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Case Studies in Environmental Medicine

17

Mercury Toxicity

Environmental ALERT...

- ***Some latex house paints release dangerous levels of mercury vapor. Check the label. Latex paints sold after August 1990 must carry a warning if they contain a mercury additive.***
- ***A recent study by the National Institutes of Health indicates that the small amounts of mercury released in the mouth by dental amalgams pose no known danger to health.***
- ***Because mercury has several forms and produces subtle effects at chronic low-level exposures, mercury toxicity can be a difficult diagnosis to establish.***

This monograph is one in a series of self-instructional publications designed to increase the primary care provider's knowledge of hazardous substances in the environment and to aid in the evaluation of potentially exposed patients. See page 23 for further information about continuing medical education credits and continuing education units.

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How to use this issue...

This issue begins with a composite case study that describes a realistic encounter with a patient. This description is followed by a pretest. The case study is further developed through Challenge questions at the end of each section. To benefit fully from this monograph, readers are urged to answer each question when it is presented. (Answers to the Pretest and Challenge questions are found on pages 21-2.) The monograph ends with a posttest, which can be submitted to the Agency for Toxic Substances and Disease Registry (ATSDR) for continuing medical education (CME) credit or continuing education units (CEU). See page 23 for further instructions on how to receive these credits.

The objectives of this monograph on mercury are to help you:

- ❑ **Explain why mercury may be an acute and chronic health hazard**
- ❑ **Describe the known factors contributing to mercury toxicity**
- ❑ **Identify potential environmental or occupational sources of exposure to mercury**
- ❑ **Identify evaluation and treatment protocols for mercury exposure**
- ❑ **List sources of information on mercury**

Contents

This issue is prepared with the assistance of those who share a common concern for physician education, public health, and the environment, including the following organizations: American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American College of Emergency Physicians (ACEP), American College of Occupational and Environmental Medicine (ACOEM), American Medical Association (AMA), Association of State and Territorial Health Officials (ASTHO), and the Society of Teachers of Family Medicine (STFM). Final responsibility for the contents and views expressed in this monograph resides with ATSDR.

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Case Study

A 3-year-old boy with irritability, digital erythema, and leg pain

A 3-year-old boy is brought to your office by his parents, who state that the child refuses to play and prefers to lie on his bed. His parents note that about a month ago, he seemed to withdraw and become cranky. Recently, he has experienced night sweats. On those nights the child has felt warm, but his parents did not take his temperature. The child has had no other symptoms, such as a runny nose or cough, and has not lost weight.

History reveals that the patient had recurring ear infections this past winter, which were treated with oral antibiotics. His growth and development have been normal; he is in the 90th percentile for weight and height. The child's immunizations are up to date, and he is on no medications.

During physical examination, the boy is uncooperative and crying. He refuses to walk or stand and says that his legs "hurt." He is afebrile, has a heart rate of 130 per minute and respirations of 16 per minute. He is sweating, and his nose, fingers, and toes are erythematous; the skin on his fingers and toes is peeling. The oral pharynx and abdomen appear normal upon examination; his lungs are clear. He does not have point tenderness in his legs, and he has full range of motion in knees and hips. His ankles are not edematous. Results of the neurologic examination are normal; there is no muscular atrophy. Other findings are unremarkable.

Three months ago, the child, his parents, and his 6-year-old sister moved into a freshly painted house. The parents report that their daughter appears healthy and is doing well in first grade. Both parents are school teachers in good health. The family has no pets and has not traveled within the past year. Until recently, the boy has enjoyed most social activities with his family.

(a) What should be included on the patient's problem list?

(b) What is the differential diagnosis for this patient?

(c) What tests would you recommend to confirm or rule out a diagnosis?

(d) What treatment and followup would you recommend?

Answers can be found on pages 21-2.

Exposure Pathways

- **Elemental mercury vapor accounts for most occupational and many accidental exposures.**
- **The major source of organic methylmercury exposure in the general population is fish consumption.**
- **Mercury-containing dental amalgams have not been proven to cause adverse health effects.**

Mercury (Hg) is a metal found in the environment in its elemental state and as organic and inorganic compounds. For 3000 years, mercury, in various forms, has been used in medicine and industry. Although most medicinal uses have been discontinued, industrial uses of mercury are increasing.

Mercury exists in three forms: elemental mercury (Hg^0), inorganic mercury salts (Hg^{1+} and Hg^{2+}), and organic mercury. Elemental mercury is a silver-gray liquid at room temperature that vaporizes readily when heated. Commonly referred to as quicksilver or metallic mercury, it is used in thermometers, thermostats, switches, barometers, batteries, and other products. Elemental mercury vapor accounts for most occupational exposures.

The intermediate oxidation state, Hg^{1+}, forms numerous mercurous salts; the best known is mercurous chloride or calomel, which was commonly used in teething powders and other medicines until its adverse effects were publicized in 1948. The highest valence state, Hg^{2+}, forms a variety of mercuric salts, which are used to inhibit bacterial or fungal growth. Most mercurous and mercuric salts readily disassociate into ions in the body.

Under appropriate conditions, Hg^{2+} can covalently bind carbon to form organomercury compounds; the most important in terms of human exposure is methylmercury (MeHg). MeHg is the form most frequently involved in mercury food poisoning. Elemental mercury and MeHg compounds have a greater ability to cross cell membranes than do the mercurous or mercuric salts and are consequently more neurotoxic than mercury salts.

The major source of atmospheric mercury is the global off-gassing of mercury from soils and surface waters. Burning of fossil fuels, particularly coal, contributes to the level of mercury in the atmosphere. The airborne level is increased by disposal of solid waste (e.g., thermometers, electrical switches, and batteries) in landfills; application of mercury-containing paints, fungicides, and pesticides; and combustion of waste oils.

Weathering of mercury-bearing rock and industrial effluents are the major sources of mercury contamination in water. Elevated mercury concentrations have been detected in approximately 25% of the groundwater and surface-water samples from 2783 hazardous waste sites tested by the Environmental Protection Agency (EPA). Groundwater surveys also have detected elevated mercury concentrations in some drinking-water supplies. Industrial processes that may produce mercury-containing effluents include chlorine and caustic soda production, mining and ore processing, metallurgy and

electroplating, chemical manufacturing, ink manufacturing, paper milling, leather tanning, textile manufacturing, and pharmaceutical production.

Any mercury compound released into the environment becomes available for potential methylation to MeHg by microorganisms indigenous to soils and waters. Higher methylating rates are associated with acidified waters. MeHg in surface waters rapidly accumulates in fish and other aquatic organisms. The mercury concentration in fish at the top of the food chain is typically biomagnified up to 100,000 times the concentration in surrounding waters.

