

Chapter 24

SELECTED TOPICS IN DEPLOYMENT OCCUPATIONAL MEDICINE

PAUL D. SMITH, DO, MPH; DOUG OHLIN, PhD; STEVE SMITH, MD, MPH; AND WILLIAM A. RICE, MD, MPH

HEALTH EFFECTS OF REPEATED IMPACT AND WHOLE-BODY VIBRATION

[PAUL D. SMITH]

- Vibration-Related Illnesses
- Musculoskeletal System
- Cardiovascular System
- Gastrointestinal System
- Neurological System
- Female Reproductive System
- Future Studies and Guidelines

HEARING CONSERVATION [DOUG OHLIN]

- Scope of the Problem
- Importance of Hearing to the Mission
- Program Implementation
- Noise Hazard Identification
- Noise Controls
- Hearing Protective Devices
- Hearing Protector Use in the Field
- Monitoring Audiometry
- Health Education

CARBON MONOXIDE [STEVE SMITH]

- Occupational Exposures in the Military
- Clinical Presentation and Treatment

SMOKE AND OBSCURANTS [WILLIAM A. RICE]

- Tactical Uses
- Delivery Systems
- Hexachloroethane Smoke
- Other Smokes and Obscurants
- Policies and Controls

P. Smith, Colonel, Medical Corps, US Army, Occupational Environmental Medicine Staff Officer, Proponency Officer for Preventive Medicine, Office of The Surgeon General, 5109 Leesburg Pike, 6 Skyline Place, Falls Church, VA 22041-3258

D. Ohlin, Occupational and Environmental Medicine, Hearing Conservation Program, US Army Center for Health Promotion and Preventive Medicine, 5158 Blackhawk Rd., Aberdeen Proving Ground, MD 21010

S. Smith, Site Medical Director, Umatilla Chemical Agent Demilitarization Facility, Umatilla Chemical Depot, 78068 Ordnance Road, Hermiston, OR 97838; formerly, Major, Medical Corps, US Army, Command Surgeon, US Army Industrial Operations Command, ATTN: AMSIO-SG, Rock Island IL 61299-6000

W. Rice, Lieutenant Colonel, Medical Corps, US Army; currently, Division Surgeon, 1st Armored Division, Germany; formerly, Director, Occupational Medicine, US Army Center for Health Promotion and Preventive Medicine-Europe, APO AE 09180

HEALTH EFFECTS OF REPEATED IMPACT AND WHOLE-BODY VIBRATION

With the dawn of the industrial age, human exposures to vibration changed dramatically. Machines of the industrial revolution resulted in vibration exposure to the individual that continues today. Vibration transmitted to humans may be classified into several common types. Whole-body vibration consists of waves of mechanical energy transmitted throughout the human body. It is differentiated from segmental vibration, which is mechanical energy generally concentrated in a segment of the body, such as an arm or leg. Vibration may be further classified by the nature of the mechanical waves. Sinusoidal vibration is characterized by a smooth repetitive cycle, such as we see in the vibrations of an electric motor. Diagrammatically, this type of vibration may be represented by a sine wave. Repeated impact vibration, also known as bump and jolt vibration, is characterized by markedly abrupt accelerations and decelerations that occur in a random and chaotic manner.

Vibration exposure is particularly pronounced in the military setting.¹ Occupants of aircraft are usually exposed to sinusoidal vibration produced by the aircraft's engines. Occupants of military ground vehicles experience a "bump and jolt" vibration as the vehicle crosses rough terrain. Tactical ground vehicles, such as armored personnel carriers and tanks, greatly exceed published exposure limits to vibration according to Griffin's *Handbook of Vibration*.² Vibration should be kept in mind when evaluating soldiers who operate equipment such as tanks, trucks, and armored engineering equipment. Patients may present with a variety of symptoms related to vibration exposure. Vibration as a cause of fatigue, pain, and other complaints should also be considered for aircrew members, particularly those flying modified or new aircraft. Unfortunately, vibration in military ground vehicles has not been adequately studied. The following section presents a review of the signs, symptoms, and health effects of vibration exposure.

Vibration-Related Illnesses

General Effects

It should be noted that nearly all illness and injury seen in vibration-exposed patients are not unique to vibration alone. Vibration is felt to be a cofactor in the development of certain diseases or is believed to accelerate certain disease processes, such as low back degenerative disease. This is well

reviewed by Dupuis.³ Such conditions are classified as an occupational illness in some European nations and by the International Labor Organization.

Vibration affects multiple body systems (Table 24-1). The effects of vibration on the musculoskeletal and gastrointestinal systems are well documented, with large-scale epidemiologic studies demonstrating increases in low back pain and esophageal dysmotility.²⁻⁶ The cardiovascular and nervous systems are also affected. Whole-body vibration, including the bump and jolt variety common in the Army and Marine Corps, acts as a general body stressor. Exposed subjects experience an overall increase in adrenaline levels.⁷ In addition, there is a general activation of the sympathetic nervous system with continued vibration exposure.^{7,8} These result in sustained muscular and cardiovascular activity with concomitant fatigue. Cortisol excretion is also increased during vibration exposure, further indicating that this exposure acts as a general body stressor and perhaps results in reduced immunity.⁹⁻¹¹

Musculoskeletal System

The human spine and supporting structures are sensitive to vibration. The spine itself may be damaged by repeated impact or from vibration that matches the spine's resonant frequencies. The lumbar spine, for instance, resonates at approximately 4 to 6 Hz.¹² These frequencies of vibration are commonly encountered in all vehicles. Vibration causes reduced disc nutrition and elevations in hydroxyproline that indicate damage to cartilage and vertebral end plates.^{2,3,13-16} Electromyography of vibration-exposed subjects reveals increases in motor activity until fatigue ensues.¹⁷⁻¹⁹ Back pain has been documented in truck drivers and train operators. This is, presumably, related to fatigue from the increase in motor activity during vibration exposure. It is likely that the same effect exists in troops using tactical ground vehicles, although this remains to be studied.

Vibration and repeated impact should be considered when seeing troops with back injury who are operators of tactical ground vehicles and truck drivers.^{4,5,20-22} In one instance, back pain and hematuria were associated with testing of the rapid response vehicle, a dune buggy modified for military use.²³ Back pain and hematuria in exposed personnel should bring vibration exposure to mind after ruling out ureterolithiasis. These symptoms and signs

TABLE 24-1

PHYSIOLOGICAL AND HEALTH EFFECTS OF REPEATED IMPACT AND WHOLE-BODY VIBRATION BY MAJOR SYSTEM

| Body System | Physiological/Health Effect | References |
|------------------|--|------------|
| Whole-body | Increased adrenaline cortisone | a-d |
| Musculoskeletal | Vertebral end plate, vertebral disc deterioration, back pain | e-r |
| Cardiovascular | Increased blood pressure, increase heart rate, hand-arm vibration syndrome, increased blood lipids | a,e,s |
| Hematological | Increased hematocrit, decreased hemoglobin, increased haptoglobin, pooling in extremities | a,b,e |
| Gastrointestinal | Esophageal dysmotility, peptic ulcer disease, gastrointestinal bleeding | e,p,q,t-w |
| Nervous System | Altered neurotransmitters, dorsal root ganglia | x-cc |
| Reproductive | Abortion, altered menses | dd,ee |

Sources:

