lead can affect the neurological system in a number of basic ways, including reducing the availability of glucose in the cerebrum; altering the production and function of neurotransmitters; and, even more fundamentally, interfering with cellular respiration.[26,42,44]

The neurological sequelae of lead intoxication have significant occupational, and particularly military, implications. Researchers at the U.S. Army Medical Research and Development Command at Fort Detrick, Frederick, Maryland, have found mild but real problems in vigilance, visuospacial perception (hand–eye coordination), fine motor control, and memory at blood lead levels as low as 40 µg/dL.[15] These effects can cause definite performance decrements. Small decrements may not pose problems in many occupational settings; however, during critical or taxing situations, especially those a soldier faces during combat or realistic training, small decrements in performance could mean the difference between life and death.

Peripheral Nervous System

One of the most characteristic findings of severe lead intoxication (ie, blood lead level > 80 µg/dL), is peripheral neuropathy (lead palsy). This neuropathy can be sensory, motor, or both. Larger myelinated motor neurons (primarily of the extensor muscles) are generally affected most severely and produce the most predominant symptoms.[44] The tendency is for the motor neuropathy to produce symptoms referable to a single muscle group; then as the intoxication progresses, additional motor groups become involved.

EXHIBIT 12-1

CENTRAL NERVOUS SYSTEM EFFECTS OF LEAD EXPOSURE

Mild and Subtle Effects
- Restlessness
- Irritability or combativeness
- Decreased libido
- Memory impairment
- Visuospacial perception problems
- Short- and long-term memory losses
- Decreased ability to manipulate information
- Sleep disturbances
- Headache
- Decreased vigilance

Severe and Obvious Effects
- Delirium
- Ataxia
- Seizure activity
- Encephalopathy
- Coma

Numerous examples of occupationally related motor neuropathies due to lead have been reported, including painter's wrist drop, file cutter's paralysis, and laborer's foot drop.[5] On examination of a worker suspected to have lead intoxication, the physician may find that the patient complains of pain and paresthesias, weakness, atrophy, and fasciculations. An interesting feature of lead palsy is that these motor neuron deficits appear to affect preferentially the most frequently used motor neuron paths. Thus, right-handed painters have been noted to develop wrist drop of the right hand. The mechanism of this phenomenon is not understood, but may be related to increased blood flow to those heavily utilized motor groups and the concomitant increase in lead distribution to those neurons.

Perhaps related to many of these PNS effects, lead can exert a toxic effect on the supportive Schwann cells, resulting in their demyelination, axonal degeneration, and slowed nerve conduction.[44] Neuronal slowing typically occurs only after prolonged exposure, and usually after severe damage has already occurred.[14] In mild-to-moderate intoxications, the axon itself is not injured, but more severe intoxications produce axonopathy. The potential that the effects can be reversed is greatest when the axon has not been injured. If the axon is involved, prognosis is, at best, fair. The effects on nerve conduction are generally not apparent until blood lead levels exceed 40 to 50 µg/dL, and even then, slowing is subtle and not observed in all patients. Some slowing has been noted with blood lead concentrations as low as 30 µg/dL.[43] Nerve conduction has been suggested as a good indicator of early lead neurotoxicity.[44] However, as a screening test, nerve conduction lacks both sensitivity and specificity. Furthermore, many conditions other than lead intoxication can slow neuronal conduction. At the present, nerve-conduction studies are best suited to determining subtle neurological effects in large populations rather than in individuals.

Central Nervous System

The CNS effects of lead poisoning (including encephalopathy) are well known. While peripheral neuropathy is primarily a problem resulting from inorganic lead, CNS effects can be the result of exposure to organic lead as well. The ease with which organic lead passes through the blood-brain barrier probably potentiates the CNS effects of organic lead, and is particularly important in the toxicity of triethyl lead.[44] Pathologically, lead can induce cerebral edema, focal degeneration and necrosis of neurons, and cerebrovascular changes.[2]

The pathological and physiological changes in the CNS can result in a broad spectrum of effects from mild and subtle, to severe and obvious (see Exhibit 12-1). Gross encephalopathy is rare in adults at blood lead levels less than 120 µg/dL, but subtle effects may occur at blood lead levels as low as 25 to 30 µg/dL. While fulminant cases of neurological disease caused by lead are easy to recognize, the effects of the lower exposures can be quite difficult to detect. Gross clinical observation may not be sensitive enough to detect the subtle, gradual changes of mild lead intoxication. Serial psychometric and psychokinetic tests are necessary to document the effects of lead intoxication. This testing usually includes written and standardized batteries and specific tests for visual memory, visuomotor coordination, and reaction times. Testing with these components has demonstrated dose-related effects of lead on memory, hand-eye coordination, depression and other affective disorders, attention span, and reaction time.[45,46] In an occupational setting, the subtle effects of lead exposure can be assessed by obtaining a preemployment baseline of psychoneural function and subsequent serial, periodic testing. Unfortunately, these tests are imprecise, time consuming, and difficult to interpret, making psychometric testing impractical as a routine occupational-surveillance tool. However, they can be useful to help document and quantify progressive effects of low-level exposure in selected individuals, and are useful research tools.

Reproductive and Developmental Effects

Numerous investigations have reported that inorganic lead is toxic to both male and female reproductive systems as well as to the developing fetus.[47,48] Recent reports strongly suggest that inorganic lead levels once considered to be low and safe can induce significant reproductive and developmental toxicity.[10,26,48,49] In contrast, there is less evidence that organic lead is toxic to the reproductive system.

Lead's effects on the female reproductive system have been recognized for centuries. Clinical reports from the early portion of this century document that numerous female lead workers, and wives of male lead workers, had increased rates of spontaneous abortions and reproductive dysfunction.[2,50] Current thinking is that toxicity to the female reproductive system may start to occur perhaps as low as 30 µg/dL,[3] and that it manifests itself through a broad range of effects including menstrual disturbances, sterility, and higher rates of premature births and spontaneous abortions.[51]

Lead is also toxic to the male reproductive system, but the adverse effects tend to occur at higher blood

lead levels. Male reproductive effects may start to occur at blood lead levels of approximately 40 µg/dL, and are readily observable at blood lead levels of 60 µg/dL or more.[32] Specifically, adverse effects include abnormal sperm morphology, low sperm count, sterility, decreased libido, and impotence.[48,51]

Developmental effects are also of significant concern, especially the neurological systems' extreme sensitivity to lead. A fetus who is maternally exposed, and young children who live in environments where lead is readily accessible (or whose parents are occupationally exposed), are at high risk of adverse developmental effects. Lead exposure to pregnant females equates with fetal exposure: blood lead levels in the umbilicus appear to correspond closely with maternal blood lead levels.[53] An estimated 400,000 fetuses per year are potentially exposed to lead via maternal occupation.[10]

The American Academy of Pediatrics has stated that, even in grossly asymptomatic children, the neuropsychological effects of lead are largely irreversible.[54] Neurological development is maximally sensitive to the adverse effects of lead exposure at 3 to 6 weeks gestational age, although the fetus remains somewhat sensitive throughout pregnancy.[55] Many studies have reported a significant correlation between the umbilical cord or childhood blood lead levels, and subsequent decreases in the exposed population's average intelligent quotient or mental development index.[39,43,49,53,56] Significantly, this effect was noted for maternal exposures as low as 15 to 20 µg/dL. These exposures are one-half the current allowable blood lead levels for workers (30 µg/dL) that OSHA recommends to minimize the risk of adverse reproductive effects.[41] In addition to lead's effect on mental development, the effects of maternal lead exposure on the developing fetus may include growth retardation, malformations, and hyperactivity.[39,43,55]

In assessing the suitability of a pregnant worker for a job in which there is potential lead exposure, occupational health physicians must be aware that

- because lead is transferred effectively through the placenta and reaches nearly identical concentrations in the maternal and fetal circulations, any workplace exposures resulting in blood lead levels of more than 15 to 20 µg/dL could potentially harm a developing fetus; and
- the pregnancy itself may cause increased lead mobilization from body stores, increasing the maternal blood lead level and therefore the lead that is available for transport to the fetus.

Most experts now agree that women who are already pregnant and male and female workers who plan to have children require a greater level of protection than that currently afforded by law.[56]

A recent Supreme Court decision is particularly relevant. In the case of Johnson Controls *v.* the United Auto Workers, the issue was an employee's right to choose to stay in the job versus the employer's right to keep workers (in particular, female workers) out of areas known to be contaminated with chemicals known to be toxic to the reproductive system. Although the Johnson Controls case could be extrapolated to apply to any reproductive hazard, the one at issue was lead.[59]

Gastrointestinal Effects

The effects of lead on the gastrointestinal system are more symptomatic than functional: abdominal pain, constipation, loss of appetite, nausea and vomiting, and a metallic taste in the mouth. Gastrointestinal symptoms usually appear at blood lead levels exceeding 60 µg/dL. At blood lead levels greater than 100 µg/dL, classic lead colic may develop. Lead colic is characterized by the sudden onset of severe, paroxysmal abdominal pain. The underlying mechanism of colic is generally believed to be due to lead's direct toxic action on the smooth muscle of the small bowel.

Cardiovascular Effects

As early as the 1930s, researchers noted a correlation between hypertension and high-level, prolonged lead exposure.[60] These studies reported exposure levels that were quite high compared to those found in current occupational settings. More recent studies and analyses of the National Health and Nutrition Examination Survey II (NHANES II) data suggest that low levels of blood lead are associated with small changes in blood pressure.[43,60] In 1988, a researcher estimated that adult male systolic blood pressure increases from approximately 1.0 to 2.0 mm of mercury for every doubling of blood lead level.[61] The correlation, although statistically significant, may not be clinically relevant, however. Precisely how lead exerts its hypertensive effect is not known. A few mechanisms have been suggested as possible causes: direct action of lead on the arteriolar smooth muscle, alteration of the renin-angiotensin system, or change in intracellular calcium balance. Certainly the possibility exists that lead may induce hypertension as a result of its nephrotoxic effect, although this effect would probably not be apparent until kidney function is notice-

ably affected. Degeneration of cardiac muscle and electrocardiographic changes have been noted from lead as well.[43]

Renal Effects

Lead is toxic to the kidneys. Proximal renal tubular dysfunction has been reported as a result of chronic exposure, leading to aminoaciduria, phosphaturia, glucosuria, and hyperphosphaturia (Fanconi-like syndrome). Hyperuricemia has also been reported. Excess retention of uric acid as a consequence of lead exposure may produce saturnine gout. Prolonged exposure can cause interstitial fibrosis, tubular atrophy, and glomerular destruction.[10]

Renal disease probably does not occur without very large, chronic doses of lead. Most effects of short-term exposure on the kidney are reversible. The likelihood of irreversibility increases with the length and degree of exposure. Early in the course of occupational lead exposure, renal-function tests probably will not show any abnormality. Typically, up to 50% of renal function must be lost before renal-function tests show changes. Thus, renal-function tests tend to be poor indicators of exposure to low levels of lead.[41]

Other Effects

Other possible effects of lead exposure include interference with the function of vitamin D and the development of cancer. Studies have demonstrated that interference with vitamin D function can affect growth and development, immunological response, and bone structure. Various researchers have suggested that lead may be a carcinogen; however, this research has yet to produce definitive proof. Advisory and regulatory agencies are mixed in their assessment of lead as a carcinogen. The International Agency for Research on Cancer (IARC) does not recognize lead as a human carcinogen, but based on studies with animals, the Environmental Protection Agency (EPA) has designated lead as a probable human carcinogen (class B2). Studies with animals have demonstrated that lead causes renal cancer in rodents, yet retrospective human epidemiological studies have been inconclusive. Studies involving lead workers have not consistently documented an increased incidence for any particular anatomic sites of cancer. Some studies did indicate a slight excess of renal, lung, and stomach cancers, although these studies' conclusions may have been confounded by other exposures.[42]

OCCUPATIONAL SURVEILLANCE

Several laboratory modalities are available to assist in assessing the received dose from an exposure and the effects of an exposure, as mentioned previously. The most frequently utilized are (*a*) blood lead level measurements, which measure lead directly; (*b*) measurements of ZPP and FEP, which quantitatively measure the effect of lead on the synthesis of hemoglobin; and (*c*) measurements of ALAD, the activity of which is inversely related to the blood lead level.

Measurement of Blood Lead Level

Of the many laboratory modalities used to assess lead exposure, the measurement of blood lead levels is often considered a cornerstone of a lead surveillance program. The blood lead level measurement demonstrates the amount of lead present in the blood compartment, and this measurement is usually a good indicator of recent exposure to lead. However, elevated blood lead levels can result from mobilized lead stores from skeletal tissues as well, and therefore relate to past exposure. Blood lead measurements do not adequately quantify intermittent or past exposure because the lead in the blood compartment turns over relatively rapidly, with a half-life of 35 days.

An important goal of monitoring the blood lead levels of employees is to ensure that blood lead levels remain below 40 µg/dL of blood in any given individual. To obtain an accurate assessment, the blood lead specimen should be collected in a heparinized container at the end of the work shift and sent to an approved laboratory for analysis. Once a reliable and accurate result is obtained, conclusions can be drawn about lead exposure in that worker. Most people in the general population have blood lead levels of 5 to 15 µg/dL.[40] A blood lead level elevated over the baseline can be a sentinel event, indicating that workplace exposure controls are less than adequate or that previously absorbed lead is being mobilized.

Measurements of Free Erythrocyte Protoporphyrin and Zinc Protoporphyrin

Unlike blood lead level measurements that actually demonstrate the concentration of lead, measurements of FEP and ZPP demonstrate lead's effect on hemoglobin synthesis. These levels are direct, quantitative

measurements of erythrocyte porphyrins in the blood. Excess porphyrins are formed when ferrochelatase, an enzyme responsible for incorporating iron into the porphyrin molecule to form heme, is blocked. Because hemoglobin is produced only in the bone marrow by *maturing* erythrocytes, lead has no effect on the erythrocytes already circulating at the time of exposure. Thus, when FEP and ZPP are measured in a peripheral blood sample, it is the effect that lead *has had* on erythrocyte development during the preceding 4 to 6 months (the normal life span of erythrocytes in the peripheral circulation) that is being measured.[1] Therefore, measuring FEP or ZPP does not replace the need to measure blood lead directly. Rather, these indirect tests provide a useful adjunct to direct blood lead level measurements. The FEP and ZPP measurements are not specific for lead intoxication. Any condition that results in the accumulation of protoporphyrin IX (eg, anemia, iron deficiency, and excess production or concentration, or both, of other prophyrins such as bilirubin, urobilinogen, and riboflavin) will result in the elevation of ZPP and FEP.

Assays for ZPP and FEP are often considered to be interchangeable, but they are not. Subtle and significant differences exist between them: FEP measures free (uncomplexed) porphyrins; ZPP measures porphyrin that has complexed with the zinc normally present in the blood. ZPP is measured using a hematofluorometer, and the erythrocyte porphyrin level is estimated from a calibration standard, which is often based on an average hematocrit for the population—children or adults—being tested. FEP, however, is measured using a much more accurate extraction method, and is a direct quantification of porphyrin. There is more variation between individuals in the measurement of ZPP compared with FEP, unless the hematofluorometer is calibrated individually for each person's hematocrit. The ZPP estimate is subject to greater error in measurement, but it is an easier, faster laboratory assay, and because an estimate is usually adequate, it is often preferred.[43]

These protoporphyrin tests are often used in conjunction with the blood lead level measurement to assess both the severity and the nature of the exposure. High levels of FEP or ZPP correlate well with lead exposure. Normal levels of ZPP should be 25 µg/dL or lower.[44] Lower levels of blood lead do not correlate well with levels of FEP or ZPP. For example, FEP and ZPP will not be increased at blood lead levels lower than 20 to 25 µg/dL. This insensitivity has significant implications for screening programs: tests for FEP and ZPP will not be useful in identifying low-level exposures to lead.

Measurements of δ-Aminolevulinic Acid Dehydratase Activity and δ-Aminolevulinic Acid in Urine

The most sensitive test currently available to detect the early effects of lead intoxication is the screening of ALAD activity. This enzyme's activity is inhibited as the blood lead level rises. As with FEP, measuring ALAD shows an effect that blood lead has caused over the past few months. Whether ALAD activity can be used in occupational surveillance is unclear. Measuring FEP is important to help document a true health effect (impaired hematopoiesis), whereas ALAD activity documents only a biochemical effect, the clinical significance of which is still under study. For example, in some cases, no deficit of hematopoiesis is noted at moderate blood lead concentrations (< 40 µg/dL), yet, ALAD activity is almost completely inhibited.[34] Furthermore, the test for ALAD activity is not yet widely available, which limits its utility in occupational surveillance.[40]

Lead and ALA concentration in the urine (ALA-U) have also been suggested as possible modes of surveillance. Urine-based assays are noninvasive, less expensive, and more convenient than blood-based assays. Although some studies have suggested that exposure to stable air concentrations of lead results in stable urinary excretion, most occupational exposures are variable and unstable.[43] Furthermore, there can be considerable individual variation in renal function, again resulting in uninterpretable laboratory values. Thus, the use of monitoring the urinary concentration of either ALA or lead as a medical surveillance tool is usually of questionable value. The best practical use for these urine-based tests is in monitoring progress during chelation therapy.

ALA-U tends to increase exponentially once the blood lead level exceeds approximately 40 µg/dL; therefore, it is sometimes used as an indicator of lead's effect on the hematopoietic system. At blood lead levels lower than 30 µg/dL, only small elevations in the ALA-U are noted. Relative to FEP, however, ALA-U determinations have these limitations:

- they tend not to be as useful for detecting sub-OSHA regulated blood lead levels,[34] and
- ALA-U tends to drop off quickly once exposure ceases.[45]

Lead content in hair has also been proposed as a useful indicator of exposure. However, technical difficulties in analysis and standardization prevent the consistency necessary for a good screening or diagnostic test.[34]

TREATMENT

When preventive measures fail and blood lead levels are exceedingly high, clinical therapy including (*a*) treatment of an acute poisoning, (*b*) supportive care, and (*c*) chelation therapy may be necessary.

The treatment of an acute poisoning and the subsequent supportive care are necessary for short-term ingestion of 0.5 g of lead. In adults, death can occur within 1 or 2 days after ingestion of 10 to 30 g.[35] Blood lead levels higher than 110 µg/dL have been fatal in children, but adults with levels twice this high have survived. Treatment for acute poisoning in known ingestional exposures includes gastric lavage to remove the lead, and hydration to minimize the toxic effects on the kidney. Subsequent supportive care may be indicated to treat symptomatology (such as abdominal pain), monitor electrolytes, and deal with complications (such as liver or kidney failure).

Chelation therapy (which chemically removes metallic ions from participation in biological reactions by causing the metal to bind to a complex ring; in heme, the porphyrin ring normally chelates the ferrous ion) can be useful for severely poisoned patients, whether the poisoning is acute or chronic. Chelation therapy is used when simple removal from further exposure will not reduce blood lead levels to an acceptable degree in an acceptable period of time, or when body stores of lead are large and would probably cause intoxication when mobilized. However, the decision to administer chelation therapy is not without risk. For example, the most frequently used chelating agent, calcium disodium edetate (Ca-EDTA), can cause zinc depletion and acute renal tubular necrosis if used improperly.

Some researchers suggest that chelation therapy be administered when blood lead levels reach 80 µg/dL,[39,52] while others favor initiating therapy at even lower levels (perhaps 70 µg/dL), particularly in children.[31] The most rational advice when approaching chelation therapy is that

- the treating physician must have adequate experience in the procedures, and
- the therapy must be based on clinical findings as well as on the blood lead level.

Chelation prophylaxis is *never* appropriate as a preventive measure for lead workers and such use is specifically prohibited by law.[12]

The Ca-EDTA lead mobilization test is a useful procedure that can help to determine the extent of body stores and whether chelation therapy may be indicated. (Other tests are currently being developed.) The mobilization test utilizes a bolus of Ca-EDTA to mobilize stored lead, and then measures the amount of lead that is excreted via the urine. Although the dose of Ca-EDTA has not been standardized, 30 mg/kg has been suggested as the recommended bolus. Many clinicians who perform this test use a bolus of 1 g. The test is considered positive and indicative of dangerous levels of lead stores if more than 600 µg of lead in 24 hours is chelated and excreted.[40,52,66] In cases of renal impairment, an excretion of more than 600 µg of lead in 72 hours is considered positive.

The chelating agent of choice is not always Ca-EDTA. It has proven to be beneficial in high-level poisonings, although no study has yet definitively indicated a benefit from chelation in asymptomatic individuals who are mildly intoxicated. Currently, some debate exists on the administration of chelation therapy for low blood lead levels in asymptomatic children. This debate centers around the indications that, in young children (< 6), even low blood lead levels can cause delayed CNS development. D-Penicillamine and British anti-Lewisite (BAL) have also been used to chelate lead. These agents are usually considered to be second choices because of their high potential toxicities. Ongoing research suggests that a number of candidate substances, such as 2,3-dimercaptosuccinic acid (DMSA), show promise as effective lead chelators.

PREVENTION AND CONTROL

Prevention and control measures must be implemented together to curtail worker exposure adequately. Control measures involve several general industrial hygiene practices such as engineering controls and PPE; however, prevention methods can be divided specifically into (*a*) primary prevention, which implies that exposure and ill effects are completely avoided, (*b*) secondary prevention, which implies an early intervention to limit the ill effects of exposure, and (*c*) tertiary prevention, which is a therapeutic or a rehabilitative action.

Standards and Regulations

Occupational and environmental standards encom-

pass elements of all three preventive strategies. As the potential hazards of lead became known, regulation and legislation to control exposure have become more commonplace. For example, the government of Great Britain developed comprehensive rules and regulations pertaining to occupational lead exposures as early as the late 19th century. In the United States, the Commonwealth of Massachusetts enacted a law in 1723 that banned the use of lead-containing materials in rum-distillation equipment.[67] Still, widespread regulation in the United States did not appear until the mid- to late 20th century, and federal regulations did not appear until the 1970s. These regulations, administered by several federal agencies, cover a number of settings for lead exposure (Table 12-3). The U.S. Army complies with all federal occupational and environmental regulations.

During this century, the worldwide trend has been downward for what are considered to be safe or acceptable blood and air levels, resulting in tighter regulation. For example, early in this century, the occupational standard for lead concentration in air was generally 500 $\mu g/m^3$,[68] it dropped to 200 $\mu g/m^3$,[69] and is now 50 $\mu g/m^3$.[70] (Interestingly, the normally more conservative Amer-ican Conference of Governmental Industrial Hygienists [ACGIH] has a recommended Threshold Limit Value [TLV] of 200 $\mu g/m^3$, which is higher than the maximum level set by OSHA regulation.[70])

As with air lead levels, blood lead levels in the 60 to 80 $\mu g/dL$ range were once considered to be generally safe.[68] The currently recognized "safe" level is also coming under a great deal of suspicion. It is now fairly well accepted that the 40 $\mu g/dL$ level is probably not safe for reproductive adults and young children.[11] In addition, under the 1974 Safe Drinking Water Act, the EPA attempted to ensure the safety of public water systems by establishing and regulating the maximum contaminant levels (MCL) for each contaminant of concern.

TABLE 12-3

SUMMARY OF LEAD STANDARDS AND REGULATIONS

Regulatory Agency	Subject of Regulation	Regulated Exposure Level	Comments
CDC	General population	25 μg lead/dL blood	Advisory level for maximum "safe" blood lead in children
FDA	General population	100 μg lead/day	Recommended maximum permitted intake via food (primarily for children ages 1–5 years)
ACGIH	Workplace air	150 μg lead/m^3 air	TLV is 3-fold > OSHA PEL
NIOSH	Workplace air	100 μg lead/m^3 air	Recommended exposure air is 2-fold > OSHA PEL
OSHA	Worker blood lead	60 μg lead/dL blood	Necessitates medical removal from job
OSHA	Worker blood lead	40 μg lead/dL blood	Necessitates mandatory detailed medical examination
OSHA	Workplace air	50 μg lead/m^3 air	PEL, 8-h TWA*
OSHA	Workplace air	30 μg lead/m^3 air	Action level, 8-h TWA
EPA	General ambient air	1.5 μg lead/m^3 air	Averaged over a calendar quarter
EPA	Drinking water	50 μg lead/L water	Enforced drinking water standard or MCL†; 5 μg/L is the proposed MCL
EPA	Drinking water	0 ppm	MCL goal (what EPA considers safe, regardless of technically achievable attainment)
CPSC‡	Paint	0.06% (600 ppm)	Maximum % (dry wt) in newly purchased or applied paint

*Time-weighted average
†Maximum containment level
‡Consumer Products Safety Commission
Source: US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. Lead toxicity. In: *Current Alert: Case Studies in Environmental Medicine*. Atlanta, Ga: USDHHS; June 1990: 17.

Occupational Regulations

Work-related lead exposure is regulated by Title 29 Code of Federal Regulations (CFR), Part 1910 § 1025.[12] Specifically, this regulation applies to all occupational lead exposures, excluding certain construction and agricultural situations that are regulated by 29 CFR 1928.[11] The construction standard is less stringent and less relevant to military lead exposures, and therefore will not be discussed further. The occupational regulation, 29 CFR 1910, hereinafter called the OSHA standard, covers exposures to metallic lead, all forms of inorganic lead, and organic lead in soaps. However, this regulation specifically excludes exposure to all other organic lead compounds.

The OSHA standard distinguishes between workers who are exposed to lead for 30 or more days in a given year, and workers who are exposed to lead for fewer than 30 days per year. This distinction is the basis for the requirements for the maximum permissible airborne lead levels and the administration of medical monitoring. Although there are many components of the OSHA lead standard, the most important categories of requirements are for air monitoring, medical monitoring, personal protection, employee notification, and employee training (Table 12-4).

TABLE 12-4

COMPONENTS OF THE OSHA LEAD STANDARD

Specific Component	Corresponding Paragraph
Permissible exposure limits	C
Exposure monitoring	D
Methods of compliance	E
Respiratory protection	F
Other protective equipment	G
Housekeeping	H
Hygiene facilities and practices	I
Medical surveillance	J
Medical removal protection	K
Employee information and training	L
Recordkeeping	N
Monitoring and observation	O

Source: 29 CFR, Part 1910 § 1025.

Air Monitoring

OSHA considers that, under most working conditions, airborne lead levels correlate well with employees' blood lead levels. Therefore, air monitoring provides the foundation for implementing the OSHA standard. The important values established by the current regulation are

- the *action level*, which OSHA defines as 30 μg/m^3, an 8-hour *time-weighted average* (TWA, the average exposure that would occur if employees were exposed to a given level of lead for a normal 8-h workday), and
- the maximum permissible exposure level (PEL), which OSHA defines as 50 μg/m^3 TWA.

If employees are exposed to air concentrations exceeding the action level, employers must initiate control measures or preventive strategies to limit exposure. These control measures may deal with exposure monitoring, medical surveillance, and training or education or both. The PEL is the average 8-hour airborne concentration, which may not be exceeded under most circumstances.

The OSHA standard requires an initial assessment to determine whether employees are exposed to airborne lead concentrations at or exceeding the action level. The standard stipulates that all employers must conduct this initial air monitoring if any of the following conditions is met:

- information, observation, or calculations indicate that employees are, or could be, exposed to lead;
- prior monitoring of airborne lead indicates possible lead exposure; or
- any employee complains of symptoms attributable to or indicative of lead exposure.

OSHA mandates the frequency of air sampling, based on the results of the initial air monitoring. If the initial air monitoring indicates that no employee is exposed to lead levels at or exceeding the action level, then the employer is required only to document the determination. The documentation must indicate the results of the air sampling, specific conditions of the sampling, (eg, the names of the individuals who conducted the air monitoring, the testing dates, and the equipment used), and the individuals (or the location) for whom the air sampling is valid. If the initial monitoring indicates that a lead exposure between the action level and the PEL is occurring, the standard requires that air monitoring be repeated every 6

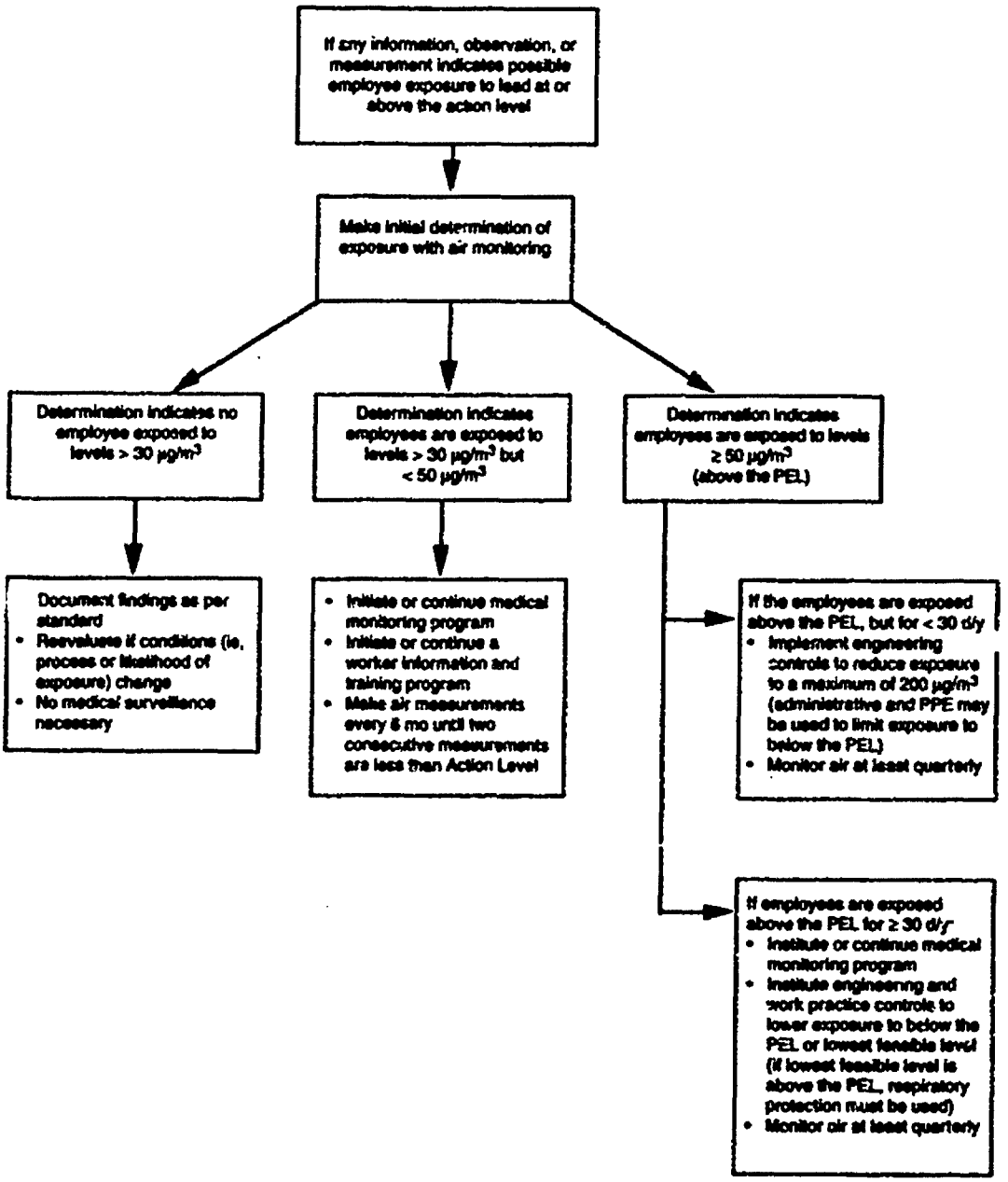

Fig. 12-11. These actions are required by 29 CFR, Part 1910 § 1025, the Occupational Safety and Health Administration's lead standard.

months, until two consecutive measurements (taken 7 d apart) demonstrate airborne lead levels to be below the action level. Finally, if initial air monitoring demonstrates exposures exceeding the PEL, quarterly air monitoring is required until two consecutive measurements demonstrate airborne lead levels below the PEL. In addition, air monitoring is required if any change in process occurs that could result in new, different, or additional lead exposure.

When initial or subsequent air-monitoring results indicate that airborne lead levels exceeding the PEL are occurring, extensive measures to limit employee exposure to below the limit, or to the lowest levels feasible, must be enacted (Figure 12-11). Where it is not possible or feasible to reduce exposures to below the PEL, then existing controls must be supplemented with respiratory protection and a respirator program. The one exception to this rule is when employees are exposed to airborne lead levels exceeding the PEL for 30 days or less per year. In this case, engineering

control measures are required to limit exposure to only 200 $\mu g/m^3$, and a combination of administrative and personal protection means can be used to effect the remaining reduction to less than 50 $\mu g/m^3$. The policies and operating procedures that detail the ways in which an employer controls lead exposure are frequently termed a *compliance program.* Compliance programs must be documented in writing and updated biannually.

Medical Surveillance

The OSHA standard requires employers to make medical surveillance benefits available, which the OSHA standard requires be provided under the supervision of a licensed physician and without cost to the employees, (*a*) to all workers who are exposed to lead at or above the action level for more than 30 days per year, (*b*) prior to assignment in areas where concentrations are above the action level, or (*c*) whenever there is a medical need for the examination based on worker complaints or medical suspicion of exposure. Employer participation is mandatory, but each employee decides whether to take advantage of these benefits (Exhibit 12-2).

The OSHA standard dictates that the maximum allowable blood lead levels in working adults is 40 µg/dL. If medical surveillance indicates that an employee has a blood lead level greater than this, the frequency of that employee's blood lead monitoring increases from once every 6 months to once every 2 months. The blood lead monitoring must continue until two consecutive blood lead measurements are 40 µg/dL. If monitoring indicates that the blood lead level is higher than 40 µg/dL, the worker must be notified of these results within 5 days. Although the OSHA standard itself and the frequency of blood lead monitoring are based on a 40 µg/dL limit, OSHA recommends a permissible maximum blood lead level of 30 µg/dL for those employees (male and female) who wish to have children.

The OSHA standard provides that occupational health physicians have discretionary power: they are allowed to set more stringent (conservative) criteria than those the OSHA standard defines for removing workers from exposure. For example, the OSHA standard mandates that employees with blood lead levels higher than 60 µg/dL, or those whose average blood lead level is higher than 50 µg/dL for three consecutive measurements, be removed from exposure until their blood lead levels drop below 40 µg/dL. However, regardless of the blood lead level findings, an occupational health physician may recommend that employees be removed from exposure if their symptoms demonstrate adverse effects from lead. If an employee is medically removed from exposure, then the OSHA standard requires that blood lead level determinations be performed every month while the employee is

EXHIBIT 12-2

OSHA-MANDATED MEDICAL SURVEILLANCE REQUIREMENTS

Determinations of blood lead level and zinc protoporphyrin levels every 6 months
Compilation of a detailed work history with attention to past lead exposures
Compilation of a habits history (smoking, drinking, pica)
Compilation of a detailed medical history (to identify potential risk factors and adverse effects associated with neurological, cardiovascular, renal, hematological, reproductive, and gastrointestinal systems)
A physical examination, with attention to the same systems mentioned above, as well as the respiratory system if respiratory protection is used
Measurement of blood pressure
Measurement of hemoglobin and hematocrit
Measurement of erythrocyte indices (including a review of the peripheral smear)
Measurement of serum creatinine
Urinalysis, including a microscopic examination
Pregnancy testing or laboratory evaluation of male fertility

removed from the worksite. Additionally, whether an employee is removed from exposure due to excessive blood lead levels or upon a physician's recommendation, the OSHA standard states that no adverse personnel actions may result from such a removal for up to 18 months (ie, the employee cannot be fired for any reason during that time).

Personal Protection and Hygiene

The OSHA standard requires that employers provide potentially exposed employees with the necessary PPE, such as coveralls and gloves, at no cost to the employee. Laundering work clothes and maintaining the cleanliness of the worksite are also the employer's responsibilities. Dry sweeping of lead dust is prohibited; it must be vacuumed up or washed down. Sometimes vacuuming with a high-efficiency particulate air (HEPA) filter may be necessary to remove lead dust. The employer must also provide clean eating, lavatory, and washing facilities.

Training and Notification

The OSHA standard holds employers responsible for informing their employees of the existence and content of the regulation, and to notify employees of their test results. The purpose for this requirement is to keep employees as informed as possible about their exposure to lead, the health implications of exposure to lead, and the protection and control practices available to them. Specifically, training must include information concerning the effects of lead, the engineering and other control measures used to deal with the hazard, and the purpose and details of the medical surveillance portion of the regulation.

Environmental Regulations

Military preventive medicine specialists who practice occupational health often are responsible for the environmental health of those who work at the installation. This responsibility can include ensuring that the post's water-distribution system is safe; that post housing, day-care centers, and other facilities are lead free and safe for habitation; and that hazardous waste is disposed of appropriately. These physicians must be cognizant of the regulations and standards that involve both environmental and occupational sources of lead exposure.

Regulations pertaining to environmental lead tend to be less specific for individuals than regulations pertaining to occupational exposure. Provisions for limiting lead exposure are contained within the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA), the Safe Drinking Water Act, the Clean Air Act, and other federal laws. These regulations cover exposure to lead from many sources such as drinking water, air, food, and consumer items such as paint. Still other regulations cover the use or disposal of lead products but a detailed discussion of all environmental regulations is beyond the scope of this chapter.

Drinking Water

The EPA's established limit of lead in drinking water—not greater than 50 µg/L—is the maximum level of contaminant for drinking water from a water-distribution system. The EPA has also proposed an ultimate goal of zero lead in drinking water and EPA regulations state that lead solder will not be used in plumbing joints. Furthermore, the public must be notified if drinking water is contaminated with lead as a result of either lead in the plumbing system or water sufficiently corrosive to cause lead to leach.

Ambient Air

The Clean Air Act regulates the level of lead in ambient air. The substantial decrease of airborne lead in recent years has been a direct result of the use of unleaded gasoline, as this regulation has required. Currently, the standard permits no more than 1.5 µg of lead per m^3 of air. The reduction of air pollution will have subsequent impact on soil and water contamination as well. Recent amendments to the original Clean Air legislation have made some administrative changes on how the air concentration is calculated and what comprises an acceptable level of air discharge.

Food

The Food and Drug Administration (FDA) has also promulgated a maximum allowable daily ingestion of 100 µg of lead from foods. The FDA has concentrated some attention on lowering lead content in canned foods, and controlling the entry of food utensils (pottery) and pesticides into this country.[26]

Paint

Lead within paint has been regulated by the Consumer Products Safety Commission to a maximum of 0.06% net weight.[11] Furthermore, the Department of Housing and Urban Development has compiled guide-

lines on lead abatement in older homes. Some of this guidance has military relevance: most installation housing and buildings now used for child-care centers are old and contain layers of lead-based paint.

Prevention and Control

Without doubt, the most effective means of preventing lead exposure is primary prevention, which includes (*a*) avoiding lead completely by substituting a less-toxic substance and (*b*) separating the worker from the lead (both spatially and temporally). These efforts require both administrative means and process and work-practice controls.

Avoiding lead and substituting less-toxic materials have been the primary means of reducing the potential for lead exposure in both occupational and environmental settings. Examples already discussed include

- the development and use of nonlead, durable paints;
- the replacement of lead plumbing, where necessary; and
- the development and use of unleaded gasoline.

However, no adequate substitute for lead is available in many situations, and the alternative is to separate the worker from the exposure. This can be accomplished through a number of ways:

- *Engineering controls.* For example, ventilation can be designed and manufactured to keep lead and its fumes and dust physically away from the worker. Care must be taken not to redirect the hazard toward others.
- *Administrative controls.* For example, work schedules and activities can be cycled to reduce the time workers spend in lead-exposure areas, which will limit their potential total exposures.
- *Substitution.* Altering the process to produce a less volatile form of lead can also be used as a control mechanism. For example, instead of burning the paint off a metal part, chipping the paint may reduce the exposure potential.

Other controls include enclosing the processes to isolate the worker from exposure and eliminating the need for human workers by using robots (as is done in some painting operations).

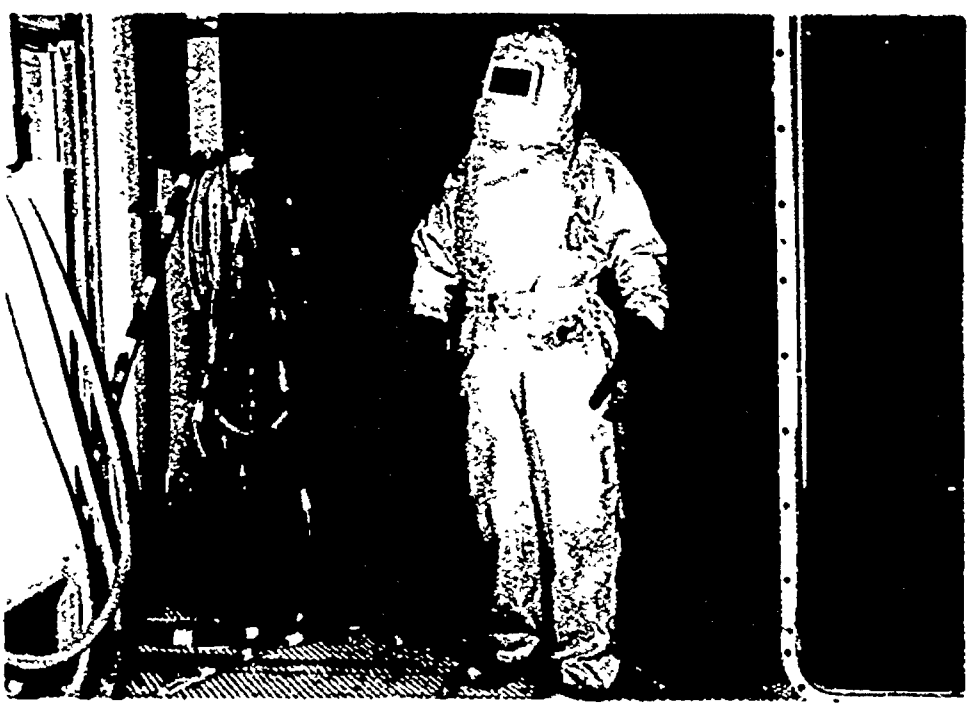

Fig. 12-12. Cumbersome protective gear (including a respirator, hooded chemical-resistant suit, gloves, and boots), worn for prolonged periods of time in sandblasting operations, imposes both physiological and psychological demands on the worker. Excessive physiological demands often manifest as heat stress. The inability to tolerate the psychological demands may manifest as anxiety or claustrophobia. Source: US Army, Anniston Army Depot, Anniston, Ala.

Often, wearing PPE (eg, respirators, gloves, or coveralls) is the only means of control available to workers. Although PPE is less effective than eliminating the exposure or altering the process, protective clothing and other devices are often used to control exposure because they are relatively low-cost. The advantages of using PPE, therefore, are availability and affordability. The disadvantages, however, can be numerous and formidable: improper fit, poorly motivated users, and ongoing maintenance.

Proper use of PPE implies that the workers are able to use the equipment. Certain conditions are incompatible with wearing PPE, however:

- Beard growth and facial deformities can make a respirator ineffective by interfering with or not conforming to its face seal; therefore the respirator will provide little or no protection.
- Cardiopulmonary insufficiency or claustrophobia may not allow a worker to tolerate the physiological or psychological demands of respirators or other heavy equipment (Figure 12-12).
- The protective equipment may even facilitate unexpected exposures; for example, spouses have been exposed via lead brought home on a worker's protective clothing.[70,71]

SUMMARY

Lead is one of the most important raw materials used in civilian and military industry. In the military, the highest likelihood of lead exposure is in operations associated with applying or removing paint, welding, and firing explosives or weapons. Lead has no known biological function, but it affects virtually every organ system and is toxic to many biochemical processes in the body. The blood lead levels at which health effects are manifest may be much lower than we once thought.

Although much about occupational and environmental lead exposure is highly regulated, we must still carefully consider sources of lead and the mechanisms of lead exposures to provide rational and effective control of lead as a hazard to human health. General workplace hygiene is important. In cleaning worksites with lead exposures, efforts must be taken to limit the amount of dust being blown or swept. Often, wetting an area to keep dust generation to a minimum is recommended. The process of lead abatement (stripping lead-based paint from accessible surfaces) can be expensive and must be performed properly to reduce exposure. When improperly performed, the process is associated with a significant degree of risk both to the abatement workers and to the occupants. Careful attention to minimizing exposure to the lead dust generated by the removal process accounts for much of the expense and tedium of deleading operations.

The potential for environmental exposures can also be modified by (*a*) using unleaded paints, (*b*) removing or enclosing lead-based paints, (*c*) replacing lead in plumbing systems, and (*d*) neutralizing acidic water. Obviously, the most effective way to achieve environmental control is similar to that of achieving occupational control: remove the sources of lead.

Despite efforts to control both occupational and environmental exposures, lead toxicity remains a not-uncommon clinical condition. Toxic levels of lead can be removed through the use of chelating agents when necessary; however, because the chelating agents are themselves toxic, the decision to treat lead poisoning should not be made lightly. Treatment should be done only by experienced, knowledgeable physicians.

REFERENCES

1. Singhal RL, Thomas JA. Lead and heme biosynthesis. In: *Lead Toxicity*. Baltimore: Urban and Schwartzberg; 1980. 79–117.

2. Hamilton A, Hardy HL. Lead. In: *Hamilton and Hardy's Industrial Toxicology*. 2nd ed. Littleton, Mass: John Wright, PSG Inc; 1983: Chap 13.

3. Grandjean P. Widening perspectives of lead toxicity: A review of the health effects of lead exposure in adults. *Environmental Research*. 1978;17:301–321.

4. Drasch GA. Lead burden in prehistorical, historical, and modern human bones. *Science of the Total Environment*. 1982;24:199–231.

5. Hunter D. The ancient metals. In: *The Diseases of Occupations*. 6th ed. London, England: Hodder and Stoughton; 1978: Chap 4.

6. Gilfillan SC. Lead poisoning and the fall of Rome. *J Occ Med.* 1965;7(2):53–60.

7. Charlesworth MP, ed. The social policy of Augustus. In: *The Cambridge Ancient History.* Vol 10, *The Augusten Empire: 44 BC–AD 70.* Cambridge, England: The University Press; 1963: 448–456.

8. Fuortes LJ. Health hazards of working with ceramics. *Postgraduate Medicine.* 1989;85(1):133–136.

9. Perrelli G, Capellaro E, Pira E, Maina G, Vergnano P. Further cases of lead poisoning from wine. *Am J Indust Med.* 1984;5:377–381.

10. Royce SE, Needleman HL, eds. Lead toxicity. In: *TSDR Case Studies in Environmental Medicine.* Atlanta, Ga: Agency for Toxic Substances and Disease Registry, Public Health Service; 1990.

11. Centers for Disease Control. Surveillance for occupational lead exposure—United States, 1987. *JAMA.* 1989;262(17):2370–2372.

12. 29 CFR, Part 1910 § 1025.

13. Zenz C. Lead and its compounds. In: *Occupational Medicine: Principles and Practical Applications.* Chicago: Yearbook Medical Publishers; 1988: Chap 36.

14. Levy BS, Wegman DH, eds. (1) Toxins and their effects. (2) Neurological and behavioral disorders. In: *Occupational Health, Recognizing and Preventing Work-Related Disease.* Boston: Little, Brown; 1983: Chaps 10 and 21.

15. Gaydos JC, moderator. Health Hazard Assessment Program review meeting. Fort Detrick, Frederick, Md; 31 May 1989.

16. Anania TL, Seta JA. *Lead Exposure and Design Considerations for Indoor Firing Ranges.* Cincinnati, Oh: National Institute for Occupational Safety and Health; 1975.

17. Robbins SK, Blehm KD, Buchan RM. Controlling airborne lead in indoor firing ranges. *Appl Occup Environ Hyg.* 1990;5(7):435–439.

18. Landrigan PJ, McKinney AS, Hopkins LC, Rhodes WW, Price WA, Cox DH. Chronic lead absorption: Result of poor ventilation in an indoor pistol range. *JAMA.* 1975;234(4):394–397.

19. Novotny T, Cook M, Hughes J, Lee S. Lead absorption in a firing range. *Am J of Public Health.* 1987;77(9):1225–1226.

20. Valway SE, Martyny JW, Miller JR, Cook M, Mangione EJ. Lead absorption in indoor firing range users. *Am J Public Health.* 1989;79(8):1029–1032.

21. Brewer F. Lead exposure in an indoor firing range. *J Occup Med.* 1989;31(4):409–410.

22. Bellamy RF, Zajtchuk R. The management of ballistic wounds of soft tissue. In: Bellamy RF, Zajtchuk R, eds. *Conventional Warfare: Ballistic, Blast, and Burn Injuries.* Part 1, Vol 5. In: *The Textbook of Military Medicine.* Washington, DC: DA Office of The Surgeon General and Center of Excellence in Military Medical Research and Education; 1991: Chap 5.

23. Hardy HL, Chamberlin RI, Maloof CC, Doylen GW Jr, Howell MC. Lead as an environmental poison. *Clin Pharmacol Therapeutics.* 1971;12(6):982–1002.

24. Elwood PC, Gallacher JEJ, Phillips KM, Davies BE, Toothill C. Greater contribution to blood lead from water than from air. *Nature.* 1984;310:138–140.

25. Zabel TF. Current standards and their relation to environmental behavior and effects—The case of lead. *The Science of the Total Environment.* 1989;78:187–204.

26. US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Atlanta, Ga: Agency for Toxic Substances and Disease Registry, Public Health Service; 1988.

27. Moore MR. Influence of acid rain upon water plumbosolvency. *Environ Health Perspectives*. 1985;63:121–126.

28. US Environmental Protection Agency. *Air Quality Criteria for Lead*. Research Triangle Park, NC: Health Effects Research Lab; 1977: 1–17. USEPA Document 600/8-77-017.

29. Chisholm JJ. Removal of lead paint from old housing: The need for a new approach. *Am J Public Health*. 1986;76(3): 236–237.

30. Chisolm JJ Jr. Lead poisoning. *Scientific American*. 1971;224(2):15–23.

31. Petrone LR. Childhood lead intoxication. *Accidents, Poisonings, and Violence*. 1990;12(1):41–52.

32. Froines JR, Liu WV, Hinds WC, Wegman DH. Effect of aerosol size on the blood lead distribution of industrial workers. *Am J Indust Med*. 1986;9:227–237.

33. Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd ed. New York: Macmillan; 1986.

34. World Health Organization. *Environmental Health Criteria 3, Lead*. Geneva, Switzerland: WHO; 1977.

35. Baselt R, Cravey, RH. *Disposition of Toxic Drugs and Chemicals in Man*. 3rd ed. Chicago: Yearbook Medical Publishers; 1989.

36. Putnam RD. Review of the toxicology of inorganic lead. *Am Indust Hyg Assoc J*. 1986;47:700–703.

37. Parmeggiani L, ed. *Lead*. In: *Encyclopaedia of Occupational Health and Safety*. Vol 2., rd ed. Geneva: International Labour Organization; 1983: 1200–1209.

38. Boggess WR. *Lead in the Environment*. National Science Foundation Report NSF/RA-770214. Washington, DC: GPO; 1977. GPO Stock No. 038-000-000338-1.

39. Landrigan PJ. Lead: Assessing its health hazards. *Health & Environment*. 1988;2(6):1–5.

40. LaDou J, ed. *Occupational Medicine*. Norwalk, Conn: Appleton & Lange; 1990: 306–309.

41. Gilman AG, Goodman LS, Gilman A, eds. *Goodman and Gilman's The Pharmacological Basis of Therapeutics*. 6th ed. New York: Macmillan; 1980.

42. Goyer, RA. Lead toxicity: A problem in environmental pathology. *Am J Pathol*. 1971;64(1):167–179.

43. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. *Toxicological Profile for Lead*. Washington, DC: USDHHS; 1990. ATSDR/TP-88/17.

44. Goetz CG. Lead. In: *Neurotoxins in Clinical Practice*. New York: Medical and Scientific Books, Spectrum Publications; 1985: Chap 1.

45. Bolla-Wilson K. Neurobehavioral Assessment in Toxic Exposure. Lecture handout presented at The Johns Hopkins University School of Medicine, Baltimore, Md; 1988.

46. Baker EL, White RF, Pothier LJ, et al. Occupational lead neurotoxicity: Improvement in behavioral effects after reduction of exposure. *Brit J Indust Med*. 1985;42:507–516.

47. Rom WN. Effects of lead on the female and reproduction. *Mount Sinai J Med*. 1976;43(5):542–551.

48. Landrigan PJ. Toxicity of lead at low dose. *Brit J Indust Med*. 1989;46:593–596.

49. Needleman HL. The persistent threat of lead: A singular opportunity. *Am J Public Health*. 1989;79(5):643–645.

50. Cullen MR, Kayne RD, Robins JM. Endocrine and reproductive dysfunction in men associated with occupational inorganic lead intoxication. *Arch Environ Health*. 1984;39(6):431–440.

51. Mitchell JW, ed. Lead toxicity and reproduction: Committee reply. *J Occup Med*. 1987;29(5):397–399.

52. Rempel D. The lead-exposed worker. *JAMA* 1989;262(4):532–534.

53. Kaul B, Davidow B, Eng Y, Gewirtz MH. Lead, erythrocyte protoporphyrin and ferritin levels in cord blood. *Arch Environ Health*. 1983;28(5):396–399.

54. Benevich T. New sources add to lead poisoning concerns. *JAMA*. 1990;263(6):790–791.

55. Needleman HL, Gastsonis CA. Low level lead exposure and the IQ of children: A meta-analysis of modern studies. *JAMA*. 1990;263(5):673–678.

56. Adamson RH, et al, eds. *A Digest Special Report: The Fourth National Environmental Health Conference*. Vol 3, No 8, Sept–Oct 1989; 3.

57. Davis M, Svendsgaard DJ. Lead and child development. *Nature*. 1987;329:297–300.

58. Landrigan PJ. Lead in the modern workplace. *Am J Public Health*. 1990;80(8):907–908.

59. Annas GJ. Fetal Protection and Employment Discrimination—The *Johnson Controls* Case. *N Eng J Med*. 1991;325(10):740–743.

60. Sharp DS, Smith AH, Holman BL, Fisher JM. *Arch Environ Health*. 1989;44(1):18–22.

61. Pocock SJ, Shaper AG, Ashby D, Delves HT, Clayton BE. The relationship between blood lead, blood pressure, stroke, and heart attacks in middle-aged British men. *Environ Health Perspectives*. 1988;78:23–30.

62. US Environmental Protection Agency. Integrated Risk Information System (IRIS). Leads and compounds (inorganic). Washington, DC: USEPA; 1989

63. Kaul B, Slavin G, Davidow B. Free erythrocyte protoporphyrin and zinc protoporphyrin measurements compared as primary screening methods for detection of lead poisoning. *Clin Chem*. 1983;29(8):1467–1470.

64. Haeger-Aronsen B. An assessment of the laboratory tests used to monitor the exposure of lead workers. *Brit J Indust Med*. 1971;28:52–58.

65. US Environmental Protection Agency. *Air Quality Criteria for Lead*. Vols 1–4. EPA-600/8-83/02aF. Washington, DC: USEPA; 1986.

66. Nadig RJ. Treatment of lead poisoning. *JAMA*. 1990;263(16):2181–2182.

67. Johnson BL, Mason RW. A review of public health regulations on lead. *NeuroToxicology*. 1984;5(3):1–22.

68. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer. Occupational Safety and Health Guideline for Inorganic Lead. In: *Occupational Safety and Health Guideline for Chemical Hazards*. Cincinnati, Oh: DHHS (NIOSH) 1988. Publication 88-186. Suppl 1-OHG.

69. American Conference of Governmental Industrial Hygienists. *Documentation for the Threshold Limit Values and Biological Exposure Indices*. 5th ed. Cincinnati, Oh: ACGIH; 1986.

70. Knishkowy B, Baker EL. Transmission of occupational disease to family contacts. *Am J Indust Med*. 1986;9:543–550.

71. Johnson D, Houghton K, Siegel C, Martyny J, Cook L, Margione EJ. Occupational and paraoccupational exposure to lead—Colorado. *MMWR*. 1989;38(19):339–345.

Chapter 13

SOLVENTS, FLUOROCARBONS, AND PAINTS

GLENN J. LEACH, Ph.D.* AND LEROY W. METKER†

INTRODUCTION

SOLVENTS
- General Toxicity
- Respiratory Uptake of Solvents
- Specific Toxicity
- Exposure Controls

FLUOROCARBONS
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- Civilian and Industrial Exposures
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- Pharmacological Effects
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PAINTS
- Toxic Constituents
- Chemical Agent Resistant Coatings
- Exposure Controls

MEDICAL SURVEILLANCE

MEDICAL TREATMENT
- Solvents and Paints
- Fluorocarbons

SUMMARY

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INTRODUCTION

A large segment of the Department of Defense (DoD) industrial workforce, including both civilian and military personnel, is employed in occupations that involve potential exposures to toxic environments. Organic solvents are a major source of exposure. A *solvent* is a material, usually a liquid, that is capable of dissolving another substance. Solvents include highly polar substances like water, as well as nonpolar organic compounds. Because the vapor pressure of water-based solvents is low, and therefore the potential for inhaling their vapors is also low, water-based solvents are not discussed in this chapter. Many of the nonpolar organic solvents, however, are relatively volatile and pose significant inhalation hazards.

The organic solvents include aliphatic and aromatic hydrocarbons, chlorinated hydrocarbons, alcohols, ether esters, and ketones. This textbook treats fluorocarbons separately from the other halogenated hydrocarbons; they are used as solvents, but their nonsolvent uses as refrigerants, propellants, and in fire-suppression systems have greater military and medical significance. Oil-based paints not only contain solvents that are used as diluents, they also contain resins, vehicles, and additives that are associated with a variety of illnesses to which occupational health professionals must respond.

SOLVENTS

The DoD is a major user of solvents. More than 25 installations—including shipyards, air logistics centers, army depots, and aviation repair and maintenance facilities—each use over 27,500 gallons of solvent per year, and at least 120 installations use lesser amounts.[1]

In general, occupational exposures to solvents in the uniformed and civilian defense workforce duplicate those found in civilian industry. Solvents are used as dry-cleaning agents, chemical intermediates in manufacturing, drying compounds, general-purpose cleaners, paint thinners and removers, and in the manufacture of materiel. This chapter considers solvents as they are used in four military applications: vapor degreasers, cold-dipping cleaners, precision cleaners, and solvents that are associated with paints.

In vapor degreasing, solvent contained in degreasing tanks is heated to the boiling point—which is low, relative to water—and the vapors form a cloud over the surface. The items to be cleaned are lowered into the vapors, which then condense on the cold item and dissolve its surface oils and greases. The degreasing tanks are usually fitted with a cooling coil near the top that condenses the vapor, minimizing the solvent loss. The solvents that are commonly used in these operations include perchlorethylene, methyl chloroform, trichloroethylene, methylene chloride, and trichlorofluoroethane.[2] Vapor degreasing is used by the army for a number of industrial operations including cleaning engines and other vehicle parts in vehicle-maintenance facilities and degreasing large-bore gun tubes after milling (Figures 13-1 and 13-2).

In cold-dipping degreasing, the item to be cleaned is simply dipped into a tank of solvent. These solvents—from petroleum distillates to mixtures that include aliphatic and aromatic hydrocarbons, ketones, cellosolves, and creosote—tend to have lower volatility than those used in vapor degreasers and therefore the risks of inhalation are less for workers. These procedures are also typical at vehicle maintenance facilities, particularly for smaller parts.

Precision cleaners—generally fluorocarbons—are also used in their liquid state, usually in cold dipping or as sprayed aerosols. They are generally used to clean sensitive electronic components. Large quantities of solvents are also used in paints and related products such as varnishes and lacquers, and paint thinners, primers, and strippers. Exposures occur during mixing, applying, or drying.

General Toxicity

The potential health effects from exposure to solvents depend on a number of factors including the physical and chemical properties of the solvents, how the solvents are used, the way in which workers are exposed, and the duration (acute or chronic) of the exposure. In the industrial setting, there are two primary routes of exposure: inhalation of vapors or aerosolized mists and dermal absorption. The concentration of solvent vapors in air is expressed in *volume per volume* units (eg, parts per million). Concentrations can also be expressed as milligrams per cubic meter, which is a *weight per volume* measure. Particulates

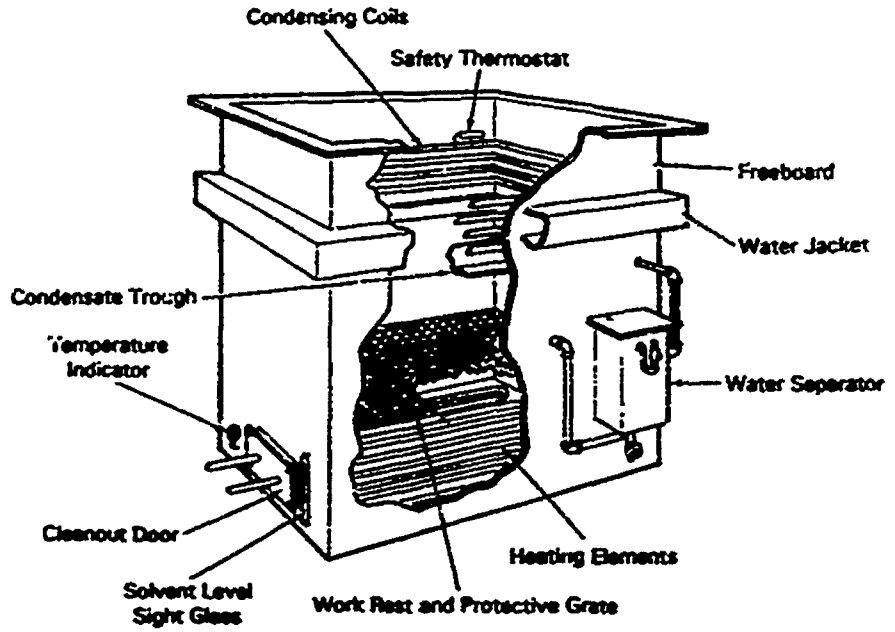


Fig. 13-1. A typical degreasing tank. Vapor degreasing is accomplished by lowering the object to be cleaned into the vapor zone above the liquid solvent. Solvent vapors condense on the cold item and loosen grease and oil, which then collect in the bottom of the tank. Condensing coils located near the top of the tank condense the solvent vapors, which collect in the condensate trough and are returned to the reservoir. The item is held in the vapor zone until the parts reach the temperature of the solvent vapor. At that time, condensation stops and the item is dry.

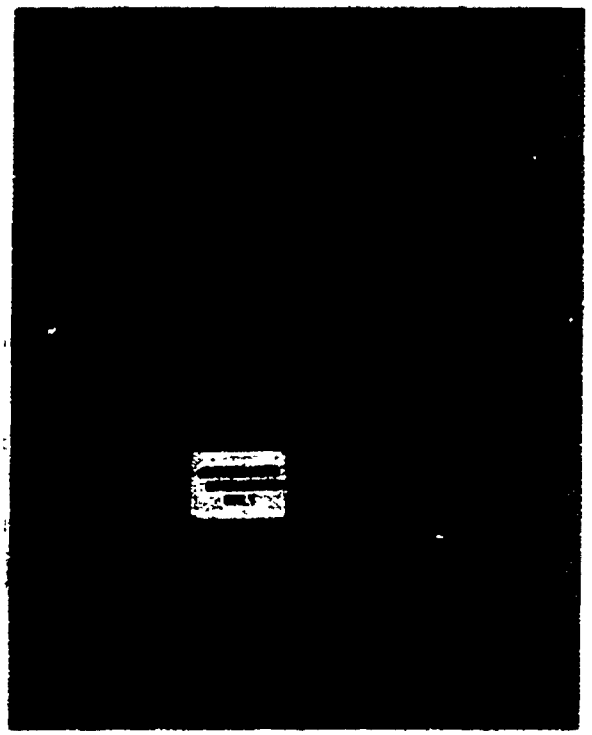


Fig. 13-2. Degreasing tanks located in a pit must be surrounded by a railing. Only authorized personnel can operate a vapor degreaser, and they must be wearing appropriate personal protective equipment. During routine operations, employees should wear chemical safety goggles and solvent-resistant aprons and gloves. During tank cleaning operations, a rescue harness, lifeline, and self-contained breathing apparatus should be worn. A second person, fully equipped to enter the tank, should be stationed outside the tank ready to assist if required.

suspended in air, such as dusts, are only expressed as weight per volume units.

Acute exposures are isolated and of short duration (minutes to several hours). In the occupational setting, acute exposures usually occur when pipes or supply lines are accidentally broken, spills occur, or workers enter chemical storage tanks to clean or paint. Acute exposures to most organic solvents cause central nervous system (CNS) depression and narcosis. Signs of toxicity include disorientation, euphoria, giddiness, and confusion, which are reversible in most cases when the victim is removed from the toxic environment. With sufficiently high exposures, the signs may progress to paralysis, convulsions, unconsciousness, and death due to respiratory or cardiovascular arrest.[3] Airborne concentrations of solvents sufficient to cause acute toxic effects are typically in the range of 1,000 to 10,000 ppm, although these are compound specific.