In the general population, diet is the major source of mercury exposure, primarily through fish consumption. Predacious fish (e.g., pike in freshwater, tuna and swordfish in marine water) can have more than 50 times the average mercury concentration found in most other fish. Between 70% and 90% of the total mercury detected in fish is in the form of MeHg. The U.S. Food and Drug Administration (FDA) is responsible for regulating commercial fish. Regulations require that marketed fish contain no more than 1 part per million (ppm) of mercury. Many states have lower advisory levels for sport fish. Other potential sources of dietary exposure are the consumption of fish-eating birds and mammals and consumption of game birds in areas where mercury-containing pesticides have been used. During the winter of 1971-72, thousands of Iraqis were poisoned by consuming homemade bread prepared from seed wheat that had been treated with a MeHg fungicide.

According to EPA, approximately 30% of interior latex paint manufactured before 1990 contained mercury compounds to prevent bacterial and fungal growth. In 1989, a case of acrodynia (a rare disease in children caused by mercury; see page 13) in a 4-year-old boy occurred 10 days after the child's home was painted with a mercury-containing interior latex paint and was not ventilated. Mercury-containing paint can raise the total indoor air mercury concentration by 1000 times the level before painting. Paint manufacturers agreed to stop using mercury in interior paint after August 20, 1990; however, sale of existing stocks of interior latex paints was allowed until July 1991. Paint manufacturers also have agreed to place labels on mercury-containing exterior paint with a warning that the paint is for outdoor use only. Mercury use in exterior paint was discontinued after September 1991. Mercury-containing joint compound, plasters, and adhesives must be labeled appropriately; sale to distributors was allowed until June 1991. Because many people keep partly used cans of paint for repainting, pre-1990 paints may continue to be a source of mercury exposure for years.

Medical treatments and some cosmetics constitute another source of potential mercury exposure. Mercurials have been used for hundreds of years for a variety of therapeutic purposes including cathartic, diuretic, antisyphilitic, antiseptic, antipruritic, anti-inflammatory, antiparasitic, and vermifuge. Metallic mercury has been used by Mexican-American and Asian populations in folk remedies for chronic stomach disorders and by Latin-American and Caribbean natives in occult practices. Mercurials are still used as preservatives in some eye drops, eye ointments, nasal sprays, vaccines, and as antiseptics and diuretics. Gammaglobulin preparations contain Merthiolate™ *, a mercury-containing biocide. Elemental mercury in a Miller-Abbott tube, which is used for intestinal decompression, provides the weight that assists the tube in traversing the GI tract.

Silver dental amalgams, which have been used for the past 150 years to fill cavities in teeth, can be 50% elemental mercury by weight. About 200 million mercury restorations are performed in the United States each year; at least one-half of those use silver amalgam. The mechanical action of chewing on an occlusive filling releases trace quantities of mercury vapor, which are partially absorbed. Typically, exposure to mercury from amalgams is less than exposure from foods such as tuna or swordfish that contain MeHg, a more toxic form of mercury. It is estimated that people with many amalgam fillings receive less than 1% of the daily mercury vapor dose that is considered occupationally safe. A National Institutes of Health expert panel recently concluded that amalgam fillings pose no significant risk of side effects and should not be replaced simply because they contain mercury.

* Use of trade names is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Challenge

(1) What are the possible exposure sources of mercury for the patient in the case study?

Who's at Risk

A 1980 survey by the National Institute for Occupational Safety and Health (NIOSH) estimated that 70,000 workers, of whom about one-third were women, were potentially exposed to mercury (primarily mercury vapor) in the workplace. Most of these workers were employed as laboratory technicians, registered nurses, and machine operators. Household members of occupationally exposed workers may also be at increased exposure risk because mercury can be brought into the home on contaminated clothes.

Personnel potentially exposed to mercury include, but are not limited to, the following:

- chlorine and caustic soda production workers
- cosmetic producers
- dental personnel
- electroplators
- explosives manufacturers
- felt makers and leather tanners
- grinding machine operators
- hazardous waste site personnel
- ink manufacturers
- laboratory personnel
- manufacturers of batteries, fluorescent lamps, mercury vapor lamps, switches, rectifiers
- metallurgists
- miners and processors of cinnabar (HgS), gold, silver, copper, zinc
- paint and pigment manufacturers
- painters
- paper millers
- pesticide/fungicide production and application workers
- pharmaceutical producers
- plumbers

Fetuses, infants, and children are at increased risk of adverse effects of MeHg. MeHg readily crosses the placenta during the prenatal stage, when the nervous system is most sensitive to mercury poisoning. Because MeHg concentrates in breast milk, nursing infants can be affected.

Children are attracted to the appearance and unique properties of liquid elemental mercury and are at risk of ingesting elemental mercury, as well as mercury-containing dust and soil, because of natural mouthing behaviors. Infants and children are at increased risk of inhaling elemental mercury because mercury vapor is heavier than air and tends to settle to the floor.

- **Workers using mercury or mercury-containing products, as well as their household members, may be at increased risk of exposure to mercury vapor.**
- **Fetuses, infants, and children are at greatest risk of MeHg's adverse effects.**
- **Children are at increased risk of exposure to elemental mercury vapor in the home because mercury vapor tends to settle to the floor.**

Because many people are increasing their consumption of fish in an effort to lower blood cholesterol concentrations, mercury exposure through diet may be increasing. Mercury is a contaminant of many fresh and marine waters. In the 1950s, hundreds of people were mercury-poisoned in Japan after consuming fish from Minamata Bay. The incident caused 41 deaths and at least 30 cases of infantile cerebral palsy. The source of contamination was effluent discharged into the bay from a factory using a mercury catalyst to make vinyl chloride.

Neurologic and behavioral disorders have been observed in persons after ingestion or dermal application of inorganic mercury-containing compounds in teething powders, skin-lightening ointments, and laxatives. Most of these products have been withdrawn from the market or are no longer available in the United States. Yellow mercuric oxide reportedly caused acrodynia (see Signs and Symptoms) in a 4-month-old boy being treated for eczema. Long-term abuse of a mercury-containing laxative was the cause of death in at least one patient.

Challenge

(2) Besides the patient, who else in the case study may be at risk of mercury exposure?

Biologic Fate

Elemental Mercury

Mercury's absorption and metabolism depend on its chemical and physical form. Inhaled as a vapor, elemental mercury is almost completely absorbed (about 80%) and diffuses rapidly across the placental and blood-brain barriers (Table 1). At the cellular level, dissolved vapor is oxidized to Hg^{2+}. Ethanol, even at nonintoxicating levels, inhibits mercury oxidation in the blood and prolongs elemental mercury's half-life in the body.

