Chapter 12

LEAD

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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 silicate	$PbSiO_3$	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.[13] The vapor is typically more respirable than the dust produced by abrasive blasting.[14] 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(pp213–215)]

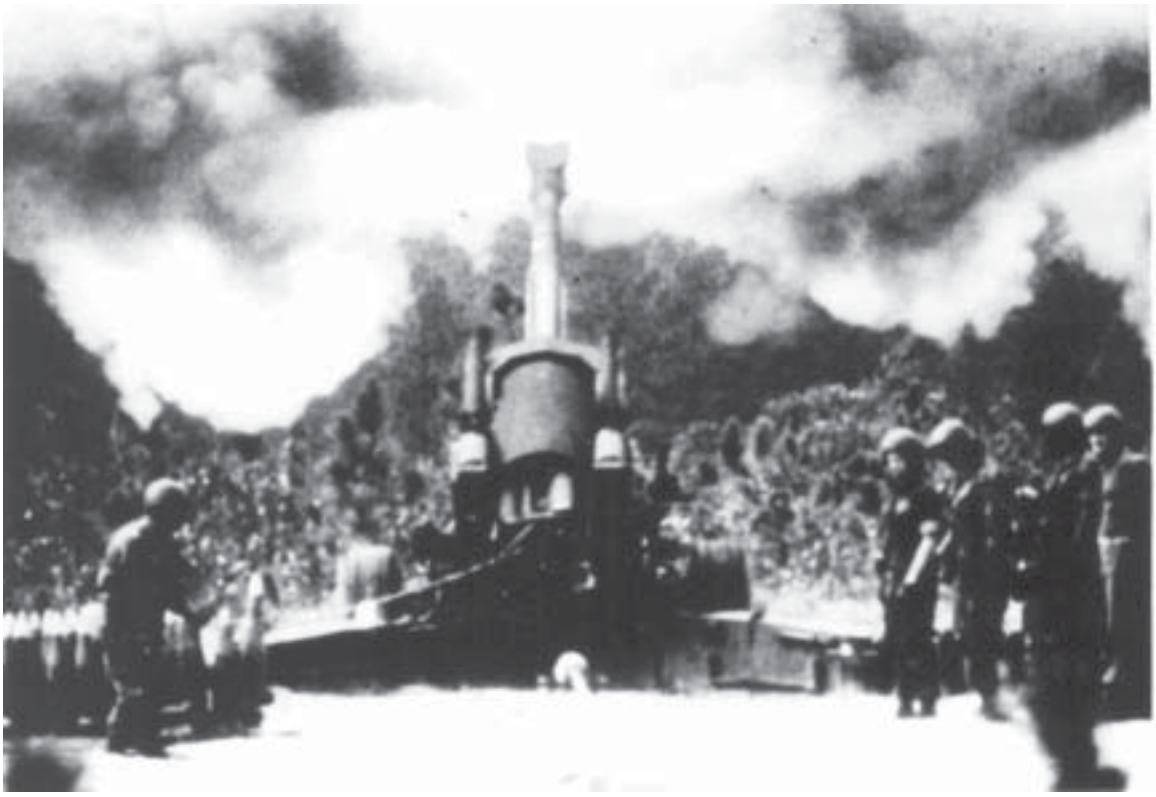

Fig. 12-7. Soldiers involved in outdoor firing operations may be briefly exposed to high concentrations of aerosolized lead from the lead azide in the ammunition primer. Source: US Army, Anniston Army Depot, Anniston, Ala.

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).