Chronic exposures to solvents are repeated, daily exposures to low concentrations. Again, concentrations sufficient to cause chronic toxicity are compound specific, but generally range from hundreds of parts per million to less than 1. Dermatitis is a common result of prolonged or repeated dermal contact. This is a result of the defatting of skin by the solvents, which causes dryness and fissuring of the skin.

The organic solvents are toxic to a number of organ systems. Well-defined CNS lesions have been described for n-hexane and n-butyl ketone. There are also reports of nonspecific behavioral, intellectual, and psychological effects among workers exposed to mixed solvents: house and car painters and jet-fuel handlers have reported impairment of visual perception, hand-eye coordination, and memory, as well as abnormal results on psychological tests.[4]

Many solvents are also toxic to the blood, liver, and kidneys. In many instances, the specific toxicity results from *biotransformation products* of the parent solvent with the formation of *reactive metabolites*.[3] The hepatotoxic effects of carbon tetrachloride and alcohol are thought to result from biotransformation products; however, they have yet to be thoroughly described. Similarly, some blood dyscrasias produced by benzene are believed to occur as a result of reactive metabolites.

Respiratory Uptake of Solvents

Among other physiological factors, the rate and depth of pulmonary ventilation and the cardiac output affect the respiratory uptake of solvents, and these ultimately influence the toxic effects. Solvent uptake in the lungs is through simple diffusion: the difference in concentration between inspired air and the blood is the driving force that causes a solvent to enter the blood and be distributed throughout the body. Gas in the alveoli equilibrates rapidly with blood in the pulmonary capillaries. Blood solvent levels depend on the solubility of the solvent vapors in blood. For very soluble solvents such as chloroform, very little remains in the alveolar gas for expiration. Soluble compounds, however, require longer to equilibrate with the blood than low-soluble compounds because a greater amount of compound is required to reach equilibrium. An increasing ventilation rate will increase the delivery of solvent vapors to the lungs and decrease the time required for equilibration. Uptake of these solvent vapors is said to be *ventilation limited* (ie, equilibration is dependent on the rate and depth of respiration). In contrast, the vapors of solvents with low solubility take less time to equilibrate with the blood, and the rate is dependent on blood flow through the lungs. Uptake of these substances is said to be *perfusion limited*.[3,5] The solubility of a solvent, the time necessary for it to reach equilibrium in the blood, and the concentration of the solvent in the blood at equilibrium are (*a*) related through the solubility coefficient (S) and (*b*) limited by ventilation, perfusion, and cardiac output (Table 13-1). The solubility coefficient represents the ratio of the concentration of a vapor or gas in an aqueous medium to the concentration in the gas phase.

Specific Toxicity

Specific toxicological effects can be described for individual solvents (Table 13-2). However, in most industrial settings exposures will be to complex mixtures; the chemical composition of these solvent mixtures will vary with the particular lot of solvent furnished by various suppliers, in many cases. The only way to be certain of the specific solvent composition is to consult the Material Safety Data Sheet supplied by the manufacturer for the specific lot of solvent in use.

Aliphatic Hydrocarbons

The aliphatic hydrocarbons include the saturated, straight-chain paraffins (alkanes) and the unsaturated olefins (alkenes). Compounds with chain lengths of 5 to 16 carbon atoms are usually liquids, and those exceeding 16 carbons are usually solids. Most compounds containing carbon chains exceeding eight units have low volatility and pose little inhalation threat. Typical aliphatic hydrocarbons include hexane, which

TABLE 13-1

RESPIRATORY UPTAKE

Chemical	S*	Equilibration Time	[Blood] at Equilibrium	Physiological Parameter Limiting Uptake
Ethanol	1,100.0	Slow	High	Ventilation
Acetone	245.0	↓	↓	
Methyl ethyl ketone	202.0			
Benzene	7.8			
Carbon tetrachloride	2.4	↓	↓	
Ethylene	0.15	Fast	Low	Perfusion

*Solubility coefficient

Source: Aharie Y. *Inhalation and Toxic Responses of Lung*. Kansas City, Kan: Mid America Toxicology Course; 1981: 285–400.

is used frequently as a solvent in adhesives and is found in many paints and varnishes[6]; kerosene, which is used as a fuel, a carrier for pesticides, and a cleaning solvent; and stoddard solvent, which can be a mixture of more than 100 different aliphatics and is used extensively as a degreasing agent.

Hexane. Commercial hexane, sometimes called hexane isomers, can consist of up to 100% n-hexane. Until the mid-1960s, n-hexane was considered to be relatively nontoxic because workers could be exposed to high concentrations of vapor with no discomfort. However, in 1964, workers using products containing n-hexane began to exhibit sensory and motor deficits. Exposures were estimated to be 500 to 2,500 ppm. Methyl n-butyl ketone (MNBK) was shown to produce a similar neuropathy.[3]

Hexane is readily absorbed and has an affinity for fatty tissues. An extensive research program in both animals and humans identified 2,5-hexanedione as a common metabolite of both n-hexane and MNBK, and this metabolite is believed to be responsible for the neurotoxicity.[5] 2,5-Hexanedione was also shown to produce a neuropathy in animals that was identical to that produced in humans by n-hexane and MNBK.[3]

The American Conference of Government Industrial Hygienists (ACGIH) has recommended a Threshold Limit Value (TLV) of 50 ppm (176 mg/m^3) for n-hexane, although recommended levels for other hexane isomers are 10-fold higher.[7] The current Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) follows the ACGIH guidance: 50 ppm for n-hexane and 500 ppm for mixed hexane isomers.[8]

Stoddard Solvent. Several commercial solvent mixtures are derived from the distillation of petroleum. The nomenclatures for stoddardlike solvents include varsol, mineral spirits, and white spirits. Although these mixtures differ in their distillation ranges and performance characteristics, their toxicological properties are similar. Stoddard solvents cause the typical hydrocarbon-solvent effects of CNS depression from high inhalation exposures and skin irritation from dermal exposures. In addition, scattered reports in the literature demonstrate that chronic exposures to stoddard solvents cause myelotoxic effects and liver toxicity.[6] Both the ACGIH TLV and OSHA PEL for stoddard solvent is 100 ppm (525 mg/m^3).[7,8]

Kerosene. Kerosene is a mixture of petroleum hydrocarbons with carbon chain lengths of 9 to 16 units, although its composition varies with the source of crude oil and the refining methods. Other common names for kerosene include astral oil, coal oil, and No. 1 fuel oil. Typical constituents of kerosene include mixtures of aliphatic, naphthenic, and aromatic hydrocarbons. Regardless of its name and exact composition, kerosene has a relatively low acute systemic toxicity, although it is a significant health hazard when the liquid directly enters the trachea and lungs or is aspirated from the stomach. Milliliter quantities of kerosene can cause pneumonitis, pulmonary edema, hemorrhage, and necrosis. Other petroleum-derived solvents pose a similar threat.[6] Dermal exposure to kerosene has also been shown to cause skin irritation, and inhalation exposures may cause bone marrow depression, although the latter response may be due to contamination of the kerosene with benzene (discussed later in this chapter).[9] TLV and PEL values have not been established for kerosene.

TABLE 13-2

TOXICITY OF REPRESENTATIVE SOLVENTS

Solvent	Toxicity Rating*	Target Organ or Effect of Chronic Toxicity	WOE†	TLV (ppm)	PEL
Aliphatic Hydrocarbons					
n-Hexane	2–1	neurotoxic, testicular atrophy	UE	50	50
Kerosene	3	pneumonitis, edema	NE	—	—
VM&P naphtha	2	CNS depression, skin irritation	NE	100	—
Stoddard solvent	1	—	—	—	—
Aromatic Hydrocarbons					
Benzene	3	CNS, aplastic anemia, leukemia	A	10	—
Toluene	3	CNS depressant, dermal irritant	D	100	100
Cresol	2	cancer, dermal irritant	C	5	—
Phenol	3	developmental toxicity	D	5	—
Xylene	2	liver	D	100	100
Chlorohydrocarbons					
1,2-Dichloroethane	2	circulatory system	B2	200	100
Trichloroethylene	3	lung, liver	B2	50	50
Carbon tetrachloride	3	liver	B2	5	2
Perchlorethylene	3	liver, blood	B2	50	25
Methylene chloride	—	lung, liver	B2	50	—
Fluorocarbons					
Dichlorodifluoromethane	1	lung, liver	NE	—	—
Trichlorofluoroethane	2–1	—	—	—	—
Trichlorotrifluoroethane	1	—	—	—	—
Chloropentafluoroethane	2–1	—	—	—	—
Bromochloro-difluoromethane	1	—	—	—	—
Dibromotetrafluoroethane	2	—	—	—	—
Aldehydes and Ketones					
Formaldehyde	3	irritant, sensitizer	B1	0.30	0.75
Acetone	2–1	liver, kidney carcinogen	D	200	200
Methyl ethyl ketone	3	CNS neurotoxin, fetotoxin	D	—	—
Methyl n-butyl ketone	2	CNS	—	—	—
Glycols					
Ethylene glycol	2–1	liver, kidney	NE	50	50
Propylene glycol	2–1	kidney, blood	NE	—	—
Glycol Ethers					
Ethylene glycol monomethyl ether	2	toxic encephalopathy, bone-marrow depression	NE	5	5
Ethylene glycol monoethyl ether	2	testicular atrophy	NE	5	5

*Toxicity Rating: a qualitative ranking of compound toxicity; categories range from 1 to 3. 1: low or slight; 2: moderate; 3: severe. Source of toxicity rating: Sax NJ. *Dangerous Properties of Industrial Materials*. 6th ed. New York: Van Nostrand Reinhold; 1984.

†Weight of Evidence: a qualitative classification of the potential for a compound to produce cancer in humans (developed by the EPA)

A: Human carcinogen (based on sufficient information from epidemiology studies to support a causal relationship)

B1: Probable human carcinogen (based on sufficient information from animal studies and limited information from human epidemiology studies)

B2: Probable human carcinogen (based on sufficient information from animal studies, but evidence of carcinogenicity in humans is inadequate)

C: Possible human carcinogen (based on limited information from animal studies)

D: Cannot be classified

E: No evidence of carcinogenicity in humans

UE: Compound is undergoing evaluation for carcinogenicity

NE: Compound has not been evaluated for its potential human carcinogenicity

Aromatic Hydrocarbons

Aromatic hydrocarbons containing an unsaturated six carbon ring structure are used extensively and are toxic to multiple organ systems. These solvents are primary skin irritants, due to their defatting properties, and their vapors are irritating to the mucous membranes and airways. Systemically, aromatic hydrocarbons are CNS depressants, and several of these compounds have toxic actions on other target organs. Aromatic hydrocarbons are important in the production of plastics and rubber products, and are used in some paints.[9]

Benzene. Benzene is the most toxic member of this group. It is a common ingredient in paint and varnish removers and paint thinners as well as a contaminant of many of the petroleum-based solvents. In addition to its CNS-depressing actions, chronic exposure to benzene suppresses the hematopoietic system. Acute and chronic lymphocytic leukemias and aplastic anemia (which has a mortality rate of 70% over a 5-y period) have been associated with worker exposures.[3,9] Epidemiological studies have also implicated benzene as a cause of acute myelogenous leukemia. The mechanisms and etiology of chronic lymphocytic leukemia and aplastic anemia have not been described; however, they appear to be initiated by an unidentified metabolite of benzene.[3] Clinically, chronic benzene exposures have been associated with decreased numbers of circulating erythrocytes and leukocytes, conditions which may be an early indication of benzene toxicity.[3] The ACGIH TLV for benzene is currently 10 ppm; however, the ACGIH has proposed lowering this value to 0.1 ppm.[7] The OSHA PEL for benzene is 1 ppm.[8]

Alkyl Benzene. The alkyl benzenes include *toluene* (methylbenzene), the *xylenes* (ortho, meta, and para isomers of dimethyl benzene), and the *cresols* (monomethyl phenols). The evidence from studies done with animals suggests that this group of solvents does not have the hematopoietic toxicity that is characteristic of benzene. The acute and chronic effects of toluene are CNS depression and dermal irritation.[10] The xylenes are also CNS depressants; the most common symptoms reported from occupational exposures are headache, fatigue, lassitude, irritability, and gastrointestinal disturbances.[9] Cresol is a strong dermal irritant with systemic effects on the kidneys and liver; its toxicity is similar to that of phenol. For work-place exposures to toluene and xylene, the ACGIH TLVs and OSHA PELs are 100 ppm; for cresol, both values are 5 ppm.[7,8]

Halogenated Hydrocarbons

These solvents are composed of carbon, hydrogen, and a halogen (usually chlorine or fluorine), and can be divided into the simple chlorohydrocarbons and the chlorofluorocarbons. The simple chlorohydrocarbons contain chlorine as the only halogen moiety and are excellent solvents for oils, fats, and other organic compounds. Simple chlorohydrocarbons are often used in both vapor and cold-dip degreasing. They are also used extensively in paint removers, solvents, and thinners.

The most common acute toxic effect of the simple chlorohydrocarbons is CNS depression; high exposures may cause respiratory depression or circulatory failure resulting in death. The vapors of the simple chlorohydrocarbons are not especially irritating to the upper airways, but repeated skin exposures may cause dermatitis due to their defatting actions.[2]

The solvent methylene chloride (dichloromethane) has a unique toxic action of which occupational health professionals should be aware. This very volatile chlorohydrocarbon is widely used in paints, paint strippers, degreasing operations, and as an aerosol propellant. In addition to the typical CNS depression, methylene chloride has also been found to be rapidly metabolized to carbon monoxide. Short exposures to high levels of this solvent have been shown to produce carboxyhemoglobin levels over 10%. This could have a significant adverse health effect in workers with existing cardiovascular disease.[9]

With chronic exposures, many of the chlorinated hydrocarbons are hepatotoxic, causing both fatty infiltration and necrosis. Carbon tetrachloride, chloroform, and 1,1,2-trichloroethane are also toxic to the kidneys; however, the other chlorohydrocarbons do not share this effect.[3] A number of chlorinated solvents have been demonstrated to cause cancer in laboratory animals (see Table 13-2). Despite extensive data from studies on animals that indicate potential carcinogenicity, however, evidence from epidemiological studies on humans has been inconclusive. Data on worker exposures to trichloroethylene, fluorocarbons, and methylene chloride have all been negative.[9]

One further concern is the potential toxicity of the thermal decomposition products of halogenated hydrocarbons. At high temperatures, the chlorohydrocarbons decompose to hydrogen chloride and phosgene. Toxic levels of these hazardous gases can be produced during welding operations in atmospheres of chlorinated hydrocarbon solvents.[10]

The chlorofluorocarbons contain both chlorine and fluorine as the halogen moiety and are used extensively in the military as precision cleaners for electronic components and in vapor degreasing. Their nonsolvent uses are discussed separately in this chapter.

Aldehydes and Ketones

Although aldehydes and ketones are grouped together based on their chemical structures, they are used for distinctly different purposes. The most common aldehyde, formaldehyde, is used in adhesives, industrial coatings, and in the production of certain dyes. The ketones are widely used as solvents.

Aldehydes and ketones also have somewhat different toxicological effects. Formaldehyde is a potent skin and eye irritant and a well-known sensitizer. Allergic responses are commonly seen in persons who come in contact with formaldehyde. Studies with animals also show that inhalation of 15 ppm of formaldehyde produces carcinomas in the nasal cavities of rats.[6] The common ketones including acetone, methyl ethyl ketone (MEK), and methyl isopropyl ketone (MIPK) are also irritants to the eyes and mucous membranes. At high concentrations, ketones are CNS depressants. MNBK is uniquely neurotoxic, as was discussed previously.

The potential health effects that distinguish the aldehydes from the ketones are also the rationale for the differences in their exposure limits. The ACGIH TLV for formaldehyde is 1 ppm, but this value may be reduced to 0.3 ppm.[7] OSHA has established a PEL of 1 ppm, with a 2 ppm *ceiling value*, and is also considering a reduction (a ceiling value is an exposure level that cannot be exceeded at any time during an 8-hour workday). Both the ACGIH TLV and OSHA PEL are 5 ppm for MNBK and 200 ppm for MEK and MIPK.[7,8]

Glycols and Glycol Ethers

Glycols and glycol ethers differ in both their degree and their mechanisms of toxicity, although they are usually grouped together by virtue of their chemical structures. The two are also used differently. Glycols are used commonly as industrial solvents for nitrocellulose and cellulose acetate, and in the production of pharmaceuticals. The glycol ethers are used extensively as solvents for lacquers, varnishes, resins, dyes, and inks.

Glycols have low vapor pressure and do not present a significant inhalation hazard unless they are heated or aerosolized. However, ethylene glycol has greater oral toxicity in humans than has been demonstrated in other mammalian species. The primary toxic action associated with ethylene glycol is metabolic acidosis, which is caused by its metabolite glycolic acid. Calcium oxalate, another metabolic byproduct of ethylene glycol, tends to accumulate in the proximal renal tubules and causes necrosis and functional changes in the kidneys.[3]

The glycol ethers include ethylene glycol monomethyl ether (EM), ethylene glycol monoethyl ether (EE), and the propylene series of glycol ethers. While they are not acutely toxic when ingested or inhaled, they have a unique reproductive toxicity: testicular atrophy and hematological effects were found when animals were administered EM and EE by various routes. The propylene series of glycol ethers does not appear to cause these reproductive effects.[3]

Due to these toxic effects, the ACGIH recently lowered the TLV for EM and EE; current recommendations are 5 ppm.[7] The OSHA PELs for these solvents are also 5 ppm.[8]

Exposure Controls

Two of the many controls used in vapor degreasing operations are condensers and local exhaust ventilators, although other controls may be designed for other operations that require solvents. Properly designed vapor degreasing units are equipped with condensers that surround the tank, which minimize the escape of solvent vapors into the ambient environment of the workplace. All of these units should be equipped with thermostatic controls mounted above the normal vapor level, which will shut off the heat source if the vapors rise above the condensing surface. Local exhaust ventilation controls may be necessary depending on the type of installation. Even with these controls, open flames, electric heating elements, and welding operations must not be located near a degreaser.[2]

Vapor degreasers also require periodic cleaning to remove the sludge and metal chips that have accumulated at the bottom of tanks. This requires that the solvent be distilled off until the heating surface is nearly exposed. The unit is then allowed to cool, and the sludge and remaining solvent are drained off. Personnel who enter a degreasing tank to perform this operation should wear respiratory protective equipment and a lifeline that is held by an attendant.[2]

The exposure controls for cold-dip degreasing and precision cleaning operations are much less extensive. Lids should be provided for the dip tanks to minimize evaporation of the solvent. Personal protective equipment (PPE) such as a faceshield and gloves must be worn to prevent the solvent from coming in contact with the skin and eyes.

FLUOROCARBONS

The first fluorocarbon, carbon tetrafluoride, was isolated in 1926.[11] Although no such compound occurs in nature, numerous additional fluorocarbons have been synthesized in substantial quantities since the 1940s. Commercial interest in fluorocarbons centered around their chemical and thermal stability and led to their extensive use as aerosol propellants, refrigerants, plastic foaming agents, heat-exchange agents, and solvents; as propellants for therapeutic agents and antiasthmatic drugs; and as general anesthetics. By the mid-1970s, production of these chemicals—originally considered to be inert refrigerants—had exceeded 2 billion pounds, most of which was released eventually into the environment.[12]

Fluorocarbons can be divided into the fully halogenated (nonhydrogenated) and the hydrogenated species. A further subdivision of these two groups—into the chlorinated and nonchlorinated species—is necessary to understand the significance that these chemical structures have in the current depletion of the earth's ozone layer (Table 13-3). This process involves the nonhydrogenated fluorocarbons, which are the more stable and therefore have the greater probability of reaching the ozone layer. Of the nonhydrogenated species, the chlorinated fluorocarbons are postulated to be the most detrimental.

TABLE 13-3

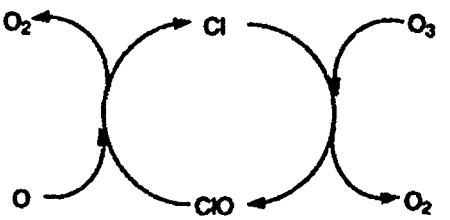
REPRESENTATIVE FLUOROCARBONS

Type	Formula
Nonhydrogenated	
Chlorinated	
Dichlorodifluoromethane	CCl_2F_2
Trichlorofluoromethane	CCl_3F
Nonchlorinated	
Octafluorocyclobutone	C_4F_8
Dibromotetrafluoroethane	$C_2F_4Br_2$
Hydrogenated	
Chlorinated	
Chlorodifluoromethane	$CHClF_2$
Chlorodifluoroethane	CH_3–$CClF_2$
Nonchlorinated	
Difluoromethane	CH_2F_2
Trifluoroethane	CH_2CF_3

The fluorocarbons are similar in composition to the chlorinated hydrocarbons, with the addition of a fluorine moiety. Numerous possible moiety combinations of carbon with hydrogen, fluorine, bromine, and chlorine have been prepared with the aliphatic hydrocarbon series. The fluorocarbons are usually clear, colorless, highly volatile liquids with a mild, somewhat ethereal odor. They are nonflammable, have high density, low viscosity, low surface tension, low toxicity, and are very stable.

Fluorocarbons do not react with most metals at temperatures below 200°C, or with most acids or oxidizing agents. However, under unusual circumstances, they can be made to react with highly reactive metals: for example, trichlorofluoromethane (CFC-11) reacts with concentrated sulfuric acid and sulfur trioxide at room temperature.

With few exceptions, the fluorocarbons are relatively inert to chemical reactions on earth and in the lower atmosphere. When compared to other halogenated compounds, the hydrolysis rates for the fluorocarbons are quite low, although there is considerable variation within the group. At atmospheric pressure, the rate of hydrolysis is too low to be measured by most analytical methods. This low rate of hydrolysis prevents the fluorocarbons from degrading in the troposphere.[13] However, it is their inherent stability—which allows them to migrate, intact, as high as the stratosphere—that is the source of concern surrounding the depletion of the ozone layer. Through photodegradation and free-radical reactions, chlorofluorocarbons provide a large reservoir of free chlorine atoms, which then catalyze the destruction of ozone:



It is postulated that this is a chain reaction; it allows one chlorine atom to continue to react, thus destroying thousands of molecules of ozone. Much of the research done on this problem is, of necessity, theoretical; furthermore, the contribution that free chlorine from natural sources might make to this process is neither well documented nor well understood. However, sufficient data exist for the international scientific community to call for a ban on the release of chlorofluorocar-

bons into the environment. The nonchlorinated fluorocarbons are more stable, are not sources of free chlorine, and are therefore not of such environmental concern.

Current Status of Ozone-Depleting Substances

The Montreal Protocol, an international agreement to limit the release and production of ozone-depleting substances, was signed in September 1987.[14] This agreement, ratified by the U.S. Senate, became effective in January 1989; it stringently restricts the international production and use of chlorofluorocarbons and *halons* (a trademarked name for several tetrafluoroethylene polymers) (Exhibit 13-1). A DoD Directive (DoDI) issued on 13 February 1989 clarified the army's position concerning specific chlorofluorocarbons and halons (Table 13-4).[15] This directive affects all DoD components, establishes policy, and assigns the responsibility for

- managing the production of chlorofluorocarbons and halons,
- identifying chlorofluorocarbon and halon applications and prioritizing their uses,
- identifying a long-term process to decrease the DoD's dependence on chlorofluorocarbons and halons,
- developing research and development programs to produce or evaluate suitable substitutes for chlorofluorocarbons and halons, and
- designing a tracking system to document the DoD's annual requirements for chlorofluorocarbons and halons.

EXHIBIT 13-1

CFC AND HALON RESTRICTIONS OF THE MONTREAL PROTOCOL

Restriction	Year of Accomplishment
Freezing of CFC consumption at 1986 levels*	1989
Freezing of halon consumption at 1986 levels	1992
Further reduce CFC consumption by 20%†	1993
Further reduce CFC consumption by 30%‡	1998

*Represents an initial reduction of approximately 15%
†Reduced from 1986 level
‡Reduced from 1993 level
Source: Treaty Document 100-10. December 21, 1987. *Montreal Protocol on Substances That Deplete the Ozone Layer.* Done at Montreal on September 16, 1987, to the Vienna Convention for the Protection of the Ozone Layer. Ratified by the US Senate March 14, 1988. A copy of the treaty document may be obtained from the Library of Congress, Washington, DC.

TABLE 13-4

CFCs AND HALONS AFFECTED BY THE DoD DIRECTIVE AS OF AUGUST 1988

Ozone Depleter	Formula	Chemical Name
CFC-11	CCl_3F	Trichlorofluoromethane
CFC-12	CCl_2F_2	Dichlorodifluoromethane
CFC-113	$C_2Cl_3F_3$	Trichlorotrifluoroethane
CFC-114	$C_2Cl_2F_4$	Dichlorotetrafluoroethane
CFC-115	C_2ClF_5	Chloropentafluoroethane
Halon 1211*	$CBrClF_2$	Bromochlorodifluoromethane
Halon 1301*	$CBrF_3$	Bromotrifluoromethane
Halon 2402*	$C_2Br_2F_4$	Dibromotetrafluoroethane

*Registered trademark of Allied Chemical Corporation.
Source: US Department of Defense. *Chlorofluorocarbons (CFCs) and Halons.* Washington, DC: DoD; 1989. DoD Directive 6050.9.

Both the Montreal Protocol and the DoDI will substantially affect the use of chlorofluorocarbons and halons in the near future. The phased-in restrictions should result in a reduction of CFCs by over 60%, as measured from 1989 consumption levels.

The effects of compliance will be at least these two: (1) the use of present compounds will be curtailed, and (2) replacement chemicals will enter the DoD's supply channels. Unfortunately, replacement chemicals have not yet been designated and therefore cannot be discussed in this chapter, but the search for replacement chemicals will obviously center around the hydrogenated nonchlorinated compounds.

The mandated phaseout of the chlorofluorocarbons and halons has generated tremendous interest in developing replacement chemicals. However, current research appears to be driven more by the need to find and produce chemicals that are environmentally safe rather than physiologically nontoxic. Occupational health professionals may soon find themselves confronting new replacement chemicals that are accompanied by little or no medical or toxicological information.

Civilian and Industrial Exposures

The fluorocarbons and halons are used as refrigerants, polymer intermediates, propellants, anesthetics, fire extinguishers, foam-blowing agents, dry-cleaning agents, and as degreasing solvents in the electronics industry. Workers who produce and package fluorocarbons are at highest risk of exposure to high concentrations of these chemicals, but significant exposures also occur when fluorocarbons are used as solvents or cleaners. Because fluorocarbons disperse rapidly when released, only minimal exposure appears to occur from their use as refrigerants and propellants. However, recent uses of fire extinguishers to protect computer and electronic areas have created the potential for short-term, high-concentration exposures when large volumes of the material are released in confined spaces. Chronic occupational exposure to hospital operating-room personnel from the use of anesthetic gases is also significant.

Militarily Unique Exposures

In addition to the civilian and industrial uses already discussed, fluorocarbon use in the military includes specialized applications such as the use of CFC-114 for submarine refrigeration to eliminate vibration that might lead to detection by the enemy. Halon 1211 and Halon 1301 are used to suppress fires in the crew compartments of tactical vehicles, aircraft, shipboard systems, and in command, control, and communication centers (see Chapter 6, Health Hazard Assessments). Significant amounts of these materials are used by DoD workers. Based on 1986 production figures, the DoD procured just under 35% of the United States's total production of Halon 1211, approximately 6% of Halon 1301, and just under 5% of the total production of the regulated chlorofluorocarbons.[14]

Fluorocarbons, as they are used in submarines and other tactical vehicles, expose military personnel to hazardous situations not usually encountered by civilians: specialized weapons systems are used in essentially closed environments. Military personnel are not always able to simply leave a contaminated environment; their duties may oblige them to stay and man their equipment, thereby exposing them to higher concentrations for longer times than may be typical in the civilian community.

For example, the crew of an M60A3 tank can be exposed to Halon 1301 if the tank's automatic fire extinguishing system is activated during a system malfunction or an actual fire. In either situation, the crew could be exposed to oxygen deficiency, the toxic effects of Halon 1301 itself, or the toxic effects of the decomposition products of Halon 1301:

> The discharge of four 7-pound (net weight) bottles of Halon 1301 into the closed, unventilated M60A3 crew compartment (estimated volume 12.7 m) would be expected to initially depress the oxygen concentration in the crew compartment to an average value of 17.5 percent. This figure is based on the assumption that the discharged Halon displaces crew-compartment air with nearly 100 percent efficiency ....[M]inimum oxygen concentrations at various locations...range from about 13 percent to 19 percent, with the lowest concentrations occurring near floor level. Exposure to oxygen concentrations in the 13 to 16 percent range can cause increased breathing and pulse rate, and slight impairment of concentration and muscular coordination.
>
> * * *
>
> [The toxic] effects of exposure to Halon 1301 concentrations in the 7 to 20 percent range are varying degrees of central nervous system depression. Exposures in the 7 to 10 percent range cause mild anesthetic effects including dizziness and tingling of the extremities. Exposures to concentrations above 10 percent are usually accompanied by pronounced dizziness, and reduced physical dexterity and mental acuity.
>
> * * *
>
> Halon 1301 decomposes upon exposure to flame. The decomposition products (hydrogen fluoride, hydrogen bromide, free bromine, and phosgene analogues) are severely irritating (even at low concentrations) to the eyes and the respiratory tract....Therefore, the occurrence of any crew-compartment fire during training necessitates immediate evacuation of the M60A3.[17pp2-9]

It is the risk of exposure to the decomposition products of halon fire-extinguishing agents within enclosed spaces that is the immediate hazard to the crew (Table 13-5). These decomposition products are all significantly more toxic than their parent compounds; even at low concentrations, they are severely irritating to the eyes and respiratory tract. As the intensity of the fire increases, the concentration of the toxic byproducts also increases; in enclosed spaces, halon decomposition products can reach extremely toxic concentrations.

Pharmacological Effects

Although they are relatively nontoxic, numerous fluorocarbon injuries have occurred. The ban on fluorocarbon propellant use has already substantially reduced human exposure, but the Consumer Product

TABLE 13-5

EXPOSURE LIMITS FOR COMBUSTION AND DECOMPOSITION PRODUCTS OF COMMON HALONS

Product	Exposure Limit* (ppm)	Type of Limit
HBr	3.0	Ceiling†
HCl	5.0	Ceiling
HF	3.0	Ceiling
Br_2	0.3	STEL‡
Cl_2	1.0	STEL
F_2	2.0	STEL
$COBr_2$ (Carbonyl bromide)	0.1§	—
$COCl_2$ (Phosgene)	0.1	8-Hour
COF_2 (Carbonyl fluoride)	5.0	STEL

*All exposure limits, except carbonyl bromide, established by ACGIH

†Ceiling: the value above which concentrations must never rise

‡STEL: short term exposure limit

§No value established (phosgene value assumed)

Source: US Department of the Army. *Health Hazard Evaluation and Test Support of the Special Study of Halon Fire Extinguishing Agents.* Washington, DC: DA; 1985. TECOM Project 1-VC-080-060-153.

Safety Commission records for 1975 (prior to any propellant restrictions) show that 5,700 aerosol-related injuries were treated in hospital emergency rooms. These injuries resulted from spraying (66%), inhalation and ingestion (12%), fragmenting of the container (10%), and cold (3%); the causes of 8% were unspecified (the published data were not rounded to 100%).[18]

Acute Exposures

Fluorocarbons as a class have very low toxicity and the predominant hazard they pose is from simple asphyxiation. Early in the history of fluorocarbon use, deaths associated with exposure were usually attributed to asphyxia, but sufficient data were accumulated during the late 1960s and early 1970s to associate mortality with abuse of these products. In particular, deaths due to propellant "sniffing" warranted closer scrutiny. The possibility of fluorocarbon toxicity and abuse was raised in 1970 when the deaths of more than 100 youths who had died while sniffing various aerosol products were investigated.[19] Other research led to similar findings; these clearly documented that fluorocarbons sensitize the myocardium to sympathomimetic drugs, which can lead to severe cardiac arrhythmia and death on subsequent exposure. Further experimental and epidemiological data tend to corroborate the unusual finding that, unlike most other sensitization reactions, the myocardial sensitization is a transient occurrence that quickly and completely disappears if the affected individual is removed from contact with the chemical.[12]

Similar and equally serious consequences resulted from the use of pressurized bronchodilator aerosols. The original bronchodilators were mixtures of the sympathomimetic drugs epinephrine and isoproterenol, which were aerosolized by both trichlorofluoromethane (CFC-11) and dichlorodifluoromethane (CFC-12). During the few years that these bronchodilators were used, physicians began to document a high incidence of bronchospasm and death. By 1968, sufficient evidence had accumulated to ban the over-the-counter sale of these devices.[20]

As a group, the fluorocarbons have a low lipid solubility and are poorly absorbed in the lung. Once in the bloodstream they are apparently not metabolized and are slowly excreted through the lung, unchanged, via expired air.

Chronic Exposure

Because fluorocarbons are rapidly disseminated in the environment, chronic exposures are limited to relatively few occupations. Workers who manufacture and package these chemicals, electronics workers who use them as cleaning solvents on printed circuit boards, refrigeration mechanics, and hospital operating-room personnel appear to have the highest exposure potential. Federal workplace exposure standards have been promulgated for most of the commercially important fluorocarbons and range from 100 to 1,000 ppm for the entire series.[21]

Current epidemiological data indicate that no significant hazards are involved with chronic exposure to most fluorocarbons at low concentrations. Their rapid dissemination limits the possibility of high exposure concentrations in most applications. Some fluoro- and chlorofluorocarbons are used as anesthetic gases; this appears to be the most prevalent route of chronic exposure to chlorofluorocarbons (see also Chapter 5, Health Hazards to Healthcare Workers). The handling and processing of waste anesthetic gases (as well as their intended use) cause the potential for daily exposure to operating room personnel. Epidemiological studies of nurse anesthetists and other female hospital personnel have indicated a correlation between exposure to anesthetic gases and the occurrence of cancers and spontaneous abortions in the study population, and congenital anomalies in their infants. Based on these findings, the National Institute for Safety and Health (NIOSH) recommended severely lowering the occupational standard for anesthetic gases to 5 ppm.[22]

A researcher who performed an extensive mutagenicity study on a series of 21 different chlorofluorocarbons concluded that they are not biologically inert: the series contains bacterial mutagens, cell-transforming agents, and rodent carcinogens. For this series of compounds at least, prokaryotic mutation does not accurately predict carcinogenic potential.[23]

Long-term carcinogenicity bioassays have been completed in rats and mice for the three major chlorofluorocarbons: trichlorofluoromethane (CFC-11), dichlorodifluoromethane (CFC-12), and chlorodifluoromethane (CFC-22). These inhalation studies ran for 104 weeks in rats and 78 weeks in mice. All three of these chemicals failed to demonstrate any carcinogenicity.[24]

Physical Effects

Exposure to fluorocarbons such as Halon 1301 can cause both traumatic auditory damage and cold injury. Military vehicles are frequently equipped with automatic fire-suppression systems that contain fluorocarbons stored under very high pressures (750 psi). These systems rapidly trigger when they sense a fire and quickly flood the vehicle. The extremely high storage pressures and short response times required of the system cause noise levels to be exceptionally high: impulse noise levels greater than 160 dBP have been measured with these systems, a level sufficient to cause permanent auditory damage. Furthermore, transitory subzero temperatures can be produced near the discharge nozzles and cause medically significant cold injury to crew members who are close to the discharge. The Health Hazard Assessment of the M60A3 tank found that

> During gunnery training and M60A3 motion, crew members are likely to be at their normal stations and are required by U.S. Army hearing conservation policy...to wear [hearing protective devices]. At these positions, the impulse noise levels caused by [the automatic fire extinguishing system] activation range up to 161 dBP at over 200 millisecond B-duration. Such levels exceed the 140 dBP hazard criterion but are within the allowable exposure range for personnel wearing [hearing protective devices]. Activation of the [automatic fire extinguishing system] at such time would not expose protected personnel to hazardous impulse noise. If personnel are not wearing [hearing protective devices] and the [automatic fire extinguishing system] is activated, permanent hearing loss may occur with repeated exposure, but is less likely for a once in a lifetime exposure.
>
> ....
>
> The rapid discharge of the 7-pound Halon 1301 bottles has the potential for freezing tissues in the discharge stream....[S]ubzero temperatures would be expected on skin surfaces for very short durations....Cold injuries...have been documented with [the automatic fire extinguishing system] in...vehicles [other than the M60A3] during accidental Halon discharge....[Dippel]

PAINTS

Paints are widely used in industry and the military for aesthetic purposes and also to produce a surface coating for protection against weathering.[2] Workers can be exposed during application of the paint, during drying and curing as a result of solvent evaporation, and during paint-grinding or -stripping operations. For this discussion, the term *paint* refers to a range of solvent-based products including conventional and epoxy paints, varnishes, enamels, and lacquers. Conventional paints consist of a pigment dispersed in a vehicle. Varnishes are usually a nonpigmented resin that is dissolved in a solvent. They dry by solvent evaporation and the oxidation and polymerization of the binder. Most enamel paints are like varnishes, but with a pigment added. Lacquers are clear or pigmented finishes, usually based on a cellulose ester in a solvent, and cure through solvent evaporation.[25] A number of water-based paints and enamels are also available, but because these coatings do not pose a significant inhalation hazard, they will not be discussed further in this chapter. Epoxy paints consist of epoxy resins in reactive diluents. These are mixed with curing agents to create tough, inert surface coatings. Generally the components are not volatile and are not an inhalation hazard.[26]

Solvent-based paints generally consist of three components: a vehicle, fillers, and additives (Table 13-6). The *vehicle*, which includes a binder dissolved in the solvent, makes up the liquid portion of the paint and allows it to be thinned to a consistency suitable for the chosen method of application. The *binder*, either a naturally occurring oil or resin or a synthetic material, cements the paint film to the substrate.[25] *Fillers* include pigments to color the coating and extenders to control gloss, texture, and viscosity. *Pigments* are usually finely powdered, insoluble solids that are dispersed in the liquid medium. In addition to providing color and opacity to the finish, some pigments also act to inhibit corrosion. *Additives* include agents that promote drying, inhibit mildew growth, and prevent the pigment from settling and the paint film from sagging. Although a variety of additives are used in formulating

TABLE 13-6

COMPOSITION OF PAINT

Vehicles	Function	Fillers	Function	Additives	Function
Solvents	Adjust viscosity (thinners)	*Pigments*	Provide color, opacity	*Driers*	Speed drying
Xylene		Titanium dioxide		*Biocides*	Prevent growth of molds and fungus
Toluene		Zinc oxide			
Benzene		Iron oxides			
Naphthas		Lead		*Flattening Agents*	Reduce luster
Perchlorethylene		Chromium			
Mineral spirits		Cadmium			
Methyl ethyl ketone		Aluminum			
Methyl isobutyl ketone		Bronze			
Ethyl acetate		Carbon black			
Trichlorethylene		Lamp black			
Butyl alcohol		*Extenders*	Build body		
Ethyl alcohol		Talc			
Cyclohexanol		Calcium carbonate			
Resins	Form film	Silica			
Alkyd		Bentonite			
Phenolic					
Acrylic					
Vinyl					
Amino					
Cellulose					
Polystyrene					
Polyurethane					
Epoxy					

Source: Burgess WA. *Recognition of Health Hazards in Industry: A Review of Materials and Processes.* New York: John Wiley & Sons; 1981.

paints, they generally compose only a small percentage of the paint.

Toxic Constituents

The toxic constituents of paints are contained in the solvents, pigments, extenders, and resins. Dermal and ocular exposures and inhalation can occur while paints are being mixed or applied; the route and degree of exposure depend to some extent on the method of application. During roller or brush painting, solvent vapors can be inhaled as these constituents volatilize from the freshly painted surface. Spray painting, however, poses a much greater respiratory hazard because both the liquid and solid constituents are aerosolized.

Chronic exposure to the mixed solvents that are found in paints can also cause a neurasthenia that is sometimes referred to as *painter's syndrome*. Toxic symptoms include headache, fatigue, difficulty in concentrating, deficits in short-term memory, irritability, depression, and alcohol intolerance. (These symptoms have also been reported by workers in the plastic boat industry and among jet-fuel handlers).[4] In general, as the severity of the solvent-related effects increase, reversibility becomes less likely.[27] The solvent-related neurasthenia was first described in the Scandinavian literature during the late 1970s, but is still not universally recognized. A recent study evaluated workers in two paint-manufacturing plants in the United States, but failed to document these specific symptoms of toxicity.[28] This study evaluated 187 workers using three standardized psychological/neurological assessment batteries. Exposure durations ranged from 6 to 36 years and concentrations were below the TLVs or PELs. No significant associations were found between solvent exposure and test scores.[29]

Solvents

Paints can contain solvents from virtually every chemical class and toxicity level (described previously in this chapter). Because many are highly toxic, such as benzene, and potentially carcinogenic, such as the halogenated hydrocarbons, attempts have been made in recent years to reduce the use of these toxic solvents in paints by substituting less-toxic solvents.[30] However, the workplace hazard remains significant because workers continue to encounter these agents in older formulations, or as constituents of current paints.

Inorganic Pigments

Pigments provide paint with opacity, color, durability, and film hardness. They typically constitute 20% to 60% of a paint by weight, and are composed of finely divided inorganic solids.[31]

Titanium Dioxide. Titanium dioxide is a white pigment that has virtually replaced the older lead-based white pigments. (It is also used in food products and cosmetics as a whitening agent.) Titanium dioxide is generally thought to be physiologically inert.[3] Other white pigments include calcium carbonate, barium sulfate, and aluminum silicate.[31] Historically, these pigments have been considered physiologically inert, and they have a low order of toxicity. Recently, however, concerns have been raised over *pulmonary alveolar proteinosis*, a physical condition thought to result from exposure to particulates. The condition has been reported among workers exposed to several particulates including the inert dusts. Other studies suggest that these may, in fact, be biologically active, and NIOSH has reported data suggesting that titanium dioxide is a potential occupational carcinogen.[8]

The ACGIH TLV for these white pigments is 10 mg/m^3. OSHA PELs are 10 mg/m^3 for barium sulfate and titanium dioxide, and 15 mg/m^3 for calcium carbonate. OSHA also set a PEL of 5 mg/m^3 as a respirable dust for all three pigments.[7,8]

Carbon Black and Lamp Black. Carbon black, produced during the incomplete combustion of petroleum gas, and lamp black, produced during the incomplete combustion of oil, are the most common black pigments used in paints. Incomplete combustion may produce a variety of polynuclear aromatic hydrocarbons (PAHs), which are likely to contaminate these black pigments. A number of the PAHs have been found to be both mutagenic, when tested in in vitro test systems, and carcinogenic in animal bioassays. No excess tumor incidence has been found in epidemiological studies of carbon black workers, however.[31] The ACGIH TLV for carbon black is 3.5 mg/m^3; OSHA has not set a PEL for this substance. No exposure limits have been set for lamp black.[7]

Iron Oxides. Iron oxides, found in red and brown inorganic pigments, have low dermal and oral toxicity. Workers in the metal or pigment industries who inhale iron oxide dust or fumes may develop a mild form of pneumoconiosis. Because the causal agent is iron, this condition is more specifically termed a siderosis, and apparently does not become fibrotic.[7,8] The ACGIH TLV for iron oxide is 5 mg/m^3; the OSHA PEL is 10 mg/m^3.[7,8]

Chromium. Chromates, used extensively in yellow and orange pigments, can act as sensitizers. Chromate dermatitis has been reported in workers in a number of industries. Exposures to chromium in the chrome-production and -pigment industries have been associated with an increased incidence of respiratory cancers.[32]

Chromium exists in oxidation states ranging from divalent to hexavalent; the trivalent state is the most common form found in nature. The carcinogenic activity associated with exposure to chromium has been attributed to the hexavalent form. Epidemiological studies suggest that the acid-soluble, water-insoluble hexavalent chromium is produced in refining operations. This form is also corrosive and reportedly can cause chronic ulceration and perforation of the nasal septum. In contrast, trivalent chromium is neither irritating nor corrosive.[8]

The ACGIH recommends a TLV of 0.5 mg/m^3 for chromium metal, di- and trivalent chromium and water-soluble hexavalent chromium compounds. Water-insoluble hexavalent chromium is a known human carcinogen and has a TLV of 0.05 mg/m^3. OSHA has a PEL of 0.1 mg/m^3 as a ceiling value for CrO_3.

Organic Pigments

In addition to the inorganic pigments, there are hundreds of organic pigments used in the paint industry. However, data on the toxicity of most organic pigments are limited: in most cases, the only data consist of acute oral LD_{50} values for rodents. The human toxicology of chronic exposures to most organic pigments remains largely unknown.[25]

Extenders

A number of minerals such as silicas, silicate clays, mica, and talc are used as extenders, which act to build body in paint formulations. While the toxicological effects of pigments depend on the specific type of pigment, *all* extenders have the potential to produce

fibrosis of the lung. Limited epidemiological data suggest that a mixed-dust pneumoconiosis is prevalent among painters, which might be due, in part, to exposure to extender materials.[25]

Resins

Resins are polymers. As a group they include the alkyd, acrylate and methacrylate, vinyl, cellulose, epoxy, and polyurethane resins. They have low volatility and are generally soluble in organic solvents and insoluble in water. In paints and varnishes, resins provide film hardness, gloss, surface adhesion, and resistance to weathering.[31]

Alkyd Resins. Alkyd resins form as a condensation product of a polybasic acid and a polyhydric alcohol. Their toxicity is relatively low and they have a long history of use without indication of a chronic hazard.[31]

Acrylate and Methacrylate Resins. Acrylate and methacrylate resins are polymers formed from acrylic and methacrylic acids. These substances are used extensively in latex paints. Unreacted monomers are primary irritants and skin sensitizers. Once they are polymerized, however, they are no longer health hazards. Reacted polymers have a low order of toxicity, and the Food and Drug Administration has approved polymethacrylate as an indirect food additive.[31]

Vinyl Resins. The vinyl resins are polymers of a number of monomers including vinyl chloride, vinylidene chloride, vinyl acetate, and derivatives of styrene. The principal health hazard associated with the vinyl resins is the potential for exposure to unreacted vinyl chloride, which the EPA considers to be a known human carcinogen. Epidemiological studies have associated workers' exposures to the vinyl resins with increased incidence of liver angiosarcoma and, possibly, brain tumors.[33] The ACGIH recommends a TLV of 5 ppm for exposure to the vinyl resins.[7]

Cellulose Resins. The cellulose resins are natural products and include nitrocellulose, cellulose acetate, and cellulose acetate butyrate. These compounds do not present any known toxic hazard.[25,31]

Epoxy Resins. The epoxy resins used most frequently in paints and coatings are usually made by reacting epichlorhydrin and *bis*-phenol-A. Exposures to epoxy resins that have not cured completely have been associated with skin and eye irritation and allergic skin reactions. Liquid epoxy resins are used primarily in two-component epoxy paints. These liquid resins are modified when reactive diluents—which are also skin, eye, and respiratory irritants—are added. Aliphatic and aromatic polyamines and polyamides are also typically used as curing agents in two-component epoxy coatings. These substances are also potential sensitizers and irritants.[25,31]

Because they are not volatile compounds, there are no TLVs or PELs. Operations such as grinding and sanding can produce nuisance dusts, however, and the TLVs for the individual components are then relevant.

Polyurethane Resins. Polyurethane resins are formed by polymerization of an isocyanate such as toluene diisocyanate (TDI). Uncured polyurethane resins contain small quantities of unreacted monomers and these monomers can cause health problems in exposed workers. Isocyanates can cause severe irritation to the conjunctiva, may cause respiratory distress, and are associated with sensitization-type reactions. Inhalation of isocyanate vapors or aerosols can produce asthmalike symptoms including constricted airways, difficulty in breathing, and a dry irritant cough. In sensitized individuals, even a very low exposure to an isocyanate can produce anaphylactic shock or other such dramatic response.[25] The ACGIH TLV and OSHA PEL are both .005 for the unreacted monomer.[7,8]

Additives

Until recently, mercury biocides were added to latex paints as a preservative to prevent bacterial and fungal growth and to control mildew on exterior surfaces. These biocides were derived from mercurial compounds including phenylmercuric acetate (PMA), 3-(chloromethoxy)propylmercuric acetate (CMPA), and phenylmercuric oleate (PMO). At least one report has linked these mercury compounds to acrodynia, a rare form of mercury poisoning found in children. This disease is characterized by pink-colored fingers and toes, peeling of the soles and palms, pain in the extremities, impaired motor control, photophobia, and mental apathy. The EPA, working with paint manufacturers, decided to remove mercury from interior paints. Only PMA will remain registered for use in exterior paints and for miscellaneous interior uses (spackling and patching compounds).[34]

Chemical Agent Resistant Coatings

Until the early- to mid-1980s, military vehicles were painted with standard alkyl and acrylic paints, but these absorbed chemical warfare (CW) agents. Up to 25% of liquid CW agent applied to surfaces painted with standard paints is absorbed within 30 minutes. The CW agent can then desorb slowly over several weeks, creating a residual toxic hazard. Standard alkyl paints are also soluble in decontamination solutions.

To avoid these hazards, the military converted to polyurethane paints (PUPs), which are chemical agent resistant coatings (CARCs), on all combat, combat-support, and tactical-wheeled vehicles, and aircraft and essential ground-support equipment.

There are two primary paints in the CARC system. The first, for exterior use, consists of aliphatic PUP applied over epoxy paint, which is used as a primer coat. The second, for interior use, consists of an epoxy-polyamide paint used over the epoxy primer.[35] Epoxies were initially considered for all CARC applications. However, because they break down with prolonged exposure to ultraviolet light, they can only be used indoors.

PUPs provide a measure of chemical resistance. However, the camouflage pigments and flattening agents required for military vehicles are more porous than are high-gloss paints. CW agents can enter the pores of PUPs, but are not adsorbed to CARC paints—unlike alkyd and acrylic paints—and they can be removed with standard decontamination procedures. CARC paints are also resistant to the components of the decontamination systems.

Two polyurethane topcoats—a two-component paint and a single-component paint—are available. The two-component system incorporates component A (containing the polyester resin, solvent, and pigment), which reacts with component B (an aliphatic polyisocyanate combined with volatile solvents) to form a tough, resilient coating. In the single-component system, the paint cures by reacting with moisture from the air.[35]

Both of the PUP CARC systems contain unreacted isocyanate groups in the uncured resin, which irritate the skin and sensitize the respiratory system. The newer PUP used currently in CARC paints includes hexamethylene diisocyanate. The monomeric hexamethylene diisocyanate is usually reacted to form a higher-molecular-weight prepolymer that is less volatile than the hexamethylene diisocyanate monomer, which tends to reduce the potential hazard from inhalation.[35]

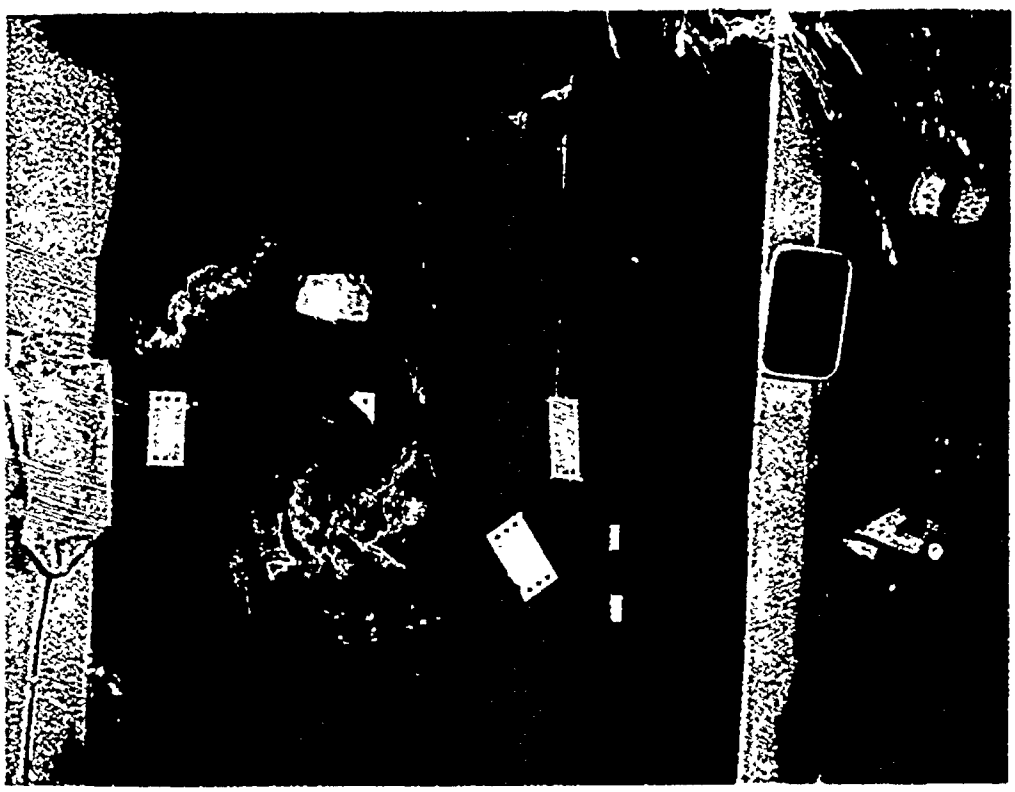


Fig. 13-3. The operator in this walk-in spray booth is wearing coveralls, gloves, a head cover, and an air-purifying respirator. He has also positioned the items to be painted between himself and the booth's exhaust ventilation. This serves two purposes: it minimizes the potential for the painter's exposure and it prevents vapors and aerosols from leaving the spray booth.

Exposure Controls

Industrial painting operations use several methods to control worker exposures and reduce potential health effects. Most flow- and spray-painting operations with solvent-based paints require exhaust ventilation to reduce the airborne paint mists and solvent vapors to acceptable levels. Ventilation controls include spray booths and rooms or tunnels, which are designed to contain the aerosol mists (Figure 13-3). These enclosures are equipped with a filtration system (or mist arrestor) to remove paint mists from the exhaust air. Spray-paint operators must wear effective PPE: goggles and a face mask with a charcoal filter. Effective spray-booth controls also require that the operator not position him- or herself between the object being painted and the point of exhaust. Personnel should also ensure that booths equipped with dry filters receive regular maintenance and cleaning to prevent the filter from clogging.[25]

The method of paint application influences worker exposures. Conventional air spraying is the most common method used in spray-finishing operations. Exposures to paint mists as a result of overspray and rebound are principal hazards of these operations. Airless spray techniques reduce overspray by approximately 50% when spraying flat surfaces. Another application method, electrostatic spraying, involves charging the paint mist so it is attracted to the item to be painted. This technique eliminates almost 90% of the overspray associated with conventional air atomization.[2,25]

Spray-finishing applications in which engineering controls are impractical, or operations in which highly toxic materials are present require that PPE, including respiratory protection, be used. Airline respirators may be required during painting operations in confined spaces such as storage tanks, boilers, ventilation ducts, or other areas where airflow is restricted. In other situations, conventional half-facepiece respirators with mist-removing prefilters and organic-vapor filters may provide adequate protection. Additional PPE should include cloth coveralls, eye protection, gloves, and head coverings. Workers should be prohibited from wearing contact lenses while painting.[35]

MEDICAL SURVEILLANCE

Workers who could be exposed to the constituents of paints should be enrolled in a comprehensive medical surveillance program designed to prevent or control occupational diseases. Exposure to solvents can occur in a variety of occupations and often involves complex mixtures; therefore, the installation's medical authority should determine the specific details concerning the scope and frequency of medical surveillance examinations. The physician can find detailed recommendations on medical surveillance from the Medical Information Module of the Occupational Health Management Information System (OHMIS), which is discussed in Chapter 4, Industrial Hygiene, or from other medical guidance.

Medical evaluations should include a preemployment examination and a regular periodic examination, both of which should include detailed medical and occupational histories. In addition to the standard evaluation for smoking and alcohol use, the physician should pay particular attention to any history of previous exposures to toxic substances, especially organic solvents or other agents associated with neurotoxic effects (see Table 13-6). Due to the neurotoxic effects of solvents, the physician should also consider obtaining a neuropsychiatric evaluation.

Because painters can be exposed not only to solvents but also to the other constituents of paint, they require medical surveillance. The most serious occupational health concerns for painters are their potential for exposures to mixed solvents and isocyanates. During the preplacement or baseline evaluation, the physician should screen for previous exposures to isocyanates. The physician should note the patient's allergies, respiratory diseases, and smoking habits. Because sensitized individuals who are subsequently exposed to isocyanates may have serious allergic reactions, *hypersensitive individuals and workers with a history of chronic respiratory illness should not work with these substances.* The physical examination and clinical tests should also thoroughly evaluate the respiratory system and include, in addition to the routine chest X ray, pulmonary function tests with forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC. A periodic examination should be performed at least annually after the preplacement examination.[35]

For a medical surveillance program to be effective, health education must accompany the evaluations. Health education includes training for both employees and employers, and should provide information on the potential hazards of the chemicals in use, the measures to control exposures, and the proper use of personal protective equipment. Training should also be updated and repeated when new chemicals are added to the workplace.[36,37]

MEDICAL TREATMENT

There are no militarily unique medical treatments for exposure to solvents and paints or to chlorofluorocarbons. In most cases, simply removing the victim from the hazardous environment is the only treatment required. Contaminated clothing should be removed to prevent additional exposure. The physician should ensure that an adequate airway and respiration are maintained. Urinary output should be monitored and fluids administered either intravenously or orally. Diazepam can be used to treat convulsions.

Solvents and Paints

Usually, the only treatment necessary for acute exposures to solvents is removal from the toxic atmosphere. However, acute exposures in high-enough concentrations can cause paralysis, convulsions, and unconsciousness that can progress to death. In instances of ingested solvents (including halogenated hydrocarbons, kerosene, and stoddard solvent), medical personnel must take care to prevent the victim from aspirating the toxic agent into the lungs. Even small quantities of a solvent like kerosene in the respiratory tract can cause pneumonitis, pulmonary edema, hemorrhage, and necrosis. If aspiration occurs, the emergency treatment is the same as for any oily liquid.

Fluorocarbons

As a group, fluorocarbons and chlorofluorocarbons have very low acute toxicity; the greatest hazard of exposure to these compounds is simple asphyxiation. Exposure to very high concentrations (> 50,000 ppm) has been shown to produce CNS depression and eventual respiratory failure. Simply removing the victim from the hazardous environment is usually the only treatment required; all toxic signs rapidly disappear when the patient is removed to fresh air. The most severe threat from fluorocarbon exposure is the ability of these chemicals to sensitize the lungs and myocardium. In some individuals this can cause bronchospasm and cardiac arrhythmias. *Should overexposures to halogenated hydrocarbons—including the chlorofluorocarbons—occur, epinephrine or other sympathomimetic amines and adrenergic activators are contraindicated.* These solvents are cardiac sensitizers and will further sensitize the heart to the development of arrhythmias. They must not be administered.

Toxic Combustion and Decomposition Products

Exposure to the combustion and decomposition products of halons when they are used as fire extinguishers is probably the most significant medical problem posed by chlorofluorocarbons. These products are extreme irritants; consequently, exposed individuals will attempt to escape from the contaminated environment. Personnel inside vehicles or confined spaces, where escape is difficult, may be exposed to long-term, high concentrations of these severe irritants and can suffer chemical burns of the eyes, skin, mucous membranes, and lungs. Medical treatment should be the same as that for any other severe irritant. Pulmonary edema will undoubtedly be a complication if exposure to high concentrations has occurred.

Physical Trauma

Halons used in fire-extinguishing or other pressurized, closed systems can cause hearing damage from excessive noise. Hearing protection should be recommended for areas where the probability of sudden release of these chemicals is high. The subzero temperatures that occur when pressurized fluorocarbons such as the halons are suddenly released can also freeze unprotected skin, eyes, and mucous membranes. Treatment for freezing injury requires no special procedures; the fluorocarbons will rapidly volatilize and the resultant injuries can be treated like any routine cold injury.

SUMMARY

Although large quantities of solvents, fluorocarbons, and paints are used annually in DoD industrial operations, exposure potentials in the military are similar to those in comparable civilian occupations. Despite the increased use of engineering controls and PPE, severe injury or debilitation can result from overexposure to these compounds.

Solvents are used extensively in the military to degrease metal parts, in paints and paint removers, and as intermediates in the manufacture of other items. Most of the organic solvents discussed in this chapter have similar acute, toxic, anesthetic-like ef-

fects to the CNS. Chronic exposures also may affect the CNS; other target organs include the liver, kidneys, and blood. Some solvents, including benzene and the chlorinated hydrocarbons, are animal carcinogens, and some are human carcinogens. Target organs for carcinogenicity are blood, liver, and skin.

Fluorocarbons as a class are relatively nontoxic after acute exposure. Chronic exposure also does not appear to pose a high level of risk. They are potentially toxic to both the cardiovascular and bronchopulmonary systems; this fact is of major significance when medical personnel are required to treat exposed individuals. The greatest medical hazard posed by fluorocarbons is the extreme toxicity of their highly irritant combustion and decomposition products. Current use in the military is significant and will probably rise. As replacement chemicals for banned fluorocarbons are selected, the toxicological and medical databases for them will be fragmentary at best.

Painters can be exposed to a variety of toxic components. The most prevalent exposures are to the solvents. Other constituents of paint can also have toxic effects. However, most constituents of paint are relatively safe in the concentrations used in paint formulations. Allergic sensitization reactions can still occur in sensitized individuals who are exposed to the isocyanates used PUP and CARC paints.

Military occupational health professionals should continue to work with industry to minimize the potential risks through engineering controls and, where possible, through substitution with less-toxic materials.

REFERENCES

1. Donahue BA, Tarrer AR, Dharmavaram S, Joshi SB. Used solvent testing and reclamation. Vol 1. In: *Cold-Cleaning Solvents*. Huntsville, Ala: US Army Corps of Engineers.1988. USA-CERL Technical Report N-89/03, AFESC Report ESL-TR-88-03.

2. Burgess WA. *Recognition of Health Hazards in Industry, A Review of Materials and Processes*. New York: John Wiley & Sons; 1981.

3. Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology* 3rd ed. New York: Macmillan Publishing; 1986.

4. US Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health. *Organic Solvent Neurotoxicity*. Atlanta, Ga: US Department of Health and Human Services; 1987. NIOSH Current Intelligence Bulletin 48; DHHS Publication 87-104.

5. Ayres PH, Taylor WD. Solvents. In: Hayes AW, ed. *Principles and Methods of Toxicology*. 2nd ed. New York: Raven Press; 1989: Chap 4.

6. Snyder R. Hydrocarbons. Vol 1. In: *Ethyl Browning's Toxicity and Metabolism of Industrial Solvents*. 2nd ed. New York: Elsevier, 1987: Chap 5.

7. American Conference of Governmental Industrial Hygienists. *1991–1992 Threshold Limit Values for Chemical Substances and Physical Agents, and Biological Exposure Indices*. Cincinnati, Oh: ACGIH; 1991.

8. 29 CFR, Part 1910.

9. Zenz C. *Occupational Medicine, Principles and Practical Applications*. 2nd ed. Chicago: Year Book Medical Publishers; 1988.

10. Finkel AJ, ed. *Hamilton and Hardy's Industrial Toxicology*. 4th ed. Boston: John Wright PSG; 1983.

11. Banks RE. *Fluorocarbons and Their Derivatives*. London, England: Oldbourne Press; 1964.

12. Aviado DM, Micozzi MS. Fluorine-containing organic compounds. Vol 2b. In: Clayton GD, Clayton FC, eds. *Patty's Industrial Hygiene and Toxicology*. New York: John Wiley & Sons; 1981: Chap 42.

13. Federal Task Force on Inadvertent Modification of the Stratosphere. *Report of Federal Task Force on Inadvertent Modification of the Stratosphere (IMOS). Fluorocarbons and the Environment*. Washington, DC: US Government Printing Office; 1975. GPO Stock No. 038-000-00226-1.

14. *Montreal Protocol on Substances That Deplete the Ozone Layer*. Treaty Document 100-10. December 21, 1987. Addendum to the Vienna Convention for the Protection of the Ozone Layer. Montreal, Canada; 1987.

15. US Department of Defense. *Chlorofluorocarbons (CFCs) and Halons*. Washington, DC: DoD; 1989. DoD Directive 6050.9.

16. US Department of Defense. Director's message. *Standardization and Data Management Newsletter*. 1990;32.

17. US Department of the Army. *Initial Health Hazard Assessment Report on the Crew Compartment Automatic Fire Extinguishing System for the M60A3 Tank*. Washington, DC: DA; 1986. RCS MED 388, Report 69-37-4615-86.

18. Hoffman BL, Klander DS. *Fluorocarbons, Environmental and Health Implications*. Draft Environmental Impact Statement. Rockville, Md: US Food and Drug Administration; 1977.

19. Bass M. Sudden sniffing death. *JAMA*. 1970;212(12):2075–2079.

20. Taylor GJ IV, Harris WS. Cardiac toxicity of aerosol propellants. *JAMA*. 1970;214(1):81–85.

21. US Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health. *Occupational Diseases, A Guide to Their Recognition*. Washington, DC: USDHEW; 1977: 319–321. DHEW publication NIOSH 77-181.

