Clinical Response to Therapeutic Agents in Poisoning from Mercury Vapor

F. WILLIAM SUNDERMAN, Sr., M.D., Ph.D.

Institute for Clinical Science,
Hahnemann Medical College and Hospital,
Philadelphia, PA 19102

ABSTRACT

Exposure to mercury vapors for an hour per working day over a period of 13 years produced in a thermometer manufacturer severe signs and symptoms of mercury poisoning. Complete disability developed insidiously over the last six months of employment.

During the first two months of observation, the patient was treated in succession with three chelating agents: 2,3-dimercapto-1-propanol (BAL)*, d-penicillamine† and sodium diethyldithiocarbamate (Dithiocarb).‡ Each agent was administered initially for a period of approximately two weeks. A second course of therapy with BAL was administered for three days.

Of the three complexing agents used, BAL gave the most dramatically favorable clinical response and yielded the highest urinary excretions of mercury. Dithiocarb was partially effective; d-penicillamine proved to be essentially ineffective.

Analyses of the patient's sweat indicated that appreciable amounts of mercury were excreted by this route. Following the alleviation of the severe symptoms by BAL, the patient was placed on a regimen of daily sweats and physio-therapy for a protracted period of several months. On this latter regimen, the mercury levels in the urine, blood serum and sweat were decreased to within the normal ranges of values. The patient made a complete and uneventful recovery.

In patients encountering psychotic and neurological disorders of undetermined etiology, consideration should be given to unsuspected or masked chronic exposure to mercury vapor as a possible cause.

Introduction

Although the toxic effects of mercury and its compounds have been known since antiquity; nevertheless, exposure to mercury continues to be an increasing danger. In 700 B.C., the Phoenicians began mining operations for cinnabar,
the sulfide ore of mercury, in Almaden, Spain.\textsuperscript{42} It is almost certain that in the extraction of mercury from its ore, some of the dangerous consequences of exposure would have become evident. Aristotle, in the fourth century, was the first to leave a written record about mercury which he called "liquid silver."\textsuperscript{42} Five centuries later, Dioscorides, a Greek physician, wrote that cinnabar had medical uses, such as healing burns, but that the ore itself was poisonous if swallowed.\textsuperscript{42} Pliny (c. 77 A.D.) was concerned with the respiratory hazards encountered by mercury workers. He advised workers to protect their faces with masks made from thin bladder skins in order to avoid the inhalation of mercurial dust.\textsuperscript{37} According to Goldwater,\textsuperscript{13} Ulrich Ellenbog in Germany wrote the first treatise on industrial metal poisonings in about 1473. Ellenbog recognized that the inhalation of metallic vapor was more dangerous than exposure to the metal itself.

During the past score of years, extensive research studies have been undertaken on the hazards of exposure to mercury and its compounds, and during the past eight years, more than 15 reviews have been published pertaining to the toxicity of mercury in relation to environmental pollution.\textsuperscript{2, 4, 8, 11, 13, 14, 29, 30, 32, 33, 38, 39, 40, 42, 45, 51} In addition, many reports have also appeared evaluating the therapeutic measures recommended for poisoning from the mercurials.\textsuperscript{7, 10, 21, 23, 35, 41}

Despite the voluminous publications on mercury poisoning, the belief is prevalent, even among scientific workers, that mercury is relatively harmless. Whereas lead and arsenic are well-established as dangerous elements, mercury is persistently overlooked as a hazard. In a recent study of laboratory technicians using mercury-filled volumetric apparatuses for gas analysis,\textsuperscript{*} Harrington\textsuperscript{16} found concentrations of mercury in urine as high as 2.4 mg per liter. Harrington expressed concern that the laboratory workers were ignorant of the potential dangers from chronic exposure to mercury vapor. The increasing use of mercury in scientific instruments, electric switches and lighting, fungicides, paints and catalysts in chemical operations will obviously increase the likelihood of mercury intoxication.