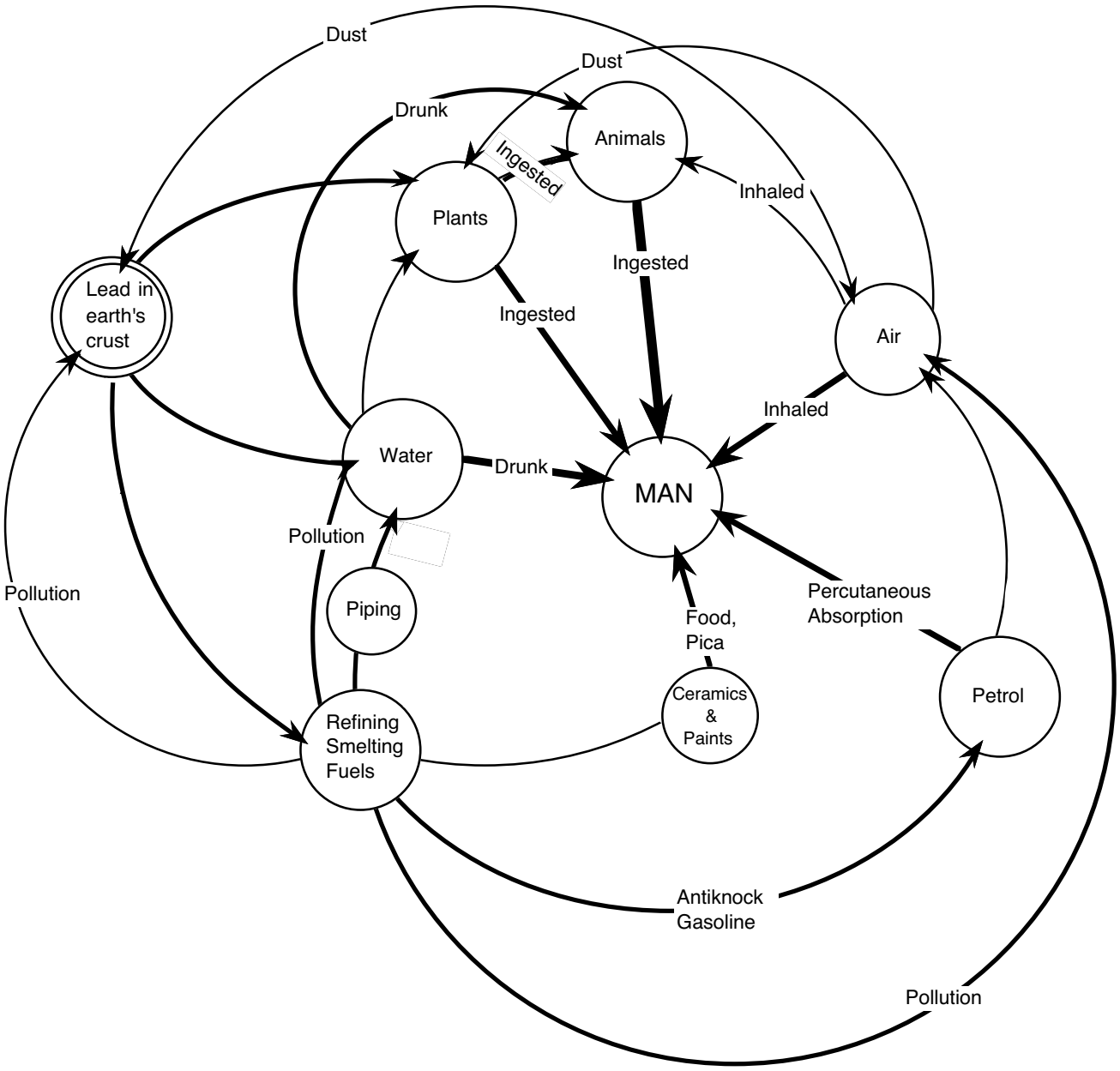

Fig. 12-8. Environmental routes of human lead exposure are indicated by arrows, with heavier arrows corresponding to more-significant exposure sources. For humans, a major route of lead exposure is ingestion of contaminated water and food. Other potential routes include exposure to lead-contaminated 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.[1(p61)]