- a. Johanning E. Back disorders and health problems among subway train operators exposed to whole body vibration. *Scand J Work Environ Health*. 1991;17:414-419.
- b. Johanning E, Wilder DG, Landrigan PJ, Pope MH. Whole-body exposure in subway cars and review of adverse health effects. *J Occup Med*. 1991;33:605-612.
- c. Daleva M, Piperova-Dalbokova D, Hadjiolova I, Mincheva L. Changes in the excretion of corticosteroids and catecholamines in tractor drivers. *Int Arch Occup Environ Health*. 1982;49:345-352.
- d. Kamenskii YN, Nosova IM. Effects of whole body vibration on certain indicators of neuro-endocrine processes. *Noise Vibration Bull*. 1989;Sept:205-207.
- e. Initial Health Hazard Assessment Report on the Fast Attack Vehicle (FAV). Aberdeen Proving Ground, Md: Environmental Hygiene Agency; 1984.1. Griffin MJ. *Handbook of Human Vibration*. London: Academic Press Ltd; 1988. RCS Med 388.
- f. Dupuis H. Medical and occupational preconditions for vibration induced spinal disorders: Occupational disease number 2110 in Germany. *Int Arch Occup Environ Health*. 1994;66(5):303-308.
- g. Nakamura R, Moroji T, Nohara S, Nakamura M, Okada A. Activation of cerebral dopaminergic systems by noise and whole-body vibration. *Environ Res*. 1992;57(1):10-18.
- h. Barron JL, Noakes TD, Levy W, Smith C, Millar RP. Hypothalamic dysfunction in overtrained athletes. *J Clin Endocrinol Metab*. 1985;60:803-806.
- i. Ariizumi M, Okada A. Effect of whole body vibration on the rat brain content of serotonin and plasma corticosterone. *Eur J Applied Physiol Occup Physiol*. 1983;52:15-19.
- j. Granjean E. *Fitting the Task to the Man*. New York: Little Brown; 1989.
- k. Pope MH, Hansson TH. Vibration of the spine and low back pain. *Clin Orthop*. 1992;279:49-59.
- l. Pope MH, Kaigle AM, Magnusson M, Broman H, Hansson T. Intervertebral motion during vibration. *Proc Inst Mech Eng [H]*. 1991;205(1):39-44.
- m. Ishihara H, Tsuji H, Hirano N, Ohshima H, Terahata N. Effects of continuous quantitative vibration on rheologic and biological behaviors of the intervertebral disc. *Spine*. 1992;17(3 suppl):S7-S12.
- n. Hansson T, Holm S. Clinical implications of vibration-induced changes in the lumbar spine. *Orthop Clin North Am*. 1991;22:247-253.
- o. Kaji H, Sato E, Nagatsuka S, et al. Evaluation of sensory disturbances using short latency somatosensory evoked potentials (SSEPs) in vibration-exposed workers. [Japanese]. *Sangyo Igaku*. 1991;33:605-612.
- p. DeLuca CJ. Myoelectrical manifestations of localized muscular fatigue in humans. *Crit Rev Biomech Eng*. 1985;4:251-278.
- q. Klein AB, Snyder-Mackler L, Roy SH, DeLuca CJ. Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. *Physical Therapy*. 1991;71(6):445-454.
- r. USAARL Contract Reports No. CR 95-1, CR 95-2, CR 95-3, CR 95-4.
- s. Boshulzen HC, Bongers PM, Hulshof CT. Self-reported back pain in fork-lift truck and freight-container tractor drivers exposed to whole-body vibration. *Spine*. 1992;17:59-65.
- t. Chatterjee S, Bandyopadhyay A. Effect of vibrating steering on the grip strength in heavy vehicle drivers. *J Human Ergology*. 1991;20(1):77-84.
- u. Gow BS, Legg MJ, Yu W, Kukongviriyapan U, Lee LL. Does vibration cause post-stenotic dilatation in vivo and influence atherogenesis in cholesterol-fed rabbits? *J Biomech Eng*. 1992;114(1):20-25.
- v. Nakamura H, Katoh A, Nohara S, Nakamura M, Okada A. Experimental studies on the pathogenesis of the gastric mucosal lesions induced by whole-body vibration. *Environmental Research*. 1992;58(2):220-229.
- w. Katoh A, Nakamura H, Nohara S, Ohmura K, Munemoto Y, Oda M. Experimental studies on the development of acute gastric lesions induced by vibration stress in rats and its pathogenic mechanisms. *Japanese J Gastroenterol*. 1992;89:469-476.
- x. Kabacinska-Knapikowa D, Paradowski L, Kwiatkowski S. Esophageal motility in personnel operating heavy self-propelled mining machines. *Materia Medica Polona*. 1992;24(3):153-155.
- y. Mecel H, Wozniak H, Sun L, Frazier E, Mason HC. Effects on rats of exposure to heat and vibration. *J Applied Physiol*. 1962;17:759-762.
- z. Nakamura H, Moroji T, Naase H, Okazawa T, Okada A. Changes of cerebral vasoactive intestinal polypeptide- and somatostatin-like immunoreactivity induced by noise and whole-body vibration in the rat. *Eur J Appl Physiol Occup Physiol*. 1994;68(1):62-67.
- aa. McLain RF, Weinstein JN. Nuclear clefting in dorsal root ganglion neurons: A response to whole body vibration. *J Comp Neurol*. 1992;322(4):538-547.
- bb. McLain RF, Weinstein JN. Ultrastructural changes in the dorsal root ganglion associated with whole body vibration. *J Spinal Disord*. 1991;4(2):142-148.
- cc. McLain RF, Weinstein JN. Effects of whole body vibration on dorsal root ganglion neurons: Changes in neuronal nuclei. *Spine*. 1994;19:1455-1461.
- dd. Nakamura H, Moroji T, Nohara S, Nakamura M, Okada A. Activation of cerebral dopaminergic systems by noise and whole-body vibration. *Environ Res*. 1992;57(1):10-18.
- ee. Bartsch C, Meihle B. The effect of vibrations in the early stages of embryogenesis on the postnatal motor and physical development of Wistar rats [German]. *Anat Anz*. 1991;173(4):239-242.

should clear once the patient's exposure to vibration has stopped.

Cardiovascular System

The cardiovascular response to vibration is well studied. This system reacts to vibration as it does to any stress, with an increase in heart rate and blood pressure. Several limited Scandinavian studies indicate a rise in blood lipids.^{3,24} Additionally, vibration results in hand-arm vibration syndrome, in which segmental vibration results in endothelial damage to the small arterioles. Patients present with vasospasm of the digits, especially in response to cold.

Gastrointestinal System

Studies reveal an increase in motility disorders in vibration-exposed workers. The prevalence of gastroesophageal reflux disease increases, and in experimental animals gastrointestinal bleeding is produced by vibration exposure.^{5,6,25-27} In troops presenting with symptoms of reflux esophagitis, gastroesophageal reflux, or intestinal bleeding, the differential diagnosis should include repeated impact vibration in the field. Although not proven by studies, reducing that exposure where practical may result in a reduction or cessation of the symptoms.

Neurological System

The peripheral and central nervous systems are both affected by vibration. Animal research has demonstrated an increase in vasointestinal peptide

and a decrease in substance P, possibly caused by a pain response.²⁸ In the dorsal root ganglia, nuclear clefting is noted after vibration exposure, denoting injury to the peripheral nervous system.²⁹⁻³¹ In the central nervous system, alterations of dopamine levels are described in experimental animals, although this has not been studied in humans.³²

Female Reproductive System

The reproductive system may be adversely affected by vibration exposure. Case studies dating to the 1950s and 1960s report spontaneous abortions in women exposed to vibration in their work. Menstrual abnormalities may be increased in vibration-exposed women.^{33,34} No studies are available at present to support or refute this effect in female service members exposed to vibration.

Future Studies and Guidelines

Adequate guidelines for vibration exposure in military ground vehicles are under development. Sinusoidal vibration is of heightened concern to the Air Force and is studied at Wright Patterson Air Force Base, Ohio. Repeated impact vibration in ground vehicles is of concern principally to the Army and Marine Corps and is actively being investigated by the US Army Aeromedical Research Lab, Fort Rucker, Ala. These efforts should result in better guidelines for controlling vibration exposure in troops by providing needed information on health effects and relating these effects to vibration levels.³⁴

[Paul D. Smith]

HEARING CONSERVATION

Scope of the Problem

Hazardous noise pervades the military environment. Moreover, the increasing demand for greater range, firepower, and speed all translate into higher and more hazardous noise levels. In 1994, among US Army enlisted combat arms (infantry, artillery, and armor), 25.5% of soldiers with more than 17.5 years of service had a significant hearing loss. Of those with 12.5 to 17.5 years, it was 15.3%. Among soldiers with less than 2.5 years, only 5.4% had a hearing loss.³⁵ Although still a problem of sizable concern, this is significant progress since 1974 when, for the same lengths of service, the prevalences of hearing loss were 54.2%, 47.8%, and 11.5%, respectively.³⁶

One training session on a firing range can wreak

havoc on unprotected ears. It is possible for a soldier to emerge from basic training with a significant hearing loss. Since the majority of noise exposure over a soldier's career is the result of routine training exercises, most noise-induced hearing loss is preventable by implementing protective measures during training.

Importance of Hearing to the Mission

Acute hearing (in both ears) is a 360° warning sense, whereas vision is only slightly better than 180°. Soldiers can identify an enemy's location by certain sounds: closing rifle bolts, loading cartridges, engaging or disengaging safety locks, clipping barbed wire, and footsteps³⁷ (Figure 24-1).

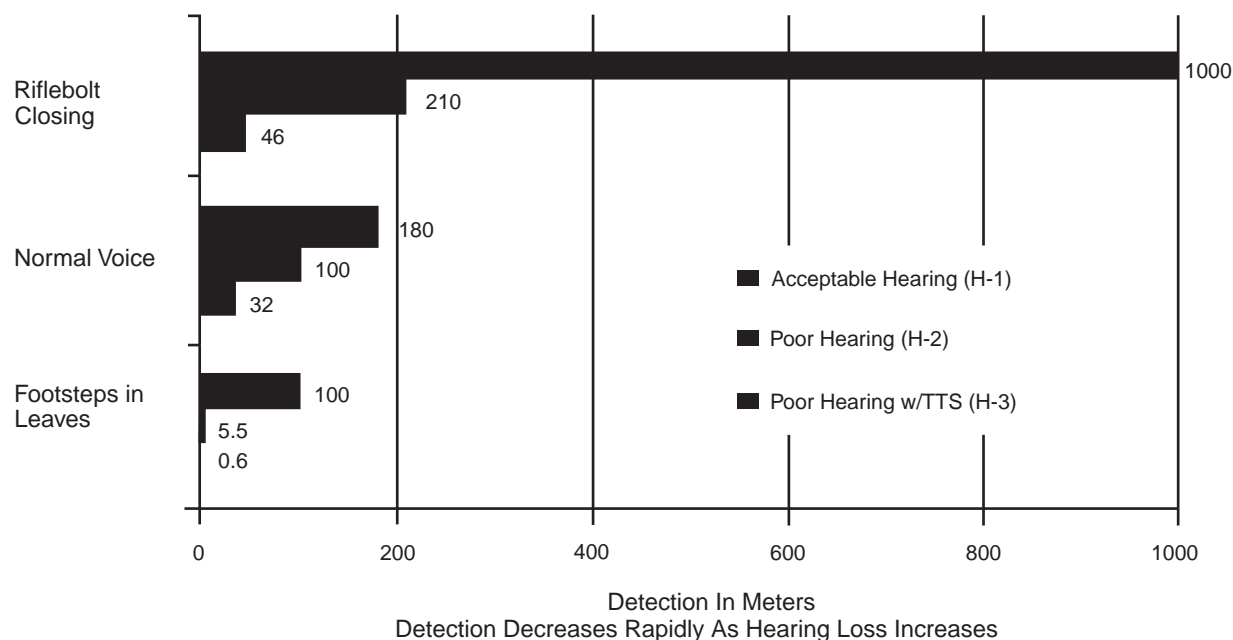


Fig. 24-1. Detection of Sound by Hearing Ability

Data source: Price GR, Kalb JT, Garinther GR. Toward a measure of auditory handicap in the Army. *Ann Otol Rhinol Laryngol Suppl.* 1989;140:42-52.