- **The chemical and physical forms of mercury determine its absorption, metabolism, distribution, and excretion pathways.**

When ingested, elemental mercury is poorly absorbed from the gastrointestinal tract (about 0.01%). The surface of the metal probably becomes coated rapidly with endogenous sulfur-laden compounds, which impairs diffusion across the gastrointestinal mucosa. A 17-year-old boy reportedly ingested 204 grams (g) of elemental mercury without systemic toxicity. Animal studies indicate that elemental mercury as a liquid or vapor can be absorbed percutaneously.

- **Elemental mercury is almost completely absorbed when inhaled, but poorly absorbed when ingested. It readily crosses the blood-brain barrier.**

Table 1. Clinical importance of various forms of mercury

Form	State	Source	Absorption*	Primary Effects	Secondary Effects
Inorganic Elemental					
Liquid Hg†	Hg^0	Thermometers, barometers	Dermal contact: minimal absorption Ingestion: poor absorption	§	
Mercury Vapor†	Hg^0	Industrial	Inhalation: 80% absorbed Percutaneous: minimal absorption	Lungs, skin, eyes, gingiva	CNS¶, kidneys
Salts					
Mercurous	Hg^{1+}	Medicines, antiseptics	Ingestion: ~10% absorbed Dermal contact: lethal doses can be absorbed by animals	Kidneys, GI tract¶	CNS
Mercuric	Hg^{2+}				
Organic					
Methylmercury†	CH_3Hg-	Fish	Ingestion: 100% absorbed Inhalation: absorbed readily	CNS	
Phenylmercury	C_6H_5Hg-	Fungicides, bactericides	Ingestion: 80%-100% absorbed Dermal contact: See Salts above	Kidneys	CNS

* In humans, the biologic half-life of all forms of mercury is 40 to 70 days.
† Crosses the blood-brain barrier.
§ Liquid elemental mercury is poorly absorbed through the intestinal tract (0.01%) or dermally; systemic toxicity is rare.
¶ CNS = central nervous system; GI tract = gastrointestinal tract

The urine and fecal elimination pathways account for most of the excretion of elemental mercury. Exhalation of mercury vapor and secretion of mercuric ions in saliva and sweat contribute to the elimination process. The biologic half-life of inhaled elemental mercury in humans is approximately 60 days.

Mercury salts

- **Mercuric salts are generally more toxic than mercurous salts.**
- **Mercury salts do not cross the blood-brain barrier as readily as elemental mercury does.**

On average, less than 10% of an ingested mercury salt is absorbed from the gastrointestinal tract. Dermal absorption of ionic mercury salts also can cause toxicity. In general, mercuric (Hg^{2+}) salts are more soluble and produce more serious poisonings than mercurous (Hg^{1+}) salts. Mercuric salts are usually colorless or white crystals or intensely colored yellow or red powders; they include mercuric chloride (antiseptic and disinfectant), mercuric cyanide and mercuric oxide (topical antiseptics), and mercuric nitrate (used in working with felt). Mercurous salts are typically colorless, white, or light yellow powders; they include mercurous acetate (antibacterial agent), mercurous chloride or calomel (cathartic, diuretic, antiseptic, and antisyphilitic agent), mercurous nitrate (used to blacken brass), and mercurous oxide (used to make electric batteries).

The tissue distribution and excretion pathways of mercury salts are similar to those of mercury vapor; however, mercuric and mercurous ions cross the blood-brain and placental barriers to a much lesser extent than inhaled elemental mercury. In humans, mercury salts have a shorter biologic half-life (about 40 days) than inhaled elemental mercury.

Organic mercury

- **Organomercurials are absorbed well regardless of exposure route.**
- **MeHg concentrates mostly in the blood and brain.**

Organomercury compounds are readily absorbed by inhalation, dermal contact, and ingestion. MeHg is distributed uniformly to all tissues, although it concentrates more in the blood and brain than elemental mercury or mercury ions do. About 90% of MeHg is found in the red blood cells, where it is metabolized to mercury ions at a slow rate. The major route of MeHg excretion (about 90%) is through bile into the feces; urinary excretion accounts for most of the remaining 10%. The biologic half-life of MeHg is about 70 days in humans.

Although considered organomercurials, phenylmercury compounds are absorbed less efficiently by the gastrointestinal system than MeHg compounds. Because phenylmercury is rapidly metabolized in the body to Hg^{2+}, its effects are similar to those of mercury salts. Metabolites of phenylmercury are excreted mainly in the urine.

Physiologic Effects

Effects of mercury toxicity manifest primarily in the central nervous system (CNS) and kidneys, where mercury accumulates after exposure. The duration, intensity, and route of exposure, and the form of mercury influence which systems are affected. The primary organ system affected by chronic exposure to elemental mercury and organomercury compounds is the nervous system; the primary organs affected by chronic exposure to mercury salts are the kidneys. In acute poisonings, the respiratory system is affected by inhaled elemental mercury and the gastrointestinal system by ingested mercury salts. The cardiovascular system may be affected secondarily.

- **The central nervous system and kidneys are key targets of mercury toxicity.**
- **In acute poisonings, the respiratory and gastrointestinal systems can be affected.**

The precise mechanism of action for all forms of mercury is unclear. Mercury ions (Hg^{1+} and Hg^{2+}) alter the structure and function of enzymes and other proteins by binding to sulfhydryl groups. Mercury may also interfere with cellular metabolism by binding to amine and phosphoryl groups. MeHg and high levels of inhaled elemental mercury are able to cross the blood-brain and placental barriers. In humans, MeHg exposure has resulted in pronounced adverse neurologic effects in the fetus. The effects of elemental mercury on the human fetus have not been studied thoroughly.

Neurologic Effects

CNS effects result primarily from exposure to elemental mercury vapor and to MeHg. These forms of mercury cross the blood-brain barrier readily and can produce irreversible brain damage. MeHg ingestion leads to delayed CNS symptoms that may not manifest until months after the initial exposure, and early symptoms are often nonspecific, such as malaise, blurred vision, or hearing loss. The peripheral nervous system also may be affected. Some investigators suggest that alteration in neurotransmission may be one mechanism of action for mercury-induced neurotoxicity.

- **MeHg and inhaled elemental mercury accumulate rapidly in the CNS.**

Renal Effects

After inorganic salts or phenylmercury compounds are ingested, a large amount of mercury may accumulate in the kidneys, producing a generalized increase in the permeability of the tubular epithelium. Exposure to mercury vapor or to mercury salts produces an apparently dose-dependent proteinuria or nephrotic syndrome. Acute tubular necrosis with resultant renal failure may occur.

