Mercury:
A Factor in Mental Disease ?

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Historically, hatters in England who used mercury in the felt hat industry got the shakes and became unstable—thus the term " Mad as a Hatter." During the Civil War, hats for the Union Army were made in Dansbury, Connecticut. Hatters there (also using mercury with felt hats) were noted for having the shakes—thus, the "Danbury Shakes." In Japan, mercury was dumped in Minamata Bay for years before it was discovered that the neurological disease seen in cats and people resulted from consuming a methylated form of this mercury in the fish they ate. Not only did victims get the shakes and emotional problems, they became debilitated with an illness called Minamata Disease (Kurland, 1961; Tsubaki, 1977; Study Group, 1968). Minamata disease somewhat resembles multiple sclerosis. Many people died of this disease.

Now mercury leaching out of silver mercury amalgams in people's teeth is becoming suspect of potentially creating or mimicking medical diseases. The purpose of this paper is to create an awareness of this potential by reporting clinical observations. Please keep in mind that these observations and data collection (processed in an interstate licensed laboratory) are done in a private practice, and that testing and funding were provided by the author alone. It is not intended to rival papers funded with millions of dollars and assembled by a host of university professors. Nonetheless, the significance of these suggestions could have a tremendous impact on dentistry, immuniology, cardiology, and psychiatric medicine. Please view this material in the light of, "How can I verify or contribute to a more thorough investigation of this concept?"

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Most Americans are aware that they have "silver fillings" in their mouths. Few are aware that less than half of that filling (around 35 percent) is silver, and that the bulk (around 50 percent) is mercury (Treptow, 1978). Dental fillings are composed of mercury, silver, tin, copper, and zinc. The approximate percentages are as follows:

±50%	mercury
33-37%	silver
12.5-13.5%	tin
0-3%	copper
0-1%	zinc

In 1973, I became aware of the potential hazards that dentally-placed mercury presents to the peripheral nervous system. Our first observation inferred that mercury toxicity creates or mimics neurological disorders both from the emotional and physical standpoint. Soon after our investigation of neurological problems began, other disorders became suspect. White blood counts (WBC) tend to come down when dental mercury amalgams are removed. Breaking the WBC down into a differential counted under Hoffman Optics shows a more definite count than the conventional light microscope, as described by Jess Clifford (Harper and Clifford, 1961). Electrocardiogram readings changed with mercury removal, as did urine mercury excretion, whole blood mercury, and the amperage created in the mouth. Obviously, this encompasses too much data to present in a brief article, but awareness is the purpose of this paper, and most importantly, the crux of mercury toxicity lies in its ability to alter different parameters in different people. As funds become available, each of these items will be explored to the best of our ability.

We have yet to find an individual who shows statistically significant, measurable changes in all areas we monitor, but most individuals show at least one dramatic change, either clinically or biochemically. Pattern inconsistency is probably the scientific stumbling block that has slowed mercury investigation over the past century.

The history of dentistry includes the "Amalgam War" of the 1840's. At that time, a dentist was removed from the roles of the American Society of Dental Surgeons if he placed silver-mercury amalgam or copper-mercury amalgam in a patient's mouth (Treptow, 1978). There was little concrete, scientific evidence to substantiate observations made at that time.

Today, we are seeing an increase in mercury interest, but we now have improved, scientific equipment unavailable to early researchers.

Mercury's action is multifocal. Specific cases that gave us reason to believe they were mercury-related included hematopoe-tic diseases like leukemia and Hodgkin's disease, neurologic diseases like multiple sclerosis, and emotional diseases so disabling that the victims are unable to function in society.

In reporting to the **Journal off Orthomolecular Psychiatry,** it would be appropriate to select a case that is primarily "psychiatric" in nature, but one which contains confusing overtones that exemplify the complexity created by a single factor—mercury.

After struggling with mercury allegations for seven years, I was not surprised to have an anxious mother call asking if I thought mercury could be a potential cause for her daughter's problems. Dentally-placed mercury had created so many non-diagnosed problems already that my attitude was one of: So what. One more ?

Frustrated parents all have one thing in mind. An instant cure. Tomorrow is never soon enough. I tried to explain that we were shooting in the dark, blindfolded, but she had already heard enough from other sources to know that even under adverse conditions (like shooting in the dark), we had seen positive changes in many patients.

This brings up a philosophical question that must be addressed. What are a dentist's limitations or obligations in the field of neurological disorders ?

David Bowerman, M.D., the pathologist on our team, said, "If dental mercury has created a problem, it is the responsibility of dentistry to investigate and correct that problem."

Upon examining the patient, I found a pimple-faced, shy, timid, 17 year old whose emotional energy rating was at the bottom of the scale. Occasionally, if something was really funny, she would move her head forward a bit; then her face would relax, and she would just exist. She wasn't really fighting the establishment, just didn't have much to say.

Her history was hardly that of a wallflower. Good grades, lots of friends, a cheerleader. . .

She had dropped out of school, couldn't be out of sight of her mother, had become introverted, highly investigated (50 practitioners had already seen her), and mildly

discouraged with life. Her "attacks" seemed to be the big thing. Attacks ? Yes. "Mother" described them as severe pains in the chest, a reversion to childlike speaking, and a concern about dying.

A few days later, Mother dropped in. "I don't think you appreciate the significance of my daughter's attacks. We were on our way through Colorado Springs when she started to have one. She's in your office. Why don't you go take a look at her ?"

My first reaction upon entering the office was one of sheer panic. "Daughter" was writhing in pain in a chair, trying valiantly to remove her heart from her chest cavity with her fingers. She would clutch her mother's arm and plead, "Mommy, I'm going to die. I'm going to die." Astute observation, I thought, under the circumstances and tended to support her diagnosis.

