

Clinical Evaluation and Management of Lead-Exposed Construction Workers

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An estimated one million construction workers are currently occupationally exposed to lead. Until 1993, construction workers were not offered the protections of OSHA's 1978 standard for lead exposure in industrial activities. Preventing exposure to lead in the construction setting presents many challenges, given the rapidly and frequently changing work environment.

This article reviews the adverse effects of lead on human health and presents an approach to the diagnosis, management, and prevention of lead-related illness. The medical aspects of the 1993 OSHA standard for lead in construction are described. Am. J. Ind. Med. 37:23–43, 2000. © 2000 Wiley-Liss, Inc.

KEY WORDS: *lead toxicity; medical monitoring; risk factors; epidemiology; construction; surveillance; treatment; intervention; prevention*

BACKGROUND

Approximately one million construction workers in the U.S. are occupationally exposed to lead [US DOL, 1993]. The majority of these are employed in commercial or residential renovation where lead-based paint may be disturbed. The lead content of paint was unregulated until 1977. Many older structures contain lead-based paint in deteriorating condition, posing a hazard primarily to young children. Increasing abatement of lead-based paint in housing and schools, driven in part by cases identified by mandated blood lead testing in children, will increase the number of workers with potential exposure to lead. Abrasive blasting, welding, cutting and torch burning on surfaces coated with lead-based paint can generate lead dust or fume, leading to inhalation and/or ingestion of lead [Osorio and Melius, 1995]. The dismantling and transport of lead-contaminated equipment and containment structures, as

well as the clean-up of lead contaminated construction rubble and debris can re-entrain lead dust, resulting in significant exposures. Types of construction activities and estimates of the number of workers exposed in these settings can be found in Table I [US DOL, 1993].

Lead-based paint covers five billion square feet of nonresidential surface area in the United States, including some 89% (90,000) of the nation's steel bridges. The transportation infrastructure is badly in need of renovation and repair, as is reflected in the passage of the federal Intermodal Surface Transportation Efficiency Act (ISTEA) in 1991 to fund infrastructure rehabilitation. In the course of rehabilitating these structures, more than 50,000 construction workers are potentially exposed to lead in this country [USDOL–OSHA, 1993]. A few states (e.g., Maryland) had their own regulations for exposure to lead in the construction setting. Until the promulgation of OSHA's lead in construction standard [USDOL–OSHA, 1993], however, most construction workers in the U.S. were not afforded the protective measures of OSHA's revised lead standard for general industry [USDOL–OSHA, 1978], although the magnitude of lead exposure in the construction trades, particularly among structural steel rehabilitators and demolition workers, has been well documented [Zimmer, 1961; Campbell and Baird, 1977; Landrigan et al., 1982; Fischbein et al., 1984; Marino et al., 1989; CDC, 1989, 1992, 1993]. The problem of lead poisoning in the

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TABLE I. Estimated Number of U.S. Construction Workers Exposed Annually to Lead

Type of construction	Number of workers	% of total	Source of lead
Commercial/institutional remodeling	546,798	58.4%	Lead paint
Residential remodeling	178,544	19.1%	Lead paint
Highway/railroad bridge repainting and rehabilitation	48,377	5.2%	Lead paint
Water lines repair and removal	41,042	4.4%	Lead pipes and solder
Housing in-place management	35,244	3.8%	Lead paint
Reinsulation over mineral wool	18,333	2.0%	Wool with lead contaminants
Lead joint work on cast-iron soil pipe	15,337	1.6%	Lead joint material
Housing lead abatement	12,238	1.3%	Lead paint
Elevator cable splicing/babbiting	9,500	1.0%	Lead splicing material
Commercial/industrial demolition	7,440	0.8%	Lead paint
Transmission/communication tower	7,333	0.8%	Lead paint maintenance
Water tank repainting	5,113	0.5%	Lead paint
Industrial facility maintenance and renovation (indoor/outdoor)	5,094	0.5%	Lead paint
Petroleum tank repainting	4,364	0.5%	Lead paint
Installation of certain roofing	576	0.1%	Lead steel roof and solder
Industrial process equipment manufacturing/repair	409	<0.1%	Lead bricks, mortar, sheets
Industrial vacuuming	392	<0.1%	Lead dust
Underground storage tank demolition	288	<0.1%	Lead paint
Stained glass window removal	208	<0.1%	Lead support rods
Installation of radiation shielding	40	<0.1%	Lead building material
Estimated total	936,670		

From US DOL, OSHA, 29 CFR Part 1926: Lead Exposure in Construction; Interim Final Rule [1993].

construction trades has been reviewed recently [Osorio and Melius, 1995].

The construction environment, unlike most industrial settings where lead exposure may occur, is marked by variability of exposure conditions, even within the course of a single work day. Changing conditions and tasks often result in “peak” episodic exposures, with intervening periods of little or no exposure to lead. “Bystander” exposure to lead dust and fume generated by the activities of nearby workers is a frequent occurrence on construction or renovation sites. In addition, the focus on protection of the surrounding environment has resulted in the practice of partial or total enclosure of some lead-disturbing tasks, with the potential for much greater exposures to workers within the containment structures. The special features of the construction setting can limit or make more difficult the use of traditional industrial hygiene measures to control exposure (e.g., exhaust ventilation), resulting in a greater reliance on personal protective equipment. Construction projects are often of short duration, and conditions which result in excessive lead absorption may not be identified by biological monitoring of workers until the project has been completed or the particular tasks performed have changed.

Given the increased sensitivity of children to the toxic effects of lead, an issue of particular concern is the phenomenon of “take-home” lead transported to the automobile and/or household on the lead-exposed worker’s contaminated clothing and shoes. In a small study based on New Jersey lead registry data, 32% of the children of workers with elevated blood lead levels were found to have blood lead concentrations $\geq 10 \mu\text{g}/\text{dL}$, compared to a prevalence of only 4.5% among all children in the third National Health and Nutrition Survey [Czachur et al., 1995]. Lead contamination of the homes of construction workers has been reported [Piacitelli et al., 1997].

THE ABSORPTION AND HEALTH EFFECTS OF LEAD

Routes of Exposure and Lead Absorption

Inorganic lead enters the body principally through inhalation and ingestion. Abrasive blasting, sanding, removal with “heat guns”, scaling, grinding, welding, cutting, and torch burning—all can produce lead particulates in the respirable range. Heat or burning lead or lead-based paint generates lead fumes, which form lead oxides as

they cool. These forms of lead are readily inhaled and absorbed [Fischbein, 1998]. Between 30 and 5% of inhaled lead is deposited in the respiratory tract and absorbed. Lead deposited in the lower respiratory tract is virtually completely absorbed. More than 75% of the lead deposited in the upper respiratory tract is transported to the gastrointestinal (GI) tract for absorption [Manton, 1985].

On the construction site, there are also frequent opportunities for ingestion of lead. Hand contamination is common, and workers frequently wipe their faces with lead-dusted hands in the course of the day's work. Studies conducted among battery workers [Far et al., 1993] and refinery workers [Karita et al., 1997] have demonstrated a relationship between hand-mouth contact as well as eating on-site with contaminated hands and lead absorption. The lack of hand washing facilities at the job site may make this an important contributor to overall exposure [ATSDR, 1998]. Workers may eat with contaminated hands at coffee or lunch breaks, and smoking with contaminated hands may permit both inhalation of volatilized lead and ingestion [Fischbein et al., 1992; al-Saleh, 1995]. In adults, approximately 10–15% of ingested lead is absorbed from the GI tract, and absorption increases under fasting conditions and with iron, calcium, phosphate, and zinc deficiency [Chisholm, 1981].

Distribution of Absorbed Lead

Lead is absorbed from the lung or GI tract and is distributed in the body in three main compartments: blood, soft tissues, and bone. Ninety-nine percent of the lead in blood is bound to red blood cells, and 1% is free in the plasma. The lead in plasma is available for exchange with the soft tissues (kidney, bone marrow, liver, and brain) and bone/teeth. The skeleton represents an accumulating reservoir containing approximately 95% of the body burden of lead in adults [Barry, 1975].

Under experimental exposure conditions in adult volunteers, the mean residence time of lead in the blood has been measured at approximately 25 days. Most of the lead in blood was excreted via urine. One fourth went to soft tissue, with a mean residence time of 40 days, after which it was excreted in bile, hair, sweat, and nails. A smaller proportion of the blood lead was deposited in bone [Rabinowitz et al., 1976]. The pattern of distribution and deposition may vary with the dose rate.