- **Severe renal damage can result from ingestion and absorption of mercury salts.**

Developmental Effects

- **Evidence indicates that MeHg causes developmental effects.**
- **Data are limited on the fetal effects caused by forms of mercury other than MeHg.**

Studies of MeHg concentrations in the blood of newborn infants show a significant correlation with maternal blood levels. In MeHg poisonings, damage to the fetal nervous system is widespread and probably involves derangement of developmental processes such as neuronal migration and neuronal cell division. MeHg also may have a high affinity for fetal hemoglobin. Infants born to women who had ingested flour made from grain treated with a MeHg fungicide had brain damage manifested by mental retardation, ataxia, deafness, constriction of the visual fields, blindness, microcephaly, cerebral palsy, and disturbances in swallowing. In experimental animals, exposure to elemental mercury vapor or administration of mercury salts has produced developmental anomalies, but the relevance of these findings to humans is unknown.

Other Effects

- **Severe tissue damage to the lungs (through inhalation exposure) and GI tract (through ingestion exposure) has been reported.**

Respiratory and gastrointestinal effects can occur in acute mercury poisonings. Inhalation of elemental mercury has caused severe pulmonary tissue damage; autopsy has revealed dilation of the right ventricle due to respiratory failure in children who died from mercury vapor inhalation. Gastritis and necrotizing ulceration of the intestinal mucosa can result from ingestion of inorganic salts. Liver damage also has been reported in cases of poisoning due to mercury salts. Both increased and decreased blood pressure have been associated with elemental mercury exposure.

Chromosomal aberrations have been found in some persons working with mercury. There is insufficient evidence to associate mercury with cancer in humans.

Challenge

(3) Additional information for the case study: The patient's mother is 2 months pregnant. Is the fetus at risk of mercury exposure?

Clinical Evaluation

History and Physical Examination

A complete history of a patient with possible mercury toxicity should contain the following information:

- occupation and hobbies of all home occupants.
- recent application of mercury-containing caulks, latex paint, and other materials in constructing or renovating homes and other buildings.
- recent move. Previous tenants may have spilled mercury, or the new home recently may have been painted with mercury-containing paint.
- use of folk medicines. Mercury compounds have been detected in folk and nontraditional healing medications.
- use of cosmetics. Mercury is contained in some mascaras and wave fixatives, and some skin lighteners sold outside the United States.
- use of over-the-counter preparations such as nasal sprays, contact lens solutions, and topical antiseptics.
- source and amount of fish consumed per week.
- use of elemental mercury in a school laboratory.
- playing with mercury. Children are attracted to liquid elemental mercury because of its unique properties.

❑ **A complete history and careful evaluation of the nervous system and kidneys are essential in diagnosing mercury toxicity.**

The nervous system and kidneys should be carefully examined. Recent behavioral changes, such as an increase in irritability or shyness and changes in short-term memory, should be documented. In children, developmental milestones should be evaluated. Blood pressure and liver function also should be assessed. If mercury salts have been ingested and corrosive injury is suspected, endoscopic examination should be performed. If elemental mercury vapor has been inhaled, a chest X ray should be obtained.

Signs and Symptoms

Acute Exposure

Elemental Mercury

❑ **Pulmonary and CNS effects result from inhaling elemental mercury.**

Inhalation of elemental mercury vapor may rapidly produce cough, dyspnea, chest pain, nausea, vomiting, diarrhea, fever, and a metallic taste in the mouth. Stomatitis, colitis, nephrotic syndrome, and salivation may occur. Later, interstitial pneumonitis, necrotizing bronchiolitis, and pulmonary edema may develop. According to one case report, a person experienced symptoms characteristic of amyotrophic lateral sclerosis after an intense elemental mercury vapor exposure; symptoms abated as the body burden of mercury decreased. Children less than 30 months of age appear to be at increased risk for pulmonary toxicity, and death may rapidly result

due to pulmonary dysfunction. Conjunctivitis and an erythematous, pruritic rash have been reported with relatively mild exposures to mercury vapor. Ingested liquid elemental mercury is not absorbed well and therefore poses only limited risk of toxicity. Contact with liquid mercury has been associated with a dermatitis characterized by a papular erythema.

Mercury Salts

❑ **The GI tract and, later, the kidneys are affected by ingestion of mercury salts.**

The acute lethal dose of most mercury salts is approximately 1 to 4 g for adults. Symptoms and signs a few hours after ingestion include a metallic taste in the mouth; nausea, vomiting, and bloody diarrhea; severe abdominal pain; tenesmus; intestinal wall necrosis leading to scarring, fibrosis, and possible stenosis; hematemesis; and cardiovascular collapse due to dehydration. The urine may contain protein, casts, and red blood cells. One day to two weeks after ingestion, urine output may diminish due to acute tubular necrosis. Death due to uremia may result.

Organic Mercury

❑ **Symptoms due to MeHg ingestion typically are nonspecific and delayed.**

The neurologic effects of MeHg ingestion have been well documented after outbreaks of poisoning in Minamata, Japan, (where fish containing MeHg was consumed) and in Iraq (where grain treated with a MeHg fungicide was consumed). In adults, the earliest signs and symptoms are nonspecific and can take months to develop. These include ataxia; paresthesias; malaise; blurred vision; and impaired hearing, taste, and smell. In Japan, the neurologic effects of MeHg were first observed in cats who ate the mercury-contaminated fish, leading to the colloquialism "cat dancing disease."

The signs and symptoms of poisoning due to aryl organomercury compounds (e.g., phenylmercuric acetate) are similar to those of mercury salts.

Chronic Exposure

Elemental Mercury

❑ **Tremor and personality disturbances are characteristic signs of chronic exposure to elemental mercury vapor.**

The most important effects of chronic exposure to elemental mercury vapor involve the nervous system. At chronic low doses, the body oxidizes most of the elemental mercury to mercuric ions (Hg^{2+}), which do not readily cross the blood-brain barrier. At high doses, the body is not able to metabolize the mercury rapidly enough and more elemental mercury reaches the brain. CNS signs and symptoms include psychological changes, insomnia, loss of appetite with weight loss, erethism (characterized by insomnia, excessive shyness, and emotional instability), irritability, headache, and short-term memory loss.

Tremor, though seldom the first sign to appear, is characteristic of exposure; it usually disappears if exposure is stopped. Other peripheral nervous system findings include distal paresthesias, motor and sensory nerve conduction delay, and limb weakness.

Acrodynia, a rare syndrome characterized by severe leg cramps; irritability; paresthesias; and painful pink fingers and peeling hands, feet, and nose, may develop in children exposed to elemental mercury, mercury salts, or phenylmercury (which is rapidly metabolized to Hg^{2+}). It is not known why children but not adults are affected by acrodynia. It is also an enigma why few children exposed to mercury develop acrodynia. If one case is diagnosed, it is likely that other persons have been exposed.

Mercury Salts

According to two case reports, the chronic ingestion of mercury salts in the form of a laxative resulted in irritability, colitis, and chronic renal failure. Gingivitis, stomatitis, and salivation also can occur.