Our previous conversation indicated that these attacks lasted from two to three hours. I asked Mother how long this had been going on. Thirty minutes. Knowing what Aca-demia would ask at this moment, I called a technician to draw her blood, an assistant to bring a camera, and I began to mix a solution of the only thing I knew at the time could counter an acute episode of mercurialism: sodium ascorbate. We had observed in many other patients that vitamin C can lower hair mercury levels. I had her drink six grams dissolved in three ounces of water and waited. Within 20 minutes, she was feeling better and was in control of herself. Incidently, the blood tests (Hycel 17 profile) did not differ significantly from those taken when she was not having an attack.

Let's review those records again. "How long have these attacks been going on ?" "Six months." "Did anything unusual happen six months ago ?" "She had dental fillings placed." "How long from placement of the fillings before you noticed these chest pains ?" "Twenty minutes."

I found an appointment the next morning. We began to replace amalgam with "composite", a type of quartz-filled plastic commonly used for filling front teeth. Shortly after starting the second amalgam, Daughter began to have another attack. I told Daughter that she probably wouldn't be any better off lying on the reception room couch than she was in my chair. I asked how she felt about continuing with the amalgam removal. She wanted to "get it over with." We placed those amalgams in an amazingly short period of time.

As they left the office, I cautioned Mother and Daughter, "Sometimes we see a big dump of mercury out of the tissue into the urine after amalgam removal. If this happens, you could be much worse tomorrow." Prophetic. The next day, Daughter had four severe attacks, multiple hallucinations, and general chaos. The third day, she started an attack, then began to laugh. "It's not going to get me. It's not going to be bad." She was right. By day four, the attacks stopped. She has been free from them for ten months at the time of this writing.

My statement about mercury dumping was based on urinary excretion studies of previous patients. Two are graphed on the following pages.

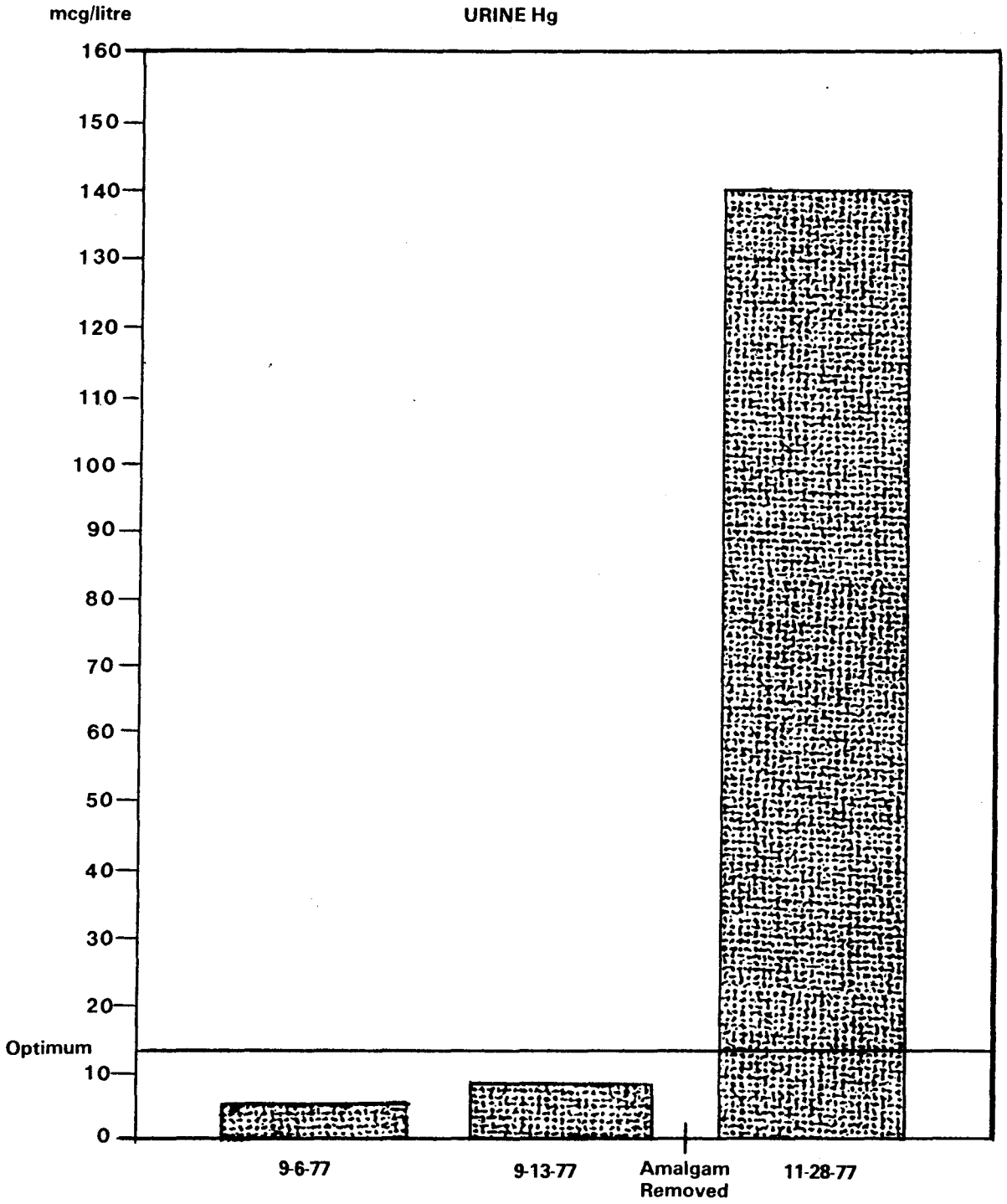
Even though our current patient had an enormous clinical reaction, her urinary excretion was actually much less than I anticipated. This is another example of individual variation.