Good hearing also enables soldiers to discriminate between friendly and enemy fire: the different acoustic signatures of M-16 and AK-47 rifles, for example. In addition, soldiers with good hearing have less trouble understanding radio transmissions over noisy communication systems. The Army Research Laboratory, Aberdeen Proving Ground, Md, has produced convincing data that have linked communication ability to performance among experienced tank crews.³⁸

Program Implementation

Hearing conservation program requirements and procedures are outlined in Department of the Army Pamphlet 40-501.³⁹ Detailed implementation procedures and technical guidance are included in various technical guides available from the US Army Center for Health Promotion and Preventive Medicine (USACHPPM), 5158 Blackhawk Road, ATTN: MCHB-TS-CHC, Aberdeen Proving Ground, MD 21010-5403.⁴⁰⁻⁴⁴

Noise Hazard Identification

For the purposes of administering the Hearing Conservation Program, a steady noise of 85 decibels (dB)A3 or greater is considered hazardous regardless of duration of exposure. Examples of

steady-state noise include generators, tracked vehicle interiors and exteriors, and aircraft. In the absence of noise data or hazard posting, there is a simple rule of thumb for determining the presence of a steady-state noise hazard if one has to raise one's voice to be heard at a distance of 1 m.

All small- and large-caliber weapons (including blanks) exceed the peak (P) impulse noise criteria of 140 dB peak (dBP). The use of hearing protection in the vicinity of weapon systems must take into account the requirement to use hearing protection within a 140 dBP contour. For small arms, this contour will extend back from the firer's position approximately 15 m. Larger-caliber weapons and shoulder-fired rockets will have much larger contours behind the firing point, depending on type of round, level of charge, and firing elevation.⁴⁴

A direct, positive correlation has been observed between the posting of hazard signs and the use of hearing protection. At no time, however, should the exteriors of tactical weapon systems be posted.

Noise Controls

Engineering controls can reduce or eliminate the noise hazard and the need for other hearing conservation measures. Although such intervention opportunities are rare in the military, exploiting

them can offer tactical advantages, as well as a reduction or elimination of a noise hazard. For example, the blast attenuating device on the end of the large-caliber mortar not only reduces the noise level at the crews' ears, but the device also makes the mortar easier to load and adds approximately 1,000 m to its range. The tactically quiet generator affords the advantages of hazard elimination and reduced detectability³⁷ (Figure 24-2).

Administrative controls are employed when hearing protection cannot protect personnel from a given exposure. Weapons system operator manuals should be consulted for any limitations on the number of rounds permitted and any time limits on noise exposure.

Hearing Protective Devices

Hearing protective devices are issued to US military personnel. The user is given a choice from among

approved protectors unless there is a medical or environmental contraindication. Approved hearing protectors are listed in Department of the Army Pamphlet 40-501.³⁹

Noise muffs are a safety item and may be purchased using a National Stock Number (NSN) or as a local purchase item. Earplugs, however, are a medical item. Choices are limited to two types of pre-formed (triple- and single-flange) and a hand-formed foam. Two exceptions to the restricted use of earplugs are custom-molded earplugs for individuals who cannot be properly fitted with approved devices and musician's earplugs for band members.

Single- and triple-flange earplugs must be fitted under medical supervision. The hand-formed earplugs can be stocked in noise-hazardous areas (eg, firing ranges, airfields) for transient personnel or for other personnel without their issued protectors. Local commanders can authorize wearing earplugs and

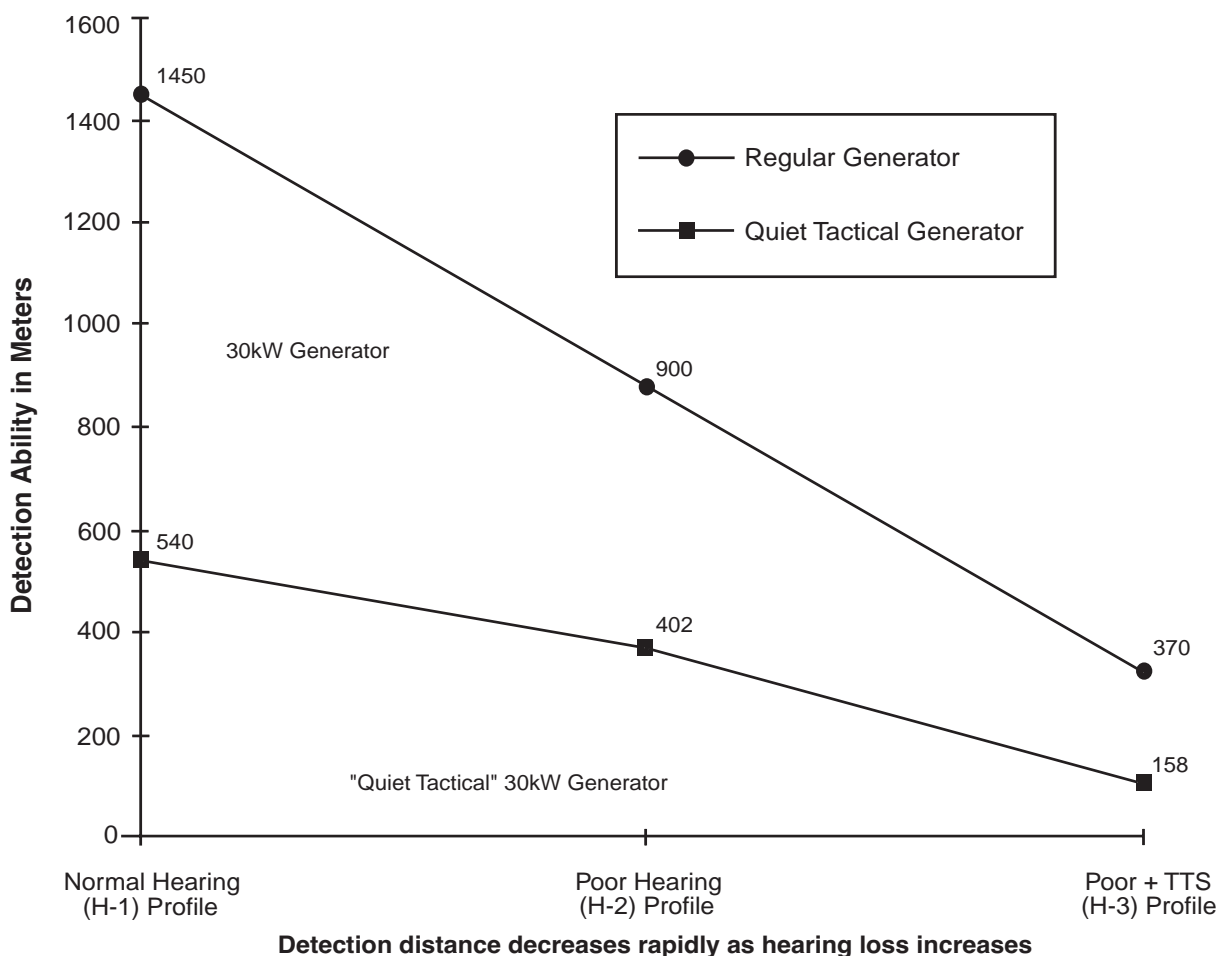


Fig. 24-2. Detectability of Generator Noise as a Function of Design and Hearing Ability
Data source: Army Research Laboratory, Aberdeen Proving Ground, Md.

the earplug carrying case on the Battle Dress Uniform and are strongly encouraged to do so.⁴⁵ The military-approved earplugs have been thoroughly tested for their noise reduction capabilities and freedom from toxic effects. Not all earplugs can make such claims. The approved foam earplug is made of a polyvinyl, closed-cell foam. Polyvinyl foam will stay rolled down long enough for proper insertion even in humid environments.

The earplug carrying case also has unique features for military applications. The olive-drab translucent color and matte finish is designed not to reflect light while allowing the contents of the case to be seen. Seating and insertion devices for the single- and triple-flange plugs are incorporated into the case lid.

Hearing Protector Use in the Field

Measures for protection against steady-state noise are the same as for impulsive noise. Hearing protection devices should be worn when the steady-state noise is above 85 dB and when impulse noise exceeds 140 dBP sound pressure level. All small arms used in the military produce impulse noise above this level. Therefore, hearing protection must always be worn on firing ranges and during field firing exercises and other forms of weapons training practice or evaluation.