22. US Public Health Service, Centers for Disease Control; National Institute for Occupational Safety and Health. *Criteria for a Recommended Standard...Occupational Exposure to Waste Anesthetic Gases and Vapors*. Washington, DC: USDHEW; 1977. DHEW Publication NIOSH 77-140.

23. Longstaff E. Carcinogenic and mutagenic potential of several fluorocarbons. *Ann NY Acad Sci*. 1988;534:283.

24. Maltoni C, Lefemine G, Tovoli D, Perino G. Long-term carcinogenicity bioassays on three chlorofluorocarbons (Trichlorofluoromethane, FC11; Dichlorodifluoromethane, FC12; Chlorodifluoromethane, FC22) administered by inhalation to Sprague-Dawley rats and Swiss mice. *Ann N Y Acad Sci*. 1988;513:261–282.

25. US National Institute for Occupational Safety and Health. *An Evaluation of Engineering Control Technology for Spray Painting*. Cincinnati, Oh: NIOSH; 1981. DHHS Publication NIOSH 81-121.

26. Hine C, Rowe VK, White ER, White KID, Youngblood GT. Epoxy compounds. Vol 5. In: Clayton GD, Clayton FC, eds. *Patty's Industrial Hygiene and Toxicology*. New York: John Wiley & Sons; 1981: Chap 32.

27. Baker EL, Fine LJ. Solvent neurotoxicity. *J Occup Med*. 1986;28(2):126–129.

28. Lindström K. Behavioral changes after long-term exposure to organic solvents and their mixtures. *Scand J Work Environ Health*. 1981;7(suppl 4):48–53.

29. Boila KI, Schwartz, Agnew J, Ford PD, Bleecker ML. Subclinical neuropsychiatric effects of chronic low-level solvent exposure in US paint manufacturers. *J Occup Med*. 1990;32(8):671–677.

30. Mehlman MA, Smart CL. A comparative toxicological evaluation of paint solvents. In: Englund A, Ringen K, Mehlman MA, eds. *Occupational Health Hazards of Solvents*. Wilmington, Del: E. I. du Pont de Nemours; 1982: 53–67

31. Krivanek N. The toxicity of paint pigments, resins, drying oils and additives. In: Englund A, Ringen K, Mehlman MA, eds. *Occupational Health Hazards of Solvents*. Wilmington, Del: E. I. du Pont de Nemours: 1982: 1–39.

32. Fan AM, Harding-Barlow I. Chromium. In: *Genotoxic and Carcinogenic Metals: Environmental and Occupational Occurrence and Exposure*. Vol 11. In: Fishbein L, Furst A, Mehlman MA, eds. *Advances in Modern Environmental Toxicology*. Princeton, NJ: Princeton Scientific Publishing; 1987: Chap 4.

33. US Agency for Toxic Substances and Disease Registry; Environmental Protection Agency. *Toxicological Profile for Vinyl Chloride*. Syracuse, NY: US Agency for Toxic Substances and Disease Registry, and the Environmental Protection Agency; 1989. ATSDR/TP-88/25.

34. US Office of Pesticides and Toxic Substances. *Mercury Biocides in Paint* Washington, DC: US Office of Pesticides and Toxic Substances; 1990. Environmental Fact Sheet G-11-OE.

35. US Army Environmental Hygiene Agency. *Guidelines for Controlling Health Hazards in Painting Operations*. Aberdeen Proving Ground, Md: USAEHA; 1987. Technical Guide 144.

36. Craft BF. Solvents and related compounds. In: Rom WN, ed. *Environmental and Occupational Medicine*. Boston: Little, Brown; 1983: Chap 44.

37. Weaver NK, ed. *The Petroleum Industry*. Vol 3, No 3. In: Occupational Medicine, State of the Art Reviews. Philadelphia: Hanley & Belfus; 1988.

Chapter 14

PESTICIDES

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SUMMARY

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INTRODUCTION

A *pesticide* is any substance or mixture of substances that prevents, destroys, repels, or mitigates any pest. A *pest* is any animal or plant that can injure the environment or the health of populations in that environment. This definition allows any of the following terrestrial or aquatic plant or animal life to be classified as pests: insects, rodents, nematodes, fungi, weeds, viruses, bacteria, or other microorganisms (except those on or within living humans or other animals). The administrator of the Environmental Protection Agency (EPA) determines which organisms qualify as pests.[1]

As part of a unified effort, all scientists, managers, and those who apply pesticides must consider the potential risks associated with the applications of pesticides before using them. Military goals are to (*a*) use pesticides judiciously and (*b*) minimize introducing these toxic materials into the environment. Nonchemical pest-control measures are given first consideration; chemical controls are initiated only if nonchemical control measures fail, or if the situation dictates that chemical controls are the only option.

Pesticides are unique among toxic materials: to be effective, they must be purposely introduced into the pests' environments. Not only other animals and plants but also humans share this environment with the pests. Excessive residues that result from misapplication and residues that migrate from target areas into areas of environmental concern, such as groundwater, are just two of the serious problems associated with pesticide use.

The risks of using pesticides must be weighed against the benefits. The problem has been and continues to be our inability to fully identify the risks associated with the introduction of pesticides into the environment. For example, the thinning of bird eggshells from the bioaccumulation of dichlorodiphenyltrichloroethane (DDT) and the controversy that surrounded the defoliant Agent Orange (a mixture of approximately 50% dichlorophenol [2,4-D] and 50% trichlorophenol [2,4,5-T], with trace amounts of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin [TCDD] contamination) after the Vietnam War were unanticipated pesticide risks. Although the acute effects on both the environment and human health may be known, there is a paucity of information on the chronic effects that result from long-term exposures to pesticide residues. Therefore, every precaution and form of protection must be taken whenever pesticides are used.

MILITARY USES OF PESTICIDES

Pesticides are necessary in the military to protect (*a*) human health, (*b*) products in storage, (*c*) natural resources, and (*d*) property, just as they are in civilian life. The military has an enormous investment in human resources, facilities, and natural resources, and adequate protection of this investment often requires the use of pesticides.

Protecting Human Health

The mission of the U.S. Army Medical Department (AMEDD) is to conserve the fighting strength. Because arthropod-borne diseases are major risks to human health, this mission requires that pesticides be used; worldwide contingency operations necessitate that AMEDD be prepared to protect its troops adequately against diseases such as malaria, typhus, plague, leishmaniasis, encephalitis, and dengue. In the United States, Lyme disease has emerged as an important tick-borne disease and has renewed the effort for using skin and clothing repellents to protect troops during field exercises. Pesticides used both as personal protection (such as repellents and pediculicides [louse powders]) and area control (such as mosquito larvicides and adulticides) are available to assist in preventing the spread of pest-borne diseases.

To protect against the health hazards associated with pesticides, the military must also conduct adequate risk-benefit analyses before using them. For example, herbicides were used as defoliants during the Vietnam War to help members of the U.S. military see the enemy in the dense jungle. Agent Orange was used widely. Its formulation was contaminated with dioxin, however, and because the contaminant is toxic, controversy raged for years in the United States over the use of Agent Orange. This controversy underscores the need for adequate risk-benefit analyses to be conducted when military forces could be present while pesticides are being applied or might be exposed to them after their application. Specifically, the assessment must evaluate the risk to the troops from the pest (arthropod-borne diseases or dense vegetation providing cover for enemy soldiers) versus the risks to troops if pesticides are used to minimize these risks. If

the benefits of using pesticides outweigh the risks associated with their use, then pesticides should be considered weapons.

Roosting birds can cause serious health problems in hospitals and dusty work areas. Bird feces can contain numerous pathogens that cause psittacosis and such mycotic diseases as histoplasmosis and cryptococcosis. These can be transmitted throughout a hospital via its ventilation system, especially if bird feces contaminate the ducting. When fecal-contaminated dust travels as suspended particulates in air and is inhaled, unprotected individuals can contract clinical illnesses such as mycoses or respiratory diseases, or develop skin lesions or subclinical infections manifested only by subtle changes in specific antibody titers.

The military uses more pesticides to control cockroaches than to control any other pest species. Cockroaches transmit disease organisms mechanically: this is the major reason for the continued effort to control them. They are associated with the spread of enteric diseases such as salmonellosis, dysentery, or typhoid. In addition, there is increasing evidence of allergies in humans, which has resulted from cohabiting indoors with cockroaches. While nonchemical control methods—such as appropriate sanitation and reduction of cockroach harborage—are far more effective in controlling cockroach populations, pesticide treatments in food areas and in military housing continue to be necessary.

Protecting Stored Products

The Stored Products Pest Management Program, conducted within the Department of Defense (DoD), consists of an integrated system of storage, quality assurance, and pest-management activities. These activities are designed to prevent or control insects and rodents that attack infestable stored products such as food and fiber items. Failure to comply with proper storage procedures may result in substantial economic losses caused by pest damage. Pesticides are an integral part of this program. Applied as residual treatments or airborne area sprays, pesticides provide a barrier between the pests and the stored products. Without such protection, insects (such as grain beetles, moths, and weevils) and rodents (such as mice and rats) would contaminate or destroy items such as flour, cereal, rice, and dried fruit.

Protecting Natural Resources and Military Property

The military, with millions of acres of land on its installations and bases, uses pesticides to protect valuable natural resources such as forests and wildlife and protect against pest populations that inhibit military activities or damage property. For example, trees are a valuable natural resource that need to be protected from the gypsy moth. Wooden buildings need to be protected from termites and other wood-destroying insects.

Populations of rodents such as prairie dogs or rats may exceed the natural carrying capacity of the area; rodenticides may be needed to reduce these populations, especially if the burrows impede the use of military equipment in the area or a plague outbreak should occur that may threaten human populations. In these instances, pesticides may be required to reduce the rodent population quickly, rather than rely on natural controls such as predation.

Pests such as birds that congregate near airports are hazardous to aircraft, crews, and passengers; an avicide can promptly reduce the hazard. Birds, particularly pigeons, can also become a serious problem when they roost in warehouses. In addition to their association with illness and contamination of stored foods, roosting birds' acidic feces can deface buildings and accelerate the deterioration of equipment.

Termites and other wood-destroying insects cause tremendous building damage and economic loss on military installations. However, as a result of the environmental persistence of the chemical constituents of pesticides and the potential for adverse human health effects from these constituents, the military uses of pesticides to control termites have changed from the use of persistent chlorinated hydrocarbon pesticides (such as chlordane) to less persistent pesticides (such as chlorpyrifos). These changes in termite management—more frequent applications of less persistent termiticides—have, paradoxically, increased the risk of exposure.

Protecting Against Exposure

The safe application of pesticides requires that precautions be taken to protect against acute or chronic exposures to pesticide residues that may cause poisoning. While acute pesticide poisoning symptoms are well documented, a paucity of information exists on effects of long-term exposure to pesticide residues. For this reason, protecting against the unknown chronic health effects, as well as acute health effects, warrants minimizing human exposure to pesticide residues.

Routes of Exposure

The purposeful introduction of pesticides into the environment can cause not only the worker who applies the pesticide (the applicator) but also unsuspect-

ing bystanders or passersby to be exposed to pesticide residues via (*a*) dermal contact, (*b*) inhalation, and (*c*) ingestion.

Dermal contact, the most frequent route of pesticide exposure, can occur during applications of liquid sprays, dusts, and granules. Preparing mixed or diluted solutions from concentrated pesticides may cause considerable dermal exposure if a spill occurs.

Pesticides can cause mild-to-severe skin injury depending on the particular pesticide and formulation involved. Severe internal poisoning may occur if sufficient pesticide is absorbed through the skin into the blood, and is transported to the internal organs.

Inhalational or respiratory exposures can also occur from pesticides during mixing or application, although this exposure route is usually much less likely than the dermal route. If inhaled, pesticides can be absorbed into the lungs and transported to other organs via the blood.

Ingestion is not usually a significant hazard for careful workers, but it can be a dangerous exposure route, especially if pesticide spray or dust is splashed into the mouth during mixing or application, or if contaminated foods or beverages are consumed. Pesticides can also be ingested if the applicator smokes while mixing or applying them. Ingested pesticides, after passing through the linings of the mouth, stomach, and intestine, are easily absorbed into the blood.

Reducing the Hazards

The hazards that pesticides present to human health are mitigated by using personal protective equipment (PPE) and the appropriate engineering controls; PPE must be used whenever pesticide exposure will occur in excess of accepted action levels[2] or the permissible exposure limit.[3] By consensus, unless otherwise defined, action levels are usually defined as a concentration equal to one-half the Occupational Safety and Health Administration's (OSHA's) permissible exposure limit (PEL), which is the statutory exposure limit. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends acceptable exposure limits, known as Threshold Limit Values (TLVs, a registered trademark of the ACGIH).[2] Protection from exposure should be provided at whichever action level is lowest (either the limit required by OSHA or the limit recommended by the ACGIH).

Protecting Pesticide Workers

Protection against pesticide exposure to military personnel who apply pesticides is afforded by both engineering controls and PPE. Engineering controls are aimed at containing or reducing the spread of pesticides. These measures are particularly important for the activities performed in pest-control facilities, such as diluting and mixing pesticides. Engineering controls can pertain to the design of ventilating systems, plumbing, fire protection, emergency shower and eyewash fountains, and personnel locker and bathing facilities.

PPE provides a barrier that precludes or limits an individual's exposure to pesticides (Table 14-1). Depending on the pest-management operation being conducted, more than one type of PPE may be necessary. For example, a waterproof jacket and pants should be worn if a spraying operation could cause the required coveralls to become wet. In addition, label instructions may indicate that a specific respirator be used with that product. Such respirators might include a dust mask, canister-type gas mask, or self-contained breathing apparatus.

Training is essential for pesticide applicators, to prevent or reduce their potential exposures. Specific issues that must be addressed in training include the benefits of using engineering controls, as well as the appropriate selection and use of PPE. Other essential training issues include the hazards of specific pesticides, possible adverse health effects from exposure, emergency first aid, decontamination procedures, and emergency responses to spills. Workers must be informed that a subtle exposure hazard potential may be associated with pesticides that have long-lasting residues. The training must emphasize that workers could be chronically exposed to pesticides if engineering controls fail to operate properly or if the PPE is defective or worn improperly.

All workers who receive, handle, store, and apply pesticides must

- demonstrate proper handling of pesticides,
- know proper clean-up procedures for pesticide spills,
- initiate appropriate spill notification or reporting, and
- have access to a pesticide-spill kit.

Cleanup procedures, including a notification channel, should be posted in the vicinity of the kit. After training, workers should be able to demonstrate that they are capable of

- recognizing the inherent toxicity of the particular pesticide products they apply,
- using available engineering controls and recognizing when the controls are functioning

TABLE 14-1

PERSONAL PROTECTIVE EQUIPMENT (PPE)

PPE	Requirement
Apron	Waterproof, made from synthetic material or rubber; use for mixing pesticides.
Boots	Waterproof, made from synthetic material or rubber.
Clothing	Clean coveralls or outer clothing; change daily; waterproof jacket or pants if using liquid formulations. Do not wear over street clothes.
Faceshield	Use when handling or mixing.
Goggles or full-face respirator	Use when handling or applying.
Gloves	Waterproof, unlined, made from synthetic material or rubber.
Hat	Waterproof, wide-brimmed with nonabsorbent headband.
Respirator	Cartridge-type approved for pesticide vapors, unless label specifies another type (dust mask, self-contained breathing apparatus).

improperly,
- determining when PPE is necessary,
- properly donning and doffing PPE to limit exposure potential,
- properly disposing of unused pesticides, and
- properly decontaminating equipment.

Protecting Bystanders

It is equally important to protect other people from exposures that may occur during or after the application of pesticides. This type of exposure is insidious because the individual may not be aware of the pesticide and its potential exposure hazard. However, the hazard to these individuals is often largely reduced if appropriate techniques are followed during application and if diluted pesticides are used.

Those who apply pesticides to buildings (offices, homes, barracks, and so forth) should carefully advise the occupants of the potential hazards. The occupants should be instructed to alert the pesticide applicators if there are signs of inappropriate application such as wet spots or puddles. The occupants should also be carefully instructed concerning their time of reentry into the treated building and safety practices associated with baits and traps. Pesticides that have a significant residual time are hazardous to humans if application procedures have been faulty.

PREPARATION OF PESTICIDES

Pesticide chemicals as they are produced by the manufacturer are usually highly concentrated, will not mix well with water, and may not be chemically stable. To enhance storage, handling, and application of pesticides, *carriers* (solvents, clays, surfactants, or stabilizers) are added to the active ingredient to create a pesticide formulation.

Formulations

The most common pesticide formulations are (*a*) sprays, (*b*) dusts, (*c*) granules, (*d*) aerosols, and (*e*) fumigants. Sprays can be prepared from a number of liquid formulations with various carriers such as water, oil, and other adjuncts (Table 14-2). Dusts are finely ground materials of either undiluted pesticide or the pesticide mixed with an inert diluent. Granular pesticides are small pellets manufactured from inert clays that are sprayed with a solution of the pesticide. Aerosols are pressurized sprays that use a propellant to disperse the pesticide, while fumigants are gaseous forms of a pesticide.

Pesticides must be formulated to improve properties such as storage, mixing, application, efficacy, and safety before they can be used. For example, certain ingredients of pesticide formulations are designed to increase water solubility for their use as diluted sprays, or incorporated into solid matrices for their use as granules. The resulting product (or formulation) includes not only chemicals (in pure or technical-grade

TABLE 14-2

FORMULATIONS OF PESTICIDE SPRAYS

Formulation Type	Definition
Emulsifiable concentrate	Concentrated oil solution of technical-grade pesticide to which an emulsifier, a detergent-like material, is added. When added to water, the emulsifier causes the oil to disperse uniformly.
Wettable powder	Pesticide dusts to which a wetting agent is added to facilitate the mixing of the powder with water.
Flowable or sprayable suspension	Blend of a technical-grade pesticide with a dust diluent and a small quantity of water. This finely ground, wet formulation mixes well with water.
Water-soluble powder	Finely ground technical-grade pesticide that dissolves when added to water.
Oil solution	Technical-grade pesticide dissolved in oil and applied as an oil spray.
Ultra-low-volume concentrate	Technical-grade pesticide dissolved in a minimum of solvent. The high concentration of the pesticide's active ingredient (usually > 50%) is applied via special ground or aerial equipment that greatly reduces the volume of pesticide formulation applied.

form) that are active as pesticides but also chemicals that are inactive (or poorly effective) as pesticides. Inactive or inert ingredients also include chemicals that are added to the pesticide, and are intended to improve its distribution and use. In this context, the term inert refers to the pesticide's activity against specific pests and is unrelated to the chemical's inherent potential toxicity to other species, including humans. For example, the formulation's carrier (or vehicle), included to enhance the solubility in water, may be a petroleum-based product that has no direct effect on the pest, but which could be a hazard to those who apply the pesticides if they were to use the product improperly.

Technical-Grade Components

Most pesticides are produced through a series of complex chemical reactions that eventually result in chemically impure mixtures of reaction products. Even when chemically pure, chemicals are used to initiate the industrial process reactions for development of the final pesticide, and the resultant product mixture is chemically diverse. Similarly, naturally occurring pesticides extracted from biological sources are chemically complex mixtures with comparable physical and chemical solvent-extraction characteristics.

Technical-grade pesticide products are complex chemical mixtures that have been developed and licensed for incorporation into commercial pesticide formulations. Technical-grade products contain numerous related, intermediate, production-process-associated compounds. The production of chemically pure organic compounds is technically difficult, expensive, and usually unnecessary to afford acceptable practical activity for use against pests. As a result, technical-grade production chemicals are often used to formulate pesticides and therefore often demonstrate chemical impurities. Technical-grade products derived from controlled chemical reactions conform to a range of chemical diversity. The acceptable range of product purity is defined by the manufacturer's design quality control specifications for each intended use. As a result, chemical mixtures generated for incorporation into pesticide formulations, or subsequent sale for use, usually demonstrate a degree of acceptable variability in the final product.

It is imperative that healthcare providers recognize that pesticides are usually impure mixtures resulting from production-process chemicals that have been combined with other chemicals to produce the final formulation. As such, "pesticides" are not mixtures of pure, chemically discrete unreacted compounds, but are technical-grade quality mixtures, and have solvents or other chemicals added to improve their dispersion or solubility properties.

Similarly, it is important for healthcare providers to recognize that pesticide products stored beyond the

stated shelf life or released into the environment might be contaminated with decomposition products. The presence of technical-grade, intermediate compounds or degradation products can substantially alter the toxic effects of the chemical mixture (in comparison with the pure compound) on biological systems.

The complexity of potential problems can be formidable. For example, the use of chlordane as a termiticide in structures owned by all military services was challenged on the basis of possible long-term sequelae among potentially exposed military members and dependents. All services were required to determine the extent of use of chlordane on every installation, and to define the possible extent of exposures of all family members. It became immediately apparent that sampling and analysis for chlordane products required definition in order to evaluate the possibility for exposure.

The commercial pesticide product chlordane is a complex chemical during both its production and its environmental degradation (Figure 14-1). It is an example of the composition of a technical-grade pesticide. The product is no longer registered for sale to the public, but it is extremely persistent in the environment. As a result, it became the focus of substantial military interest during the 1980s. The controversy concerning potential acute and chronic health effects of this product on military members and their dependents will probably continue to resurface periodically. Therefore, a review of the chemical composition of chlordane serves both as a point of historical interest and as a practical demonstration of the complexity of human exposure and health-risk determination.

Toxicological testing of several of the individual components has resulted in the assignment of differential human-health risks from exposure to the separate compounds of technical-grade chlordane. For example, both chlordane and heptachlor demonstrate variable degrees of activity at the same target organs (ie, both are potential hepatotoxins). Reported differences in acute toxicity in rodents usually indicate that heptachlor is approximately 5-fold more potent than chlordane. In addition, heptachlor has been recognized as a substantially more potent carcinogen than chlordane.[4]

In addition to the concern for environmental degradation over time, the technical-grade composition of chlordane had been reported to change with time. Formulations of chlordane produced before 1951 are known as "early chlordane." The temporal differences in specific composition of technical grade may be related to differences between the incidence of reported hepatoma in mice in 1977 to 1979 and 1989. The later studies have failed to demonstrate an increased incidence of hepatoma reported in earlier studies.[5]

Toxicological and biological activity associated with chemical stereoisomers of the same chemical moiety have been documented for numerous compounds. As an example, the acute toxicity of permethrin is directly correlated with the *cis/trans* ratio, with most mammalian toxicity attributable to the *cis* isomer.[6,7]

The best-recognized example is probably the undesirable human toxicity associated with exposure to an intermediate, unwanted, contamination product found in a technical-grade, market formulation of Agent Orange (Figure 14-2). An intermediate compound in the production of the herbicide 2,4,5-trichlorophenol (2,4,5-T) was found to cause a positive response in the rabbit ear toxicology evaluation.[8] The compound that was subsequently identified as the cause of chloracne in humans and hyperkeratosis in livestock was the contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). The chemical TCDD is an undesired reaction

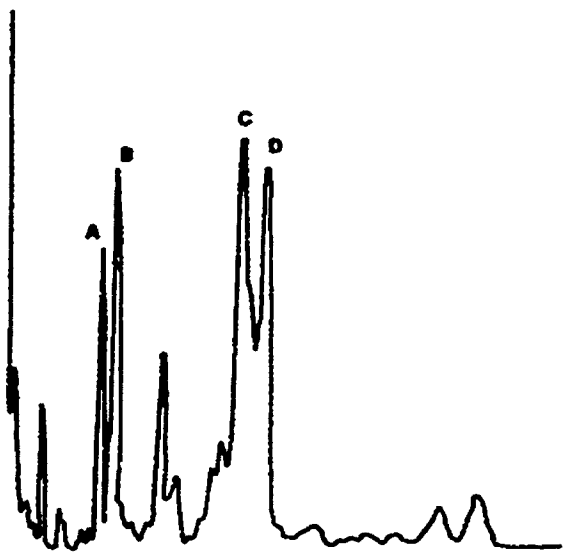


Fig. 14-1. A chromatogram is produced during the analysis of chlordane on federal installations. Careful review reveals a complex composition. Technical-grade chlordane has been identified and selectively defined for compound identification by the ratio of the percentage composition of four of the major components in the mixture. The four major components are electively labeled A, B, C, and D, based on their retention times—from shortest to longest, respectively—using carefully prescribed chromatographic specifications. Peak A is an otherwise unspecified chemical substance known as "compound C." Peak B represents the chemical compound heptachlor, peak C represents *trans*-chlordane, and peak D represents *cis*-chlordane. If the ratio of peaks A+B/C+D approximates 0.8 (range 0.4–1.6), the sample is accepted as representative of technical-grade chlordane.

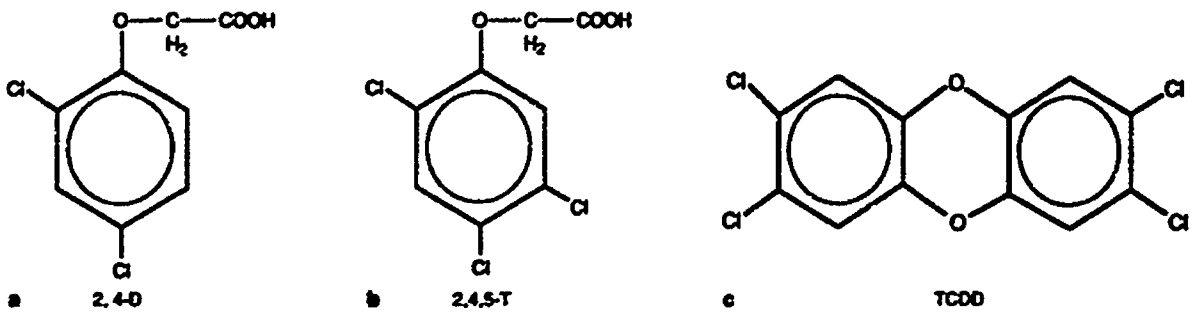


Fig. 14-2. Agent Orange. The chemical structures of the herbicides (a) dichlorophenoxyacetic acid (2,4-D) and (b) 2,4,5-trichlorophenoxyacetic acid (2,4,5-T); (c) represents the structure of the undesirable intermediate dioxin contaminant 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD).

product identified in the production of phenols and hexachlorophene, which is now often chemically removed from technical-grade products before they are formulated. TCDD has neither pesticide nor phenol activity. It is, however, by scientific consensus, the chemical believed to represent the main health hazard associated with exposure to Agent Orange. Another example of undesirable reactions associated with unreacted chemical intermediates was the dermal and respiratory toxicity identified for the chemical hexachlorocyclopentadiene, a contaminant of early chlordane. Specifications for chlordane produced after 1951 limited hexachlorocyclopentadiene concentrations to less than 1%, with a resultant decline in acute adverse dermal and respiratory effects.[5]

In addition to recognizing the differences in toxicity between separate compounds in the marketed pesticide formulation, it is important to recognize the possibility for chemical interactions that may result in exposure consequences for an individual. In selected cases of patient poisonings, healthcare providers may be required to carefully review the available toxicology database for a pesticide to determine if the testing has been done on the isolated, separate, chemical components, technical-grade chemicals, or pesticide formulations.

CLASSIFICATION AND TOXICITY OF PESTICIDES

Pesticides must be approved for use by and registered with the EPA. They can be classified in numerous ways, including their mechanism of action and their chemical structure.

Pesticides do not necessarily kill pests, but in fact may kill, repel, or attract them (Exhibit 14-1). Pesticides are also used as plant regulators, defoliants, and desiccants. Some pesticides are used as *chemosterilants*, which prohibit or limit propagation of the next pest generation. *Growth regulators* retard the growth of plants and insects; *defoliants* remove the leaves of plants; and *desiccants* enhance the destructive drying of plants. Even *antimicrobials* (which include disinfectants, sanitizers, and bacteriostatics) are classified as pesticides and must be registered with the EPA. However, for purposes of this chapter, the word *pesticide* is used in its more traditional sense and is limited to the major categories: insecticides, rodenticides, and herbicides (Table 14-3).

Specific toxicological evaluations to identify potential adverse biomedical responses that could affect workers who apply pesticides, the general public, and the environment are required before the EPA will

EXHIBIT 14-1

CLASSIFICATION OF PESTICIDES BY ACTIVITY

Amphibian and reptile poisons and repellents
Antimicrobial agents
Attractants
Bird poisons and repellents
Defoliants
Desiccants
Fish poisons and repellents
Fungicides
Herbicides
Insecticides
Invertebrate animal poisons and repellents
Mammal poisons and repellents
Plant regulators
Rodenticides
Slimicides

TABLE 14-3

CATEGORIES OF PESTICIDES

Category	Example
Insecticides	
Chlorinated hydrocarbons	DDT, lindane, aldrin, chlordane
Organophosphates	malathion, naled, dichlorvos, parathion
Carbamates	carbaryl, propoxur, carbofuran
Foramidines	chlordimeform, amitraz
Dinitrophenols	dinitrocresol, dinoseb
Organotins	cyhexatin, fenbutatin-oxide
Botanicals	pyrethrum, nicotine, rotenone
Pyrethroids	allethrin, d-phenothrin, fenvalerate
Synergists	piperonyl butoxide, MGK 264
Inorganics	sulfur, arsenic, boron
Fumigants	methyl bromide, ethylene oxide, ethylene dibromide
Microbials	*Bacillus thuringiensis*, Heliothis
Insect growth regulators	methoprene, diflubenzuron
Repellents	diethyl-m-toluamide (DEET)
Rodenticides	
Phosphorus	zinc phosphide, yellow phosphorus
Coumarins	warfarin, fumarin
Indanediones	pindone, diphacinone
Botanicals	red squill, strychnine
Organochlorines	endrin
Herbicides	
Inorganic	ammonium sulfate, sodium tetraborate
Organic arsenicals	cacodylic acid, disodium methanearsonate
Phenoxyaliphatic acids	2,4-D; 2,4,5-T; silvex
Substituted amides	propanil, diphenamid, alachlor
Nitroanilines and substituted ureas	benefin, trifluralin, monuron, diuron
Carbamates	propham, terbucarb
Thiocarbamates	pebulate, metham, butyiate
Heterocyclic nitrogens	atrazine, simazine, prometon, picloram
Aliphatic acids	dalapon, trichloroacetic acid (TCA)
Arylaliphatic acids	dicamba, dimethyl tetrachloro-terephthalate (DPCA)
Phenol derivatives	dinoseb, pentachlorophenol
Substituted nitriles	dichlobenil, bromoxynil
Bipyridyliums	diquat, paraquat

Source: Ware GW. *Fundamentals of Pesticides—A Self-instruction Guide*. Fresno, Calif: Thomson Publications; 1982.

register a pesticide. A number of specific types of toxicological evaluations are considered before EPA registration and as an integral part of the Agency's ongoing review. The classical descriptive toxicological tests are customarily divided into two broad categories: acute or chronic, based on the duration of exposure to the administered toxicant under study.

An acute toxicological evaluation is based on measured biological responses to a single dose (or occasionally several doses) of a test compound within a 24-hour period. When the toxicity of a compound is low, the necessary volume to achieve the required dose often cannot be administered as a single dose, but must be administered in repetitive doses given within that 24-hour period. The observation period following exposure is customarily 7 days, but effects may be recorded for up to several weeks following the administration of acute exposure doses in selected circumstances. The most common acute studies simply record lethal effects; however, some studies record observations of toxic signs such as ataxia, feeding difficulty, or lethargy. (Like its use in clinical medicine, a "sign" in animal studies is defined as a discrete event that can be seen by the observer.)

Toxicological effects are either *quantum* or *continuum* responses. A quantum response is a discrete, yes or no, all or none, numeric phenomenon such as lethal outcome. A continuum response is a graded response

associated with a normal, or accepted, numerical range as well as abnormal levels (those that are reported outside the normal range, such as quantitative concentrations of enzymes identified in a sample of whole blood).

The EPA requires carefully controlled performance and documentation of acute lethality studies, using selected animal models, before a pesticide product can be registered. A specific, acute, toxic response to exposure required by the EPA is the LD_{50} (the dose of the pesticide that is lethal to 50% of the test population of animals under specified test conditions).[1]

Other types of toxicological evaluations that may be useful during the EPA registration process require repetitive administration of test doses of the pesticide over progressively increasing periods of time. Based on the duration of repetitive dosing, toxicology tests have been categorized as subacute, subchronic, or chronic. Subacute toxicological evaluations employ repeated administration of doses of the test compound over a duration of several days to 1 month. Subchronic evaluations are defined as repetitive exposures administered over a period of 1 to 3 months, while chronic studies require administration of the toxicant for longer than 3 months. While subchronic evaluations are customarily used to identify effects of long-term, low-dose exposures on target organs, both subchronic and chronic exposures often focus on the occurrence of carcinogenicity as the measured response. A common form of chronic toxicological study is the 2-year, rodent-lifetime study.

Several additional kinds of evaluations may be required for scientists, regulators, and health professionals to more completely assess potential pesticide toxicity. These evaluations include screening tests for the in vivo or in vitro changes in genetic material that could subsequently be associated with possible carcinogenic sequelae. Of these studies, the most commonly performed screening tool is the Ames test. In this test, the use of a specific strain of histidine-requiring bacteria allows scientists to measure changes in growth and replicative ability of the organisms after exposure to the test compound (see also Chapter 9, Explosives and Propellants). As an adjunct, extracts of rat liver homogenate are added to the usual culture media to simulate the hepatic metabolism of the test material in the "activated" Ames test.

In simplistic terms, positive findings reported from the Ames test result from the interaction of genetic materials and the test pesticide or its activated product. Positive results are recorded following an increased number of bacterial colonies (genetically altered clones) on the test-compound-treated growth media, when compared with the number of colonies recorded on the control bacterial-growth medium. In other words, the potential of the test compound to affect genotoxic alterations is recorded as an increase in the number of colonies on the histidine-deficient, test-compound-treated media, compared to histidine-deficient control culture plates.

Other forms of possible toxicological evaluations include assessments for potential skin or eye effects (the rabbit Draize test), reproductive effects (dominant lethal or multigeneration tests), and human effects (patch testing or epidemiological studies). The approved uses and cumulative toxicity reports associated with any specific pesticide are monitored as an ongoing, continuous EPA administrative process. As a result of these routine reviews or pesticide-use complaints from the general public, a pesticide product registration may be revised or revoked based on accumulated toxicological data and human epidemiological experience.

Both acute and chronic adverse health effects from a host of possible, likely, presumed, and confirmed pesticide exposures have been reported in humans. When applied to the circumstances surrounding human exposures, and subsequent effects, the differentiation between acute and chronic human health effects is based on the total frequency and duration of the exposure profile. Acute exposure effects are those that occur after short-duration, high-concentration, or high-potency exposures. This type of exposure usually results from cutaneous contact during improper application or handling of the more potent pesticide products. Chronic exposures occur over extended periods, with the slow accumulation of the pesticide ultimately resulting in signs or symptoms of exposure. Chronic poisoning can occur as a result of partial failures of PPE, engineering controls, or worker complacency. As a result, repeated exposures to dilute or relatively nontoxic, but biologically cumulative, pesticide products can cause chronic poisoning in pesticide applicators.

"Chronic poisoning" is not comparable to the term "chronic health effect." The health effects associated with chronic poisoning may vary between transient, totally remitting signs or symptoms and permanent, adverse, health sequelae. For example, one who enjoys gardening could suffer exposure to chlordane retained in previously contaminated soil. As a result of the accumulated dose from exposures over time, the gardener could develop a nonspecific convulsive disorder. Correct diagnosis and removal from subsequent exposure could result in complete recovery without adverse sequelae. In comparison, a chronic health effect is a persistent, possibly unremitting consequence of either acute or chronic exposure.

Individuals who have adverse effects after either

acute or chronic exposures are routinely reported in the medical literature in the generic category of "pesticide poisoning." Information gleaned from animal-toxicity studies must be combined with the reported human health effects from pesticide exposure to compile comprehensive disease prevention and medical care programs. Data from both animal-toxicity studies and reported human health effects provide clinically useful information for employee occupational health promotion and symptomatic patient management.

Management of both asymptomatic, healthy workers and symptomatic, poisoned workers can be specifically tailored for the individual, based on the known chemical composition and associated toxic effects of the pesticides in question. Essential information related to biological monitoring, medical surveillance, patient examination, and medical management can be recommended and provided using available cumulative data. However, healthcare providers must exercise caution when human experiences with the pesticide products are limited, or when toxicology information is indirectly extrapolated from animal data without reference to compound-specific, relative interspecies variability.

In workers, acute toxic or chronic exposure effects can occur during manufacture, formulation, mixing, or application of pesticide products. If short-term exposures to high pesticide concentrations occur, the symptomatic onset of poisoning is usually rapid and easily correlated with the coincident exposure profile. In contrast, when relatively low-dose exposures occur over prolonged periods, the pathophysiological responses from the chronic-exposure profile may be extremely difficult to correlate clinically with pesticide-associated sequelae. For example, the etiology of seizure activity associated with acute poisoning during pesticide application is rather easily identified during a cursory history. But the cumulative, chronic retention of pesticide residues may not be recognized as the cause of seizures in the gardener who experiences subtle, prolonged exposures to carbamate pesticides during grounds maintenance, but who does not display the usual associated acetylcholinesterase depression.

Workers associated with pesticide application, the general population, and the environment can be affected either acutely or chronically by exposure to pesticide products or residual products of degradation. Chronic biomedical responses to low-level, long-term exposure profiles may range from no apparent adverse health impact to overt clinical signs of poisoning. The possibility of chronic health impacts resulting from subtle exposures provides the theoretical and philosophical bases for biological monitoring and medical surveillance in pesticide-associated workers.

Pesticides that resist biological degradation or have limited environmental translocation are said to be environmentally persistent; such pesticides can cause subtle, prolonged human exposures. There may be no known correlation between low-level exposures to residual pesticide concentrations and known health effects. However, residual levels of organochlorine insecticides that have low comparative acute toxicities have been associated with the onset of seizures in gardeners, and have been predicted to cause an increased risk for carcinogenicity in exposed populations.[4]

Acute and chronic adverse health effects based on the duration of exposure to pesticides have been reported in humans. Patients said to be "poisoned" or "intoxicated" have adverse clinical signs or symptoms following pesticide exposure. Acute poisoning can result from mixing and applying pesticides, when the exposure to relatively high concentrations of the pesticide occurs over a relatively short time. Obviously, this type of intoxication is of particular concern to individuals who manufacture, formulate, or apply pesticides. These are the workers most likely to be seen with the chronic toxic effects that result from long-term exposures to relatively small quantities of pesticides that produce no apparent acute adverse biomedical effect, but which produce an accumulative, detectable alteration with increasing exposure time.

Workers who apply pesticides and those in the general public who continually experience involuntary exposures secondary to environmental contamination can have adverse health effects from chronic exposure to pesticides. The public may be exposed to pesticide residues on foods, at work, during recreational activities, and in their homes. Pesticides that are resistant to environmental degradation may be extremely persistent and thus may result in prolonged exposure potentials. As noted previously, the environmental degradation products may vary depending on the specific pesticide and environmental circumstances (such as temperature, soil type, and moisture). As a result, it is not possible to make a generic statement concerning pesticide degradation and the possible, consequent, proportional environmental toxicity.

Toxic Categories and Labeling Requirements

When pesticides are registered with the EPA, they are assigned a toxicity category that indicates the degree of toxicity that has been determined by animal experimentation and documented instances of human poisoning. The toxicity classification includes four categories (I, II, III, and IV) for each of five hazard indicators: three related to the LD_{50} associated with

oral, inhalational, and dermal exposures; and two related to topical eye and skin exposure effects (Table 14-4).[9] Within these categories, a particular pesticide may be highly toxic for one hazard indicator, such as the oral LD_{50}, while it has only a slight degree of toxicity by another indicator, such as the dermal LD_{50}. In cases where hazard indicators differ, the most toxic hazard indicator determines the official EPA registration category.

The EPA also requires the use of *signal words* (DANGER, POISON, WARNING, CAUTION) on labels to indicate the potential hazard of the pesticide. The most toxic pesticides are classified as Category I and must display the signal word DANGER. Additionally, if this category is based on the oral, dermal, or inhalation LD_{50}, the product label must contain the word POISON in red on a contrasting background, and the skull-and-crossbones symbol, which is familiar to most adults. Category II pesticides are moderately toxic and are labeled WARNING. Pesticides in Categories III and IV are both labeled CAUTION. With few exceptions, all pesticides are also labeled, "Keep out of the reach of children."

The toxicity classification and corresponding labeling apply primarily to pesticides on the basis of their acute toxicity. If a pesticide is known to cause significant chronic toxic effects in humans (such as carcinogenicity or teratogenicity), the pesticide is also subject to a restricted-use classification, which is noted on the label. This classification requires that the pesticide be applied by or under the direct supervision of trained and certified pesticide applicators.

If medical personnel are aware of the different requirements for EPA registration and of the spectrum of potential adverse health effects that pesticides can cause, they can provide better medical care to patients who present with signs and symptoms of possible pesticide poisoning. Pesticide labels must include medically useful information, such as acute toxicology categorization and a statement of practical poisoning treatment. In addition, medical surveillance, patient examination, and biological monitoring can be specifically tailored to individuals who may be exposed to pesticides, based on the known toxicological effects. As a result of possible differences in toxicological responses between species, however, care must be exercised when toxicological information is extrapolated from the animal model to patients.

Comparative Hazards

The differences between organophosphorus and organochlorine pesticides can be used to illustrate the comparative levels of hazard and the relative potentials to cause adverse health effects (Table 14-5). Organochlorine insecticides were used extensively from the 1940s through the 1960s because they were highly efficacious and were generally less acutely toxic than organophosphorus compounds of the same period. The organochlorine derivatives have recently fallen

TABLE 14-4

TOXICITY CATEGORIES AND HAZARD INDICATORS OF PESTICIDES

Toxicity Category	Hazard Indicators					Signal Word on Label
No./Description	Oral LD_{50} mg/kg*	Inhal. LD_{50} mg/L*	Dermal LD_{50} mg/kg*	Eye Hazards	Skin Effects	
I Highly toxic	< 50	< 0.2	< 200	Corrosive; corneal opacity not reversible within 7 d	Corrosive	DANGER POISON (only if Cat. I based on Oral, Inhal., and Dermal LD_{50}s)
II Moderately toxic	50–500	0.2–2	200–2,000	Corneal opacity reversible within 7 d; irritation persisting for 7 d	Severe irritation at 72 h	WARNING
III Slightly toxic	500–5,000	2–20	2,000–20,000	No corneal opacity; irritation reversible within 72 h	Moderate irritation at 72 h	CAUTION
IV Practically nontoxic	5,000	> 20	> 20,000	No irritation	Mild or slight irritation at 72 h	CAUTION

*Numbers listed apply to the pure substance only; dilutions may be considerably less toxic

TABLE 14-5

SELECTED PESTICIDES: ACUTE LETHALITY LEVELS IN RATS

Pesticide	Acute Oral Lethality (mg/kg) (Rat LD_{50})
Organochlorines	
DDT (powder)	500–2500
Lindane	88–200
Chlordane	150–700
Aldrin	10–74
Organophosphoruses	
Schradan	9–42
Parathion	3–30
Dichlorvos	46–80

Sources: (1) Smith AG: Chlorinated hydrocarbon insecticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press, Harcourt Brace Jovanovich; 1991; Chap 15. (2) Gallo MA, Lawryk NJ: Organic phosphorous pesticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press, Harcourt Brace Jovanovich; 1991; Chap 16.

into disfavor, however, because of their persistence in the environment, tendency for bioaccumulation within the food chain, tendency to accumulate in human tissues, and possible potential to cause human carcinogenicity. Commonly used organochlorine pesticides of the early periods of production and use included DDT, lindane, aldrin, and chlordane.

An example of the perceived safety of the organochlorine class of pesticides is demonstrated by the large number of scientific studies directed toward the evaluation of DDT pharmacokinetics after oral administration in humans. DDT was administered to healthy human volunteers in single doses as high as 1,500 mg and, in separate studies, repetitive doses of 35 mg/person/day for 18 months.

In contrast, many of the early organophosphorus compounds were found to be highly toxic during acute exposure. Some were produced as weapons by several countries as nerve agents, a class of chemical warfare gases. Despite their demonstrated potency, several of the early organophosphorus compounds were developed and used as medications for treatment of myasthenia gravis and glaucoma. When the environmental persistence and the possibility of adverse, cumulative toxicity of the organochlorine compounds were recognized, however, their use fell into disfavor. Less toxic organophosphorus compounds were substituted because of their more rapid environmental biodegradation and increasing availability. Later, selected carbamate pesticides, which were less persistent and more specific against the targeted pests, were substituted for many of the organophosphorus compounds.

Hazard as a Function of Toxicity

The toxicity of a pesticide is not synonymous with the hazard associated with use of the chemical. The hazard involved in the application of a pesticide is a result of the interactions among three elements: toxicity, exposure, and time. This relationship is expressed by the equation

$$\text{Hazard} = \text{Toxicity} \bullet \text{Exposure} \bullet \text{Time}$$

where *hazard* is the risk of pesticide poisoning, *toxicity* is a measure of the pesticide's potential for harm, *exposure* is a multivariate function, and *time* is associated with the cumulative dose.[10] In addition to time, the exposure dosage is dependent on the physical state of the pesticide formulation, application technique, and degree of personal protection. The cumulative association between the exposure concentration and the duration of the exposure is often called the exposure dosage and is expressed by the abbreviation *Ct* (ie, *concentration • time*). If any of the three variables on the right side of the equation equal zero, the hazard also equals zero.

Although a pesticide's toxicity can never actually be zero, selection of an appropriate formulation can reduce the associated hazard. Toxicity can be decreased by selecting a pesticide with (*a*) a lower dermal toxicity, (*b*) a less toxic formulation, and (*c*) a lower concentration for application. As a practical example, a relatively toxic pesticide could represent a diminished hazard if it is applied in a very dilute form or in a formulation that restricts its skin absorption potential.

It is possible to reduce the hazard of application, based on the hazard equation, in several practical ways. For example, a pesticide applicator could elect to substitute malathion for parathion in an eradication effort. As another example, the use of appropriate engineering controls or PPE would serve to decrease the exposure and, therefore, diminish the hazard.

The physical state of a pesticide's formulation influences the degree of hazard associated with its use. In general, solids are less hazardous than liquids. Liquid emulsifiable concentrates are probably the most widely used formulations, but these can pose significant hazards due to the highly flammable solvents that may be in the product. The presence of solvents may result in an increased potential for dermal absorption.

Of the liquid formulations, solutions penetrate skin more readily than suspensions.[10]

The exposure potential and correlated hazard potential are related to both the physical state of the pesticide formulation and the application technique employed. To minimize the hazard potential, the techniques and equipment should allow the pesticide to be directed as accurately as possible at the pest and should minimize the pesticide's unwanted drift or movement.

Indoor pesticide applications present greater exposure potentials to both the applicator and the occupants. Pesticides that are considered extremely hazardous should not be used indoors, especially if the product label does not contain the indoor application site.

Pesticide Selection

While efficacy is a major consideration in the selection of a pesticide, selection should be influenced equally by the inherent hazard, of which the acute toxicity of the active ingredient plays an important role. One simple method to evaluate the suitability of several pesticides is to compare the ratio of mammalian to pest toxicity. The higher the ratio of the LD_{50} for a mammal, such as a rat, and the lower the LD_{50} for a given pest, such as a German cockroach, the lower the potential hazard for humans. That is, the higher the mammalian LD_{50}, the safer the product may be for humans, while the lower the LD_{50} for a pest species, the less active ingredient may be required to control the pest. Although toxicity/efficacy ratios provide an indication of relative hazards, they do not provide information on the effectiveness of the pesticide against the pest in the field.

Other factors that influence the level of control that is achieved for a specific pest population include the pest's resistance to the pesticide, the application techniques, and the type of surfaces treated. For example, military use of the pesticide d-phenothrin was seriously hampered when cockroach resistance to this pesticide was found. The application of pesticides to cracks and crevices, rather than broad baseboard treatments, has been found to be more effective because the pesticide is placed in locations where the pest is more likely to be found. Some surfaces such as wood, concrete or surfaces painted with latex paint may absorb the pesticide application, reducing the amount of surface pesticide available when contacted by the pest.

Although oral and inhalational toxicities may be significant, dermal absorption may represent a more significant hazard to those who apply pesticides. Careful selection by comparing the dermal toxicities of selected pesticides and calculating dermal toxicity versus efficacy ratios may be helpful in keeping the applicators safe.

EXPOSURE EPIDEMIOLOGY

There are no published epidemiological data about pesticide exposures resulting from military applications. Because of the federal regulatory requirements, however, civilian and military applications of pesticides are similar; therefore, data accumulated through civilian reports may represent potential qualitative health effects in military applications.

The requirements for reporting medical data are stringent in California, especially for worker's compensation: health-effects data from patients with suspected acute pesticide poisonings have been collected and reported for more than 40 years.[11] As a result of the increasingly stringent medical reporting requirements, these data may be the best quantitative source on which to base estimates of the potential for symptomatic human pesticide exposures, both occupational and nonoccupational. The epidemiological value of the data is compromised (degraded), however, by a number of factors that influence the adequacy of the data collection. In an uncompromised quantitative ratio, the number of patients who demonstrated signs or symptoms caused by pesticide exposure (the numerator) would be compared to the total number of individuals with pesticide exposure (the denominator). Uncertainties that could influence the numerator include physiological differences among patients, failure of minimally or mildly symptomatic patients to seek healthcare, failure of medical personnel to report all pesticide-associated illnesses, and the possibility that the etiology of a presumed pesticide-associated illness is inaccurately assigned.

Even if technically flawed for strict scientific extrapolation, the California epidemiological data provide valuable insights into the magnitude of potential pesticide exposures and the subsequent health effects. Another indicator of the widespread potential for pesticide exposure (and the subsequent health effects) is the quantity sold: 268,749,526 kg of pesticides were sold in California in 1988.[11] The *California State Abstract* for 1989 estimates that the population of California was 28,514,000 in 1988; therefore, pesticide use would have approximated 9.5 kg of pesticide per Californian.

Of these quantities, approximately 49% were used in agriculture, 17% in the home or garden, 19% in industry, and 13% in institutions within the state. The *U.S. Census of Agriculture* for 1987 estimates that about 31 million acres were available for agricultural use in California in 1988. If all the purchased pesticides in the state were applied on agricultural land within its borders, approximately 8.7 kg would have been applied per agricultural acre.

The earliest summary of information that provides insight into the epidemiology of pesticide poisonings was collated in 1950. In that year, a total of 293 cases of reported occupational diseases were associated with agricultural chemicals.[11] Unfortunately, absolute numbers, rather than incidence rates, of poisonings were reported. For 1950, in California, the profile of the leading causes of pesticides poisoning was parathion (n=52), DDT (n=27), sulfur (n=21), arsenic (n=9), nicotine (n=8), and tetraethyl pyrophosphate (n=6).

The introduction of about 500 new EPA-registered pesticides between 1949 and 1970, associated with more carefully regulated use practices and withdrawal of registration for some compounds, resulted in a change in the exposure profile. In 1987, approximately 17,000 pesticide poisonings were reported in California; of these, 1,507 were occupational illnesses. Of the several hundred registered products identified, the most serious poisonings were reported from exposures to the cholinesterase inhibitors and methyl bromide.[12] The organophosphate insecticide parathion was the most frequently reported pesticide correlated with systemic poisonings (n=90) in California in data reported for the years 1982 through 1986; these data are similar to those reported in 1950. Mevinphos (n=58), methomyl (n=51), methamidophos (n=44), methyl bromide (n=32), sulfur (n=28), dimethoate (n=27), dinitrophenol (n=25), methidathion (n=22), and malathion (n=20) were the next-most-commonly reported compounds.[12]

The American Association of Poison Control Centers reported 1,581,540 human poison exposures in 1989. With respect to the poisonings caused by pesticides, insecticides were the most commonly reported class with regard to the frequency of poisoning report, number of patients treated in healthcare facilities, number of symptomatic patients, and number of patient lethalities (n=12). Of the insecticides, organophosphates were by far the most commonly reported poisons in all age groups and were associated with seven of the reported deaths. Arsenicals, the next-most-implicated class of pesticides, were associated with three deaths.[13]

PHARMACOLOGY OF PESTICIDES USED BY THE MILITARY

Like other chemicals, pesticides can be categorized a variety of ways. The characteristics pesticides are most commonly used for are (*a*) mechanism of action (eg, the cholinesterase-inhibiting substances); (*b*) chemical composition (eg, the chlorinated hydrocarbons); (*c*) target pest classes (eg, the rodenticides); and (*d*) source of derivation (eg, the botanical extracts). Although the pharmacology of pesticides is not militarily unique, medical officers need to be familiar with

- the classes of pesticides most frequently used by the military,
- their mechanisms of toxicity,
- the manifestations of acute poisoning,
- the antidote, reversal, or therapeutic intervention,
- the biological monitors and surveillance parameters, and
- the potential long-term effects.

Table 14-6 summarizes these aspects of the following pesticide classes: organophosphates, carbamates, chlorinated hydrocarbons, anticoagulants, boric acid, and the pyrethroids. This chapter treats only the major pesticides that are used by the military.

Information concerning the pharmacology of pesticides and the medical management of poisonings accumulates exponentially. Consequently, most military emergency rooms subscribe to information sources such as the POISONDEX Information System.[14] Information compiled from this source is the basis for much of the management practices that follow. The three-volume *Handbook of Pesticide Toxicology*, published in 1991, thoroughly reviews the current, scientific literature related to general pesticide toxicology.[15] Additional sources of information concerning pesticide toxicity and the emergency medical response to poisoning include

- local poison control centers;
- the EPA publication *Recognition and Management of Pesticide Poisonings*, which provides valuable information for emergency management of a wide range of pesticide intoxications;
- the National Pesticide Telecommunications Network (the telephone number is 1-800-858-7378); and
- the DoD Pesticide Hot Line (the telephone number is 1-410-671-3773).[16-18]

TABLE 14-6

MECHANISIMS OF ACTION AND RECOMMENDED MEDICAL MANAGEMENT OF SELECTED PESTICIDE CLASSES

Chemical Class	Mechanism of Action	Manifestations of Acute Poisoning
Organophosphate	Cholinesterase inhibition, phosphorylation, time-dependent aging	Muscarinic, nicotinic, and CNS effects
Carbamate	Cholinesterase inhibition, carbamylation (rapidly reversible without aging)	Muscarinic, nicotinic, and CNS effects
Chlorinated hydrocarbon Chlorinated ethane derivative (eg, DDT)	Na, K, Ca channels	Highly variable, nonspecific: psychological, sensory, motor
Chlorinated cyclodiene, (eg, dieldrin)	CNS stimulation (transmitter release at synapse)	Convulsions Cardiac arrythmias possible
Anticoagulant (rodenticide baits)	Antimetabolites of vitamin K	Internal hemorrhage Prolonged prothrombin times, depressed levels of factors II (prothrombin), VII, IX, X
Pyrethroid (repellants, insecticides)	Delayed closure of Na channels	Skin irritation, allergy Eye irritation, allergy Paresthesia Allergic bronchospasm Rare: salivation, tremor, vomiting, incoordination
Boric acid (roach control)	Metabolic acidosis Electrolyte abnormalities	Nausea, vomiting, diarrhea, anuria, electrolyte imbalance, tremors, convulsions, skin erythema progressing to desquamation

Cholinesterase-Inhibiting Insecticides

Military applications of insecticides are usually restricted to selected organophosphates (including chlorpyrifos, parathion, diazinon, and malathion) and carbamates (including aldicarb, propoxur, and carbaryl), which are cholinesterase inhibitors; and organochlorines and borate derivatives, which are discussed later in this chapter.

The toxicities of these insecticides vary widely depending on their route of absorption, pharmacological interactions after absorption, degree of metabolic degradation, degree of reversible enzymatic binding, and rate of excretion. Although these insecticides can also be absorbed via inhalation or ingestion, most occupational effects have been reported following dermal exposures.

Organophosphate and carbamate insecticides exert

Antidote, Reversal, Therapeutic Intervention	Biological Monitor and Surveillance Parameter	Long-Term Effects
Atropine (antidote) 2-PAM Cl (reversal) Anticonvulsant	RBC acetylcholinesterase (AChE), (detectible acute or chronic decremental change from individual baseline) Plasma cholinesterase (detectible acute decremental change)	Delayed neuropathy possible Possible neurotoxic esterase effect
Atropine (antidote) 2-PAM Cl CONTRAINDICATED Anticonvulsant	RBC AChE (possible short-term, acute depression; usually near normal level)	None reported
No known antidote	Specific chemical analysis possible; standard levels unknown; results related to known toxic response case reports	Bioaccumulation in lipid tissues Environmental persistence
Anticonvulsant (cholestyramine may enhance biliary excretion of some compounds)	Chemical epoxides in some cases	Possible carcinogenesis
Vit. K_1 is the SPECIFIC ANTIDOTE (K_3 and K_4 ineffective), fresh blood for bleeding	Prothrombin time	Warfarin reported teratogenic
Topical corticosteroids Topical therapy Vit. E oil for paresthesia Antihistamines, occasionally bronchodilators or steroids No known antidote	None reported	Possible carcinogenesis
Syrup of ipecac for acute indigestion (charcoal not efficacious) Anticonvulsant, monitor EKG, Fluid management, dialysis Topical skin therapy	Serum borate level (mg/L blood) (0–7.2 = normal; < 340 mg/L rarely shows toxic effect)	No direct sequelae reported

their pharmacological influences as a result of their inhibition of a family of cholinesterase enzymes in various tissues. There are clear toxicological similarities between both classes of cholinesterase inhibitors, but there are also significant differences between their pharmacodynamic interactions and the recommended medical therapeutic managements. Regulatory controls introduced by the EPA, greater awareness of the associated hazards by applicators, and an intensive effort by pesticide producers to develop safer, more efficacious products, have resulted in a decline in both toxicity and the numbers of human poisonings associated with these chemicals. As a group, the ready availability, widespread use, and relatively toxic nature of the cholinesterase inhibitors still cause a number of human fatalities annually.[13,17]

To review, an enzyme is a protein molecule that induces chemical changes in another molecule with-

out being changed itself; therefore, an enzyme is a catalyst. The substrate is the molecule on which the enzyme exerts an influence and that is changed to generate the enzymatic reaction product. For example, the enzyme acetylcholinesterase catalyzes the hydrolytic chemical conversion of the neurotransmitter acetylcholine. An inhibitor is a chemical that competes with or prohibits the enzymatic interaction with the substrate. Many enzymes are named on the basis of the customary substrate or characteristic chemical reaction type, followed by the terminal identifier *-ase*. For example, the enzyme acetylcholinesterase catalyzes the hydrolytic chemical conversion of the neurotransmitter acetylcholine.

Cholinesterase enzymes are widely distributed within the body. They are localized within numerous tissues, fluids, and cells including heart muscle, cholinergic synapses, myoneural junctions, plasma, and erythrocytes. Although a number of different cholinesterase enzymes are known to be distributed through the body, this discussion will focus specifically on the enzyme acetylcholinesterase, the cholinesterase enzyme characteristically associated with the erythrocyte. A different enzyme of the cholinesterase class, pseudocholinesterase (also called butyrylcholine esterase), is found in the plasma fraction of whole blood. Although the chemical substrate interactions and substrate degradation mechanisms are similar between these two types of cholinesterase enzymes, they differ with respect to their preferred substrate, rate of enzymatic activity, site of production, and rate of regeneration after poisoning. These differences will be discussed later in this chapter, with reference to specific pesticide poisoning, emergency medical management, and occupational surveillance.

To catalyze the hydrolysis of acetylcholine, its preferred natural substrate, the acetylcholinesterase protein molecule utilizes a specific, selective binding site (Figure 14-3). The acetylcholine binding site is thought to be composed of two specific binding areas that have been identified on the enzyme molecule.[18] A negatively charged *anionic* site of the enzyme is believed to attract and form an electrostatic bond with the positively charged nitrogen atom of the choline molecule of the transmitter acetylcholine. The ionic bond, and secondary attractions between the methyl groups of the choline moiety, and surface of the enzyme molecule appear to be prerequisites to the bond formed between the protonated acidic carboxyl group of the acetylcholine ester molecule at the *esteratic* site.[19] In the process of acetylation, a covalent linkage forms between the transmitter and enzyme at the esteratic site of the enzyme. Under normal metabolic conditions, the enzyme-substrate complex rapidly dissociates to form a molecule of choline and an acetylated enzyme. The acetylated enzyme undergoes a hydrolytic reaction, releasing acetate and regenerating the active enzyme.

Cholinesterase-inhibiting insecticides preferentially bind with cholinesterase enzymes, and as a consequence, cause interference with the normal enzymatic activity. This causes acetylcholine, the substance responsible for impulse transmission, to accumulate. Poisoning occurs in exposed humans because of interactions between the inhibitors and the enzyme within central and peripheral cholinergic synapses and within myoneural junctions. As a consequence of its functional activity, this enzyme controls nerve-impulse transmission from nerve fibers to autonomic ganglia, muscle, glandular cells, and other nerve cells within the central nervous system (CNS). Should an individual be exposed to a sufficient dose of cholinesterase inhibitors, the loss of the enzyme function results in the accumulation of acetylcholine at the cholinergic receptor sites. The pathophysiological response to

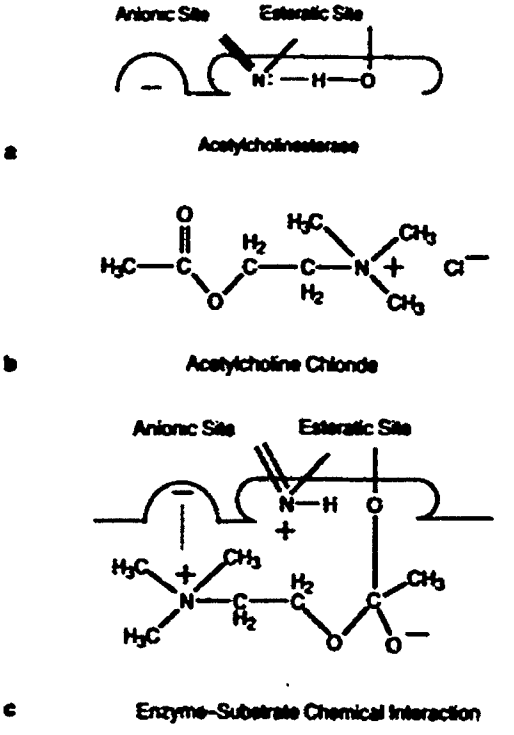


Fig. 14-3. These diagrams illustrate (a) the active site of the acetylcholinesterase molecule; (b) its usual natural substrate, acetylcholine chloride; and (c) the enzyme-substrate interaction. The electron-rich anionic site of the acetylcholinesterase molecule is represented as a negatively charged area; the serine residue at the esteratic site is represented in the hydroxylated state.

unimpeded overstimulation of neuromuscular tissues by acetylcholine is manifested by a spectrum of potential medical signs and symptoms related to the degree of poisoning. While clear toxicological similarities are demonstrable between the organophosphate and carbamate insecticides, there are significant differences between the pharmacodynamic interactions and recommended medical therapeutic management.

Organophosphates

The first organophosphate pesticide, the highly toxic compound tetraethyl pyrophosphate (TEPP), was introduced as an insecticide in 1939, although it was initially chemically synthesized and identified in 1894.[20] Closely related, highly toxic compounds later classified as nerve agents were identified during attempts to synthesize alternative pesticides and were secretly produced in Germany during World War II. Although they were never used as insecticides, warfare nerve gases such as tabun and soman were produced in substantial quantities and stored for possible use during the war. The partnership between applied toxicology and the chemical pesticide industry has resulted in the production and use of safer, more specific organophosphate pesticides for the target pest.

Many organophosphate pesticides have been developed, but those most commonly used for military applications are chlorpyrifos, diazinon, and malathion (Table 14-7).[20] Parathion is occasionally used; however, the EPA has currently been reviewing its registration. As a result of the intense scrutiny, the registration of parathion might be revised by the manufacturer with EPA approval, or revoked or revised by the EPA.

Route of Exposure. Most organophosphate insecticides are readily absorbed by all routes of exposure. Intentional ingestion of pesticide products is a commonly reported form of attempted suicide. Therefore, much of the information on medical management of organophosphate poisoning is derived from suicide attempts. Military scientific and medical experiences with the chemically similar nerve agent war gases have resulted in substantial contributions in the basic scientific literature and accumulated medical knowledge concerning organophosphate pesticides.

While some compounds such as mevinphos are more toxic when the exposure route is dermal, most of these compounds are more toxic if they are ingested. Some of the compounds, such as trichlorfon and diazinon, are severalfold more toxic if ingested or inhaled than if they absorbed through the skin. Chlorpyrifos and malathion are readily absorbed and may manifest equally toxic effects as a result of cutaneous contact, inhalation, and ingestion.[14]

Mechanism of Action. Much of the toxicological information concerning the mechanism of action of organophosphates has been gained through study of the more toxic chemicals such as the nerve agents and parathion.