The inhalation of inorganic and organic mercury vapors and their absorption through the lungs present special toxicological problems. These problems pertaining to mercury absorption from the respiratory tract have been delineated by Kudsk\textsuperscript{24, 25, 26, 27, 28} and others. After exposure to mercury vapor, mercury accumulates in brain tissues in quantities that are far greater than the quantities found when equivalent amounts are administered either orally or parenterally.\textsuperscript{5, 6, 34} Thus, intoxication from the inhalation of mercury vapor may produce symptoms that are not characteristically observed in other forms of mercury poisoning.

The time-honored treatment for persons chronically ill from exposure to mercury vapor in mining and industrial operations in Europe has been, in the main, directed to diaphoresis and physiotherapy. Health regulations in Spain that are two centuries old, limit cinnabar miners to work for only eight days per month. As a consequence, these men hold second jobs in the various trades and their exposure to mercury is intermittent and limited. Nonetheless, many become chronically ill from mercury vapor. Putnam\textsuperscript{42} describes the treatment given in a hospital in Almaden, Spain. The hospital contains a special room lined with heat lamps and has a floor marked with a circular path: "Sometimes a man inhales too much mercury vapor in the mine and develops a tremor. If it's a severe case, the doctors send him here (i.e., the room) for treatment. He strips and walks round and round (the circular path) in the heat,
sweating out the mercury. Most respond rapidly and are returned to work. A few don't, they are pensioned.”

In the early part of this century, the treatment of mercury poisoning was directed, for the most part, to persons attempting suicide by the ingestion of lethal amounts of mercuric chloride.” For this purpose, Lambert and Patterson advocated the use of diuretics, both orally and by rectum, supplemented by diaphoresis. These measures were combined with the administration of albumin in the form of milk and frequent flushings of the stomach and colon. Sansum expressed the view that Lambert and Patterson's treatment owed its value chiefly to the colonic irrigations.

As an adjunct to the diuretic and diaphoretic treatment of Lambert and Patterson, Hayman and Priestley decapsulated the kidneys and administered saline fluids in a patient suffering from severe bichloride of mercury poisoning. Sunderman, Austin and Camac studied the electrolyte components of this patient's serum and reported a marked decrease in the concentrations of chloride and total base owing to the suppression of the tubular absorption of sodium, chloride and water. The decrease in serum chloride was partially compensated by increases in base bound by phosphate, sulfate and organic acid. In the light of these findings, the patient of Hayman and Priestly was given appropriate saline fluid replacement and made an uneventful recovery.

Chelating Agents

The use of chelating agents in the treatment of heavy metal poisoning has been under investigation since the development of dimercaprol (BAL) during World War II as an antidote for arsenic poisoning. In general, BAL and d-penicillamine have been recommended as antidotes of choice for mercury poisoning. However, as Gabard has recently pointed out, the experimental and clinical data upon which these recommendations were based have been meager and contradictory. It should also be noted that most of the reports have placed emphasis on the response of chelating agents in the treatment of intoxication from organic mercury, particularly methyl mercury, and that comparatively few studies pertain to the treatment of poisoning from inorganic mercury compounds.

For the treatment of uremia in acute mercurial intoxication, mention should be made of the beneficial response obtained with hemodialysis. Sanchez-Sicilia pointed out that the supportive therapy of hemodialysis is especially useful in patients who respond unfavorably to BAL and other forms of chemotherapy.

The efficacy of dimercaprol (BAL) as an antidote in poisoning by mercuric chloride was studied in experimental animals by Gilman et al, Stocken and Adam. Clinical studies have also been reported by Longcope and Luetcher, Matthes et al and by Haddad and Stenberg, Eastmond and Holt and others. Longcope and Luetcher used BAL for the treatment of 23 persons attempting suicide by the ingestion of bichloride of mercury. All but one of the treated cases recovered. Matthes and coworkers reported four cases of acute severe pneumonitis from the inhalation of mercuric vapor. One of the patients was dead on arrival at the hospital, another died within 30 hours after admission. Both of the other patients received BAL and one of them survived. Haddad and Stenberg reported two patients with acute bronchitis following the inhalation of mercury vapor. Both patients received BAL and recovered.