Hearing protection can significantly degrade communication ability when protection is needed the most, that is, during weapons firing. During the relatively quiet periods between high-level impulse noise exposures, hearing protection can interfere with communication. Loud speaker systems at fixed firing points are designed to overcome the sound attenuation of the hearing protectors and ensure that voice signals are received at sound levels no lower than those of conversational speech. For best results, loud speakers must be spaced at appropriate intervals to maximize the benefits of amplification.

Such amplification is also effective on close combat courses and grenade ranges when standard commands and instructions are used.^{46,47} These recommendations are limited to operations in wind speeds of less than 25 miles per hour. On grenade ranges, hearing protection can be waived for pit noncommissioned officers if they insert their fingers into their ears as soon as the tower announces "frag out."⁴⁶

A new earplug has been developed in partnership with the French Army that addresses these communication issues for the dismounted soldier or Marine. The combat arms earplug affords protection from weapons fire over a wide range (up to 190 dBp) while marginally interfering with voice communications and the detection and localization of environmental (combat) sounds.

The filter inserted in the stem of the earplug is "nonlinear." Noise reduction capability increases with the noise level of the weapons fire. At low sound levels, however, there is little or no sound reduction and speech is understood without shouting. A detection model, developed at the Army Research Laboratory predicts that a normal-hearing soldier can detect a truck at the same distance (800 m) with or without the combat arms earplug. That detection capability is cut in half (400 m) with conventional foam plugs. The double-plug design includes a solid linear earplug that can be used for steady-state noise, such as in helicopters or armored personnel carriers.

Blank rounds fired from M-16 rifles, although not as noise-hazardous as live ball ammunition, are still hazardous to hearing. Blank suppressors attached to rifle muffles can reduce noise levels 4 to 7 dB, for example, from 147 to 143 dBP.

This does not imply that hearing protection should be worn when actually in combat, except where weapons are fired from positions out of the immediate zone of fighting. Even in the immediate zone of fighting, however, hearing protection may sometimes be advantageous. The effective loss of hearing produced by use of a hearing protector can be quickly eliminated by removal of the protector. On the other hand, the loss of hearing produced by the action of noise on the unprotected ear could be permanent or, at best, can require many hours, sometimes even days, before recovery of hearing sensitivity.

The use of hearing protection during transport in tracked-wheeled personnel carriers, trucks, helicopters, or other aircraft should be enforced as a practical and feasible procedure for enhancing communication during transport and for preserving hearing for both training or wartime missions. 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.⁴² The result was that the troops arrived at the prison camp, removed their earplugs, and found their hearing unimpaired from the noise of the helicopters. This is not an isolated example. Most soldiers appreciate that under these conditions, earplugs function like sunglasses that cut down excess glare. First Lieutenant Clarence Briggs told of the Panama fight:

Our CH-47 [helicopter] descended rapidly, the-ramp dropped open and we charged out... Suddenly we stopped. Troops were quickly coming up behind us, and I could barely hear Crittenden yelling! "They landed in pairs!" He was trying to tell me that the two Hueys had landed 10 minutes before us instead of just 30 seconds, as had been the plan. I couldn't

hear. My ears were deafened by the noise of the CH-47's engines. "Next time we'll wear earplugs," I thought. "This craziness might get us killed." Before I knew it, CPT Dyer was kneeling next to me. We yelled at each other, unable to hear anything. Somehow we started to move toward the edge of the golf course, where 1st Platoon was securing the linkup site. Talking on the radio was useless. We blundered into them and moved into the bush just before the railroad tracks.⁴⁸

Monitoring Audiometry

Through detection of small decrements in hearing sensitivity, individuals susceptible to noise can be identified before their hearing loss becomes a social or occupational handicap. All monitoring audiometry in the Army's Hearing Conservation Program is conducted through the Defense Occupational and Environmental Health Readiness System in Hearing Conservation (DOEHRS-HC) application, which will be integrated into the Composite Health Care System II (CHCS II). The DOEHRS-HC provides automated

hearing testing, as well as data for installation and Army-wide program management.

Health Education

Since noise-induced hearing loss is a relatively painless and bloodless process, an individual will not always be aware of the damage incurred until the loss has reached moderate-to-severe levels. Effective health education is essential to reinforce the faithful and proper use of hearing protection. The widespread use of earplugs presents a special challenge. Unless an expert performs a visual inspection of the earplug in the ear canal and tests the earplug stem or tab for tension, only the user knows whether or not the earplugs are properly inserted. Hence, there is an imperative that the user be convinced that hearing protection must be used and know how to maintain and insert them properly. Training aids, unique to the military and designed for this purpose, are available through publication and audiovisual support centers.³⁹

[Doug Ohlin]

CARBON MONOXIDE

Carbon monoxide's high toxicity and extensive exposure potential have made it one of the most widespread poisons in the world, the most significant toxic gas in the workplace, and another element of danger for the service member. Carbon monoxide is toxic by several mechanisms, though principally by carboxy-hemoglobin formation—an affinity for hemoglobin well known since the last century. Manifestations of its toxicity run from mild headaches to coma and death (Table 24-2).

Carbon monoxide poisoning has manifested itself in every modern armed conflict. In World War I, the French identified propellants, combustion engines, and explosives as CO sources that created casualties in enclosed spaces. In World War II, carbon monoxide policy in the US Army started at the Armored Medical Research Laboratory, Fort Knox,⁴⁹ when scientists developed an infrared gas analyzer to study carbon monoxide levels in armored vehicles. World War II also saw the first study incriminating low levels of carbon monoxide as a cause of neurobehavioral defect.⁵⁰

Operational Exposures in the Military

Today, the army is literally "an army on wheels." Armored fighting vehicles move more soldiers, have bigger engines, burn more propellant throwing bigger projectiles faster, and are sealed against the external environment, allowing fumes and gases to accumulate. Improved bore extraction and ve-

hicle ventilation systems have reduced the incidence of carbon monoxide intoxication, but there continue to be intermittent occurrences.

The foot soldier is not immune from carbon monoxide intoxication. He or she may be exposed to it while in transport, whether in a "HumVee," "Bradley Fighting Machine", "deuce and a half," Blackhawk, Chinook, or US Air Force transport; while operating weapon systems including machine guns, rocket launchers, and recoilless rifles; and even while sleeping in tents, buildings, or vehicles heated with combustion heaters or charcoal. There is additional risk in enclosed spaces. Service members cannot smell it. It has no color. Chemical masks will not protect against it. Only a knowledge of its sources and continuous vigilance may preserve the service member from intoxication or demise.

Clinical Presentation and Treatment

In the initial evaluation, mild effects are often incorrectly attributed to mild viral syndromes or food poisoning. Failure to identify the carbon monoxide source may lead to more serious presentations or even fatalities. Unexpected symptoms of "inebriation" may actually be advanced carbon monoxide intoxication, requiring immediate intervention to prevent further or permanent injury to the service member or loss of an entire weapon sys-

TABLE 24-2
MANIFESTATIONS OF CARBON MONOXIDE TOXICITY

| 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 | worsening symptoms, increased possibility of collapse and syncope, increased respiration and pulse |
| 50–60 | syncope, increased respiration and pulse, coma with intermittent convulsions, Cheyne-Stokes' 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: Weyandt TB, Ridgeley CD Jr. Carbon monoxide. Deeter DP, Gaydos JC, eds. *Occupational Health: The Soldier and the Industrial Base*. Part III, Vol 3. In: *Textbook of Military Medicine*. Washington, DC: Office of The Surgeon General; 1993: 412.

tem. When available, carboxyhemoglobin determinations (or breath carbon monoxide measurement) should be made if carbon monoxide intoxication is even remotely suspected.⁵¹

Intervention begins with recognition of the hazard and removal from the exposure. In the absence of hyperbaric capabilities, supplemental oxygen will assist in recovery. Hyperbaric oxygen is preferred to reduce the incidence of late sequelae, recognized as a problem in serious incidents of carbon monoxide intoxication and even with apparently less-severe intoxications. Sequelae can range from minor psychoneural deficits (eg, slight memory loss, fine tremors) to visual, hearing, IQ, and motor deficits. Hyperbaric therapy also reduces recovery time to a fraction of that required for supplemental oxygen therapy. Although its availability is limited in battlefield situations, it is increasingly available for treatment of intoxication resulting from peacetime training activities in the United States and even overseas.

While there is still disagreement as to what levels of carboxyhemoglobin first lead to measurable impairment, current Army policy (first established in 1981) calls for aviators to be below 5% and the

general Army population to be below 10%.⁵² Military vehicles and weapon systems are usually designed with this in mind. Evidence in the literature suggests that the majority of the force would experience no measurable impact at levels of up to 15% to 20%.⁵³ Unfortunately, there may be subpopulations who experience impairment at levels below 5%.⁵⁴ What the PM community should do with this information is undecided. To date, the 5% and 10% standards remain in effect, and there is no initiative to change them.

Carbon monoxide has been a significant problem for 20th century armies. A more complete account of the US Army and carbon monoxide appears in another volume of the *Textbooks of Military Medicine* series.⁵⁵ That chapter provides a detailed history of carbon monoxide experiences of modern armies. The chapter also reports an important equation—the Coburn-Forster-Kane equation—used by engineers designing weapon systems for estimating carboxyhemoglobin values from known concentrations of carbon monoxide. The reader is cautioned to consult the article by Smith and colleagues,⁵⁶ before attempting to use this equation.