The mean residence time in cortical bone is 30 years. Lead stored in bone may be released under conditions of pregnancy, lactation, immobility, thyrotoxicosis and other states where demineralization of the skeleton may occur, resulting in endogenous exposure [Silbergeld et al., 1988; Goldman et al., 1994]. A contribution of skeletal lead stores lead to blood lead levels after the cessation of occupational exposure has been reported [Nilsson et al., 1991].

Blood lead levels best reflect recent exposure; i.e., that which has occurred within the prior month, but are poor indicators of total body burden. Because blood lead concentrations increase quickly after inhalation or ingestion, they are useful as a guide to the need for preventive interventions to reduce exposure. Blood lead levels associated with exposure to only the natural lead concentration in the biosphere, without the contribution from industrial sources, are estimated to range from 0.06 to 0.12 $\mu\text{g}/\text{dL}$ [Flegal and Smith, 1992]. While the second National Health and Nutrition Examination Survey (NHANES II) in 1976 found a geometric mean blood lead concentration of 12.8 $\mu\text{g}/\text{dL}$ for the U.S. population, NHANES III in 1991 found a marked decrease to 2.8 $\mu\text{g}/\text{dL}$, largely due to the removal of lead from most gasoline and from soldered cans [Brody et al., 1994]. Levels in excess of 10 $\mu\text{g}/\text{dL}$ are now considered to reflect exposures above "background."

X-ray fluorescence (XRF) measurement of lead concentrations in bone has emerged in recent years as an index of cumulative exposure to lead. The technique is non-invasive and exposes the examinee to a dose of x-irradiation less than one-tenth that of a chest radiograph [Todd et al., 1992]. The sensitivity of XRF detectors has improved, permitting bone lead determinations in populations with chronic, low-lead exposure [Hu et al., 1990]. Correlation between bone lead concentrations and time-integrated, prior blood lead levels in occupationally exposed groups has been demonstrated, permitting estimation of previous average blood and plasma lead levels [Bergdahl et al., 1998]. Two skeletal compartments have been demonstrated: cortical bone, e.g., the tibia, from which lead is mobilized only very slowly; and trabecular bone, e.g., the patella, from which lead can be mobilized more readily [Hu et al., 1996].

Health Effects

Lead exerts its toxic effects on many organ systems, across a wide range of exposure levels. Beginning with subtle, subclinical disturbances of enzyme function and biochemical aberrations, the toxic effects of lead can progress to severe, clinically evident disease with disruption of multiple organ functions [Fischbein, 1998]. Disruption of subcellular, mitochondrial energy metabolism by interference with enzymatic function, competition with essential metals for binding sites, and disturbance of ion transport mechanisms are likely to account for many of the subclinical and clinical manifestations of lead toxicity [US EPA, 1986].

Health Effects—Acute/Severe

In adults, the effects of high-dose exposure to lead have long been recognized and described [Landrigan et al., 1990]. The clinical picture is characterized by anemia,

abdominal colic, peripheral neuropathy (extensor weakness, “wrist/ankle drop”), central neuropathy with toxic encephalopathy, nephropathy, and sterility. With very severe poisonings, the frequency of which has decreased in recent years, tremors, stupor, intractable seizures, and coma may result when blood lead levels rise rapidly to exceed 100 µg/dL.

Health Effects—Chronic/Subclinical

The Adult Blood Lead Epidemiology and Surveillance (ABLES) program of the CDC, which monitors laboratory-reported elevated blood lead levels among adults in 27 states, has published data which indicate a decrease in the absolute numbers and the proportion of all levels ≥ 25 µg/dL from 1997 to 1998 [CDC, 1999]. In the U.S., more than 95% of elevated blood lead levels in adults result from workplace exposure [Rabin et al., 1992]. CDC monitoring data also indicate a decrease in absolute numbers of elevated lead levels from 1992, when the ABLES program was initiated, to 1998, with a corresponding decrease in the number of severe poisonings [CDC, 1992a]. Reflecting these trends, in recent years attention has turned increasingly to the toxicity resulting from chronic exposure to lower levels of lead than give rise to acute, severe poisoning [Loghman-Adham, 1997].

Workers with lower-level, chronic or recurrent exposure to lead may remain asymptomatic or develop vague, non-specific symptoms (e.g., myalgias, fatigue, irritability, headaches) reflecting more subtle organ system damage and impairment than is seen in acute intoxications. Such individuals often continue to work and come to clinical attention only as the result of blood lead screening or monitoring programs (now mandated by OSHA).

The relationship between blood lead concentrations and physiological impairments/health effects is graphically illustrated in Fig. 1 [ATSDR, 1990]. Studies are lacking which would more precisely define these relationships, especially at low to moderate cumulative dose levels. Data indicating disturbance of neurological development and vitamin D metabolism in children at blood lead concentrations of 10 µg/dL [Rosen, 1989] suggest that pathological changes may also occur in adults with blood lead levels below 40 µg/dL.

Recent evidence suggests a biochemical basis for interindividual differences in susceptibility to the toxic effects of lead. Genetic polymorphism in the heme biosynthetic enzyme ALA-D may account for the variability in the adverse effects of lead observed among individuals with comparable blood lead levels [Astrin et al., 1987]. ALA-D is markedly inhibited by lead. Significant correlations have been found among ALA-D phenotype, blood lead levels, and bone lead concentrations [Smith et al., 1995]. ALA-D allele-specific differences in kidney function

have been found in lead-exposed workers [Bergdahl et al., 1997].

Blood Forming Organs

Lead inhibits several enzymatic steps in the synthesis of hemoglobin. Delta-aminolevulinic acid dehydratase (ALA-D) is inhibited in a dose-dependent fashion, beginning at blood lead levels of 10 µg/dL [Schutz and Skerfving, 1976]. ALA-D activity can be 70% inhibited at blood lead levels of 40 µg/dL, the level permitted by OSHA under the current construction standard [USDOL–OSHA, 1993].

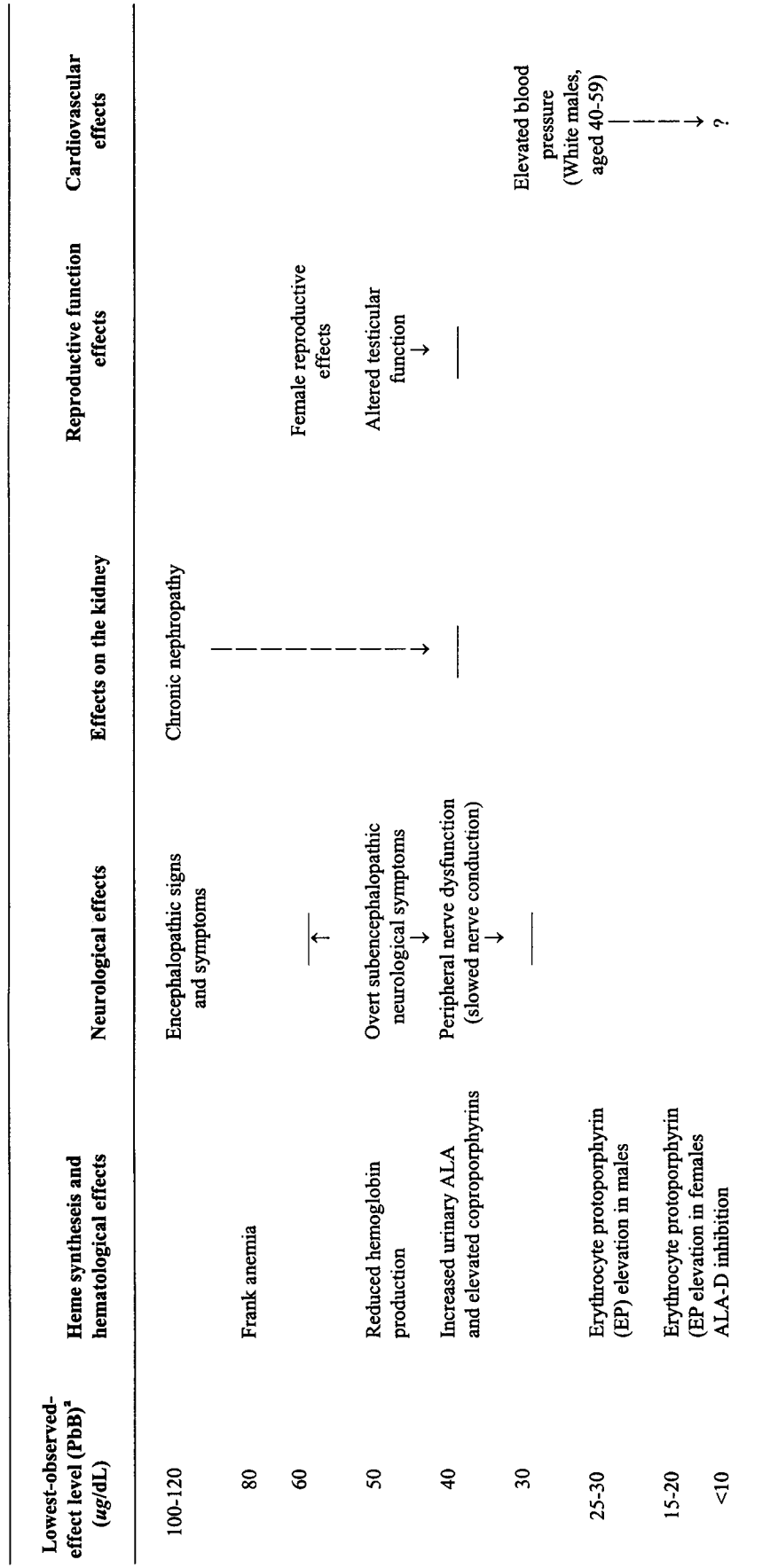
Inhibition of the mitochondrial enzyme, ferrochelatase, results in accumulation of erythrocyte protoporphyrin, as fetal erythrocyte protoporphyrin (FEP) or zinc protoporphyrin (ZPP), in the red blood cells and increased urinary excretion of coproporphyrin [Wildt et al., 1987]. Because ZPP remains in the erythrocyte for 120 days, the average life span of the red blood cell, the blood ZPP level reflects averaged exposure over a three month period [Eisinger et al., 1978].