We have records of one patient whose urinary excretion remained above 100 micrograms per liter for five years.

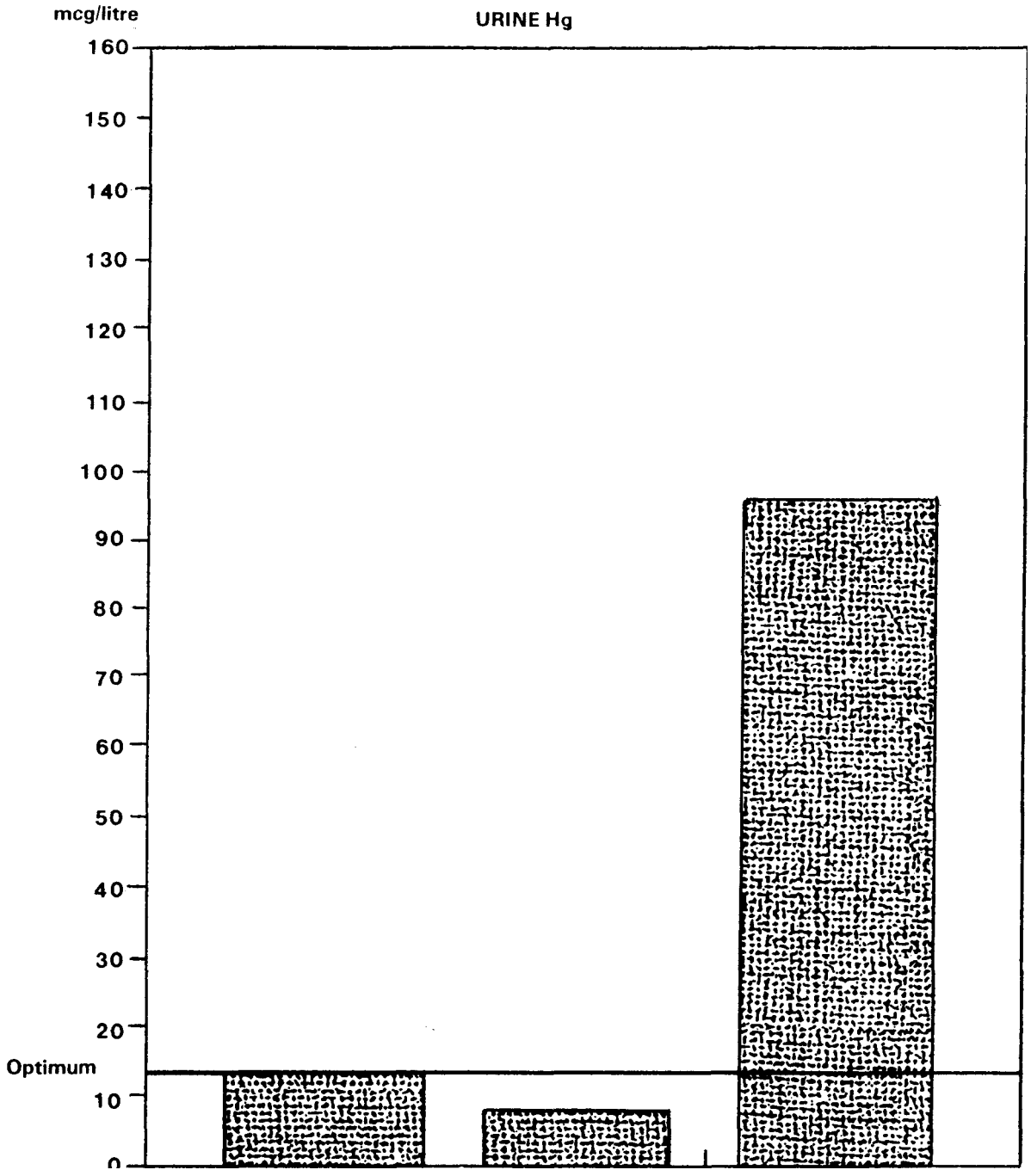
I feel that there is much to be learned from this patient's experience by reviewing her six month terror of multiple diagnoses. Without knowledge of mercury's ability to mimic or create a broad spectrum of disorders, her diagnostic transfer from specialist to specialist had left her condemned to the consensus of opinion: confinement in a mental institution.

There is a diversification of opinion as to who is best qualified to handle such a patient. Patients have to rely upon medical opinion and back fence gossip. In addition to the dozens of doctors who saw her during trips to hospital emergency rooms, it was recommended that she see

an internist	she did
a psychiatrist	she did
a hospital for testing	she did
an allergist	she did
an osteopath	she did



Graph 1, patient 1



Graph 2. patient 2

a cardiologist	she did
a minister	she did
a psychologist	she did
a gynecologist	she did
a chiropractor	she did
a psychotherapist	she did
a dentist	she did

All but one of the suggestions sound reasonable. Mercury toxicity supplied each practitioner with many signs and symptoms worthy of investigation. The trouble was that each investigation ended in a referral (in this case, another word for blind alley).

"Patient" had a history of mild episodes of psychiatric disturbances in July, 1972. She had one silver-mercury amalgam placed in June, 1972, which was approximately six weeks before her first appointment with a psychiatrist. The winter of 1973, her symptoms subsided. Dental records indicate that the baby tooth with the only filling in her mouth was removed at that time.