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

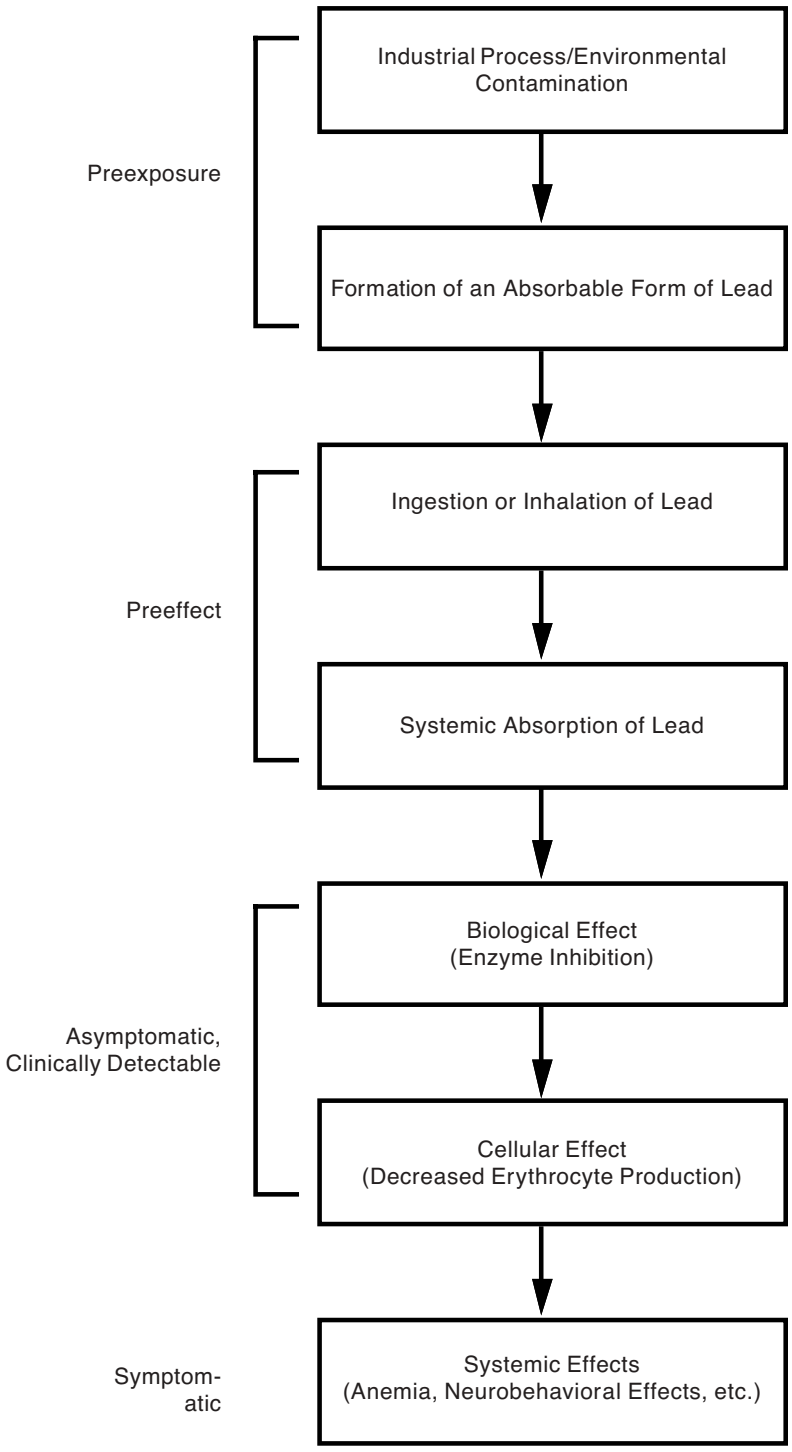

Fig. 12-9. Lead poisoning involves a definite sequence of events. In the preexposure period, absorbable lead is generated from environmental or industrial sources. After ingestion or inhalation, lead is systematically absorbed during the preeffect period. An asymptomatic period ensues, during which biological 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 var-iations 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.[38,39] 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