Organic Mercury

The signs and symptoms of chronic exposure to MeHg include a tingling sensation in the extremities; tunnel vision; impaired hearing, taste, and smell; incoordination; tremor; irritability; memory loss; depression; and insomnia. As with acute MeHg exposure, the effects of chronic exposure may be delayed for months. Chronic exposure to MeHg may result in permanent CNS damage.

- **Permanent CNS damage may result from chronic exposure to MeHg.**

Laboratory Tests

Direct Biologic Indicators

Mercury can be measured in blood, urine, and hair. Since mercury has a short half-life in blood (3 days), blood analysis is typically performed shortly after an acute exposure; urine is the best biologic specimen when chronic mercury exposure is suspected. Hair analysis can provide evidence of MeHg exposure.

- **Blood is an appropriate specimen for analysis after acute mercury exposure; a 24-hour urine specimen is preferred in cases of chronic exposure.**

For acute high-level mercury exposure, whole blood is a valid indicator of body burden (and brain concentration of MeHg); for low-level exposure, plasma should be analyzed separately. Blood samples should be collected in vacutainers containing heparin and then refrigerated. In unexposed adults, the blood mercury level rarely exceeds 1.5 micrograms per deciliter (μg/dL); a blood concentration of 5 μg/dL or greater is considered the threshold for symptoms of toxicity.

- **In adults, the background mercury concentration is generally less than 1.5 μg/dL in blood and less than 20 μg/L in urine.**

A 24-hour urine specimen collected in an acid-washed plastic container is the preferred specimen for patients who have been chronically exposed to elemental mercury or mercury salts. A first morning void can provide a close approximation of a 24-hour collection, particularly if it is adjusted for the concentration of the urine (using specific gravity or amount of creatinine present). Since organic mercury is usually excreted through the biliary system, urine levels are not useful in evaluating MeHg exposure.

Urine should be analyzed for mercury by cold vapor atomic absorption spectrophotometry. The Reinsch test, a screening test for heavy metals, is not sufficiently specific or sensitive to detect low levels of mercury. A urinary mercury concentration of less than 20 μg/L in adults is considered background. Urine mercury concentrations from 20 to 100 μg/L are associated with subtle changes on some tests, even before overt symptoms occur (Table 2). Background or toxic urinary mercury concentrations have not been determined for children.

Generally, levels of mercury in hair are not useful in evaluating a patient clinically. A properly handled hair sample can provide evidence of MeHg exposure because MeHg accumulates in hair where its concentration remains constant. Maternal hair samples have been used to provide an estimate of fetal MeHg exposure.

Table 2. Relationship of urinary mercury concentration with effects

Urinary Mercury Concentration (μg/L)	Signs and Symptoms
<20	None
20 to 100	Decreased response on tests for nerve conduction, brain-wave activity, and verbal skills Early indication of tremor on testing
100 to 500	Irritability, depression, memory loss, minor tremor, and other nervous system disturbances Early signs of disturbed kidney function
500 to 1000	Kidney inflammation Swollen gums Significant tremor and nervous system disturbances

Indirect Biologic Indicators

If acute inorganic mercury poisoning is suspected, baseline BUN, creatinine, electrolytes, and urinalysis should be obtained; these values should be monitored continually to evaluate renal toxicity. Urinary $ß_2$-microglobulin and retinol-binding protein levels may be useful in determining renal status. Liver function tests also should be performed.

Challenge

(4) What problem list could you construct for the patient in the case study?

(5) What is your differential diagnosis?

(6) What tests would you order to confirm or rule out your diagnoses?

Treatment and Management

The treatment of inorganic mercury poisoning usually involves the use of chelating agents. Chelating agents contain sulfhydryl groups, which bind mercury ions and facilitate their excretion through urine and feces. Dimercaprol (British anti-Lewisite or BAL) was the first chelating agent used for mercury toxicity and is still widely used for inorganic mercury poisoning. BAL is contraindicated for MeHg poisoning because it has been shown to increase the concentration of MeHg in the brain and therefore exacerbates symptoms. BAL is anticipated to be effective in treating phenylmercury poisoning because phenylmercuric acetate is rapidly oxidized to Hg^{2+} in the body; hence, phenylmercury is similar to inorganic mercury. Possible side effects of BAL include nausea and vomiting, headache, tachycardia, fever, conjunctivitis, blepharospasm, and lacrimation.

In some cases, an alternative or adjunct to parenterally administered BAL is orally administered N-acetylpenicillamine (NAP). Side effects of NAP can include fever, rash, leukopenia, eosinophilia, and thrombocytopenia.

- **Chelation therapy has been used successfully in treating patients who have ingested mercury salts or inhaled elemental mercury.**
- **No antidote exists for patients poisoned with organic mercury; supportive care is recommended.**

Newer derivatives of BAL, such as dimercaptosuccinic acid (DMSA) and 2,3-dimercaptopropane-1-sulfonate (DMPS), are more effective than BAL in experimental studies. DMSA (Succimer™*) is available in the United States; consult your regional certified poison control center or a physician experienced in chelation therapy for more information. DMPS is still an investigational drug and must be used under FDA guidelines. When DMPS was administered to two workers exposed to high levels of elemental mercury vapor, it decreased the mercury excretion half-life from 33.1 days to 11.2 days.

Elemental Mercury

- **Acute inhalation of mercury vapor may require chelation.**
- **Ingestion of elemental mercury in amounts typically found in thermometers does not usually require treatment.**

Patients who have experienced acute elemental mercury inhalation should receive supportive care; give supplemental oxygen as needed and monitor closely for development of acute pneumonitis and pulmonary edema. Chelation may be required.

Elemental mercury is usually nontoxic when ingested; the amount contained in a clinical thermometer typically presents little risk. In some circumstances, increased or enhanced absorption after a relatively small dose may occur in patients with inflammatory bowel disease. Rarely, mercury becomes trapped in the appendix or intestine and requires surgical removal.

To clean up a spill of metallic mercury, an ordinary household vacuum cleaner is of little use and may be harmful since it will vaporize the mercury and increase the airborne mercury concentration. Professional toxic clean-up with a self-contained vacuum system or a mercury clean-up kit should be used. Contaminated carpeting or porous tile should be discarded after clean-up.

Mercury Salts

- **Chelation therapy is recommended for serious systemic intoxication due to mercury salt ingestion.**

When a patient has ingested mercury salts, the goals of therapy are to remove mercury from the body and to prevent dehydration and shock. Inorganic mercury can be removed from the gastrointestinal tract by emesis, catharsis, or lavage. It is imperative that adequate intravenous fluids be administered to prevent dehydration and to reduce the concentration of mercury in the kidneys. BAL or other appropriate chelating agent should be administered immediately; its usefulness depends on rapid administration. With a potentially lethal mercury dose, early peritoneal dialysis or hemodialysis should be considered to enhance mercury removal and to support renal function.

