KALAMAZOO CENTER FOR MEDICAL STUDIES

Mercury Exposure Aboard an Ore Boat

Richard R. Roach and Stephanie Busch

Department of Internal Medicine, Kalamazoo Center for Medical Studies, Kalamazoo, Michigan, USA

Two maritime academy interns (X and Y) were exposed to mercury vapor after spilling a bottle of mercury on the floor in an enclosed storeroom while doing inventory aboard an ore boat. During a 3-day period, intern Y suffered transient clinical intoxication that resolved after he was removed from the environment and he showered and discarded all clothing. His initial serum mercury level dropped from 4 ng/mL to < 0.05 ng/mL. Intern X had an initial level of 11 ng/mL, which continued to rise to a maximum of 188.8 ng/mL. He complained of tremulousness, insomnia, and mild agitation and was hospitalized. He had showered and discarded all clothing except his footwear earlier than intern Y. Intern X's continued exposure due to mercury in the contaminated boots during the 2 weeks before hospitalization was presumed to be the cause. Removing his footwear led to resolution of his toxic symptoms and correlated with subsequent lowered serum mercury levels. Chelation was initiated as recommended, despite its uncertain benefit for neurologic intoxication. Mercury is used in the merchant marine industry in ballast monitors called king gauges. New engineering is recommended for ballast monitoring to eliminate this hazard. *Key words:* ballast gauge, chelation therapy, dimercaptosuccinic acid, mercury intoxication, neurotoxicity. *Environ Health Perspect* 112:910–913 (2004). doi:10.1289/ehp.6798 available via http://dx.doi.org/ [Online 10 March 2004]

Case Report

In June 2000 a mercury spill occurred aboard an iron ore carrier in an unventilated steel storage compartment measuring approximately 10 ft × 10 ft × 30 ft. The compartment was located above the boiler, so the floor temperature varied from 105°F when in dock to as much as 130°F when the ship was underway. The ceiling of the storeroom was the upper deck, and the walls were steel with a tight-fitting steel door at one end (Figure 1). Within this enclosed compartment, two maritime academy interns (referred to in this report as interns X and Y) were working on supply inventory. Intern X started his inventory in the storeroom farthest from the door. He opened a drawer to find approximately 40 lb of mercury stored in a polyethylene bottle with a screw-top lid. Unaware of its weight, he dropped the bottle onto the floor, spilling a portion of the metal across the floor as well as splashing it onto his coveralls and into his boots. He was aware that his coveralls were contaminated, but he claimed he did not realize that the inside of his boots were also contaminated. Intern Y was working at the opposite end of the storeroom, nearest the door. The incident was not reported immediately; both interns continued to work for 3 days in the storeroom

after the spill occurred, completing their inventory. Both showered the day of the spill but continued to work in contaminated coveralls.

After their inventory was completed, the interns notified the chief engineer of the spill. The chief engineer confiscated and discarded the contaminated coveralls, immediately cleaned the exposed area with a sulfur amalgam compound according to Occupational Safety and Health Administration (OSHA) protocol (Lawrence Livermore National Laboratory 2001), and secured the storeroom, allowing no further access until the ship docked and the storeroom was decontaminated. Medical and personnel authorities were notified when the chief engineer was informed of the spill. Serum mercury levels of the interns were ordered as they left the ship. Intern Y was assigned to another ship and did not have his serum drawn until the second ship docked. Intern X felt he had the "flu" and decided to take his vacation, so he left for home. He continued wearing his boots and possibly contaminated socks, but his other clothing was confiscated and discarded by the chief engineer.

Intern Y reported feelings of lethargy, headaches, lightheadedness, and excessive thirst that lasted for several days aboard the

second ship. These symptoms resolved by the time the second ship arrived at port and he had his serum tested. Two mercury levels were evaluated. His first serum level, obtained 15 days after the initial exposure, was 4 ng/mL; the range in the general population is 0–9 ng/mL, and a serum level of 50 ng/mL is considered toxic (Mayo Clinic 2002). He reported complete resolution of his symptoms at the time his serum was drawn 19 days after initial exposure. At that time, his mercury level was < 0.05 ng/mL; his serum creatinine of 1.4 mg/dL was not considered sufficiently abnormal with respect to his mercury levels to warrant reevaluation. Intern Y was examined by R.R.R. 17 days after the initial exposure and was found to have no mental status changes, ataxia, tremor, or protein in his urine.