The therapeutic use of the penicillamines has also received consideration. In 1958, Aposhian proposed the use of d-penicillamine as an oral protective agent against lethal doses of mercuric
chloride. Chisolm\textsuperscript{7} reported favorable response by this mercury-binding agent. He concluded that in severe cases of mercury poisoning, the administration of penicillamine is attended by an impressive clinical response following by a long period of slow but steady improvement. Penicillamine was used by Pagnotto et al\textsuperscript{41} in the treatment of a chemist suffering from chronic mercury poisoning. These investigators found that the urinary excretion of mercury was increased during treatment with penicillamine although the amount of mercury excreted was not as great as with other complexing agents.

Gabard\textsuperscript{10} has recently studied the influence of 15 selected chelating agents on the excretion and distribution of inorganic mercury in rats. He concluded that among the chelating agents tested that DMPS (sodium 2,3-dimercaptopropane-1-sulfonate) exhibited the highest efficacy in mobilizing mercury from the body tissues. Moreover, DMPS having an LD\textsubscript{50} of 6.4 mmol per kg of body weight is far less toxic than BAL with an LD\textsubscript{50} of 0.85 mmol. Gabard noted that the cumulative excretion of mercury in urine and feces when penicillamine was used as an antidote was less than either DMPS or BAL.

The chelating agents studied by Gabard\textsuperscript{10} did not include Dithiocarb (sodium diethyldithiocarbamate). It has been shown by West and Sunderman\textsuperscript{52,53} that the administration of Dithiocarb is relatively free of protracted undesirable side effects that are observed with both BAL and penicillamine and that Dithiocarb is more effective than either of these agents in antidotal activity to nickel and other divalent cations. Soldatovic and Petrovic\textsuperscript{46} have shown that Dithiocarb is an effective antidote for guinea pigs administered lethal doses of bichloride or mercury. These investigators reported that 75 percent of the Dithiocarb-treated animals survived the lethal doses of mercury that were given and that 15 percent of the treated animals lived longer than the animals from the control group.

**Chronic Exposure to Mercury Vapor**

The present study is concerned with the treatment of a thermometer manufacturer who had been chronically exposed to hot mercury vapor (230° to 260°C.) for an hour per working day over a period of 13 years. Severe symptoms of both organic and inorganic mercury poisoning developed insidiously in the patient during the last six months of employment and finally reached the degree in which the patient became totally disabled.

**Protocol of History and Physical Findings**

A caucasian man 41 years of age complained of “shaking all over” to such an extent that he could no longer perform his manual work skills, write legibly, groom his body, feed himself, walk well or operate his automobile. He experienced a continuous unpleasant taste and had a foul breath. He had lost ten pounds of weight which he considered to be due to poor appetite and difficulty in feeding himself. The onset of symptoms began with a fine intention tremor of his hands which became progressively severe over a period of eight months. During this interval, unsteady walking, tremulous speech and a tremor of his mouth developed. This abrupt increase of symptoms was attended by fleeting paresthesias of the right arm. Subjective visual acuity decreased although his vision was found on admission to be 20/20 with intact visual fields.

For the past 13 years, the patient was the owner, employer and principal worker in a business which manufactured precision thermometers. He worked over a standup bench in a room without a hood or exhaust fan and performed the opera-
tion of filling the thermometers. Metallic mercury was heated to 230° to 260°C and then introduced into a vacuum system wherein evacuated thermometer tubes became filled with hot mercury by capillary action. After the thermometer tubes were filled, the vacuum was released, the mercury allowed to cool and the tubes were sealed. This operation required one hour each working day. It is noteworthy that the patient's manufacturing plant had received annual state inspections and that the plant had always been given approval for the maintenance of satisfactory and safe working conditions.