Organophosphate insecticides interact with the esteratic site of the acetylcholinesterase enzyme molecule by the process of phosphorylation. The interaction, and subsequent chemical events that may occur between the enzyme and insecticide inhibitor, can be shown by the equation:

$$EOH + IL \leftrightarrow \{EOH\}\{IL\} \rightarrow L^- + H^+ + EOI \rightarrow EOH + I^- + L^-$$

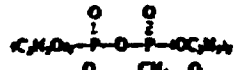
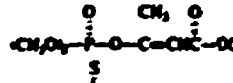
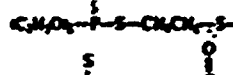
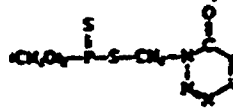
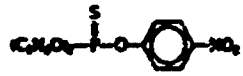
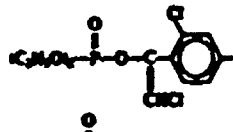
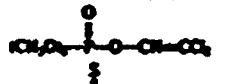
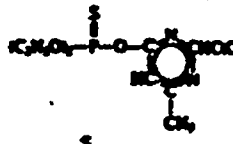
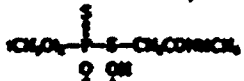
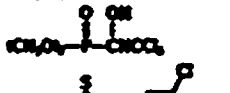
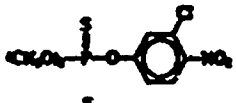
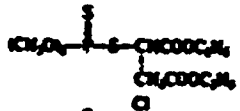
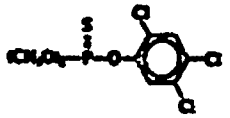
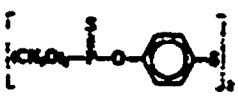
where *EOH* represents the enzyme, *IL* represents the pesticide, *{EOH}{IL}* represents the reversible enzyme-insecticide complex, L^- represents the leaving group, H^+ represents the hydrogen ion, *EOI* represents the phosphorylated or carbamylated enzyme, and I^- represents the pesticide remnant that remains after hydrolytic dephosphorylation or decarbamylation has occurred.

Hepatic metabolic activity may influence organophosphate pesticide activity as a result of pesticide degradation, activation, or both.[4,14,20] In addition to the enzymatic degradation of organophosphate pesticides through the acetylcholinesterase pathway, hepatic metabolization of organophosphate insecticides is sometimes important in pesticide detoxification. Enzymes that perform phase I metabolic activation in the liver—through hydrolysis, oxidation, or reduction of the parent insecticide—are primarily localized within the hepatocyte endoplasmic reticulum. Hydrolytic, oxidation, or reduction rates and the types of metabolic products vary, depending on the particular pesticide. With some insecticides, breakdown may be sufficiently slow that temporary storage of the pesticide can occur in body fat. With other insecticides (eg, parathion, chlorpyrifos, and malathion), the metabolites of hepatic enzymatic pathways are more potent than the parent compound (the marketed pesticide). For example, the hepatic metabolites paraoxon and malaoxon cause much more pronounced toxic (cholinesterase inhibitory) effects than those produced by the parent compound.[4,14,20] For those compounds that are metabolically activated by the liver (including parathion, malathion, and chlorpyrifos), the onset of signs and symptoms may be delayed for several hours after exposure because the actual toxicity is almost exclusively due to its metabolic product, an oxygen analog.[14,15]

If the chemical reaction of the phosphorylated cholinesterase enzyme complex (EOI) results in hydrolytic dealkylation rather than dephosphorylation, an

TABLE 14-7

TOXICOLOGY OF SOME ORGANOPHOSPHATE INSECTICIDES

COMPOUND	STRUCTURE	LD50 IN SMALL RATS (mg/kg)*		"NO EFFECT LEVEL"*	ADI‡
		Oral	*Dermal*	*mg/kg/day*	*mg/kg*
TEPP		1.1	2.4	—	—
Malathion		6.1	4.7	—	—
Disulfoton		6.8	15	—	—
Azinphosmethyl		13	220	Rat—0.125 Dog—0.125	0.0025
Parathion		13	21	Rat—0.05 Man—0.05	0.005
Methylparathion		14	67	—	—
Chlorfenvinphos		15	31	Rat—0.05 Dog—0.05	0.002
Dichlorvos		80	107	Rat—0.5 Dog—0.27 Man—0.005	0.004
Diazinon		108	200	Rat—0.1 Monkey—0.05 Dog—0.02 Man—0.02	0.002
Dimethoate		215	200	Rat—0.4 Man—0.04	0.02
Trichlorfon		650	>2000	Rat—2.5 Dog—1.25	0.01
Chlorthion		800	1500—4000		
Malathion		1375	>4000	Rat—0.5 Man—0.02	0.02
Bromel		1250	>3000	Rat—0.5 Dog—1.0	0.01
Abate		8000	>4000		

*Values obtained in standardized tests in the same laboratory

†Maximum rate of intake (usually 3-mo, 2-yr feeding studies) that was tested and did not produce significant toxicologic effects (as listed in the monographs issued jointly by the Food and Agriculture Organization of the United Nations and the World Health Organization, as developed by joint meetings of expert panels on pesticide residues held annually, 1965–1972)

‡Acceptable daily intake (ADI) = the daily intake of a chemical that, during a lifetime, appears to provide the practical certainty that injury will not result (in man) during a lifetime of exposure. Figures taken from World Health Organization (1973).

Reprinted with permission from Murphy SD. Toxic effects of pesticides. In: Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd ed. New York: McGraw-Hill; 1986: 529.

irreversible enzyme-phosphate product (phosphoryl adduct) is generated.[16] As a result, the enzyme cannot be regenerated and remains inactivated; that is, it is said to become *aged*. The reaction can be shown by the equation:

EOI —> alkyl group product + phosphoryl adduct

For example, in the enzymatic reaction with the organophosphate diisopropyl fluorophosphate (DFP), the initial enzyme-DFP complex releases its leaving group (fluoride) as a result of the hydrolytic reaction. The reaction results in the generation of the phosphorylation product, a diisopropylphosphoryl-enzyme complex and hydrofluoric acid. Subsequent hydrolytic dephosphorylation of the phosphorylated enzyme complex results in the release of a phosphoric acid derivative, diisopropyl phosphate, and the regeneration of the active enzyme. Because the rate of dephosphorylation is slow for the diisopropylphosphoryl-enzyme complex, an alternative degradation pathway through hydrolytic dealkylation is possible. As a consequence, the concentration of the enzyme-monoisopropyl phosphate complex (the aged enzyme) rises following DFP exposure. The rate of organophosphate-enzyme detoxification by hydrolytic dephosphorylation and resultant active enzyme regeneration strongly depends on the type of organophosphorus inhibitor involved. (In contrast, as a class, carbamate insecticides are considered *reversible* inhibitors of cholinesterase and are not associated with aging.[20])

Parathion is another organophosphate insecticide that ages the cholinesterase molecule (Figure 14-4). During the conversion process, parathion is metabolized to the active cholinesterase inhibitor, paraoxon, by desulfuration within the endoplasmic reticulum. Paraoxon reacts with the esteratic site of cholinesterase by phosphorylation. As a result of dearylation, *p*-nitrophenol is released as the leaving group from the initial, transient enzyme-insecticide complex. After dearylation, the phosphorylated-enzyme complex is very stable, with only limited subsequent hydrolysis to regenerate the active enzyme and release diethyl phosphate. Most of the phosphorylated complex is slowly converted to the extremely stable (aged) ethylphosphonate-enzyme adduct by dealkylation.[19,21]

Reactivation of a phosphorylated enzyme is possible using oxime therapy. However, reactivation of the aged enzyme-phosphorylated adduct complex is not possible and is, therefore, refractory to oxime therapy. The rate of pesticide-enzyme complex aging depends on the specific organophosphate inhibitor involved. For example, the nerve agent soman rapidly reacts with the enzyme through phosphorylation and the enzyme becomes aged within seconds to a few minutes. Other selected organophosphate-enzyme complexes may be reactivated 1 or 2 days after initial binding. The aged acetylcholinesterase associated with erythrocytes in the peripheral circulation is normally regenerated only as a consequence of erythrocyte

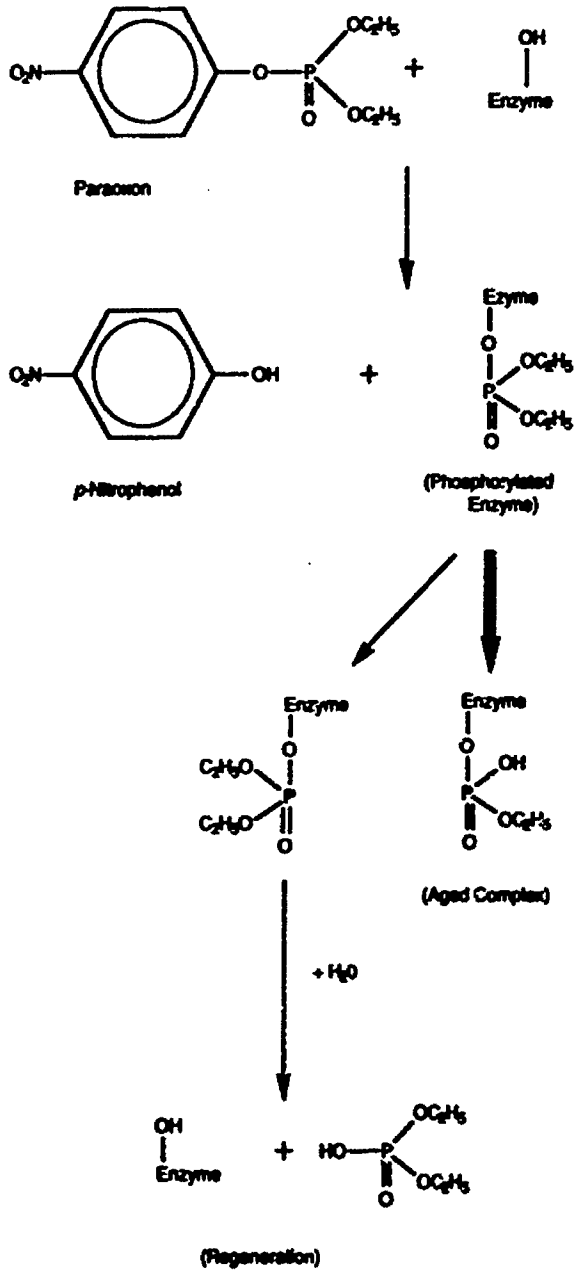


Fig. 14-4. The acetylcholinesterase molecule ages after organophosphate poisoning with parathion. The chemical reactions between paraoxon, the active chemical metabolite of parathion, and acetylcholinesterase favor enzyme aging; enzyme regeneration also occurs, but at a slower rate.

production and replacement in the circulation. Consistent with the life span of the mature erythrocyte, regeneration of erythrocyte-associated aged acetylcholinesterase occurs at a rate of about 1% per day.

Chlorpyrifos. Chlorpyrifos is one of the safer organophosphorus insecticides. When sufficient doses are absorbed to elicit a toxic response, however, chlorpyrifos produces clinical effects in humans that are indistinguishable from other organophosphorus compounds. In contrast to many organophosphate insecticides, chlorpyrifos is an active inhibitor of plasma cholinesterase, but is characterized as only a moderate inhibitor of the erythrocyte-associated enzyme. As a result, normal exposure causes selective depression of cholinesterase activity in the plasma rather than in erythrocytes. Depression of erythrocyte-enzyme levels is often seen, however, when systemic effects are clinically apparent.[14]

When ingested by humans, oral doses of chlorpyrifos of 0.03 mg/kg/day had no detectable effect on plasma cholinesterase (also called pseudocholinesterase) levels. Enzyme depression (inhibition) of 70% has been reported to cause only mild symptoms in some cases. Human subjects who ingested 0.1 mg of chlorpyrifos/kg/day for 4 weeks were found to have statistically significant decreases in plasma cholinesterase. Pest-control operators who were exposed to 8-hour time-weighted average (TWA) exposures of 27.6 mg of chlorpyrifos/m^3 of air revealed significant inhibition of plasma acetylcholinesterase when compared with age- and sex-matched controls; however, they had no clinical signs or symptoms of exposure.[14]

Information related to chronic neurological sequelae of chlorpyrifos exposure in humans is limited. An adult male ingested 300 mg/kg of chlorpyrifos. He exhibited varying degrees of severity of cholinergic signs for more than 2 weeks. Although his electrophysiological studies of peripheral nervous function were reportedly normal 1 month after ingestion, his neurotoxic esterase (lymphocytic neuropathy target esterase, NTE) was approximately 60% inhibited. About 2 weeks later, the patient complained of paresthesia and lower-extremity weakness. A clinical examination and laboratory evaluation demonstrated classical findings of delayed axonal peripheral neuropathy.[14]

Delayed neurotoxicity has been reported following chlorpyrifos administration in the standard hen assay; however, the effects were reported to be reversible.[19] Delayed neurotoxicity was not seen following administration in the mouse model.[14] However, it is the hen, not the mouse, that is considered to be the standard assay of neurotoxic effect.[1]

The delayed CNS neuropathy following acute exposures to the organophosphate insecticides may be slowly reversible or remain irreversible, associated with axonal degeneration. A delayed onset, mixed sensory-motor peripheral neuropathy has been reported, with onset between 6 and 21 days after malathion exposure. After malathion exposures, recovery from the delayed neuropathy may be slow or incomplete. After diazanon exposures, sensory-motor peripheral neuropathy has occurred; however, the onset of the neurological abnormalities may be delayed for several weeks. Recovery may be slow or incomplete.[14]

Organophosphorus ester–induced delayed neurotoxicity (OPIDN) is the classical clinical syndrome associated with organophosphate insecticides. The early clinical description of OPIDN was associated with workplace exposures to tri-*ortho* cresol phosphate. The clinical findings of OPIDN include a rapidly progressive paralysis of the lower and upper extremities with limited recovery. The classical pathological lesion occurs within the CNS and peripheral nervous system (PNS) and is characterized as axonal degeneration and demyelinization of the long motor and sensory neurons. (The adult female hen is the preferred and accepted scientific standard model for the study of neurotoxic effect.) Neurotoxic esterase has been reported to be the putative target of OPIDN. The enzyme 2′,3′-cyclic nucleotide 3′-phosphohydrolase (CNPase) has recently been reported as a sensitive indicator of myelin loss, and may be a sensitive indicator for OPIDN.[22]

Chlorpyrifos exposure did not result in teratogenic or fertility effects in the rat. Chlorpyrifos demonstrated no carcinogenic potential following chronic administration in studies using rats and mice. No changes in microbial mutation or sister chromatid exchanges have been reported following chlorpyrifos exposures. Semen quality changes have been reported in bulls exposed to chlorpyrifos.[14]

Parathion. In a human dose-response study, adults who consumed more than 6 mg of parathion per day for 30 days had some decrement in cholinesterase level. Several individuals who consumed more than 7.5 mg for 16 days had erythrocyte cholinesterase levels inhibited to 50% and 52% of pretest levels. No adverse signs or symptoms were noted in any of the study subjects. In a separate study, a total daily dosage of 0.078 mg/kg of parathion resulted in depression of both erythrocyte cholinesterase (16% decrease) and plasma cholinesterase (33% decrease) levels compared to baseline values. The no-effect daily dose of parathion administered to adults was between 0.058 and 0.078 mg/kg when administered over time durations between 25 and 70 days. The acute dose that could be lethal in an adult has been estimated to be 120 mg.[14]

Parathion has been shown to be fetotoxic, but not teratogenic, in laboratory studies. However, methyl parathion has been associated with human birth defects. Although parathion has demonstrated effects of possible genotoxicity in rodents (induced DNA alterations) and in vitro studies (Ames and sister chromatid exchange assays), it is not considered to be carcinogenic.[14]

Diazanon. In contrast to parathion, the estimated adult oral fatal dose for diazinon is approximately 25 g. In addition to cholinesterase inhibition, increased prothrombin time has been reported with both malathion and diazinon exposure.[14]

Malathion. Manifestations related to the degree of acute toxic response seen after malathion exposure depend on the total dose absorbed, manner of exposure, and duration of the exposure profile. The toxicity of malathion is probably due to its metabolic oxidation to malaoxon, which has been estimated to be approximately 1,000-fold more potent than malathion itself as a cholinesterase inhibitor. The cholinergic toxicity is regarded as the principle hazard associated with exposure. Malathion and its metabolites appear to affect both erythrocyte cholinesterase and serum butyrylcholine esterase enzymes. Malathion has relatively low acute toxicity: an oral dose of 24 mg/day was required to depress cholinesterase activities in adult volunteers. The estimated fatal oral dose exceeds 70 mg/kg.[14]

Malathion has been reported to cause mild skin and upper-respiratory irritation in humans, and repetitive exposures have been reported to cause allergic cutaneous sensitization. A case of transient renal dysfunction secondary to a malathion-induced, immune-complex nephropathy has been reported.[14]

Malathion exposure has been studied in human lymphoid cell culture. An increase in sister chromatid exchange was noted with increasing doses of cellular exposure. Metabolic activation with liver homogenate had no demonstrable effect on the exchange. At the highest dosage, cytotoxicity was demonstrated by the loss of approximately 50% of the cultured cells. A study of malathion-intoxicated individuals identified an increased frequency of chromatid breaks and unstable structural chromosomal aberrations. However, a causal association could not be demonstrated.[14]

Signs and Symptoms of Intoxication. Clinical signs and symptoms of organophosphate insecticide poisoning depend on the type and exposure dosage of the chemical pesticide involved and are usually reported within several hours of exposure (Table 14-8). The asymptomatic individual may have cholinesterase depression. Clinical signs of exposure vary from limited local effects to severe systemic effects such as coma.

TABLE 14-8

SIGNS AND SYMPTOMS OF ORGANOPHOSPHATE POISONING

Organ System	Effects
	Muscarinic (parasympathetic)
Visual	Dimness of vision, blurring of vision, unilateral or bilateral miosis
Respiratory	Rhinorrhea, breathing difficulty, cough, tightness of chest, bronchoconstriction, increased bronchial secretions, wheezing
Cardiovascular	Bradycardia, systemic hypotension, atrial or ventricular arrhythmia
Gastrointestinal	Salivation, nausea, vomiting, diarrhea, involuntary defecation
Cutaneous	Sweating
Genitourinary	Frequent, involuntary urination
	Nicotinic (sympathetic and neuromuscular)
PNS (ganglia)	Peripheral vasoconstriction, tachycardia, hypertension, hyperglycemia
Musculoskeletal (striated)	Localized or generalized fasciculation, respiratory insufficiency or paralysis, weakness, cramps, twitching
	CNS (mixed muscarinic and nicotinic)
CNS	Anxiety, giddiness, restlessness, headache, emotional lability, excessive dreaming, nightmares, confusion, tremor, ataxia, coma, cardiorespiratory depression, cyanosis, hypotension, convulsions, apnea

Signs and symptoms of acute cholinesterase inhibition and subsequent cholinergic intoxication are highly dose dependent. Direct skin contact can cause local fasciculation or sweating without other effect. Similarly, local ocular exposure to an organophosphate insecticide aerosol may cause unilateral or bilateral miosis without systemic manifestations.[14]

The systemic effects of this group of pesticides are the (*a*) muscarinic, (*b*) nicotinic, and (*c*) the combined, more severe CNS responses.[15]

Muscarinic Response. The classical postjunctional muscarinic response to acetylcholine stimulation is associated with activation of specific receptor sites of postganglionic parasympathetic effector cells. Postganglionic muscarinic receptors are found primarily in smooth muscle, exocrine glands, and the heart. Muscarinic signs and symptoms are associated with cardiac, ocular, pulmonary, cutaneous, genitourinary, and gastrointestinal manifestations. Severe bradycardia, miosis, wheezing associated with bronchoconstriction and bronchial secretions, sweating, involuntary urination, nausea, vomiting, diarrhea, and involuntary defecation are common muscarinic responses. Exposed workers may present with giddiness, complaints of blurred vision, headache, nausea, abdominal cramps, breathing discomfort, or with various degrees of more severe distress. Military medicine uses the acronym SLUDGE to aid rapid field recognition of the signs and symptoms of the muscarinic response: *s*alivation, *l*acrimation, *u*rination, *d*efecation, gastrointestinal complaints, and *e*mesis.

Atropine, in sufficient dosage, is an effective antidote for muscarinic signs associated with the cardiac, respiratory, and CNS responses. Atropinization causes the gastrointestinal and genitourinary effects to improve; however, atropinization is only partially efficacious because it neither results in enzyme regeneration nor affects nicotinic receptors.

Nicotinic Response. Nicotinic responses to acetylcholine occur at myoneural junctions of striated muscle, preganglionic autonomic synapses with ganglia, and within the CNS. Although several types of site-specific nicotinic receptors have been identified, the acetylcholine effect on nicotinic sites is independent of those differences. Muscular responses to stimulation of nicotinic receptors range from easy fatigue, mild weakness, twitching, or localized fasciculation, to severe, generalized fasciculations that result in respiratory embarrassment and cyanosis.

The effects of stimulation of the nicotinic receptors of sympathetic ganglia can result in pallor. At higher levels of sympathetic nicotinic stimulation, hypertension and hyperglycemia may occur. In addition, tachycardia that results from ganglionic nicotinic receptor stimulation, which overrides the bradycardic effects of muscarinic stimulation, may be observed. The nicotinic receptors of the CNS appear to be important in nicotine dependence; headaches, paresthesia, and tiredness have been reported with nicotine administration.[14] Other nicotinic actions on the CNS cause tremor, convulsions, initial respiratory stimulation followed by respiratory depression, and vomiting (the latter action being caused by direct action on the area postrema of the brain stem).[7]

Central Nervous System Response. Signs and symptoms of CNS poisoning include anxiety, apathy, toxic psychosis, restlessness, fatigue, headache, nightmares, tremors, seizures, and depression of cardiac and respiratory centers, which can progress to coma.[16,18] Atropinization and oxime therapy are efficacious for management of the CNS toxic effects. Anticonvulsants are indicated to provide therapeutic management of seizure control.

Chronic health effects from both high-dose, short-term and low-dose, chronic exposures to organophosphate compounds have been reported in humans.[16,23] Neuromuscular signs and symptoms associated with this neuropathy include paresthesias, easy fatigability, cramps, and may progress to gait abnormalities. Pathologically, the delayed peripheral neuropathy demonstrates peripheral demyelinization. The delayed peripheral effects may be related to organophosphate binding of a "neurotoxic esterase" enzyme.[16] Neurobehavioral signs and symptoms include many different complaints such as anxiety, depression, insomnia, and irritability. Most residual neurological symptoms appear to resolve within a year following acute intoxications.[18]

Parathion Intoxication. The onset of signs and symptoms induced by parathion has been uniformly accepted as the standard general description of organophosphate poisoning. It is important to recognize that the signs or symptoms can recur for days, despite therapeutically efficacious medical management. Careful patient monitoring and administration of indicated therapeutic management must be assured for days after parathion or other organophosphate poisoning.

Clinical signs and symptoms seen in children are most often seen by alterations of central neurological status. CNS depression, stupor, flaccidity, and coma are the most common signs in children. Dyspnea is commonly seen in children.

Ingestion usually results in nausea, followed by increased salivation, abdominal cramps, vomiting, and diarrhea. Hypothermia may occur as an early sign, but is not a usual finding. Alterations in mental status can manifest as confusion, anxiety, or giddiness.

Inhalation is usually followed by rhinorrhea, then

chest tightness as exposure doses increase. Although miosis may occur with eye pain, ciliary muscle spasm, and blurring of vision in topical or inhalation exposure, miosis is not a dependable sign for ingestion or cutaneous exposures. In fact, mydriasis is not an uncommon finding, possibly as a result of a sympathetic, reflex, adrenal response.

Although alveolitis, followed by progressive pulmonary fibrosis, has been reported in a parathion-intoxicated patient,[14] alveolitis and fibrosis are much more common following paraquat exposures.[24] Chemical alveolitis is probably associated with other chemicals in the pesticide formulation, rather than as a result of exposure to the cholinesterase-inhibiting insecticide itself.

CNS effects such as decreased vigilance, altered expressive language, diminished cognitive function, impaired memory, depression, anxiety, and irritability have been reported.[14] Other signs and symptoms may include visual hallucinations, auditory hallucinations, and psychosis.

Cardiac signs of organophosphate insecticide poisoning characteristically include bradycardia and hypotension, although reflex tachycardia, despite significant poisoning, has been reported. Heart rate, alone, is an unreliable sign for both the degree of exposure and efficacy of therapy. It has become increasingly apparent that acetylcholine in the coronary circulation can cause intense vasospasm resulting in atrial arrhythmias, hypotension, chest pain, and heart block.[14,25]

Acute respiratory failure is the major cause of death in organophosphate poisoned patients. Dyspnea, bronchorrhea, and tachypnea represent significant clinical signs of respiratory difficulty. Bronchospasm occurs as a typical pharmacological muscarinic effect. Respiratory responsiveness, such as diminished respiratory secretions and decreased ventilatory resistance are reliable indicators of inhalation toxicity and the efficacy of medical management. Delayed respiratory crisis may occur for 2 to 3 weeks following acute poisonings.[14]

Chlorpyrifos Intoxication. Muscular weakness, fatigability, and fasciculations are commonly reported in association with chlorpyrifos poisoning. They may be delayed in onset and paralysis may occur.[14]

Miosis, lacrimation, and blurred vision are common signs of chlorpyrifos poisoning. Mydriasis is unlikely, but has been reported in association with severe poisonings.[14] Excessive salivation is a common post-exposure sign.

Sweating is a common sign of chlorpyrifos exposure, but does not occur as a universal finding. Dermal irritation and sensitization have been reported but are uncommon. Other uncommon effects of exposure include a reported alteration in prothrombin time and the occurrence of hyperglycemia in severe poisoning.[14]

In addition to nausea, vomiting, and diarrhea, abdominal pain and fecal incontinence may occur with cholinesterase inhibition. Urinary frequency and, in severe cases, urinary incontinence have been reported.

Diazanon Intoxication. Nausea is often the first symptom that follows diazinon exposure. Other signs and symptoms range in severity from mild gastrointestinal or respiratory effects to cholinergic crisis. Vomiting, diarrhea, abdominal cramps, and salivation are commonly reported, especially with cutaneous or gastrointestinal absorption. Inhalation exposures are more commonly associated with rhinorrhea and chest tightness. Ocular effects from exposure include tearing, miosis, ciliary muscle spasm, and eye pain. Paradoxical mydriasis has been reported with diazinon poisoning and probably is the result of a sympathetic reflex response.[14] Weakness, local fasciculations, drowsiness, dizziness, headache, and behavioral changes represent mild to moderate neuromuscular responses to exposure. Loss of muscular coordination, generalized twitching, and convulsions represent more severe neurological consequences of poisoning with diazinon.

Malathion Intoxication. It is important to note that symptoms occur after high-dose exposures to malathion, which are possible only in unusual circumstances such as an incorrect application. Inhalation of malathion results in ocular and respiratory effects as first signs of exposure. Ingestion results in a loss of appetite, nausea, vomiting, abdominal cramps, and diarrhea, which may appear within several hours. After skin absorption, local signs of sweating and twitching may occur within minutes or may be delayed for several hours. Severe signs may occur following exposure by all routes.

Clinical manifestations of malathion exposure in children may differ from the predominant signs and symptoms associated with adults exposure. CNS signs, such as CNS depression, stupor, loss of muscular tone, and coma are the most commonly reported signs of exposure in children; respiratory difficulty has also been reported.[14]

Other signs and symptoms have also been reported: fever may persist for several days; alterations in prothrombin time may occur; and hyperglycemia, glycosuria, and metabolic acidosis without ketosis have been reported associated with severe poisoning.[14]

Medical Treatment. If a strong likelihood of acute organophosphate poisoning exists, the patient should be treated immediately without waiting for laboratory results. The usual ABCs of emergency care apply:

healthcare providers must ensure that the patient has a patent airway, is breathing, and has adequate circulatory function without apparent hemorrhage. Oxygen should be provided, if the patient's condition indicates. Individuals who attend the victim should avoid direct contact with heavily contaminated clothing, vomitus, skin, and hair by wearing PPE such as rubber gloves (at a minimum).

Plasma and erythrocyte cholinesterase enzyme activities should be measured, but the degree of correlation between the levels of cholinesterase inhibition and clinical effects is imprecise. In some cases, a depression of only 50% of the enzyme activity may be associated with signs of cholinergic crisis. The correlation between cholinesterase levels and clinical effects is unreliable and should not be used for medical management.

In addition to the determinations of erythrocyte and plasma cholinesterase levels, some clinical laboratories may perform urinalyses for *p*-nitrophenol, the parathion leaving group. As with cholinesterase levels, the determination of parathion-metabolite levels is often not a readily available emergency test procedure. As a result, medical management of the parathion-poisoned patient should be directed by the patient's clinical responsiveness, with cholinesterase or pesticide-metabolite levels in urine serving only as subsequent measures of confirmation of exposure.

Clinical decisions concerning medical management must be based on the type and degree of signs exhibited by the acutely poisoned patient. Medical therapeutic intervention should not depend on the degree of depression of measured cholinesterase activity for the following reasons:

- The correlation between enzyme levels and clinical effects is poor.
- The test is not universally available.
- Laboratory report times are too time consuming.
- Baseline information is absent in many acute poisoning cases.

As a result, the measured enzyme inhibitions may confirm the diagnosis of cholinesterase inhibitor intoxication, but will not contribute substantially to acute patient management.

Asymptomatic patients with documented depression of cholinesterase levels should be carefully monitored, but they require no atropine unless signs and symptoms of poisoning evolve. Follow-up evaluations of cholinesterase levels for adequately treated or clinically stable asymptomatic patients whose levels have been acutely depressed may be done at weekly intervals unless the patient's condition dictates more frequent analyses.

Because early-onset respiratory depression and generalized convulsions are expected after serious exposures such as intentional ingestion, induction of emesis is contraindicated. If necessary, gastric aspiration or lavage can be performed. Protection of the airway is critical during nasogastric procedures and may be accomplished by cuffed endotracheal intubation. If lavage is performed, the return volume should approximate the amount of fluid administered.

In the management of ingestion, an activated charcoal slurry should be administered as quickly as possible. A total of 30 to 100 g of charcoal should be administered to adults, and 15 to 30 g to children.[14] Administration of a cathartic, either with the charcoal or separately, is recommended.

Atropine Therapy. In addition to respiratory distress, patients with severe signs of intoxication have profuse nasal, oral, and airway secretions. It is imperative to maintain control of a patent airway, using suction if necessary, until the degree of atropinization is adequate to control secretions and relieve bronchospasm. If hypoxia is suspected, administration of oxygen (if available) before atropine is injected is recommended. Atropine administration has been associated with the precipitation of ventricular fibrillation in hypoxic patients.

Atropine administration for symptomatic patients is imperative. Timely administration is crucial, regardless of the route of pesticide exposure. Atropine sulfate should be given intravenously, if possible, but is effective when injected intramuscularly, especially if administered via the current military Mark I atropine autoinjector. Signs of adequate atropine administration include drying of the airway secretions and improvement of respiratory efforts. Atropine administration should be continued as necessary until signs of organophosphate poisoning no longer recur, sometimes days after the acute poisoning event. The fever, disorientation, and delirium associated with atropine use reflect signs of excessive atropine administration; they indicate at least temporary discontinuation of atropine. Atropine administration does not affect acetylcholinesterase regeneration.

The typical adult dose of atropine is 2 to 4 mg, which can be administered every 10 to 15 minutes as needed. The dosage of atropine useful for managing a poisoned child is 0.05 mg/kg every 10 to 15 minutes as needed.[14] Treatment of cholinesterase inhibition is required for hours or days, depending on the individual patient and the circumstances of exposure. The treatment of patients poisoned with organophosphate insecticides may require a total of several grams of

atropine over the course of acute recovery from poisoning.

Oxime Therapy. There is no effective, medically approved antidote for the nicotinic effects of cholinesterase-inhibiting substances. If therapeutic intervention precedes enzyme aging, oxime therapy, such as administration of 2-pyridine aldoxime methyl chloride (2-PAM Cl), acts as a reversal compound (Figure 14-5). Improved levels of active enzyme within the acetylcholine-receptor regions results in improved disposition of free acetylcholine, with resultant improvement in the patient's signs and symptoms. Oxime therapy is recommended for patients with signs of severe pesticide intoxication such as severe twitching or fasciculation, significant weakness, or respiratory embarrassment.[16,23] 2-PAM Cl does not relieve bronchospasm or bronchorrhea, which are treated with concurrent administration of atropine.[23]

Pralidoxime Therapy. Pralidoxime therapy is often helpful in acute organophosphate poisonings. *It is imperative that pralidoxime be administered to severely poisoned patients* who have neuromuscular effects such as fasciculations, weakness, and respiratory paralysis. The typical dose for individuals who are 12 years old or older is 1 g of pralidoxime delivered intravenously over a minimum of 2 minutes. Children younger than 12 years of age should be given an intravenous dose of 20 to 30 mg/kg slowly, over at least 2 minutes.[14]

Pralidoxime is often helpful in acute organophosphate poisonings and is indicated in severe cases of organophosphate insecticide poisoning that are accompanied by profound weakness and respiratory depression. The recommended adult dose of pralidoxime is 1.0 g, administered intravenously, at a rate of 0.5 g/minute or infused in 250 mL of normal saline over 30 minutes. For initial management, the dose can be repeated up to three times. It may be administered in intervals of 6 to 12 hours if muscle weakness is not relieved or if the patient remains comatose. A continuous pralidoxime infusion (500 mg/h) may be administered; however, this alternative is considered to be controversial.[14]

For a poisoned child, pralidoxime may be administered intravenously at a dosage of 25 to 50 mg/kg over 30 minutes. Further administration may be necessary if muscle weakness and associated respiratory depression remain uncorrected.[14]

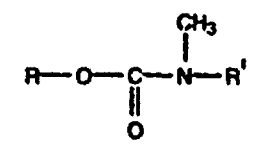
Anticonvulsant Therapy. Medical personnel should be prepared to promptly administer benzodiazapines (diazepam) as anticonvulsants for seizure activity associated with poisoning. The occurrence of clinically apparent convulsions has been recognized as a sign of neurological electrophysiological seizure activity. If convulsions occur, timely administration of diazepam as a direct intravenous bolus is imperative to preclude further neurological damage from hypoxia. The typical adult dose is 5 to 10 mg initially, which may be repeated every 15 minutes, as necessary, up to 30 mg. For the convulsing child, a dosage of 0.25 to 0.4 mg/kg up to 10 mg total dose is recommended. Intramuscular injections are slowly absorbed and should be avoided, if possible. If seizures are uncontrollable or recur, phenytoin or phenobarbital should be administered.[14] *Physostigmine, succinylcholine, or other cholinergic agents are contraindicated and should not be administered.*

Complications. Pulmonary edema may result from inhalation of pesticide formulations or occur as a complication of medical management. Oxygenation and ventilation must be maintained and arterial blood gases must be carefully monitored. If Po_2 remains low in spite of oxygen administration, it may be necessary to add positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). Careful fluid management is essential and a central line or Swan-Ganz catheter should be placed to monitor fluid status.

If significant inhalation exposure or coincident aspiration occurs, a baseline X ray should be obtained. This is especially important if the pesticide formulation was concentrated, contained irritant or hydrocarbon compounds, or was of unknown composition. Determining arterial blood gases and testing pulmonary function may be necessary during complicated medical management of some patients.

Hypotension may occur and should be treated by administering intravenous fluids and placing the patient in the Trendelenburg position, if necessary. Patients with blood pressure that is unresponsive to fluid administration may require careful pressure titration using dopamine (2–5 μg/kg/min) or norepinephrine (0.1–0.2 μg/kg/min).[14]

Carbamates



Carbamate insecticides are cholinesterase inhibitors (see Table 14-6). These compounds are an associated group of chemical esters with the general structural composition shown above, where *R* represents an oxime, alcohol, or phenol, and is the leaving group associated with inhibition of the cholinesterase molecule[16]; *R'* represents an N-methyl or hydrogen atom.[2,20,26] The most commonly used carbamates in

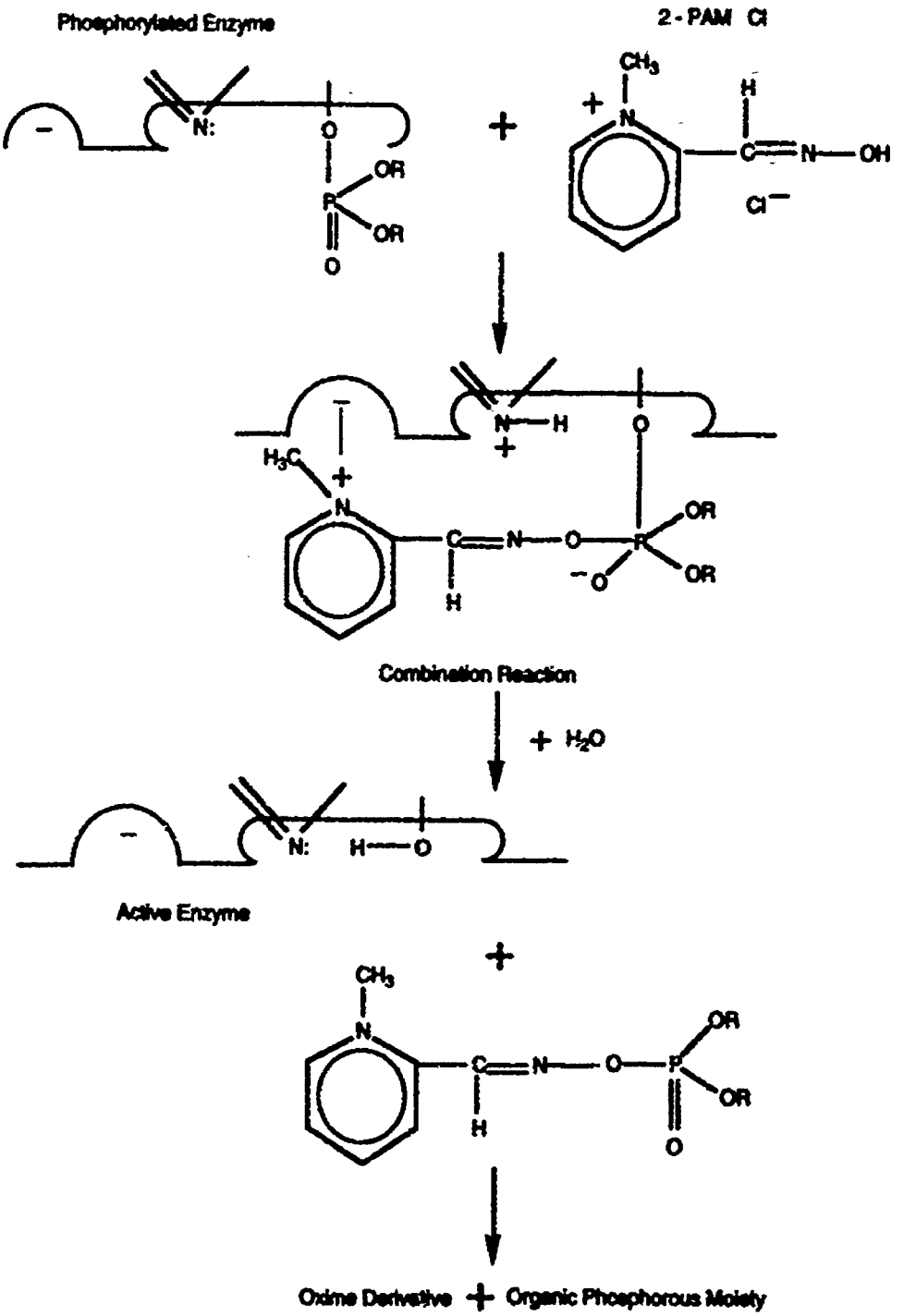


Fig. 14-5. If organophosphate-inhibited acetylcholinesterase is treated *before it ages* with: pralidoxime chloride (2-PAM Cl, an oxime), the enzyme can be reactivated.

the military are carbaryl and propoxur, representatives of the N-methylcarbamate group (Table 14-9).[20]

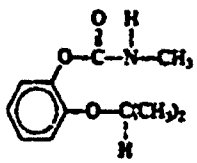
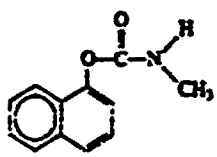
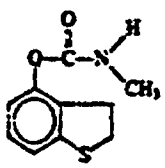
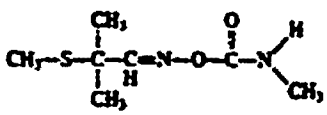
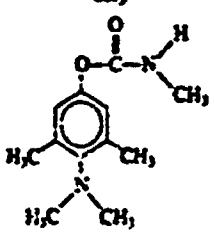
Route of Exposure. Carbamates are absorbed via all routes of exposure, although dermal absorption is slight.[16] The degree of acute toxic effect depends on both the rapidity of absorption and the cumulative dose. Many carbamate insecticides have low dermal toxicity.[16]

Mechanism of Action. Inhibition of the acetylcholinesterase molecule by the N-methylcarbamate pesticide group differs from the phosphorylation reactions in organophosphate insecticides. In contrast to the rather stable organophosphate phosphorylation at the esteratic site, carbamylation of the enzyme appears to involve attachment of the carbamate at both the anionic and esteratic sites, which is similar to the action of acetylcholine. Because of the ready dissociation of the enzyme-carbamyl complex and the subsequent regeneration of the active enzyme molecule, carbamate compounds are rapidly reversible inhibitors of acetylcholinesterase.

Like other cholinesterase-inhibiting substances, carbamates are not directly measured in blood. Indirect measurement of exposure to carbamates is determined by measuring blood cholinesterase activity. Because the interaction between the carbamate and cholines-

TABLE 14-9

EXAMPLES OF RANGE OF ACUTE TOXICITIES OF SOME CARBAMATE INSECTICIDES

		LD50 IN MALE RATS[a] (mg/kg)	
		Oral	*Dermal*
Baygon (Propoxur)		83	>2400
Carbaryl		850	>4000
Mobam		150	>2800
Temik (Aldicarb)		0.8	3.0
Zectran		37	1500–2500

[a]Values obtained in standardized tests in the same laboratory

Reprinted with permission from Murphy SD. Toxic effects of pesticides. In: Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons.* 3rd ed. New York: McGraw-Hill, 1986: 540.

terase is spontaneously reversible, the residual cholinesterase activity usually fails to correlate with the clinical significance of the exposure.[14]

Rapid reversibility of the carbamylated enzyme complex decreases the duration of the clinical signs of poisoning, allows a wider dosage range between the onset of symptoms and death, and decreases the slight chance of documenting the level of depression of enzyme levels, unless a blood sample is analyzed almost immediately after exposure.[14,18,20]

Metabolism of carbaryl involves N-demethylation, hydroxylation, hydrolysis, and conjugation. Hydrolysis results in the urinary excretion of (*a*) 1-naphthol, which accounts for more than 20% of an administered dose, and (*b*) *p*-hydroxycarbamyl, which accounts for approximately 4% of the administered dose. Urinary concentration of 1-naphthol has been used as a biological exposure index of carbaryl exposure. Unexposed subjects have been reported to have urinary concentrations of 1-naphthol below 0.23 mg/L. Asymptomatic workers who were exposed to ambient air concentrations of carbaryl as high as 31 mg/m^3 were found to have urinary 1-naphthol concentrations of more than 42 mg/L. Although no standards have been established for carbaryl metabolites in urine, urinary 1-naphthol concentrations in excess of 4 mg/L of urine may represent significant exposure to carbaryl.[14]

Dose-dependent inhibitions of platelet aggregation and arachidonic acid metabolism in platelets have been demonstrated to be inhibited by carbamate insecticides. In these evaluations, the most potent carbamate compound found was carbaryl, which was shown to inhibit platelet aggregation and diminish products of the enzyme cyclooxygenase at concentrations as low as 10 µM. However, radiolabeled carbaryl was shown to bind covalently with numerous platelet proteins, in contrast to acetylsalicylic acid, which acetylates only a single platelet protein. Acetylsalicylic acid is known to specifically inhibit cyclooxygenase enzyme activity, which is similar to the action of carbaryl.[14]

Despite its widespread use by the World Health Organization as an insecticide to control the mosquito vector of malaria, only a few mild cases of propoxur poisoning have been reported. A human volunteer ingested 1.5 mg/kg of propoxur. The erythrocyte cholinesterase fell to 27% of the baseline within 15 minutes. The subject experienced nausea, vomiting, blurred vision, sweating, and tachycardia. By 2 hours following ingestion, the subject had no residual signs or symptoms and the enzyme levels were within normal limits. Adult humans have ingested single 90-mg doses without any apparent symptoms.[14]

Although delayed neurotoxicity has been reported with carbaryl exposure in one 75-year-old man, epidemiological studies of human carbaryl exposures have not demonstrated delayed neuropathy. Male human volunteers who ingested carbaryl dosages of 0.06 and 0.12 mg/kg for a study period of 6 weeks had no demonstrable changes in their electroencephalogram patterns. Inadequate data from human studies and the uncertain relevance of existing data from animal studies limit the final conclusions that can be drawn concerning delayed neurotoxic or myotoxic effects in human populations.[14]

Signs and Symptoms of Exposure. Much of what is known concerning the signs and symptoms of human exposures to carbamates has been based on studies of a closely related chemical compound, physostigmine.[26] The clinical signs and symptoms of carbamate poisoning are identical to those associated with organophosphate poisoning (see Table 14-6). Carbamates are believed to have a wide safety margin because the signs of cholinesterase poisoning, which resolve soon after the exposure is discontinued, are rapidly reversible. The most common signs and symptoms include lacrimation, salivation, miosis, convulsion, and death.[4]

Exposure to carbamate insecticides may lead to clinical manifestations of cholinergic crisis similar to those found with the organophosphate insecticides. The classic signs and symptoms associated with cholinergic activity may include increased salivation, lacrimation, urinary incontinence, diarrhea, gastrointestinal cramping, and emesis. Clinical CNS signs and symptoms of carbamate poisoning are less intense and shorter in duration than those associated with comparable organophosphate pesticides.[14]

Ocular signs of exposure, including miosis, tearing, ciliary spasm, severe ocular or retroorbital pain, and diminished accommodation, may occur. Miosis may be either unilateral or bilateral and is often recognized by the patient as visual blurring, especially in a darkened room. Mydriasis may occur as a result of reflex adrenergic stimulation, although this is unusual.[14]

Respiratory responses to carbaryl intoxication include rhinorrhea, increased bronchial secretions, bronchospasm, wheezes, rhonchi, and rales. The patient may also experience chest tightness.

The major cutaneous sign associated with local dermal absorption is localized sweating. On careful observation of the skin, fasciculations may be observed in the underlying skeletal muscle.[14]

In addition to the localized fasciculations, other neuromuscular effects may include generalized loss of muscle tone, widespread muscular twitching, and overt convulsive activity, which can result from systemic stimulation. Other neurological responses include weakness, lassitude, incoordination, and slurred speech. Death is primarily the result of central respi-

ratory depression and paralysis, and is usually preceded by or associated with generalized convulsions, fecal or urinary incontinence or both, and coma.

In addition to the direct effects of the pesticide, bronchopulmonary sequelae may result from inhalation of the components of the pesticide formulation. For example, inhalation of the supposedly inert dust or the hydrocarbon vehicle may cause throat irritation, dyspnea, pneumonitis, or pulmonary edema.

If the individual who is exposed to the pesticide demonstrates cardiac effects, bradycardia is the most common sign; tachycardia can occur, however, possibly as a result of reflex response to bradycardia.

Disseminated intravascular coagulation and kidney damage have been reported, and delayed peripheral neuropathy, similar to that caused by some organophosphorus compounds, was reported in one case.[14]

With more severe exposures, mental confusion, loss of muscle coordination, tremors, or convulsions may occur. Death can result from respiratory arrest of CNS origin, paralysis of respiratory musculature, or intense bronchorrhea with bronchoconstriction.[14]

Medical Treatment. The administration of basic life-saving practices and decontamination of the skin with soap and water, if indicated by circumstances, are the essential elements of early first aid and medical care. Analysis of the plasma and erythrocyte cholinesterase levels are of no benefit in the medical management of these patients. Even in circumstances where depressed enzyme levels could be demonstrated, the delay in obtaining the results would severely compromise the usefulness of the enzyme levels as diagnostic and prognostic tools. Because the interaction between the carbamate pesticides and cholinesterase molecules is readily reversible, enzyme levels may be normal despite overt signs of poisoning. In addition, the tremendous variability between laboratory methodologies and reported cholinesterase activity units severely compromises meaningful interpretation of reported results in typical patients.

Induced emesis is not recommended following carbamate ingestion. After nasogastric suction has been performed, administration of activated charcoal and a cathartic is recommended. The adult dose of activated charcoal is usually between 30 and 100 g; for children, 15 to 30 g.[14]

Direct eye contact or splash should be treated by irrigation with copious amounts of water for at least 15 minutes. Medical evaluation is recommended in most circumstances, especially if signs of irritation such as pain, chemosis, lacrimation, or photophobia persist.

If significant inhalation exposure or coincident aspiration occur, a baseline chest X ray should be obtained. This is especially important if the pesticide formulation was concentrated, contained irritant or hydrocarbon compounds, or was of unknown composition.

Atropine administration for symptomatic patients is imperative. Timely administration is crucial, regardless of the route of pesticide exposure. Although best administered intravenously in life-threatening circumstances, atropine is also effective if administered intramuscularly, particularly if delivered by the atropine autoinjector. Atropine administration should be titrated to the patient's need based on resultant signs of atropinization. In cases of carbamate poisoning, the most reliable sign of atropine adequacy is the degree of drying of the pulmonary secretions.

The typical adult dose of 2 to 4 mg may be carefully administered every 10 to 15 minutes as needed. The dosage of atropine useful for the management of a poisoned child is 0.05 mg/kg every 10 to 15 minutes as needed.[14] The treatment of cholinesterase inhibition associated with carbamate poisoning may be required for hours or days, depending on the individual patient and the circumstances of exposure.

Clinically apparent convulsions have been accepted historically as a sign of neurological seizure activity. *If convulsions occur, timely administration of diazepam as a direct intravenous bolus is imperative.* The typical adult dose is 5 to 10 mg initially, which may be repeated every 15 minutes as needed up to 30 mg. For the convulsing child, the recommended dosage is 0.25 to 0.4 mg/kg, up to 10 mg total dose. Intramuscular injections are slowly absorbed and should be avoided, if possible. If seizures are uncontrollable or recur, phenytoin should be administered.[14] *Physostigmine administration is contraindicated, and its use should be avoided.*

The administration of pralidoxime is contraindicated in pure carbamate poisonings. The addition of oximes such as 2-PAM Cl markedly increase carbamate toxicity and may cause death.[14,18,27] Use of pralidoxime is controversial in carbamate poisonings that are complicated by other factors such as known organophosphate-combined poisoning or unknown circumstance of poisoning. Although atropine is effective against the muscarinic manifestations of carbamate poisoning, it is ineffective against the nicotinic manifestations.

However, some authorities recommend that pralidoxime should be given when life-threatening symptoms are present.[14] The recommended adult dose is 1.0 g administered intravenously at a rate of 0.5 g/min, or infused in 250 mL of normal saline over 30 minutes. The dose may be repeated up to three times for initial management. Praladoxime may be administered in intervals of 6 to 12 hours if muscle

weakness is not relieved. As an alternative, an infusion of 500 mg/hour may be administered.

For a poisoned child, pralidoxime may be administered intravenously at a dosage of 25 to 50 mg/kg over 30 minutes. Further administration may be necessary if muscle weakness and associated respiratory depression remain uncorrected.[14]

Organochlorine Insecticides

The four classes of organochlorine insecticides are (1) DDT and related derivatives of chlorinated diphenylethane; (2) cyclodienes, including dieldrin, heptachlor, and chlordane; (3) lindane (the gamma isomer of hexachlorocyclohexane); and (4) toxaphene (a mixture of chlorinated terpenes).[25] Substances in this group are known for their low biodegradability in the environment, accumulation in human and animal adipose tissues, and carcinogenicity in laboratory animals.

The acute toxicity and human hazard potential of organochlorine insecticides are highly variable and are influenced by the rate of exposure, route of exposure, and specific chemical compound (see Table 14-6). For example, dermal absorption is much greater of hexachlorocyclohexane (the technical grade includes the gamma isomer lindane) and the cyclodiene derivatives than for the ethane derivatives. The potential acute human toxicity hazard for these compounds is, from highest to lowest, endrin, aldrin, dieldrin, chlordane, toxaphene, chlordecone, heptachlor, DDT, and methoxychlor. Dicofol, methoxychlor, and hexachlorobenzene have limited CNS toxicity; however, in extreme overdoses, CNS depression may occur.[14]

Organochlorine insecticides are primary representatives of the broader group of chlorinated hydrocarbon pesticides. Other representatives of the chlorinated hydrocarbons have been used as fumigants, herbicides, fungicides, and nematocides.[5] The chemical structures and selected toxicological activities for representative organochlorine insecticides are found in Table 14-10.

The precise mechanism of action of many of the organochlorine insecticides remains unknown.[26] Since 1944, DDT has been known to affect the nervous system; however, the precise mechanism of action remains incompletely described.[26] DDT is thought to exert its complex toxicological action through its impact on the fluxes of sodium, potassium, and calcium across the nerve cell membrane, but measurements of ionic potentials differ among species. In addition, in rabbit brain, DDT has been shown to inhibit the action of Na^{+}–Mg^{++} adenosine triphosphatase (ATPase), an enzyme responsible for ion movement in the nervous system.[26] The net effect of these ionic and enzymatic interactions is a slowed closing of the sodium channel, with prolongation of the hyperexcitable state of the nerve cell following nerve-impulse transmission.[25,29]

In contrast to DDT, lindane and the cyclodienes (eg, dieldrin) appear to exert their toxic effects at the nerve synapse, where their action results in increased spontaneous and evoked release of neurotransmitter.[28] Lindane and dieldrin act on the ganglion, rather than the axon, where increased membrane permeability to calcium was demonstrated. In addition, they appeared to inhibit Ca^{++}–Mg^{++} ATPase, which influences the rate of calcium extrusion. Dieldrin, lindane, and heptachlor epoxide have all recently been shown to be potent, competitive, stereospecific inhibitors of the picrotoxin receptor. As a result of this inhibition, γ-aminobutyric acid (GABA) and GABA-related transmission is affected. GABA-induced chloride permeability is inhibited, with the result that the nerve cell remains hyperexcitable after stimulation. DDT and mirex were not found to bind to the picrotoxin receptor.[29,30]

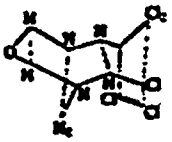
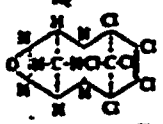
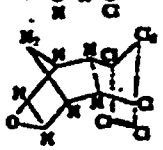
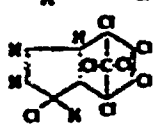
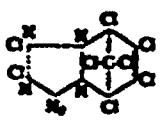
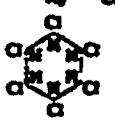
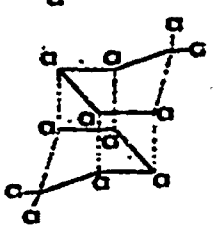
Animal experimentation and human epidemiological studies have demonstrated that chronic exposure to chlorinated hydrocarbons results in bioaccumulation in adipose tissues. Experimental observations within these test groups demonstrate significant differences in the metabolism and storage in the adipose tissue depots.

Numerous nonspecific signs and symptoms have been reported after acute organochlorine exposures. Sensory disturbances such as hyperesthesia or paresthesia may be either early or low-dose, acute effects of DDT poisoning. Headache, dizziness, vomiting, incoordination, and tremor may progress to myoclonic jerking, convulsions, or both. Individuals who are exposed to cyclodienes may present with convulsions as the first sign of poisoning as long as 48 hours after acute exposure. Increased neuronal irritability results in excitation, convulsions, and possibly coma at high doses. Cardiac dysrhythmia may also occur.[14,23]

Several of the organochlorine insecticides (eg, aldrin and heptachlor) stimulate hepatic microsomal enzymes and undergo xenobiotic transformation through epoxide derivative intermediates (Figure 14-6). The development and storage of these intermediates, and, in animal models, evidence for their carcinogenic potential, have reinforced the concern that many of these compounds are suspect human carcinogens.[4] Histopathological changes have been noted in the livers of chlordecone workers; however, elevation of their hepatic enzymes indicative of damage was not observed. Reductions in sperm counts have been noted in workers who have been exposed to chlordecone and dichlorobromopropane. Blindness has been reported in sheep that have been exposed to

TABLE 14-10

TOXICOLOGY OF SELECTED ORGANOCHLORINE INSECTICIDES

COMPOUND	STRUCTURE	LD50 IN MALE RATS ORAL	DERMAL	NO EFFECT LEVEL[*†]	ADI[‡]
Aldrin		39	98	Rat—0.025 Dog—0.025	0.0001
Dieldrin		46	90	Rat—0.025 Dog—0.025	0.0001
					
Endrin		18	31	Rat—0.05 Dog—0.025	0.0002
					
Heptachlor		100	~195	Rat—0.25 Dog—0.05	0.0005
Chlordane		335	840	Rat—1.0 Dog—0.05	0.001
Lindane		88	1000	Rat—1.25	0.0125
Mirex		740	>2000	—	—

[*] Values obtained in standardized tests in the same laboratory

[†] Maximum rate of intake (usually 3-mo, 2-yr feeding studies) that was tested and did not produce significant toxicologic effects (as listed in the monographs issued jointly by the Food and Agriculture Organization of the United Nations and the World Health Organization, as developed by joint meetings of expert panels on pesticide residues held annually, 1965–1972)

[‡] Acceptable daily intake (ADI) = the daily intake of a chemical that, during a lifetime, appears to provide the practical certainty that injury will not result (in man) during a lifetime of exposure. Figures taken from World Health Organization (1973).

Reprinted with permission from Murphy SD. Toxic effects of pesticides. In: Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd ed. New York: McGraw-Hill; 1986: 540.

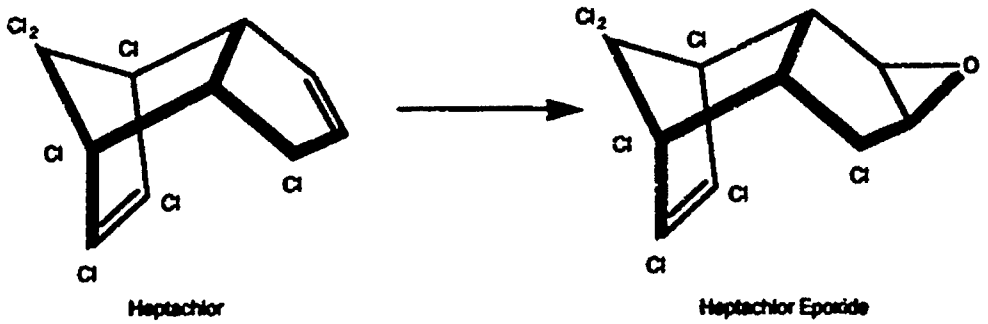


Fig. 14-6. When the parent pesticide compound *heptachlor* is metabolized by the hepatic P-450 enzyme system, the resultant metabolite *heptachlor epoxide*, a suspect carcinogen, is formed. The metabolism of chlordane to chlordane epoxide, also a suspect carcinogen, is similar.

endosulfan, and mirex has caused cataracts in the rodent model.[16,20,23] Chronic exposure to chlordecone has been associated with weight loss, weakness, tremor, slurred speech, cognitive changes, and abnormal liver-enzyme profiles.

Chlordane

Chlordane has been widely used as an environmentally persistent, highly effective termiticide. As a result of its efficacy and popularity, large numbers of pesticide operators and members of the general population have been exposed. The xenobiotic metabolism of chlordane has been evaluated in pest-control operators: chlordane and its metabolites (oxychlordane, heptachlor epoxide, and *trans*-nonachlor) were identified in the blood of some of the pest-control operators but not in the control subjects. The concentration of chlordane and its metabolites correlated well with the number of spraying days in the previous 3 months. As a result, the concentration of chlordane and its metabolites has been suggested as a biological monitor for chlordane-exposed workers. Although the chemical compounds can be identified at rather low concentrations in blood, no correlation between blood concentrations and health effects has been published. Consequently, the utility of the biological monitoring capability for chlordane and its metabolites remains speculative and is not routinely recommended.[14]

Purified chlordane has been reported to induce cytotoxic effects in human liver cell cultures. In that system, chlordane exposure resulted in growth inhibition and alterations of cellular morphology.

The fatal dose of chlordane for an adult has been estimated to be between 6 and 60 g. Onset of clinical signs and symptoms would be expected between 45 minutes and several hours.[14]

Route of Exposure. Chlordane and its formulations are lipophilic and may be readily absorbed following skin contact, inhalation, and ingestion. As a result of its widespread use, persistence, ease of cutaneous absorption, lipophilic nature, and preferred in vivo lipid-affiliated storage, the EPA identified almost ubiquitous presence of chlordane in samples of body fat and breast milk from human populations.[4]

Signs and Symptoms of Exposure. Because chlordane and related pesticides disrupt nervous system functions, patients may present with a spectrum of CNS-stimulation findings. The earliest signs of poisoning are related to increased sensitivity to stimuli. Hyperexcitability of the CNS usually manifests as nervousness, agitation, irritability, amnesia, and generalized hyperactive reflexes. Cycles of excitement followed by depression may occur repeatedly. More-severe signs include muscle twitching, tremor, incoordination, and ataxia. The most severe neurological signs include clonic convulsions, with or without coma.[14]

Respiratory depression may occur as an unusual result of chlordane intoxication. Aspiration of chlordane formulations that contain petroleum distillates may result in chemical pneumonitis.[14]

Gastrointestinal effects such as nausea, vomiting, and diarrhea have been reported following ingestion of chlordane. Extensive cutaneous contact may result in dermal irritation.[14]

The EPA considers chlordane and related compounds to be potential human carcinogens.[4] However, epidemiological studies of workers in the chlordane-producing industry have failed to identify an increased occurrence of cancer. Exposure to chlordane, heptachlor, or both has been related to aplastic anemia and acute leukemia in several cases, but the relationship between these chemicals and the adverse

health effects was inadequate to reflect a causal association. In another retrospective study, an uncertain cause-and-effect relationship was suggested between the occurrence of neuroblastoma in several children and possible chlordane or heptachlor exposure.[14]

In a prospective mortality study directed toward employees of a chlordane production plant, workers with the highest risk of exposure appeared to demonstrate an inverse relationship between exposure and cancer risk. The study identified an unexplained excess of deaths due to cerebrovascular disease.[14]

Medical Treatment. As with any pesticide-poisoned patient, basic life support and early decontamination procedures are essential elements of medical management. After the contaminated clothing has been removed, the patient should be decontaminated with soap and water, then topical alcohol, and then soap and water again.[14]

In alert patients, emesis should be induced with syrup of ipecac in cases of recent, substantial ingestion. In addition, administration of activated charcoal and a cathartic is recommended.[14]

As a consequence of the increased myocardial irritability associated with organochlorine poisoning, administration of epinephrine or other adrenergic amines may precipitate refractory ventricular arrhythmias.[14]

Seizure activity should immediately be treated with diazepam administered by intravenous bolus. The typical adult dose is 5 to 10 mg initially, which may be repeated every 15 minutes, as needed, up to a total dose of 30 mg. For seizure activity in a child, the recommended dosage of 0.25 to 0.4 mg/kg should be administered intravenously. In the child, diazepam doses may be repeated up to a total dose of 10 mg. Uncontrollable or recurrent seizures should be managed through administration of phenytoin.[14]

Excretion of chlordane (and a related organochlorine pesticide, chlordecone) may be accelerated by oral administration of cholestyramine, which fosters removal from the cycle of enterohepatic circulation. Dialysis, exchange transfusion, and hemoperfusion are probably ineffective.[14]

Heptachlor

Heptachlor epoxide has been identified as a metabolite of heptachlor in both animals and humans. In transformed human cell cultures, exposures both to heptachlor and to its epoxide induce unscheduled DNA synthesis, indicating possible genetic damage.[14] Heptachlor is absorbed following cutaneous contact, inhalation, and ingestion. The dose required to induce acute toxic effects in humans varies with the route and rate of exposure (see Figure 14-6).