* Use of trade names is for identification only and does not imply endorsement by the Public Health Service or the U.S. Department of Health and Human Services.

Organic Mercury

Damage to the nervous system after MeHg exposure is usually permanent. Because of evidence that BAL increases the MeHg concentration in the brain, BAL (and perhaps other chelating agents) should not be used to treat MeHg toxicity. Since sulfhydryl groups bind tightly to mercury ions, oral polythiol resin (for mercury ingestion) and regional hemodialysis with L-cysteine may be of some benefit, but these therapies are unproven. Exchange transfusion also has been used in an attempt to reduce the body's mercury burden.

❑ **Administration of BAL for MeHg poisoning is contraindicated.**

(7) What treatment would you recommend for the patient in the case study?

(8) What follow-up measures would you recommend for managing this exposure?

Standards and Regulations

The regulations and guidelines pertaining to mercury and mercury compounds in air, water, and food are summarized in Table 3.

Workplace

Air

The workroom air standard mandated by the Occupational Safety and Health Administration (OSHA) is a time-weighted average (TWA) of 6.1 parts per billion (ppb) or 0.05 milligrams per cubic meter of air (mg/m^3) for inorganic mercury vapor, and 1.2 ppb or 0.01 mg/m^3 for organomercury compounds. NIOSH recommends a concentration no greater than 6.1 ppb as a TWA exposure for an 8-hour workday. Subjective psychological complaints, subtle decrements in

some neuropsychological and neurophysiologic parameters, and the appearance of proteinuria have all been reported to occur after exposure to airborne mercury at concentrations as low as 1.2 ppb (0.01 mg/m^3). Correlating airborne mercury levels with health effects is difficult because almost all studies have used area sampling, rather than personal sampling, to determine air concentrations of mercury.

Environment

Air

The EPA National Emission Standards for mercury from various industrial sources include the following: mercury ore processing facilities—2300 g mercury maximum per 24-hour period; mercury cell chlor-alkali plants, sludge incineration plants, other wastewater treatments—3200 g mercury maximum per 24-hour period. Ambient air contains mercury at about 2.4 parts per trillion (ppt); however, concentrations near certain industrial areas, such as mercury mines and refineries, can be nearly 1800 ppt.

Water

The World Health Organization (WHO) guideline for all forms of mercury in drinking water is 1 ppb (1 μg/L). The EPA standard for drinking water is 2 ppb. EPA estimates that, for an adult of average weight, exposure to 21 μg of inorganic or organic mercury per day in food or water will probably not result in any harm to health. The FDA limits mercury in bottled water to 2 ppb.

Food

The FDA regulation for mercury in fish is 1 ppm (1000 ppb). Mercury concentrations in most non-fish foodstuffs are generally less than 0.02 ppb, although levels of up to 0.2 ppb have been detected in meat and poultry. The average concentration of mercury in most fish is less than 0.2 ppb.

Biologic Standards

Mercury is being considered for inclusion in the biological exposure indices (BEI) established by the American Conference of Governmental Industrial Hygienists (ACGIH). BEIs are reference values intended as workplace guidelines for evaluating potential exposure hazards by measuring appropriate determinants in specimens collected from workers at specified times. The proposed BEI for total inorganic mercury in urine, collected preshift, is 35 micrograms per gram (μg/g) creatinine. The proposed BEI for total inorganic mercury in blood is 1.5 μg/dL, collected at the end of the workweek.

Table 3. Standards and regulations for mercury

Agency*	Focus	Level	Comments
ACGIH	Air-workplace Organo (alkyl) mercury compounds	1.2 ppb (0.01 mg/m^3) 3.6 ppb (0.03 mg/m^3)	Advisory; TLV-TWA† Advisory; STEL§
	Mercury vapor	6.1 ppb (0.05 mg/m^3)	Advisory; TWA
	Mercury (aryl and inorganic)	12 ppb (0.10 mg/m^3)	Advisory; TWA
NIOSH	Air-workplace	6.1 ppb (0.05 mg/m^3)	Advisory; TWA
OSHA	Air-workplace Organo (alkyl) mercury compounds Mercury vapor Mercury (aryl and inorganic)	 1.2 ppb (0.01 mg/m^3) 6.1 ppb (0.05 mg/m^3) 6.1 ppb (0.05 mg/m^3)	 Regulation; TWA
EPA	Drinking water	2 ppb (2 µg/L)	Regulation; MCL¶
	Air Mercury ore processing	2300 g Hg/24-hr period maximum	Regulation; National Emission Standard
	Mercury cell, chlor-alkali plants, sludge incineration and wastewater treatment plants	3200 g Hg/24-hr period maximum	
FDA	Food and water Fish Bottled drinking water	 1 ppm 2 ppb (2 µg/L)	 Regulation
WHO	Drinking water	1 ppb (1 µg/L)	Guideline

* ACGIH = American Conference of Governmental Industrial Hygienists; EPA = Environmental Protection Agency; FDA = Food and Drug Administration; NIOSH = National Institute for Occupational Safety and Health; OSHA = Occupational Safety and Health Administration; WHO = World Health Organization

† TLV-TWA (Threshold Limit Value-Time-Weighted Average) = a time-weighted average concentration for a normal workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed.

§ STEL (Short-Term Exposure Limit) = a 15-minute TWA exposure which should not be exceeded at any time during a workday.

¶ MCL (Maximum Contaminant Level) = enforceable level for drinking water.

Suggested Reading List

Reviews

Clarkson TW. Mercury. Ann Rev Public Health 1983;4:375-80.

Clarkson TW. Mercury. J Am Coll Toxicol 1989;8(7):1291-5.

Sunderman FW. Perils of mercury. Ann Clin Lab Sci 1988;18(2):89-101.

Environmental Exposure Sources

Agocs MM, Etzel RA, Parrish RG et al. Mercury exposure from interior latex paint. N Engl J Med 1990;323:1096-1101.

Friberg L, Vostal J, eds. Mercury in the environment: an epidemiological and toxicological appraisal. Cleveland, Ohio: CRC Press, 1987.

Government Documents

Agency for Toxic Substances and Disease Registry. Toxicological profile for mercury. Atlanta: US Department of Health and Human Services, Public Health Service, 1989.

Environmental Protection Agency. Mercury health effects update; health issue assessment. Washington, DC: Government Printing Office, 1984; DHEW publication no. 8-84-019F.

Sources of Information

More information on the adverse effects of mercury and the treatment and management of mercury-exposed persons can be obtained from ATSDR, your state and local health departments, university medical centers, and the National Pesticide Telecommunications Network 24-hour toll-free hotline (1-800-858-7378). *Case Studies in Environmental Medicine: Mercury Toxicity* is one of a series. For other publications in this series, please use the order form on the back cover. For clinical inquiries, contact ATSDR, Division of Health Education, Office of the Director, at (404) 639-6204.