Acute, high-level lead absorption can cause hemolytic anemia, while chronic absorption of lead can cause anemia by inhibition of heme synthesis and decreasing red blood cell survival. A relationship has been found between hemoglobin and hematocrit levels and lead concentrations in trabecular bone [Hu et al., 1994]. Frank anemia is not generally an early manifestation of lead poisoning, but becomes evident only after a prolonged period of elevated blood lead levels.

Renal Effects

The clinical picture of acute lead related nephropathy, following a period of markedly increased absorption of lead with rapidly rising blood and soft tissue lead levels (as in childhood lead poisoning following ingestion of lead-based paint chips), has been described and includes dysfunction of the proximal tubules, associated with the appearance of intranuclear inclusion bodies and swelling of the mitochondria in proximal tubular cells [Goyer et al., 1968]. Glycosuria, aminoaciduria, and phosphaturia (the Fanconi syndrome) are the laboratory manifestations of the tubular disorder. Unlike the more insidious nephropathy associated with long-term exposure at lower levels, acute lead nephropathy appears to be largely reversible following treatment with chelating agents [Loghman-Adham, 1997].

Chronic nephropathy, with proximal tubular damage and eventual destruction, can result from long-term excessive absorption of lead. The tubular nephropathy can progress to diffuse, interstitial renal fibrosis and renal failure [Legge and Goadby, 1912]. Lead-induced nephropathy has been shown in an experimental model: prolonged adminis-



^aPbB = Blood lead concentrations.

Adapted from ATSDR, 1990

FIGURE 1. Summary of lowest-effect levels for key lead-induced health effects in adults.

tration of lead to rats caused tubular atrophy, leading to interstitial nephritis and fibrosis [Goyer, 1971].

At blood lead levels of 40–80 $\mu\text{g}/\text{dL}$, intranuclear inclusion bodies consisting of lead–protein complexes can form in the cells lining the proximal tubules [Beaver, 1961]. Impairment of proximal tubular function can cause a Fanconi-like syndrome, as well as decreased excretion of uric acid and hyperuricemia. Increases in blood urea nitrogen (BUN) or creatinine levels are not generally seen until over 50% of the nephrons are destroyed, and initially the renal changes are clinically silent [Lilis and Landrigan, 1988].

In 1958, a relationship between lead intoxication among Australian children and increased rates of hypertension and renal insufficiency in later life was reported. Lead poisoning during childhood was also associated with a higher risk of subsequent death from chronic nephritis. Granular, contracted kidneys and increased bone lead levels were found among those who died of renal failure [Henderson, 1958]. A similar study of adults with a history of childhood lead poisoning in Boston failed to demonstrate significant renal dysfunction in that group, perhaps because they had undergone earlier chelation therapy than in the Australian experience [Tepper, 1963]. Renal interstitial fibrosis, tubular degeneration, glomerular degeneration, and intranuclear inclusion bodies were found among chronic consumers of lead-contaminated “moonshine” whiskey in the U.S. [Reynolds et al., 1983].

Blood lead levels, ranging from 2.3 to 72.5 $\mu\text{g}/\text{dL}$, were shown to correlate with measures of glomerular dysfunction in a study of a large European population [Staessen et al., 1992]. Measures of renal function in asymptomatic lead-exposed workers demonstrated evidence of nephropathy in half of those examined [Wedeen et al., 1975]. A relationship between blood urea nitrogen concentrations and blood lead levels has been shown in a number of occupationally exposed groups [Lilis et al., 1980]. Low-level exposure to lead has been shown to impair renal function and accelerate age-related loss of renal function in asymptomatic middle-aged and older men, among whom increased serum creatinine levels were positively correlated with blood lead levels [Kim et al., 1996]. Impaired glomerular function has been reported to be correlated with the time-integrated blood lead history among lead-exposed workers [Chia et al., 1995]. Increased mortality from chronic renal failure has been found in lead-exposed smelter workers [Selevan et al., 1985] and battery manufacturing workers [Cooper, 1985].

Urinary excretion of high molecular weight proteins, e.g., albumin, can be measured as an index of early glomerular dysfunction [Mutti, 1989]. Measurement of creatinine clearance remains, however, the “gold standard” for assessing glomerular function [Todd et al., 1996].

Injury to the proximal tubules causes failure of tubular reabsorption of low molecular proteins, with increased excretion of *N*-acetyl- β -glucosaminidase (NAG), γ -gluta-

myltransferase (GGT), lactate dehydrogenase (LDH), β -2-microglobulin (β -2-MG), and retinol binding protein (RBP) [Lauwerys and Bernard, 1989]. NAG excretion may be particularly responsive to recent blood lead absorption, rather than long-term accumulation [Chia et al., 1994]. Blood lead levels ranging from 30 to 69 $\mu\text{g}/\text{dL}$ were associated with increased urinary NAG excretion, but not β -2-MG excretion [Kumar and Krishnaswamy, 1995].

Hypertension

Both toxicological and epidemiological studies have implicated lead absorption in the development or exacerbation of hypertension. Systolic and diastolic blood pressures have been shown to be higher in lead exposed workers than in controls [Verschoor et al., 1987], and systolic blood pressures were positively correlated with blood lead levels in a number of studies [Maheswaran et al., 1993; Schuhmacher et al., 1994]. A positive association between systolic blood pressure and blood lead concentration has been demonstrated, even in the absence of abnormal renal function, indicating a mechanism for hypertension distinct from the nephropathic effects of lead [deKort et al., 1987]. An effect of lead on diastolic, but not systolic, blood pressure has been reported in some studies [dos Santos et al., 1994]. Others have failed to demonstrate an effect of lead exposure on blood pressure in occupationally exposed groups [Parkinson et al., 1987; Wu et al., 1996].

Studies of the general population in NHANES II have shown a relationship between blood lead levels and systolic and diastolic blood pressures, observed at blood lead levels as low as 10–20 $\mu\text{g}/\text{dL}$ [Harlan et al., 1985]. Blood lead concentrations were associated with left ventricular hypertrophy in the NHANES II population [Schwartz, 1995]. Using bone lead concentrations as an index of chronic exposure, a number of studies have found a relationship with hypertension [Fleet, 1996; Hu et al., 1996]. Lead poisoning in childhood has been shown to increase the risk of clinically significant hypertension during adult life [Hu, 1991; Hu et al., 1996]. In a meta-analysis of 23 human studies (13 general population and 10 occupationally exposed groups), including 33,141 subjects, a two-fold increase in blood lead concentrations was associated with a 1.0 mmHg rise in systolic blood pressure and a 0.6 mmHg rise in diastolic blood pressure [Staessen, 1995].

The relationship between lead absorption and blood pressure has been shown with animal experimental models as well. Rats exposed to lead during development exhibit hypertension as adults [Aviv et al., 1980]. The hypertensive effects can be independent of nephropathy in animal models as well [Victory, 1988].

The mechanism of lead-induced hypertension is not fully understood, but may be mediated through lead's action on vascular smooth muscle [Lilis et al., 1968], increased

response to α -adrenergic stimulation [Boscolo and Carmignani, 1988], decreased β -adrenergic response [Chang et al., 1997], or response to catecholamines [Skoczynska et al., 1986]. Interference with the renin–angiotensin system [Vander, 1988] and disruption of the calcium signaling mechanism involved in smooth muscle contraction [Schwartz, 1995] may play roles as well.