The current, advanced, bizarre symptoms began after placement of six small amalgam fillings ending on June 30, 1980. A brief outline of doctor referrals and their diagnoses follow. Many of these disorders have already appeared in the literature as being related to mercury, but few mention silver-mercury amalgam fillings as a potential source of mercury.

The following is a 1980 clinical summary with diagnoses:

June 27 Silver-mercury amalgams placed. No other fillings were present at this time.

June 30 More amalgams placed. Had fading sensation in the dental office. Diagnosis: after-effect of brief exposure to nitrous exide.

June 30 -
July 4 Patient had "under the weather" or "not up to par" general feeling.

July 4 Hyperventilated—thought she was going to die. Diagnosis: reaction to Permathene diet pill.

July 19 Difficulty in breathing—"allergy" doctor sent her to hospital for chest x-rays. Hyperventilated—given shot to calm her and dismissed. Diagnosis: Nerves. Referred to family doctor for checkup. July 21 To chiropractor for colonic. Constipated and experiencing pain in chest and back. Colonic made her sick. Diagnosis: Colonic stirred up toxins. July 22 Paramedic attention at rodeo. Couldn't breathe. Pain in chest & back. Dismissed. Referred to family doctor. July 23 Family doctor hospitalized her for 13 days of extensive testing. Diagnosis: Nerves. Referred to psychiatrist. Aug. 11 Psychiatrist saw her and referred her to a social worker for therapy. Attacks of pain in the chest now associated with pain running down the left arm. Aug. 19 Patient upset and "scared". Wanted her mother to hold her like a baby. Aug. 22 Referred to a cardiologist. EKG, thyroid and liver scans done. Referred to psychotherapist. Sept. 9 -
Sept. 16 Psychotherapy. Sept. 22 -
Oct. 2 To hospital again for tests. Diagnosis: Nerves.
Oct. 3 Referred to chiropractor
Oct. 13 Very painful period—unusual. Referred to gynecologist. Diagnosis: Possible hepatitus—referred to psychiatrist. Oct. 15 To psychiatrist. Oct. 16 To osteopath. Diagnosis: Possible gall bladder problem.
Oct. 30 An M.D. recommended hospitalization for psychotherapy.
Nov. 20 Much pressure to send her to a mental hospital for drug therapy.
Nov. 26 Patient now suicidal. Referred for council with a minister.
Nov.28 Emergency room trip because of intense pain in chest and dying sensation—near hysteria. Diagnosis: Nerves—referred to family doctor.
Nov. 30 Emergency room. Patient was fiery red from waist up. Felt like

she was burning up. Diagnosis: Nerves—referred to family doctor.

Dec. 1 Thyroid function tests—negative.

Dec. 4 Gall bladder series—negative.

Dec.8 Referred to dentist for mercury sensitivity evaluation.

Dec. 11 Amalgam removal

Dec. 12 Hallucinations, dizziness, dark brown urine, green stools, hysteria.

Dec. 16, 1980 All symptoms stopped. Back to school—graduated in June, 1981

Oct 1981 Ten months without recurrence of symptoms.

Before and after removal of silver-mercury amalgams.

Considering how many specialists were diverted to diseases other than mercury poisoning, is it possible that more people are experiencing the same thing ? In these cases, the psychiatrist is more apt to see the majority of these patients and should be aware of this route of investigation.

What are the tests that indicate mercury sensitivity ? This, of course, is the primary obstacle. Before and after tests show a variety of changes. A single test is of little diagnostic value, with the exception of the white blood count.

There are varying degrees of mercury sensitivity, but in patients who have experienced post-amalgam recovery from more severe symptoms, there is a suggestion of a correlation between the WBC and mercury sensitivity. Granted there are many diseases that produce an elevation of the WBC—infectious diseases in particular. Dr. Olympio Pinto of Rio de Janeiro has pioneered mercury studies for two decades. (His dentist father was noting disease-mercury relationships as early as the 1920's.) It was his suggestion, "remove amalgams when the WBC remained over 11,000 for three months without visible medical cause," that initiated my investigation into mercury sensitivity. In our practice, we have noted a trend for the WBC to seek a level around 5,000 to 7,000 count after amalgams are removed.

Trakhtenberg (1974) reports in **Chronic Effects of Mercury on Organisms** that his work presented in 1974 was the only work in the literature at that time demonstrating "the immunobiological effects and immune reactions of mercury or its derivatives." Our interest in studying the white cells and their "immunostatus differential" was increased after seeing Trackhtenberg's report. His work involved measuring the effects on neutrophils when white rats were exposed to 0.01 mg/M^3 - Hg in the air (six hours per day exposure).

U.S. safety standards for mercury exposure, as established by the Occupational Safety and Health Act of 1970 (1972) is 0.1 mg/M^3—ten times this amount. Later, in 1972, the standard was lowered to 0.05 mg/M^3 (ANSI, 1972) based on the studies of Smith et al. (1970). In the October issue of the ADA Journal, it recommends 0.02 mg/M^3 (Kantor and Woodcock, 1981). Russia established 0.01 mg as their maximum allowable count ration more than 30 years ago (Smelyanskiy and Nlanova, 1959).