Intern X, who had spilled the mercury, reported symptoms of fatigue, drowsiness, and trouble sleeping when his mercury level was 11 ng/mL. Eleven days after the initial exposure his level had risen to 56 ng/mL. A third test 15 days after the initial exposure showed an increase to 89 ng/mL. At this point, Intern X's mother reported by telephone conversation that he was "nervous and tremulous." She was advised to seek medical attention for her son immediately. He was seen by his primary physician and admitted to the hospital 16 days after the initial exposure; his mercury level was 188.8 ng/mL. Upon admission to the hospital, the nurse took off his footwear, including the boots, which she reported was visibly contaminated with mercury. Intern X

had been wearing the boots daily since the initial exposure. It is not known from the record whether he had consistently worn socks or whether he changed socks. The toxicologist, consulted by his primary physician, reported to one of us (R.R.R.) that the boots were not analyzed but were "contaminated with visible mercury."

Seventeen days after the initial exposure, while Intern X was in the hospital, a 24-hr urine collection revealed a mercury level of 356 μg/L and creatinine of 1.2 mg/dL. Liver function studies were normal. Chelation therapy was initiated with succimer, 700 mg orally every 8 hr for 5 days, and then every 12 hr for another 14 days. His blood pressure was normal, and his initial creatinine level was 1.1 mg/dL (17 days after the initial exposure) rising to 1.3 mg/dL 11 days after hospitalization before normalizing at 0.9 mg/dL 21 days after hospitalization. Intern X's serum mercury levels drawn 21 days and 38 days after the initial exposure were 182 ng/mL and 17 ng/mL, respectively. By 2 months after the initial exposure, his levels were < 0.5 ng/mL (Figure 2).

Although Intern X described tremulousness, no tremor or ataxia was noted by the attending physician, even though he specifically looked for them. The clinician found no alteration of mental status, and neuropsychologic testing was not performed. A neurologist was not consulted because the symptoms were improving and the toxicologist with the primary internist had begun chelation therapy. One of us (R.R.R.) assessed the patient before he returned to his maritime duties and found no cognitive impairment, tremor, abnormalities of reflexes, or ataxia.

Environmental Testing

Mercury vapor readings were assessed by a certified industrial hygienist, as described by Norman (2000), with a Jerome model 431-X mercury vapor analyzer (Certified Industrial Hygienist Services, Duluth, MN) that can detect mercury concentrations as low as 0.001 mg/m^3. The American Conference of Governmental Industrial Hygienists defines a concentration of 0.025 mg/m^3 as the threshold limit value (OSHA 2004) for an 8-hr time-weighted average limit for toxic exposure. OSHA (2004) uses 0.10 mg/m^3 as the permissible exposure limit. Mercury level readings were obtained throughout the storage compartment (Figure 1). Inside the locker of the storage compartment where the mercury was kept, the mercury levels were 0.999 mg/m^3 (maximum meter reading). Measurements taken in the interns' quarters were 0.012 and 0.025 mg/m^3. The lower reading was in intern Y's bunk, and the higher reading was recorded in intern X's bunk. A reading of 0.025 mg/m^3 was recorded in the shower area.

Because the engine control room and the captain's bridge were the locations of the king gauges, measurements were also made in those two areas. In the engine control room, a level of 0.015 mg/m^3 was detected under the king gauge and at the engine control panel. Just above the reservoir, a mercury level of 0.024 mg/m^3 was detected. On the captain's bridge near the captain's chair, the reading was undetectable. However, above the carpet under the gauge where there was visible liquid mercury, a level of 0.006 mg/m^3 was found, whereas above the capped reservoir the mercury level was 0.028 mg/m^3. Significantly, these areas were air conditioned to below 70°F. Measurements for mercury were also taken in the food processing areas, dining room elevator, engineer's quarters, random cabins of other officers and sailors, and the hallways leading to the interns quarters. All these readings were at undetectable levels.