Neurological Effects

Peripheral neurotoxicity resulting from high dose lead absorption first involves the motor axons, with segmental demyelination and axonal degeneration [Fullerton, 1966]. When sufficiently severe, the classic clinical manifestation of wrist or ankle “drop”, characterized by extensor muscle weakness at the wrist or ankle, may be seen, although this has been found much less frequently since governmental regulation has resulted in better controlled exposures [Fischbein, 1998].

A precursor to frank extensor paresis is likely to be the peripheral neuropathic changes demonstrable on nerve conduction velocity testing. Slowed ulnar nerve conduction has been found when blood lead levels reached 30–40 $\mu\text{g}/\text{dL}$ among new workers in the lead industry [Seppalainen et al., 1983]. Sensory neuropathies may also be present, resulting in the paresthesias experienced by some lead-intoxicated workers. Vibratory thresholds, sensory amplitudes and sensory and motor conduction velocities were negatively correlated with time-integrated blood lead concentrations in lead battery workers [Kovala et al., 1997]. Subclinical tremor has been found among lead-exposed workers with blood lead levels in excess of 70 $\mu\text{g}/\text{dL}$ [Repko et al., 1978]. Reversibility of the lead-induced peripheral neuropathy following chelation therapy has been demonstrated in some studies [Araki et al., 1980]. An effect of lead on the development of entrapment neuropathy, with reversal of electrophysiological abnormalities following chelation with EDTA, has been reported in a lead-exposed worker with evidence of cubital tunnel syndrome [Kajiyama et al., 1993].

In severe, acute lead poisoning, dramatic disturbances of central nervous system function may occur, more frequently seen in lead-poisoned children with blood lead levels $>100\ \mu\text{g}/\text{dL}$. The clinical picture may include convulsions, delirium, and coma [Fischbein, 1998]. More common among workers is the toxic encephalopathy associated with longer-term, lower level exposure to lead, accompanied by non-specific, often subtle symptoms, including headache, dizziness, sleep disturbances, short-term memory deficits, depression, fatigue, irritability, joint and/or muscle pain, and loss of libido [Lilis et al., 1977].

Impaired cognitive performance has been demonstrated in relation to chronic low-level exposure to lead in a number of studies of occupational groups [Valciukas et al., 1978;

Johnson et al., 1980; Stollery et al., 1989; Balbus-Kornfeld et al., 1995]. Recently, neuropsychological decrements have been found to persist among lead battery workers with a history of blood lead levels ranging from 50 to 100 $\mu\text{g}/\text{dL}$ with subsequent low-level exposure to lead, indicating long-lasting and perhaps permanent functional impairment [Hanninen et al., 1998]. Central nervous system impairment with intellectual and cognitive dysfunction can occur at blood lead levels in the 40–70 $\mu\text{g}/\text{dL}$ range, and there is evidence that impaired short-term memory and difficulty in concentrating may occur when blood lead levels reach 40 $\mu\text{g}/\text{dL}$ [U.S. EPA, 1986]. Emotional lability, with irritability, sleep disturbance, fatigue, and depression may be among the earliest symptoms experienced by workers with excessive absorption of lead and have been shown to occur when blood lead concentrations exceed 40 $\mu\text{g}/\text{dL}$ [Baker et al., 1983]. In a recent study of elderly women, blood lead levels as low as 8 $\mu\text{g}/\text{dL}$ were significantly associated with decrements in cognitive function measured by neuropsychological testing [Muldoon et al., 1996]. Quantitative EEG changes have been demonstrated in relation to long-term lead exposure [Kovala et al., 1997].

The mechanism by which lead disturbs CNS function has been the subject of recent investigation. Effects of lead on neurotransmitter system functions in specific areas of the brain have been demonstrated in rats following low-level exposure in rats [Kala and Jadhav, 1995]. Brain mitochondrial respiration and adenosine diphosphate (ADP) phosphorylation is affected *in vitro* by lead [Dumas et al., 1985].

Gastrointestinal Effects

Lead may exert a direct effect on visceral smooth muscle tone and vascular supply in the gastrointestinal tract, leading to non-specific symptoms of epigastric discomfort, nausea, anorexia, weight loss, and dyspepsia. At higher blood lead levels ($>80\ \mu\text{g}/\text{dL}$), severe abdominal cramping and constipation may occur (lead colic) [Fischbein, 1998].

Reproductive Effects

The toxicity of high-dose lead exposure to reproductive function in both sexes has been known since early twentieth century [Legge, 1901; Oliver, 1914]. Lead has been used as an abortifacient in the U.K. and elsewhere [Hall and Cantab, 1905].

In female workers, lead exposure at high doses has been reported to result in an increase in the frequency of miscarriages and stillbirths. Increased prevalences of menstrual disturbances, spontaneous abortion and threatened abortion were reported to occur among lead exposed women. Decreased volume of ejaculate, prolonged latency of semen liquifaction, reduced sperm counts, and decreased sperm mobility and viability occurred among lead-exposed

men with blood lead levels $>40 \mu\text{g/dL}$ [Xuezi et al., 1992]. Lead crosses the placenta readily, with equilibration of blood lead levels in the fetus and mother. Reduced birth weight [Gonzalez-Cossio et al., 1997] and premature birth are associated with maternal lead exposure, as well as impairment of central nervous system development [Bellinger et al., 1987; Bornschein et al., 1985].

The fertility of male battery manufacturing workers was reported to decrease significantly after the onset of exposure to lead [Gennart et al., 1992]. A comparative study of the fertility of lead-exposed workers and unexposed bus drivers revealed decreased fertility among the lead-exposed group, even when age, race, education, and residence were controlled for [Lin et al., 1996]. An increased prevalence of congenital epilepsy and heart disease was found among the children of lead-exposed male workers. Testing of their semen quality revealed an increased rate of teratospermia and hypospermia, along with decreased LDH-X and succinodihydrogenase levels in sperm [Hu et al., 1992]. Among men with blood lead levels in the $50\text{--}70 \mu\text{g/dL}$ range, decreased sperm counts and concentration, impaired motility of sperm and abnormal morphology have been reported [Lancranjan et al., 1975; Cullen et al., 1984]. Effects on testicular function at lower blood lead levels have not been studied adequately. Effects on sperm quality appeared to be independent of disturbance of endocrine function in some studies [Assennato et al., 1987], while in others an effect of lead exposure at high levels on LH secretion at the hypothalamic-pituitary level has been found [Braunstein et al., 1978]. Decreased libido and difficulties in maintaining erection have been reported by men with blood lead levels of $40\text{--}50 \mu\text{g/dL}$, and appear to occur concomitantly with manifestations of central nervous system dysfunction. Chelation therapy has resulted in improved semen quality among lead-poisoned male workers [Cullen et al., 1984]. Women with lead-exposed husbands have been reported to miscarry at higher rates [Lindbohm et al., 1991; Anttila and Sallmén, 1995].

Many animal experimental studies have demonstrated the toxicity of lead at high doses to both male and female reproductive function [US EPA, 1986].

Carcinogenic Effects

The International Agency for Research on Cancer (IARC) in 1987 evaluated the evidence for the carcinogenicity of lead and concluded that lead and its inorganic compounds were possible human carcinogens (IARC group 2B), based on sufficient evidence for carcinogenic effects in animal studies but inadequate evidence for carcinogenicity in humans [IARC, 1987].

Animal experimental studies have shown that inorganic lead compounds can induce renal adenomas and carcinomas in rodents [IARC, 1987]. Renal tubular tumors, including

carcinomas, have been shown to occur in mice treated with lead acetate during gestation and lactation, even in the absence of lead-related nephropathy [Waalkes et al., 1995]. Two case reports have appeared in the literature associating renal cell carcinomas with lead exposure [Baker et al., 1980; Lillis, 1981].

A recent meta-analysis of available epidemiological data found evidence among heavily exposed workers to indicate associations between lead exposure and overall cancer, stomach, lung, and bladder cancer. A non-significant increase in kidney cancer risk was found. The analysis was limited by the lack of information about potential confounders, including smoking habits and exposure to other carcinogens [Fu and Boffetta, 1995].

Lead has a direct genotoxic effect on mammalian cells at high doses, but has a more pronounced comutagenic effect when administered in combination with other DNA-damaging agents. This indirect effect appears to be mediated through lead's interference with DNA repair processes [Hartwig, 1994]. Lead-binding proteins in rat kidneys facilitate the transport of lead into the cell nucleus to form intranuclear inclusion bodies and to alter renal gene expression [Fowler et al., 1994].