Trackhtenberg (1974) states, "Changes in immunological reactivity brought on by low mercury concentration generally fall into two periods, one of stimulated immunological reactivity, and the second in which it begins to decline." Excerpts from his studies demonstrate these changes:

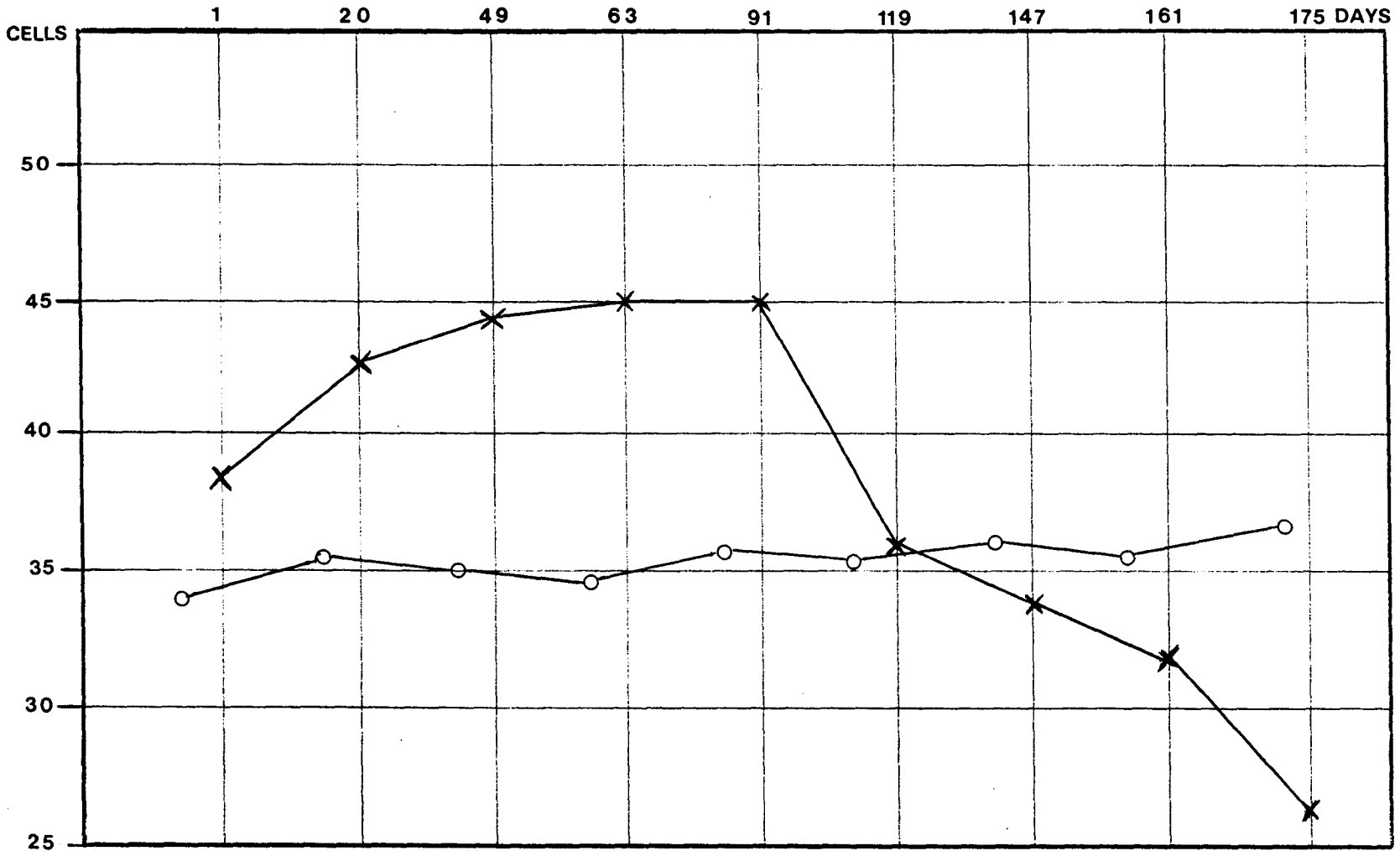
Changes in Neutrophil Count Due to Exposure *to* 0.01 mg/M3 HG

Day Number	1	20	49	63	91	119	147	161	175
Experimental Group (20 animals)	38.5	42.0	44.0	44.9	44.9	36.8	34.0	32.2	26.2
Control Group (18 animals)	35.0	36.1	36.0	35.9	36.8	37.0	38.4	37 5	38 6

In graph form, these figures appear on the following page.

On a limited basis, we have observed the same reaction upon placement and removal of amalgams. Primarily, we observe the WBC after removal of amalgams since we do not place amalgam in our practice. The initial counts above 6,000 tend to come down, and those below 5,000 tend to come up after amalgam removal. This subject needs carefully controlled study due to the many factors that influence white cell counts.

We have been monitoring the WBC, blood chemistry profile, hair analysis for minerals, the electrocardiogram, body temperature, white cell morphological changes, urinary excretion of mercury, whole blood mercury levels, urine vitamin C, specific gravity and pH, electrical current (amperage) generated in the oral cavity, and symptoms. Changes have appeared in all of these



Graph 3
o - control
x - experimental

parameters in some patients, but not always in a consistent or predictable fashion.

There are many cases of suggested mercury poisoning in the literature. Most of these references are in obscure journals and contain little more than anecdote type material. I feel that we have not known where to look, or what to document.

Urinary excretion has been mentioned by Goldwater in many articles as a potential source of diagnosis of exposure (Goldwater et al., 1962; Goldwater and Joselow, 1967). Recently, other researchers have noted that urinary excretion in people known to be exposed was lower than in non-exposed people (Frisberg and Vostal, 1972). Our feeling is that there is no correlation between exposure, onset of symptoms, and urinary mercury excretion. Many researchers agree with this statement (Smith and Moskowitz, 1948; McGill et al., 1964; Frisberg and Nordberg, 1972). There is one significant phenomenon that we have noted. That is, the condition we term "biological inversion."