Signs and Symptoms of Intoxication. Increased sensitivity to external stimuli and CNS hyperexcitability are the first adverse signs of exposure to heptachlor. Acute toxic signs of exposure include hyperactive reflexes, muscular twitching, tremors, ataxia, and clonic convulsions with or without coma. Respiratory depression may occur concurrently with convulsions. Repetitive cycles of excitability and depression may be seen. Cardiac irritability and contractility alterations can initiate dysrythmias.[23] Ingestion may cause nausea, vomiting, diarrhea, gastroenteritis, anorexia, or a delayed-onset hepatitis. Skin irritation has been reported following cutaneous contact.[14]

Aplastic anemia and neuroblastoma have been reported in patients following possible heptachlor exposures; however, a causal association has not been identified. If ingestion and subsequent aspiration of heptachlor pesticide formulations occurs, chemical pneumonitis should be anticipated.[14]

Medical Treatment. Patients contaminated with heptachlor should be decontaminated with soap and water. Some authorities recommend an alcohol wash following the soap and water, followed, in turn, by another soap and water wash. Three soap-and-water decontamination procedures have been recommended. Particular attention to decontaminating the hair is essential.[14]

Obtaining levels of chlorinated hydrocarbons or heptachlor in whole blood or serum is not useful in acute toxic exposures.[14] In most cases, blood levels of heptachlor or its metabolites reflect cumulative, rather than acute, exposures. Furthermore, these blood-chemistry analyses are usually not routinely available in most clinical laboratories. Because laboratory results will probably not be available to assist with early diagnosis or therapy, treatment must be based on a careful workplace—or intentional poisoning—historical profile. As with other organochlorine poisonings, if a history of heptachlor exposure is obtained, *the administration of adrenergic amines is to be avoided: they may increase myocardial irritability and precipitate refractory ventricular arrhythmias.*[14]

If an alert patient has ingested a possibly toxic dose of heptachlor, emesis should be induced with syrup of ipecac. Emesis is most effective within 30 minutes of ingestion of the poison. An activated charcoal slurry and cathartic should also be administered. Through interference with the enterohepatic circulation of the compound, cholestyramine has been reported to increase excretion of chlorinated hydrocarbons such as chlordecone and chlordane. Hemodialysis and ex-

change transfusion probably would not be effective therapeutic modalities for heptachlor poisonings.[14]

If seizures occur, diazepam should be carefully administered as an intravenous bolus of 5 to 10 mg. Diazepam may be repeated every 15 minutes, not to exceed a cumulative dose of 30 mg. If convulsions fail to respond to diazepam, or if they recur after initial therapy, phenytoin should be administered.[14]

Borate Insecticides

Boric acid, H_3BO_3, is also known as boracic acid or orthoboric acid. Its main use is as a common tablet formulation for pesticide management of cockroach infestation. Although the mechanism of action in humans is unknown, the end result is a metabolic acidosis with associated electrolyte abnormalities. Signs or symptoms of toxic exposure are not seen unless boron concentrations in the brain reach levels above 10 ppm (see Table 14-6).[31]

Routes of Exposure

Crawiing children can be exposed to boric acid if pesticide applications of the powder or pellet formulations are careless or inappropriate. Intact skin provides an effective barrier to absorption. Abraded or burned skin allows efficient absorption, however, although no mechanism for enhanced absorption across abraded skin has been proposed.

Borates are well absorbed following ingestion. In rare instances, infants have been inadvertently poisoned when powder formulations were mixed in their infant formula.[31]

Signs and Symptoms of Exposure

The signs and symptoms associated with exposure to and absorption of borate insecticides include abdominal pain, nausea, protracted vomiting, diarrhea, and hematochezia. These have been associated with absorption across burned or abraded skin. Topical exposure has been associated with a bright erythematous rash that may progress to extreme exfoliation. Restlessness, headache, weakness, and tremors may precede convulsions in severely poisoned patients. Cyanosis and shock may precipitate acute renal failure associated with metabolic acidosis from boric acid in severe cases of intoxication. Poisoning may be confirmed by blood borate concentrations. Normal ranges in nonexposed individuals are between zero and 7.2 mg of borate per liter of blood with a mean of 1.4 mg/L. Concentrations lower than 340 mg/L have rarely been associated with toxicity. Urine borate tests may yield false-positive results.[14]

Medical Treatment

Potentially contaminated skin should be washed with soap and water. Treatment of boric acid poisoning includes administering syrup of ipecac to children who weigh less than 30 kg, if the child has ingested more than 200 mg/kg as an acute dose. Larger individuals who have ingested a dose of more than 6 g are also treated with syrup of ipecac. If acute doses exceed these levels, emergency medical specialists may prefer to use gastric lavage as an alternative to inducing emesis.[14]

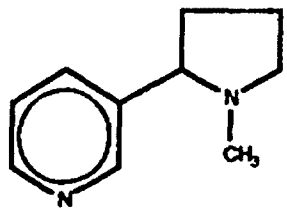
Activated charcoal does not absorb borate and should not be used unless there are special ingestion circumstances (eg, the ingestion of multiple substances). A blood sample drawn 2 to 3 hours after ingestion should be obtained to assess the severity of poisoning. If massive quantities have been ingested over several days, careful monitoring and medical management to prevent the adverse sequelae of metabolic acidosis and electrolyte abnormalities must be provided.[14] Excretion of boric acid is efficient and forced diuresis may afford some benefit. Exchange transfusion and peritoneal dialysis are efficacious.[31] Anticonvulsants are indicated for treatment of convulsions.[14]

Botanical Pesticides

Pesticides that are derived from living biological systems are chemically and pharmacologically diverse. In the broadest sense, this group includes relatively simple but potent molecules such as nicotine, complex proteinaceous poisons such as ricin, and the neurotoxin produced by *Clostridium botulinum*, which causes botulism. Many individuals share the common belief that a pesticide derived from natural sources has a greater margin of safety than a commercially manufactured pesticide. Carefully performed testing using standardized and accepted laboratory models has demonstrated, however, that some of the most potent poisons are derived from natural sources.

The mechanisms of action and possible antidote therapeutics for selected toxic compounds of biological origin have been carefully evaluated in the military's biological defense program. However, review and discussion of these toxins, their toxic mechanisms, and antidotes are not relevant to this discussion. Numerous pesticides derived from biological sources are available; however, the military uses only a few botanical derivatives such as the pyrethrins to eradicate or manage pests.

Nicotine



Nicotine is one of the most frequently recognized plant-derived pesticides. In addition to its commercial use as a fumigant and stomach poison for leaf-eating insects, its scientific use has provided valuable insight into the functions of the cholinergic components of the human nervous system. The toxicological mechanisms for cholinesterase inhibitors were clarified because of nicotine's cholinergic properties and reaction with specific sites (nicotinic receptors) within the CNS and PNS. Nicotine is not itself a cholinesterase inhibitor, but does have CNS, autonomic, and neuromuscular effects similar to excessive acetylcholine stimulation at nicotinic sites. Nicotine can exist in two isomeric forms: the synthetic R isomer is up to 8-fold more toxic than the natural S isomer.

There is no specific antidote for nicotine poisoning, but atropine administration may be somewhat efficacious in emergency medical management. In nicotine-poisoned dogs, artificial respiration has been shown to be life-saving if respiratory assistance was instituted prior to severe hypotension.[7]

Pyrethrum, Pyrethrins, and Pyrethroids

The insecticidal properties of the *Chrysanthemum* (Dalmatian pyrethrum flower) have been known for more than 100 years. Commercial growth of the flowers and production of the natural pyrethrum extracts (pyrethrins) made pyrethrum and pyrethrins available for domestic and agricultural pesticide use.[6] Widespread agricultural uses were initiated during the 1970s.[7] The naturally occurring pyrethrum components are produced from an oleoresin extract of dried chrysanthemum flowers and contain six active insecticidal ingredients collectively known as pyrethrins. These are used in a number of pesticide products, particularly aerosol products for indoor pest control. Advantages of pyrethrum and pyrethrins include their relatively low mammalian toxicity, rapid "knock down" and kill of pests, and the absence of environmental persistence. Disadvantages include the high cost of application and poor light stability.

As a result of efforts to decrease the unit production cost and to improve light stability, more than 1,000 synthetic compounds known as *pyrethroids* have been synthesized, some of which show significant structural differences and pest selectivities from the parent pyrethrum molecule (Figure 14-7).[7] Pyrethroids are light stable, biodegradable, and highly specific. For example, deltamethrin is approximately 3,000- to 5,000-fold more toxic to houseflies than to rats.[7,14,20,23]

Permethrin, one of the pyrethroid products, is a repellent that is used extensively by the military as a uniform impregnant (see Table 14-6). Some pyrethroids are approved for use as clothing spray formulations and others are used as pediculocides.

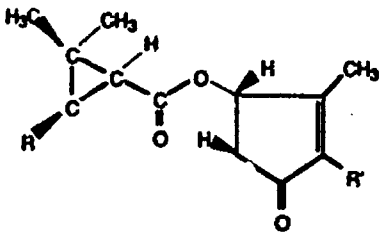
Route of Exposure. Pyrethrins are poorly absorbed via the dermis but appear to be well absorbed via the inhalational and ingestional routes. Although pyrethroids, if they are administered intravenously into laboratory animals, may cause extreme neurotoxic effects (convulsions), dermal and inhalation exposures are associated with only limited systemic toxicity.[16]

Mechanism of Action. Pyrethrins and pyrethroids have a high affinity for the sodium channel of the afferent neuron and produce their toxic effects as a consequence of neuronal hyperexcitability. As a consequence, pyrethroids cause a delay in channel closure, which results in a prolonged tail current of the action potential. The interaction slows the influx of sodium during the end of the depolarization phase, increases the depolarizing afterpotential, and results in repetitive discharges.[7,14,20] Pyrethroids are *open* channel blockers (ie, they selectively affect the active, or open, sodium channel). At high concentrations of pyrethroid, the nerve membrane may depolarize completely and excitability may be blocked.[7]

Allethrin and DDT have been shown to cause sodium channel effects in the lateral line organ of the toad. At the receptive portion of the peripheral afferent neuron, both chemicals cause a hyperexcitable stimulus response that results in the generation of a repetitive series of impulses. In the conductive portion of the afferent neuron, both compounds appear to impede the closing of the sodium channel, which results in an increased phase of hyperexcitability.[29]

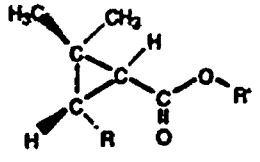
Within the CNS, pyrethroids exhibit effects consistent with several mechanisms of action. Proposed CNS mechanisms of pyrethroid effects include antagonism of the GABA-transmitter pathway, modification of nicotinic cholinergic transmission, enhancement of norepinephrine release, and alteration of calcium-ion fluxes. The primary effects of pyrethroids may be the result of changes in the functional sodium channel activity of the afferent neuron; the toxic CNS effects appear to be secondary.[7]

Pyrethrins of the Form



Compound	R	R'
Pyrethrin I	$(CH_3)_2C{=}CH-$	$-CH_2-CH{=}CH-CH{=}CH_2$
Pyrethrin II	$CH_3-O-C(O)-C(CH_3){=}CH-$	$-CH_2-CH{=}CH-CH{=}CH_2$
Cinerin I	$(CH_3)_2C{=}CH-$	$-CH_2-CH{=}CH-CH_3$
Cinerin II	$CH_3-O-C(O)-C(CH_3){=}CH-$	$-CH_2-CH{=}CH-CH_3$
Jasmolin I	$(CH_3)_2C{=}CH-$	$-CH_2-CH{=}CH-CH_2-CH_3$
Jasmolin II	$CH_3-O-C(O)-C(CH_3){=}CH-$	$-CH_2-CH{=}CH-CH_2-CH_3$

Pyrethroids of the Form



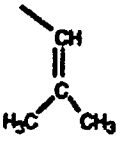
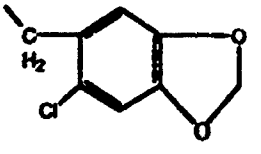
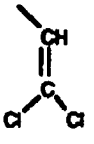
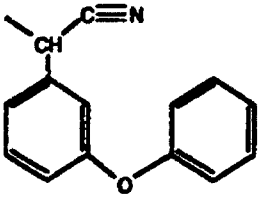
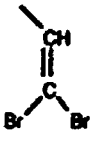
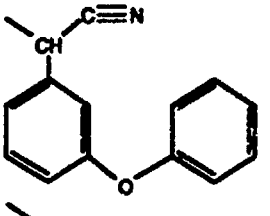
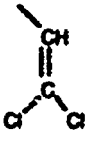
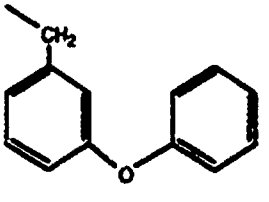
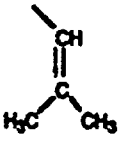
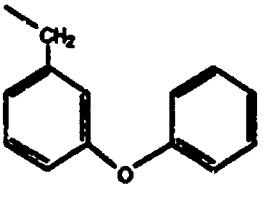
Compound	R	R'
Barthrin		
Cypermethrin		
Deltamethrin		
Permethrin		
Phenothrin		

Fig. 14-7. The chemical structures for representative pyrethrin and pyrethroid pesticides. Source: Ray DE. Pesticides derived from plants and other organisms. In: Hayes WJ Jr and Laws EP Jr, eds. *Handbook of Pesticide Toxicology*. New York: Harcourt Brace Jovanovich, Academic Press Inc; 1991; Chap 13.

Each pyrethrin and pyrethroid compound may form at least four isomers, and interaction with the sodium channels is highly dependent on the isomeric form. Permethrin and pyrethrum form isomers at the third carbon of the cyclopropane ring, with the *cis* and *trans* forms both demonstrating insecticidal activity. In mammalian systems, however, the *cis* isomers are approximately 10-fold more potent than the *trans*.[7]

Crude pyrethrum is a dermal and respiratory allergen; the refined pyrethrin products are less sensitizing and less irritating. A strong cross-reactivity with ragweed pollen has been shown. Pyrethrins do not inhibit the enzyme cholinesterase.[7,16] The rapid rate of hydrolysis of both the natural and synthetic molecules within the hepatic circulation probably accounts for the low acute toxicity in mammals, compared to the selective toxicity for insects.[7,16,20]

Most authorities agree that topical and systemic toxicities of the pyrethroids should be considered separately. In addition, most agree that there are two distinct types of systemic poisoning syndromes.[6,7] The different syndromes have been used to separate most pyrethroid compounds into two groups, types I and II, based on their clinically different manifestations of poisonings. In the rodent, the type I syndrome (tremor) is first recognized by an increased aggressiveness, followed by the rapid onset of tremor, hyperactivity, hyperthermia, clonic convulsions, and death. The type II syndrome (choreoathetosis with salivation) begins with profuse salivation, followed by a coarse body tremor, spontaneous writhing, tonic-clonic convulsions, and death.[6]

Permethrin and cismethrin are examples of type I compounds. Effects of cismethrin poisoning can be induced by placing a small quantity of the chemical within the CNS; as a result, the poisoning effects are believed to arise from central stimulation. In contrast, type II compounds such as deltamethrin and cyfluthrin act on a broader range of tissues and produce a more complex poisoning syndrome.[7]

Signs and Symptoms of Exposure. Acute intoxication with pyrethroids is uncommon. Patients may present with signs or symptoms of allergic skin reaction, eye irritation, skin irritation, or pulmonary allergic mediated bronchospasm. Extreme doses may cause salivation, tremor, incoordination, vomiting, and diarrhea. Irritability to sound has also been reported. Paresthesia has been associated with exposure to pyrethroids in humans who have experienced effects through local volatilization or liquid contact. Facial discomfort is reported most commonly; however, paresthesia of the neck, forearms, and hands are sometimes noted. Heat, sweating, sun exposure, and moisture may aggravate symptomatology. Paresthesia, itching, and/or burning sensations may progress to numbness.[16] Possible long-term effects in humans include the potential for carcinogenesis.[32]

Medical Treatment. Rapid decontamination is a mainstay of therapy; therefore, contaminated skin should immediately be washed with soap and water. Antihistamines are efficacious in controlling most allergic reactions. Severe asthmatic reactions may require bronchodilators and corticosteroid therapy. Vitamin E oil preparations are effective in relieving symptoms of local paresthesia. Corn oil is somewhat helpful, but zinc oxide aggravates the sensation.[7,16] Contact dermatitis may require prolonged use of topical corticosteroid preparations.[16]

After acute ingestion, careful gastric lavage may be followed by administering activated charcoal and a cathartic. There are no approved antidotes.[16]

As a precautionary note, the EPA states that although several drugs have been useful in the treatment of intentionally poisoned animals, none has actually been tested in humans. Therefore, neither the efficacy nor the safety of the therapeutic compounds has been evaluated.[16]

Some authorities recommend that therapy should be directed toward managing the functional neuropathological effects (hyperthermia, choreoathetosis, and seizures) until the pyrethroids are metabolized. The sedatives phenobarbital and pentabarbitol are effective against type I neurological effects when administered in anesthetic concentrations. Clomethiazole has been shown to be beneficial in deltamethrin poisoning (type II), especially when used with diazepam and atropine. Diazepam, when used alone, was found to be of limited benefit in the rodent model and only moderately effective in the canine model.[7]

Rodenticides

Rats and mice may ingest or spoil large quantities of stored food. Rodents may also directly transmit disease and cause discomfort or disease through bites. In addition, rodent parasites can be disease vectors. Efforts to control the rodent population may be directed toward removal of harborage, introduction of predators, use of traps, and use of chemicals directed toward poisoning of the rodent species.

Although numerous chemical compounds have been listed as rodenticides, including thallium, strychnine, phosphorous, phenyl- (PNU) and thio- (ANTU) ureas, and red squill,[23] civilian and military uses are usually associated with the anticoagulant compounds because they have a wider margin of safety (see Table 14-6).

The anticoagulant dicoumarol, first isolated from spoiled, sweet-clover hay, was identified as a cause of

lethal hemorrhagic disease in cattle during the 1920s. The beneficial medical therapeutic and possible rodenticidal properties were readily recognized and several related compounds were rapidly synthesized. Warfarin, a synthetic analog, was introduced for clinical trial in 1952 and used to treat President Dwight D. Eisenhower in 1955.[33]

Anticoagulant rodenticides are typically applied as baits, either in liquid or solid form. Bait formulations must meet these four requirements: they must be (1) effective in small quantities, undetected by rodents; (2) formulated to avoid bait shyness; (3) lethal without the rodents' becoming suspicious of the cause; and (4) used in a concentration safe for accidental human ingestion or formulated in a concentration specific to the target species.[29]

Mechanism of Action

Regardless of the anticoagulant bait form, the active ingredient is a derivative of coumarin, such as warfarin or fumarin; or a derivative of 1,3-indanedione, such as pival or diphasin. These compounds have similar modes of action. They are highly toxic when pure, but are incorporated into bait formulations in low concentrations. For example, warfarin concentrations incorporated into most baits range between 0.025% and 0.05%. Although detectable reductions of prothrombin can be identified in rodents within 24 to 48 hours of initial ingestion, the onset of actual hemorrhage in rodents generally follows ingestion of 1 to 2 mg daily for one week.[14,23]

Coumarins and indanediones are effective anticoagulants. In addition to its anticoagulant properties, warfarin damages capillary integrity, which precipitates internal hemorrhage. Indanediones induce signs of neurological and cardiopulmonary injury in the rodent toxicological model, but have not been shown to cause neurological or cardiopulmonary effects in humans.[14,23]

Warfarin is an antimetabolite of vitamin K and as such, warfarin depresses the hepatic vitamin K–dependent synthesis of essential clotting factors II (prothrombin), VII, IX, and X. The antiprothrombin effect is the best known and is the basis for detection and assessment of clinical poisoning. Lengthened prothrombin time from a toxic dose of coumarins or indanediones usually reaches a maximum 36 to 72 hours after acute ingestion. Newly developed superwarfarin compounds are much more potent and their anticoagulation effects more persistent. Warfarin has also been identified as a human teratogen that causes microcephaly, brain malformations, optic atrophy, nasal hypoplasia, and mental retardation.[14,23]

Route of Exposure

Gastrointestinal absorption of anticoagulant rodenticides is efficient, with warfarin well absorbed within 2 to 3 hours of ingestion. In a study of 14 subject volunteers, warfarin was administered orally at a dosage of 1.5 mg/kg. Maximal concentrations of warfarin were measured in plasma between 2 and 12 hours after ingestion. The maximal decrease in prothrombin activity was demonstrated between 36 and 72 hours after ingestion.[33]

Dermal absorption is slow but measurable in the rodent. Dermal absorption has not been reported as a cause of human poisoning. Toxic ingestion may occur if the bait is unintentionally consumed by a child or is intentionally consumed in a suicide attempt.

Signs and Symptoms of Intoxication

Signs and symptoms of anticoagulant ingestion include epistaxis, bleeding gums, petechial rash, hematomas, hemarthrosis, cerebral hemorrhage, shock, and death.

Medical Treatment

Medical treatment is probably not required if only a few grams of bait are ingested. If larger amounts have been ingested, syrup of ipecac followed by activated charcoal and cathartics may be efficacious. If the amount of the anticoagulant rodenticide ingested is unknown, phytonadione (vitamin K_1) given orally will protect against the anticoagulant effect with minimal risk to the patient. Phytonadione specifically is required: vitamin K_3 and vitamin K_4 are not antidotes for these anticoagulants.

If the patient is bleeding actively, careful intravenous administration of vitamin K_1 consistent with the specified dosage-administration rates is indicated, recognizing that adverse reactions and fatalities have been reported in association with this procedure. Actively bleeding patients should receive fresh frozen plasma or fresh blood transfusions in cases of severe bleeding.

Prothrombin times may be helpful in judging the severity of intoxication if the ingestion occurred during the preceding 15 days. The peak prothrombin effect occurs after about 3 days and prothrombin times should be followed to document peak effect and recovery.[14,23]

Herbicides

The compound 2,4-dichlorophenoxyacetic acid (2,4-D) has been used as a growth-regulating substance

with herbicidal activity since the early 1940s (see Figure 14-2).[8] 2,4-D has only moderate oral toxicity when administered to a variety of animal species. Large doses cause death quickly in animals, probably as a result of ventricular fibrillation. Lower doses were associated with myotonia, ataxia, paralysis, and coma. Of the mammalian species tested, the dog was the most sensitive, with an oral LD_{50} of 100 mg/kg. The no-effect level in a 2-year canine study was approximately 12 mg/kg/day.[34]

Limited information is available with respect to human exposures and toxic doses. In humans, 100% of the radiolabeled dose administered by intravenous injection was recovered in the urine. In contrast, only 5.8% of the topically applied dose was excreted in the urine, suggesting that dermal absorption is limited.[34]

Route of Exposure

Workers exposed to herbicides via inhalation, ingestion, or dermal contact may experience clinical effects. Skin contact is the more common exposure route; however, dermal absorption is not efficient.[34] Inhalation and ingestion are also possible routes of exposure.[14]

Signs and Symptoms of Intoxication

Although a wide variety of clinical reactions to exposure is possible, depending on the exposure route and dose, irritant responses are most commonly reported effects of exposure.[14] Irritant responses have been reported from cutaneous contact, airborne or inhalation exposure, and ingestion. Chloracne from the dioxin contaminants in the related herbicide 2,4,5-T has been reported in heavily exposed workers (see Figure 14-2).

Eye, nose, and throat irritation may be associated with airborne exposures. Pulmonary edema has been reported following inhalation exposures. Ingestion has been reported to cause mouth, esophagus, and stomach irritation sometimes associated with vomiting and diarrhea. Elevated liver enzymes—lactic dehydrogenase (LDH), serum glutamic-oxalacetic transaminase (SGOT), and serum glutamic-pyruvic transaminase (SGPT)—have been reported.[14]

Neurological consequences of occupational exposures have been reported to include vertigo, headache, malaise, and paresthesias. Higher doses may produce muscle fasciculations, followed by profound muscle weakness and unconsciousness. Rhabdomyolysis and myotonia have been reported in severely poisoned persons.[14]

Tachycardia has been reported as a common manifestation of cardiac toxicity but the occurrence of actual arrhythmia has been uncommonly reported. Reported renal effects include albuminuria and azotemia.[14]

Headache, dizziness, stomach pains, nausea, leukopenia, fever, urinary incontinence, hypertonia, and constipation have been reported in humans after accidental or intentional ingestion. Skeletal muscle fasciculations have also been reported. Although myotonia is the most frequently identified sign of poisoning in animals, it is unusual in poisoned humans. Degeneration of the renal convoluted tubule, glomerular protein deposition, and limited fatty infiltration of the renal parenchyma have been reported in an accidental ingestion by a farmer.[34] Serious acute human poisonings have been reported after the ingestion of multigram doses.[14] An analog of 2,4-D, clofibrate, is used clinically to lower cholesterol levels.[34] These herbicides can be measured directly in plasma and urine by gas liquid chromatography. They do not affect cholinesterase levels.[14]

Medical Treatment

If eye exposure occurs following a splash, the eyes should be irrigated with copious amounts of water for at least 15 minutes. If a direct splash into the eye has occurred, the patient should be taken to a medical treatment facility (MTF) for evaluation and necessary eye care.[14]

In cases of skin contact, the affected area should be thoroughly cleansed with soap and water. If acute irritation occurs, evaluation at the health clinic is indicated.[14]

Although massive overexposure could require the basic life-saving interventions such as airway management, this is usually unnecessary. Respiratory depression, hypotension, metabolic acidosis, hyperthermia, or seizures may occur in severely poisoned individuals.[14]

Emesis is indicated, using syrup of ipecac, if intentional ingestion has occurred and if the patient is conscious. This emetic is most effective when administered within 30 minutes of ingestion. Removal of the stomach contents via nasogastric intubation is indicated if the patient is obtunded, comatose, or convulsing. Activated charcoal administration and catharsis are recommended following ingestion of chlorphenoxy pesticides.[14]

If ingestion could result in toxic clinical manifestations, the following baseline determinations should be obtained: complete blood count, arterial pH, bicarbonate, serum creatinine, blood urea nitrogen (BUN), liver enzyme levels, urinary protein, myoglobin, and erythrocyte losses; urinary output should also be measured. Liver enzymes should be monitored to evaluate

the significance of possible hepatic injury. Because muscular tissue destruction may occur, baseline levels of serum creatine phosphokinase and myoglobin should be measured. Ongoing evaluations of liver enzymes and myoglobin levels should be performed following intentional ingestion. When significant myoglobinuria is seen, alkaline diuresis should be instituted to enhance elimination.[14] Alkaline diuresis appears to improve renal clearance of 2,4-D.

If inhalation has occurred in an enclosed area, the patient should be removed to fresh air. The patient may experience irritation of the respiratory tract as burning discomfort in the airway. Cough and respiratory distress may follow high-dose inhalation exposures as evidence of pulmonary edema. Bronchitis or pneumonitis may occur. Supplemental humidified oxygen or assisted ventilation may be required in extreme cases.[14] The treatment of 2,4-D exposure is symptomatic, with this exception: quinidine sulfate has been used in the management of tachycardia and may be helpful in treating the skeletal muscle dystonia.[34]

Many chronic adverse health effects from exposures to this group of pesticides have been alleged. However, in a 20-year follow-up of the health status of U.S. Air Force veterans who were exposed to herbicides in Vietnam, only basal cell carcinoma was more frequently identified among the herbicide handlers.[35] The possibility that the association was spurious has been noted.[36]

Sodium Arsenite

Sodium arsenite ($NaAsO_2$) is used as aqueous solution for weed control, and it has limited use as an insecticide.[16] Arsenical compounds demonstrate a spectrum of toxicity based on their chemical composition and arsenic valence state. The generally accepted order of toxicity is from the more toxic arsine (trivalent); through the organo-arsine derivatives, arsenites (trivalent); arsenoxides (trivalent); arsenates (pentavalent); other pentavalent organic compounds; arsonium metals (monovalent); to the least toxic, metallic arsenic. The active component of sodium arsenite is the arsenite, or trivalent arsenic, moiety. Arsenite has been shown to be much more toxic than the pentavalent form, arsenate.[14,16]

Mechanism of Action

Acute ingestion of more than 100 mg of the arsenite compound has been reported to be associated with significant toxicity. Ingestion of 200 mg of a related compound, arsenic trioxide, may be fatal in an adult.[14]

Sodium arsenite has been reported to be a potent cause of a number of cutaneous lesions, including corrosive ulceration. In addition, among pesticide applicators, it has been associated with erythema, papular dermatitis, and folliculitis. Among previously sensitized patients, some cases of folliculitis may actually be seen at low-exposure concentrations as a result of allergic cutaneous responsiveness. Ulceration of the hands, feet, and scrotum have been reported. While trivalent arsenic compounds are associated with skin corrosion, arsenic trioxide and pentoxide compounds are usually reported as skin sensitizers.[14]

Although arsenic compounds have been associated with both skin and pulmonary cancer in human epidemiological studies, arsenic has not yet been reported as a carcinogen in standard laboratory animal models. Arsenic is identified as a human carcinogen in the statutory occupational requirements promulgated by OSHA. The EPA has been actively reviewing the issue of potential carcinogenicity and has recently acted to withdraw product registrations.

Small doses of arsenic-containing compounds induce vasodilation. Increasing doses stimulate capillary dilatation and increase capillary permeability. Transudation of a large volume of plasma may cause profound hypotension with secondary arteriolar and myocardial damage. Abnormalities of the electrocardiographic record may persist for months following acute poisonings.[14]

Inorganic arsenicals cause increased blood flow through the bone marrow, which alters the marrow's cellular composition. Moderate doses of inorganic arsenicals depress the production of both erythrocytes and leukocytes, possibly through inhibitory actions on folic acid interactions.[14]

Inorganic arsenicals are considered to be potent hepatotoxins. Poisonings have been associated with central necrosis, fatty infiltration, and cirrhosis. Acute yellow atrophy and death may occur.

In a 1977 report concerning the state of California, 291 of 2,228 pesticide exposures involved sodium arsenite ingestion. Dosages as low as 1 mg/kg may induce serious toxicity and dosages as low as 2 mg/kg may be lethal.[14]

Route of Exposure

Acute poisonings are associated with both intentional and accidental ingestion. Chronic oral intoxication is associated with arsenical compounds used as medicaments. Skin contact may result in localized pathology but has not been associated with poisonings. Inhalational exposure is possible, depending on the exposure circumstances.[14]

Signs and Symptoms of Intoxication

Acute symptoms usually occur within 30 minutes to 1 hour from the time of ingestion unless simultaneously consumed food delays absorption. A garliclike odor may be noticeable in the breath or feces. Dysphagia, esophageal pain, stomach pain, colic, and profuse, watery—sometimes bloody—diarrhea have been reported. Dehydration, hypotension, tachycardia, and fluid and electrolyte disturbances are common. Hypovolemic shock, sometimes with associated intestinal blood loss, and cardiac abnormalities including tachycardia, QT interval prolongation, alterations in the T wave, and ventricular fibrillation have been reported in acute exposures. Chronic exposures have been associated with myocarditis.[14]

Acute exposures by direct contact with trivalent arsenic-containing compounds can cause eye, mouth, and skin corrosion. Chronic exposures have been associated with cutaneous and nasal septal ulcerations, and nasal septal perforations have been reported. Cutaneous responses to chronic arsenical exposures may include hyperpigmentation, keratoses, and epidermoid carcinomas.[14]

Acute arsenic exposures have been associated with CNS and PNS effects including alterations of mental status, convulsions, toxic delirium, chemical-induced encephalopathy, and delayed peripheral neuropathy. Hematuria and acute tubular necrosis have been reported complications of acute arsenic poisoning.[14]

Acute poisoning can cause hemolysis. Chronic arsenic exposures have been associated with bone marrow depression, pancytopenia, aplastic anemia, and leukemia.[14] Inorganic arsenicals may cross the placenta and have been associated with fetal death if chelation therapy was not prompt.[14]

Medical Treatment

Individuals with a history of possible acute arsenic ingestion require careful medical evaluation and timely medical management. A complete blood count, urinalysis, and baseline levels of serum electrolytes, liver enzymes, BUN, and creatinine should be obtained immediately. Urinary and blood arsenic levels should be obtained. A 24-hour urine specimen should be analyzed for arsenic excretion. Urinary arsenic excretions exceeding 100 µg have been reported to indicate abnormal excretory levels.[14] However, individuals who consume diets rich in seafood may excrete 200 µg or more of arsenic per day.[16] Individuals with possible surface contamination should be washed with soap and water. Decontamination of all areas, including the hair, should be thorough.[11,16]

Chest and abdominal X rays should be obtained for all patients being evaluated for arsenical ingestion. Timely, thorough gastric lavage is indicated for patients with acute, possibly toxic, ingestion. Whole-bowel irrigation has been recommended if radiography identifies arsenicals in the intestinal contents. Although activated charcoal has somewhat speculative benefit, its use is recommended following lavage. If there is no primary diarrhea associated with the arsenic ingestion, the administration of sodium sulfate as a cathartic should be considered.[14] Morphine may be administered in cases of intense abdominal pain.

Aggressive fluid and electrolyte management is required for individuals who ingest arsenicals, and is especially critical for those who are hypovolemic. A high output of alkaline urine should be maintained.[14] In some circumstances, pulmonary edema has been associated with arsenic poisoning.[14]

Symptomatic patients should be promptly treated with the chelating agents dimercaprol (2,3-dimercaptopropanol, also known as British anti-Lewisite [BAL]) and penicillamine. To avoid adverse effects from BAL administration, it should be administered intramuscularly at a rate of 3 to 5 mg/kg/dose every 4 to 12 hours. The dose requirement and frequency of administration should be correlated with the degree of arsenical poisoning. Tapered doses of BAL should be continued for 5 to 8 days in patients who are allergic to penicillin.[14] Another authority recommends somewhat lower dosages: 2.5 to 3.0 mg/kg/dose every 4 hours and tapered over about 2 weeks.[16]

As the signs and symptoms of acute arsenic poisoning subside as a consequence of BAL therapy, penicillamine should be prescribed as soon as possible for patients who have no history of penicillin allergy. For adults, the recommended dose for oral administration of D-penicillamine is 100 mg/kg/day up to 2 g daily, provided in four divided doses for a total of 5 days. The recommended dosage for children is 25 mg/kg/day in four divided doses daily, not to exceed a total of 2 g.[14] Another authority recommends, for children younger than 12 years, 100 mg/kg/day in 4 divided doses, not to exceed 1 g daily.[16]

Combined BAL and penicillamine therapy should be considered for severely poisoned patients. The dosages of chelating agents must be adjusted if renal complications occur as a result of the arsenic exposure. Hemodialysis may be necessary if renal function is impaired and if removal of the chelated arsenical complex is desired. Dimercaptosuccinic acid is currently being investigated as an alternative to BAL.[14]

Complications associated with the administration of BAL include acute signs and symptoms such as nausea, headache, restlessness, anxiety, paresthesia,

pain, tearing, tachycardia, and hypertension. In selected patients, antihistamines may be beneficial in managing these complications. Other common side effects from BAL administration include maculopapular rash, fever, depressed leukocyte counts, eosinophilia, lymphadenopathy, and joint pain. BAL therapy has also been implicated in a number of other, less common complications.[14,16]

PESTICIDE LEGISLATION

Numerous federal, state, local, and DoD regulations have been promulgated to ensure the proper use and disposition of pesticides. A brief history of federal pesticide legislation may help the reader understand the political concerns about pesticides.

Pesticide legislation is in a constant state of flux. Current laws continue to be amended and new laws continue to be enacted. The purpose of this legislation is to protect those who apply pesticides, the bystanders, and the environment from the harmful effects of pesticide residues. Therefore, all participants in the pesticide section of the occupational health program must be familiar with the current, pertinent military regulations and the requirements of federal, state, and local regulations.

The Federal Food, Drug, and Cosmetic Act

The first federal law regarding pesticides was the Food and Drug Act of 1906. It required that food shipped in interstate commerce be pure and wholesome. Although pesticide residues were not addressed in this act, their exclusion was eventually identified and the act was completely rewritten. The 1938 legislation was called the Federal Food, Drug, and Cosmetic Act; it established the allowable levels of pesticide residues on foods. This legislation was the federal government's first attempt to protect consumers from foods that had been contaminated with pesticides.

In 1954, Public Law 518, commonly called the Miller Amendment, amended the 1938 Federal Food, Drug, and Cosmetic Act. This amendment established pesticide *tolerances* (allowable residues on a raw agricultural commodity). The Miller Amendment stated that a commodity could be considered adulterated if (*a*) it contained a pesticide residue that had not been cleared for safety or (*b*) it exceeded the allowable tolerance.

The Food Additives Amendment, enacted in 1958, further amended the Federal Food, Drug, and Cosmetic Act in 1958; it regulated the use of food additives and established pesticide tolerances in processed foods. An extremely important part of this amendment was the Delaney Clause, which prohibited the use of a pesticide or a food additive that had been shown to cause malignant tumors in laboratory animals at any dose.

The Federal Insecticide, Fungicide, and Rodenticide Act

The first law regulating the transportation of pesticides in interstate commerce was enacted in 1910: the Federal Insecticide Act was designed to protect farmers from the distribution of fraudulent and substandard pesticide products. In 1947, the Federal Insecticide, Fungicide, and Rodenticide Act was enacted. It superseded the 1910 Act and required that pesticide products be registered and labeled with the U.S. Department of Agriculture before being shipped in interstate commerce. The Federal Insecticide, Fungicide, and Rodenticide Act also attempted to ensure the safe use of pesticides by requiring them to be labeled with

- the manufacturer's name and address;
- the name of the pesticide;
- the net contents;
- a statement of ingredients;
- warnings to prevent injury to humans and other animals, plants, and organisms that are not targets; and
- directions for use that would protect the user and the public.

In 1972, the most important revision of the Federal Insecticide, Fungicide, and Rodenticide Act was completed; this revision, entitled the Federal Environmental Pesticide Control Act, prohibited the use of any pesticide that was inconsistent with the warnings and directions on the label. In other words, *the label was the law*. Another provision of the 1972 revision regulated pesticides within states, not only those involved in interstate commerce; this was an important development in the regulation of pesticides on a national level.

The Federal Environmental Pesticide Control Act also required that pesticides be categorized as *General Use Pesticides* and *Restricted Use Pesticides*. General Use Pesticides are those pesticides that the public uses. Restricted Use Pesticides must be applied by, or under the direct supervision of, trained and certified personnel. A certification program was mandated within each state to train and certify the personnel who could apply the pesticides included in the Restricted Use category.

The Environmental Protection Agency

In 1969, the National Environmental Policy Act became a law and established the EPA. The legislation transferred the authority for pesticide regulation and registration from the U.S. Department of Agriculture to the EPA. At the same time, the authority to establish pesticide tolerances was transferred from the Food and Drug Administration to the EPA. However, the authority for enforcement of tolerances remains with the Food and Drug Administration.

Occupational Safety and Health Regulation

In 1970, the Williams-Steiger Occupational Safety and Health Act was enacted into law. OSHA was established as the regulatory authority to implement the act and establish policy under the U.S. Department of Labor. The National Institute for Occupational Safety and Health (NIOSH) was established, as an integral requirement of the act, to provide scientific guidance and recommendations for the regulatory authority. Executive Order 12196, dated 26 February 1980, required federal agencies to comply with OSHA requirements. OSHA promulgates regulatory requirements and documents the current annual requirement in the most recent volume of Title 29, Code of Federal Regulations (CFR), part 1910, *Occupational Safety and Health Standards*. OSHA publishes periodic updates and revisions of 29 CFR 1910 in the Federal Register (FR) and documents revisions to the published requirement or interim supplementation with new requirements. When the annual copy of the CFR is published, the new edition automatically incorporates all interim changes published in the periodic FR supplements. In addition to providing regulatory requirements, the CFR also documents the current, mandated PELs. In some cases, specific medical requirements are identified.

DoD Directive 1000.3 (29 March 1979, with Change 1, 17 April 1979) established military policy and provided implementation guidance for occupational safety and health. Health specific requirements for the Department of the Army (DA) are identified in Army Regulation (AR) 40-5, *Preventive Medicine, Health and Environment*.

For occupational health professionals to provide appropriate care for military employees and soldiers, they must be aware of the legal and regulatory requirements that direct the provisions for care. Federal legal requirements mandate that occupational illnesses and injuries are to be reported on the OSHA log. Army regulatory requirements specify that workers diagnosed with occupational illnesses, such as symptomatic pesticide-exposed workers, are to be identified in military occupational and safety health reports. State legal requirements are variable. Maryland and California are examples of states that require physicians who are licensed by the state to report all illnesses of occupational etiology.

THE U.S. ARMY PESTICIDE OCCUPATIONAL HEALTH PROGRAM

Any organization, installation, or activity that uses or stores pesticides must have an Occupational Health Program to monitor pesticide use and to implement procedures to protect the health of workers. The program addresses (*a*) health reports, records and forms, (*b*) protective equipment, (*c*) emergency medical treatment, (*d*) pesticide handling and applications, (*e*) pest-control equipment and facilities, (*f*) field occupational health, (*g*) medical surveillance, and (*h*) action levels for medical removal and return-to-work policies.

Health Reports, Records, and Forms

Command headquarters (eg, Health Services Command [HSC] or Army Materiel Command [AMC]) must be informed—through mandatory command health reports—of environmental releases and health-related problems involving pesticides. Environmental accidents resulting from the use, storage, or disposal of pesticides should be identified on any health report sent to the command headquarters. Confirmed or suspected health-related problems associated with occupational exposure to pesticides must also be reported. The purposes of the report are to (*a*) provide essential information about the circumstances associated with the problem, (*b*) identify additional resources that are necessary to solve the problem, and (*c*) provide information concerning problem resolution.

Extensive occupational health records, personnel records, and exposure monitoring records are required to provide and document the healthcare provided to all workers who handle pesticides (see Chapter 3, U.S. Army Health Programs and Services). The personnel office should work closely with the pesticide program management to assure that all job descriptions clearly delineate the worksite requirements and the medical conditions that cannot be accommodated in the pesticide-workshop environment.

After employment, preplacement examinations must be provided for pesticide applicators. These

document the patient's baseline health status and ensure that PPE can safely be used (eg, respirator fit and heat tolerance). Careful administrative management procedures must be provided to ensure that there is appropriate coordination between all health and safety personnel who are required to generate or maintain (or both) records related to employment.

Comprehensive records and forms should be maintained for all pest-control or pesticide-handling workers. To ensure that working conditions are in compliance with OSHA requirements, appropriate industrial hygiene site-visits, pesticide-applicator monitoring data, and safety-surveillance records are vitally important. Accurate sampling, analysis, recording, and interpretation are necessary in work areas where potential pesticide exposures may occur (eg, in pest-control shops and pesticide-storage warehouses). These results should also be prominently posted in the work area to notify employees and their supervisors.

An important method used to identify the need to mitigate exposure or to modify work practices is to obtain workplace data concerning pesticide concentrations associated with storage or use. As noted previously, the concentrations and durations of potential exposure to a pesticide are critical variables in this hazard evaluation. Exposure data should be carefully obtained by industrial hygienists when circumstances indicate that unacceptable exposures could occur in the workplace. The industrial hygienist should (*a*) collect data and keep a documented record, (*b*) formally notify the employee, area supervisor, and occupational healthcare provider, (*c*) identify corrective actions, and (*d*) ensure their implementation to reduce potential exposures, if monitoring data reveal concentrations of pesticides above the action level. Documented exposures above the action level and all pertinent information must be provided to the occupational health clinic in order to allow appropriate planning for medical care.

Individuals who are required to perform potentially hazardous operations (using engineering controls or PPE to mitigate or prevent their exposures) should also be afforded appropriate medical surveillance examinations to document that their health is maintained. The absence of adverse health effects among these workers may be useful to demonstrate the efficacy of engineering controls and PPE.

The health history obtained from employees prior to, periodically, and at the termination of employment must be carefully documented and retained for a minimum of 30 years after the termination of employment.[3] In addition, results from any atmospheric sampling should be included and retained in the military or civilian worker's medical records. These results should be identical to those that have been posted in the work area. The results of continuing medical surveillance or exposure-related medical examinations or clinical laboratory analyses must be carefully reviewed, compared with the preexisting information, and documented. Special care should be exercised to properly document and report workplace accidents, injuries, and illnesses in the medical records. Healthcare providers must ensure that Department of Labor forms for reporting illness or injury and compensation are completed in a timely fashion.

Emergency Medical Treatment

All employees who store, handle, and apply pesticides should be trained and able to practice emergency measures that ensure both the safe removal of exposed individuals from the contaminating source and their careful decontamination. All employees should receive some basic life-support training. Employees should demonstrate the appropriate use of PPE to preclude self-contamination and be able to implement the necessary procedures to assure that the spread of contamination will be limited.

Because pesticide antidotes are prescription medications, such antidotes should not be provided for employees to use unless the individual employees are properly trained and granted clinical privileges to provide emergency-response medical care. In certain circumstances where extremely toxic chemicals are involved—such as military nerve agents—employees may be allowed to carry the antidote, administer buddy aid, and inject themselves. Nonmedical personnel who are expected to use prescription antidotes and perform emergency care must learn to recognize the

- events that precipitate use,
- quantity and frequency of administration, and
- adverse effects associated with such use.

Unless access to first-aid kits can be carefully controlled, they are used only by trained individuals, or the work is being performed at a remote site, first-aid kits should not be available or used at the workplace. If managers decide that first-aid kits should be placed in work areas, medical personnel must approve the kits' contents, and pesticide workers must receive approved first-aid training. Procedures for safe first-aid kit use, kit resupply, accident reporting, and appropriate medical follow-up evaluation should be carefully documented.

The healthcare facilities that provide occupational health or general medical services must maintain a pharmaceutical inventory with necessary quantities of specific and appropriate antidotes, reversal agents,

and anticonvulsants. In addition, the facility must incorporate sufficient emergency-support equipment to manage an emergency situation, and healthcare providers must be properly trained and medically credentialed. Emergency-support agreements with nearby evacuation services and hospitals should be implemented and continuously revised to assure that timely support is available.

Pest-Control Equipment and Facilities

Industrial hygiene, safety, and occupational health personnel should ensure that (*a*) pest-control equipment is compatible with the pesticide formulation that is being applied, (*b*) the equipment is available and calibrated properly, and (*c*) the pesticides are transported properly. Engineering controls and PPE should be certified as operational. The appropriate care and use of the equipment should be documented. The following factors must also be considered in the transportation of pesticides:

- Vehicles used to transport pesticides, particularly pest-control vehicles, should be equipped with lockable storage areas and separate cabs for passengers.
- Transporting pesticides in the cabs should be prohibited.
- Vehicles assigned to the pest-control shop should be used only for pest-control activities.
- Pesticide spill kits should be placed on each vehicle.
- A portable eyewash should be available on vehicles that are located at remote pesticide-application sites.
- Emergency telephone numbers should be posted on pest-control vehicles.

Pesticide storage and mixing facilities must conform to not only federal workplace safety and health requirements, but also to state and local fire codes.[3,37] Pesticide labels, Material Safety Data Sheets, safety data prepared by the manufacturer, and a current pesticide inventory for the pesticides that are stored and in use should be available for the employees' review. Plans to adequately contain a pesticide fire at the facility should be prepared and updated annually. Copies should be provided to the local fire department, police department, hospitals, and safety offices.[38]

Pesticide Applications

Only personnel who are trained and certified should apply pesticides or supervise their application. Scheduled, periodic pesticide treatments should be prohibited unless a pest-control professional specifically approves, and these preventive treatments should be done only if surveillance has indicated past or current problems with pests. Furthermore, at least two pest controllers should perform pesticide operations such as fumigation that are particularly hazardous.

Food-handling areas and MTFs require special considerations for pest management; nonchemical pest-control methods should be attempted before chemical measures are considered. In food-preparation areas, pesticide treatments should be conducted only (*a*) when the food-preparation area is not in operation and (*b*) according to the pesticide label's instructions. Pesticides should not be applied routinely in MTFs, but *only* when the pest infestation warrants the use of a pesticide and then *only* administrative or storage areas should be treated. Pesticides should *not* be applied in patient-sensitive areas such as intensive care wards, emergency rooms, or infant nurseries. It is particularly important that pesticides *not* be applied in neonatal wards: infants have low blood cholinesterase; cholinesterase-inhibiting pesticides used in neonatal wards could cause serious health problems.

A pest-management coordinator should be appointed in each MTF. All pest sightings should be reported to the coordinator and any actions taken to control pests in the facility, including the use of pesticides, should be documented and maintained by the coordinator. Specific guidance for pest management operations in medical treatment facilities is provided in Armed Forces Pest Management Board Technical Information Memorandum 20.[39]

Medical Surveillance

Comprehensive medical surveillance is an essential element of a functional occupational health program. As a program element, the term *medical surveillance* is a misnomer. The surveillance element is composed of (*a*) general medical and specific occupational exposure history review, (b) target-organ-system-focused medical examination, (*c*) selected clinical laboratory analyses, and (*d*) medical intervention, depending on examination findings. In contrast to its use as a program element, in general preventive medicine the term medical surveillance is a separate, descriptive term. In that context, medical surveillance is a type of secondary prevention, directed toward identification of exposure effects at the time of organ-system injury, prior to the onset of permanent damage (impairment). For comparison, the term biological exposure index (BEI) is used as an element of primary prevention to identify and measure the specific chemical, or its metabolites,

in biological fluids. Measurement of the chemical or its metabolite may not prove to be medically useful except to define whether a possible exposure has occurred. For the BEI to have maximal utility, a dose-response relationship for humans must have been determined for the chemical or metabolite that has been measured.

The toxicities of the chemical materials, the potential for cumulative effect, and the potentially serious medical consequences of long-term, low-dose exposures make a medical surveillance program essential for pesticide workers. The interdependent industrial hygiene, safety, and medical factors that exert important influences on the type and extent of medical surveillance programs, include

- the number, amount, and toxicity of the pesticides being handled;
- the potential hazards associated with the formulations;
- the potential hazards associated with the applications that are being performed;
- the presence or absence of a well-ventilated, properly designed and constructed pest-control shop or warehouse;
- the degree of compliance with procedures that are intended to minimize pesticide health hazards; and
- the extent of industrial hygiene surveys related to personnel exposures and the results that are obtained with a workplace environmental survey program.

As a tool to tailor medical surveillance for the individual pesticide worker, a health and safety hazard evaluation is necessary to fully analyze the cumulative importance of the factors. For example, potential exposures of pesticide workers are, as a rule, not restricted to one particular substance. Usually a number of pesticides, formulation components, solvents, and cleaning agents with very different modes of action and degrees of toxicity are used intermittently and simultaneously. The comprehensive set of employee-specific exposure possibilities represents the individual's *exposure profile*. A comprehensive medical surveillance program is optimally developed to identify the employee who has had a gradual decrement in cholinesterase as a result of ongoing organophosphate exposure before the worker experiences symptoms from the application of a carbamate insecticide.

Medical surveillance examinations—preplacement, periodic, and termination—should be specifically designed for each employee: the contents of the medical evaluation should be determined by the potential exposure profile of that employee. Specific medical surveillance tests or appropriate biological exposure indices should be identified for the employee on a case-by-case basis. If a generalized exposure potential to numerous chemicals exists, an extensive surveillance examination is usually required. The healthcare provider should coordinate with industrial hygienists, safety professionals, and supervisors to design specific examinations focused to the individual employee. Although coordination among these individuals fosters a better medical understanding of the workplace, the healthcare provider must remember to maintain the confidentiality of medical information.

Medical surveillance examinations of pesticide workers are provided for all employees who have the potential for exposure to pesticides in excess of either the statutory (29 CFR 1910) or the recommended exposure levels. The ACGIH annually publishes the current, revised set of recommended exposure limits and action levels. Both OSHA and ACGIH limits identify levels that should be safe for the traditional worker exposure (40 h/wk for the duration of employment). This examination is provided for any employee who has the potential to become exposed at or above the legal, regulatory, or advisory exposure levels despite the presence or use of engineering controls and PPE or if administrative work practices fail.

Preplacement Examination

Preplacement examinations are performed before an employee is assigned to the worksite. As a result of the Americans with Disabilities Act (ADA, which became effective in 1992, and by 1994 will cover companies with more than 15 employees), the character and type of examination performed prior to employment or job assignment has changed dramatically.[40] In short, before the statutory requirement was implemented, individuals presumed to be at increased risk from exposure could be excluded from employment or particular positions. Since the ADA became effective, however, it is illegal to refuse employment or placement for the individual who may be ill-suited to perform the job unless specific conditions of employment are published in the job description. If a potentially susceptible employee is hired in the absence of specific exclusions (conditions of employment), the work site must be reconfigured to accommodate the employee.

The preplacement examination for potential pesticide workers, performed by a physician or properly privileged and supervised healthcare provider, should include

- a comprehensive medical and work history;

- a physical examination with particular attention to the cardiovascular and respiratory system to evaluate the employee's ability to use respiratory protective equipment;
- an examination of the hepatic and renal systems to ensure that employees will not be unusually susceptible to ill effects from pesticides, formulation products, solvents, or cleaning materials;
- examinations of the musculoskeletal and nervous systems to identify preexisting neurological disorders, including
  - evaluations of PNS and CNS functions, and
  - mental status and limited neuropsychiatric evaluations;
- a chest X ray;
- spirometry, including
  - forced vital capacity (FVC) and
  - forced expiratory volume at 1 second (FEV_1);
- a complete blood count;
- liver function tests (such as SGOT and LDH);
- renal function tests (such as creatinine and BUN).

The preplacement examination may include a variety of other elements if indicated by the potential exposure profile. For example, for individuals who will be required to work with the organophosphate insecticides, one component of the examination is the determination of the baseline erythrocyte cholinesterase level. Subsequent analyses during periodic examinations or after suspected exposures will be evaluated by comparing with the baseline level.

The erythrocyte cholinesterase baseline determination is defined as the average value of three separate erythrocyte-associated cholinesterase measurements obtained during a 9- to 14-day period. Cholinesterase measurement methods must be subjected to judicious quality control procedures, both inside and outside the laboratory. Quality control is essential to assure consistently reproducible and reliable results. Laboratory consistency is critical because "normal" levels vary widely among individuals, but change very little over time in the same individual. Several methods have been developed for the analysis of cholinesterase levels.[41] Extreme care must be taken to ensure that all subsequent samples are analyzed by the same, carefully controlled, technique. Results of each test must be reviewed by an individual who has been granted clinical privileges to provide this type of care.

The erythrocyte cholinesterase baseline and all subsequent test results should be documented graphically (Figure 14-8). Individuals whose erythrocyte cholinesterase is depressed more than 25% should receive an immediate medical evaluation and then be removed from further exposure to cholinesterase inhibitors. Individuals can be cleared to return to work when the enzyme levels reach 80% of the patient's baseline.

Plasma cholinesterase (sometimes called pseudocholinesterase or butyrylcholinesterase) may be significantly changed with short-term, relatively high-dose exposures to cholinesterase inhibitors. The reactivation and replacement kinetics of the plasma-associated enzyme do not permit their being used in routine surveillance, although the plasma measurement can be used to confirm a very recent pesticide exposure.

Periodic Examinations

A physician or privileged healthcare provider should perform a periodic examination, based on the worker's potential for exposure to pesticide levels above the action levels identified by OSHA or the ACGIH. The examination should be focused within the scope of the preplacement examination and should be dependent on the potential exposure hazard. The frequency of this examination is arbitrary; it can range from annually to a frequency that depends on the worker's age and health. For example, an age-related examination could be performed on individuals with a minimal potential for pesticide exposure: workers younger than 40 years of age would be examined every 4 years; workers 40 to 49 years of age would be examined every 2 years; and workers 50 years and older would be examined annually.

Periodic examinations must be performed on workers who require PPE to verify that the level of protection is adequate. The examining healthcare provider is responsible for coordinating with the personnel who characterize and document exposure profiles so that appropriate and timely periodic examinations are provided to all appropriate workers.

The need for more extensive surveillance increases in situations when

- more toxic or hazardous pesticides are used,
- medical examinations indicate that more frequent monitoring is necessary, or
- a health- or safety-hazard evaluation reveals that potential pesticide exposures could affect workers' health.

If the medical surveillance program is extended because of potential—or actual—unfavorable working conditions, the extension should be considered as an interim measure until more-effective controls are installed.

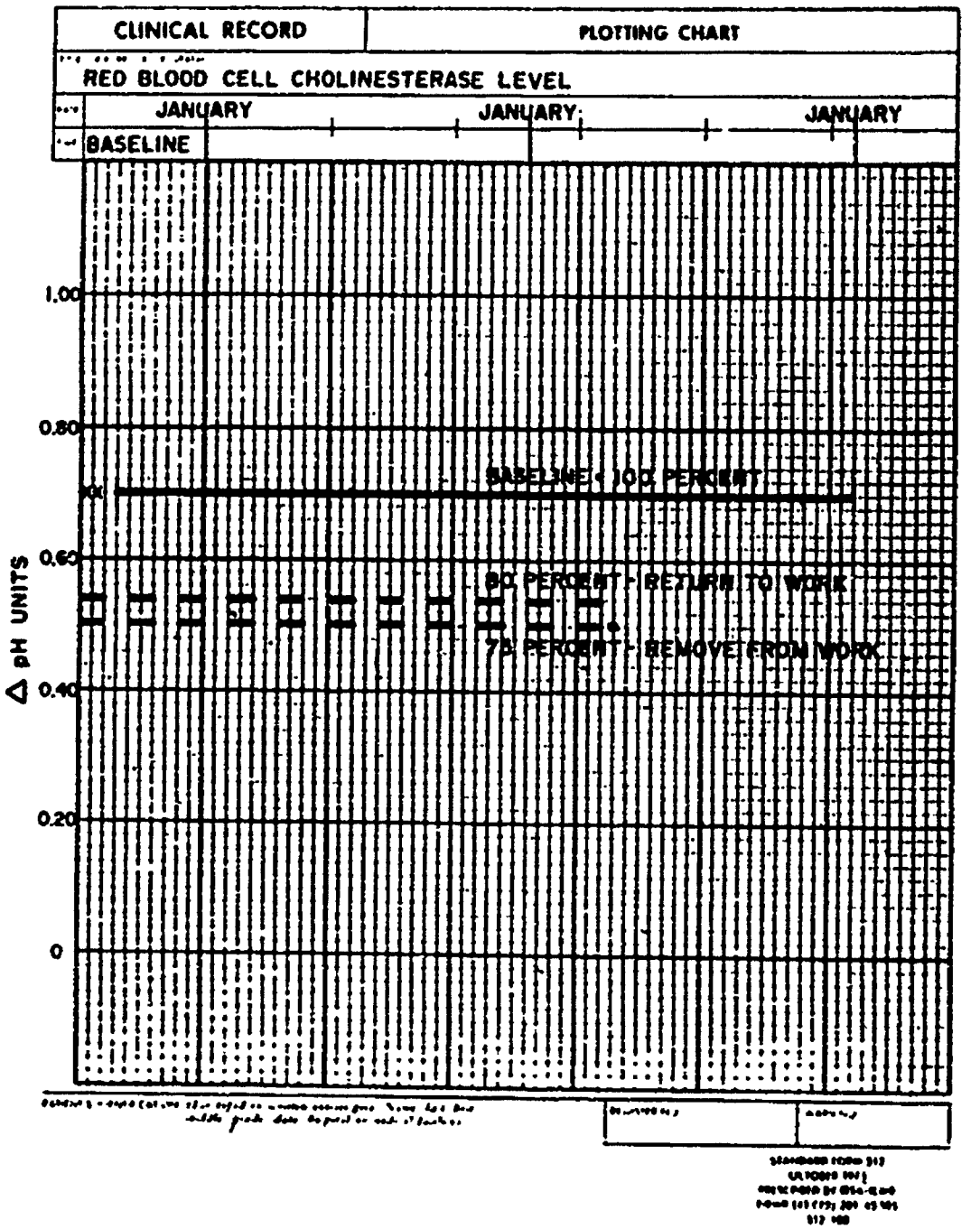


Fig. 14-8. The Clinical Record-Plotting Chart (SF 512) is an approved form for entry in a medical treatment facility's employee outpatient medical record. The graphic record should indicate an *action line*, (eg, a 20% depression from baseline values). When an employee approaches the action line, a set of patient-management practices, such as careful medical examination or return to work, may be implemented. A *medical removal line* should be drawn at the level of 25% depression. The change in pH (Δ pH units, the ordinate) is the difference in the pH of the erythrocyte environment before and after the addition of a buffer, as a measure of cholinesterase activity.

Regardless of the worker's age, a brief, focused interim review of history should be documented. A carefully elicited history of a general pesticide worker who has a wide potential for exposure, should, at a minimum, evaluate the organ systems mentioned in Table 14-11. Liver- and kidney-function tests and a complete blood count should be performed annually. If other occupational exposures exist (such as high noise levels from the vehicles or aircraft used for spraying pesticides), appropriate medical surveillance of the effects from these other occupational exposures should also be provided.

The frequency of periodic determinations of erythrocyte cholinesterase levels should be carefully planned for each individual, focusing on the potential for significant exposure to organophosphate insecticides or similar, militarily unique substances. If an emergency or a worker's breach of protection has caused an exposure, the results of the cholinesterase analysis should be charted immediately on the clinical graph and compared to the individual's baseline. In addition, a cholinesterase determination and comparison with the baseline are required when a symptomatic individual is evaluated.

Determinations of erythrocyte cholinesterase levels should be performed more frequently when workers are required to handle, store, mix, or use organophosphate insecticides. For example, if frequent applications are required during the summer, monthly determinations of the enzyme level should be documented as a prudent medical practice. In winter, if no potential exists for exposure, no determination of cholinesterase levels would be required.

The results of periodic examinations should be negative. If deviations from the normal baseline occur for occupationally related reasons, more frequent or more extensive examinations may be indicated. At the same time, an investigation should be initiated into the cause of the deviation, with specific attention directed toward engineering controls, PPE, and work practices.

Pretermination Examination

For employees who have been associated with pesticide use, pretermination examinations should be performed within 30 days of the termination of their employment. And as was previously discussed, the employee's total employment health history—obtained

TABLE 14-11

HISTORY AND PHYSICAL EXAMINATION FOR PESTICIDE WORKERS

Evaluation Categories	Areas of Emphasis
General History	Appetite, unexplained weight change, fatigue, work-site exposure potential
Visual	Acuity, need for prescription inserts, dimness and blurring of vision, unilateral or bilateral miosis, pressure, chemosis, allergic conjunctivitis
Respiratory	Rhinorrhea, breathing difficulty, cough, tightness of chest, bronchoconstriction, increased bronchial secretions, wheezing asthma, recurrent respiratory allergies, respirator wear (use test), claustrophobia, pulmonary-function testing, if needed
Cardiovascular	History of cardiovascular difficulty, atrial or ventricular arrhythmia, fainting, evidence of cardiac susceptibility, blood pressure history, family history
Gastrointestinal	History of ulcer or chronic bowel disease, neurological assessment for sphincter tone, history of incontinence or soiling
Cutaneous	Sweating disorders, heat tolerance, metabolic or genetic disorders, beard pattern, eczema, exfoliation, contact dermatitis, hematoma, easy bruising, petechiae
Genitourinary	Frequency, incontinence, history of renal disease
Musculoskeletal	Localized or generalized fasciculation, respiratory insufficiency (paralysis), weakness, cramps, twitching, strength, symmetry, family history
Nervous	Anxiety, giddiness, restlessness, depression, emotional lability, excessive dreaming, tremor, nightmares, confusion, headache, ataxia, apnea, convulsions, paresthesia, mental status exam, general neurological evaluation, affect, mood, memory, judgment
Hematological	Erythrocyte cholinesterase baseline (for organophosphates)

from preplacement, periodic, illness or injury, and pretermination examinations—must be retained for a minimum of 30 years.[3] All normal findings, together with any details of exposure and abnormal findings that could be ascribed to pesticide exposure, must be evaluated and documented before the employee's record is further disposed or retired.

Action Levels for Removal and Return-to-Work Policies

In general, any abnormal finding that could be related to pesticide exposure should cause the employee to be removed from further exposure until a complete evaluation is made with respect to the extent, cause, and significance of the finding. The employee's return to work should not be recommended if pesticide exposure could further harm the worker's health, *even if pesticide exposure did not cause the abnormality.* If the worker is found to have depressed levels of erythrocyte cholinesterase, which is fully reversible over time if no additional exposure occurs, the following policies should be adopted:

- The worker must be removed from work when the erythrocyte cholinesterase activity is depressed to 75% or less of its baseline value.
- The worker should be permitted to return to work when the erythrocyte cholinesterase activity has returned to 80% or more of its normal value, provided that this level is confirmed by a second test. In addition, the worker must be asymptomatic and have had no exposure to cholinesterase inhibitors for at least 1 week.
- The erythrocyte cholinesterase levels should not routinely be evaluated more frequently than once per week because the normal recovery rate is approximately 1% per day.

SUMMARY

Pesticides are used to prevent, destroy, or mitigate pests. To be effective, however, they must be applied into the pest's environment—the same environment shared by other animals, plants, and humans. To minimize adverse effects of pesticides to the environment or human health, the risks of applying a pesticide must be weighed against the benefits of its use. The inability to fully identify the risks assoeiated with introducing pesticides into the environment is a continuing problem.

Although in some instances the acute effects may be known, there is a paucity of information on chronic effects that result from long-term exposures to pesticide residues—not only to those who apply pesticides but also to bystanders. For this reason, the safe application of pesticides requires that precautions be taken to protect against acute or chronic exposures to the residues. Human exposures to pesticides during their application are minimized by using PPE and appropriate engineering controls.

The pharmacology of pesticides is not militarily unique. However, medical officers need to be familiar with the mechanisms of toxicity, their signs and symptoms of intoxication, and the recommended medical management practices, occupational exposure surveillance end-points, and long-term effects. Medical officers also need to be familiar with the numerous pesticide-related federal, state, local, and DoD regulations.

The inherent hazards to humans, during and after pesticide application, dictate that a comprehensive occupational medicine program be implemented. This includes monitoring the workplace for pesticide use and disposition. In addition, the program provides mechanisms to investigate alleged incidents of pesticide exposure.

REFERENCES

1. 40 CFR, Parts 152–180.