Answers to Pretest and Challenge Questions

Pretest

Pretest can be found on page 1.

(a) The patient's problem list includes painful extremities; erythematous and peeling skin on nose, toes, and fingers; personality changes; tachycardia; sweating; and possible intermittent low-grade fever.

(b) Diagnoses you might consider are the following:

(1) acute rheumatic fever. ARF occurs most commonly between the ages of 5 and 15 years when streptococcal infection is relatively common. Sore joints and fever are characteristic.

(2) leukemia. This is the most common cancer in young children, and symptoms can include sweats and low-grade fever. This diagnosis would not explain the erythematous and peeling skin of the fingers and toes.

(3) Kawasaki disease. The patient does not have some of the common signs of this disease: e.g., he does not have bilateral conjunctivitis; lymphadenopathy; a red rash on his body; red and sore lips, mouth, or throat. However, he is under 5 years of age and does have red and tender hands and feet with peeling skin. He may have had a fever, but it is not well characterized. Kawasaki disease is relatively rare—only 5 to 10 of every 100,000 children acquire the disease.

(4) tuberculosis. The patient's night sweats and possible low-grade fever make this a possibility; however, he has no cough, and tuberculosis is not associated with erythematous and peeling skin on the fingers and toes.

(5) measles. Although immunized against measles, the patient could have experienced primary vaccine failure. However, he does not have Koplik's spots, cough, conjunctivitis, coryza, or a typical rash, making this diagnosis unlikely.

(6) boric acid poisoning. Irritability and erythema and peeling of the skin and mucous membranes can occur with boric acid poisoning. However, the patient does not exhibit renal toxicity or other common symptoms of boric acid toxicity such as nausea, vomiting, and diarrhea.

(7) acrodynia. The patient exhibits many of the symptoms common to this disease. See the problem list in (a) above. This disease of infancy and early childhood is caused in most, if not all, instances by exposure to mercury.

In addition, Stevens-Johnson syndrome, fifth disease, scarlet fever, rubella, systemic lupus erythematosus, and drug rashes (due to an unsuspected ingestion) should be considered.

(c) The best test to confirm or rule out chronic mercury exposure is a 24-hour urinary mercury concentration and creatinine clearance. The urine should be analyzed by cold vapor atomic absorption spectrophotometry; the Reinsch test—a heavy metal screening test—is not sufficiently specific or sensitive. In addition, the following tests would be useful to help exclude other diagnoses in the differential: complete blood count with differential; erythrocyte sedimentation rate or C-reactive protein; chest and hip X rays; serum creatinine and blood urea nitrogen; urinalysis; tuberculin skin test with controls; streptoccocal antibody titers (ASO); and throat culture for streptococcus.

Answers to Pretest and Challenge Questions

Pretest *continued*

(d) If test results indicate the patient has a high urinary mercury concentration, chelation therapy should be considered, and a physician experienced in chelation therapy should be consulted. It is also important to ensure that the patient is no longer exposed to the mercury source.

All family members should have their urinary mercury concentration measured. A common exposure is quite likely, particularly if the source is mercury vapor in the home. If the source is a product used in the home, other persons using the product may be at risk. The county or state health department should be contacted to identify and eliminate the mercury source and to evaluate the potential exposure to members of the community. Medical follow-up for mercury-exposed persons includes monitoring nervous system and renal function status.

Challenge

Challenge questions begin on page 4.

(1) In a patient so young, sources of chronic mercury exposure are most likely to be linked to the home. Within the home, the possible mercury sources include off-gassing of paint on interior walls and liquid mercury from a spill embedded in floors or carpets. Possible ingestion sources include contaminated drinking water, mercury-containing medicinals, or folk remedies.

(2) If the mercury source is in the home or diet, all members of the family could be exposed. Other persons in the community who ingest contaminated food or drink might also be affected. In addition, if paint is the source of exposure, consumers using the same paint brand may be exposed.

(3) Yes, if the source is elemental mercury vapor released from paint in the home, the mother, and subsequently, the fetus, are likely to be exposed. Although the adverse developmental effects of MeHg are known, the long-term neurologic consequences to the human fetus of chronic low-level exposure to mercury vapor have not been documented well.

(4) See pretest answer (a).

(5) See pretest answer (b).

(6) See pretest answer (c).

(7) See pretest answer (d).

(8) See pretest answer (d), paragraph 2.

Posttest and Credits

Continuing education credit is available to health professionals who use this monograph and complete the posttest. The criterion for awarding continuing medical education (CME) credits and continuing education units (CEU) is a posttest score of 70% or better.

The Centers for Disease Control and Prevention (CDC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians, and by the International Association for Continuing Education and Training (IACET) to sponsor continuing education units for other health professionals.

The Agency for Toxic Substances and Disease Registry, in joint sponsorship with CDC, is offering 1 hour of CME credit in Category 1 of the Physician's Recognition Award of the American Medical Association and 0.1 hour of CEU for other health professionals upon completion of this monograph.

In addition, the series *Case Studies in Environmental Medicine* has been reviewed and is acceptable for credit by the following organizations:

The **American Academy of Family Physicians (AAFP)**. This program has been reviewed and is acceptable for 1 prescribed hour by the American Academy of Family Physicians. (Term of Approval: beginning January 1992.) For specific information, please consult the AAFP Office of Continuing Medical Education.

The **American College of Emergency Physicians (ACEP)**. Approved by the American College of Emergency Physicians for one hour per issue of ACEP Category I credit.

The **American Osteopathic Association (AOA)**. AOA has approved this issue for 1 credit hour of Category 2-B credit.

The **American Association of Occupational Health Nurses (AAOHN)**. AAOHN has approved this program for 1.2 contact hours. Applicant will receive the assigned code number in the award letter.

The **American Board of Industrial Hygiene (ABIH)**. ABIH has approved this program for 0.5 certification maintenance (CM) point per 3 Case Studies. The CM approval number is 2817.

To receive continuing education credit (CME or CEUs), complete the Posttest on page 24 in the manner shown in the sample question below. **Circle all correct answers.**

Which of the following is known to precipitate migraine headaches?

(a) fatigue
(b) alcohol
c. grapefruit
(d) sunlight
e. sleep

After you have finished the Posttest, please transfer your answers to the answer sheet on the inside back cover and complete the evaluation on the lower half of that page. Fold, staple, and mail the back cover to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333. Your confidential test score will be returned with an indication of where the correct answers can be found in the text. Validation of earned CME credit and CEU will also be forwarded to participants, and their names, if requested, will be placed on the mailing list to receive other issues in the *Case Studies in Environmental Medicine* series.