We do not fully understand the phenomenon of "biological inversion," but would like to describe what we have observed. The normal range for urinary excretion of mercury is 2 to 20 mcg/liter. After the removal of the last amalgam—in some people, not all—the urinary excretion may go up substantially.

Our observations have led us to suggest that before biological inversion, the body accepts and absorbs some of the mercury that leaches out of amalgam fillings. When the last amalgam is removed, the body tends to rid itself of its mercury by excreting it in great quantities. This is what we term biological inversion—letting the mercury out of the cell instead of storing it. How long does it last ? The longest time we have been able to follow one patient is five years. Surprisingly, after five years, she was still excreting over 100 mcg/liter.

Even though mercury excretion continues for an extended period of time, there is usually a dramatic change in symptoms and other test parameters within days. White cell reaction can occur within 24 hours when they are elevated due to the presence of mercury. As an example of this, the following patient had maintained a WBC of 15,000 to 17,000 for about two years. Graph 4 shows the changes concomitant with amalgam removal.

She had run a "sub" temperature of around 1 degree Fahrenheit for at least ten years and, after the amalgams were out, it went up to normal within 24 hours. We offer no explanation for this, but submit it as another avenue open to investigation. Again, not all people running subnormal temperatures experience a change to normal when amalgams are removed.

Another interesting observation involving the "mental" patient described in this paper occurred during the first seven days after amalgam removal when 50 percent of her acne disappeared. Within 14 days, 50 percent of what remained was gone and, within a month, her skin was clear.

Let's take a brief look at all the diagnoses that had been considered in her case, keeping in mind that none of these diseases had been present before the amalgams were placed, and they all disappeared after the amalgams were removed: reaction to N_2O, hyperventilation, reaction to diet pills, chest pains, cardiovascular pain, thyroid, liver, gall bladder, painful menstruation, hepatitus, severe complexion problems, half the body turning red, and the most popular: nerves and mental disease. Strangely enough, most of these reactions are mentioned in the literature as being associated with mercury toxicity.

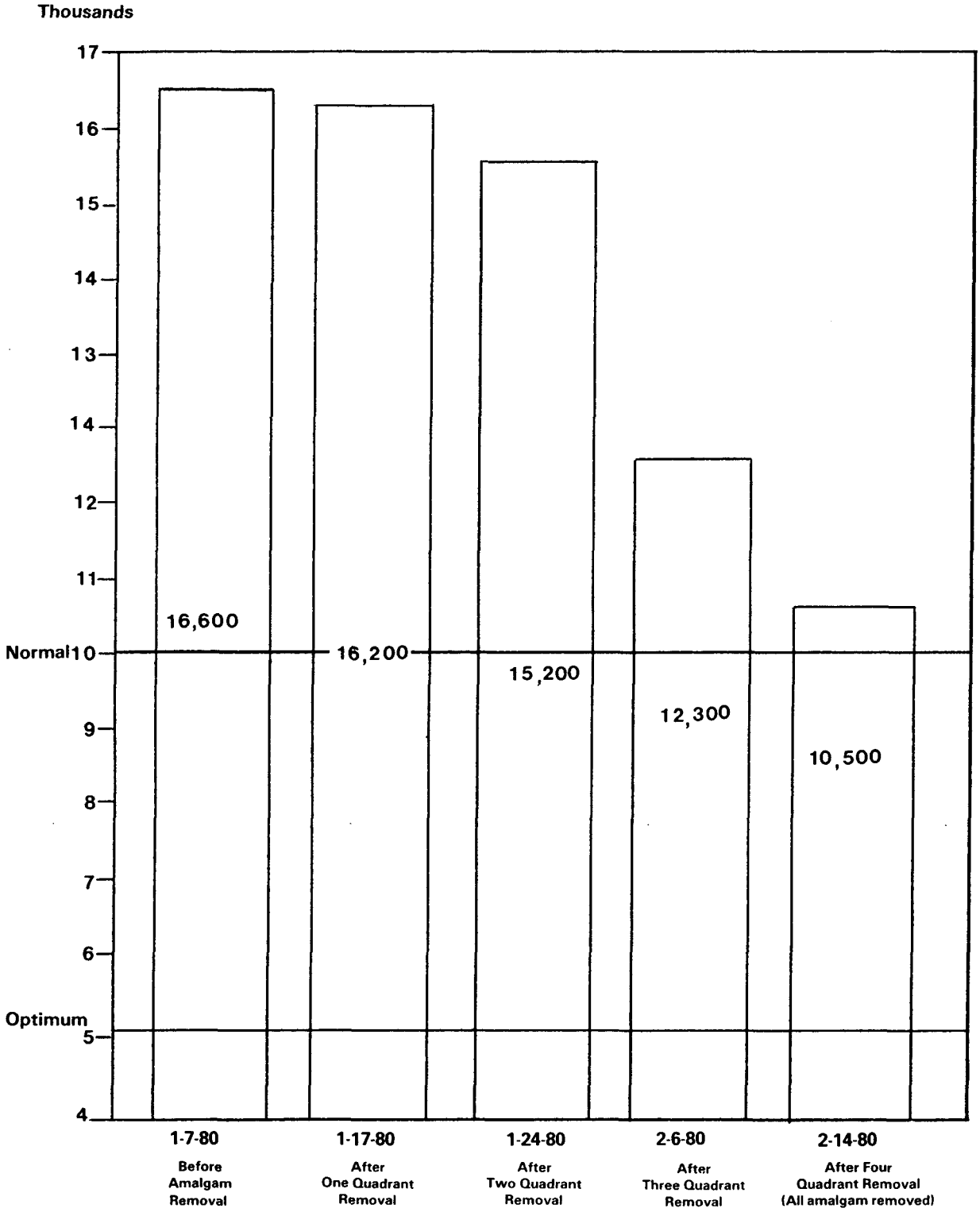
Schwarzkopf (1970), reporting in the **Pacific Coast Homeopathic Bulletin,** describes a case of severe stabbing heart pain and difficulty in breathing which was resolved upon removal of six amalgams.