After the decontamination procedure, readings of 0.000–0.003 mg/m^3 in the storage compartment documented adequate removal of the mercury vapor source. The drawer where the mercury container was stored registered 0.007 mg/m^3. Levels in the interns' quarters were undetectable, and levels under the king gauges after cleanup were 0.004 mg/m^3 in the engine control room and 0.013 mg/m^3 on the captain's bridge.

Discussion

Mercury has been used for industrial and medicinal purposes for several millennia (Sunderman 1988). Although most medicinal use of mercury has been discontinued, cultural use (Wendroff 1995) and industrial use of mercury are ongoing (Agocs et al. 1992; Ozuah 2000). In 2001, the American Association of Poison Control Centers reported a total of 3,550 cases of mercury exposure in the United States (Litovitz et al. 2001). Occupational exposure, now the principal source of mercury intoxication, typically results from improper handling of mercury, accidental spills, and poor ventilation in the work environment (Ozuah 2000). Elemental mercury vapor accounts for most of those reported cases (Agocs et al. 1992).

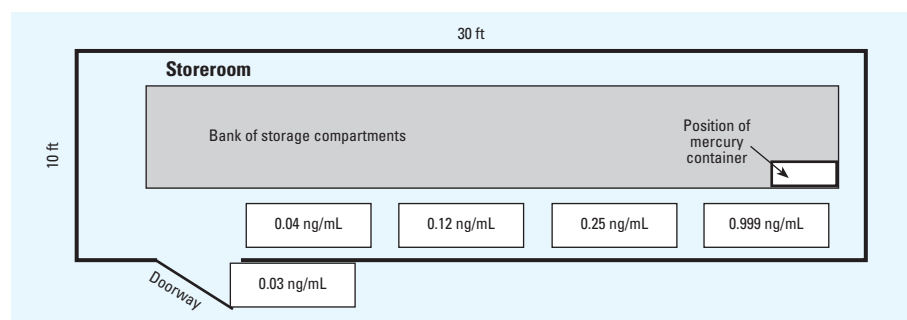U.S. Steel Great Lakes Fleet shipping companies use king gauges extensively for

Figure 1. Diagram of the storeroom where the spill occurred. The levels of mercury vapor are shown where they were recorded in the storeroom.
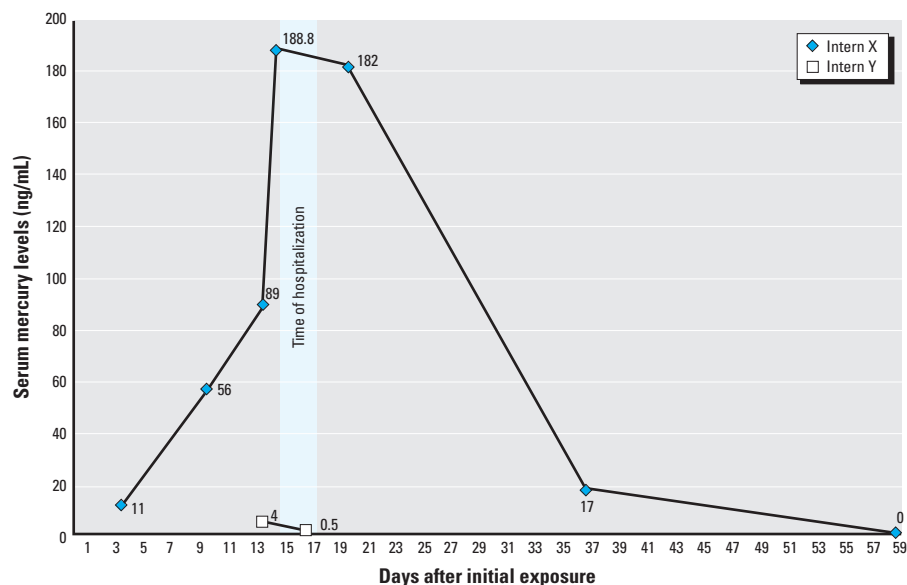