DIAGNOSING AND MANAGING LEAD POISONING

The Clinical Evaluation of the Lead-Exposed Worker

The assessment of the patient with potential lead toxicity involves an evaluation of the exposures which could have resulted in excessive absorption of lead, a determination of whether the patient has developed symptoms consistent with lead poisoning, whether physical signs compatible with lead toxicity are present, and whether laboratory findings demonstrate evidence of excessive lead exposure and/or of organ system damage consistent with lead-related effects.

A comprehensive approach to the evaluation of such patients is necessary for the diagnosis of lead intoxication and its adverse effects on organ function, the management of lead poisoning, and the consideration of the risk of further exposure for individuals who have already sustained organ system impairment, whether as a consequence of exposure to lead or from other causes.

Current Lead-Related Symptoms

In cases of slow-onset lead poisoning in adults, many workers with elevated blood lead levels will report no complaints, even after detailed inquiry. The earliest symptoms to appear in chronic, insidious exposure circumstances most often include non-specific, frequently vague

TABLE II. Lead-Related Symptoms

- Muscle aches or pain
- Joint pain
- Loss of appetite
- Weight loss of 10 lbs or more without dieting
- Nausea/vomiting
- Unusual taste in mouth/change in taste of food
- Abdominal cramping/pain
- Constipation
- Headaches
- Paresthesias
- Sleeplessness
- Diffuse muscle weakness
- General fatigue/lethargy
- Depression
- Irritability
- Nervousness
- Change in personality
- Tremulousness
- Difficulty concentrating
- impairment of short-term memory
- Loss of libido
- partial or total impotence (men)
- History of infertility
- History of adverse reproductive outcomes
- Abnormal menses (women)
- History of blood pressure lability

complaints, including increased general fatigue, myalgia and arthralgia, loss of appetite, headaches, irritability, sleep disturbance, and/or diminished libido (Table II). With increasing exposure, symptom intensity may increase, and difficulty in concentrating, impairment of short-term memory, depression, incoordination, paresthesias, abdominal pain, constipation, and impotence may develop. In rapid-onset, severe poisonings, frank paresis, extreme lethargy, abdominal colic, and changes in consciousness may be reported by patients.

The clinical presentation for most patients depends on the rate at which excess absorption of lead occurs, the blood lead level achieved, and the duration of excessive exposure. While the examiner should inquire about all lead-related symptoms listed in Table II, the history should, therefore, focus on those symptoms which are likely to be associated with excessive lead absorption under the specific patient's exposure conditions. The likelihood that symptoms reported by the lead-exposed worker are attributable to lead absorption increases as the blood lead level rises above 30 µg/dL [Cullen et al., 1983].

For each symptom reported, the time of onset, any change in frequency or intensity, and the relation of such

symptoms to recent exposure conditions should be determined where possible. If blood lead data obtained over time are available, a relationship between the onset of lead-related symptoms and increments in blood lead levels should be sought. As described below, it is of value to attempt to identify particular work processes or tasks which place workers at the greatest risk for lead intoxication. Correlation of work task performed with increases in blood lead levels is at times possible for individual patients.

Prior Lead Poisoning History

Inquiry should be made regarding previous tests for lead absorption. The dates and frequency of such testing and the availability of prior test results should be determined. The patient should be asked whether any test results were reported as abnormal, whether "prophylactic" chelation was employed during a period of blood lead testing, and whether he/she was ever treated for lead poisoning. Detail about episodes of known lead intoxication should be collected, and medical records should be obtained, since patients are often not able to recall specific facts about their past clinical course and/or treatment which may be relevant to current management.

Medical History (Pre-Existing Organ System Damage)

History of previously diagnosed gastrointestinal, hematologic, renal, cardiovascular, reproductive, and neurological conditions should be obtained, both to recognize other possible contributors to current symptomatology and to identify pre-existing medical conditions which place the patient at special risk for the effects of lead (e.g., previously diagnosed renal disease, history of oligospermia).

Family Exposure

Inquiry should be made regarding the presence of young children (six years of age or younger) in the patient's household who may have been exposed via lead-dusted clothing brought home from work. The patient should be asked whether such children have undergone lead testing and what the results indicated. Testing should be encouraged if not already performed. Blood lead testing of the worker's spouse may also be indicated if lead-contaminated work clothing has been brought into the household or family vehicle.

Recent and Past Exposure to Lead

The specific trade of the patient should be determined, as well as the date of entry into the trade, including apprenticeship. The date of onset of work at the current job

should be established, along with the dates of employment at other job sites where lead may have been encountered during the prior several months. In order to approximate the magnitude of lead exposure, since air and biological monitoring data are frequently unavailable, the particular job tasks performed by the patient should be asked about (e.g., torch cutting, welding, rivet busting, grinding/sanding/scaling, heat gun use, sweeping up lead-containing debris, cable splicing, etc.). The total time spent during the work day in such tasks should be assessed. The physical characteristics of the job site should be described, including the degree of enclosure of the patient's work area and the proximity of other lead-disturbing activities nearby. Work performed inside containment structures should be detailed. The use of methods to control exposure, e.g., prior paint removal, the use of vacuum-shrouded tools, wet cleaning, and other exposure reducing measures should be asked about and described.

The use of personal protective equipment should be detailed, including whether and by what examination the medical clearance for respirator use was obtained. Fit testing and training for respirator use should be inquired about. The type of respirator and filter, the frequency with which filters are replaced and the respirator cleaned, and the method of storage should be described. The patient should be questioned about which specific tasks he/she uses a respirator for and which are carried out without respiratory protection.

The availability of hand washing facilities at the worksite, separate lead-free eating areas, and clean change rooms should be determined. Smoking at the worksite or immediately after work should be discussed. The patient should be questioned about whether changes of work clothing are provided and whether lead-contaminated work clothing is ever worn or brought home for laundering.

Given the complex and changing exposure environment found on most construction sites, only by capturing information about the above-described exposure factors can the occupational medicine physician estimate individual exposure levels, identify high-risk activities, and develop strategies for exposure reduction with industrial hygienists.

The patient should also be asked about previous or concomitant exposure to lead in other work settings, including battery manufacturing, metal mining/smelting, secondary smelters, cable/wire manufacturing or reclamation, cable splicing, plumbing, radiator repair, shipbuilding/repair (other than as a construction worker), wire/metal soldering, leaded glass manufacturing, printing/typesetting, firearms instruction, and other work with lead. Dates of exposure and estimates of degree of exposure should be considered in relation to current symptoms and laboratory findings.

A general exposure history should be obtained, with focus especially on agents which can impair organ functions

affected by lead as well, e.g., organic solvents and other heavy metals. Hobbies which entail potential exposure to lead should be asked about, including loading/reloading ammunition, use of a shooting range, home rehabilitation, stained glass work, and making lead fishing sinkers.

Physical Examination

In the majority of cases of insidious-onset lead poisoning, symptoms are not accompanied by specific findings on physical examination. Classically-described findings such as pallor, lead lines on the gums, wrist and/or ankle drop are rarely encountered in current clinical practice in the U.S. A general, comprehensive physical examination should be performed, to identify signs of possible lead toxicity and to identify conditions which place the patient at special risk with regard to additional exposure to lead. The examination should focus on the elements listed in Table III.

Laboratory Tests

The blood lead concentration

This is the best indicator of recent lead absorption and is the current biological standard for exposure to inorganic lead in the U.S. With the exception of the individual whose blood lead level is reflective of an elevated body burden of lead from a previous, high exposure, the blood lead concentration is a good indicator of exposure which has occurred in the 2–3 weeks prior to testing. The relationship between blood lead levels and lead-related health effects is presented in Fig. 1.

TABLE III. Physical Examination in Lead-Exposed Workers

- Blood pressure
- Pallor
- Appearance of chronic illness
- Lead (blue-black) lines on gums
- Abdominal tenderness
- Motor/sensory/cerebellar neurological deficits
- Tremor
- Cognitive function
- Mood and affect

For individuals with fertility/reproductive concerns (to exclude other causes of impaired reproductive function):

- Degree of sexual maturation
- Testicular atrophy or enlargement
- Cryptorchidism
- Orchitis

All blood lead levels in New York State are required to be reported to the New York State Department of Health lead registry. Other states have similar registries. Analyses are to be performed only in OSHA approved laboratories certified by the State Department of Health on the basis of quality control performance. Blood lead concentrations should be obtained for all individuals under clinical evaluation for lead exposure, with repeat determinations to be guided by the initial level, opportunities for ongoing exposure, clinical complaints/findings, and other clinical factors. In cases where the blood lead concentration is $\geq 40 \mu\text{g/dL}$, blood lead monitoring should generally be performed at least once in every two weeks at the outset to be sure that the levels are in fact declining.

