Evidence Implicating Amalgam in Alzheimer's Disease

Excerpts from memo by Kip Sullivan - August 1996

Kip Sullivan is an attorney from Minneapolis that has recovered from an "incurable" illness after having his amalgam fillings removed. Since then he has been an advocate for making this treatment more readily accepted and available to others suffering from neurological diseases, and has done extensive research which is documented in a memo that he is presenting to several well known Alzheimer's medical researchers in the Minneapolis area. The following are excerpts from the memo, presented here with Kip's permission, without the extensive footnotes, references and tables contained within the memo. Anyone wishing additional information with regard to the content presented here should contact Kip at (612)823-1459.

Robert Terry et al. are the authors of a book which reviews current knowledge about Alzheimer's Disease (AD). The book was recently described as "the best single book on the topic" by the New England Journal of Medicine and as "a most useful reference for practitioners of medicine" by the Journal of the American Medical Association. At page 363 of this book, Terry et al. state: "Taken together, these studies suggest that chronic low level Hg toxicity in AD should be considered as a potential pathogenetic factor in AD." Because amalgam is the dominant source of human exposure to mercury, one may paraphrase this conclusion as follows: "Chronic low level exposure to mercury from amalgam should be considered as a factor in AD."

The scientific evidence supports the following statements:

1. People with amalgams have much more mercury in their bodies, including their brains, than people without amalgams; the extra mercury carried by people with amalgams constitutes half to three-fourths of all mercury found in their bodies;
2. People who die of AD have elevated levels of mercury in their brains; in rat brains and human brain homogenate, mercury blocks a biochemical process that is also blocked in the brains of AD victims; mercury causes emotional and mental symptoms frequently found in AD patients;
3. Twin studies suggest an environmental cause of AD; epidemiological evidence (the temporal and racial distribution of AD) suggests mercury from amalgams plays a role in AD;
4. The hypothesis that mercury causes AD is consistent with other hypotheses about the etiology of AD, namely, that apolipoprotien E status is a strong predictor of AD, that education and estrogen protect against AD, and that trauma to the head may trigger AD;
5. Mercury may be a cause of other diseases of the central nervous system, including amyotrophic lateral sclerosis, multiple sclerosis, and Parkinson's Disease.

Overview of mercury body burden studies

The typical amalgam filling is 50% mercury, 30% silver, and 20% other metals. The appendix presents the research which documents the conclusion that mercury escapes amalgam and accumulates in tissue throughout the body, including the brain. The great majority of these studies were performed on people, not animals. I summarize them briefly here.

Mercury escapes from the amalgam in three ways: (a) corrosion, caused by electrical currents passing through the filling; (b) what one expert calls "direct dry evaporation;" and (c) removal of small pieces of the fillings caused by chewing. The liberated mercury is taken into the body via (a) inhalation, (b) swallowing and (c) absorption into nearby mouth and nasal tissue. People with amalgams have at least twice as much mercury vapor in their mouths, twice as much mercury in their blood, three to six times as much in their urine, at least six times as much in their kidneys, and double the amount of

mercury in their brains and other body parts as people without amalgams. Several of these mercury burden studies found a correlation between the number of amalgam surfaces and mercury levels in the brain. One of the latest and most disturbing of these studies found that women with ten or more filled teeth give birth to babies with twice as much mercury in their bodies as mothers with two or fewer filled teeth.

Estimates of amounts of mercury absorbed from amalgams

That amalgams can double the body's mercury level and triple the urine's mercury level suggests that mercury from amalgams is the source of at least half to two-thirds of all the mercury humans are exposed to. Over the last four or five years, a substantial body of evidence has emerged which indicates that most of the mercury absorbed by people in industrial nations comes from their amalgam fillings.

In 1991 the World Health Organization..... concluded the daily dose of mercury from the environment is 2.6 ug..... If the correct figure for absorption of mercury from amalgam turns out to be 8 ug, then the total mercury absorbed per day from all sources -- the environment plus amalgam -- is 10.6 ug, according to the WHO data. This means amalgam mercury constitutes three-fourths of all mercury absorbed by the body (8 ug is 75% of 10.6 ug). If 10 ug turns out to be the correct figure for mercury taken up from amalgams, then the total absorbed is 12.6, which means about four-fifths of all mercury absorbed comes from amalgams (10 ug is 79% of 12.6 ug).

In a 1995 report prepared for Health Canada (Canada's national health department), Richardson estimated that amalgam mercury constitutes half of the mercury absorbed daily by adult Canadians.

Whether amalgam mercury is the source of 50% of all mercury absorbed, as Richardson's data indicates, or three-fourths, as the WHO data suggests, mercury from amalgams contributes a substantial portion of the mercury absorbed each day, not a "very small" portion as the ADA would have the public believe.

All the studies that directly link mercury to Alzheimer's Disease have been done at the University of Kentucky. Three of these found higher mercury levels in AD brains than in age-matched, neurologically normal brains. The first of these, published by Ehmann et al. in 1986, examined levels of 16 trace elements in the cerebral cortex of 14 AD and 28 control brains. The authors reported significant differences in eight of the 16 elements; the largest differences were in mercury and bromine (elevated in AD brains) and rubidium (reduced in AD brains). Two years later Thompson et al. examined levels of 14 trace elements in three areas of the brain that undergo marked change in AD victims -- the hippocampus, amygdala, and nucleus basalis of Meynert (nbM) (11 AD and 11 control brains). They found the same imbalances in these regions of the brain that they had found in the cerebral cortex in their earlier study (with the exception of rubidium). The mercury imbalance in nbM was the largest of these differences. In 1990, Wenstrup et al. measured 13 trace elements at the "subcellular level" in material taken from the temporal lobe of ten AD patients and 12 normal controls. They reported significant differences for five elements: elevated mercury and bromine, and diminished selenium, zinc, and rubidium. The authors noted that selenium "is known to play a protective role in biological tissue against mercury toxicity" and that zinc may also play a protective role "by forming a zinc-thioneine complex with which the mercury will replace zinc forming a less toxic mercury-thioneine complex."