He describes a 39 year old patient with thyrotoxicosis, endocarditis, and a pulse of 125. Severe weight loss was also present. Three amalgams were removed. In two weeks, the pulse was 96. In six months, the pulse was 72, all symptoms were gone, the patient was back at work and had gained 28 pounds towards normal weight.

He describes another "thyroid" problem of the opposite kind—an 18 year old who was excessively overweight and bloated in

WHITE BLOOD CELLS

GRAPH 4



appearance. Four amalgams were removed. The bloated appearance and overweight condition were resolved, and he grew taller.

Several researchers (Trakhtenberg, 1974; Ruskin and Johnson, 1949; Gessler et al.; Dahhan and Orfaly) implicate mercury in cardiovascular disturbances from the aspect of enzyme inhibition—specifically, those conditions containing the -SH or thiol receptor. Trakhtenberg (1974) says, "Thiol poisons affect several aspects of cardiac activity, mediatory and extra-cardiac nerve activity, myocardial contractility, and electrical activity." Chernyavskiy (1960) states that "chemoreceptors are related to tissue receptors." Others (Ruskin and Ruskin, 1953; Turpayev, 1950) say ... as a consequence of enzyme reactions affected by thiol poisons, including mercury, which are complex irritants of chemoreceptors. Additionally, toxic substances block thiol poisons, altering the metabolism of the nerve endings themselves and suggesting changes in the acetylcholine metabolism which will be mentioned later.

Saytanov (1960) found in rabbits exposed to low concentrations of mercury vapor that EKG "changes were characterized by a lowering and broadening of the P-waves." In our "mental" patient's case, a reversal of this P-wave broadening was seen when the mercury was removed. Her EKG P-wave changed as is shown below:

Electrocardiogram Changes

12-8-80		12-11-80	12-18-80	12-30-80
P-wave amplitude	1.5 mm	amalgam removal	1.0 mm	0.5 mm

We are not prepared to comment on the significance of these changes, but they do correlate with patient improvement and are the reverse of what Saytanov found regarding exposure to mercury.

Commenting on acetylcholine, Trakhtenberg (1974) states: "Currently many facts have accumulated indicating that cardiac function usually depends on cholinorecep-tor properties. The vagus nerve endings control acetylcholine having a highly specific effect on the heart muscle which incorporates cholinoreceptors (proteins), the active principle of which are -SH groups. The -SH group reaction capacity is affected by mercury and can change aspects of acetyline formation and tissue response to it. That is why we can conclude that mercury effects produce specific shifts both in the biochemical dynamics of the cardiac muscle and in its response."

More -SH information was presented by Trakhtenberg (1974), who stated, "Functional disturbance in the myocardiac is a consequence of disruption of extracardiac heart activity, regulation as a result of the continuous toxic effect of mercury on the myocardium and heart value, primarily through -SH blockage." Turpayev (1950), proposed that, "suppression of the contractile properties of the myocardium through the action of thiol poisons occurring as a consequence of -SH enzyme inactivation is directly linked with the energetic contractile act of the myocardium."

Trakhtenberg (1974) exposed mice to 0.05 mg Hg/M3 (the maximun U.S. standard for safety) for up to three months. Exposure time was six hours daily. There was no visible difference in behavior between the control and the experimental group until the second month. By the third month, the experimental group "acquired heightened reflex excitability, some increase in motor activity, and then, adynamia and depression, decreased appetite, and lowering of response to external stimuli."

From **Mercury in the Environment,** we see several suggestions that relate to our primary case under consideration. On page 117, it defines the classification of "mercury etiology" as having three or more of the following objective symptoms: tremor, thyroid, enlargement, increased uptake of radio-iodine in the thyroid, hematological changes, hypotension, liable tachycardia, dermographism, and gingivitis.

Even our patient's flush of redness can be found in the literature. 'For many years a rather uncommon disease of infants has

been recognized and called 'pink disease', infantile acrodynia, or erythredema, and is characterized by a redness and swelling of the extremities and certain other skin areas along with photophobia, irritability, loss of reflexes, and muscular hypotonia" (Wark-any and Hubbard, 1948). Dr. Warkany of the Children's Hospital of Cincinnati examined an infant suffering from this disease in 1945 and found a urinary concentration of 360 mcg/liter. A summary of 20 cases showed that most infants with pink disease had definitely elevated mercury levels—75 percent had more than 50 mcg/liter and 10 percent more than 400 meg/liter while most control infants showed undetectable levels.

Kantarjian (1961) summarized mercurialism in part as follows: anorexia, various emotional alterations, such as mood depression and timidity. Erethism, or blushing, is often common, but whether it is due to emotional disturbances or alterations of autonomic vascular control is unknown.

There is some mention of liver function abnormality and mercury. (Our patient had several liver scans done in the hospital.) Foulerton (1961) stated, "that mercury has an affinity for lipids, not only because of the solubility of HgCl2 in fat, but also due to formation of oleates, and is transported to the liver in the circulating blood fat. Liver damage then results in defective lipid metabolism.