POSTTEST: MERCURY

Circle all correct answers and transfer your answers to page 25.

1. Which of the following statements about mercury sources is true?
   a. Fish consumption is the main source of MeHg exposure in the general population.
   b. Ingesting small amounts of elemental mercury is lethal.
   c. Burning fossil fuels contributes to the level of mercury in the atmosphere.
   d. A mercury spill can be safely cleaned up using a household vacuum cleaner.
   e. By law, medicines or over-the-counter preparations can no longer contain mercury in any form.

2. Which of the following are or were potential sources of low levels of mercury vapor?
   a. new carpeting
   b. latex paint
   c. fish
   d. dental amalgams
   e. BAL

3. Which of the following statements are true of the mercury in a mercury salt such as mercuric chloride?
   a. It is excreted mainly in the bile and feces.
   b. It has a whole-body clearance of 3 to 5 days.
   c. It will cause only CNS injury.
   d. It does not readily cross the blood-brain barrier.
   e. It is readily reduced to elemental mercury in the body.

4. Of the following, who is likely to be at increased risk of mercury exposure?
   a. welders
   b. fungicide applicators
   c. children
   d. laboratory technicians
   e. dentists

5. Which of the following statements are true about laboratory tests and mercury exposure?
   a. The best test to confirm chronic inhalation exposure is a urinary β_2-microglobulin level.
   b. A chest X ray will reveal deposits of mercury in the lungs up to 5 hours after inhalation exposure.
   c. A blood mercury concentration of 20 µg/dL indicates mercury poisoning.
   d. Hair analysis is useful in clinically evaluating all mercury-poisoned patients.
   e. A urinary mercury concentration of less than 20 µg/L in adults is considered a background level.

6. The standard of care for mercury-poisoned patients may include
   a. chelation therapy to reverse neurologic damage caused by MeHg ingestion
   b. chelation therapy for a patient who has swallowed a bead of liquid mercury
   c. an abdominal X ray for a patient who has recently consumed grain treated with MeHg
   d. renal supportive therapy for a patient who has ingested mercuric chloride
   e. chelation therapy for a patient who has ingested a significant quantity of mercuric chloride

7. Clinical evidence of mercury poisoning due to ingestion of fish (MeHg form) may include
   a. tunnel vision
   b. peeling toes and fingers
   c. hearing loss
   d. tremor
   e. ataxia

8. Which organs or systems will require monitoring in a patient with a urinary mercury level of 575 µg/L?
   a. nervous system
   b. spleen
   c. kidneys
   d. bone marrow
   e. thyroid

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CASE STUDIES IN ENVIRONMENTAL MEDICINE: MERCURY TOXICITY

If you wish CME credits or CEU, please indicate your answers to the Posttest questions on page 24 by circling the letters below for the correct answers. Complete the evaluation questionnaire and fill in the information requested on the reverse side. Tear off this last page, fold, staple, and mail to Continuing Education Coordinator, Agency for Toxic Substances and Disease Registry, Division of Health Education, E33, 1600 Clifton Road, Atlanta, GA 30333.

1. a b c d e
2. a b c d e
3. a b c d e
4. a b c d e
5. a b c d e
6. a b c d e
7. a b c d e
8. a b c d e

Evaluation Questionnaire

Please complete the following evaluation by circling the appropriate number.

	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
1. As a result of completing this monograph, I will be able to:					
Explain why mercury may be an acute and chronic health hazard.	1	2	3	4	5
Describe the known factors contributing to mercury toxicity.	1	2	3	4	5
Identify potential environmental or occupational sources of exposure to mercury.	1	2	3	4	5
Identify evaluation and treatment protocols for persons exposed to mercury.	1	2	3	4	5
List sources of information on mercury.	1	2	3	4	5
2. The monograph addressed the objectives printed on the inside front cover.	1	2	3	4	5
3. I am more likely to ask patients questions regarding possible environmental exposures as a result of reading this issue.	1	2	3	4	5
4. Independent study was an effective teaching method for the content.	1	2	3	4	5
5. How much time (in minutes) was required to read this monograph and complete the posttest?	40	60	80	100	120

Comments:_____

To obtain credit, provide the information below.

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Specialty _____

To be placed on mailing list, check here. ☐

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Please send me the following *Case Studies in Environmental Medicine.*

- ❑ Arsenic
- ❑ Asbestos
- ❑ Benzene
- ❑ Beryllium
- ❑ Cadmium
- ❑ Carbon Tetrachloride
- ❑ Chlordane
- ❑ Cholinesterase Inhibitors
- ❑ Chromium
- ❑ Cyanide
- ❑ Dioxins
- ❑ Ethylene/Propylene Glycols
- ❑ Exposure History
- ❑ Gasoline
- ❑ Jet Fuel
- ❑ Lead
- ❑ Mercury
- ❑ Methanol
- ❑ Methylene Chloride
- ❑ Nitrates/Nitrites
- ❑ Pentachlorophenol
- ❑ Polyaromatic Hydrocarbons (PAHs)
- ❑ Polychlorinated Biphenyls (PCBs)
- ❑ Radiation
- ❑ Radon
- ❑ Reproductive and Developmental Hazards
- ❑ Skin Lesions
- ❑ Stoddard Solvent
- ❑ Tetrachloroethylene
- ❑ 1,1,1-Trichloroethane
- ❑ Trichloroethylene
- ❑ Toluene
- ❑ Vinyl Chloride

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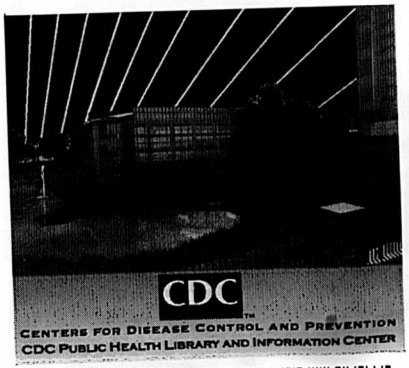
staple or tape



The state of knowledge regarding the treatment of patients potentially exposed to hazardous substances in the environment is constantly evolving and is often uncertain. In this monograph, the Agency for Toxic Substances and Disease Registry (ATSDR) has made a diligent effort to ensure the accuracy and currency of the information presented but makes no claim that the document comprehensively addresses all possible situations related to this substance. This monograph is intended as an additional resource for physicians and other health professionals in assessing the condition and managing the treatment of patients potentially exposed to hazardous substances. It is not, however, a substitute for the professional judgment of a health care provider and must be interpreted in light of specific information regarding the patient available to such a professional and in conjunction with other sources of authority.

DEPARTMENT OF
HEALTH & HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Agency for Toxic Substances and Disease Registry
Atlanta, GA 30333