[Steve Smith]

SMOKE AND OBSCURANTS

On perceived or actual attack by a predator, an octopus will release a cloud of ink into the water to conceal it from its attacker, while it escapes. Humans have developed a tactic that is similar to that

of the octopus by the use of smoke on the battlefield. Since the 1930s, the US Army has extensively used smokes and obscurants during training and combat. Smokes are products produced by burn-

ing or vaporizing substances such as oils and organic fuels. Obscurants are suspended particles, such as fog, mist, and dust, that block or weaken transmission of a part or parts of the electromagnetic spectrum, such as infrared and visible radiation or microwaves.⁵⁷ Their uses have included obscuration, screening, deception, identification, and signaling. During World War II, the strategic and tactical use of smoke was advanced to a point never before attained in warfare. Using newly developed smoke devices, it was possible to screen whole cities from the sight of enemy aircraft for the first time in history. Laying down smoke in strategic areas was effective in keeping down the number of casualties and in greatly reducing the effectiveness of enemy air operations.⁵⁸

Throughout the modern battlefield, forces acquire and engage targets based on visual, infrared, and millimeter wave technologies. To disrupt these engagements, several types of smokes and obscurants have been developed; each targets particular portions of the electromagnetic spectrum to disrupt enemy surveillance, target acquisition, and weapons guidance systems. The effectiveness of each smoke depends on its ability to reflect, refract, and scatter light rays to obscure visibility. For this reason, all military smokes consist of aerosols with particle dimensions approximating the wavelength of the portion of the magnetic spectrum that they are designed to obscure.⁵⁹ The list of smokes and obscurants is long and includes hexachloroethane, fog oil, diesel oil, white and red phosphorus, colored smokes, brass, graphite, titanium dioxide, and terephthalic acid. Depending upon the smoke or obscurant being used and its intended purpose, these may be delivered by projectiles, grenades, smoke pots, or smoke generators.

Tactical Uses

Smokes and obscurants can be used in either hasty or deliberate operations. Hasty smoke covers a small area for a short period of time. Deliberate smoke is usually employed over large areas for extended periods and is planned at the brigade level or higher.⁶⁰

Smoke is used in four ways: to obscure, to screen, to deceive, and to identify or signal. Smoke that is referred to as "obscuring" smoke is used on enemy positions to degrade their sighting capabilities and their command and control. In contrast to this, "screening" smoke is used in friendly operational areas or between friendly and enemy forces. Smokes can also be used to deceive the enemy; this is especially useful in defensive retrograde troop movements. Colored smokes, to include white smokes,

are used for signaling and identification purposes only, such as marking a landing zone or communicating pre-arranged signals (Figure 24-3).

There are three types of smoke screens: smoke blankets, smoke hazes, and smoke curtains. A smoke blanket is a dense and horizontal development of smoke in friendly operational areas or between friendly and enemy forces. It is used to screen friendly forces from enemy ground and aerial observation. However, the high density of smoke used in a smoke blanket can hamper friendly operations (Figure 24-4). In contrast to a smoke blanket, a smoke haze is only a light concentration of smoke. It is used to restrict the accuracy of enemy observation and fire but it is not dense enough to disrupt friendly operations. A smoke curtain is a dense, vertical development placed between enemy and friendly forces. It restricts ground observation but not aerial observation (Figure 24-5).

US forces will use smokes and obscurants whenever the tactical advantage to be gained outweighs the potential degradation to friendly operations. When smoke is used in the above manner, the smoke will deny information to the enemy, reduce the enemy's effectiveness in target acquisition, create conditions to surprise the enemy, deceive the enemy, disrupt organized enemy movement, operations, and command and control, and restrict nap-of-the-earth (NOE) and contour approaches by enemy aircraft. NOE flights occur when pilots fly as close to the terrain as the speed and performance contours of the aircraft will allow.



Fig. 24-3. In conditions where verbal communication is difficult, such as when being masked, colored smokes such as the violet seen here are sometimes used for communicating prearranged signals.

Photograph: Courtesy of Commander, US Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Md. Photographer: W. Ben Bunger III.



Fig. 24-4. This is an example of a smoke blanket being produced by HMMWV-mounted smoke generators vaporizing fog oil. Over a friendly area, this would prevent observation by the enemy from both the ground and the air. A lighter density of smoke—a smoke haze—would make operations within the smoke easier.

Photograph: Courtesy of Commander, US Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Md.

Delivery Systems

There are several ways in which to employ smoke on the battlefield. These include projectiles, smoke grenades, smoke pots, smoke generators, vehicle engine exhausts. Smoke can come via air and sea. Methods of projectile employment include mortars,



Fig. 24-5. This is an M113 track vehicle with smoke generators mounted on it. It can quickly develop a long smoke curtain.

Photograph: Courtesy of Commander, US Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Md.

field artillery, tank guns, and grenade launchers. Grenade launchers can be mounted on armored vehicles. These grenade launchers fire eight or twelve grenades simultaneously, with firing arcs of 90° to 110°. The vehicle commander launches the grenades as soon as the enemy fires on the vehicle. The individual smoke clouds from each of the eight or twelve grenades quickly coalesce into one continuous smoke curtain. Evasive action may then be taken behind that curtain (Figure 24-6).

The smoke grenades are classified according to the color of their smokes. The M8 grenade contains hexachloroethane and so produces a white smoke. The M18 grenades produce red, green, yellow, and violet smokes.

There are several types of smoke pots, and each has different capabilities. The M1 and M5 smoke pots differ only in their weights and thus their burn times. The M1 smoke pot weighs 10 lbs and lasts 5 to 8 minutes, while the M5 smoke pot weighs 30 lbs and has a burn time of 12 to 22 minutes. The M4A2 and the M207A1 smoke pots weigh 30 lbs and have fuzes so that they can be delivered by helicopter. The M4A2 smoke pot floats, so that it can be used in water operations.

The M3 smoke generator, which is for use over large areas, vaporizes fog oil. The generator burns about 40 gallons of fog oil per hour. The M56 turbine generator is under development. It can be mounted on a HMMWV (High Mobility Multipurpose Wheeled Vehicle). It will be able to produce a cloud that will obscure both outside and within the visible spectrum by burning fog oil and grinding and blowing graphite particles out of an ejector (Figure 24-7).

The vehicle engine exhaust smoke system is mounted on M1 and M60 series tanks. The engine's fuel pump injects about 1 gallon of diesel fuel per minute into the hot engine exhaust system, causing the fuel to flash-vaporize and exit with the engine's exhaust gases. However, the Army has switched to JP8 as the standard vehicle fuel, and it produces inadequate obscuration due to its high volatility. To overcome this problem, the Army has developed a bolt-on reservoir for fog oil that mounts on the rear of the M1 Abrams tank. Fog oil is pumped from the reservoir using existing VEES hardware into the M1's exhaust.

Hexachloroethane Smoke

The hexachloroethane-based smoke munition is referred to as "HC smoke." The precursor to the HC smoke munition was developed by Captain E.F. Berger of the French Army. Berger's mixture con-



Fig. 24-6. This is a smoke curtain being developed by a grenade launcher mounted on an armed vehicle. These eight clouds will quickly coalesce into one continuous smoke curtain.

Photograph: Courtesy of Commander, US Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Md.

tained carbon tetrachloride, powdered zinc, and zinc oxide. When ignited, it produced an aerosol of zinc chloride and sooty carbon particles. Between the two world wars, the US Army's Chemical Warfare Service made several refinements of Berger's mixture. The first refinement replaced the volatile



Fig. 24-7. This is a ground-level view of the smoke blanket shown in Figure 24-4. The blanket is being developed by a HMMWV-mounted M56 turbine generator. This generator is under development and will be able to produce a multispectral cloud by burning fog oil and grinding and blowing graphite particles out of an ejector. Photograph: Courtesy of Commander, US Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, Md.

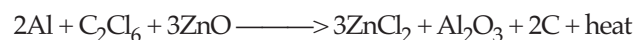
liquid carbon tetrachloride with hexa-chloroethane, which is a solid at room temperature. Later changes in the chemical mixture were made to overcome difficulties caused by the limited availability of some of the chemical ingredients and the explosive nature of others, leading to the Type C composition widely employed today in HC smoke-producing devices.⁵⁹

HC smoke pots are ordinarily used to provide screens for small areas, to supplement other smoke sources by filling holes in screens, and to help rapidly establish screens. HC smoke pots were used extensively in World War II to provide preliminary screens during the approximately 5 minutes needed to set up large thermo-mechanical fog oil smoke generators. In addition, HC smoke pots were used by the US Army and Navy to cover harbors in Italy and North Africa and in both the European and Pacific theaters. They were used on land and water to screen supply routes, bridge construction, amphibious operations, tanks, ammunition dumps, troop concentrations, and ground operations. They were also used to hide mortar flash.⁵⁹ In the assault on Salerno, Italy, on September 9, 1943, the first infantry waves burned HC smoke pots, and a chemical smoke unit landed with the mission of concealing the beaches from aerial observation. For the first few days, until the mechanical generators arrived, the screens were made and maintained only with smoke pots. Although the Salerno beaches were under constant enemy air attack and observed artillery fire for nearly 2 weeks, the German fire could not be directed accurately to a single target on or off the beach. Priceless lives and vital equipment were saved, and smoke as an instrument of assault had proved itself.⁵⁸

Although fog oil is the most widely used smoke, the smoke munition that has caused the most service member morbidity and mortality is the HC smoke munition. Between 1945 and 1993, many individuals have been hospitalized and have died due to exposure to HC smoke.⁶¹ These cases have involved the use of HC smoke in enclosed spaces or in extremely close proximity. The morbidity and mortality have been largely caused by pulmonary edema or secondary bronchopneumonia. Zinc chloride, a combustion product of the HC smoke munitions, is the most likely cause of these health effects.