2. American Conference of Governmental Industrial Hygienists. *1990–1991 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices.* Cincinnati, Oh: ACGIH; 1990.

3. 29 CFR, Parts 1900–1910.

4. US Environmental Protection Agency. *Analysis of the Risks and Benefits of Seven Chemicals Used for Subterranean Termite Control.* Washington, DC: EPA; Nov 1983. Technical Report 540/99-83-005.

5. Smith AG. Chlorinated hydrocarbon insecticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press, Inc; 1991: Chap 15.

6. Gray AJ. Pyrethroid structure-toxicity relationships in mammals. *Neuro Toxicol*. 1985;6:127–138.

7. Ray DE. Pesticides derived from plants and other organisms. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 13.

8. Gasiewicz TA. Nitro compounds and related phenolic pesticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press 1991: Chap 18.

9. 40 CFR, Part 156 § 10.

10. Plestina R. *Prevention, Diagnosis and Treatment of Insecticide Poisoning*. Geneva, Switzerland: World Health Organization, Vector Biology and Control; 1984. WHO Document WHO/VBC 84.889.

11. Maddy KT, Edmiston S, Richmond D. Illness, injuries, and deaths from pesticide exposures in California 1949–1988. *Reviews of Environmental Contamination and Toxicology*. 1990;114:57–123.

12. Krieger R, Edmiston S. *Ranking of Pesticides According to the Number of Systemic Illnesses Related to Agricultural Pesticide Use in California 1982–1986*. California Department of Food and Agriculture, Division of Pest Management, Environmental Monitoring and Worker Health and Safety Branches, 1220 N Street, PO BOX 942871, Sacramento, California 94271-0001; n.d.

13. Litovitz TL, Schmitz BF, Bailey KM. 1989 Annual Report of the American Association of Poison Control Centers National Data Base System. *American Journal of Emergency Medicine*. 1990;8:394–442.

14. Rumack BH, Spoerke DG. POISINDEX Information Syst—. Denver, Colo: Micromedex Inc. CCIS CD-ROM Vol 68; edition exp May 1991.

15. Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991.

16. US Environmental Protection Agency. *Recognition and Management of Pesticide Poisonings*. Washington, DC: EPA; 1989. Technical Publication EPA 540/9-88-001.

17. Douville J, Licker MD, eds. *International Pest Control*. New York: Von Nostrand Reinhold; 1990.

18. Koelle GB. Anticholinesterase agents. In: Goodman LS, Gilman A, eds. *The Pharmacological Basis of Therapeutics*; 4th ed. New York: Macmillan; 1970: Chap 22.

19. Gallo MA, Lawryk NJ. Organic phosphorous pesticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 16.

20. Murphy SD. Toxic effects of pesticides. In: Klaassen CD, Amdur MO, Doull J, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 3rd ed. New York: Macmillan; 1986: Chap 18.

21. Hodgson E, Silver IS, Butler LE, Lawton MP, Levi PE. Metabolism. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press 1991: Chap 3.

22. Olajos EJ. Biochemical evaluation of organophosphorus-ester induced delayed neuropathy (OPIDN): The role of 2′,3′-cyclic nucleotide 3′-phosphohydrolase. *Neuro Toxicol*. 1987;8:211–222.

23. Ellenhorn MJ, Barceloux DG. Medical toxicology. In: *Pesticides*. New York: Elsevier Science Publishing; 1988: Chap 38.

24. Santone KS, Powis G. Mechanisms of and tests for injuries. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 4.

25. Roth A, Zellinger I, Arad M, Atsmon J. Organophosphates and the heart. *Chest*. 1993;103:576–582.

26. Baron RL. Carbamate insecticides. In: Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 17.

27. Clark JH, Sultan WE, Meyer HG, Lieske CN. *Studies of the Amplification of Carbaryl Toxicity by Various Oximes*. US Army Medical Research Institute of Chemical Defense; 1990. Technical Report USAMRICD-TR-90-13.

28. Woolley DE, Zimmer L, Dodge D, Swanson K. Effects of lindane-type insecticides in mammals: Unsolved problems. *Neuro Toxicol*. 1985;6:165–192.

29. Woolley DE. Application of neurophysiological techniques to toxicological problems: An overview. *Fundamental and Applied Toxicology*. 1985;5:1–8.

30. Matsumura F. Involvement of picrotoxin receptor in the action of cyclodiene insecticides. *Neuro Toxicol*. 1985;6:139–164.

31. Clarkson TW. Inorganic and organometal pesticides. In: WJ Hayes Jr, ER Laws Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 12.

32. Archibald SO, Winter CK. Pesticide residues and cancer risks *California Agriculture*. 1989;43(6):6–9.

33. Pelfrene AF. Synthetic organic rodenticides. In: WJ Hayes Jr, ER Laws Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 19.

34. Stevens JT, Sumner DD. Herbicides. In: WJ Hayes Jr, ER Laws Jr, eds. *Handbook of Pesticide Toxicology*. New York: Academic Press; 1991: Chap 20.

35. Wolfe WH, Michalek JE, Miner JC, et al. Health status of Air Force veterans occupationally exposed to herbicides in Viet Nam: I. Physical health. *JAMA*. 1990;264:1824–1831.

36. Emmett EA. Herbicides and pesticides. Critical reviews. In: Emmett EA, Brooks SM, Harris RL, Schenker MB, eds. *Yearbook of Occupational Medicine 1992*. St. Louis, Mo: Mosby=Year Book; 1992: 89–90.

37. US Department of the Army. *Design of Pest Management Facilities*. Washington, DC: DA; 1991. Military Handbook MIL-HDBK-1028/8A.

38. Armed Forces Pest Management Board. *Pesticide Fires: Prevention, Control and Cleanup*. Washington, DC: AFPMB; 1981. Technical Information Memorandum 16.

39. Armed Forces Pest Management Board. *Pest Management Operations in Medical Treatment Facilities*. Washington,DC: AFPMB; 1989. Technical Information Memorandum 20.

40. Americans with Disabilities Act. Pub L No. 101-336. 26 July 1990.

41 Watson AP, Munro NB. *Reentry Planning: The Technical Basis for Offsite Recovery Following Warfare Agent Contamination*. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1990 Technical Report ORNL-6628.

RECOMMENDED READING

Ecobichon DJ, Joy RM. *Pesticides and Neurological Diseases*. Boca Raton, Fla: CRC Press; 1982.

Hayes WJ Jr, Laws ER Jr, eds. *Handbook of Pesticide Toxicology*. , New York: Academic Press; 1991.

Morgan DP. *Recognition and Management of Pesticide Poisonings*. 4th ed. Washington, DC: Environmental Protection Agency; 1989. US EPA Report 540/9-88-001.

Ware GW. *Fundamentals of Pesticides—A Self-Instruction Guide*. Fresno, Calif: Thomson Publications; 1982.

Chapter 15

NONIONIZING RADIATION

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INTRODUCTION

CHARACTERISTICS OF ELECTROMAGNETIC RADIATION
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- Interactions with Matter

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- Microwave Radar
- Radar During World War II
- Physical Parameters That Determine Energy Transfer
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LASERS
- Specific Properties of Lasers
- Direct Biological Effects
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- Military Applications
- Laser Protection Standards
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MEDICAL SURVEILLANCE

SUMMARY

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INTRODUCTION

The electromagnetic (EM) radiation spectrum has been divided into somewhat arbitrary frequency regions (Figure 15-1). The spectral divisions are usually based on the radiation's originating process and the manner in which this radiation interacts with matter. The most useful divisions are between ionizing radiation (X rays, gamma rays, and cosmic rays) and nonionizing radiation (ultraviolet [UV] radiation, visible light radiation, infrared [IR] radiation, and radio-frequency [RF] waves). The division between ionizing and nonionizing radiation is generally accepted to be at wavelengths (λ) around 1 nm, in the far-UV region.

Ionization of matter occurs when an electron that is orbiting a stable atom is expelled. Atoms of all elements can become ionized, but only gamma rays, X rays, alpha particles, and beta particles have enough energy to create ions. Because ions are charged particles, they are chemically more active than their electrically neutral forms. Chemical changes that occur in biological systems may be cumulative and can be detrimental or even fatal.

Another obvious division occurs at wavelengths of approximately 1 mm between the optical and RF radiation regions. These can be further divided into narrower bands ad infinitum. The radiation produced in this portion of the spectrum, however, does not possess energy sufficient to ionize matter. This nonionizing radiation excites atoms by raising their outer electrons to higher orbitals, a process that may store energy, produce heat, or cause chemical reactions (photochemistry). The biological effects of nonionizing EM radiation are caused by thermal stress (the accumulation of heat). When heat is dissipated, the effects do not persist (they are not cumulative). When the thermal stress is extreme, however, persisting injuries such as erythema, cataracts, or burns can occur. These are not minor injuries: for RF radiation, the burn can be internal and life threatening; for laser systems, the injuries occur to the eye.

CHARACTERISTICS OF ELECTROMAGNETIC RADIATION

Two complementary concepts have been used to describe EM radiation: the wave model and the particle model.[1] Certain EM phenomena are easier to conceptualize with the wave model, and others are easier to conceptualize with the particle model. The wave model characterizes EM radiation as *the propagation of energy through transverse oscillations of the electric and magnetic fields.* These EM waves are measured by

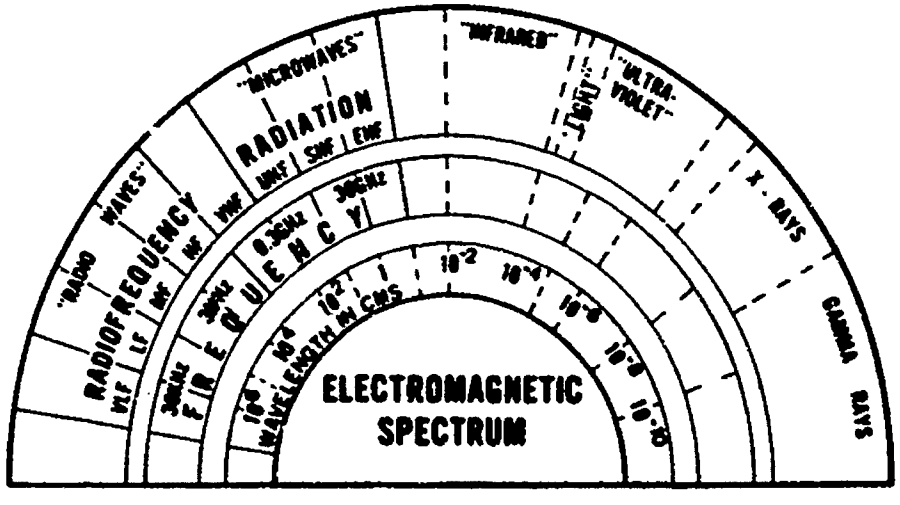

Fig. 15-1. The divisions of the electromagnetic spectrum are arbitrary, overlapping regions on a continuum. The generally accepted divisions are cosmic rays $\lambda < 0.005$ Å; gamma rays λ 0.005–1.4 Å; X rays λ 0.1–100 Å; ultraviolet (UV) λ 40–400 nm; visible light λ 400–700 nm; infrared (IR) λ 700 nm–1 mm; microwave (MW) λ 1 mm–1 m; radio-frequency (RF) λ >1 m.

four parameters: frequency, wavelength, polarization, and amplitude (field strength). The *frequency* of a wave is its number of oscillations per second as measured in hertz; the *wavelength* is the distance between successive waves, or the distance between the peaks. *Polarization* is the relative orientation of the EM radiation (horizontal, vertical, or circular). *Amplitude* is the absolute strength of the EM radiation as measured in volts per meter or amperes per meter. Frequency and wavelength are related to each other through wave *velocity* such that

$$c = \lambda \cdot f$$

where c represents the wave velocity, λ represents the wavelength, and f represents the frequency. The speed will change with different media but will never exceed the speed of light in a vacuum ($3.0 \cdot 10^8$ m/s).[1]

The particle model proposes that EM radiation consists of entities called *photons*, which can possess only discrete amounts of energy (*quanta*). Photons can only exist in motion, which, for them, can only mean moving at the speed of light. Photons can interact with other particles, exchanging energy and momentum through elastic and inelastic collisions.

Higher-frequency EM radiation has higher energy. Planck's constant ($6.62 \cdot 10^{-34}$ J/sec) relates the actual energy value of a quantum to frequency in the equation

$$E = h \cdot f$$

where E represents the actual energy value, h represents Planck's constant, and f represents the frequency.

The particle model is useful in conceptualizing certain phenomena such as scatter, and is also used to describe the phenomenon of *stimulated emission* (a quantum mechanical phenomenon that results in the emission of two photons in the same direction with the same energy and spatial coherence).[1] This is the essence of the *laser* (light amplification by stimulated emission of radiation), which is a technology, not a type of radiation. However, current usage employs the term laser to refer both to the technology and to the highly collimated beam of nonionizing radiation that it produces.

The terms *energy* and *power* are not synonyms. Energy refers to the ability to do work, whereas power is the ability to do work per unit time.

The characteristics of EM radiation can be categorized as shared properties and distinctive interactions with matter. All types of EM radiation share certain properties that make them alike. However, when EM radiation interacts with matter, the differences become evident. For example, visible light can be absorbed by a thin sheet of black paper, but RF radiation passes through the paper essentially uninhibited.

Physical Properties

All types of EM radiation—ionizing and nonionizing—share the properties of (*a*) divergence, (*b*) interference, (*c*) coherence, and (*d*) polarization, and aside from having differing amounts of energy, they do not differ in their physical properties.

Divergence

The term *divergence* is used to describe how the radiation emitted from a source spreads out. It can be calculated using the formula

$$D = \frac{b - a}{r}$$

where D represents divergence, b represents the diameter of the beam at the point measured, a represents the diameter of the beam at the point of emission, and r represents the length of the ray in question (Figure 15-2).

Divergence is related to the diffraction limit, which is the degree to which nonionizing radiation interacts with matter. It is impossible for divergence to be less than the diffraction limit.[2] (With lasers, the divergence is almost equal to the diffraction limit.) Other factors that contribute to divergence include the size of the source, the means of radiation production, the geometry of the emitter's aperture, and the medium of propagation.

Isotropic and collimated radiation emitters exemplify two contrasting concepts. By definition, radiation from an isotropic emitter spreads out uniformly in all directions surrounding the source. The intensity of the radiation decreases with the square of the distance from the source: at triple the distance from the source, the intensity of the radiation decreases by a factor of 9 (Figure 15-3).[3]

Collimated radiation, however, has an asymmetrical or directional spatial pattern. The intensity of the radiation does not decrease with the square of the distance but gradually decreases with distance. Lasers are highly collimated sources of radiation. The light from automobile headlights is somewhat collimated; it has a larger divergence than that from a laser but a smaller divergence than that of an isotropic emitter (such as a tungsten light bulb).

Interference

The principle of *superpositioning* maintains that amplitudes of intersecting waves combine to produce a resultant wave (Figure 15-4). Therefore, the net effect of interference for two waves of the same frequency will be either *constructive* (the amplitude will increase)

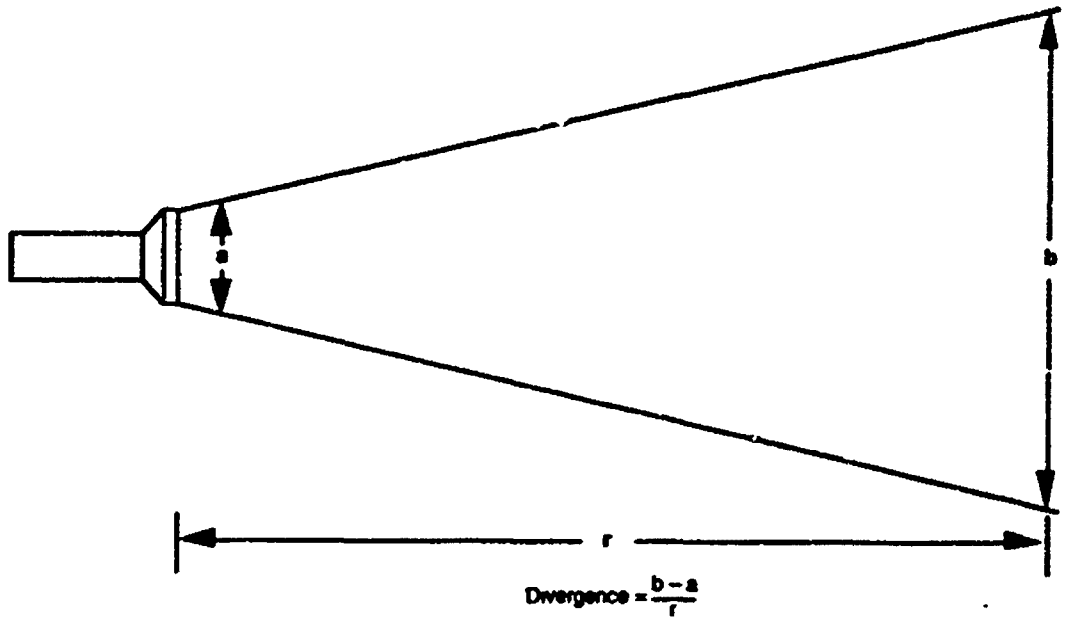

Fig. 15-2. Divergence can be measured as the change in the diameter of the beam divided by the distance of propagation.

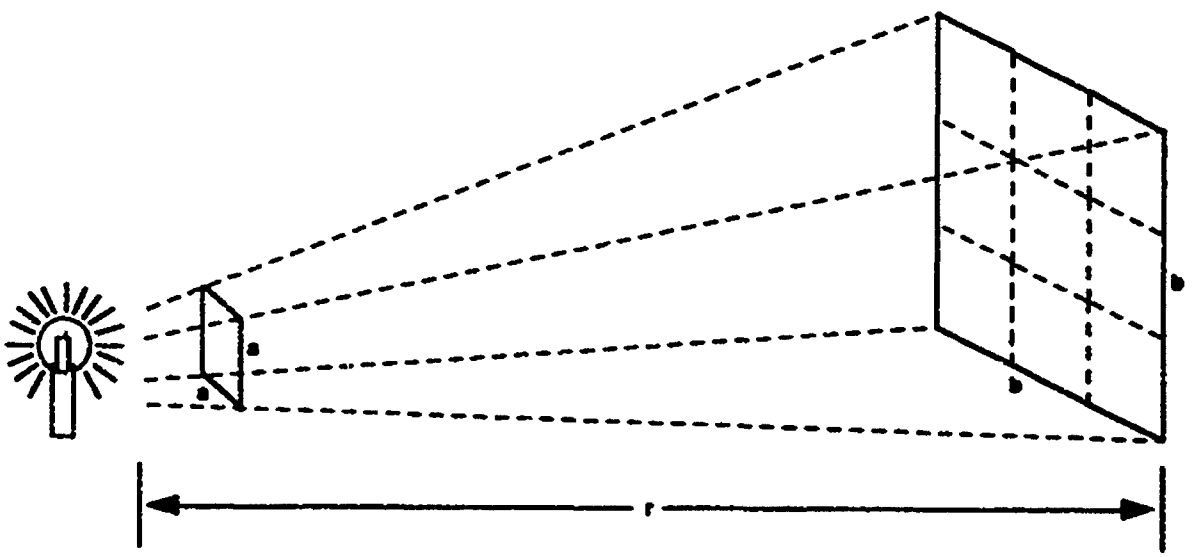

Fig. 15-3. When the distance from an isotropic em[illegible] increases by 3-fold, the light fills a 9-fold greater area. Inversely, the amount of light falling on a given area decreases by ⅑ (the Inverse Square law).

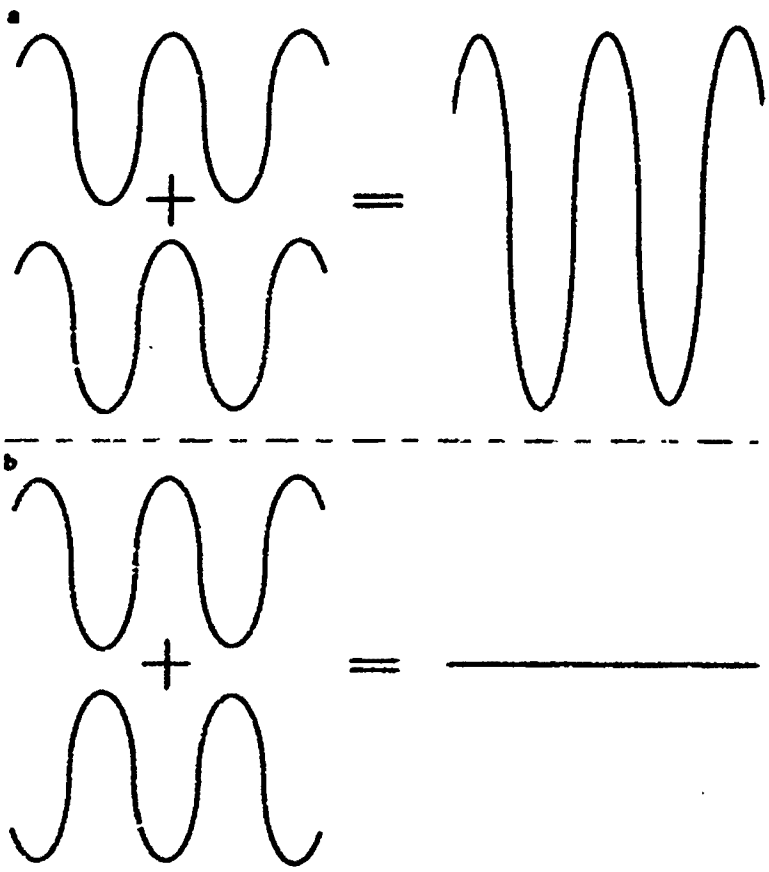

Fig. 15-4. Constructive and destructive interference. (a) Two waves that are in phase, in which peaks match with peaks, can be combined to produce a wave the amplitude of which is the sum of the peaks. (b) Two waves that are out of phase can nullify each other. Note that these waves are precisely in or out of phase. Waves only slightly out of phase will result in other types of waves.

or *destructive* (the amplitude will decrease). Constructive interference occurs when two waves of equal amplitude are in phase (their crests overlap); the result is a single wave with twice the amplitude. Destructive interference occurs when two waves are out of phase (a peak overlaps a trough) and their energies nullify each other. The interference phenomenon can be illustrated by illuminating two diffraction slits with spatially coherent light. The intersection of the two diffraction patterns produces alternating constructive and destructive interference bands.[1]

Coherence

The coherence of EM radiation implies organization and means literally "sticking together" with respect to phase. As the amplitude of the EM field varies cyclically, likewise so does the wave phase. In addition, EM radiation coherence may be either spatial or temporal; the two differ greatly (Figure 15-5). A temporally coherent radiation source is monochromatic and requires equal amounts of time for the phase peak of the radiation rays to pass. A spatially coherent radiation source emits rays with *like* phases all passing a point at the same time. The term *coherence* usually refers to spatial coherence.[4]

Polarization

The *orientation* of the electric and magnetic fields (ie, their polarization) affects the radiation's interaction with matter. Most natural sources of EM radiation do not exhibit a preferred orientation and are therefore unpolarized. However, EM radiation can gain a preferred direction of oscillation by reflection or by transmission through a material. For example, sunglasses with polar-

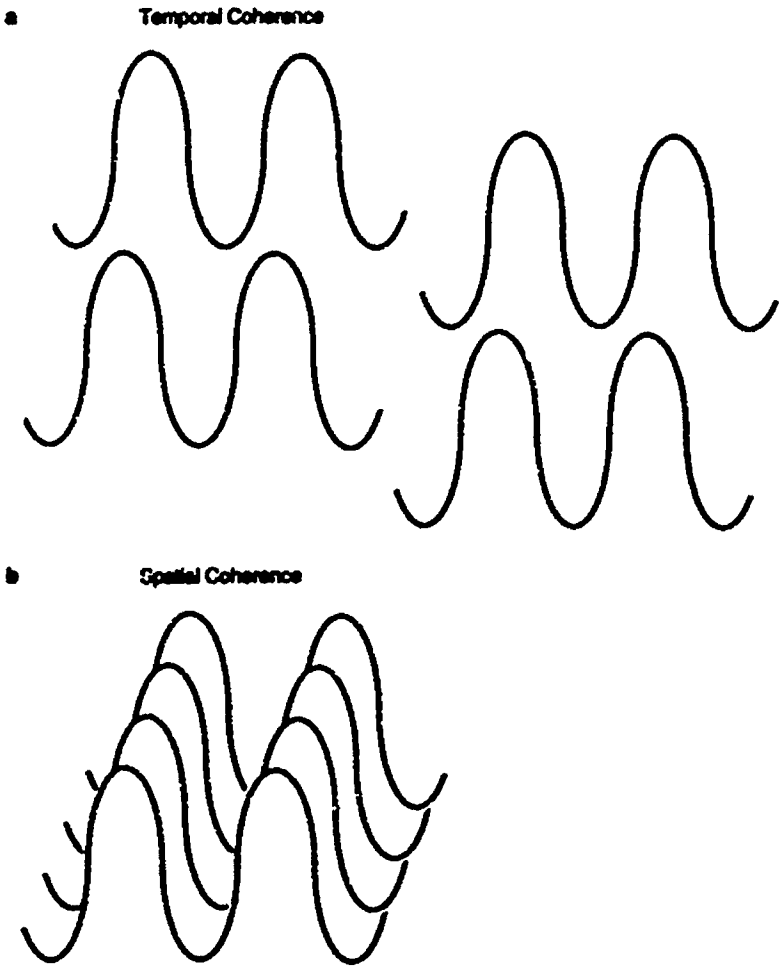

Fig. 15-5. (a) Temporally coherent waves have identical wavelengths, but they are not necessarily *in phase* (aligned). (b) Spatially coherent waves not only have the same wavelength, they are also all in phase.

izing filters prevent glare by blocking horizontally polarized light that reflects off surfaces such as car hoods.

Polarized radiation is classified according to its structure. *Linear* polarization, both horizontal and vertical, occurs when the electric and magnetic fields oscillate in a constant plane. In comparison, *elliptical* polarization (which includes circular polarization) occurs when the plane of oscillation rotates about the axis of the direction of propagation. Polarization can be modulated and thus can be used to transmit information. Elliptical polarization is commonly encountered in RF radiation work and is also important in the field of optics.[1]

Interactions with Matter

When EM radiation contacts matter, it interacts with the atoms in the medium and behaves in some respects like a particle and in some respects like a wave. The particlelike behaviors include scattering, reflection, and absorption. The wavelike behaviors include reflection, refraction, transmission, diffraction, and absorption. (Note that reflection and absorption are characteristics shared by both particles and waves.) The resulting effect of the radiation on matter depends on numerous factors including the wavelength components of the radiation, the sending medium, the receiving medium, the polarization components of the radiation, and the angle of incidence.

Reflection, Refraction, and Scatter

Reflection depends on the smoothness of the material's surface relative to the wavelength of the radiation. A rough surface will affect both the relative direction and the phase coherency of the reflected

wave. Thus, this characteristic determines both the amount of radiation that is reflected back to the first medium and the purity of the information that is preserved in the reflected wave (Figure 15-6). A reflected wave that maintains the geometrical organization of the incident radiation and produces a mirror image of the wave is called a specular reflection.[1]

The speed of EM radiation in any medium depends on (*a*) its wavelength and (*b*) the medium's physical properties, although it always will be slower than its speed in a vacuum (3.0×10^8 m/sec). The medium's index of refraction determines the speed of EM radiation through a specific material. If more than one material is involved in the passage of EM radiation, the propagation direction is subject to bending, which is called refraction. This is exemplified when light passes from a substance with one index of refraction (such as water) into another substance (such as air). Refraction is the property that enables a lens to form images by bending light.[1] When parallel rays of EM radiation from an object converge after passing through a lens, an image of the object is formed at the focal point. Prescription eyeglasses utilize this principle to aid the eye in focusing on an image at the retina rather than in front of or behind it.

However, if the reflection process fails, then the reflecting medium does not preserve the information to produce an image but scatters the radiation in all directions and destroys the image. When radiation passes through the medium, it loses coherence because of scattering. A reflecting medium that fails to produce an image is described as *diffuse*; a medium that loses an image during transmission is described as *translucent*.

The scattering mechanisms depend on the size of the particles composing the medium and the wavelength of the incident radiation.[1] The radiation exhib-

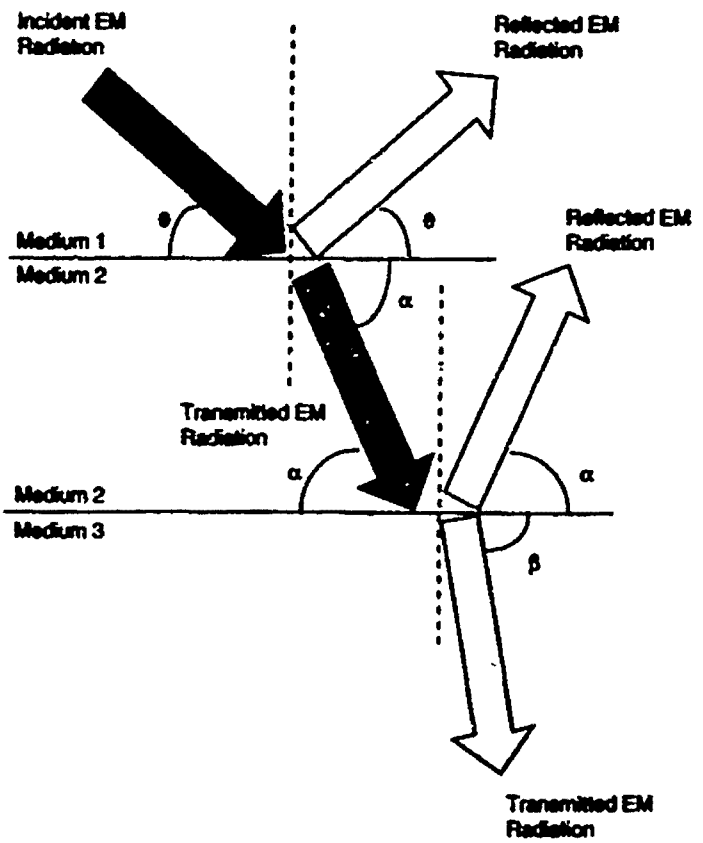

Fig. 15-6. When electromagnetic radiation encounters an interface between media (eg, between air and glass) the incident beam splits into reflected and transmitted beams. The angle of reflection (θ) is equal to the angle of incidence (θ) of the incident beam. The direction of the transmitted beam differs from that of the incident beam, a phenomenon known as refraction. The angle of refraction (α) of the transmitted beam depends on the physical properties of both media at their interface. Likewise, the energy transmitted through Medium 2, or reflected back from Medium 1, depends on both the physical properties of the media at their interface and the angle at which the incident beam strikes Medium 1.

its *Rayleigh scattering*, which is nondirectional when the size of the particles is on the order of the radiation's wavelength.[4] The diffusion by larger particles is called *Mie scattering*, which is not as wavelength-dependent as Rayleigh scattering. This scattering profile is dependent on particle size and can produce forward and backward scattering.

Transmission

The wavelength of the radiation greatly influences transmission and absorption because a given material can be transmissive at one wavelength and absorptive at another. For example, red glass transmits light with wavelengths near 650 nm; it absorbs the complementary color green, which has wavelengths near 550 nm. Transmission of radiation occurs when materials lack the properties necessary for absorption.

Absorption

Absorption is both particlelike and wavelike behavior. When EM radiation interacts with matter, it can be absorbed, transferring the energy of the radiation to the medium. For a particle, this interaction is an inelastic collision. For a wave, the wave energy is transferred from EM wave energy into energy modes in the absorbing medium. The absorption process is divided into categories that correspond to modes of molecular energy storage and include thermal, vibrational, rotational, and electronic modes (Figure 15-7). How energy is absorbed depends on the frequency of the radiation, the intensity of the beam, and the duration of exposure.

Thermal modes of energy storage consist of translational movement modes, in which atoms move horizontally and vertically about their lattice points in a medium, and which is commonly referred to as heat. Thermal absorption is common in the IR and other longer-wavelength spectrums. Vibrational energy modes consist of intramolecular vibrations between component atoms. Rotational energy modes consist of inertial energy stored in the orientation of spinning polarized molecules in local electrical fields that are found within some materials and can be stimulated by RF radiation. Electronic modes consist of the differ-

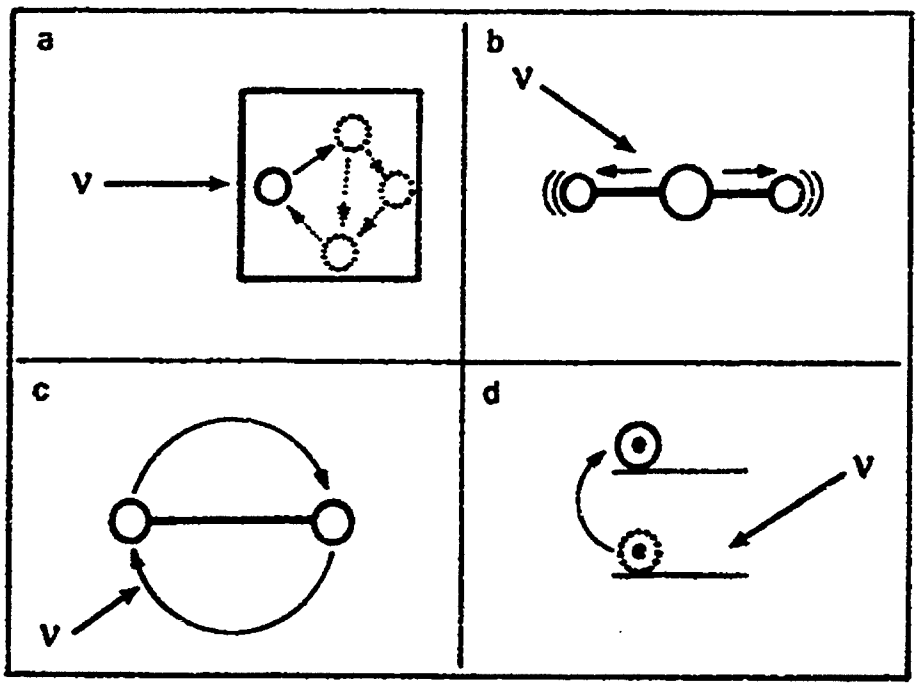

Fig. 15-7. Radiation interacts with matter and its energy is absorbed in several modes; in this drawing, v represents the incident radiation. (a) In the thermal mode, the radiation is absorbed by an atom, which then begins to move about its locus within the molecular lattice of the medium. (b) In the vibrational mode, radiation is absorbed and results in vibrations between neighboring atoms within a molecule. (c) In the rotational mode, energy is absorbed by polar molecules, which reorient themselves relative to local electric fields within the medium, storing energy. (d) In the electronic mode, electromagnetic radiation is absorbed by electrons, raising them to higher orbitals, storing energy that can be reradiated when the electrons return to their original energy state.

ent orbital energy states to which electrons can be excited, and these modes can produce new radiation energies as the excited electrons drop back to their original orbitals. Both electronic and vibrational modes can be stimulated by visible light and microwave EM radiation.

The amount of energy that a material will absorb from nonionizing radiation depends on (*a*) the frequency of the radiation, (*b*) the intensity of the beam, and (*c*) the duration of exposure. The most important of these parameters is frequency. Microwaves, IR radiation, and RF radiation can excite translational modes and generate heat. Microwaves are suspected to excite vibrational modes, and RF radiation excites rotational modes.

The intensity of the beam is also a factor in determining how much energy is absorbed. The greater the intensity of the beam, the more energy is available to be transferred. The duration of exposure is another factor. The longer the duration of exposure, the more energy that will be absorbed.

Diffraction

A phenomenon called diffraction allows EM radiation to bend, pass through small apertures, and move around small particles of matter. The smaller the aperture or particle, the more the light rays will bend This bending is quantitatively referred to as the diffraction limit. Because stars are so distant from earth, their light is almost pe. ectly collimated and the angle of subtense is infinitesimal. However, what we on earth see is actually a star's diffraction patterns, which occur when starlight passes through galactic dust.[1]

RADIO-FREQUENCY RADIATION

In 1864, James Clerk Maxwell proposed mathematically that energy can be transferred by electric and magnetic fields traveling together at a finite speed. It was not until 1886, however, that Heinrich Hertz experimentally proved Maxwell's theory of electromagnetism. To accomplish this, Hertz constructed the first oscillator-transmitter. This consisted of two metal spheres that were each connected to the end of a rod that had a spark gap in the center. The receiving antenna consisted of a loop with a tiny gap cut into it. With this equipment, Hertz conducted experiments that demonstrated the similarity between radio waves and light waves, and the polarization, refraction, and reflection of EM waves. Although Hertz's experiments were performed with relatively short wavelength radiation (50 and 450 megahertz [MHz]), later work in radio was performed at longer wavelengths.[5]

Radio Communication and Radar Technology

Guglielmo Marconi put the theories and experiments of Maxwell, Hertz, and others to practical use. In 1901, Marconi succeeded in establishing coherent wireless communications (using short-wavelength radiation) across the Atlantic Ocean. By 1907, regular commercial wireless service had been established between North America and Europe. Marconi not only predicted and successfully demonstrated radio communication between continents, he also recognized the potentialities of short-wavelength radiation for *radar* (*ra*dio *d*etecting *a*nd *r*anging), the radio detection of objects. However, he was unsuccessful in attracting support for this application. Marconi's suggestions did stimulate some experimental work at the Naval Research Laboratory, which resulted in the first radio detection of a wooden ship in 1922, and the first detection of an aircraft in 1930. By 1932, equipment operating at 33 MHz was capable of detecting the presence of an aircraft at distances of 50 miles. However, target-position information such as range and bearing could not be readily determined.[6]

From Marconi's primitive radar equipment, development efforts continued during the 1930s and 1940s. The U.S. Navy tested the first true radar, the XAF, aboard the battleship *New York* in 1939. This radar operated at 200 MHz with a range of 50 miles. By October of the same year, orders were placed for a manufactured version, the CXAM, and by 1941, 19 of these radars had been installed on major ships of the fleet. The army also conducted research in radar development. During the 1930s, the U.S. Army Signal Corps began efforts to develop radar, which intensified in 1936 when its first radar was tested. In 1938, the army introduced the first operational radar for aiming antiaircraft fire, the SCR-268. This radar was used in conjunction with searchlights because its angular accuracy was poor, although its range accuracy was superior to any optical methods in use at that time. The SCR-268 was the standard fire-control radar until early 1944, when the SCR-584 microwave radar replaced it. The SCR-584 was developed through work conducted at the Radiation Laboratory of the Massachusetts Institute of Technoiogy. In 1939, the U.S. Army developed the SCR-270, a long-range, early-warning radar. This

radar detected the first signs of the attack on Pearl Harbor in 1941, but these were ignored until after the bombing began.[7-9]

Microwave Radar

The first efforts to develop radar that could operate at microwave frequencies were initiated in 1936, when two papers were published that discussed replacing conventional transmission lines with waveguide—metal tubing—to operate radar systems at microwave frequencies.[10] Second, a successful cavity magnetron was developed in Great Britain in 1940, and for the first time it was possible to generate substantial amounts of power at microwave frequencies. During that year, prototypes of the cavity magnetron were transported to the United States, and the Radiation Laboratory at the Massachusetts Institute of Technology began research and development efforts in the microwave field. Much of this early work was directed toward the design of airborne microwave radar equipment because the microwave frequencies permit relatively small antenna structures. The term radar was first applied to a specific type of microwave equipment that was used to "see" electronically by means of a transmitted radio wave that reflected from the object that was "seen" (ie, a receiver detected the reflection and translated it to indicate an object's range, azimuth, and elevation). Microwave radar equipment revolutionized the existing very high frequency (VHF) equipment. By using the shorter-wavelength microwave spectrum, the newer radar devices not only could be made smaller but also could have greater range and versatility.[9]

Radar During World War II

The value of radar was not recognized in the civilian sector during the early years of World War II because its development was a successfully guarded secret of war preparation. Radar equipment was installed on battleships, submarines, and in airplanes—often against the wishes of the commanding officers. One of the first and best-known uses of radar in naval warfare occurred off the coast of Greenland in May 1941, in an engagement fought by the German battleship *Bismarck* and the cruiser *Prinz Eugen* against the British battlecruiser *Hood* and the battleship *Prince of Wales*. The commanding officer of the *Hood* preferred an optical rangefinder's readings to that of his newly installed radar (λ = 0.5 m) because the radar was unable to measure distances with the accuracy required for the main caliber guns to score hits. Nevertheless, the *Hood*'s companion ship, the *Prince of Wales*, did use an air-warning radar and scored three hits on the *Bismarck* after the *Hood* had been sunk.[11] Germany had developed a naval radar in 1939 (λ = 0.8 m), but it was used primarily for target search and had only limited use for fire control since it could not provide target course and speed for accurate fire-control plot. Therefore (and indicative of the primitive state of radar development), the *Bismarck* did

> ... not use her radar for rangefinding; it was the stereoscopic rangefinders, with their ability to measure accurately great distances in conditions of adequate visibility, that had allowed the quick destruction of the *Hood*.[12]

Although the radar used in the Battle of Britain operated at VHF, it still provided accurate range and tracking data, and the ability to function in spite of fog, clouds, and darkness reduced the threat from Hitler's bombers. The introduction of microwave techniques not only sharpened these abilities, but the reduced size and weight of the equipment and the extended applications of radar made improved radar a decisive factor in winning the war.

Until 1942, allied airborne antisubmarine radar, operating at a frequency of approximately 200 MHz, had neutralized the effectiveness of German submarines in the North Atlantic. At that time, many German submarines were equipped with listening receivers operating at the radar frequency. A German submarine with a directional antenna could now determine the direction of allied antisubmarine aircraft and estimate their range from the strength of the signal received. The effectiveness of allied antisubmarine aircraft decreased greatly because German submarines, warned of impending attack, dived before the aircraft were positioned to drop depth charges. However, allied aircraft countered the effect of the submarines' listening receivers by using an attenuator inserted between the radar transmitter and the transmitting antenna. During the final phase of attack, the radar operator would adjust the attenuator to reduce the radiated signal level. The operator of the listening receiver in the submarine would then note a decrease in the signal strength and conclude that the aircraft was moving away, when in fact the aircraft was approaching for attack.

When the allied forces introduced microwave radar, the German forces mistakenly believed that some sort of IR equipment operating on heat from the submarine had replaced the VHF radar. Because the German military made no attempt to develop microwave listening receivers, allied antisubmarine operations increased in effectiveness. By 1943, microwave equipment operating at a wavelength of approximately

10 cm replaced most of the VHF airborne radar. Germany's Grand Admiral Karl Dönitz said, "The enemy has rendered the U-boat war ineffective ... through his superiority ... in the modern battle weapon-detection (radar)."[7]

During the next 2 years, new types of radar were developed including airborne-targeting radar and ground-controlled approach (GCA) radar equipment. The airborne-targeting radar allowed airborne bombers to accurately locate targets on the ground in overcast conditions. GCA equipment permitted ground operators to direct an aircraft to a safe landing under zero visibility conditions. Neither of these two technologies had been practicable before the advent of microwave radar: the required antenna directivity was not possible using small VHF antennas.

Since World War II, microwave equipment has been used for various types of communication systems such as microwave relay installations that handle telegraph, telephone, or television signals. The wide microwave band affords significant data-handling capacity, offers great antenna directivity, and requires relatively low-power transmitting equipment.[13]

Physical Parameters That Determine Energy Transfer

RF energy is typically transferred to the body through conduction, coupling, and absorption mechanisms, which are dependent on both the length of the RF wavelength and the body's distance from the radiating source. Distances from the source in wavelengths and their corresponding mechanisms are

- 0 λ = *conduction* (contact),
- 0–0.2 λ = *coupling* (direct transfer of a charge), and
- > 0.2 λ = *absorption* (conversion to internal heat at frequencies > 1,000 MHz).

Conduction

Conduction occurs when the body makes contact with an RF source (eg, when an individual touches an antenna element or an exposed transmission line). The detrimental effects usually associated with conducted energy are electrical shock and burn. At frequencies above 100 kilohertz (kHz), most of the energy delivered through contact with an RF source will be absorbed within a few millimeters of the RF current's travel through the tissue. In this case, the specific absorption rates (SARs) involved may be significant if a small volume of tissue absorbs a large amount of energy.

Even if the induced current from RF conduction is not sufficient to create a thermal injury, it can stimulate the nervous system and cause a response similar to that invoked by an electrical shock. The individual may jerk involuntarily or reflexively, and the resulting movement could cause an injury to the victim or to someone nearby.[14]

The exposure limits (ELs) to control RF shocks and burns are intended to limit induced RF-current flow through the body for frequencies less than 100 MHz.[15] From 100 kHz to 100 MHz, the standard limits the RF current through each foot, and at the contact point to 100 mA. The current through both feet is limited to 200 mA. This limit is conservative, so even if this current enters the body through the smallest area of tissue, such as a fingertip, it would not be great enough to produce RF shock or burn. The RF current limits change with frequency below 100 kHz (ie, they decrease from 100 mA at 100 kHz to a lower limit of < 3 mA at 3 kHz). This decreased current limit is primarily due to the increased depth that RF current penetrates the body. At frequencies lower than approximately 3 kHz, the penetration is sufficient to interact with the CNS and other electrically sensitive organs such as the heart. At these frequencies, the biological effects associated with RF current flow in the body are clearly discernible and the physiological effects are well understood. They include (in addition to shock and thermal injury) electronarcosis, ventricular fibrillation, and involuntary movement. However, allegations of low-level effects have been made regarding the frequency region lower than 3 kHz, with the largest cluster of questions presently centered at the 60 Hz power-line frequency.[15-17]

Coupling

An individual can be exposed to the stored energy fields—at frequencies lower than 1,000 MHz—that are present close to element antennas or transmission lines. The body will absorb this energy through capacitive or inductive *coupling*, which is the direct transfer of a charge from one conductor (an antenna) to another (the body). Physical contact with the source need not occur, but inductive coupling can occur only if the second conductor is within 0.1 to 0.2 λ of the antenna or transmission line. At distances greater than 0.1 to 0.2 λ from the source, the radiation fields dominate. The actual exposure levels and SARs for coupled fields are difficult to predict and measure. Therefore, most cases of suspected RF radiation overexposure due to coupling must be investigated using dosimetric measurements on tissue-equivalent models.[18]

The type of energy-transfer mechanism involved in a suspected overexposure does not affect the resulting biological effects. Likewise, the exposure standards designed to prevent thermal insult will remain constant throughout the low-frequency region, with this exception: when a conducting object, such as the human body, is placed in an RF radiation field, the object will absorb 3- to 5-fold more RF energy due to coupling if the field is at the object's *resonant* frequency. Resonance occurs when an object's dimensions approximate one-half the wavelength of the incident energy. The human body standing in a vertically polarized field is resonant in the frequency band between 30 to 100 MHz. For example, a person 175 cm in height would be resonant at 85 MHz, where 175 cm is 0.5 λ. This resonance exists because the body attracts the RF current by acting as an antenna that appears to have an increased cross-sectional area. As body size decreases, the frequency for resonance increases (Figure 15-8).

The permissible exposure level (PEL) in the resonant frequency region for humans (30–300 MHz) is reduced 10% from the PEL in the nonresonant region. The SAR still remains 0.4 W/kg and the effects are still thermally induced. Only the PEL, which is derived from the SAR, changes.[19]

Absorption

The body will absorb RF radiation when it is located more than 0.2 λ away from the radiation source. Absorption is the principal mechanism of energy transfer for frequencies greater than 1,000 MHz. At lower frequencies, RF energy transfer occurs through a combination of radiation conduction, coupling, and absorption.

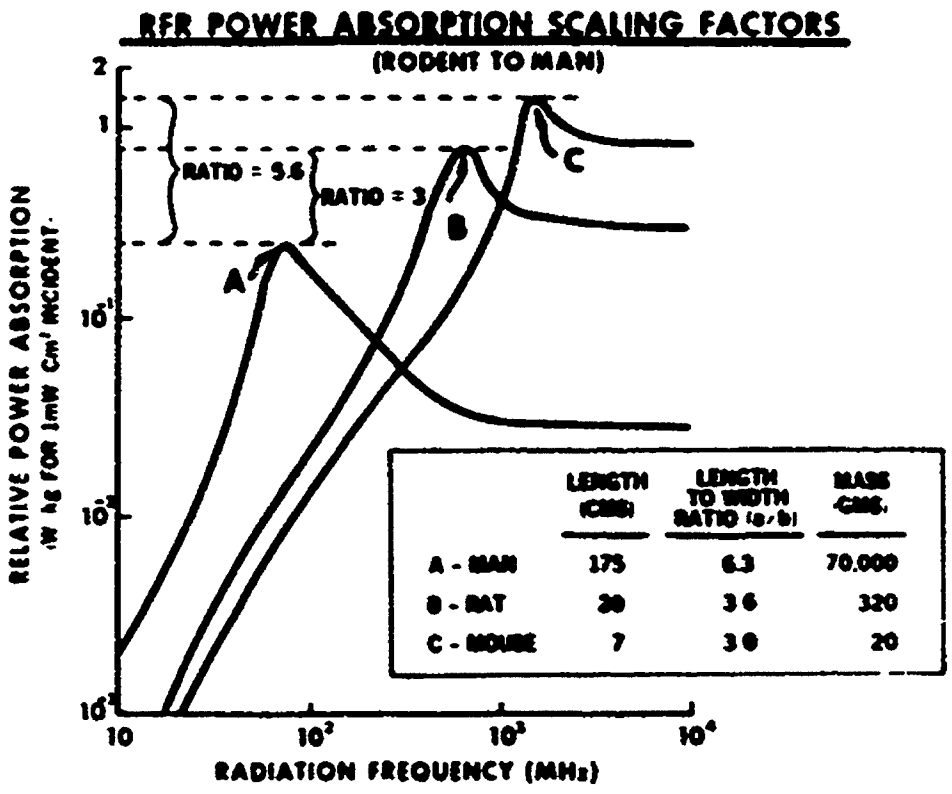

Fig. 15-8. The power absorption rate is related to the complex absorbing and scattering properties of the organism. The rate of absorption for mice, rats, and humans is associated with the length and mass of the animal such that the maximum absorption rate occurs at different frequencies. The absorption rate is independent of the intensity of the incident field and is a critical parameter for establishing exposure limits in humans. For humans, the maximum absorption rate occurs around 70 MHz. Reprinted with permission from DeLorge JO. Disruption of behavior in mammals of three different sizes exposed to microwaves: Extrapolation to larger mammals. In: Stuchly SS, ed. *Symposium on Electromagnetic Fields in Biological System*. Edmonton, Canada: International Microwave Power Institute; 1979: 215–228.

The PEL at frequencies greater than 1,000 MHz is based on a whole-body SAR of 0.4 W/kg. This SAR corresponds to absorbing 100% of the RF radiation energy incident on the body and equates to a measured power density of 10 mW/cm^2. For energy absorption at frequencies greater than 1,000 MHz, the biological effects are induced thermally.

The SAR threshold for thermal damage, even to the lens of the eye, is considered to be much greater than 4.0 W/kg. This SAR corresponds to absorbing 100% of the RF radiation energy incident on the body and equates to a power density of 100 mW/cm^2.

Direct Biological Effects

The absorption of energy is the key mechanism by which EM radiation affects living cells. Energy is transformed from electric and magnetic fields of radiation into one or more types of energy modes in the target material. When translational modes are excited, the ambient cell temperature rises due to the heat generated by these modes. If the temperature rise is sufficient, proteins denature and a burn results. In the case of lasers, the energy beam may be so intense that extreme heating can occur almost instantaneously, resulting in a superheated ionized gas (plasma). A secondary, mechanical effect of this ionized gas is a shock wave that is created as the gas expands. This shock wave and associated cavitation has been used surgically to disrupt certain fragile tissues such as the posterior capsule in the lens.

Depositing RF radiation energy into the body increases its overall thermal load. The thermoregulatory system responds to the increased thermal load by transfer of energy to the surrounding environment through convection, evaporation of body water, and radiation (primarily IR). When the RF radiation causes localized heating of certain organs, such as the eyes, prolonged exposure to this thermal stress can directly damage that organ. However, short duration exposure to RF-induced thermal load will usually not cause damage and the heat will be dissipated. For this reason, RF radiation exposure is not cumulative, unlike ionizing radiation exposure. The biological effects of RF radiation are thoroughly treated in textbooks such as *CRC Handbook of Biological Effects of Electromagnetic Fields*.[20,21]

Burns caused by exposure to nonionizing radiation exposure are different from conventional and electrical burns in that contiguous tissues are not necessarily affected: which tissue will be affected depends on the frequency of the incident EM radiation. For example, microwaves excite thermal modes in water molecules, and tissues with high water content such as skin and muscle are affected more severely than tissues with low water content such as fat. Therefore, microwave-induced burns tend to damage skin and muscle preferentially, and (relatively) spare the subcutaneous fat layer that separates these two structures. In addition, tissue interfaces such as organ capsules and fascial planes tend to be more susceptible to microwave damage.

Burns induced by nonionizing radiation also differ from electrical burns. For example, charring is minimal with electrical burns and is usually localized only to the site of entry. *Nuclear streaming* (the cellular nuclei align along the direction of current flow) is characteristic of electrical burns,[22] but is not a characteristic of radiation burns.

The lens of the eye is recognized as the most sensitive site for thermal damage. Studies on rabbits show that cataracts can be induced after repeated exposure to RF radiation in the FM frequency band (Figure 15-9).[23] However, the cataract-producing phenomenon could not be reproduced in a primate model; it may be a species-specific phenomenon. The nonionizing radiation absorption characteristics of the human eye are shown in Figure 15-10. (Although whether cataracts are induced in humans by chronic exposure to RF radiation remains an issue of debate, near-instantaneous cataract formation can be induced thermally by acute exposure to UV lasers with $\lambda = 365$ nm. UV lasers can also induce cataracts by a photochemical process, but this can take as long as 24 hours after exposure.[24] Laser injuries are discussed later in this chapter.)

EM radiation exciting electronic energy modes can cause significant biological effects by making photochemical reactions occur. Atoms do not need to be ionized to react chemically. Covalent chemical bonds can form when outer-shell electrons are excited to higher-energy states. For example, UV radiation creates thiamine dimers in skin tissue, a process that is associated with solar keratosis, premature aging, and skin cancer. Because sunlight contains UV radiation, sun exposure is a risk factor for skin cancer.

Low-Level Effects of Electromagnetic Radiation

Controversy surrounds the possibility that EM radiation at frequencies between 300 kHz and 300 gigahertz (GHz) may cause harmful biological changes in the absence of demonstrable thermal effects.[25] Thermal effects can affect all body tissue and are induced by measurable temperature increases in the affected tissue. These thermal effects occur at levels at least 10-fold greater than the officially mandated PELs. RF environments in the U.S. Army are relatively safe, but

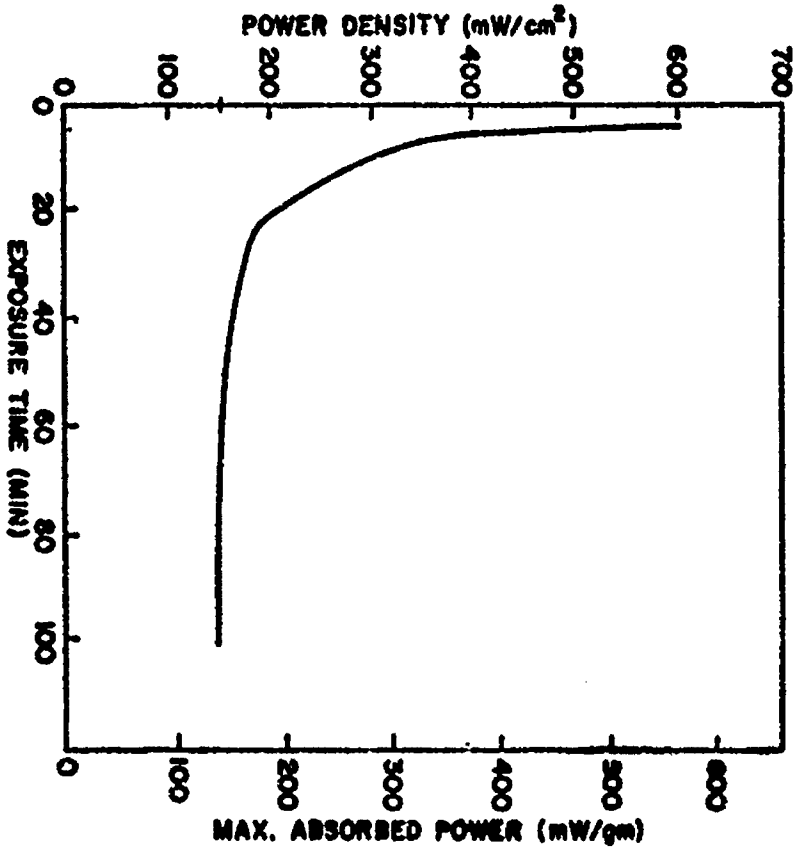

Fig. 15-9. Using 2450 MHz of radio-frequency radiation, the minimum power density required to induce a cataract in the eye of the rabbit is greater than 100 mW/cm² (the specific absorption rate is 4.0 W/kg). Reprinted with permission from Guy AW, et al. Effects of 2450 MHz radiation on the rabbit eye. *IEEE Transactions on Microwave Theory and Techniques*. T-MTT, 1975;23(6):495. © 1975 IEEE.

medical officers must realize that the effects can range from nuisance, to minor discomfort, to serious injury, to death. Serious effects such as extensive burns are obvious, but low-level effects may not be so clear cut.

Effects of low-level RF radiation, actual or alleged, involve temperature increases that are (*a*) too small to measure, (*b*) transient, or (*c*) are too localized to distinguish.[26] The significance of these effects on health is a subject much debated among scientists, public health officials, and various special-interest groups. The most common allegations of low-level effects encountered today involve frequencies lower than 3 kHz (the present lower limit of U.S. Army exposure standards). Low-level effects have also been alleged at frequencies above approximately 3 kHz. This includes low-level effects linked to use of video display terminals.[27]

Although thermal effects are the basis for current RF radiation exposure standards, scientific investigations during the 1980s have focused on effects of low-level exposure. Cancer, birth defects, behavioral changes, and other detrimental effects have been investigated using low-level RF energy below the current PELs. These investigations include epidemiological studies, animal studies, and other research efforts. No conclusive evidence substantiates claims of low-level RF radiation effects.[1]

Indirect Biological Effects

RF radiation indirectly poses a threat to health through electromagnetic interference (EMI) with electronic devices by disrupting their normal operation. EMI affects medical personnel by causing interference with sensitive healthcare devices (such as electrocardiograph equipment, operating-room monitors, or cardiac pacemakers), and affects military personnel by causing electronic weapons platforms (such as helicopters) to fail. Currently, the U.S. Army Medical Department (AMEDD) is only addressing concerns associated with the interference of medical devices, specifically from those EMI sources that the Department of Defense (DoD) produces and operates. This concern justifies the ongoing evaluation of the biological effects associated with the RF energy that these

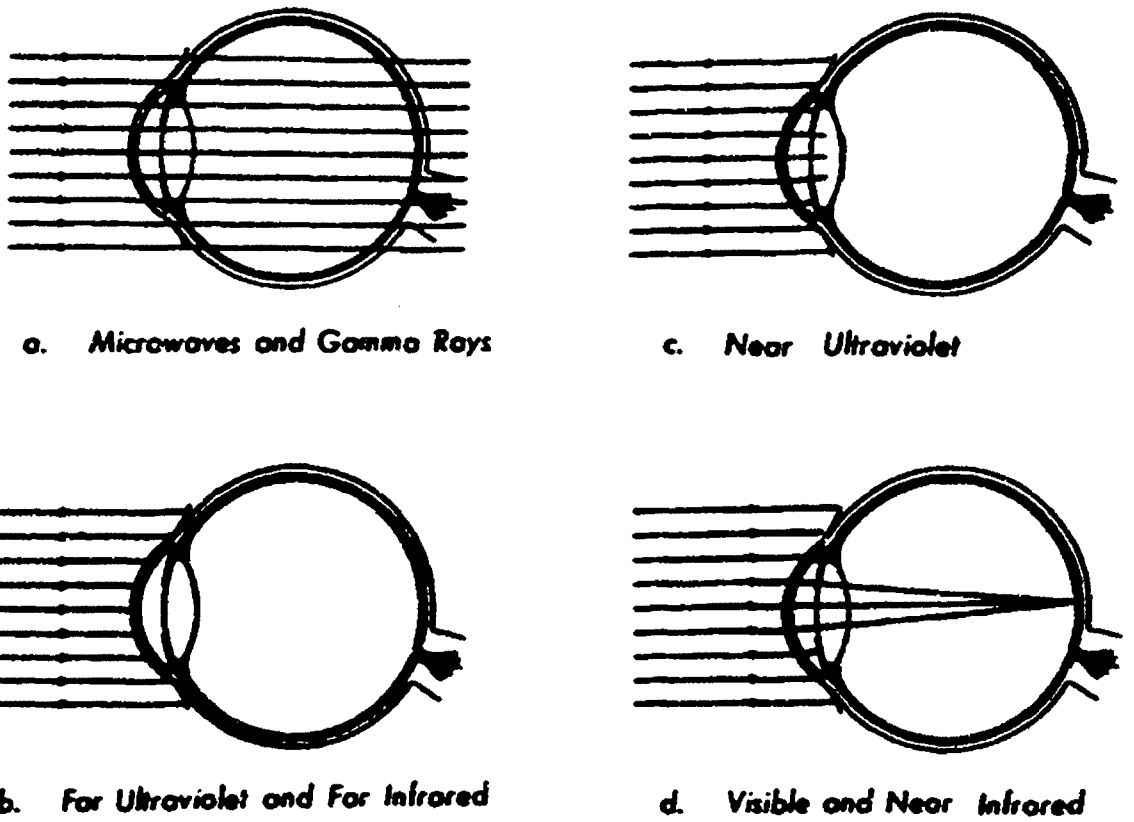

Fig. 15-10. (a) Microwave (MW) and gamma ray radiation can penetrate the human eye with some absorption in all tissues. (b) Far ultraviolet (UV) and far infrared (IR) do not penetrate the eye at all. (c) Near UV penetrates into the anterior portion of the eye, where it can damage the lens and posterior capsule. (d) Visible and near IR are focused by the lens onto the retina, where they can cause retinal damage. Adapted with permission from Sliney DH, Wolbarsht ML. *Safety with Lasers & Other Optical Sources*. New York: Plenum; 1980.

systems produce. These evaluations determine system characteristics such as *rise time* (the time required for the RF pulse to reach peak intensity), modulation, duration, and peak amplitude of the RF radiation.

Most manufacturers carefully design sensitive healthcare devices (that are intended to be used in a fixed setting such as a hospital) to operate satisfactorily in conventional RF environments. The U.S. Army Environmental Hygiene Agency (USAEHA) is tasked to study EMI problems with healthcare devices at army hospitals. The USAEHA maintains special measuring equipment capable of detecting the very low EMI threat levels. Typical EMI threat levels may be 7 to 10 orders of magnitude below the PEL associated with direct biological effects.

Implantable electronic devices such as cardiac pacemakers may be subject to EMI-related failure in certain RF radiation environments, such as near electromagnetic pulse (EMP) generators. Most implanted pacemakers provide a stimulating pulse to the heart only when the heart's own pacemaker fails to do so. The implanted pacemaker monitors the biological pacemaker, but the artificial pacemaker is sometimes susceptible to EMI. Fortunately, the device's shielding and the surrounding tissue act to reduce the threat of EMI.

During 1987 to 1989, a citizens group protested the use of an EMP generator that supposedly posed an EMI threat to implanted cardiac pacemakers.[28] The army and other military services used this generator to test the susceptibility of electronic weapons platforms to the effects of EMP. A thorough study of the alleged threa. was conducted in response to the protest. The results showed that EMPs can indeed interact with modern, implanted cardiac pacemakers. Individuals who work close to the EMP source (EMI test personnel) are at risk and require radiation protection controls; however, the EMP levels actually encountered by the general public will not produce EMI interactions.

Presently, although the army's RF environments pose no known uncontrolled EMI threats to pacemak-

ers or similar medical devices, pacemaker wearers are denied access to controlled areas that might pose an undetermined threat. Such devices and sources of EMI are continually evaluated through the U.S. Army Radiation Protection Control Program.[29-31]

Radio-Frequency Radiation Protection

Every installation or activity commander is responsible for ensuring that nonionizing radiation controls are implemented. The radiation protection officer (RPO), who is designated by the commander, usually manages the entire Radiation Protection Program including nonionizing radiation controls. Each installation also maintains a radiation control committee, which meets periodically to advise the commander on radiation hazards and methods for controlling them.