The patient sought medical attention during the six months prior to our contacts with him. His complaints to other physicians were concerned mainly with tremors, ataxia and blurred vision for fine print. It was noted that he was hostile, bellicose and irritable. Prior to our contacts, the patient had been intensively studied at another hospital where his disabilities were diagnosed as a psychosis with benign essential tremor. In the main, medication consisted of the oral administration of various tranquilizers. Mental institutional care was recommended; however, before being institutionalized, he was referred to a neurosurgeon in a second hospital for evaluation of the possibility of an intracranial tumor. It was at this hospital that an alert resident obtained the history of exposure to mercury and referred him for opinion.

Outstanding observations on physical examination were the marked resting tremors on all four extremities, head, tongue and mouth; gross intentional tremors; and fasciculations of pectoral muscles. The patient was ataxic and could not walk in tandem. He had a stuttering speech, was anxious, irritable, self-conscious and had impaired concentration and recall of recent memory. At times he would become belligerent and confused. He was unable to feed or groom himself. Although the patient complained of pain in his teeth, there was no evidence of periodontal disease, mobility of the teeth, soft tissue lesions or acute abscess. Evaluations of heart, lung, liver and gastro-intestinal tract were essentially normal. Visual acuity and fields were within the normal limits.

The routine laboratory analyses were essentially within the normal ranges of values although on admission the urine contained a trace amount of glucose and the concentrations of serum glucose and urea nitrogen were slightly elevated to 160 and 25 mg per dl, respectively. Cerebrospinal fluid, cerebral blood flow, brain scan, computerized brain transaxial tomogram, electromyogram, nerve conduction velocities, electroencephalogram and electrocardiogram were all normal.

Results

The diagnosis of chronic mercury vapor poisoning was established by the findings of 2.9 mg of mercury in a 24-hour collection of urine (normal, 0.006 to 0.020 fig) and by an increased concentration of 240 fig of mercury per dl of serum (normal, 5 to 20). The mercury analyses were made by the method of Toffaletti and Savory.50

A resumé of the clinical data and the results of mercury analysis in serum, urine and sweat are given in table I.

Three chelating agents were used in succession during the first two months of treatment: 2,3-dimercapto-1-propanol [British Anti-Lewisite (BAL)]; d-penicillamine and Dithiocarb (sodium diethyldithiocarbamate). The first day after receiving BAL, the urinary excretion increased to 25.2 mg and the concentration of mercury in serum remained essentially unchanged. Following the initial dose of BAL, the patient developed a severe reaction for an hour which included chills, sweating, violent tremors and burning sensations over the entire body. BAL therapy was continued for two
TABLE I
Resumé of Clinical Data