The health hazards to the service member using the HC smoke munitions can be disastrous, and physicians practicing acute care near military installations need to be aware of the importance of taking a good exposure history, the time course to the development of symptoms and pathology, and the acute treatment.

Uncombusted Type C composition HC munitions contain approximately 7% grained aluminum and roughly equal parts of hexachloroethane and zinc oxide. The HC munition is ignited by a pyrotechnic starter and is then self-perpetuating. Below is the ideal combustion equation in which the aluminum, hexachloroethane, and zinc oxide are converted into zinc chloride, aluminum oxide, and particulate carbon in an exothermic reaction⁶²:



Zinc chloride, which constitutes approximately two thirds of the smoke and accounts for the acute toxic effects, is hygroscopic and becomes coated with water molecules, which in turn produce the smoke effect.

The chemical equation for HC smoke combustion is merely an ideal equation. In reality and so that the rate of combustion can be controlled, there is stoichiometrically not enough aluminum to balance this equation. Therefore, there are side reactions that produce additional combustion products as chlorinated vapors (Table 24-3 and Exhibit 24-1). The proportion of the various side-products depends on atmospheric conditions, especially relative humidity. For example, hydrochloric acid at lower humidities only is produced at much higher levels than the other additional combustion products.⁶² HC smoke munitions produce both severe respiratory irritants, an asphyxiant, and known or suspected carcinogenic substances.

The uncombusted HC smoke munition contains several impurities: lead, cadmium, arsenic, and mercury. Lead and cadmium are present at higher levels than arsenic and mercury. The impurities, though, are present in relatively small amounts compared with the other munition ingredients. They are present at levels of less than 0.2%.⁶³ The impurities are also present in the smoke cloud as oxides and are likewise present in low levels.

In training and in combat, the service member may be exposed to HC smoke through the skin, through the gastrointestinal tract, and through open wounds. However, by far the most significant route of exposure is inhalation. The acute health effects from HC smoke are thought to be due to zinc chloride, which makes up two thirds of the resultant smoke from a combusted HC smoke munition. Zinc chloride is a corrosive astringent compound that is highly soluble in water. Its water-solubility results in easy deposition in the upper airways. In lower doses, zinc chloride will deposit primarily in the nasal passages and the pharynx. In higher doses,

TABLE 24-3

APPROXIMATE COMPOSITION OF HC SMOKE

| Constituent | Estimated Mass Fraction, % |
|-------------------------|----------------------------|
| Total particulate phase | 89.2 |
| Zinc chloride | 62.5 |
| Zinc oxide | 9.6 |
| Iron oxide | 10.7 |
| Aluminum oxide | 5.4 |
| Lead oxide | 1.0 |
| Chlorinated vapors* | 10.8 |

*chlorinated vapors are listed in Exhibit 24-1

Source: DeVaul GE, et al. Analysis Methods and Results of Hexachloroethane Smoke Dispersion Experiments Conducted as Part of Atterbry-87 Field Studies. Fort Detrick, Md: US Army Medical Research and Development Command; 1989. Contract No. 84PP-4822.

zinc chloride molecules will deposit in lower respiratory anatomical sites, to include the alveoli in extremely high doses.

Pathological changes are variable but are largely dose-dependent. Pathologic changes reported in the literature include laryngeal tracheal, and bronchial mucosal edema and ulceration; interstitial edema; interstitial fibrosis; alveolar obliteration; bronchiolitis obliterans; and subpleural emphysematous blebs complicated by pneumothorax.^{61,64,65}

EXHIBIT 24-1

ADDITIONAL CHLORINATED VAPOR COMBUSTION PRODUCTS OF HC SMOKE MUNITIONS

HCl (hydrochloric acid)
 COCl₂ (phosgene)
 CCl₄ (carbon tetrachloride)
 C₂Cl₄ (tetrachloroethane)
 C₂Cl₆ (hexachloroethane)
 C₆Cl₆ (hexachlorobenzene)
 CO (carbon monoxide)

Source: Katz SA, et. al. Physical and Chemical Characterization of Fog Oil Smoke and Hexachloroethane Smoke. 1980. Final Report. Contract DAMD17-78-C-8085. NTIS: AD A080936.

Most extremely high doses are a result of exposures to HC smoke in confined spaces, such as in tunnels during MOUT (military operations in urbanized terrain) training. There have been many instances in which individuals have died from high-dose acute exposure to HC smoke. In Malta during World War II, 70 people were exposed to HC smoke after enemy bombs ignited a store of 79 smoke munitions located 80 yards into a 200-yard tunnel. Thirty-four of the exposed people passed through the nearby first-aid station and one other was taken on board a ship in the harbor where he died within a few minutes. Of the 34 treated at the first-aid station, 2 died there; 15 were sent to a nearby general hospital, of whom 6 died; 17 were sent to their homes. Of the 17 sent home, 3 were subsequently admitted to the general hospital, and 1 of these died, making the total number of deaths from this exposure 10.⁶¹

This case illustrates several points. First, exposure to HC smoke in an enclosed space is life-threatening. Second, serious pulmonary complications can appear in a delayed fashion. Three of the seventeen people who were sent home after an initial medical evaluation were subsequently admitted to the hospital and one of these died. It is extremely important for clinicians to ascertain an estimate of the dose of inhaled HC smoke. The exposure history should include the location of exposure (eg, confined space, distance from munition), approximate visibility, duration of exposure, respiratory protection used (eg, M17 mask), and the elapsed time since exposure. Given this information, the clinician is better able to decide whether someone who appears to have only mild respiratory symptoms may later develop life-threatening respiratory distress.

Treatment of the HC smoke-exposed casualty begins on the battlefield or training site. The service member should be removed from exposure; masking the casualty may be necessary if the smoke cloud is large and removal from exposure may be prolonged. Humidified supplemental oxygen (100%) should be administered with assisted ventilation as required. Exposed skin and eyes should be copiously flushed with water. Respiratory tract irritation, if severe, can progress to pulmonary edema, which may be delayed in onset for up to 72 hours. Therefore, the HC smoke-exposed patient should be closely monitored for respiratory symptoms and signs for at least 48 hours. Adequate ventilation and oxygenation should be maintained, with close monitoring of arterial blood gases. If pO_2 cannot be maintained above 50 mmHg with inspiration of 60% oxygen by face mask or mechanical ventilation, then positive end-expiratory pressure in intu-

bated patients or continuous positive airway pressure in non-intubated patients may be necessary. Crystalloid solutions must be administered carefully to avoid a net positive fluid balance. Central hemodynamic monitoring should be considered for ascertaining current fluid status. Morphine is not recommended since respiratory depression may occur. Antibiotics are indicated only when there is evidence of infection. Finally, although corticosteroids may prolong the onset of noncardiogenic pulmonary edema, it is unclear whether early administration of corticosteroids can prevent its eventual development.⁶⁶

Another smoke munition, terephthalic acid smoke is being fielded as a training replacement for HC smoke. Although it is considered to be much safer than HC smoke, it does contain respiratory irritants. Unfortunately, the quality of the smoke is not sufficient to produce the desired amount of obscuration for actual combat, so the terephthalic acid smoke munition will only be used in training.

Other Smokes and Obscurants

Fog Oil

The smoke generated by injecting mineral oil into a heated manifold is termed fog oil. The oil is vaporized when heated and condenses when exposed to the atmosphere to produce an opaque mist. Fog oil is produced from naphthenic oils. Such oils have been associated with increased incidence of skin cancer. The military specification for fog oil was changed in 1985 after it was determined that untreated naphthenic oils were carcinogenic. The new specification requires that the oil be processed to remove known carcinogenic and potentially carcinogenic substances.⁵⁷

Diesel Oil

Diesel oil smoke is formed by using the vehicle engine exhaust smoke system described above. The fuel is vaporized, resulting in a suspension in air of 0.5 to 1.0 mm droplets. The vaporized fuel consists mostly of saturated hydrocarbons, substituted benzenes, and 2-3 ring aromatic hydrocarbons. The respiratory tract is the major organ system of concern. Mortality from acute exposures is a function of the duration of exposure in addition to the concentration of the diesel oil smoke. Repeated exposure studies have shown that pulmonary toxicity is dependent on the frequency of exposure. Pulmonary lesions increase when exposure frequency exceeds twice weekly. The pulmonary lesions are largely inflammatory in nature.⁵⁷

Red Phosphorus

Red phosphorus is combined with styrene-butadiene rubber (butyl rubber) in a 95:5 mixture. The butyl rubber reduces the cloud-pillar effect found with pure red phosphorus. This mixture also contains insulating oil (1.25% by weight) and talc or silica (1% by weight), which is added to improve uniformity of the pattern. Red phosphorus reacts slowly with oxygen and water vapor to produce the highly toxic phosphine gas. However, this reaction is extremely slow at normal temperatures and humidities and is therefore not significant to military operations. The combustion products from red phosphorus in butyl rubber have been characterized. The particles consist primarily of various species

of phosphoric acid. Phosphoric acid is an irritant that may result in terminal bronchiolar fibrosis.⁵⁷