Radiation Controls

The PELs for RF radiation have changed dramatically over the past three decades. Until the mid-1960s, the maximum PEL for both the military and private industry was 10 mW/cm^2 regardless of the exposure duration. However, the increasing power output levels of new radar and other RF radiation sources, as well as certain tactical requirements of the military, made compliance difficult. Therefore, the military adopted new criteria that permitted higher exposure levels for determined lengths of time. Levels of exposure greater than 10 mW/cm^2 were permitted if the average exposure over a 6-minute period did not exceed 10 mW/cm^2. In 1974, the U.S. Army and the C-95.1 Committee of the American National Standards Institute (ANSI) adopted new standards that related the exposure level to the duration and were more frequency specific. ANSI Standard C95.1-1974 recommended a limit of 10 mW/cm^2 for frequencies lower than 10 MHz. Like the previous standard, ANSI Standard C95.1-1974 permitted exposure above the ELs, provided the average level did not exceed these limits for longer than 6 minutes. The U.S. Army further refined the standards in 1981 for frequencies lower than 10 MHz: a PEL of 66 mW/cm^2 averaged over a 6-minute period.

The DoD adopted the army's current PELs in April 1987, based on the recommendations of ANSI C95.1-1982 Standards Committee.[32] These standards encompass the frequency range 10 kHz to 300 GHz. They state that personnel should not be exposed to RF radiation fields that would cause a whole-body SAR of 0.4 W/kg. The threshold for biological effects is 4.0 W/kg. Thus, these control standards are based on levels of direct biological threats.

Derived equivalent PELs that correspond to exposures of 0.4 W/kg—which were determined theoretically and experimentally—were also specified in the 1987 standard for power density and levels of electric and magnetic field strength. They are further divided into areas restricted and nonrestricted for human occupancy to correspond to a whole-body SAR of 0.4 W/kg. These derived equivalent PELs are valid only under the conditions for which they were designed to be applicable.

Many RF radiation sources—most radars and some communications systems—can radiate bursts of energy rather than continuous energy. The pulses can contain power-density levels far in excess of the PEL. But the PEL is based on an average exposure over a 6-minute period. Therefore, it is possible to derive an equivalent average power density using the formula

$$Pd = \frac{6 \cdot PEL}{t}$$

where *Pd* represents the power density level under consideration and *t* represents the exposure duration measured in minutes. It is this derived PEL that is compared to the standard, not the maximum power density contained in a single pulse.

A new ANSI Standard C.95.1 was approved by the Institute of Electrical and Electronic Engineers (IEEE) in 1992. This new standard maintains the use of 0.4 W/kg SAR for calculating PELs, but adds limits for RF shock and burn. This standard will likely be adopted by the DoD in the near future. The DoD has considerable input to this standard-setting committee and has consistently adopted its recommendations. The limits are self-imposed by consensus agreement with the findings of the IEEE in the absence of federal regulations.

Attenuation of the Beam

The direction of maximum radiation of some RF radiation sources can be changed by physically turning the antenna or electronically steering the beam. A person standing near one of these systems would experience the maximum power density for only a fraction of the total time the system is transmitting. Again, a time-weighting factor can be used to derive the equivalent power-density level for comparison with the standard. However, regardless of the weighting factor, no individual should be exposed to an average power density level that exceeds the PEL by 5-fold (as averaged over 6 min). This is calculated by treating the scanning system as if it were radiating a fixed beam.

Threat Analysis and Evaluation

The evaluation of the exposure potential associated with RF radiation sources generally has several steps. First, the USAEHA determines if the system is capable of emitting field-intensity levels that exceed the PELs. Next, the USAEHA investigates the engineering controls that have been incorporated into the system to reduce the potential for exposure. Then, the degree of threat is determined by measuring the actual field intensities to which personnel may be exposed. To this end, the USAEHA is required to evaluate new RF radiation sources both to ascertain each system's threat and to recommend appropriate engineering modifications or system controls.

Emission of Radio-Frequency Radiation

Minimally, RF radiation emission requires (*a*) a generator or source, (*b*) a means to direct or guide the radiation, and (*c*) an antenna. Most practical systems for transmitting and receiving information require additional components that are relevant to the waveform, but this discussion will be limited to these three components of all RF emitters.

The Generator

RF radiation sources, or generators, convert electrical power into RF radiation using appropriate technologies such as oscillators or magnetrons. The radiation requirements of the system determine the type of generator or RF radiation source used. Some of the parameters are the power-output requirements, efficiency, size, bandwidth, frequency, and the modulation requirements. An oscillator is the most basic RF radiation source and consists of a tuned resonant circuit that is usually equipped with amplification stages and positive-feedback circuits. This basic RF radiation generator is often used as the input to other high-power amplifiers.[33] These amplifiers, such as the klystron and traveling wave tube, increase the power of an oscillator's output. Both amplifiers use a similar technique—injecting a beam of electrons into a vacuum tube—and use the input from the oscillator to alternately accelerate and decelerate the beam at the desired frequency. The oscillating beam of electrons is extracted via an electrical conductor.[34]

In comparison, a magnetron is a vacuum tube with resonant cavities. Its static magnetic field bends the electron beam from the cathode to the anode. The bent electron beam passes the resonant cavities and induces an alternating current at the desired frequency for radiation. These generators do not require an oscillating source or amplifier.[34]

The Transmission Line

Once RF radiation has been generated and information has been imparted to the signal through modulation stages, the next task is to guide the energy from the generator to an antenna. This can be accomplished with waveguide, coaxial cable, or wires. Waveguide is a long, hollow conductor, the dimensions of which can be designed to accommodate the transmission of any frequency. However, waveguide is impractical at frequencies lower than a few hundred MHz, and is usually used at frequencies of 3 GHz and higher. Coaxial cable, like that used for cable television, will transmit frequencies up to 3 GHz before significant transmission losses make it impractical. A colinear pair of wires will usually suffice for RF radiation up to 100 MHz.[35]

The Antenna

The final phase in transmitting RF radiation from a system is the *antenna*, a device used to make an efficient transition from a guided to a radiated wave. The complex design of an antenna, which has been the subject of many textbooks, will be influenced by such requirements as size, weight, frequency range, power output, directivity, propagation technique (such as troposcatter, line-of-sight, or ground wave), polarization, and electrical impedance.[3]

These requirements explain the wide variety of antenna designs available for different functions. In many respects, the characteristics of the antenna are the most important aspect of radiation hazard evaluation. The antenna will determine the direction and range of the radiation. A parabolic reflector antenna has the advantage of propagating RF radiation over long distances, but to achieve this, the energy is contained in a limited region. A monopole antenna will radiate equally in azimuth, but the RF radiation has a shorter range.

Military Applications

It should be understood that many military uses of RF radiation are not associated with weapon systems. For example, the military uses RF radiation for communication; target detection; imaging; electronic countermeasures; therapeutic medical diathermy; industrial heating, drying, and hardening of metals; and food preparation.

Radio communication is used to transmit voices and data between distant locations; radar technology is used to detect targets on the ground or in the air. The military also uses RF radiation sources offensively, as electronic countermeasures to interfere with hostile communication or detection devices. Although the military is experimenting with high-power sources as potential weapons, the main applications continue to be information gathering and broadcasting. Much of the hardware discussed below is known by a descriptive code, from which the user can determine the installation, type of equipment, and its purpose. For the most part, these codes will be used to identify the equipment and weapons systems.

Information Gathering

Ground-surveillance radar, lower-power radio, and air-defense radar are the most significant information-gathering uses of RF radiation.[36] Others, not discussed further in this chapter, include

- mine-detection devices;
- avionics radios and weather radars (which are also used in broadcasting);
- ground-mapping radars;
- navigational aids, transponders, and altimeters;
- missile tracking and guidance systems;
- object classification and identification;
- security systems and motion detectors;
- mortar source location;
- projectile tracking; and
- personnel location.

Ground Surveillance Radar. The AN/FPN-30 is a ground-control approach radar system that is used to assist aircraft landings when weather conditions and visibility are poor. It comprises an antenna group, a receiver-transmitter group, a control-indicator group, and a power-supply group. The AN/FPN-40 transmitter is able to produce average power of 180 W in a frequency range of 9.0 to 9.16 GHz. The PEL, which has the force of law and is established by the Occupational Health and Safety Administration (OSHA) for soldiers and civilian employees at this frequency range, is 10 mW/cm^2 for continuous exposure.

Two antennas can be used to obtain both azimuth and elevation information on aircraft positions. This information is obtained through the scanning or surveillance operation mode, or through the precision operation mode. In the scanning or surveillance operation mode, the system uses the azimuth antenna to search the horizon for aircraft. In this scanning mode, personnel around the system will not be exposed to RF radiation in excess of the PEL because the RF radiation can only be experienced for a fraction of the total time it is radiating. In the precision-operation mode, the system uses both antennas to provide accurate azimuth and elevation information during the final approach of aircraft attempting to land. In this mode, personnel located within 28 m may almost continuously experience the main beam from either antenna. Therefore, in the precision mode, a 28-m exclusion zone is observed immediately in front of the antennas.

Low-Power Radio. SINCGARS-V is a new family of VHF-FM (very high frequency-frequency modulation) radios for tactical communications designed for simple, quick operation using a push-button tuner. It is capable of voice, frequency shift key, or digital data communications. The frequency range is 30 to 88 MHz, with a nominal output power of 5 W and a maximum output power of 50 W when equipped with a power amplifier. The antennas used with these systems are typical low-gain element models such as a monopole whip, the same type used with citizens band (CB) radios. Physical contact with the antenna when it is equipped with a power amplifier may cause an RF radiation shock. Without the amplifier, the system is not subject to control procedures for RF radiation protection.

Air-Defense Radar. The purpose of the Hawk air-defense system is to acquire and engage hostile targets in the air. To this end, the system is equipped with several radars. Details of system performance and parameters are classified.

The pulsed acquisition radar (PAR) is one of the Hawk radars. The PAR provides information on the range and azimuth direction of acquired objects such as airplanes. Range information is obtained by transmitting pulses of RF radiation and measuring the time for one pulse to return or echo back to the PAR. The azimuth direction is known by observing the direction from which the echo is received.

The antenna is mechanically scanned in azimuth through 360 degrees. In this scanning mode, the RF radiation levels do not exceed the PEL for continuous exposure due to the short duration of exposure that personnel would experience during each rotation. However, the RF radiation levels in front of the system will exceed the PEL to a range of 17 m when the system is operated with the mechanical scan disabled for maintenance. More importantly, soldiers who perform these maintenance operations must never place themselves between the feed horn and the reflector of the antenna; extremely high levels of RF radiation are present at the feed horn.

Stabilotron and thyratron amplifier tubes generate high-power RF radiation. These tubes can produce X

rays, which must be shielded to preclude exposing soldiers who are working in the vicinity of these devices.

Broadcasting

In addition to high-power radio and satellite communications terminals, other broadcasting uses of RF radiation include electronic countermeasures, directed energy, avionic radios, and weather radars.

High-Power Radio. The AN/GRC-106 is a high-frequency (HF), single-sideband (SSB) radio set used primarily as a mobile link in a communications network. It may also be used in fixed and semifixed applications. The AN/GRC-106 has an amplitude modulation (AM) mode to make it compatible with standard AM radio sets. It is now being used as the basic radio set with all the newer radio teletype (RATT) configurations.

The RF power (400 W in the frequency range of 2–3 MHz) is fed to an element antenna. The RF radiation levels from this system will not exceed the PEL beyond 1.5 m from the antenna. More importantly, however, an RF shock or burn can be induced if an individual makes physical contact with the antenna while it is transmitting.

Satellite Communication Terminals. The AN/TSC-93 satellite communications (SATCOM) terminal contains receiver and transmitter equipment for voice, data, and teletypewriter communications via geosynchronous orbit satellites. The system is transportable for field operation with all but the antenna components contained in a standard military vehicle. The antenna is a transportable, high-gain, aperture model that is set up next to the vehicle.

The nominal power output is 500 W in the frequency range of 7 to 8 GHz, but typically this system is operated at a much lower power output (< 100 W). The maximum power density in the main beam of the antenna will exceed the PEL to a range of 110 m from the antenna when transmitting at full power. In normal operation, the antenna is set up on high ground, away from elevated structures and is directed toward the sky to acquire a satellite. This procedure will preclude overexposing personnel on the ground in the vicinity of the system.

Exposure Incidents

The severity of an alleged personnel overexposure to RF radiation determines the extent of the medical and technical investigations. When overexposure to RF radiation is suspected, the local medical department activity must examine the person involved and complete a *Special Telegraphic Report of Selected Condition* (Requirement Control Symbol [RCS] MED-16 [R4]), which is to be transmitted to the Office of The Surgeon General (OTSG), Preventive Medicine Division. This medical evaluation initiates a technical evaluation to analyze the following RF radiation parameters and incident data:

- operating frequency,
- antenna gain,
- average output power of the transmitter,
- transmission losses between the transmitter and the antenna,
- distance from the antenna to the location of the alleged overexposure, and
- duration of the exposure.

Another consideration that may influence the evaluation is the calibration of the measuring instrumentation. The state of the art of measuring technology does not permit measurements more accurate than ± 20%. Therefore, it is often necessary to calculate the measurement tolerance by calibrating the instrumentation. To maintain consistency, calibrations should be traceable to National Institutes of Science and Technology standards.

The initial technical evaluation permits the investigator to determine the maximum possible exposure level the individual could have encountered. However, an incident is not classified as an overexposure solely because the exposure level exceeded the PEL. For example, the power density may be averaged over a 6-minute period to determine if the exposure level exceeds the PEL by a factor of 5. A factor 10 for safety is integrated into the PEL. Army regulations do not allow actual exposures of more than 5-fold greater than the PEL over 6 minutes.

If the initial technical evaluation indicates that the person could have been exposed to RF radiation exceeding the PEL by a factor of 5, then (*a*) medical personnel will administer a complete medical evaluation but the only manifestation of overexposure that the evaluation will diagnose is thermal injury; (*b*) the USAEHA will conduct an investigation; and (*c*) an investigation report, which includes the following components, will be published: on-site interviews, evaluation of the radiation source, measurements at the incident cite (or at a similar site using the same equipment), consideration of other evidence to establish the extent of the overexposure, and recommendations for improving deficiencies in the Radiation Protection Program.

The following incidents of alleged exposures were selected to demonstrate the investigation process from

start to finish. Most exposure investigations do not proceed beyond the initial phase: determining if an overexposure could possibly have occurred. Usually investigations find that the field intensity was not *excessive* (5-fold greater than the PEL) or the duration of exposure was too short. In the first case study described below, an overexposure was determined not to have occurred, but this fact could only be determined after full investigation. In the second case study, an overexposure to RF radiation did occur and was investigated fully by the USAEHA. Interestingly, in the first case, RF radiation exposure was alleged to be responsible for an actual injury, whereas in the second case, no discernible injury was found.

Case Study 1

One RF radiation exposure incident involved a soldier working near a high power illuminator radar (HIPIR) unit that was tracking aircraft (Figure 15-11). The soldier was dismantling a tent 35 to 40 m from the HIPIR when he reported feeling localized heating on his back. As the soldier turned around to discern the heat source, he noticed the HIPIR directed toward him. Immediately, he entered a nearby equipment shelter, which put the HIPIR out of sight (thus making the soldier feel safe). The soldier soon began to notice sunburnlike reddening on the backs of his arms and on his upper back.

Although the soldier's commanding officers did not direct him to a medical facility, he later visited the installation hospital for treatment of the burned areas and the general uncomfortable feeling he was experiencing. The burned areas, described as superficial burns, were 1 cm^2 on the arm and 3 cm^2 on the upper back. There was no generalized erythema. All vital signs were normal and the soldier was released after being treated with Silvadine ointment (a 1% suspension of silver sulfadiazine in a hydrophilic base). The hospital created a MED-16 report of the occupational injury because RF radiation was a possible cause. (AR 40-400, *Patient Administration*, requires this report.)

[The actual medical records, which would have told exactly what physical signs were present, were not available for this case report. Medical officers might ask (*a*) if the burns were first, second, or third degree, or (*b*) if localized, rather than generalized, erythema were present. The use of Silvadine suggests that bullae characteristic of second-degree burns were present, but if so, the lack of erythema would be most unusual.—*Eds.*]

In addition to the initial medical examinations, the OTSG

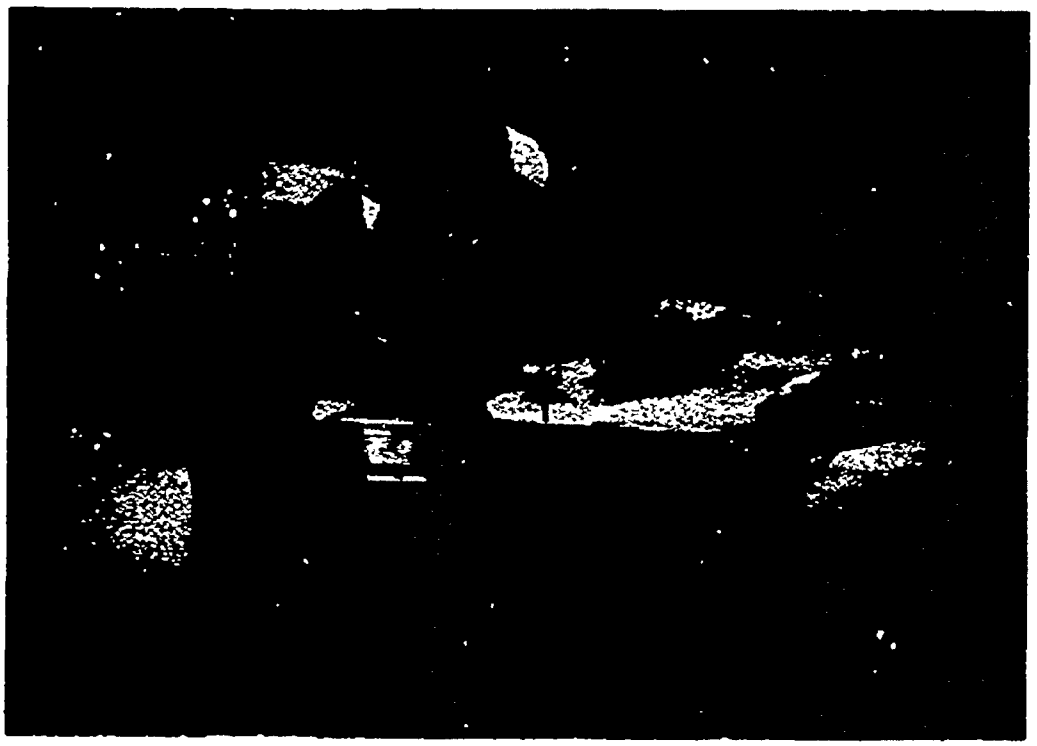

Fig. 15-11. The high-power illuminator radar (HIPIR) is an acquisition radar of the Hawk air-defense system.

requested that the USAEHA conduct an investigation of the circumstances surrounding the incident. This investigation, which was performed within several weeks of the incident included (*a*) a determination of the actual RF radiation levels to which the soldier may have been exposed, (*b*) the duration of any RF radiation exposure, and (*c*) the possible biological effects resulting from an RF radiation exposure. The initial evaluation was conducted via telephone interviews. The preliminary findings indicated that the maximum RF radiation levels that the soldier may have experienced were between 110 and 150 mW/cm^2. However, the actual exposure duration could not be determined. Because the RF radiation levels exceeded the PEL by more than 5-fold, the USAEHA initiated a full investigation of the incident, including a visit to the incident location to obtain the actual power-density measurements.

The investigation team who were dispatched to the incident site consisted of medical and engineering personnel. The team conducted extensive interviews with personnel associated with the operation of the HIPIR at the time of the incident, the soldier who allegedly was injured by radiation from the HIPIR, personnel who were working with the soldier at the time of the incident, and hospital personnel who examined the soldier. These interviews attempted to create an overall picture of the events surrounding the incident.

In addition, the team engineers conducted measurements of the actual RF radiation levels at 35 to 40 m from the HIPIR source. Figure 15-12 shows the relative positions of the HIPIR and the tent. The measured values agreed with those the initial evaluation predicted. The team also determined that the HIPIR was in a tracking mode at the time of the incident. When a HIPIR loses its target, the radar will coast (continue in the same direction with the same velocity) in an attempt to reacquire the target. If the HIPIR does not reacquire the target within a few seconds, the radar will return to its primary target (the direction is preset by the operator), which, in this instance, was away from the tent. It is likely that the soldier was exposed to the main beam of the HIPIR during this coasting function.

The investigation team determined that no RF radiation overexposure had occurred. The PEL in the frequency range of the HIPIR is 10 mW/cm^2 when averaged over 6 minutes. The duration of the exposure could not have exceeded a few seconds. Therefore, the soldier was not exposed to RF radiation in excess of the PEL. The burn may have been caused by another heat source such as the sun. The sensation of heat from the RF energy may have exacerbated the soldier's injury, but did not cause it. Nevertheless, unauthorized personnel will not be permitted within the RF radiation control range established for the HIPIR.

The soldier continued to report fatigue and chest pains. A series of examinations including liver function tests, blood counts, pulmonary function tests, and ophthalmic examinations were conducted over the next 6 months. All these examinations found no abnormalities. Later examinations included an electroencephalogram (EEG) and a computed tomography (CT) scan. These examinations also found no abnormalities. The attending internist recommended regular neurological evaluations, which also found no abnormalities.

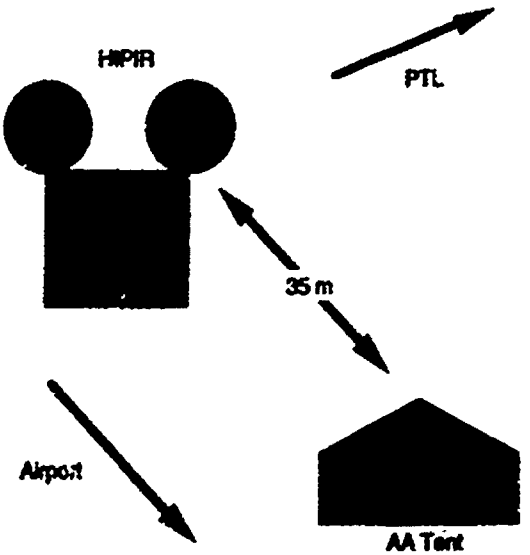

Fig. 15-12. During a training exercise, the operators of the high-power illuminator radar (HIPIR) were tracking aircraft near a local airport. The air assault (AA) tent was located between the airport and the HIPIR. After the radar had attempted to reacquire an aircraft that had descended below the line of sight, it automatically returned to its preassigned primary target line (PTL), away from the tent.

Case Study 2

Another RF radiation exposure incident involved two soldiers performing maintenance operations on a HIPIR. They were attempting to determine why the antenna's arc-detector crystal—located inside the transmitter housing—repeatedly burned out. While the radar was transmitting, the two soldiers removed the transmitter housing cover and visually examined the inside. After having examined the inside for 10 minutes, they noticed that a rigid waveguide was positioned incorrectly. They shut off the radar to remove the antenna pedestal head assembly. Upon further inspection after removing the assembly, they found the source of the repeated crystal burnout: a section of flexible waveguide that transfers high-power RF radiation to the antenna had been severed (Figure 15-13).

Within hours of discovering the severed waveguide, the two soldiers independently reported nausea and general malaise, and they reported the incident to the RPO. The RPO used an RF radiation meter to attempt to determine the actual power-density levels to which the soldiers may have been exposed. The measured levels exceeded the

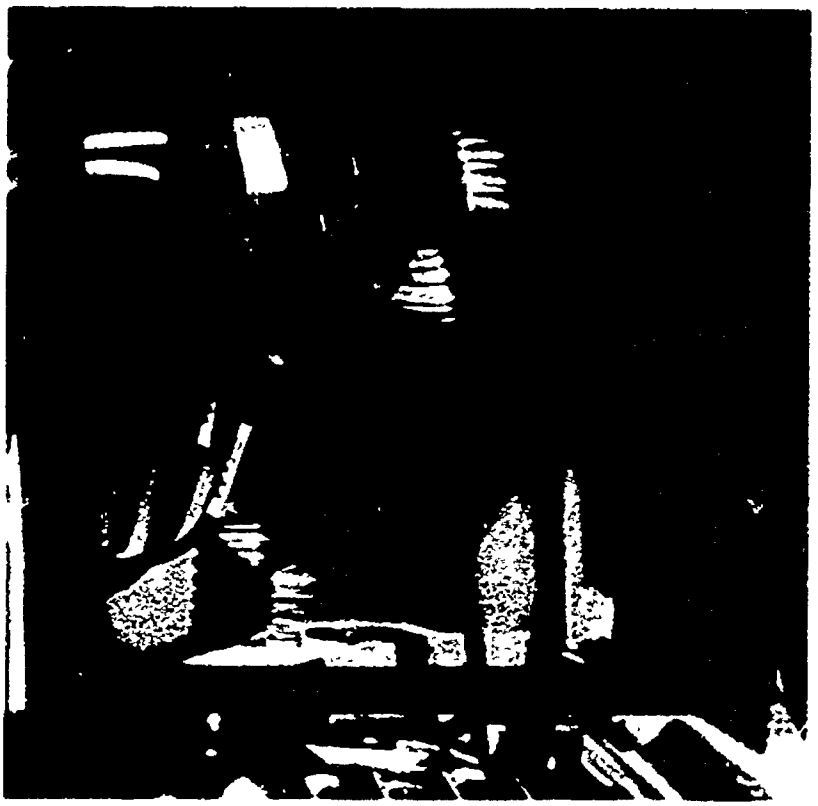

Fig. 15-13. There is no visible indication that RF radiation is being emitted by severed or cracked waveguide

100 mW/cm^2 limit of the meter, and therefore the RPO requested the services of the USAEHA.

On the day after the incident, before the USAEHA investigation team arrived, the soldiers received ophthalmic examinations. The examinations included tests for visual acuity, a slit-lamp evaluation of the crystalline lens, ophthalmoscopy, and an intraocular pressure test. The examinations revealed no abnormalities in either of the soldiers' eyes. The soldiers also reported that their initial nausea had subsided.

The USAEHA medical and engineering investigation team conducted an investigation. They interviewed the two soldiers, the RPO, and medical personnel at the installation. In addition, they measured the actual power-density levels in the regions where the soldiers were troubleshooting. The RF radiation levels ranged from 50 mW/cm^2 to 250 mW/cm^2. Assuming that at least the minimum exposure level was experienced for a maximum of 10 minutes, there is no question that the soldiers were exposed to RF radiation more than 5-fold greater than the PEL (10 mW/cm^2).

The primary recommendation of the investigation team was to test the HIPIR to determine why the flexible waveguide section had broken. (This was apparently not the first time the waveguide had broken in this manner.) The investigation team also recommended that maintenance personnel should always perform visual inspections around the transmitter housing with the power to the transmitter turned off.

The Radio-Frequency Radiation Protection Program

The installation or activity RPO is responsible for assuring that a comprehensive Nonionizing Radiation Protection Program (NIRPP) is implemented in accordance with U.S. Army regulations. The NIRPP for each installation or activity should include the specific systems, environments, and controls involved at the installation or activity. Most programs contain elements in common, including (*a*) inventories of RF radiation sources, (*b*) engineering controls, (*c*) administrative controls, (*d*) training programs, and (*e*) emergency procedures. No personal protective equipment (PPE) is used by DoD for RF radiation.[c]

Inventories of Radio-Frequency Radiation Sources

The installation RPO and the activities that operate the RF radiation sources must maintain inventories of all RF radiation sources (with their corresponding

threat classifications). From these inventories, the RPO can recommend appropriate engineering and administrative controls to curb exposure.

Engineering Controls

Any device or method that modifies the design, construction, or operation of the system to prevent undesired radiation is considered to be an engineering control. This includes safety interlocks that are incorporated into a system to prevent its operation under less-than-optimum conditions. Engineering controls restrict radiation levels and radiation zones through methods such as warning sirens, flashing warning lights, built-in dummy loads, azimuth- and elevation-limiting switches, couplers, and attenuators.

Administrative Controls

Physical barriers (such as fences, warning signs, lights, or alarms) that identify the radiation-control area at its perimeters and access routes are administrative controls. They are used to preclude individuals from RF radiation exposure. Administrative controls rely on the individual to ensure his or her own radiation protection. Standing operating procedures (SOPs) are developed to inform those who use the system about RF control procedures. Warning signs are placed around the device to further remind users of the potential for exposure.

Training Programs

The personnel who should receive radiation safety training include all those responsible for operating, maintaining, or repairing RF radiation sources that are capable of emitting levels at or exceeding the PEL. This training should be conducted when an individual is first employed and annually thereafter. The RPO should maintain a record containing a brief outline of the instructions for each training session and a list of individuals who received the training. Training sessions should include instruction concerning

- exposure potential associated with specific pieces of equipment,
- biological effects associated with overexposure to power density levels exceeding the PEL,
- proper use of protective equipment and devices such as barriers, signs, and lights,
- accident-reporting procedures,
- routine radiation-safety surveys, and
- procedures for maintaining an operational log for recording radiation-safety-related events (such as radiation-zone violations or overrides of warning signs or safety interlocks).

Emergency Procedures

Specific individuals—designated in writing—will be notified in the event of an emergency (such as equipment malfunctions or alleged overexposures) that involves RF radiation levels that possibly exceed the PEL. A list of those to be notified should be posted in control areas. The individual so designated will report the circumstances of the emergency to the installation or activity RPO, who will determine if an RF radiation exposure 5-fold greater than the PEL has occurred. If the exposure did exceed the PEL by 5-fold, the OTSG will request that the USAEHA conduct an on-site investigation.

The OTSG will also ensure that potentially exposed individuals receive an appropriate medical evaluation within 24 hours of the incident, and will develop and transmit an RCS-MED-16 report.[38] At a minimum, this examination will include an ocular examination, which consists of the following:

(1) Ocular history, with emphasis on previous eye injury or disease and medication use (especially any photosensitizing medications).

(2) Distance visual acuity (with correction) in each eye. If the corrected distance visual acuity is poorer than 20/20 in either eye, a refraction will be performed to obtain the best corrected acuity.

(3) Amsler grid or similar pattern will be used to test macular function for distortions and scotomas.

(4) A slitlamp examination of the lens and cornea and an ophthalmoscopic examination of the fundus, both with a rapidly acting, short-duration mydriatic (eg, tropicamide) unless the use of a dilating agent is contraindicated by medical history and/or professional judgement. The following, as a minimum, are to be recorded:

(*a*) Presence or absence of opacities in the media.
(*b*) Sharpness of the outline of the optic nerve head.
(*c*) Cup to disk ratio.
(*d*) Ratio of the size of the retinal arteries to retinal veins.
(*e*) Presence or absence of a well-defined macula.
(*f*) Presence or absence of a foveal reflex.
(*g*) Any retinal abnormality, however small or subtle.
(*h*) A color fundus photograph (the preferred method) that includes the optic nerve head and macula may be used in place of (*b*) through (*g*) above.[39]

LASERS

Laser technology developed in a sequence that integrated the findings of both theoretical and experimental investigators. When he stated his theory of the hydrogen atom in 1913, Niels Bohr first proposed his contentions that atoms could (*a*) exist in discrete energy states and (*b*) radiate light of well-defined frequencies in transitions between these energy states. In 1917, during the course of a theoretical investigation of blackbody radiation, Albert Einstein showed that a third process, stimulated emission of radiation, was necessary to account for the observed form of the blackbody's radiation spectrum.

The field of electronics and the extension of available sources of radiation in the RF region developed steadily during the first half of the 20th century. In the visible and IR regions, however, no similar extension occurred until 1958, when knowledge and technologies began to proliferate (Exhibit 15-1).

The current inventory of fielded laser systems in the United States military consists primarily of three types of lasers: (1) ruby, (2) neodymium:yttrium aluminum garnet (Nd:YAG), and (3) gallium arsenide (GaAs). Prototypes of military laser rangefinders were developed during the 1960s: a ruby laser mounted on the M551A1 Sheridan vehicle was fielded in the early 1970s, followed shortly by another type of ruby laser rangefinder that was fielded on the M60A2 missile-firing tank. During the 1980s, the Nd:YAG laser rangefinders and target designators were fielded in infantry, armor, artillery, and aviation units. The third type of laser system—the direct-fire simulator, which uses the luminescent diodes of GaAs—was fielded during the early 1980s. These were developed to provide more realistic training in the use of direct-fire weapons.

EXHIBIT 15-1

PROLIFERATION OF LASER TECHNOLOGY

Townes and Schawlow showed the feasibility of operating a maser (microwave amplification by stimulated emission of radiation) in the optical or near-IR region in 1958.

Schawlow proposed the use of ruby as a laser material in 1960.

Maiman reported the first operating laser using ruby as the medium in 1960.

Javan, Bennett, and Herriott successfully demonstrated an operational, continuous gas laser in 1961.

Keyes and Quist discovered highly efficient luminescent diodes of gallium arsenide (GaAs) in 1962.

Specific Properties of Lasers

Lasers are sources of nonionizing radiation that can operate in the IR, visible, and UV regions of the EM radiation spectrum. Laser technology utilizes three basic components: a lasing medium, a pumping system, and a resonant optical cavity. Although only these three components are necessary for lasing action, other components such as lenses, shutters, and mirrors can be added to the system to change the output.

The fundamental physical process in the lasing medium is the stimulated emission of photons (discrete bundles or particles of energy), which requires that (*a*) the particles of the medium be in an energetic state and capable of photonic emission, and (*b*) the emission must be stimulated (a photon interacts with an atom, triggering an energy-state collapse, which allows another photon to be released) (Figure 15-14). Stimulated emission differs from spontaneous emission, which permits the particle to collapse to a lower

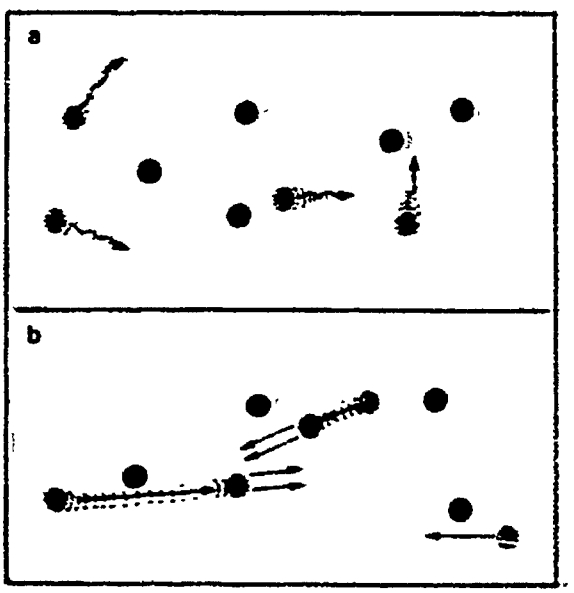

Fig. 15-14. (a) Spontaneous emission occurs when an excited system drops to a lower energy state and emits a photon. This process occurs by itself and the departing photon has no preferred direction. (b) Stimulated emission occurs after collision with a photon. The two emitted photons leave in exactly the same direction and with the same energy.

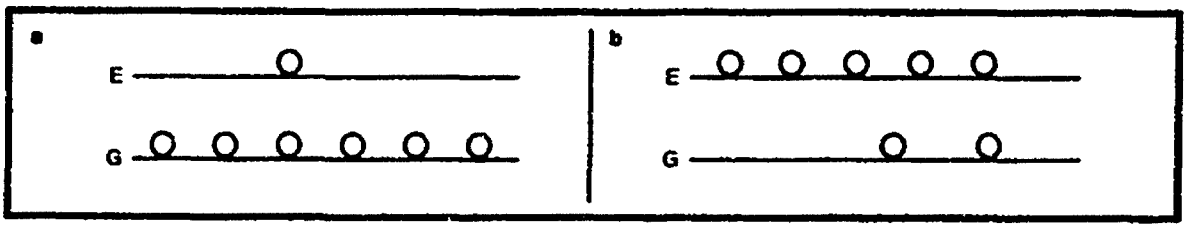

Fig. 15-15. (a) A normal electronic population distribution, where the ground state G is the most populous. (b) An electronic population inversion occurs when the excited state E becomes more populous than the ground state.

energy and emit a photon, but in a random direction. Laser operation requires that each of the photons resulting from stimulated emission amplify the radiation by stimulating two other photons, and so forth. This amplification depends on the preexistence of a condition called *population inversion* (more electrons exist in the excited than in the lower-energy state) (Figure 15-15).[40]

When matter absorbs energy, population inversion can be encouraged by special techniques such as *pumping*.[41] Several different systems are available. *Optical* pumping uses a strong source of light, such as a xenon flashtube or another laser (usually of a shorter wavelength). *Electron-collision* pumping is accomplished by passing an electric current through the lasing medium. *Chemical-reaction* pumping is based on the energy release that occurs during the formation and breakdown of chemical bonds.

A mirror at each end of the lasing medium forms a resonant optical cavity, which permits a beam of light to be reflected from one mirror to the other (Figure 15-16).[41] Lasers are designed so that as this light passes through the lasing medium many times, the number of emitted photons (stimulated emission) is amplified with each passage. One of these mirrors is only partially reflective, thus allowing part of the beam to leave the optical cavity. This is the laser beam.

The requirements for the construction of a laser are few and the means of achieving these requirements are numerous.[42] Many materials can act as laser media provided a proper stimulation system and a means of gain exist. The lasing medium can be composed of organic or inorganic materials in any of the four phases of matter (gas, liquid, solid, or plasma). It can be pumped into higher energy states through electrical discharge, flashlamp discharge, chemical reaction,

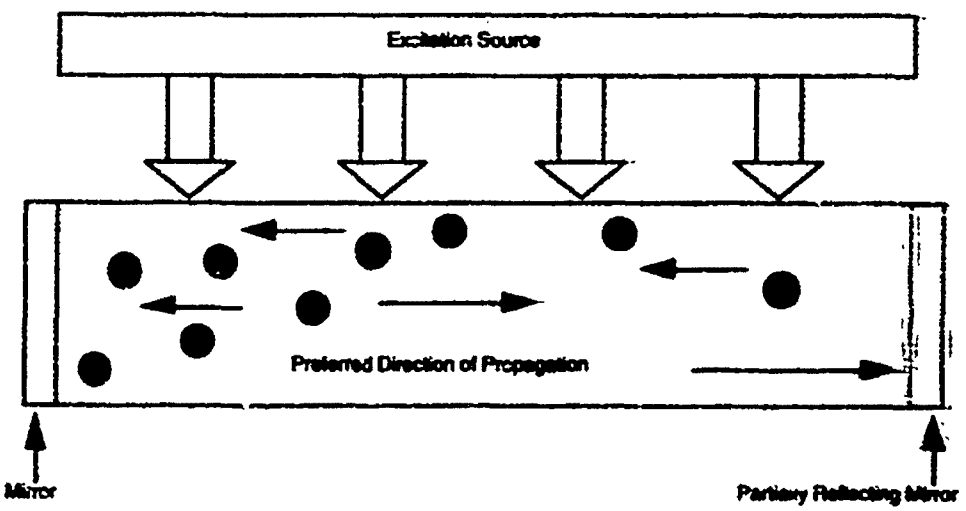

Fig. 15-16. The basic components of a common laser include a lasing medium (or cavity) with a preferred direction of propagation, an excitation source to create a population inversion, and mirrors to amplify the laser beam.

electron injection, and RF stimulation. Laser emission in some systems is fixed at certain wavelengths, while the output from other systems is tunable.

Direct Biological Effects

The biological effects associated with exposure to the laser-optical region of the EM radiation spectrum involve the skin and the retina, lens, cornea, and conjunctiva of the eye. The mechanism of injury for most effects is either photochemical or thermal; some authorities believe that these injury mechanisms are not understood fully.[42,43] Thermal effects predominate from the long-wavelength-end of the visible region, toward the microwave end of the spectrum. Photochemical effects usually predominate in the UV and short-wavelength-light region, where photon energies are greatest. (See Chapter 8, Conserving Vision, for additional discussion of laser injuries to the eye.)

Because the nature and degree of injuries vary with wavelength, it is useful to consider the effects in the seven optical spectral bands that the International Commission on Illumination (*Commission Internationale de l'Eclairage*, CIE) has adopted (Figure 15-17). *Actinic* UV radiation (UV-C [100–280 nm] and UV-B [280–315 nm], which produce photochemical changes) characteristically produces erythema and photokeratitis (welder's flash). UV-A can also produce these effects, but to a far lesser extent. Unless the exposed individual is also being treated with photosensitizers, which make him or her more sensitive to UV-A, exposure to UV-A (eg, from UV-A black lights such as those used in industry) seldom produces an adverse effect. The recent identification of the injurious wavelengths (the *action spectrum*) associated with UV cataracts concluded that only radiation of 295 to 325 nm was effective in producing a temporary or permanent lenticular opacity for acute exposures.[44]

Although lasers exist that can produce thermal effects on the skin, *currently fielded military lasers are not powerful enough to produce any thermal effects on skin.* Effects on the eye are the greatest concern when perform-

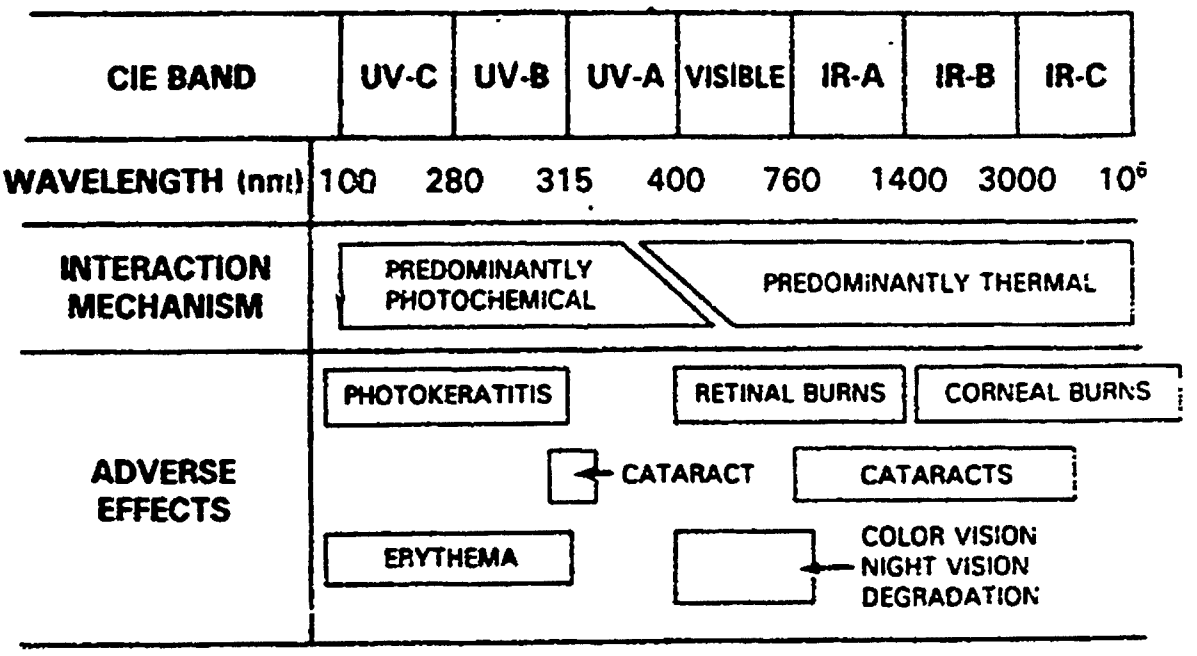

Fig. 15-17. International Commission on Illumination (*Commission Internationale de l'Eclairage*, CIE) spectral bands with their corresponding adverse effects. The direct biological effects of optical radiations are frequency dependent. In the visible and infrared (IR) regions, the interaction mechanism is primarily thermal. In the ultraviolet (UV) region, the interaction mechanism is predominately photochemical, although thermal injury is also present. The biological effects for IR radiation are corneal burns and cataracts. The biological effects for visible radiation are retinal burns, cataracts, and degradation of color or night vision. The biological effects for UV radiation are photokeratitis, cataracts, and erythema. UV-A and UV-B also cause skin cancer.

ing a hazard analysis. There is an obvious interest in defining the sites of principal absorption. Another consideration is not only how much energy is absorbed in tissue, but also its relative biological effectiveness once absorbed. This is particularly important *outside* the spectral region where thermal effects predominate, at wavelengths shorter than approximately 400 nm.

Mechanisms of Injury

Distinguishing the category of injury mechanism is of paramount importance in proposing ELs or maximum permissible exposures (MPEs) for personnel and in predicting the potential long-term, delayed, or chronic effects of exposure. One property that aids in understanding the differences between injury-mechanism categories is *reciprocity* (the product of irradiance, or exposure rate, and the time necessary to produce an effect), which is constant over a wide range of exposure durations. Exposure reciprocity is assumed to hold for up to 8 hours (one workday) and there appears to be very little (although measurable) additivity for multiple exposures if carried over one day.

Photochemical Injury. UV effects and blue-light retinal injury are considered to be photochemical in origin. Photochemical processes involve breaking or forming molecular bonds, or both, and result from the stimulation of electronic energy modes. The property of reciprocity is important in understanding photochemical processes: they obey the reciprocity rule for exposure duration from microseconds to hours. For example, 20 mJ/cm^2 of UV radiation produces the same degree of erythema whether it is delivered as 20 kW/cm^2 for 1 µsec, or as 20 µW/cm^2 for 1,000 seconds, provided the same wavelengths are employed. Exposure guidelines such as the American Conference of Governmental Industrial Hygienists' (ACGIH's) Threshold Limit Values (TLVs) or MPE for eye or skin exposure to UV radiation are therefore expressed as *radiant exposure durations.*

Thermal Injury. Reciprocity does not hold for thermal injury; therefore, it is always necessary to specify the exposure duration when studying a thermal injury. For thermal injury of the skin or eye from a pulsed source, it is the duration of the pulse that determines the threshold irradiance, TLV, or MPE for a given wavelength and effect. The rate-process nature of thermal injury suggests that for exposure durations of less than approximately 10 to 100 µsec, the rate of delivery of thermal energy to the tissue plays only a minor role because heat conduction cannot occur in so short a time.

Even the ELs established for lasers tend to reflect this fact. For example, for ultrashort laser exposures of picoseconds, the biological effect is nonlinear and does not appear to be thermal. For the body to sustain thermal injury, exposure to a higher-intensity laser (J/cm^2) for a duration greater than 100 µsec is required because the body's blood flow and heat conduction away from the exposed site tend to provide some protection. Thus, if thermal injury has not occurred within a few seconds, it is unlikely to occur because a minimal critical temperature (perhaps 45°C) would not be reached through further exposure.

Laser beams may be capable of forming shock waves within tissue. The shock wave is believed to result from the rapid expansion of a plasma, which has been caused by the near-instantaneous heating of a tiny volume of tissue to approximately 10,000°F. Lasers are used in this manner to perform posterior capsulotomies in the treatment of cataracts, and to pulverize gallstones during laparoscopic surgery. Perhaps the clicking or popping sound anecdotally reported by humans who have inadvertently gazed into a laser's beam can be attributed to such a shock wave.[45,46]

Comment on Injury Processes

The entire injury process is better understood when the distinction between injury categories is established. Until recently, the thermal-injury mechanism was thought to be associated with the retinal injury that is sustained when individuals view bright light sources such as the sun. Researchers were puzzled that the calculated retinal temperature rise for an individual who stares at the sun was only 2° to 3°C for a 2-mm pupillary diameter. Laboratory studies of thermal retinal injury also showed that a 10° to 20°C temperature elevation in the 160-µm solar image was required to produce retinal thermal injury within a few seconds. At this duration, short-wavelength light proved to be far more damaging than longer wavelengths, and reciprocity was maintained over a period of hours. Research concluded that the actual mechanism of retinal injury was photochemical, not thermal.[47]

The discovery of the blue-light retinal-injury process and the theory of photochemical injury answered unexplained questions about solar retinitis (eclipse blindness). Researchers had not understood why individuals who gazed at the sun for 2 to 3 minutes during a solar eclipse at midday developed eclipse scotoma, but individuals who gazed for several minutes at the sun while it was low in the sky—and lacking blue light—did not even sustain solar retinitis.[47]

The exact chromophores and details of the injury mechanisms for UV erythema and keratitis also re-

main largely unknown. However, UV erythema and keratitis, like retinal injury, are apparently multistaged because the manifestations of injury are delayed from several hours to 2 days.

Both UV radiation and blue light are most intense at noon. The solar spectral irradiance at 300 nm drops 10-fold by 1500 hours (Standard Time, not Daylight Saving Time). For this reason, an exposed individual does not receive enough UV-B to develop photokeratitis when the sun is near its zenith, but at the same time the skin is more directly exposed to solar UV-B and the individual may sustain erythema solare.

Indirect Biological Effects

Many laser systems used in both research and development and industry contain or are associated with other potential ancillary sources of adverse biological effects such as chemical burns, loss of hearing, exposure to airborne contaminants, and electric shock.[48] These sources include chemical reactants and byproducts, target-generated contaminants, cryogenic fluids, dyes and solvents, ionizing radiation, noise, and high voltage. Consensus standards (such as those from the ACGIH, local and state agencies, and the Occupational Safety and Health Administration) govern many of these sources to limit exposure to contaminants associated with laser operation (Table 15-1).

Current laser systems used in the military for aiming weapons are not expected to pose any ancillary hazards to the operators. However, a potential for exposure to lethal voltages or other harmful radiation hazards might exist when protective covers are removed for service or maintenance. Safety precautions

TABLE 15-1

REPRESENTATIVE CONTAMINANTS ASSOCIATED WITH LASER OPERATIONS

Contaminant	Probable Source	OSHA Allowable TWA	OSHA Ceiling Value
Asbestos	Target backstop	0.2 F*/cc	—
Beryllium	Firebrick target	0.002 mg/m^3	0.025 mg/m^3
Cadmium oxide fume	Metal target	0.1 mg/m^3	0.3 mg/m^3 (0.05 mg/m^3)
Carbon monoxide	Laser gas	50 ppm	200 ppm
Carbon dioxide	Active laser medium	10,000 ppm	30,000 ppm†
Chromium metal	Metal targets	1.0 mg/m^3 (0.5 mg/m^3)	—
Cobalt, metal fume, and dust	Metal targets	0.05 mg/m^3	—
Copper fume	Metal targets	0.1 mg/m (0.2 mg/m^3)	—
Fluorine	HF chemical laser	0.1 ppm	(2 ppm)†
Hydrogen fluoride	Active medium of laser	3 ppm	6 ppm† (3 ppm)
Iron oxide fume	Metal targets	10 mg/m^3 (5 mg/m^3)	—
Manganese fume	Metal targets	1 mg/m^3 (1 mg/m^3)	3 mg/m^3†
Nickel and insoluble compounds	Metal targets	1 mg/m^3 (0.05 mg/m^3)	1 mg/m^3†
Nitrogen dioxide	GDL‡ discharge	(3 ppm)	1 ppm† (5 ppm)†
Ozone	Target & Marx generators	0.1 ppm	0.3 ppm† (0.1 ppm)
Sulfur dioxide	Laser exhaust	2 ppm (2 ppm)	5 ppm† (5 ppm)†
Sulfur hexafluoride	Saturable absorber	1,000 ppm	—
Uranium (soluble/insoluble)	Target	0.05/0.2 mg/m^3 (0.2 mg/m^3)	0.6 mg/m^3† (0.6 mg/m^3)†
Vanadium fume	Target	0.05 mg/m^3 (0.05 mg/m^3)	—
Zinc oxide fume	Target	5 mg/m^3 (5 mg/m^3)	10 mg/m^3† (10 mg/m^3)†

Values in parentheses denote level recommended by ACGIH
*Fibers > 5 μm in length
†Short-term exposure limits
‡Ground designator laser

are provided in the appropriate technical manuals.

Many chemical fuels and exhaust products are associated with the operation of some laser systems (Exhibit 15-2). For example, the use of high-energy hydrogen fluoride or deuterium fluoride chemical lasers can cause atmospheric discharges of helium, oxides of nitrogen and sulfur, and several fluorinated compounds (including hydrogen fluoride or deuterium fluoride, which are corrosive and environmentally toxic). Normal ventilation techniques, such as dilution and local exhaust, and other engineering and administrative controls for industrial hygiene can reduce the concentrations of chemical reactants and their byproducts.

The *target* of a laser operation can itself generate airborne contaminants during laser material processing, beam termination, and interactions with metal surfaces (such as arc welding). The ACGIH has recommended limits for welding fumes to provide protection from arc-welding contaminants. Control of airborne contaminants can also be achieved through local and dilution exhaust ventilation, and other engineering and administrative controls.

Cryogenic fluids such as liquid nitrogen and others are utilized to cool some lasers and many high-sensitivity photodetectors. When cryogenic fluids evaporate, they displace breathable oxygen and thus should be used only in areas of good ventilation. Another safety hazard associated with the use of cryogenic fluids is the possibility of explosion from ice collecting on a valve or a connector. Both protective clothing and face shields should be used when handling large quantities of liquid nitrogen. Those using gas canisters and cryogenic Dewar flasks are required to follow numerous safety procedures (which are beyond the scope of this chapter) to prevent serious accidents.

EXHIBIT 15-2

LASER FUELS AND EXHAUSTS

Carbon monoxide
Methane
Sulfur dioxide
Sulfur hexafluoride; other sulfur compounds
Nitrogen
Helium
Fluorine
Lithium
Carbon disulfide
Hydrofluoric acid
Hydrogen
Carbon dioxide
Various fluorides
Nitrogen oxides

Organic dyes and solvents are often used as lasing media. Solvents usually compose 99% of the dye solution by weight and are commonly flammable and toxic by inhalation or percutaneous absorption. Control measures for dyes and solvents include exhaust ventilation and proper storage and handling of flammable chemicals.

Ionizing radiation—X rays—are generated from some high-voltage power tubes and electron-beam lasers. This ionizing radiation can be controlled through proper monitoring and shielding procedures. Manufacturers can successfully shield lasers to prevent X-radiation leakage, provided that the lasers are operated with the shields in place.

Noise levels that exceed the acceptable standards are generated by certain high-energy lasers and transversely excited atmospheric pressure lasers, although most lasers operate silently. However, these hazardous noise levels occur only near the laser or its target, where personnel are not permitted. Other safety considerations thus obviate the need for noise-control measures.

Lethal voltage levels often are generated inside the laser-system enclosure. Personnel can be exposed to these voltages if the system covers are removed or if the electrical interlocks are defeated. Standard electrical safety precautions can reduce the risk of electrocution.

Military Applications

Using light for long-range, line-of-sight communication is not new. Paul Revere received a coded message in light before his ride in 1775. Morse code utilizing light was widely used during World War II. Current military uses of laser systems (both hand-held and mounted on vehicles or aircraft) include rangefinding or distance measurement, tactical target designation, and simulation of ballistic characteristics for training purposes. Lasers can also be used as part of fire-control systems and in conjunction with night-vision and IR-sensing technologies.

Rangefinders

Rangefinder laser devices emit a single pulse or series of pulses toward a target. A counter is activated

when the pulse is emitted. When the light contacts the target, a diffuse reflector, it is scattered in all directions. Optical sensors receive the light reflected back to the rangefinder and deactivate the counter. Thus, the distance from the rangefinder to the target can be calculated from the time of travel between the laser and target and the speed of light using the formula

$$r = \frac{c \cdot t}{2}$$

where r represent the range in meters, c represents the speed of light (3×10^8 m/sec), and t represents time in seconds for the pulse of light to travel the round trip, which is why it is necessary to divide by 2.

The military frequently uses Nd:YAG as a rangefinding and designating laser medium. The wavelength of this laser is 1,064 nm, in the near-IR region of the spectrum. Both the hand-held AN/GVS-5 rangefinder and the vehicular-mounted AN/VVG-3 rangefinder on the M1 tank employ a single pulse of Nd:YAG. The AH-1F Cobra helicopter employs a multiple pulse of Nd:YAG in its Laser Augmented Airborne Tow (LAAT) rangefinding system. Although the Nd:YAG laser is used frequently, it is not used exclusively in military laser systems. For example, the M60A3 tank employs the AN/VVG-2, which is a single-pulse ruby laser rangefinder that operates in the visible region of the spectrum at 694.3 nm.

Tactical Target Designators

Laser systems accomplish tactical target designation by emitting a series of pulses toward a diffuse reflection target, which scatters the light. Programmed optical sensors respond to the particular code of pulses that the designator emits and direct munitions toward the target. Several laser systems for tactical target designation also have rangefinding capability (such as the G/VLLD [ground/vehicle laser locator designator]), but others are capable only of tactical target designation (such as the hand-held AN/PAQ-1 [portable, invisible radiation, special purpose]).

Directed Fire Simulator

Lasers are used extensively in military training to simulate ballistic characteristics of live-fire weapons. The most frequently used laser training system is the Multiple Integrated Laser Engagement System (MILES), which uses laser diode technology. In most cases, the diode used is GaAs, which operates in the 905-nm region of the spectrum. The laser diode is programmed to emit a code of pulses to simulate a particular weapons system. The sensors attached to the target (such as a tank, aircraft, or personnel) receive the code of laser pulses and interpret the code as a *kill* or a *near miss*. A near miss will signal the target as being engaged, and a kill will shut down the MILES.

New Applications

The military also uses lasers in conjunction with night vision and IR-sensing technology. For example, diode lasers operating in the near-IR region can illuminate a target to enhance its signature, thus making the target more visible through a night-vision or IR-sensing device. Similarly, low-power gas lasers, usually composed of helium and neon (He-Ne) and operating at 632.8 nm, are used in association with small-caliber arms for sighting targets.

Other new applications of lasers include relatively safe, low-powered lasers that are being developed for guided optical communication systems or fiber optic networks, and short-to-medium range or line-of-sight communicators.

Likewise, technology is being developed to use the laser as an optical countermeasure: a high-power laser with a rather large beam divergence can be focused on a target to overwhelm the target's biological (the eyes) or electronic optical sensors that are being used in conjunction with its fire-control system. Also, with the development of technology, very high power lasers will be used in directed energy warfare to engage targets as a direct-fire weapon. The laser will transfer EM energy to a target and cause the target, or one of its critical components, to overheat and malfunction.

Four types of high-energy laser technologies are potentially suitable for weapons applications: the gas dynamic laser, the electric-discharge laser, the chemical laser, and the free-electron laser. However, each of these systems has significant limitations regarding military utility; therefore, no high-energy laser system has been fielded.

Laser Protection Standards

Until 1973, when the first ANSI Standard (Z136.1) pertaining to lasers was published, only general standards for the use of lasers existed. This standard laid the groundwork for a multitude of laser standards, including standards pertaining to lase use in industry and the military, performance standards, and environmental and international laws. Today, ANSI Standard Z136.1[49] is updated triennially. Additional ANSI standards encompass other laser uses: ANSI Standard Z136.2[50] for fiber optics systems, and ANSI Standard

TABLE 15-2

U.S. ARMY GUIDANCE PERTAINING TO LASER USE

Regulation	Title	Purpose
AR 40-5	*Preventive Medicine*	Describes the radiation control program
AR 40-46	*Control of Health Hazards from Lasers and High Intensity Optical Sources*	Establishes U.S. Army policies, procedures, and standards for protection of personnel from the hazards of optical radiation
AR 385-63	*Policies and Procedures for Firing Ammunition for Training, Target Practice, and Combat*	Provides procedures for operating lasers outdoors on a U.S. Army range
AMC Reg 385-29	*Laser Safety*	Defines the use of lasers at U.S. Army Materiel Command installations
TB MED 524	*Control of Hazards to Health from Laser Radiation*	Provides exposure limits and guidance, and establishes responsibilities for personnel protection from radiation; applies to active U.S. Army, U.S. Army National Guard, U.S. Army Reserve, and Corps of Engineers facilities

Z136.3[51] for medical lasers. ANSI Standard Z136.4 concerning laser measurement is also being developed. Although the U.S. Navy uses ANSI Standard Z136.1 directly, the U.S. Air Force maintains its own standard (Air Force Occupational Safety and Health [AFOSH] Standard 161-10)[52] and the U.S. Army maintains several regulations and technical bulletins pertaining to laser use (Table 15-2).

Title 21, Code of Federal Regulations, Part 1040, dictates the performance standards for all laser devices manufactured after 1976, but some exemptions have been made for the military.[53] Soon after this regulation was promulgated, the DoD obtained an exemption for tactical laser systems, outdoor training lasers, and lasers that were classified in the interest of national defense. In addition, alternate design criteria were developed for the army, navy, and air force, and were published in Military Standard (MIL-STD) 1425A.[54] MIL-STD-882C addresses safeguards from other, related potential hazards.[55]

Even certain environmental laws affect the use of lasers and laser facilities. Congress created the *National Environmental Policy Act of 1969* to establish national policy to protect the environment and to minimize adverse environmental consequences of federal actions.[56] Certain provisions of the act are incorporated into other federal regulations including the *National Historic Preservation Act of 1966*[57] and the *Endangered Species Act of 1973*.[58] Army Regulation 200-2 contains U.S. Army policy pertaining to these matters.[59] Because lasers can be used outdoors on a range, the effect of laser radiation on endangered species and other wildlife must be considered.

Lasers are also a subject of concern on the international and national scenes. The United States is a part of the North Atlantic Treaty Organization, which maintains a standardization agreement on laser radiation, *Standardization Agreement* (STANAG) 3606.[60] New York and Texas have also set state restrictions for laser use.

Official standards do not yet address the use of high-intensity optical sources other than lasers. Safety guidelines for these sources are provided in the ACGIH publication, *Threshold Limit Values and Biological Exposure Indices*.[61] This document provides guidelines for the use of intense visible sources, which can produce retinal thermal injury; sources of intense blue light, which can produce retinal photochemical injury; and IR radiation, which can adversely affect the lens. These guidelines will probably provide a basis for future army standards regarding these sources.

Exposure Limits

PELs are not used in the field of laser technology. The terms used are ELs or MPEs. The ELs for laser sources, like those for RF radiation sources, are based on the biological damage level.[48] (These values should be used as guides in the context of exposure. They are based on the best available information from experimental studies.) The interior of the eye is transparent, to a varying extent, to wavelengths between 400 and 1,400 nm. The visual response of the eye is wavelength dependent. The amount of incoming optical energy that is first transmitted through the anterior portions

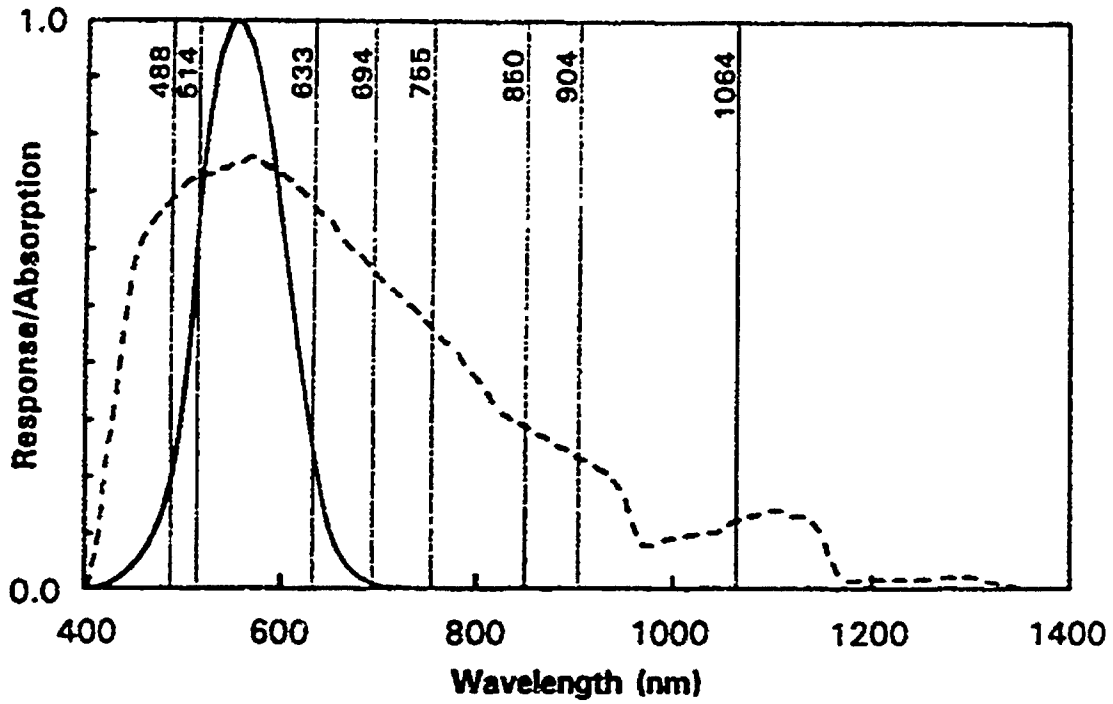

Fig. 15-18. The eye's absorption of nonionizing radiation depends on the wavelength of the incident radiation. Human visual response is limited to wavelengths of approximately 400 to 700 nm, but the retinal pigment epithelium (RPE) absorbs from wavelengths 400 to 1,400 nm. Although we may not be able to see the laser, the eye still responds and focuses the energy.

of the eye and then absorbed by the retinal pigment epithelium (RPE) is also wavelength dependent (Figure 15-18). Separate ELs have been established for all wavelengths. The cornea and lens of the eye concentrate the energy of a laser beam 100,000-fold greater than the unfocused energy; therefore, extremely small amounts of optical energy can injure the retina.