<table>
<thead>
<tr>
<th>Date</th>
<th>Therapeutic Agents</th>
<th>Hg Urine mg/day</th>
<th>Hg Serum ug/l</th>
<th>Hg Sweat ug/l</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/15</td>
<td>Before therapy</td>
<td>2.9</td>
<td>240</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/16-17</td>
<td>BAL (1st day)</td>
<td>25.2</td>
<td>235</td>
<td></td>
<td>Severe reaction.</td>
</tr>
<tr>
<td>1/17-30</td>
<td>BAL (average 3 days)</td>
<td>14.8</td>
<td>340</td>
<td></td>
<td>Rapid improvement. Severe ulcerative stomatitis.</td>
</tr>
<tr>
<td>1/31-2/13</td>
<td>Penicillamine (average 3 days)</td>
<td>0.5</td>
<td>255</td>
<td></td>
<td>Return of symptoms.</td>
</tr>
<tr>
<td>2/14-16</td>
<td>None</td>
<td>0.4</td>
<td>78</td>
<td></td>
<td>Mental confusion, dermatitis &amp; urticaria.</td>
</tr>
<tr>
<td>2/17-3/4</td>
<td>Dithiocarb (average 4 days)</td>
<td>1.2</td>
<td>240; 370</td>
<td></td>
<td>Favorable response, alert.</td>
</tr>
<tr>
<td>3/5-7</td>
<td>BAL</td>
<td>1.4</td>
<td>290</td>
<td></td>
<td>Mentation &amp; aids for daily living improved.</td>
</tr>
<tr>
<td>3/8-6/20</td>
<td>Sweats &amp; physio-therapy</td>
<td>0.7</td>
<td>290; 100; 32</td>
<td>45</td>
<td>No defects of mentation, calculation or judgment.</td>
</tr>
<tr>
<td>6/20-11/17</td>
<td>Sweats &amp; physio-therapy</td>
<td>0.2-0.08</td>
<td>40; 4</td>
<td>6</td>
<td>Slight intention tremor, - eventually disappeared.</td>
</tr>
<tr>
<td>After 6 months</td>
<td>Continuing sweats &amp; physio-therapy</td>
<td>0.017</td>
<td>10; 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>After 1 year</td>
<td></td>
<td>0.006-0.20</td>
<td>5-20</td>
<td>0-trace</td>
<td></td>
</tr>
</tbody>
</table>

NORMAL VALUES
Urine 0.006-0.20  Serum 5-20  Sweat 0-trace

weeks and, during this period, the average daily urinary excretion amounted to 14.8 mg of mercury. The concentration of serum mercury became increased during this period to 340 μg per dl. This increase is believed to reflect the mobilization of mercury from the tissues into the blood at a rate more rapid than that at which mercury was being excreted in urine and feces. Under BAL medication, the patient improved rapidly. For the first time in several months, he was able to get out of bed and feed himself. However, after two weeks of BAL medication, the patient developed a severe ulcerative stomatitis and BAL had to be discontinued.

After the discontinuance of BAL, the patient was placed on d-penicillamine medication (2 to 5 g orally) for approximately two weeks. During this period, the urinary excretion of mercury decreased to 0.5 mg per day, the patient experienced the return of severe tremors, ataxia and the inability to groom himself. After a period of two days without medication, the patient was placed on a schedule of Dithiocarb therapy (1 g daily in divided doses) for two weeks. Under Dithiocarb medication, the urinary excretion of mercury became increased to an average of 1.2 mg per day and the concentration of mercury in the serum became increased to 340 μg per dl. However, during this period, the patient developed intermittent episodes of mental confusion, incoherency, nausea, vomiting, dermatitis and urticaria. The patient was again placed on BAL medication for a period of three days and then made a dramatically favorable clinical response. He became alert, had diminished tremors and was able to groom himself. The urinary excretion of mercury averaged 1.4 mg per day and the concentration of serum mercury was 290 μg per dl.

On the basis of the clinical response, the increased excretion of mercury in urine and the increased concentration of mercury in serum, presumably from the mobilization of mercury from tissue stores, there is no doubt that BAL was the most effective of the three chelating agents administered for the treatment of the patient. Dithiocarb medication was
Effective in increasing the urinary excretion of mercury; d-penicillamine appeared to have little or no effect in relieving symptoms or in increasing the amount of mercury excreted in the urine.

Following the second course of BAL medication, the patient was placed on a daily regimen of sweats and physiotherapy, principally walking at first and then jogging. Parenthetically, diaphoresis and exercise have been used in Spain for many years for the treatment of mercury poisoning in cinnabar miners. During the first three months in which our patient followed a regimen of diaphoresis and exercise, the concentrations of mercury in his sweat ranged from 30 to 45 \( \mu \text{g} \) per dl. After six months of sweats and physio-therapy (and with complete abstinence of all medication and alcohol), the concentrations of mercury in the sweat decreased to 10 and 6 \( \mu \text{g} \) per dl and the amounts of mercury excreted in the urine and the concentrations of mercury in serum returned to values that were within the normal ranges.