Free erythrocyte protoporphyrin (FEP) or zinc protoporphyrin (ZPP) levels in the blood

These reflect the effect of lead on the heme synthetic pathway and indicate lead exposure over the three month period prior to testing (determined by the average 120 day survival time of the erythrocytes). ZPP levels begin to rise above the normal value of $35 \mu\text{g/dL}$ when blood lead concentrations rise above $30 \mu\text{g/dL}$; ZPP levels decline more slowly than blood lead levels following reduction or cessation of exposure. OSHA regulations require that both blood lead levels and ZPP concentrations be monitored for workers exposed to lead in the construction setting. These should be repeated with the same frequency as blood lead determinations.

Patients in whom ZPP or FEP levels are elevated should have a *CBC, with a peripheral smear for RBC morphology*, and a *serum iron level* performed to rule out anemia, which can result in increases in erythrocyte protoporphyrin levels independent of lead's effect. A *routine urinalysis* (specific gravity, sugar, and protein determinations, with microscopic examination) should be obtained, as well as *blood urea nitrogen* and *serum creatinine* levels to identify clinically significant nephropathy. Twenty-four hour excretion of *N-acetyl- β -glucosaminidase (NAG)*, although primarily a research tool, may be measured as a subtle indicator of early renal tubular dysfunction, which may be useful in monitoring workers with pre-existing renal disease or who have one remaining kidney.

Nerve conduction velocity (NCV) testing

This may be of value in documenting the presence and characteristics of a lead-related peripheral neuropathy. Findings are not specific to lead, but impaired conduction rates may be an early sign of subclinical neurotoxicity. NCVs should be considered when persistent symptoms

and/or clinical findings suggest the presence of a peripheral neuropathy and especially if blood lead levels exceed $80 \mu\text{g/dL}$.

Neurobehavioral testing

This is to assess cognitive function. It may provide evidence of an organic brain syndrome associated with lead absorption, which may not be detectable by routine mental status assessment. Where persistent impairment of cognitive function is suspected and when blood lead levels exceed $80 \mu\text{g/dL}$, neurobehavioral testing should be considered.

Sperm analysis

This may be of value in determining the effect of lead absorption on reproductive function in men. Where the reproductive history indicates problems with fertility, sperm analysis is indicated. The OSHA standard for lead exposure in construction provides for a medical consultation, which may include a spermatogenesis assay, for any male worker who has concerns about his reproductive status.

CaNa₂EDTA challenge testing

This has been used to assess the body burden of lead and as a guide to indicate the necessity for chelation therapy. The test is considered positive for an increased lead burden when the twenty-four hour urinary excretion of lead exceeds $600 \mu\text{g}$ following intravenous injection of 2 g Ca-EDTA. Its value as a measure of chronic, cumulative exposure has been called into doubt, since the lead excreted following challenge is drawn mainly from the soft tissue pool [Skerfving et al., 1993]. Use of such challenge testing is declining also due to questions raised about the possibility of redistributing lead from skeletal stores to the brain and other sensitive organs, and the increasing availability of XRF determination of bone lead concentrations as a measure of chronic, cumulative exposure [Fischbein, 1998].

Bone lead concentrations measured by x-ray fluorescence (XRF)

This is a relatively new technology for the determination of the cumulative body burden of lead and, therefore, of chronic exposure. This is based on the observation that approximately 95% of absorbed lead ultimately is deposited in the skeleton, with only very slow release after deposition (half-life >25 years). Clinical correlation with elevations in bone lead concentrations are currently under study and have been reported with increasing frequency (see Hypertension above). XRF detection equipment is still available only in a

few research centers, and the technique remains largely a research tool at present, although its increasing clinical application can be anticipated.

The decision to obtain specific tests of organ system function, e.g., neurobehavioral studies or NCVs, must be based on clinical findings, including the severity and duration of symptoms and the observation of abnormal signs on physical examination.

The Management of Excessive Exposure to Lead (Lead Poisoning)

Reduction of exposure is a necessary component of the management of all cases of excessive absorption of lead. As described above, when lead level rises to 50 $\mu\text{g}/\text{dL}$, mandatory removal from exposure is required by the OSHA standard. This can take the form of the individual's transfer to a lead-free setting or remaining off work at full pay until lead levels fall to below 40 $\mu\text{g}/\text{dL}$. Given the accumulating information indicating adverse effects on enzymatic function, peripheral nerve conduction, and reproductive function at levels below 40 $\mu\text{g}/\text{dL}$, reduction of exposure should be attempted when the blood lead level is above 25 $\mu\text{g}/\text{dL}$ and/or shows an increasing trend. This may involve improvements in engineering design of the task(s), improved personal protection, hand washing, and other changes. Intervention strategies should be guided by an investigation of exposure conditions by a trained industrial hygienist. Such investigations and interventions should always follow the identification of workers with blood lead levels above 50 $\mu\text{g}/\text{dL}$.

Expected Fall in Blood Lead Levels Over Time

In many cases of lead poisoning, removal from exposure is the only treatment required [Fischbein, 1998]. Lead has a half-life in blood of approximately 25 days; in soft tissues, about 40 days; and in the non-labile skeletal compartment, over 25 years. In general, following complete removal from a recent, brief exposure, the blood lead level decreases by approximately 3–5 $\mu\text{g}/\text{dL}$ per week [Williams, 1984]. This rate, however, is influenced by the total lead burden present in blood, soft tissue, and labile skeletal stores, as well as by impairment of renal function.

Chelation Therapy

The decision to chelate a worker with evidence of excessive lead absorption should be based largely on several considerations: the current blood lead level; evidence of current adverse clinical effect, including disabling, lead-related symptoms; the duration of excessive exposure (as

determined by the exposure history or longitudinal blood lead data); and the duration of symptoms.

In general, when the blood lead level rises to 80 $\mu\text{g}/\text{dL}$, chelation should be considered, especially in the presence of symptoms and/or signs suggesting central or peripheral nervous system dysfunction [Fischbein, 1998]. For blood lead levels between 60–80 $\mu\text{g}/\text{dL}$, evidence of neurological impairment or such symptoms as severe abdominal pain, muscle/joint aching and marked irritability or depression may represent the basis for beginning treatment, especially when these symptoms have been present for more than one to two months. Chelation should rarely be considered when the blood lead level is below 60 $\mu\text{g}/\text{dL}$, with the exception of circumstances in which the affected individual experiences persistent (for more than two months), disabling lead-related symptoms or has reasons to achieve conception as soon as possible. In no circumstances should chelation therapy be instituted when opportunity for ongoing exposure to lead persists.

Chelation therapy in lead-poisoned adults has been reported to result in improved CNS function [Grandjean et al., 1991; Linz et al., 1992], male reproductive function [Cullen et al., 1984], and acute, lead-induced nephropathy [Loghman-Adham, 1997], as well as in preclinical lead nephropathy [Kim et al., 1996]. There are, however, no controlled clinical trials which have studied the efficacy of chelation treatment in lead-intoxicated adults. The most common chelating agents used currently include CaNa_2EDTA (sodium calcium edetate) and DMSA (2,3-dimercaptosuccinic acid).

CaNa₂EDTA therapy

Treatment with CaNa_2EDTA should usually be accomplished in a hospital setting and only after adequacy of renal function is established. The treatment should be overseen by an experienced physician. One gram of CaNa_2EDTA in 250 ml of 5% dextrose in water is infused intravenously over 4–6 h on each of 5 successive days, with daily determination of BUN, creatinine, and urinalysis results. Twenty-four hour collections of urine should be obtained daily during the period of treatment to measure the amount of lead excreted. Treatment should be discontinued if evidence of renal dysfunction (acute tubular necrosis) appears. Blood lead levels should be determined at the outset and conclusion of the five-day course. After an initial fall in blood lead concentration, a “rebound” in blood lead levels may be observed as lead is dynamically released from soft tissues and mobilizable skeletal stores. Persistence or recurrence of symptoms in the presence of rebound elevations in blood lead concentrations may require repeat treatment courses separated by 3–4 weeks. Again, evidence of renal compromise requires cessation of therapy.

DMSA (2,3-dimercaptosuccinic acid, Succimer) therapy

This is an orally administered chelating agent and has recently found use as a replacement for EDTA in adult lead poisoning, although initial clinical investigations were carried out on pediatric populations [Graziano, 1986]. The dose used in studies of DMSA chelation in adults has not been consistent, but treatment regimens utilizing 30 mg DMSA/kg in three divided doses daily for 5 days have been well tolerated and have been shown to result in marked increases in urinary lead excretion and decreases in blood lead concentrations. The effects of DMSA have been comparable or superior to the effect of EDTA therapy, without depletion of iron, calcium, or magnesium. Mild, transient increases in SGPT levels occurred in 2 of 18 adults treated [Graziano, 1986]. Other studies of DMSA chelation have used dose regimens of 30 mg/kg for 2 days and 20 mg/kg for an additional 5 days. Again, as with EDTA, rebound in blood lead levels and recurrence of symptoms may indicate the need for additional courses of treatment. Hepatic and renal function should be monitored closely.