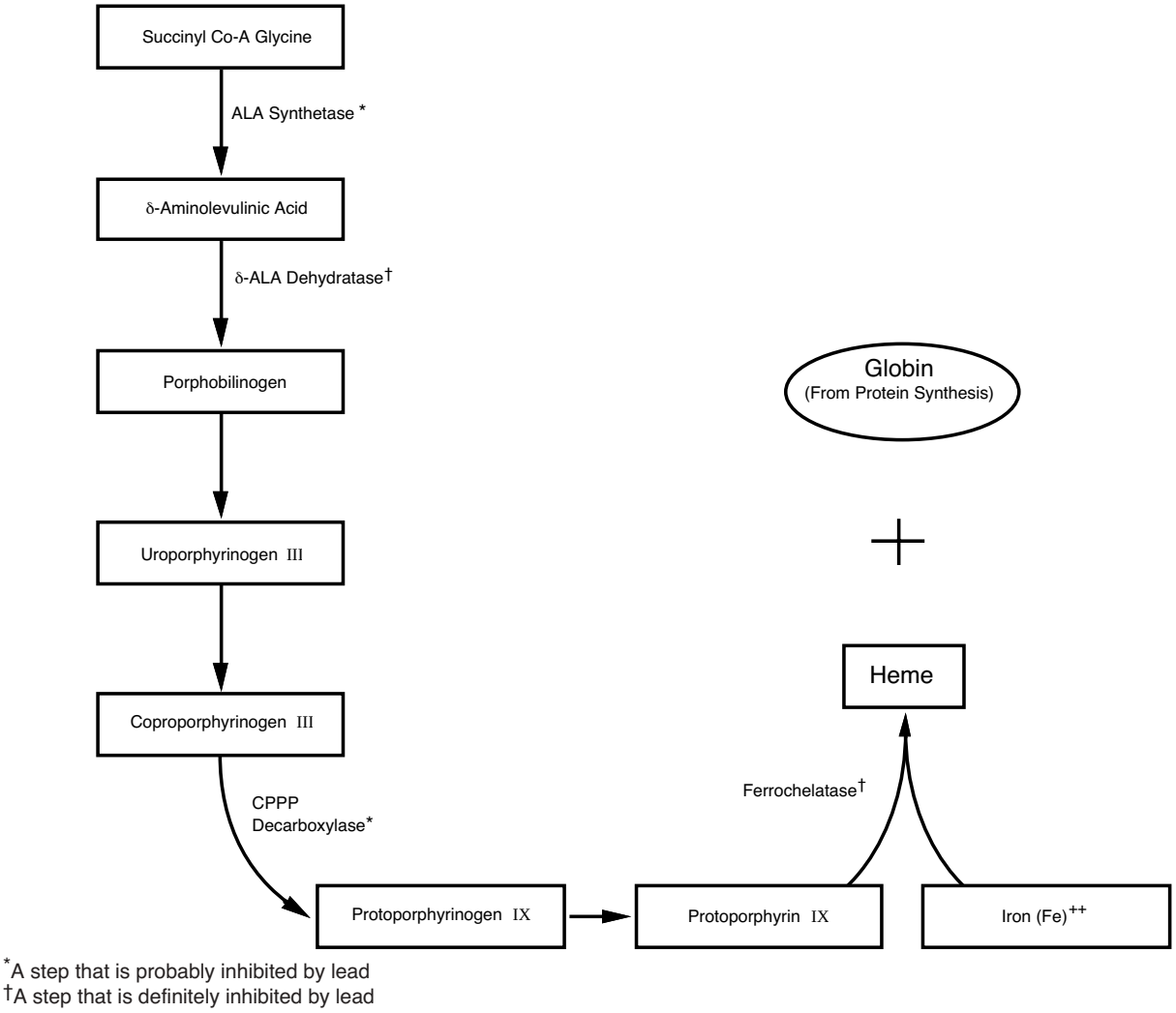

Fig. 12-10. The effects of lead on the biochemical pathway for the synthesis of hemoglobin. The synthesis of hemoglobin begins with the conversion of succinyl Co-A and glycine to aminolevulinic acid (ALA). This reaction is catalyzed by ALA synthetase, an enzyme that may be inhibited by lead. ALA dehydratase (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.[52] 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.[26] 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.[39,43,49,55,56] 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.[43] 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.[26,43,57]

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.[58]

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 80 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.[48] 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 noticeably 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.[62]

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.[63]

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.[64] 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.[65]

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,[35,52] 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 μg/m^3,[68] it dropped to 200 μg/m^3,[69] and is now 50 μg/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 μg/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 μg/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 μg/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