The patient made a complete and uneventful recovery. Previous defects of locomotion, mentation, calculation, judgment and memory all disappeared. A specimen of the patient's handwriting before treatment, a specimen of his handwriting five months after admission and his signature eight months after treatment are all shown in figure 1.

The initial diagnosis made by the attending physicians at the hospital where he was first studied was a functional one, i.e., psychosis with benign essential tremor requiring institutional care. Although the etiology of the psychosis had not been ascertained, the studies at the first hospital were comprehensive.

**Diagnostic Considerations**

Intoxication from chronic exposure to mercury vapor is usually of occupational origin. It is encountered in a variety of

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**Figure 1.** (A) Signature on hospital admission; (B) Handwriting five months later; (C) Signature after eight months of therapy.
industries that include mercury mining operations, chlor-alkali plants, felt hat, mirror and battery industries, instrumentation and electrical equipment manufacturing plants using mercury and also in chemical, research and industrial laboratories. Chronic intoxication to mercury vapors is unlikely to occur from the non-occupational exposure to the trace amounts of mercury normally present in the atmospheric environment.

A listing of many of the symptoms encountered in the syndrome of chronic mercury poisoning is given in table II. It should be emphasized that the signs and symptoms may be quite variable from patient to patient.

Attempts have been made to differentiate between the symptoms of chronic poisoning due to inorganic and organic mercurials. The point of view has been held that the two forms of mercury produce two separate disease entities. For example, in an intensive clinical and animal experimental study on poisoning by methyl mercury compounds, Hunter et al found severe generalized ataxia, dysarthria and gross constriction of the visual fields to be present in all of their patients. In their studies on experimental animals, certain parts of the nervous system were selectively damaged: the peripheral nerves and posterior spinal roots first and the posterior columns and granular layer of the middle lobe of the cerebellum later. The triad: ataxia, dysarthria and constricted visual fields—are regarded as the classical signs of organic mercury poisoning. On the other hand, chronic inorganic mercury poisoning is characteristically associated with tremors (Danbury shakes), mental changes (erythmismercurialis, erethism), dermatitis, stomatitis, renal, gastro-intestinal, muscular and other disturbances.

Changes in mentation appear to be more prevalent in persons exposed chronically to the inhalations of mercury vapor than those otherwise exposed. Investigations in experimental animals suggest a reasonable explanation for this clinical observation. Thus, Berlin et al have shown that the inhalations of mercury vapor in mice is attended by as much as a ten-fold greater concentration of mercury in the brain than in animals receiving injections of equivalent amounts of mercuric salts. Magos and Berlin et al extended these investigations to rats, rabbits and monkeys. When mercury vapor is absorbed through the alveoli, it was found that, owing to lipid solubility of the vapors, more mercury diffuses into brain tissues than when mercury is otherwise introduced into the body.

It is noteworthy that poisoning from the inhalation of mercury vapor is not usually attended by the severe renal lesions, such as those that develop when inorganic mercurial salts are ingested. Furthermore, delayed skin sensitivity reactions appear to occur more often in persons who have been chronically exposed to organic rather than inorganic mercurials. Such a reaction, previously