Conclusion

It is clear that many ramifications of physical and mental health need investigation in light of the potentially hazardous effects of mercury toxicity. Suggestions have been forwarded that mercury leaching out of dental amalgam filling can affect the peripheral nervous system, immune system, and cardiovascular system, and that mercury in a biological system appears to create or mimic many disorders in these three areas. The case of a young girl has been presented which had been thoroughly investigated through hospital and direct evaluation by psychiatrists, internists, osteopaths, chiropractors, psychologists, and members of the clergy. The consensus of opinion was to place a previously healthy and productive individual in a mental institution—not because of any malice or neglect on the part of the professionals she consulted, but because of sheer lack of knowledge about the potential effects of mercury toxicity. Their conclusion was based on the vast array of physical and emotional problems the patient exhibited, even though none of these problems were present before her amalgams were placed, or after they were removed. When I received her graduation announcement, it hit me how great a tragedy had been averted—and what if hers is not the only case ?

Because it is common to refer patients like her with multifocal problems defying diagnosis to a psychiatrist or psychologist, it is especially important that professionals in these fields become more aware of the possibility of a common denominator for many such cases—mercury toxicity.

References

AMERICAN NATIONAL STANDARDS INSTITUTE (ANSI).: American National Standards Allowable Concentrations of Mercury. New York. 1972.

CHERNYAVSKIY. M.I. et al.: Hygiene and Sanitation 6. p. 100. 1960.

DAHHAN. S.S. and ORFALY. H.: Electrocardiographic Changes in Mercury Poisoning. Amer. Inst, of Cardiology 14. pp. 178-83.

FOULERTON. A.G.R.: Neurology II. p 639. 1961.

FRISBERG. L. and NORDBERG. G.F.: Inorganic Mercury: Relations Between Exposures and Effects. In: Mercury in the Environment. CRC Press. 113-140. 1972.

FRISBERG. L. and VOSTAL. A. (Eds.) Mercury in the Environment. CRC Press. 1972.

GESSLER, U.. JUNER. E. and GESSLER. Z.: Exptl. Med. 133. 178-183. and p. 394.

GOLDWATER. L.J.. JACOBS. MB and LADD. A.C.: Relationship of Mercury in Blood and Urine. Arch. Environ. Health 5. p. 537. 1962.

GOLDWATER. L.J. and JOSELOW. M.M.: Absorption and Excretion of Mercury in Man. Arch. Environ. Health 15. p. 327. 1967.

HARPER. H.W. and CLIFFORD. W.J.: The Immunostatus Differential. J. Int. Acad. Prev. Med. VI. 1. 1961.

KANTARJIAN. AD: Neurology II. p. 639. 1961.

KANTOR. ML. and WOODCOCK. R.C.: Mercury Vapor Exposure in the Dental Office. J. Amer. Dental Assoc. 103.3. Sept.. 1981.

KURLAND. L.T.. FARGO. S.N. and SEIDLER. H.: Minamata Disease. World Neurology 5. Nov.. 1961.

McGILL. CM. et al.: Mercury Exposure in a Chlorine Plant. J. Occup. Med. 6. 335-337. 1964.

MONTAGUE. P. and MONTAGUE. K.: Mercury. New York. Sierra Club. 1971.

OCCUPATIONAL SAFETY AND HEALTH ACT OF 1970.; Sec. 1910.93 of Title 29, Federal Register 37. No. 202. 22134-22144. Oct. 18. 1972.

RUSKIN. A. and JOHNSON. J.E.: Proc. Soc. Exptl. Biol. Med. 72. p. 572. 1949.

RUSKIN. A. and RUSKIN. B.: Journal of Pharmacal. Exptl. Therapy 107. p. 327. 1953.

SAYTANOV. A.O.: 1960. as quoted by Trakhtenberg: Chronic Effects of Mercury on Organisms. U.S. dept. of Health. Education and Welfare. National Institute of Health, p. 200. 1974.

SCHWARZKOPF, H.: Are All Dental Fillings Harmless? Pacific Coast Homeopathic Bulletin XXVII. No. 12. Dec. 1970.

SMELYANSKIY. Z.B. and NLANOVA. IP.: New Standards for Permissable Levels of Toxic Gases. Fumes, and Dust in the Air of Work Areas. Gige Trud. Prof. Zabol 5. 7-15. 1959.

SMITH. A.R. and MOSKOWITZ. S.: Urinary Excretion of Mercury. Monthly Review. New York State Dept. of Labor. Div. of Ind. Hyg. Safety Standards 27. 45-47. 1948.

SMITH. R.G. et al.: Effects of Exposure to Mercury in the Manufacture of Choline. Amer. Ind. Hyg. Assoc. J. 31. 687-700. 1970.

STUDY GROUP OF MINAMATA DISEASE.: Minamata Disease. Japan. Kumamoto University. 1968.

TRAKHTENBERG. I.M.: Chronic Effects of Mercury on Organisms. U.S. Dept. of Health. Education, and Welfare, National Institute of Health. 1974.

TREPTOW. R.S.: Amalgam Dental Fillings. Parts I and II. Chemistry. April. 17-20. May. 15-19. 1978.

TSUBAKI. T.: Minamata Disease. New York. Elsevier Scientific Publishing Company. 1977.

TURPAYEV. T.M.: 1950, as quoted by Trakhtenberg: Chronic Effects of Mercury on Organisms. U.S. Dept. of Health. Education and Welfare. National Institute of Health, p. 204. 1974.

TURPAYEV. T.M.: Biochemistry (USSR) 20. p. 456. 1955.

WARKANY. J. and HUBBARD. DM.: Lancet 1. p. 829. 1948.