The use of DMSA in pregnant women is thus far unstudied. There are animal experimental data which demonstrate a teratogenic and fetotoxic effect of high-dose DMSA in pregnant mice [Domingo et al., 1988]. At the current time, the clinical indications for treating a pregnant woman with DMSA must be weighed against the potential hazard to the fetus from the fetotoxic effects of DMSA.

Rats dosed with lead acetate exhibited no redistribution of or increase in the concentration of lead in the brains of animals chelated with DMPS (2,3-dimercapto-1-propanesulfonic acid), structurally similar to DMSA [Aposhian et al., 1996]. DMSA treatment has been shown to be effective in reducing the neurotoxicity of lead in a rat model [Gong and Evans, 1997].

Sub-Populations at Special Risk

Construction workers with pre-existing hypertension, neurological, renal, or reproductive dysfunction represent groups for whom exposure to lead poses potentially greater risk. There is scant information in the literature regarding the consequences of lead intoxication for progression or exacerbation of such conditions, whether related to previous

episodes of lead poisoning or due to other causes. There are no provisions in the OSHA standard for lead exposure in construction which address this issue, and the application of the Americans with Disabilities Act to this setting has not been adequately tested as a possible recourse.

A worker with a pre-existing illness should be advised of the information available about the adverse health effects of absorption of lead, with particular focus on the toxic effects relevant to the individual's specific disease. If the individual chooses to remain in his or her trade, even when potential exposure to lead is an inevitable aspect of the work tasks, close clinical monitoring of the functional status of the affected organ system is warranted, and the need to reduce exposure to the lowest level feasible should be vigorously emphasized. If evidence of further functional compromise emerges during clinical monitoring, the physician may choose to recommend discontinuance of work which entails exposure to lead. Discussion of workers' compensation is appropriate in such instances.

In similar fashion, men who wish to father children and women who are pregnant or contemplating pregnancy should be advised of the possible reproductive toxicity associated with exposure to lead and the potential effects on development of the fetus. The possible mobilization from skeletal stores during pregnancy should be discussed as well. Whether to continue in a work setting where lead exposure may occur remains the woman's choice, but the decision should be guided by an understanding of what is known and the remaining uncertainties regarding the risks involved in further exposure to lead.

PREVENTING LEAD POISONING IN THE CONSTRUCTION SETTING

Provisions of the OSHA Standard for Lead Exposure in Construction (29 CFR 1926.62)

The physician plays a pivotal role in many aspects of the OSHA standard for exposure to lead in the construction setting, promulgated in 1993. It is necessary, therefore, that the physician be well acquainted with the provisions of standard in order to protect the welfare of the lead-exposed worker. Specific provisions of the standard are triggered by defined levels of exposure or by the performance of defined tasks, as follows:

Action level (AL): 30 $\mu\text{g}/\text{m}^3$

Exposure at or above the AL requires the employer to provide blood testing, worker training and air monitoring.

Permissible exposure limit (PEL): 50 $\mu\text{g}/\text{m}^3$, 8 h time-weighted average (TWA)

Exposure at or above the PEL requires that the employer:

- Develops and implements a written compliance program, including the roles and responsibilities of personnel involved in implementation, the competent person, engineering and work practice controls, and record keeping.
- Provides air monitoring and notifies workers of results.
- Sets up hygiene facilities, including change areas, clean eating areas, showers, and hand washing stations.
- Uses HEPA vacuums or wet methods for cleaning.
- Posts warning signs: no eating or smoking.
- Provides respirators, fit testing, and medical evaluations.
- Inspects, cleans, maintains, and stores respirators properly.
- Provides protective clothing and other equipment.
- Provides blood lead, ZPP/FEP monitoring.
- Trains workers about lead hazards, controls, and the use of respirators.
- Has competent persons make daily inspections and keep logs.
- Provides medical examinations if blood lead levels are $\geq 40 \mu\text{g/dL}$ or if symptoms are reported.
- Removes workers from exposure if blood lead levels are $\geq 50 \mu\text{g/dL}$.

Task-Based Exposure Presumptions

The OSHA standard presumes specific levels of exposure for various common lead-disturbing tasks and

requires the provision of respirators, protective clothing, hygiene facilities, and housekeeping procedures until air monitoring results show that these protections are not needed.

Task	Presumed exposure ($\mu\text{g}/\text{m}^3$)	Respirator required
Manual demolition, manual scraping, power tool cleaning with dust collector	50–500	Half-face APR with HEPA
Rivet busting, grinding, scaling, needle gunning without dust collector, moving containment, cleaning up abrasives	500–2500	Full face APR with HEPA, forced air, or PAPR
Torch cutting, burning, welding, abrasive blasting	2500+	Airline with pressure demand

APR, air purifying respirator; HEPA, high efficiency particulate air filter; PAPR, powered air purifying respirator.

Biological Monitoring of Exposure to Lead

The OSHA standard is specific with regard to the necessity for and the frequency of biological monitoring for lead exposure in the construction setting. The standard

explicitly points to the value of blood lead monitoring in protecting the health of exposed workers and identifying opportunities for industrial hygiene intervention. The provisions for testing at first hire and for ongoing biological monitoring are as follows.

Initial medical surveillance (blood testing at onset of work on a “lead project”)

- Not mandatory for employees, but must be provided by the employer.
- Required for all workers exposed to AL for at least one day.
- Blood lead analyses must be performed in a laboratory approved by OSHA.
- Blood lead monitoring includes blood lead level; zinc protoporphyrin (ZPP) or free erythrocyte protoporphyrin (FEP) level at baseline.
- Employer is to notify employee of the results of blood lead testing within five days of receipt.
- If the blood lead level at baseline is $\geq 40 \mu\text{g/dL}$, biological monitoring is to be continued every two months until two consecutive tests show blood lead levels to be $<40 \mu\text{g/dL}$.

Medical surveillance program (ongoing biological monitoring)

- Not mandatory for employees, but must be provided by the employer.
- Required for all workers exposed to the AL for ≥ 30 days per year.
- Blood lead analyses must be performed in a laboratory approved by OSHA.
- Employer is to notify employee of results of blood lead testing within five days of receipt.
- Blood lead monitoring includes blood lead level; zinc protoporphyrin (ZPP) or free erythrocyte protoporphyrin (FEP) level at least every two months for the first six months and every six months thereafter.
- If the blood lead level is ≥ 40 $\mu\text{g}/\text{dL}$, the monitoring frequency is increased to at least every two months and not reduced until two consecutive tests show blood lead levels to be <40 $\mu\text{g}/\text{dL}$.
- If the blood lead level is ≥ 40 $\mu\text{g}/\text{dL}$, the employer must notify the employee that the standard requires Medical Removal Protection benefits (see below) when the blood lead level is ≥ 50 $\mu\text{g}/\text{dL}$.
- If the blood lead level is ≥ 50 $\mu\text{g}/\text{dL}$, a second blood lead test must be performed within two weeks after the employer receives the first result.

The OSHA standard also provides for clinical evaluations of lead-exposed workers who meet specific criteria:

Medical examinations

- To be provided at least annually for all workers with blood lead levels ≥ 40 $\mu\text{g}/\text{dL}$, at any time during the preceding 12 months, who are exposed at or above the AL for ≥ 30 days per year.
- Not mandatory for employees, but must be made available by the employer.
- To be performed by or under the supervision of a licensed physician.
- To be provided without cost to employees at a reasonable time and place.
- The annual examination includes work and medical histories; physical examination; blood lead level; ZPP or FEP level; hematocrit, hemoglobin, peripheral smear morphology and RBC indices; routine urinalysis (specific gravity, sugar, and protein determinations, microscopic examination); blood urea nitrogen, and serum creatinine.