<table>
<thead>
<tr>
<th>TABLE II</th>
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<tbody>
<tr>
<td>Main Symptoms of Chronic Poisoning from Mercury Vapor</td>
</tr>
<tr>
<td><strong>NEUROLOGIC</strong></td>
</tr>
<tr>
<td>Headache, vertigo, peripheral neuritis, numbness in extremities, ataxia, tremors, agraphia, dysmetria, dysdiadokokinesia.</td>
</tr>
<tr>
<td><strong>PSYCHIATRIC</strong></td>
</tr>
<tr>
<td>Erythmismercurialis, erethism, irritability, excitability, forgetfulness, irrational outbursts of temper, anxiety, depression, insomia.</td>
</tr>
<tr>
<td><strong>ORAL AND GASTRO-INTESTINAL</strong></td>
</tr>
<tr>
<td>Salivation, stomatitis, metallic taste, foul breath, spongy gums, swollen salivary glands, nausea, vomiting, diarrhea.</td>
</tr>
<tr>
<td><strong>GENITOURINARY</strong></td>
</tr>
<tr>
<td>Polyuria, anuria (occasionally glycosuria).</td>
</tr>
<tr>
<td><strong>DERMATOLOGIC</strong></td>
</tr>
<tr>
<td>Dermatitis, urticaria, delayed hypersensitivity of skin, dermatographia.</td>
</tr>
<tr>
<td><strong>OCULAR</strong></td>
</tr>
<tr>
<td>Eyelid fasciculations, constrictions of normal fields, discolorations of lens capsules.</td>
</tr>
<tr>
<td><strong>RESPIRATORY</strong></td>
</tr>
<tr>
<td>Symptoms of pneumonitis with low grade fever (Leucopenia?).</td>
</tr>
<tr>
<td><strong>NON-SPECIFIC</strong></td>
</tr>
<tr>
<td>Muscular weakness, ready fatigue, anorrhexia, anosmia.</td>
</tr>
</tbody>
</table>
reported by us, was observed in an organic chemist following an accidental spill of phenyl mercuric acetate over his hands.

Kark and coworkers hold the opinion that inorganic and organic mercury poisoning need not be considered entirely distinct diseases. It is our feeling that although the inorganic and organic forms of mercury poisoning manifest over-lapping signs and symptoms; nevertheless attempts to separate them into two entities is justified within reasonable limits. Moreover, poisoning from inorganic mercury may be further subdivided, since a clear differentiation may be made between poisoning caused by the ingestion of inorganic salts of mercury and that caused by the inhalation of mercury vapor.

**Discussion**

Conceivably, the response to the three chemotherapeutic agents might have been different if they had been administered in a different sequence. However, on the basis of the present study, our inclination is to recommend the intermittent short courses of BAL combined with sweatings and physio-therapy. After the severe symptoms are alleviated, sweating and physio-therapy may be continued throughout convalescence until the concentration of mercury in serum and the content of mercury in urine have decreased to the normal ranges of values.

**Mercury in Air**

Lauwerys and Buchet have recently pointed out that there is no international agreement on the concentrations of mercury vapor in the air to which workers may be exposed. In the United States, the American Conference of Governmental Industrial Hygienists established the toxic threshold limit value (TLV) for mercury vapor in air to be 100 µg per cubic meter. However, on the recommendations of an international committee that met in Stockholm in 1968, the threshold limit was reduced to 50 µg per cubic meter. In the Soviet Union, the maximum allowable concentration of mercury in air at industrial sites was established in 1944 to be 10 µg per cubic meter. The recent monograph by Traktenberg suggests that even this low value has little if any margin of safety.

Studies undertaken by the Soviet investigators and reviewed by Traktenberg direct attention to a syndrome which is referred to as micro-mercurialism. The Russian investigators have shown that warm-blooded animals subjected chronically to concentrations of mercury vapor from 6 to 50 µg per cubic meter developed "proteinemic shifts" (i.e., binding by SH groups), changes in conditioned reflex activity, disturbances of the thyroid gland and changes in the cardio-vascular system. On the basis of their experimental studies, Traktenberg concludes that the maximum allowable limit should be reduced below 10 µg per cubic meter.

In a survey of 40 laboratory technicians exposed to metallic mercury for several years, Lauwerys and Buchet found a significant correlation (r = 0.47; P < 0.05) between the urinary mercury concentrations and the levels of mercury exposure. These investigators suggested a level of 50 µg per liter as the maximum allowable concentration for urine. If concentrations increase above this level, they propose that corrective measures should be taken to reduce the environmental contamination.

The summarization is given in the abstract.

**Acknowledgment**

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References