Because pulsed lasers operate at pulse widths of less than one nanosecond to several seconds, the ELs depend on the pulse width, repetition, and expected duration of exposure. An individual might be exposed to a visible laser for 0.25 seconds, until the natural aversion response to light causes the exposed individual to turn away. In comparison, exposure to invisible wavelengths can last 10 seconds or longer, before the individual becomes aware that he or she is being exposed. This is because invisible wavelengths do not invoke the natural aversion response to light.

Laser-control standards keep exposure levels low enough to preclude any known risk, even repeated exposures day after day. The radiant exposure that causes a minimum injury, such as a visible lesion on the retina, cornea, or unprotected skin, is generally 10- to 50-foid higher than the established ELs.[62]

Threat Analysis and Evaluation

An analysis of the hazards that laser systems pose must (*a*) verify that the engineering controls that have been incorporated into the system comply with the control standards, (*b*) determine the requirements for eye protection, and (*c*) determine the nominal ocular hazard distances (NOHDs) for both the unaided eye and the eye that is aided by magnifying optics. The NOHD is the distance required for the beam to expand to the point where the EL is not exceeded. The NOHDs for military designator lasers extend 10 to 20 km for unaided viewing and 40 to 50 km for optically aided viewing; the NOHD for military rangefinders is generally 5 to 10 km. However, these lasers are known to cause various levels of eye injury at shorter distances. The ruby laser rangefinder exceeds the hemorrhage level within 100 m (Figure 15-19), and can inflict a retinal lesion within 1,100 m if the victim is standing directly in the beam. A laser designator can inflict a retinal lesion within 5 km, but again, the victim must be standing directly in the beam to sustain this injury, and, in the judgment of USAEHA's Laser Branch, the probability that this will occur is very small. An optical

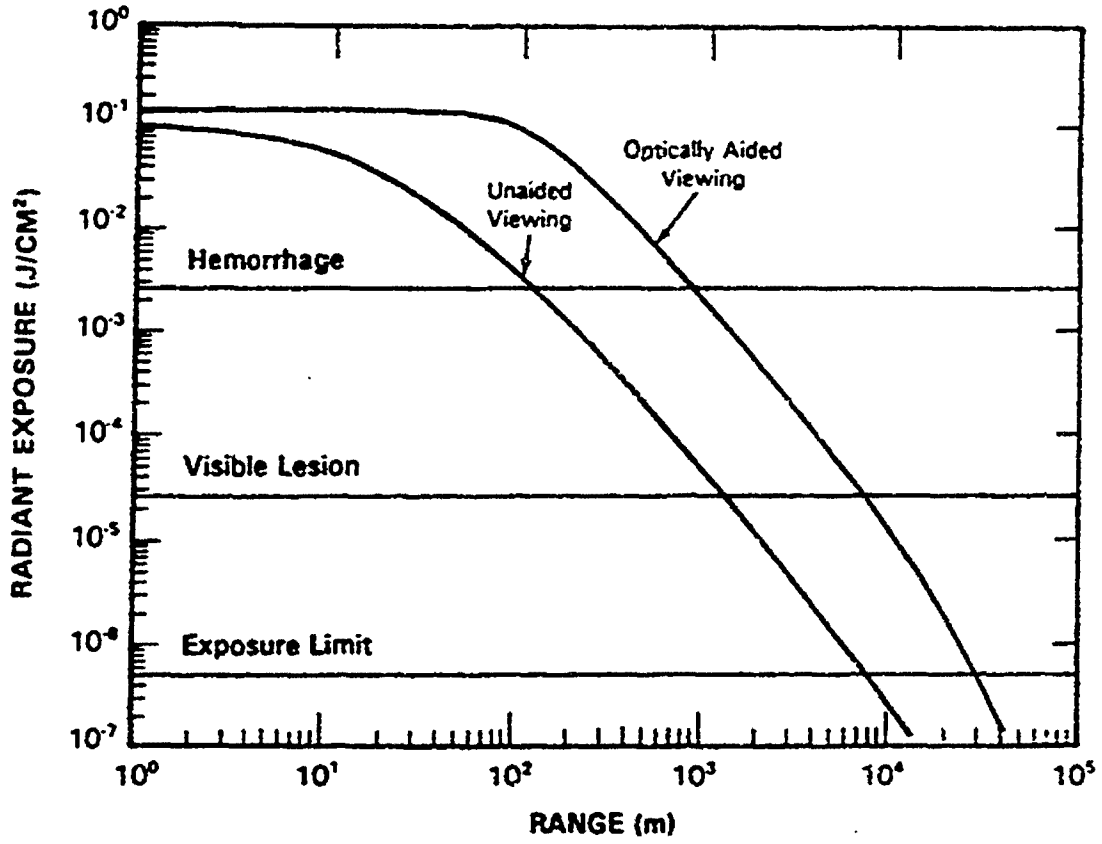

Fig. 15-19. The concentration of ruby laser light is plotted as a function of distance from the laser (range). The graph predicts that a retinal hemorrhage would occur at a distance less than 100 m, a visible lesion would occur at a distance less than 1,100 m, and that the exposure limit would be exceeded at a distance less than 8,000 m.

viewing system can extend these distances by magnifying the power of the system.

Laser and Optical Radiation Exposure Incidents

Unlike RF radiation exposures, few incidents of laser and optical radiation exposures have been reported since 1981, when the USAEHA began investigating them. Eight incidents were formally investigated from 1981 through 1987, although approximately 16 were known to have occurred. From 1988 to 1992, one additional laser incident occurred, and in 1988, a formal procedure was established for the investigation of laser and RF radiation exposure incidents (USAEHA Regulation 40-13).[63] This procedure may involve a formal investigation followed by an official report, but only when directed by the OTSG.

The following four case studies of laser and optical radiation exposures have been selected to illustrate the range of incidents that the USAEHA investigates. The first two concern two of the three serious retinal injuries that have occurred since 1981 (the third, which occurred in 1984 at Fort Bragg, North Carolina, involved the AN/PAQ-1 and was similar to these two). (See Figures 8-17 and 8-18 in Chapter 8, Conserving Vision, for other laser injuries.) The third and fourth case studies describe incidents where the reported ocular effects were inconsistent with the potential laser exposure.

Case Study 1

On 3 March 1987 a civilian employee at Aberdeen Proving Ground, Maryland, was adjusting a Nd:YAG-pumped dye laser when he reported seeing a single orange flash. The laser was operating at a wavelength of 620 nm with a pulse-repetition frequency of 10 Hz. The individual was not wearing laser eye protection at the time because it inhibited his view, and viewing the beam is essential for performing adjustments.

During the eye examination that was conducted after the exposure, the individual reported seeing a central reddish scotoma approximately 2 feet in radius in his right eye when observing a large object 20 feet away. The examination showed visual acuity of 20/x [not measurable] for the right eye, and 20/20 for the left eye. A funduscopic

examination showed a macular hemorrhage approximately 1.5 disc diameters wide in the right eye and a normal left eye. Retinal photographs were taken on the day of the injury and on followup eye examinations (Figure 15-20).

The examinations and investigation hold that the individual probably received a total intraocular exposure of approximately 550 μJ, which is 3,000-fold greater than the occupational exposure limit of 0.19 μJ. The injury was consistent with the exposure parameters. The individual did regain 20/20 vision in the injured eye, but continues to experience a slight visual degradation in his visual field when using the affected eye for monocular vision.

Case Study 2

On 18 July 1989 a U.S. Army soldier stationed in the Federal Republic of Germany reported two laser exposures induced by an MX-9838 AN/GVS-5 laser IR observation set. The soldier claimed that he was exposed to the direct beam at 10 to 12 inches from the source. With each exposure, the soldier reported seeing a whitish flash, hearing a click, and then immediately seeing a dark spot in his visual field. Later, he reported seeing what appeared to be dark jellyfish tendrils in his field of view, which appeared red in high-level ambient illumination.

Because the GVS-5 is a single-shot laser rangefinder, a maximum of one injury is expected per exposure. The retinal examination, however, showed four separate lesions in and around the macula. The ophthalmologist, who examined the soldier the day following the injury, discovered the right eye to demonstrate poor visual acuity (20/400), and the left eye to demonstrate 20/20 visual acuity (Figure 15-21). Although the ophthalmologist found the left eye to be normal, the right eye had sustained multiple macular and penmacular laser burns with edema, subretinal hemorrhages, rupture of the internal limiting membrane, and vitreal hemorrhage.

The eye examination and the USAEHA investigation concluded that the maximum intraocular exposure for each pulse could be 15 mJ at 1,064 nm, if the exposed eye collected all the radiant energy emitted. The occupational exposure limit for a pulse less than 50 μs at 1,064 nm is 1.9 μJ. The potential exposure was therefore approximately 8,000-fold greater than this limit. Although the severity of the injuries was consistent with the exposure, the number of injuries was not consistent with the incident as reported.

Case Study 3

On 4 October 1984 an individual at Jefferson Proving Ground, Indiana, reported being exposed to a helium-neon laser for 1 to 2 minutes. The individual reported seeing a dark afterimage in a uniform circle, approximately the size of a golfball. The afterimage gradually disappeared, and it had completely disappeared within 1 hour after the alleged exposure.

The individual had two eye examinations after the alleged incident. An examination performed 6 hours after the incident showed visual acuity of 20/30 for the right eye, and 20/20 for the left eye. The individual reported no visual disturbances. Six days later, a followup examination demonstrated 20/20 visual acuity in both eyes. Neither of the two examinations revealed any ophthalmoscopically visible retinal changes.

Like the eye examination, the USAEHA investigation of the incident failed to prove that an overexposure had occurred. The investigation showed that at the time of the alleged exposure, the individual was located approximately 8.1 m from the transmitter. The laser was rigidly mounted and the direct beam's height was too far above the ground to expose the individual's eyes. If the individual had been exposed to the direct beam at this distance, he would have received a corneal irradiance of 170 $\mu W/cm^2$. Approximately 1 minute of exposure to 170 $\mu W/cm^2$ is permitted, and similarly, 8 hours of exposure is permitted for 17 $\mu W/cm^2$. The actual exposure was probably approximately 1.7 $\mu W/cm^2$, which is far below the 8-hour limit. Therefore, the individual was not exposed beyond the occupational limit, and the persistence of the afterimage was not consistent with the exposure level.

Case Study 4

On 2 July 1986, while inside the turret of an M60A3 tank at Fort Indiantown Gap, Pennsylvania, an individual performed a self test on a ruby laser rangefinder operating at 694.3 nm. Within one-half hour after performing the self test, the individual complained of an irritation in his left eye. Inflammation increased, becoming more intense over the next 4 days. When the individual returned to work after a holiday, he was sent for an examination. The civilian ophthalmologist's examination proved normal, except for a preexisting nevus on the fundus of the left eye, and episcleritis and keratitis of the left eye. The ophthalmologist did not attribute the nevus to laser exposure, but did attribute the episcleritis and keratitis to accidental laser exposure because the individual had been working with lasers at the onset of these conditions.

U.S. Army ophthalmologists examined the individual's eyes and disagreed with the findings of the civilian ophthalmologist. The patient was then flown to Walter Reed Army Medical Center for examination 4 days after the initial eye examination. Army ophthalmologists there agreed with the clinical findings of the civilian ophthalmologist, but were more guarded about attributing the other two findings to laser exposure. Contrary to the civilian ophthalmologist's conclusion, episcleritis is a relatively common inflammation of the anterior segment of the eye and its cause is usually unknown. Similarly, the civilian ophthalmologist's conclusion that a ruby laser induced the keratitis is inconsistent with scientific and clinical evidence that suggests that red light at 694.3 nm cannot damage the cornea unless it is of sufficient intensity to cause catastrophic injury to postcorneal ocular tissue. The onset of the episcleritis and keratitis after the laser self test was coincidental. In addition, radiometric measurements verified that no laser radiation was present inside the turret during the self test.

Fig. 15-20. (a) The retinal photograph of an accident victim's right eye shows macular hemorrhage. (b) The retinal photograph of the patient's right eye, taken 3 months after the accident, shows recovery to 20/20 vision.

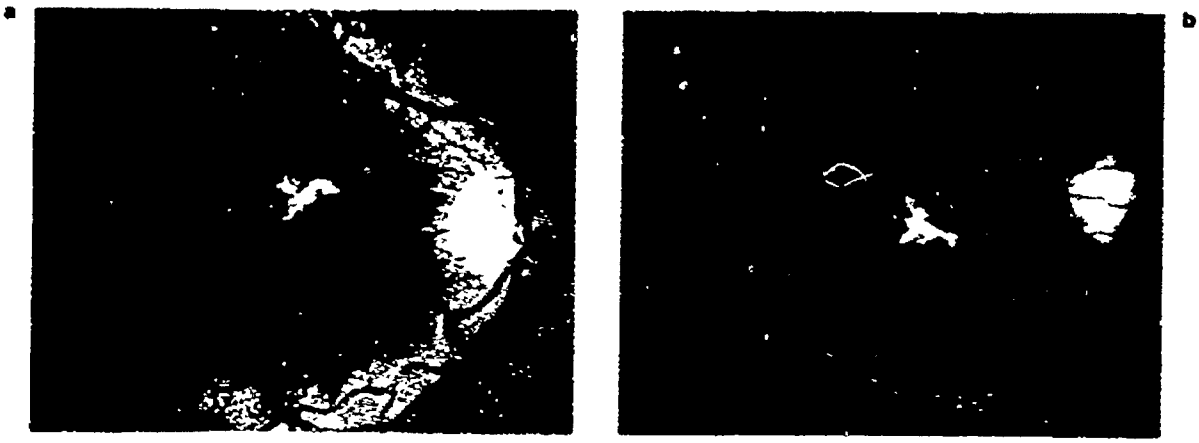

Fig. 15-21. (a) This retinal photograph of the patient's right eye was taken 3 weeks after the incident. Little change is noted and vision has not improved beyond 20/400. (b) This retinal photograph of the patient's right eye was taken approximately 1 month after exposure to an AN/GVS-5 laser.

The Laser Protection Program

The installation or activity commander has control over the Laser Protection Program elements; the installation or activity RPO is responsible for implementing the program. A typical Laser Protection Program includes elements such as (*a*) laser inventory and threat classification, (*b*) engineering controls, (*c*) training, (*d*) administrative controls, and (*e*) laser eye protection.

Inventory and Threat Classification

Complete program files should be maintained by the RPO to include the current records of inventory, SOPs, and records of related safety instruction.

Lasers and laser systems are evaluated to determine the severity of hazard that they are capable of posing. Each laser or laser system is classified in one of four basic hazard categories, with Class 1 being the least hazardous and Class 4 being the most.[44] Class 1 lasers do not pose a hazard to personnel, even if all the energy emitted can be focused into a person's eye. A few training lasers and laser-diode systems fall into this category, but most *open-beam* (unenclosed) lasers are too powerful. In many systems, however, powerful Class 4 lasers are enclosed inside interlocked cabinets within the system. These *enclosed* lasers present no hazard during normal operation and are therefore categorized Class 1.

Class 2 lasers, which are visible, only pose a hazard if an individual overcomes his or her normal aversion response and stares into the laser for more than 0.25 seconds. Lasers less than 1 mW are categorized Class 2, and lasers that do not exceed the EL for 1,000 seconds (approximately 16 min) are categorized Class 2a.

In general, lasers that exceed the Class 1 and Class 2 criteria, but are less than 0.5 W in their average power, are categorized Class 3. This category is subdivided into Class 3a and Class 3b, with Class 3a posing the lesser threat. Class 3a lasers do not pose an immediate hazard to personnel who either view the source directly or view a reflection from a specular surface. However, Class 3a lasers pose an *immediate hazard* to personnel who are using magnifying optics. A visible laser is categorized Class 3a if the output power is 1- to 5-fold greater than the Class 2 accessible exposure limits (AELs). A UV or near-IR laser is classified Class 3a if the output power or energy is 1- to 5-fold greater than the Class 1 limit. Class 3 lasers, or laser systems that do not meet the previous requirement (such as single-pulsed neodymium rangefinders), are classified as Class 3b.

Class 4 lasers have an average output exceeding 0.5 W. Some are capable of producing thermal injuries to the skin; others pose hazards from combustion or diffuse reflection.

Engineering Controls

Title 21, Code of Federal Regulations, Part 1040 (1988), dictates the type of engineering controls required for laser equipment, based on the laser's class.[44] This standard requires that all lasers (*a*) contain an interlocked protective housing and (*b*) display proper labeling and appropriate user and service information. In addition, Classes 2, 3, and 4 lasers must contain an emission indicator and a beam attenuator. Classes 3b and 4 lasers are further required to have (*a*) an emission indicator that is activated prior to laser emission, (*b*) a connector that can deactivate the laser remotely, and (*c*) a key-operated switch to prevent unauthorized use. Alternate control measures that do not interfere with the military mission are contained in MIL-STD 1425A for military-exempt lasers. Laser laboratories must also maintain engineering controls to preclude personnel exposure, including warning lights and signs; filtered view ports; and, for Class 4 lasers, door interlocks.

Administrative Controls

SOPs and other administrative controls such as protocols, operators manuals, and good safety practices are important components of laser-protection control. Every installation or activity that uses Classes 3 or 4 lasers should publish an SOP for proper laser use. This SOP should specifically address the hazards of the lasers in the environment where they are used and should prescribe procedures for their safe use.

Training Programs

Laser-safety training is necessary for all who operate potentially hazardous optical equipment. This training should include instruction concerning the hazards of the particular pieces of equipment, protection methods, and emergency procedures. Instructors involved in the training of laser safety and range safety personnel should receive further instruction concerning basic optics, biological effects of lasers, laser safety standards, laser-protective clothing (eyewear, gloves, and flame-retardant clothing), and preparation of laser range areas.

Eye Protection

The type of laser eye protection that the U.S. Army uses is rapidly changing due to new technology. The

Fig. 15-22. The Ballistic/Laser Protective Spectacle. The brown spectacle provides ballistic and sun protection. The clear spectacle provides ballistic protection only. The blue-green frontsert is a laser eye protector that can be worn over either spectacle. Photograph Courtesy of the US Army Environmental Hygiene Agency.

army is planning to distribute the Ballistic/Laser Protective Spectacle (BLPS) to all personnel. BLPS is designed to provide protection both against moving objects and against all currently fielded DoD laser equipment (Figure 15-22; see also Figure 8-29 in Chapter 8, Conserving Vision). However, BLPS is not required in a training environment, due to the administrative controls already in place. BLPS is currently available to a select few (details are classified), but other types of laser eye protection are available through commercial vendors for most army personnel.

Laser eye protectors are designed for protection against a specific wavelength and have a certain amount of attenuation known as optical density (OD). Therefore, the laser's wavelength and the required OD must be known before selecting the proper eye protection. Because the U.S. Army frequently uses the Nd:YAG laser at 1,064 nm and the ruby laser at 694.3 nm, eye protection with an OD of 6 or greater at these wavelengths will provide adequate protection from most lasers fielded by the army.

MEDICAL SURVEILLANCE

There is no scientific basis or epidemiological evidence to suggest that medical surveillance for RF radiation workers is necessary and the army's program of periodic ocular examinations for them was eliminated in February 1992. However, the army has a medical surveillance program for laser workers, which employs both screening and diagnostic examination protocols.

The ocular surveillance examinations within the program are divided into three categories: preplacement, immediate, and termination. These examinations are based on whether the individual is classified as an *incidental* or a *laser* worker. Incidental workers are employees whose work makes it possible, but unlikely, that they will be exposed to laser energy sufficient to damage their eyes. For example, operators of fielded equipment are considered incidental workers. Laser workers are employees who routinely work in laser environments and have a higher risk of accidental overexposure; for example, laser-maintenance workers and research, development, test, and evaluation personnel work in situations where adequate protective measures cannot be provided. Regardless of their classification, all individuals who begin working with lasers must undergo a preplacement examination to determine their baseline visual status prior to potential exposure and a termination examination on termination of employment or military occupational specialty.

If at any time during employment, an individual suspects that he or she has been exposed to a laser beam in excess of the ELs, that individual must be examined within 24 hours of the suspected incident.[45] This action initiates a preliminary investigation and records the incident in a registry of alleged laser overexposures.

Two different protocols—screening and diagnostic—are used for ocular surveillance examinations. The screening protocol is used for preplacement and termination examinations of incidental and laser workers. The diagnostic protocol is used for immediate examinations, and will be performed by an optometrist, ophthalmologist, or physician who possesses the necessary skills.[39]

SUMMARY

EM radiation may be ionizing or nonionizing. Nonionizing radiation includes EM radiation with wavelengths greater than 1 nm and is classified as UV radiation, visible light, IR radiation, microwave radiation, and RF radiation. Nonionizing radiation possesses a variety of physical characteristics such as divergence, interference, coherence, and polarization. These characteristics, together with the media with which it interacts, determines how the radiation is scattered, absorbed, transmitted, refracted or diffracted.

When nonionizing EM radiation is absorbed by matter, either translational, vibrational, rotational, or electronic energy modes of constituent atoms and molecules are excited. Excitation of translational modes generates heat. Excitation of electronic modes generates photochemical reactions.

Nonionizing EM radiation can interact with tissue in a variety of ways, the most medically important being absorption and excitation of electronic modes. Therefore, all direct biological effects from exposure to nonionizing radiation are thermally or photochemically induced. When radiation energy is absorbed by biological tissue, it can be converted to heat. If the total energy absorption exceeds the capacity of the tissue, then a thermal effect may be produced. Optical radiation effects occur to the eyes and the skin. RF radiation can affect all organs of the body; however, the eyes and skin are generally the most sensitive.

The direct biological effects of RF radiation exposure are dependent on the radiation frequency and mechanism of energy transfer. Conduction of RF energy requires physical contact with an active RF source and can cause an RF shock or burn. An RF burn is generally deeper than an ordinary burn. Coupling of RF energy from an RF source at the resonant frequency of the body will increase the energy 3- to 5-fold. Absorption is the only mechanism for energy transfer when the recipient is at least 0.2 λ from the source. The PELs for RF radiation are based on limiting the specific absorption rate to no more than 0.4 W/kg. The biological effects from RF radiation absorption include lenticular cataracts, erythema, and tissue burns.

The direct biological effects of optical radiations are frequency dependent. In the visible and IR regions, the interaction mechanism is primarily thermal. In the UV region, the interaction mechanism is predominantly photochemical, although thermal injury is also present. The biological effects for mid- to far-IR radiation are corneal burns and cataracts. The biological effects for visible and near-IR radiation are retinal burns and degradation of color or night vision. The biological effects for UV radiation are photokeratitis, cataracts, and erythema.

Lasers are a special case of nonionizing radiation. Lasers are highly collimated, monochromatic, and intense sources of radiation; therefore the injuries caused by lasers are generally acute, and consist mostly of localized burns or retinal injuries. The military applications of laser technology consist mainly of lasers for targeting devices; these lasers do not cause injury to the skin but can cause permanent retinal injury when a soldier stares directly into the laser's beam.

Indirect biological effects may follow the use of nonionizing radiation sources. EM interference with electronic devices can disrupt the operation of life-support equipment such as pacemakers. Interference can occur at levels below the PEL for RF exposure of humans. Ancillary sources of health effects associated with lasers include caustic chemicals, noise, airborne contaminants, and electricity. Ionizing radiation, such as X rays, are also produced by some RF and optical sources where high-voltage vacuum tubes are employed.

The military uses RF radiation for communication, target detection, imaging, electronic countermeasures, medical diathermy, industrial heating, and food preparation. Ground surveillance radar assists aircraft landing under adverse weather conditions. Radios permit commanders to direct their troops and obtain information from their troops. Air-defense radars direct weapons to hostile targets and provide early warning of an attack. Satellite communication terminals provide for long-range voice and data transmissions with greater speed and capacity than radio transmissions.

The protection of army personnel from overexposure to nonionizing radiation is accomplished through a comprehensive Radiation Protection Program. The implementation of the program is the responsibility of the installation or activity commander to ensure the safety and health of his or her personnel. To this end, consensus standards for occupational exposure to nonionizing radiation have been developed and are enforced through army regulations. The primary regulation is AR 40-5. Procedures have been established for the investigation of alleged overexposure incidents.

The incidence of accidental exposure to nonionizing radiation in excess of established limits has been rare, especially considering the number and variety of sources in use today and the types of environments where they are used. The best medicine is preventive. But in the event of an accidental overexposure, it is the responsibility of the attending physician to determine if an injury has occurred and to prescribe a treatment. An eye examination should be performed as a minimum.

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REFERENCES

1 Sears FW, Zemansky MW, Young HD *University Physics*. 5th ed Reading, Mass. Addison-Wesley Publishing, 1979.

2. Yariv A. *Introduction to Optical Electronics*. New York: Holt, Rinehart and Winston; 1985.

3. Stutzman WL, Thiele GA. *Antenna Theory and Design*. New York: John Wiley & Sons; 1981.

4. Jenkins FA, White HE. *Fundamentals of Optics*. New York: McGraw-Hill; 1976.

5. Santo B. Making waves at 100. *IEEE Spectrum*. 1988;(May):58–60.

6. *Encyclopedia Britannica*. Vol 14. Chicago: William Benton; 1972. 856

7. Adams JA Electrotechnology in World War II: Detecting the enemy. IEEE *Spectrum* 1987;(Aug).37–41.

8. Fitzgerald K. Electrotechnology in World War II. Targeting the enemy. *IEEE Spectrum*. 1987;(Aug)·42–48

9. *Encyclopedia Britannica*. Vol 14. Chicago· William Benton; 1972; 995–1001.

10. Southworth GC. *Principles and Applications of Waveguide Transmission*. Princeton, NJ: Van Nostrand; 1950.

11. Raven A, Roberts J *British Battleships of World War Two*. Annapolis, Md: Naval Institute Press, 1976. 347–351

12 Garzke WH, Dulin RO. *Battleships—Axis and Neutral Battleships in World War II*. Annapolis, Md. Naval Institute Press; 1985; 290

13 US Army Environmental Hygiene Agency. *Radiofrequency Radiation and Ultrasound Course Manual* Aberdeen Proving Ground, Md: USAEHA; 1990.

14 Chatterjee I, Wu D, Gandhi P Human body impedance and threshold currents for perception and pain for contact hazard analysis in the VLF-MF band. *IEEE Transactions on Biomedical Engineering*. 1982;33(5).486–494.

15 American National Standards Institute *American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 3kHz to 300 GHz.* New York. The Institute of Electrical and Electronics Engineers, Inc; 1990.

16 Bernhardt JH Evaluation of human exposures to low frequency fields In. AGARD Lecture Series No. 138. *The Impact of Proposed Radio-Frequency Radiation Standards on Military Operations* Neuilly Sur Seine, France: NATO Advisory Group for Aerospace Research and Development (AGARD), 1985 [Available from NATO Advisory Group for Aerospace Research and Development (AGARD), 7 Rue Ancelle 92200, Neuilly Sur Seine, France]; 1985.

17 Gandhi OP, Chen JY, Riazi A Currents induced in a human being for plane-wave exposure conditions 0-50 MHz and for RF sealers *IEEE Transactions on Biomedical Engineering.* 1986,33.757–767

18 Chatterjee I, Gandhi OP, Hagmann MJ Numerical and experimental results for near-field electromagnetic absorption in man *IEEE Transactions on Microwave Theory and Techniques.* 1982;33(5):1000–1005.

19 Gandhi OP, Sedigh K, Beck GS, Hunt EL. Distribution of electromagnetic energy deposition in models of man with frequencies near resonance In *Biological Effects of Electromagnetic Waves.* Rockville, Md US Department of Health, Education, and Welfare; 1977. DHEW Publication FDA 77-8011.

20 Polk C, Postow E. *CRC Handbook of Biological Effects of Electromagnetic Fields.* Boca Raton, Fla. CRC Press; 1986.

21 DeLorge JO Disruption of behavior in mammals of three different sizes exposed to microwaves Extrapolation to larger mammals In Stuchly SS, ed *Symposium on Electromagnetic Fields in Biological Systems* Edmonton, Canada International Microwave Power Institute; 1979 215–228.

22 Alexander RC, Surrel JA, Cohle SD. Microwave oven burns to children An unusual manifestation of child abuse *Pediatrics.* 1988,79(2).255–260

23 National Council on Radiation Protection and Measurements. *Biological Effects and Exposure Criteria for Radiofrequency Electromagnetic Fields* Washington, DC: NCRPM; 1986

24. Hu H. Effects of ultraviolet radiation. *Medical Clinics of North America.* 1990;74(2).509–514.

25 Sagan LA Epidemiological and laboratory studies of power frequency electric and magnetic fields *JAMA.* 5 Aug 1992;268(5) 625–629.

26 Stuchly MA Interaction of radiofrequency and microwave radiation with living systems, a review of mechanisms. *Rad Environ Biophys* 1979;16 1–14

27 US Department of Health and Human Services. *An Evaluation of Radiation Emission from Video Display Terminals.* Rockville, Md: Bureau of Radiological Health; 1981. DHHS Publication FDA 81-8153.

28 Craig G Central location for pulse tests urged by EPA officials. *Potomac News* 7 March 1989.

29 Ruggera PS, Elder RL. *Electromagnetic Radiation Interference with Cardiac Pacemakers.* Rockville, Md. U.S. Department of Health, Education, and Welfare; 1971.

30 World Health Organization *Extremely Low Frequency (ELF) Fields.* Geneva, Switzerland. WHO, 1984. Environmental Health Criteria 35.

31 Duchene AS, Lakey JRA, Repacholi MH *IRPA Guidelines on Protection Against Non-Ionizing Radiation* Highstown, NY. McGraw-Hill; 1992.

32. American National Standards Institute *American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz* New York. The Institute of Electrical and Electronics Engineers, Inc, 1982. ANSI Standard C95.1.

33 Millman J *Microelectronics. Digital and Analog Circuits and Systems.* New York: McGraw-Hill, 1979

34 Stimson GW. *Introduction to Airborne Radar* El Segundo, Calif. Hughes Aircraft Co; 1983.

35. Magnusson PC. *Transmission Lines and Wave Propagation* Boston. Allyn and Bacon; 1965.

36 US Department of Army *Hazard Criteria for CECOM Radiofrequency and Optical Radiation Producing Equipment.* Washington, DC: DA, 1991. Technical Bulletin 43-0133.

37 World Health Organization. *Radiofrequency and Microwaves.* Geneva, Switzerland World Health Organization, 1981. Environmental Health Criteria 16.

38 US Department of the Army. *Patient Administration.* Washington, DC: DA; 1983. Army Regulation 40-400.

39. Office of The Surgeon General. *Surveillance of Laser and Radiofrequency Radiation Personnel.* Memorandum. US Department of the Army, 1992. Memorandum.

40 Charschan SS, ed. *Lasers in Industry.* New York: Van Nostrand Reinhold; 1972.

41 Milonni PW, Eberly JH Specific lasers and pumping mechanisms. In: *Lasers.* New York. John Wiley & Sons; 1988: Chap 13.

42. Hillenkamp F, Pratesi R, Sacchi CA. *Lasers in Biology and Medicine.* New York: Plenum Press; 1980.

43 Mueller GJ, Kar H, Helfmann J, Berlien HP. The value of lasers in medicine—A state of the art review. In: Siomos K, ed. *Lasers in Medicine, Proceedings of the First European Workshop on Lasers in Medicine.* Kolimpari, Chania, Crete, Greece: The Commission of the European Communities Directorate General for Science, Research and Development, Joint Research Center; 1988.

44 Pitts DG, Cullen AP, Hacker PD. Ocular effects of ultraviolet radiation from 295 to 365 nm. *Inv Ophthal and Vis Sc.* 1977;16910:932-939.

45. Keller J. Lasers for tomorrow's battlefield. *Military Intelligence Magazine.* 1981;7(2):39-43.

46 Decker CD. Accident victim's view. *Laser Focus.* August 1977 6.

47. Sliney DH. Eye protective techniques for bright light. *Ophthalmology* 1983;90(8):938-944.

48. Sliney DH, Wolbarsht M *Safety with Lasers and Other Optical Sources* New York: Plenum Press; 1980

49. American National Standards Institute. *American National Standard for the Safe Use of Lasers.* Toledo, Oh: The Laser Institute of America; 1986. ANSI Standard Z136.1.

50 American National Standards Institute. *American National Standard for the Safe Use of Optical Fiber Communication Systems Utilizing Laser Diode and LED Sources* Toledo, Oh: The Laser Institute of America; 1988. ANSI Standard Z136.2.

51 American National Standards Institute. *American National Standard for the Safe Use of Lasers in Health Care Facilities.* Toledo, Oh. The Laser Institute of America; 1968. Standard ANSI Z136.3.

52. US Department of the Air Force. *Health Hazards Control for Laser Radiation.* Washington, DC: Department of the Air Force; 1980. AFOSH Standard 161-10.

53. 21 CFR, Parts 1000 and 1040

54. US Department of Defense *Safety Design Requirements for Military Lasers and Associated Support Equipment.* Washington, DC: DoD, 1991. Military Standard 1425A.

55. US Department of Defense *Systems Safety Program Requirements.* Washington, DC: DoD; 1993. Military Standard 882C.

56. *National Environmental Policy Act of 1973.* Pub L No. 93-205.

57. *National Historic Preservation Act of 1966*. Pub L No 89-665.

58 *Endangered Species Act of 1973*. Pub L No. 93-205.

59. US Department of the Army. *Environmental Effects of Army Actions*. Washington, DC DA, 1988 Army Regulation 200-2.

60 North Atlantic Treaty Organization. *Evaluation and Control of Laser Hazards on Military Ranges* 1986. Mons, Belgium. NATO Standardization Agreement (STANAG) 3606.

61 American Conference of Governmental Industrial Hygienists. *Threshold Limit Values and Biological Exposure Indices for 1990-1991*. Cincinnati, Oh: ACGIH, 1990.

62. US Department of the Army. *Occupational and Environmental Health, Control of Hazards to Health from Laser Radiation* Washington, DC: DA; 1985. Technical Bulletin MED 524.

63 US Army Environmental Hygiene Agency. *Laser and Radiofrequency Radiation Indices. Reporting Procedures and Registry Maintenance*. Aberdeen, Md: USAEHA; 1988 Army Regulation 40-13.

64. 21 CFR, Part 1040.

65 US Department of the Army *Accident Reporting and Records* Washington, DC. DA, 1987 Army Regulation 385-40.

Chapter 16

IONIZING RADIATION

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- Procedures for Whole-Body Exposures
- Protecting the Medical Team
- Controlling Contamination in the Medical Treatment Facility

U.S. ARMY RADIATION PROTECTION PROGRAM
- Program Responsibilities
- Program Elements

SUMMARY

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INTRODUCTION

Wilhelm Roentgen could not have envisioned the impact that his 1895 discovery of X rays would have. That discovery, Marie Curie's discovery of radium, the discovery and development of atomic fission and fusion, and other discoveries described in this chapter led to military and civilian applications of ionizing radiation (Exhibit 16-1). With these uses came the need for occupational health programs to control exposures.

Most military occupational exposures are minimal due to the safety procedures and engineering controls in place and the nature of the sources of the radiation. However, many sources do have the potential to deliver significant exposures and a large number of military and civilian employees are routinely exposed to low-level radiation. Thus, occupational exposure to ionizing radiation in the military demands recognition and attention, through strict adherence to all aspects of safety requirements. Clearly, exposures from a nuclear detonation pose the greatest ionizing radiation hazard to the soldier. However, because these effects are described in *Medical Consequences of Nuclear Warfare*, Part I, Volume 2 in the Textbook of Military Medicine series, they will not be discussed in detail here.

PROPERTIES OF IONIZING RADIATION

Radiation is categorized according to its origins and its properties. For radiation to be considered *ionizing*, it must have sufficient energy to strip electrons from the outer shell of neutral atoms or molecules. This stripping of electrons liberates free electrons and positive ions, which can cause a biological effect. Ionizing radiation can be characterized as either *particulate* or *electromagnetic* (EM).

Particulate Radiation

Particulate radiation is composed of alpha particles, beta particles, and neutrons. Alpha particles, which are equivalent to helium nuclei, are heavy and have a double positive charge. They are emitted from nuclei of heavy radioisotopes and can travel up to 10 cm through air, and up to 0.1 mm through tissue. Because alpha particles are easy to shield against and cannot penetrate the outer layers of skin, exposure from external sources causes little biological damage. However, alpha particles that are deposited internally can cause considerable biological damage.

Beta particles are equivalent to electrons: they weigh far less than alpha particles and have a single negative charge (or, in the case of positrons, a single positive charge). They are emitted from the nuclei of radioisotopes, and can travel up to 10 m through air and up to 8 mm through tissue. Beta particles can cause biological damage if they remain on exposed skin and if they are deposited internally. The best shields against beta particles are plastics, or metals with low atomic numbers.

Neutrons have no charge. They are produced in nuclear reactions or are emitted by certain heavy, artificial radioisotopes and can travel up to 3,000 m through air. Because neutrons can penetrate tissue easily, exposure to external sources can cause biological damage to deeper tissues. The best substances to shield against neutrons are hydrogenous materials such as water, paraffin, and concrete.

Electromagnetic Radiation

Gamma rays and X rays are types of EM ionizing radiation, but they differ in their origins: the nuclei of most radioisotopes emit gamma rays, whereas the orbital shells of virtually all radioisotopes emit X rays. X rays can also be machine produced. Both gamma and X rays can travel up to 3,000 m through air. Typically, gamma and X rays can penetrate tissue easily, but their range through tissue depends on their energy. Gamma and X rays can cause biological damage from external exposure or internal deposition of emitting radioisotopes. The best shields against these radioisotopes are heavy, dense metals such as lead, steel, and depleted uranium.

DISCOVERY AND APPLICATIONS OF X RAYS

Roentgen's discovery of X rays was a culmination of the research of scientists such as Wilhelm Hittorf, William Crookes, Heinrich Hertz, and Philipp Lenard. Roentgen's discovery was sparked on 8 November 1895 when he saw the barium platino-cyanide screen fluorescing on a table some distance from the cathode ray tube with which he was working.[1,2] This occurrence stimulated his interest, and he worked feverishly over the next few days to comprehend and document the observed phenomenon. By turning the current on

EXHIBIT 16-1

KEY DEVELOPMENTS IN ATOMIC FISSION

Year	Development
1897	J.J Thomson identified the electron. Ernest Rutherford identified alpha and beta rays emanating from uranium and later correctly identified them as helium nuclei and electrons, respectively.
1898	Villard recognized gamma rays and observed their similarities to the roentgen ray.
1905	Albert Einstein proposed his famous equation, $E=mc^2$, stating the relationship of energy to mass.
1910	F. Soddy suggested an explanation for atoms with slightly different weights, but identical chemical properties, and called them *isotopes*.
1911	Rutherford proposed the atomic theory with a distribution of mass and charge that is essentially the one that we accept today.
1913	Niels Bohr suggested an atomic structure involving a central nucleus with orbital electrons in layers around it.
1919	Rutherford bombarded nitrogen atoms with alpha particles and observed the production of hydrogen and oxygen. This milestone was the first controlled experiment in which one element was artificially transformed into another.
1931	Ernest Lawrence invented the cyclotron, a chamber in which it is possible to accelerate particles to immense speeds for use as projectiles.
1932	James Chadwick of Cambridge University recognized the neutron.
1934	Enrico Fermi first split an atom of uranium by neutron bombardment. Lise Meitner, a German physicist, explained the process and termed it *fission*; it was quickly realized that large amounts of energy were released in this process.
1939	Fermi approached the U.S. Navy Department about the prospects for an atomic weapon, and expressed his fear that Germany would produce and use such a weapon. The importance and power of atomic fission was clear to many scientists. Some also foresaw and were frightened by the implications of its use as a weapon. A letter, drafted by Leo Szilard and signed by Einstein, was forwarded to President Franklin D. Roosevelt, and Roosevelt started the process that would result in the development of the atomic bomb.
1940	D W Kerst constructed a betatron, in which electrons were accelerated to energies of 20 million electron volts (MeV), and later to 300 MeV, by magnetic induction.
1941	The Manhattan Project began, consolidating the fragmented efforts at atomic weapons development. Brigadier General Leslie Groves (a civil engineer) was appointed as the project's director, and J. Robert Oppenheimer (a physics professor at the University of California, Berkeley) was selected as the scientific director.
1942	On 2 December Fermi successfully initiated the first self-sustaining nuclear chain reaction in a uranium pile at the University of Chicago.
1945	On 16 July the first atomic bomb (a plutonium-fueled implosion device) was detonated in New Mexico. On 6 August an atomic bomb (a gun-assembly, uranium-fueled device code-named Little Boy) was dropped on Hiroshima, Japan. On 11 August a second atomic bomb (a plutonium-fueled implosion device code-named Fat Man) was dropped on Nagasaki, Japan.

Source: Dewing SB. *Modern Radiology in Historical Perspective*. Springfield, Ill: Charles C Thomas; 1962.

and off, Roentgen observed that the fluorescence was related to discharge within the tube. Roentgen concluded that he had found a new phenomenon, which emanated from the tube.

In testing this phenomenon's ability to penetrate various materials, Roentgen was startled to see the image of the bones of his own hand on a photographic plate. After this discovery, Roentgen observed and recorded the differential development of photographic plates using materials of various densities. He even produced an image of his wife's hand with a 15-minute exposure.[2] To document the findings of these experiments, Roentgen wrote a paper describing the rays' means of production and their important properties. In December 1895, he submitted it, entitled *A New Kind of Rays*, to the Wurzburg Physical-Medical Society. On 6 January 1896 Roentgen's discovery was announced to the world, creating an immediate stir in the scientific community.[2] Others apparently had observed the photographic effects of X rays but had failed to recognize the significance of the phenomenon.

Medical Uses

The medical community in general, and the U.S. Army in particular, were quick to embrace the new technology that followed the discovery of X rays. Within a year, several examples of the use of X rays for diagnoses were available.

The army attempted to experiment with X rays within 3 months of Roentgen's discovery when the curator of the Army Medical Museum, Major Walter Reed, applied to The U.S Army Surgeon General for authority to obtain a roentgen-ray apparatus. Although Surgeon General George Sternberg initially denied Reed's request, there is evidence that the museum possessed a roentgen-ray apparatus by June 1896. Admission records of Garfield Hospital in Washington, D C., show that a 17-year-old female patient was admitted with a penetrating gunshot wound to the hip, which had been inflicted when her brother accidentally discharged a .22-caliber weapon. Dr. Joseph S. Wall accompanied the patient in a horse-drawn ambulance to the Army Medical Museum, where Dr. William Gray could assist in identifying the bullet's exact location with a Roentgen tube (Figure 16-1). The patient was exposed to X rays for 1 hour before a roentgenogram showing the bullet's location could be obtained. After this examination, the patient was returned to Garfield Hospital, where the bullet was successfully removed.[3]

Although the army began experimenting with X rays soon after their discovery, other countries had actually employed them in treating military casualties

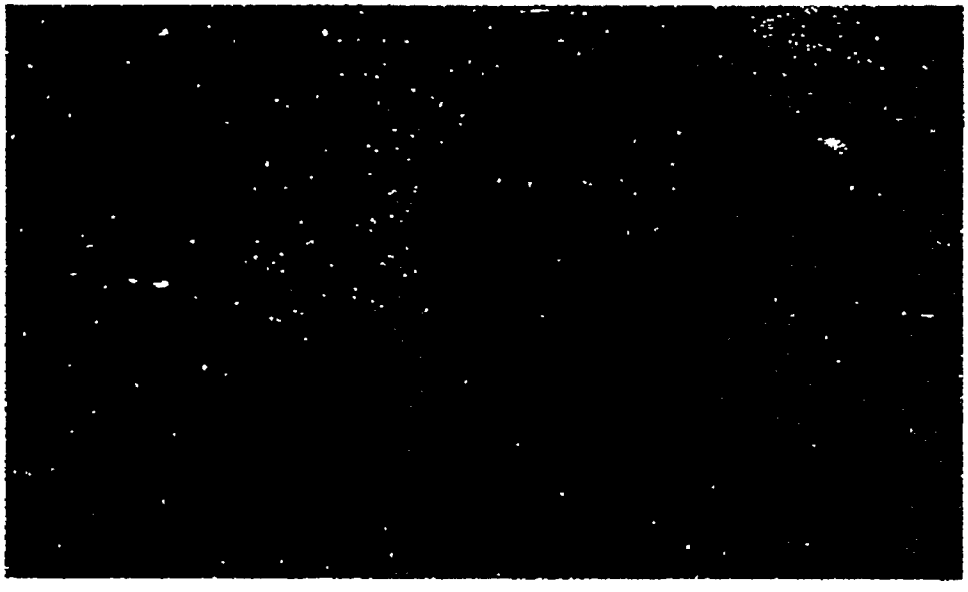

Fig. 16-1 A roentgen-ray tube similar to the one possessed by the U.S. Army Medical Museum that was used to locate a bullet lodged in a patient in 1896. Crude tubes of this type were the first X-ray machines used by the U.S. Army. Source: Henry RS. *The Armed Forces Institute of Pathology, its First Century 1862–1962* Washington, DC: Office of The Surgeon General, DA, 1964.

early roentgenograms may leave much to be desired by today's standards, they were, in fact, remarkable for their clarity and utility (Figure 16-3).

By the time the United States entered World War I, radiology was becoming established as a medical discipline. However, the use of X rays was limited because the equipment and supplies were unsuited to mass use and too few radiologists were available. In fact, in April 1917 the U.S. Army had only one radiologist: Colonel Philip Huntington.[4]

While no real distinction existed between military and civilian medical applications of roentgenology, the military's differing circumstances required specialized apparatus. For example, portable and bedside X-ray units, not used in the civilian sector, were tailored to military needs (Figure 16-4). The army also recognized that X-ray capabilities were necessary in mobile hospitals and surgical units, and therefore modified a standard army ambulance to house a field-portable X-ray apparatus and one bedside unit. In May 1918 the first X-ray ambulance was tested and found to be successful.

On 25 November of that same year, the army refined its methods for using X rays and published the *United States Army X-ray Manual* under the direction of the Division of Roentgenology of the Office of The Surgeon General.[6] This manual served as a guide and textbook for military roentgenologists. By the end of World War I, the United States had shipped 150 complete base hospital X-ray units, 250 bedside X-ray units, 264 portable X-ray units, and 55 X-ray–equipped ambulances overseas.[7]

Radiology as a specialty made tremendous strides during the interval between World War I and World War II: equipment was improved, radiologists were formally trained, and radiological technologies were developed and clinically applied. By the onset of World War II, the use of X-ray technology was well

Fig. 16-3. This famous radiograph of the hand of Prescott Hall Butler showing multiple, retained shot was made by Michael I. Pupin in New York City, probably on 14 February 1896. It was "the first roentgen plate to guide a surgical operation in New York...[and] is the best of all early roentgen prints as far as technical quality (and bone detail) is concerned which is quite unusual when one considers the fact that the x rays were produced in the glass of the tube, and were in no way focused." Reprinted from Gngg EPN. *The Trail of the Invisible Light: From X-Strahlen to Radio(bio)logy*. Springfield, Ill: Charles C Thomas, 1965: 312.

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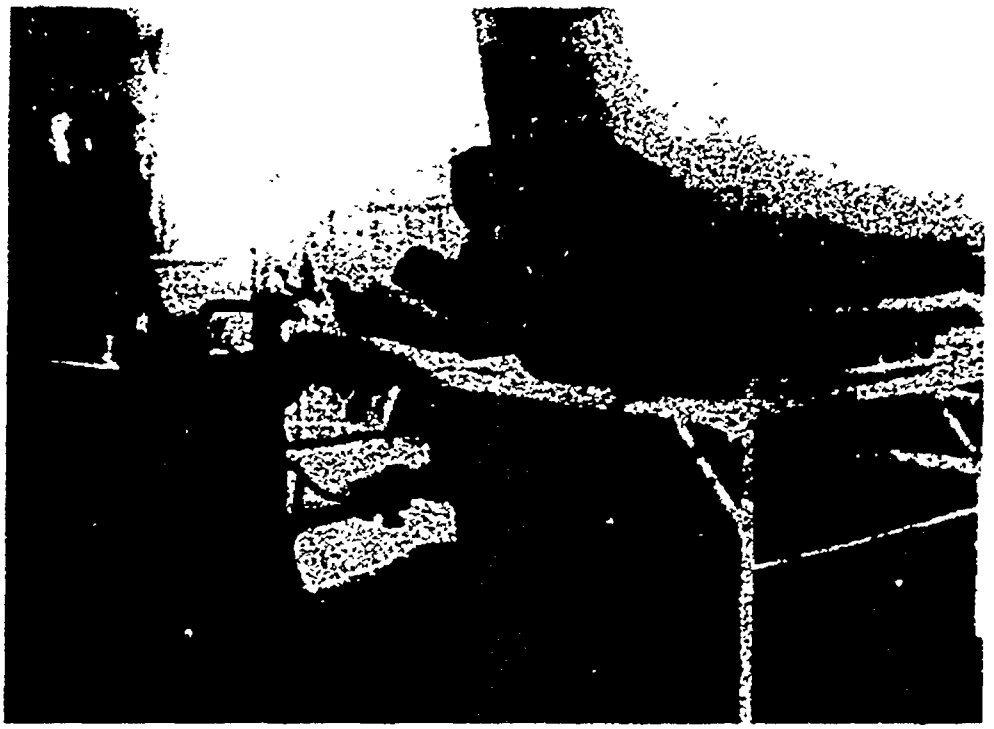

Fig. 16-4. The Waite and Bartlett Army bedside unit, shown at the base hospital in Grand Blottereaux in 1915, was the first stock X-ray equipment that used a Coolidge hot-cathode tube. The examiner looked into a *cryptoscope*, the hand-held fluoroscope Source Feldman A A sketch of the technical history of radiology from 1896 to 1920. *RadioGraphics*. 1989;9(6) 1113–1128 Photograph Courtesy of Arnold Feldman, Methodist Medical Center, Peoria, Ill

established as a diagnostic and therapeutic tool. Radiology as a recognized medical specialty was an integral part of every hospital, and radiology teams were part of auxiliary surgical groups that performed front-line surgery.

Providing radiological services was not effortless, however. Once basic equipment was supplied, radiologists and technicians had to maintain it, often with great difficulty and improvisation. Battlefield needs sparked further developments in mobile and portable X-ray systems such as the U.S. Army Field X-ray unit, which was widely used both at front- and rear-echelon military medical facilities (Figure 16-5).[8] Despite the advances in radiology and training techniques, radiologists were constantly in short supply during World War II In an effort to meet radiological needs, training courses were provided for medical officers and technicians at institutions such as the U.S Army School of Roentgenology.[4]

The importance of radiology during World War II was also reflected in the structure of the U.S Army Surgeon General's Office. The Radiation Branch, later renamed Radiology, was established on 12 July 1942 under the direction of Major Michael E. DeBakey. This branch, a part of the Surgery Division, later became the Surgical Consultants Division.[4]

Great advances in radiological technology were made beginning in the 1950s, partly resulting from the military uses of radiology during World War II. During the Korean and Vietnam Wars, X rays were used extensively in the diagnosis and treatment of casualties (Figure 16-6).

Also during the 1950s and 1960s, A. M Cormack, a South African, did the original work on projection imaging that set the stage for computed tomography (CT). However, the evolution of that technology from experimental curiosity to clinical reality was largely due to the efforts of English engineer Godfrey Hounsfield.[9]

CT was introduced into medical practice in the early 1970s This technology made cross-sectional imaging with X rays possible, which greatly enhanced the physician's ability to see abnormalities in a variety of anatomical structures. Vast technological improvements have been made in CT technology since the Hounsfield scanners were introduced Within only 4 years, major improvements (four generations) of the CT scanner decreased minimum scanning times from

Fig. 16-5. A portable field X-ray unit in action in World War II. The unit shown was developed by the Picker Corporation, which became the sole supplier of the U.S. Army Field X-Ray unit during World War II. Source: Krohmer JS. Radiography and fluoroscopy 1920–1989. *RadioGraphics* 1989;9(6):1129–1153. Photograph: Courtesy of Jack S Krohmer, PhD, Georgetown, Tex.

Fig. 16-6. The X-ray section of a forward surgical hospital during the Korean War. The advances in X-ray technology and techniques that had been developed since World War II permitted field hospitals to practice quality imaging in their treatment of battlefield casualties. Source: Howard JM, ed. The battle wound: Clinical experiences. In: *Battle Casualties in Korea, Studies of the Surgical Research Team*. Vol 3. Washington, DC: Army Medical Service Graduate School, Walter Reed Army Medical Center; 1955.

5 minutes to 5 se .ds; during the next 2 years, the minimum scanning times were reduced to 2 seconds.[9] Advances and refinements continue to achieve enhanced imaging and resolution and to further reduce scan times to milliseconds. Clinical medicine has benefited from cross-sectional imaging, and the field of radiology continues to evolve as medicine advances with the computer era Current approaches being explored employ radiation sources at wavelengths not now used for imaging.

Parallel to their diagnostic uses, the therapeutic uses of X rays date back to 29 January 1896, when Emil H. Grubbe reported that he, in collaboration with Dr. R. Ludlum in Chicago, had treated a carcinoma of the breast with 18 X-ray treatments.[10] During the next few years, therapeutic X rays were tried on conditions ranging from malignancies to excess facial hair. This experimentation resulted in many disappointing outcomes as well as radiation injuries However, the number of successes was sufficient to maintain the interest of scientists and physicians in the therapeutic value of X rays, particularly with respect to tumors.

In the early years, the efficacy of therapeutic X rays was limited by the low kilovoltage that the equipment could achieve, which enabled the X-ray beam to penetrate only shallowly.[10] Thus, *brachytherapy* (ie, the application of an encapsulated radioactive source or sources to deliver a radiation dose at a distance not greater than a few centimeters) using radium was more useful than external-beam therapy (teletherapy) until approximately 1921, when higher-energy external-beam systems became available.[10] In 1937, the

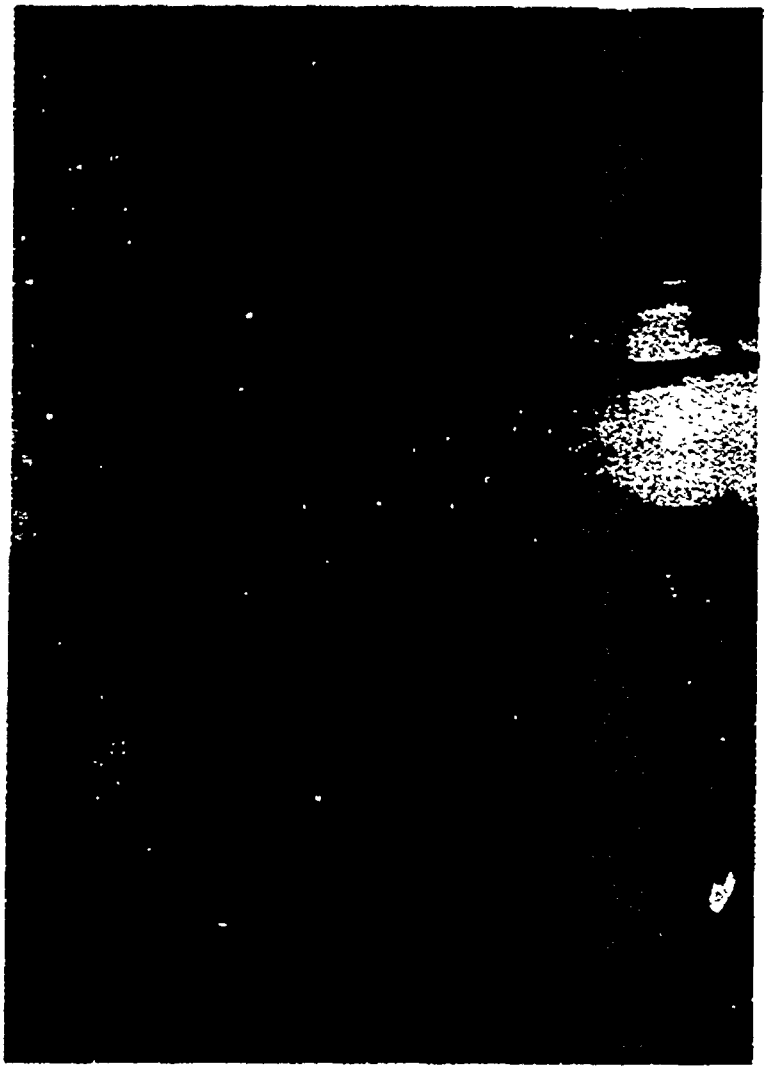

Fig. 16-7. Dr. Ralph Phillips and a patient to be treated using the 1-million-electron-volt (MeV) therapy installation at St. Bartholomew's Hospital, London. The unit created high-energy, penetrating X rays that could be used for treating cancers and other tumors The immediate benefit to the patient of the eradication or reduction of the tumor generally was thought to outweigh the risk of developing future cancers from the high radiation dose delivered by such a therapy device. Source: Laughlin JS. Radiation therapy. *RadioGraphics*. 1989;9(6):1252. Photograph: Reprinted with permission from *Brit J Radiology* British Institute of Radiology, London, England.

earliest type of supervoltage teletherapy unit (Figure 16-7) was used on patients.[11] This 1-million-electron-volt (MeV) unit was used at St Bartholomew's Hospital in London, England, under the supervision of Dr. Ralph Phillips and George Innes.

The therapeutic use of X rays progressed when high-energy sources became available. In 1940, Donald W. Kerst of the University of Illinois developed the betatron (Figure 16-8), which functioned as an electron accelerator. This first betatron operated at 2.3 MeV, the second at 20 MeV, and the third at 300 MeV. In 1948, Kerst collaborated with Dr. Henry Quastler at the University of Illinois in the first treatment of a tumor using these high-energy rays. Localized irradiation from the betatron was administered to a graduate student at the university whose brain tumor had been partially excised. The patient eventually succumbed to cancer, but the autopsy revealed no viable neoplastic cells in the irradiated region.[11] The Allis-Chalmers Manufacturing Company developed a commercial version of the betatron in 1948 with improvements for medical use.

The development of the linear accelerator further advanced the therapeutic use of X rays. Before and during World War II, oscillator tubes capable of relatively high-power output at microwave frequencies were developed and applied to radar.[11] At the end of World War II, the technology was refined and applied to the advancement of the linear accelerator, which has become the predominant modality for delivering modern radiation teletherapy treatment.

Industrial Uses

Industrial radiography sprang from Roentgen's mention of the radiograph of a piece of metal in his 1895 paper. Metallurgists seized this concept as a nondestructive method of examining metals. As early as 1896, the war departments of Germany, Austria, and the United States were using X rays to examine cannons. In 1922, a 200-kV, 5-mA industrial X-ray unit was assembled at the U.S. Army Arsenal at Watertown, Massachusetts.[2] During the 1940s, betatrons were also used extensively in industrial radiography. New tech-

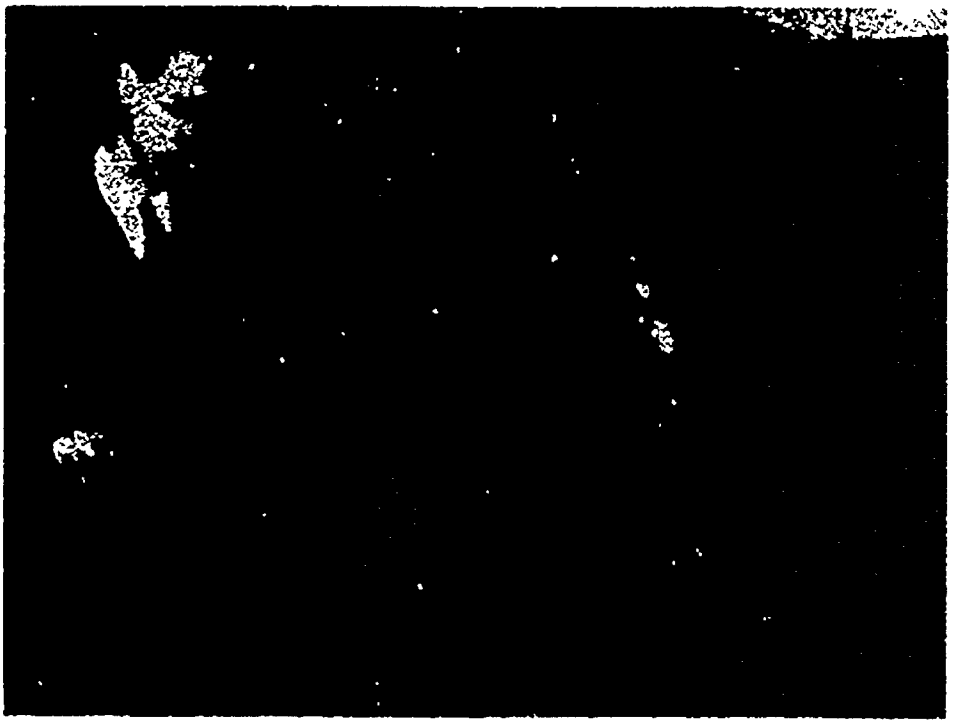

Fig. 16-8. Professor Donald Kerst with two of his betatrons (1940). A betatron is an electron accelerator. These betatrons were compact and able to accelerate electrons to high energies. Electrons that reach sufficiently high energies are able to penetrate deeply into tissue, therefore, accelerated electrons can be used therapeutically. Additionally, the betatron-accelerated electrons were relatively monoenergetic and their energy was easy to control. Source: Laughlin JS. Radiation therapy. *RadioGraphics* 1989;9(6):1254. Photograph: Courtesy of John S. Laughlin, Medical Physics Department, New York, NY.

nology and increased and diversified uses of conventional applications, such as radiography, have proliferated the industrial uses of machine-produced ionizing radiation.

During World War II, General Electric Company physicist E. Dale Trout was assigned to work with the military on industrial radiography. Trout assured the quality of all aircraft templates for the B-17s, B-24s, B-29s, and B-50s with X rays. He claimed that during his work with the military, every shell of 155 mm or larger, all aircraft bearings, and all rocket propellant grains were X rayed, on continuously operating equipment. Trout and the military also assembled a 1-MeV unit at Hayward, California, to radiograph the outboard struts on ships built at Mare Island and at Hunter's Point.[12]

As part of its production-line quality control, the U.S. Army inspects materiel by means of radiographic, fluoroscopic, and continuous automatic inspection. Radiographic inspection to detect a defective weld was attempted within 1 year after the discovery of X rays However, industrial radiography was used very little until 1920 because neither the equipment nor the film available were suited for that purpose. Today, the army has several industrial radiographic units, which range in size from the small portable unit used to inspect pipeline welds to the 25 MeV betatron used to inspect armor plate and missiles.

Fluoroscopy, which produces X-ray images in real time, lends itself to use on conveyor production lines or assembly lines, and is also used for nondestructive, noninvasive inspection of packages and luggage. In the past, fluoroscopic inspection on production lines was limited to thin, lightweight metals and nonmetallic goods, but the development of state-of-the-art image intensifiers permits inspection of heavier materials. Continuous automatic inspection uses devices such as thickness and height-of-fill gauges. Thickness gauges, which automatically control the production machinery, are used to continuously measure the thickness of sheet metal, glass, and rubber These measurements are made by passing the product between an X-ray–emitting tube and the detector. Height-of-fill gauges also operate by passing filled containers between the X-ray tube and the detecting element. Containers not filled to the predetermined level permit more X rays to pass through, which activates a device that automatically removes the underfilled containers from the conveyor.

The military and private industry also employ ionizing radiation to analyze materials by means of X-ray diffraction and X-ray absorption photometry. Because crystals diffract X rays in a specific diffraction pattern, X rays permit qualitative and quantitative analyses of crystalline materials. X-ray absorption photometry is also an analyzing technique, but this method utilizes the differences in absorption of the various elements.

The military, like private industry, uses electron-beam generators to deliver massive doses of radiation. One device for electron-beam processing is the Van de Graaff apparatus, which is an electron accelerator. Another is the 1- or 2-MV resonant transformer X-ray apparatus. Some applications of electron-beam processing include sterilizing foods and drugs, exterminating insects in seeds, toughening polyethylene containers (which induces cross-linkage of polyethylene molecules), and activating chemical reactions in petroleum processing.

DISCOVERY AND USES OF RADIOISOTOPES

In 1896, Henri Becquerel followed Wilhelm Roentgen in exploring the idea that naturally fluorescent minerals might emit rays similar to roentgen rays. On 1 March 1896, while studying the influence of light on the fluorescence of uranium salts, Becquerel placed a sample of uranium in direct sunlight to study the degree of development of a shielded photographic plate he had placed under the sample. When the sky became cloudy, Becquerel interrupted the test and set the cassette aside. He processed this cassette a few days later and found that its emulsion had developed identically to that from cassettes that had been exposed to bright sunlight. Recognizing the importance of his finding, Becquerel announced to the Paris Academy of Science in November 1896 that he had detected the spontaneous emission of rays.[2] The emanations of uranium were initially named Becquerel rays; however, this discovery received surprisingly little attention until consequent work was done by Marie and Pierre Curie. In fact, the Curies coined the term *radioactivity* to describe the phenomenon.

Becquerel conducted much work on radioactivity with the Curies after Marie Curie took an avid interest in Becquerel's report in 1897. In July of 1898, the Curies and Becquerel positively identified a new element and named it *polonium*. In December they identified another and dubbed it *radium*. However, it was not until 1902 that they refined a pure sample, which allowed them to establish the atomic weight of radium as 226. In 1910, Marie Curie purified radium metal in