Figure 2. Diagram of the serum levels of the two interns.

determining the ballast necessary to keep the ship balanced during loading of iron ore, grain, and limestone. The king gauge is a labeled row of parallel upright mercury-filled half-inch-diameter glass columns. Each column reflects the ballast in one of compartments of the ship's hold. Although each mercury-filled column is capped, small amounts of mercury can spill out of the column or vaporize. The ship carries a supply of mercury in order to refill the columns when they reach a minimum threshold. Frequently, small amounts of mercury are found on the floor under the gauge. The two king gauges on the ship are on the bridge and in the engine control room.

Elemental mercury is poorly absorbed through the skin at a rate of 0.025 mg/m^3 (OSHA 2004). Only 0.01% of mercury is absorbed through the gastric mucosa after ingestion (Agocs et al. 1992; Ozuah 2000). However, when elemental mercury is vaporized, it is 80% absorbed (Agocs et al. 1992) through alveolar membranes and distributes into red blood cells and tissue (Centers for Disease Control and Prevention 1991). About 7–14% of inhaled mercury is exhaled within a week after exposure (U.S. EPA 1997). Once absorbed, mercury diffuses throughout the body, concentrating in the liver, kidneys, and brain. Diffusion of the metal across the blood–brain barrier can result in irreversible damage to the central nervous system (Bradberry and Vale 2001), including the cerebellum (Florentine and Sanfilippo 1991).

The major route of excretion (90%) of mercury is through bile into the feces. Urine accounts for another 10% of the elimination process (Agocs et al. 1992). Renal storage significantly lowers serum levels acutely once exposure has ceased (Maiorino et al. 1996), but the elimination of mercury through biliary and urinary excretion is a slow process, with a half-life in excess of 40 days (Ozuah 2000). Therefore, elimination of the source is the primary approach to therapy. Chelation therapy, used since the 1950s, is based on the concept of chemically binding the metal and promoting its urinary excretion (Bruno 1999; Frumkin et al. 2001). Dimercaptosuccinic acid (DMSA) is considered the safest, most effective treatment for mercury poisoning (Aaseth et al. 1995), but it apparently does not affect mercury retained in the brain (Kostial et al. 1997). Polythiol, a nonabsorbable resin, can aid in removal by binding mercury in excreted bile (Cikrt and Lenger 1980). Although in common use, these therapies have yet to be approved by the Food and Drug Administration.

The value of chelation therapy after inorganic or elemental mercury exposure remains controversial. Several studies have documented that DMSA treatment results in increased urinary excretion of mercury (Roels et al. 1991; Torres-Alanis et al. 1995). In a randomized, double-blind, parallel group design study with oral administration of DMSA or a placebo for 14 days, Englund et al. (1994) demonstrated an increase in mercury excretion, but there was no evidence that chelation therapy alleviated neurologic symptoms in the study. Studies such as these question whether excretion equals efficacy in resolving toxicity (Kosnet 1992; Kostial et al. 1997). According to other researchers, there is no evidence that chelation removes mercury from the brain (Buchet and Lauwerys 1989; Louwerse et al. 1995; Nierenberg et al. 1998). Several studies report that chelation increases urinary excretion of mercury (Buchet and Lauwerys 1989; Goyer et al.1995; Louwerse et al. 1995; Roels et al. 1991; Torres-Alanis et al. 1995). A controlled animal study of rats exposed to mercury vapor with subsequent administration of chelating agents demonstrated that chelators mobilize mercury only from the kidneys and not from the brain (Buchet and Lauwerys 1989). Chelation did not effect brain levels, even with increasing doses of chelator.

Mercury poisoning can cause a nonfocal permanent encephalopathy (Bradberry and Vale 2001). Acute exposure to elemental mercury vapor is characterized by euphoria, irritability, anxiety, and emotional lability, whereas chronic exposure results in mental confusion, altered level of consciousness, and permanent tremor (LaDou 1997; Letz et al. 2000) or cerebellar ataxia (Cherry et al. 2002).