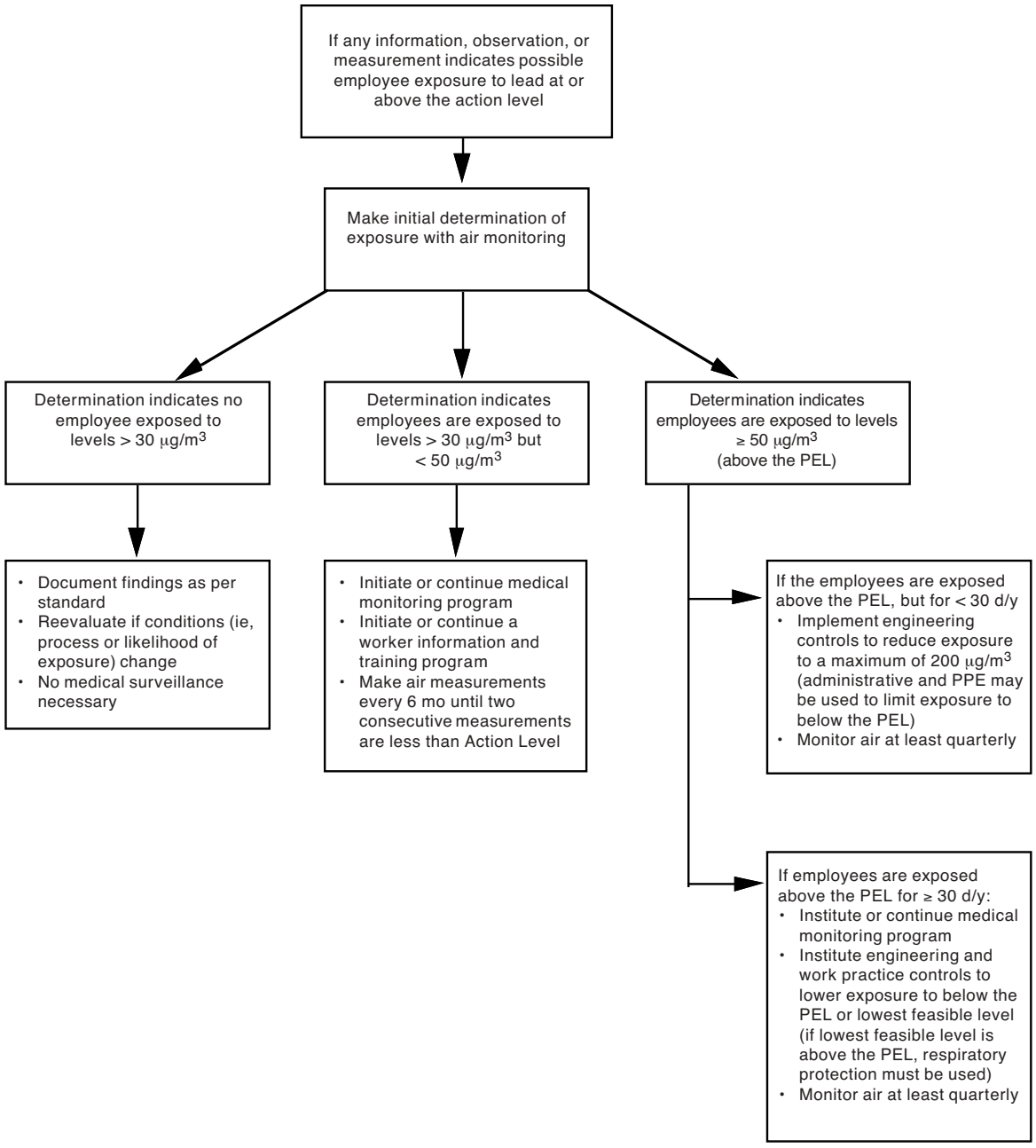

Fig. 12-11. These actions are required by 29 CFR, Part 1910 § 1025, the Occupational Safety and Health Administration's lead standard.

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 μg/m^3, and a combination of administrative and personal protection means can be used to effect the remaining reduction to less than 50 μg/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

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

while the employee is 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 per-sonnel 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.

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