In addition, the standard ensures access to physician consultation under other defined circumstances:

Medical consultations

- **Required to be provided as soon as possible if:**
- The employee notifies the employer that he/she has developed symptoms commonly associated with lead toxicity.
- The employee notifies the employer that the employee desires advice concerning the effect of lead on his/her reproductive capacity.
- The employee notifies the employer that she is pregnant.
- The employee has difficulty in breathing during fit testing or the use of a respirator.
- The employee is removed under the Medical Removal Protection provision.
- **The content of the examination during a consultation is at the discretion of the physician.**
- **A pregnancy test or male fertility test (at minimum: analyzing sperm number, motility and morphology) must be provided on the request of the employee.**

When clinical assessment has been performed for any of the above-listed reasons, the worker has a right to another physician's evaluation and opinion:

Multiple physician review mechanism

- Following the first clinical evaluation, the employer must notify the employee that he/she has the right to a second medical opinion. The employee may designate a second physician to review the findings, determinations or recommendations of the medical consultant chosen by the employer. Notification of the employee's desire to seek a second opinion must be given within 15 days of the employer's notification that a second physician's review is available. The employee must take steps to obtain an appointment for this review within the 15 days.
- The second physician may conduct examinations, consultations, and laboratory tests deemed necessary to facilitate the review. If there are differences in findings, determinations or recommendations from the first clinical evaluation, the two physicians are to communicate to resolve these differences.
- A third physician may resolve disagreements between the first two physicians if their communication does not resolve the conflicts. The cost of this multiple physician review process is to be borne by the employer.
- Where a final medical determination results in a recommendation for special protective measures or limitations on an employee's exposure to lead, the employer must follow the recommendation.
- The special protective measures or limitations may be removed by the employer when a subsequent final medical determination concludes that these are no longer necessary.

The standard provides for the medical removal of workers with blood lead levels $\geq 50 \mu\text{g/dL}$ or with other medical basis for removal, requiring that such employees be placed on tasks which entail much less exposure to lead or be permitted to remain off work while drawing full pay. While removal is absolutely required when blood lead levels

exceed $50 \mu\text{g/dL}$, removal should be considered for levels $>40 \mu\text{g/dL}$, especially when there is evidence of a trend of increasing levels. These provisions of the standard do not preclude the employee from filing a workers' compensation claim for the effect on his/her health resulting from excessive absorption of lead.

Medical removal protection

- The employer must remove an employee from work entailing exposure to lead at or above the AL on each occasion that a blood lead test and a follow-up blood lead test are both $\geq 50 \mu\text{g/dL}$. The follow-up test must occur within two weeks of the first.
- The employer can return the employee to former job status when two consecutive blood samples are $\leq 40 \mu\text{g/dL}$.
- The employer must remove an employee from work which entails exposure to lead at or above the action level on each occasion that a physician's medical determination results in a finding that the employee has a medical condition which places the employee at increased risk of "material impairment to health" from exposure to lead.
- The employer can return the employee to former job status when a subsequent medical determination concludes that the employee no longer has a medical condition which places the employee at increased risk of "material impairment to health" from exposure to lead. This decision can be appealed to multiple physician review.
- If a removed employee files a claim for workers' compensation payments for lead-related disability, the employer must continue to provide medical removal protection benefits pending disposition of the claim. The employer's medical protection obligation may be reduced by the amount awarded to the employee for earnings lost during the removal.
- For employees removed because of an elevated blood lead level whose blood lead level has not declined in the past 18 months of removal enough to permit return to former job status (to $\leq 40 \mu\text{g/dL}$), the employer must make available a medical examination to determine whether the employee can be returned to former job status. If return is not judged possible, the final medical determination should state what steps should be taken to protect the employee's health. Medical removal benefits are to be provided by the employer until the employee is returned to former job status or a final medical determination concludes that the employee is incapable of ever returning to former job status. Despite having an otherwise unacceptable blood lead level, if an employee is returned to former job status, removal in the future must be decided by a final medical determination.

The OSHA standard states unequivocally: Prophylactic chelation of any employee by anyone employed, retained, supervised or controlled by the employer is prohibited.

Limitations of the OSHA Standard for Exposure to Lead in Construction

The promulgation of the OSHA standard represented an important enhancement of the protections afforded to construction workers, and, when implemented, has been shown to be capable of reducing blood lead levels among lead-exposed workers [Levin et al., 1997]. Given the short duration of many construction projects, however, and the variability of conditions on any specific project, it is reasonable to question the adequacy of the frequency of blood testing required by the standard.

Blood lead levels can rise quite rapidly under conditions of excessive exposure [Marino et al., 1989]. If biological monitoring is to protect the health of individual workers and to serve as a guide to industrial hygiene intervention to reduce exposure to lead, blood testing every two months is likely to miss opportunities to develop and implement strategies for worker protection. The integration of industrial hygiene strategies for exposure reduction with the biological monitoring program is key to a public health approach to preventing lead toxicity in the construction environment.

In similar fashion, the exclusive use of threshold levels ($\geq 40 \mu\text{g/dL}$) to trigger testing on an every two month basis fails to make use of information which could be derived from an analysis of trends in individual and group blood lead concentrations. For example, an increase of $10 \mu\text{g/dL}$ in an individual's blood lead level in a given testing interval should prompt an industrial hygiene assessment of exposure conditions, even if the absolute blood lead level is below $40 \mu\text{g/dL}$.

In response to the latter concern, the New York State Department of Transportation has adopted guidelines for bridge and highway repair projects which incorporate an increase of $10 \mu\text{g/dL}$ or more between successive tests as a trigger event for an on-site investigation and the implementation of a corrective action by the industrial hygienist [NYS DOT, 1995].

The OSHA standard provides for repeat blood testing as long as two weeks after a blood lead level of $\geq 50 \mu\text{g/dL}$ is obtained before removal from exposure to lead is required. Under heavy exposure conditions, e.g., burning through lead-based paint, there is a considerable risk of severe lead poisoning within the ensuing two week period if exposure is not better controlled. There is no requirement that a blood lead level in excess of $50 \mu\text{g/dL}$ should initiate an industrial hygiene assessment of the exposure conditions for the affected worker.

To identify poorly controlled exposures in the changing construction setting, blood lead testing on a monthly basis, at the outset of work on a lead-disturbing project and at such time as the content and/or location of the work changes, can provide information to guide prompt evaluation and intervention. If an individual worker's blood lead level rises to $\geq 25 \mu\text{g/dL}$ or if the interval increment in blood lead concentration for an individual is $\geq 10 \mu\text{g/dL}$, an industrial hygiene investigation should be carried out and monitoring on a monthly basis should be continued. When blood lead concentrations remain below $25 \mu\text{g/dL}$ and interval increments remain below $10 \mu\text{g/dL}$ for three months, the blood testing frequency can be maintained on a bi-monthly schedule. After six months of monitoring during which blood lead concentrations remain below $25 \mu\text{g/dL}$ and interval increments remain below $10 \mu\text{g/dL}$, testing can be performed every six months, so long as the content of the work and the exposure conditions remain stable. This biological monitoring scheme offers a reasonable opportunity to control exposure before workers experience excessive absorption of lead.

Education

A key element in the prevention of lead poisoning in the construction environment is worker and management education about health effects of lead and the control of exposure. Such training is a requirement of the OSHA standard. The identification of excessive lead absorption in an individual or group of workers should always prompt educational efforts to increase awareness regarding the hazards associated with such exposures and how exposure can be reduced.

Under the provisions of the OSHA standard, training and education are the responsibilities of the employer, as is the medical surveillance of lead-exposed workers. A cogent argument has been made for locating these responsibilities in public health agencies, which could prepare accurate educational materials and provide education regarding the health risks, safe work practices, and regulatory requirements under the standard [Keogh and Gordon, 1994].

Industrial hygiene assessment of the work site should be attempted, prompted by the identification of index cases of excessive exposure or rising blood lead levels among the work force. This may be accomplished by working through the construction contractor and the consultant industrial hygienist employed on the project or may involve the services of an industrial hygienist associated with an independent clinical center, if access to the work site can be secured. Those tasks which disturb lead-based paint or otherwise generate a lead aerosol should be evaluated with regard to the use of engineering controls and the use of adequate personal protective equipment (respiratory protection). The availability of hand washing facilities, the

prevention of smoking at the job site, and other factors which can influence exposure should be assessed.

Industrial hygiene interventions to correct inadequate controls on lead exposure should be attempted, by the contractor's consultant or, when possible, by the clinical center's industrial hygienist. This often will include a review of the training and education program provided to potentially lead-exposed workers.

Coordination with workers' representatives or unions to enlist their assistance in educating workers at risk of lead poisoning and accomplishing necessary changes at particular worksites should be attempted. This should be done only with the full understanding and cooperation of the affected workers, since issues of confidentiality must be taken into account.

Coordination with public health agencies may be necessary when attempts to correct identified exposure problems are met with unwillingness to make necessary changes, as evidenced by persistently elevated blood lead levels among workers on the project or new cases of lead intoxication arising from the specific worksite. The involvement of such agencies as OSHA should be discussed fully with the worker(s) involved, since individual employment jeopardy must be considered.

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