Acute exposure to mercury affects the pulmonary, renal, and nervous systems. Initial evaluation of the patient with acute inhalation of mercury vapor, according to LaDou (1997), should address the potential for cyanosis, tachypnea, and pulmonary edema; therefore, arterial blood gas measurement and a chest X ray are important if respiratory symptoms are present. Gastrointestinal symptoms of nausea, vomiting, and diarrhea are not usually life threatening. Renal injury presents with a diuretic phase and then proteinura and can result in renal failure. Neuropsychiatric manifestations include symptoms of agitation, anxiety, and tremulousness followed by true resting tremor and eventual truncal ataxia. Lack of clinical tremor does not exclude subclinical neurologic effects if evaluated with a computer-based test method described by Netterstrom et al. (1996). Peripheral neuropathy symptoms of paresthesias can also be presenting symptoms. Decision to treat should be based on neurologic and renal toxicity; therefore, a neurologic examination and assessment of serum and urine mercury, renal function, and presence of proteinuria should be part of the exposed patient's evaluation. Formal psychologic testing may be necessary depending on the patient and family observations or persistence of personality change or psychiatric complaints (LaDou 1997).

Remarkably, our patients reported no pulmonary complaints, even upon suggestive questioning. They both manifested transient gastrointestinal symptoms. Intern Y complained of lethargy and headache but no neurologic abnormalities were found on examination; also, his renal function remained normal with no proteinuria. Intern X was reported by his mother to have a sense of tremulousness, but his primary physician did not observe any objective tremor or ataxia. Transient slight elevation in serum creatinine and subsequent normalization suggested that he had mild renal toxicity, but no proteinuria was noted despite the documented renal excretion of mercury. His 24-hr excretion of 356 µg/L with a creatinine of 1.2 µg/g is below the 500 µg/g creatinine associated with significant renal or neurologic toxicity (LaDou 1997).

After the cleanup, several recommendations were made to the merchant marine (Norman 2000): *a*) replacement of all king gauges with non-mercury-containing instruments; *b*) secondary containment of mercury instrumentation devices; *c*) storage of elemental mercury in sealed containers within an air-conditioned locker that, upon impact, cannot allow leakage; *d*) training technicians in protocols for filling king gauge reservoirs; and *e*) availability of rubber gloves, chemical splash goggles, and respirators with mercury vapor cartridges for cleanup on board ship.

Training procedures for cleanup of spills and refilling of gauges were initiated, containment devices were installed, and the mercury was thereafter stored in sealed containers in a cooled environment. However, at present, merchant marine ballast monitors still use mercury, and no acceptable engineering alternatives have as yet been designed or installed.

Conclusion

The clinical courses of these two interns illustrate several important pathophysiologic principles. Intern Y certainly had a toxic inhalation exposure, but his symptoms quickly resolved when the exposure ended. Intern X clearly had more inhalation exposure because he was working in the less-ventilated part of the storage room. The door was on the other side, and there was no other ventilation (Figure 2), but his increasing mercury levels after the initial exposure suggested continued exposure (Figure 1).

Symptoms, interpreted as "nervousness" by intern X's mother, resolved within 4 days of removing his boots at the time of hospitalization. By the time he presented to one of us (R.R.R.) for return to work, neurologic evaluation showed no signs of ataxia or dementia.

Computer-based tremor testing was not performed.

Although mercury toxicity may result from consumption of fish and from amalgam fillings, the marked decrease in his serum levels correlated with the removal of his footwear. We are not aware of previously reported cases of cutaneous elemental absorption leading to toxicity. Increased inhalation is possible from the mercury in the intern's boots, because contamination of skin and clothing has been shown to increase the mercury in the microenvironment (Henderson 1973). However, we speculate that cutaneous absorption of vapor may have been a contributing factor leading to continued elevation of his serum mercury levels. On the basis of the studies reviewed, we conclude that the neurologic symptoms of both interns resolved with the removal from mercury exposure, storage in the renal compartment, and physiologic mercury excretion, although chelation was initiated in the more severely affected intern.

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