Colored Smokes

These smokes come in yellow, green, red, and violet. The colored smokes are to be used for signaling and identification purposes only. Quite often, these smokes are used inappropriately, resulting in unnecessary exposure to personnel. Each color has its unique formulation. (Some of the colored smokes are undergoing product improvement so that old and new compositions exist in the inventory). They commonly contain an anthraquinone dye and other components that are added to effect a controlled combustion of the munition. Soldiers

EXHIBIT 24-2

DEPARTMENT OF THE ARMY SAFETY POLICY REGARDING TRAINING IN SMOKE

The following precautions apply to all smoke training:

- A. Personnel will carry the protective mask when participating in exercises which include the use of smoke.
- B. Personnel will mask:
 - 1) before exposure to any concentration of smoke produced by M8 white smoke grenades or smoke pots (HC smoke) or metallic powder obscurants.
 - 2) when passing through or operating in dense (visibility less than 50 m) smoke such as smoke blankets and smoke curtains.
 - 3) when operating in or passing through a smoke haze (visibility greater than 50 m) and the duration of exposure will exceed 4 hours.
 - 4) any time exposure to smoke produces breathing difficulty, eye irritation or discomfort. Such effects in one individual will serve as a signal for all similarly exposed personnel to mask.
 - 5) personnel will mask when using smoke during military operations in urban terrain (MOUT) training when operating in enclosed spaces. Note: The protective mask is not effective in oxygen deficient atmospheres. Care must be taken not to enter confined spaces where oxygen may have been displaced.
 - 6) smoke generator personnel will mask when it is impossible to stay upwind of the smoke.
- C. Showering and laundering of clothing following exercises will eliminate the risk of skin irritation following exposure to smoke. Troops exposed to smoke should reduce skin exposure by rolling down sleeves.
- D. Special care must be taken when using HC smoke to ensure that appropriate protection is provided to all personnel who are likely to be exposed. When planning for the use of HC smoke in training, specific consideration must be given to weather conditions and the potential downwind effects of the smoke. Positive controls (observation, control points, communications) must be established to prevent exposure of unprotected personnel.
- E. Request applicable publications and standard operating procedures be reviewed to ensure appropriate safety precautions for use of smoke are included in training and employment guidance. Commanders should ensure this policy is widely disseminated.

Source: US Department of the Army, DAPE-HR. *Smoke Safety*. Washington, DC: DA; 1985. Message.

are exposed to the combustion products of these mixtures. The combustion products can include irritants that can result in significant pulmonary effects.⁶⁷ The mutagenicity and carcinogenicity of the combustion products of the anthraquinone dyes are variable. A combustion product of the old red smoke grenade, 2-aminoanthraquinone, is classified as a suspected animal carcinogen by the International Agency for Research on Cancer.⁶⁸

Graphite

Graphite flakes are disseminated mechanically into the atmosphere. Graphite dust behaves biologically as a nuisance dust; dust-laden macrophages are seen in the lungs as well as type II pneumocyte proliferation. However, the presence of crystalline silica in graphite may increase fibrogenic potential and result in a pneumoconiosis.⁶⁹ Fortunately, the particle sizes being disseminated are typically larger than the alveolar respirable range.⁷⁰

Policies and Controls

The Army's first safety policy on the use of smokes was developed in 1985 in response to sporadic epi-

sodes of soldier exposures to HC smoke that resulted in illnesses and deaths⁷¹ (Exhibit 24-2). The safety policy was a common sense approach to minimizing exposures. The policy has been criticized by some as being too conservative with regard to masking conditions. The concern has been that soldiers are less effective when wearing respiratory protection due to decreased visual fields and diminished communication. This diminution of effectiveness can lead to a decreased level of safety from other dangers that outweigh the risks from exposure to the smokes.

To formulate a safety policy that is founded on more objective data, the Army Surgeon General sought the assistance of the National Academy of Science's Committee on Toxicology to formulate exposure guidance levels.⁵⁷ The National Academy of Science used existing health data from peer-reviewed scientific literature to formulate the exposure guidance levels using a health risk assessment method.⁷² These levels, coupled with industrial hygiene exposure data from the field, may be used to refine the level at which masking must take place to prevent health effects to military personnel. These levels may also be used to determine safe locations for the employment of smoke on training installations and may be used to assess an individual's postexposure risk.

[William A. Rice]

REFERENCES

1. Gaydos JC, Thomas RJ, Sack DM, Patterson R. Armed Forces. In: JM Stellman, ed. *Encyclopedia of Occupational Health and Safety*. 4th ed. Geneva: International Labor Organization; 1998: 95.14–95.17.
2. Griffin MJ. *Handbook of Human Vibration*. London: Academic Press Ltd; 1988. RCS Med 388.
3. Dupuis H. Medical and occupational preconditions for vibration induced spinal disorders: Occupational disease number 2110 in Germany. *Int Arch Occup Environ Health*. 1994;66(5):303–308.
4. Johanning E. Back disorders and health problems among subway train operators exposed to whole body vibration. *Scand J Work Environ Health*. 1991;17:414–419.
5. Johanning E, Wilder DG, Landrigan PJ, Pope MH. Whole-body exposure in subway cars and review of adverse health effects. *J Occup Med*. 1991;33:605–612.
6. Kabacinska-Knapikowa D, Paradowski L, Kwiatkowski S. Esophageal motility in personnel operating heavy self-propelled mining machines. *Materia Medica Polona*. 1992;24(3):153–155.
7. Daleva M, Piperova-Dalbokova D, Hadjiolova I, Mincheva L. Changes in the excretion of corticosteroids and catecholamines in tractor drivers. *Int Arch Occup Environ Health*. 1982;49:345–352.
8. Kamenskii YN, Nosova IM. Effects of whole body vibration on certain indicators of neuro-endocrine processes. *Noise Vibration Bull*. 1989;Sept:205–207.
9. Nakamura R, Morojoji T, Nohara S, Nakamura M, Okada A. Activation of cerebral dopaminergic systems by noise and whole-body vibration. *Environ Res*. 1992;57(1):10–18.
10. Barron JL, Noakes TD, Levy W, Smith C, Millar RP. Hypothalamic dysfunction in overtrained athletes. *J Clin Endocrinol Metab*. 1985;60:803–806.

11. Ariizumi M, Okada A. Effect of whole body vibration on the rat brain content of serotonin and plasma corticosterone. *Eur J Applied Physiol Occup Physiol*. 1983;52:15–19.
12. Granjean E. *Fitting the Task to the Man*. New York: Little Brown; 1989.
13. Pope MH, Hansson TH. Vibration of the spine and low back pain. *Clin Orthop*. 1992;279:49–59.
14. Pope MH, Kaigle AM, Magnusson M, Broman H, Hansson T. Intervertebral motion during vibration. *Proc Inst Mech Eng [H]*. 1991;205(1):39–44.
15. Ishihara H, Tsuji H, Hirano N, Ohshima H, Terahata N. Effects of continuous quantitative vibration on rheologic and biological behaviors of the intervertebral disc. *Spine*. 1992;17(3 suppl):S7–S12.
16. Hansson T, Holm S. Clinical implications of vibration-induced changes in the lumbar spine. *Orthop Clin North Am*. 1991;22:247–253.
17. Kaji H, Sato E, Nagatsuka S, et al. Evaluation of sensory disturbances using short latency somatosensory evoked potentials (SSEPs) in vibration-exposed workers. [Japanese]. *Sangyo Igaku*. 1991;33:605–612.
18. DeLuca CJ. Myoelectrical manifestations of localized muscular fatigue in humans. *Crit Rev Biomech Eng*. 1985;4:251–278.
19. Klein AB, Snyder-Mackler L, Roy SH, DeLuca CJ. Comparison of spinal mobility and isometric trunk extensor forces with electromyographic spectral analysis in identifying low back pain. *Physical Therapy*. 1991;71(6):445–454.
20. Bovenzi M, Zadini A. Self-reported low back symptoms in urban bus drivers exposed to whole-body vibration. *Spine*. 1992;17:1048–1059.
21. Boshulzen HC, Bongers PM, Hulshof CT. Self-reported back pain in fork-lift truck and freight-container tractor drivers exposed to whole-body vibration. *Spine*. 1992;17:59–65.
22. Chatterjee S, Bandyopadhyay A. Effect of vibrating steering on the grip strength in heavy vehicle drivers. *J Human Ergology*. 1991;20(1):77–84.
23. *Initial Health Hazard Assessment Report on the Fast Attack Vehicle (FAV)*. Aberdeen Proving Ground, Md: Environmental Hygiene Agency; 1984.
24. Gow BS, Legg MJ, Yu W, Kukongviriyapan U, Lee LL. Does vibration cause post-stenotic dilatation in vivo and influence atherogenesis in cholesterol-fed rabbits? *J Biomech Eng*. 1992;114(1):20–25.
25. Nakamura H, Katoh A, Nohara S, Nakamura M, Okada A. Experimental studies on the pathogenesis of the gastric mucosal lesions induced by whole-body vibration. *Environmental Research*. 1992;58(2):220–229.
26. Katoh A, Nakamura H, Nohara S, Ohmura K, Munemoto Y, Oda M. Experimental studies on the development of acute gastric lesions induced by vibration stress in rats and its pathogenetic mechanisms. *Japanese J Gastroenterol*. 1992;89:469–476.
27. Mecel H, Wozniak H, Sun L, Frazier E, Mason HC. Effects on rats of exposure to heat and vibration. *J Applied Physiol*. 1962;17:759–762.
28. Nakamura H, Moroji T, Naaase H, Okazawa T, Okada A. Changes of cerebral vasoactive intestinal polypeptide- and somatostatin-like immunoreactivity induced by noise and whole-body vibration in the rat. *Eur J Appl Physiol Occup Physiol*. 1994;68(1):62–67.
29. McLain RF, Weinstein JN. Nuclear clefting in dorsal root ganglion neurons: A response to whole body vibration. *J Comp Neurol*. 1992;322:538–547.

30. McLain RF, Weinstein JN. Ultrastructural changes in the dorsal root ganglion associated with whole body vibration. *J Spinal Disord.* 1991;4(2):142-148.
31. McLain RF, Weinstein JN. Effects of whole body vibration on dorsal root ganglion neurons: Changes in neuronal nuclei. *Spine.* 1994;19:1455-1461.
32. Nakamura H, Moroji T, Nohara S, Nakamura M, Okada A. Activation of cerebral dopaminergic systems by noise and whole-body vibration. *Environ Res.* 1992;57(1):10-18.
33. Bartsch C, Meihe B. The effect of vibrations in the early stages of embryogenesis on the postnatal motor and physical development of Wistar rats [German]. *Anat Anz.* 1991;173(4):239-242.
34. USAARL Contract Reports No. CR 95-1, CR 95-2, CR 95-3, CR 95-4.
35. Ohlin DW. *20 Years Revisited: The Prevalence of Hearing Loss Among Selected U.S. Army Branches.* Aberdeen Proving Ground, Md: US Army Center for Health Promotion and Preventive Medicine; (Draft). Hearing Conservation Special Study No. 51-FK-5367-96.
36. Walden BE, Prosek RA, Worthington DW. *The Prevalence of Hearing Loss Within Selected U.S. Army Branches.* Washington, DC: U.S. Army Medical Research and Development Command; 1975. Interagency IAO 4745.
37. Price GR, Kalb JT, Garinther GR. Toward a measure of auditory handicap in the Army. *Ann Otol Rhinol Laryngol Suppl.* 1989;140:42-52.
38. Garinther GR, Peters LJ. Impact of communications on armor crew performance. *Army Res Development Acquisition Bull.* 1990;January-February:1-5.
39. US Department of the Army. *Hearing Conservation.* Washington, DC: DA; 1991. DA Pamphlet 40-501.
40. US Army Environmental Hygiene Agency. *Hearing Evaluation Automated Registry System (HEARS) Audiometer Operation Manual.* Aberdeen Proving Ground, MD: USACHPPM; 1991. Technical Guide 167A. Available from the USACHPPM, 5158 Blackhawk Road, ATTN: MCHB-DC-CHC, Aberdeen Proving Ground, MD 21010-5422.
41. US Army Environmental Hygiene Agency. *Hearing Evaluation Automated Registration System (HEARS) Manager's Module Operation Manual.* Aberdeen Proving Ground, Md: USACHPPM; 1991. Technical Guide 167B.
42. Ohlin, DW. *Personal Hearing Protective Devices, Fitting, Care and Use.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1975. USACHPPM Technical Guide 041.
43. *Noise Dosimetry and Risk Assessment.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1994. USACHPPM Technical Guide 181.
44. Goldstein J. *Noise Hazard Evaluation, Sound Level Data of Noise Sources.* Aberdeen Proving Ground, Md: US Army Center for Health Promotion and Preventive Medicine; 1975. USACHPPM Technical Guide 040.
45. US Department of the Army. *Wear and Appearance of Army Uniforms and Insignia.* Washington, DC: DA: 1992. Army Regulation 670-1.
46. Ohlin DW. *Evaluation of Communication Abilities with Hearing Protective Devices on a Grenade Range.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1974. Bio-Acoustics Special Study, No. 34-048-75.
47. Ohlin DW. *Evaluation of Communication Devices with Hearing Protective Devices on a Close Combat Course.* Aberdeen Proving Ground, Md: US Army Environmental Hygiene Agency; 1974. Bio-Acoustics Special Study, No. 34-029-74/7s.
48. Briggs CE III. *Operation Just Cause: Panama, December 1989: A Soldier's Eyewitness Account.* Harrisburg, Penn: Stackpole Books; 1990.

49. LTC Hatch, CPT Nelson, CPT Horvath, LT Eickma, LT Walpole. *Control of Gun Fumes in M-4 Series Medium Tanks*. Fort Knox, Ky: US Army Armored Force Medical Research Laboratory; 1943. Project Nos. 3-1 and 3-5, File No. 724.41.
50. McFarland RA, Roughton FJW, Halperin MH, Niven JI. The effects of carbon monoxide and altitude on visual thresholds. *Aviation Med.* 1944;Dec:381–394.
51. Dalton BA. Carbon monoxide in US Army tactical vehicles. *Med Bull US Army Med Dept.* 1988;Feb:11–13.
52. US Department of Defense. *Military Standard: Human Engineering Design Criteria for Military Systems, Equipment and Facilities*. Washington, DC: Government Printing Office: 1989. MIL-STD-1472D.
53. US Environmental Protection Agency. *Air Quality Criteria for Carbon Monoxide*. Washington, DC: EPA; 1991. Document EPA/600/8-90/045F.
54. Benignus VA, Petrovick MK, Newlin-Clapp L, Prah JD. Carboxyhemoglobin and brain blood flow in humans. *Neurotoxicol Teratol.* 1992;14:285–290.
55. Deeter DP, Gaydos JC, eds. *Occupational Health: The Soldier and the Industrial Base*. Part III, Vol 3. In: *Textbook of Military Medicine*. Washington, DC: Office of The Surgeon General; 1993.
56. Smith SR, Steinberg S, Gaydos JC. Errors in derivations of the Coburn-Forster-Kane equation for predicting carboxyhemoglobin. *Am Ind Hyg Assoc J.* 1996;57:621–625.
57. Committee on Toxicology, National Research Council. *Toxicity of Military Smokes and Obscurants*. Vol 1. Washington, DC: National Academy Press; 1997.
58. The Chemical Corps Association. *The Chemical Warfare Service in World War II*. New York: Reinhold Publishing Corporation; 1948.
59. Eaton JC, Lopinto RJ, Palmer WG. Health effects of hexachloroethane (HC) smoke. Fort Detrick, Md: US Army Biomedical Research and Development Laboratory; February 1994. Technical Report 9402.
60. US Department of the Army. *Deliberate Smoke Operations*. Washington, DC: DA; 1984. US Army Field Manual 3-50.
61. Evans EH. Casualties following exposure to zinc chloride smoke. *Lancet.* 1945;2:368.
62. Cichowicz JJ. HC Smoke. Vol 4. In: *Environmental Assessment, Programmatic Life Cycle Environmental Assessment for Smoke/Obscurants*. Aberdeen Proving Ground, Md: Chemical Research and Development Center, US Army Armament, Munitions and Chemical Command; 1983. ARCSL-EA-83007.
63. Katz SA, et al. *Physical and Chemical Characterization of Fog Oil Smoke and Hexachloroethane Smoke: Final Report*. 1980. Contract DAMD17-78-C-8085. NTIS: AD A080936.
64. Milliken JA, Waugh D, Kadish ME. Acute interstitial pulmonary fibrosis caused by a smoke bomb. *Canadian Med Assoc J.* 1963;88:36–39.
65. Matarese SL, Matthews JI. Zinc chloride (smoke bomb) inhalational lung injury. *Chest.* 1986;89:308–309.
66. Micromedex Tomes Medical Management System. Zinc chloride. Volume 27. Englewood, Colo: Micromedex Inc; 1996.
67. Rubin IB, Buchanan MV, Moneyhun JH. *Chemical Characterization and Toxicologic Evaluation of Airborne Mixtures: Chemical Characterization of Combusted Inventory Red and Violet Smoke Mixes*. Oak Ridge, Tenn: Oak Ridge National Laboratory; 1982. ORNL/TM-8810, AD A131527.
68. National Institute for Occupational Safety and Health. *Registry of Toxic Effects of Chemical Substances, 1983–84 Supplement to 1981–82 Edition*. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, Centers for Disease Control, NIOSH H; 1984.

69. Driver CJ, et al. Environmental and health effects review for obscurant graphite flakes. Aberdeen Proving Ground, Md: Edgewood Research, Development and Engineering Center; 1993. Report CR-056.
70. Guelta MA, Banks DR, Grieb RS. Particle size analysis of graphite obscurant material from an XM56 smoke generator. Edgewood Research, Development and Engineering Center Technical Report 053, 1993.
71. US Department of the Army, DAPE-HR. Smoke safety. 272030Z Nov 85. Message.
72. Committee on Toxicology, National Research Council. *Criteria and Methods for Preparing Emergency Exposure Guidance Level (EEGL), Short-term Public Emergency Guidance Level (SPEGL), and Continuous Exposure Guidance Level (CEGL) Documents*. Washington, DC: National Academy Press; 1986.