In this last study, the authors discussed three mechanisms by which mercury could cause AD symptoms: reducing the brain's ability to utilize tubulin, a protein used to manufacture microtubules; alteration of cell membranes; and other forms of cell dysfunction caused by the loss of zinc and selenium, a loss "possibly" due to the role zinc and selenium plan in detoxifying mercury. The University of Kentucky researchers have conducted several studies examining the first hypothesis.

Tubulin is a protein found in every cell. In nerve cells, tubulin is an element of microtubules, described by one writer as "neuronal railroad tracks, transporting molecules between the cell body and nerve terminals." The hypothesis that AD brains cannot use tubulin to make microtubules is at least ten years old. The contribution of the University of Kentucky researchers has been to show that mercury may be the cause of this defect. Three studies by these scholars support this hypothesis. The first demonstrated that the tubulin-guanosine-5'-triphosphate (GTP) interaction is greatly reduced in AD brains; the second that EDTA "complexed" with mercury reduces GTP-tubulin interaction in normal human brain homogenates; the third that the brains of rats exposed to mercury vapor demonstrate the same marked reduction in tubulin-GTP interaction demonstrated in humans in the first study. In the third study (which was done with scholars at the University of Calgary medical school), rats were exposed to mercury vapor at 300 micrograms per cubic meter for four hours a day for periods ranging up to 28 days. The authors noted that 300 micrograms is "a level detectable in mouths of some human subjects with large numbers of amalgam fillings." The authors found that by the fourteenth day the rate of tubulin-GTP binding had been reduced by 75 percent. "The identical neurochemical lesion of similar magnitude is evident in Alzheimer brain homogenates. . .," stated the authors.

A large body of research on the effect of accidental mercury poisoning indicates that mercury causes emotional and mental disturbances very similar to those that appear in many AD victims. Here is a summary of the emotional and mental symptoms commonly associated with AD:

Patients with Alzheimer's exhibit changes in personality and social skills. They . . . may become socially uninhibited or lose all initiative and interest in activities. These patients often have delusions, hallucinations, and sleep disorders. They sometimes show grossly inappropriate judgement and sometimes are misdiagnosed as being depressed or psychotic.

Every one of these symptoms are common symptoms of mercury poisoning.

The American Dental Association and its state affiliates cite a 1995 study of 129 elderly nuns as conclusive evidence that amalgams cannot cause AD. But this study lacked a control group, and is therefore useless. The authors divided 129 nuns into five groups depending on how many teeth and how much occlusal amalgam surface they had. The authors referred to these groups as "risk categories," which is to say the authors assumed that an old woman's risk of contracting Alzheimer's-like symptoms is higher if, at the time of the study, she had teeth with amalgams than if she had no teeth. The reader is led to think, in other words, that the edentulous (toothless) nuns were the least likely to get AD because their amalgam load at the time of the study was lower than that of nuns in the other four categories. This is an absurd assumption. No one other than the authors of this study postulates that mercury from amalgams causes AD overnight. If amalgam mercury causes AD in elderly women, it does so over decades, beginning possibly when the women got their first fillings, which for most people occurs when they are children. Having made this untenable assumption, the authors proceeded to measure the mental abilities of the nuns with the Mini-Mental State Examination, a commonly used test of mental function, and other tests, and found no difference in test scores among the five "risk categories." "In fact," note the authors in an attempt to get the reader to think the edentulous group was the control group, "the group with the highest amalgam surface area had the same mean Mini-Mental State Examination score as the edentulous group." Nowhere in this article does the phrase "control group" appear.

What the authors of this study should have done was to measure the lifetime exposure of the nuns to amalgam mercury as well as mercury levels in their brains during autopsy, and see if there is a correlation between lifetime exposure and brain mercury levels, or between either of these variables and performance on cognitive skills tests. In such a study, nuns edentulous by the time of the study may turn out to be the "risk category" with the highest exposure to amalgam mercury. After all, a toothless person has almost certainly suffered from greater tooth decay than someone with teeth, and, therefore, has probably been exposed to more amalgam mercury for a substantial period of time than someone who remains dentate. Because mercury stays in the brain for decades, one could be edentulous for a very long time and still have more brain mercury than someone who is dentate and has a lot of amalgams.

Like Parkinson's and other adult-onset diseases of the central nervous system that now afflict humanity, AD was rare or nonexistent prior to the onset of the Industrial Revolution. The Industrial Revolution, which greatly increased human exposure to mercury in the work place and in the environment, got underway in the mid-1700s. Amalgam was invented in Europe around 1820 and was widely used throughout Europe and North America by 1850. Parkinson's, MS, and ALS were first mentioned in medical journals in the 1800s. It was not until 1906 that German psychiatrist Alois Alzheimer announced that he had found a "strange disease of the cerebral cortex" in the course of performing an autopsy on a demented 56-year-old woman. The woman's brain contained an unusual number of the plaques and tangles now considered to be the defining symptoms of AD.

Scholars affiliated with the University of Minnesota and the Minnesota Pollution Control Agency have demonstrated that atmospheric mercury levels rose dramatically during the 1800s. In a 1992 article published in Science, they reported that the rate at which mercury accumulated in seven lakes "increased by a factor of 3 to 4 during the past 140 years." Because the seven lakes are at great distance from industrial sources of mercury and are spread out over a large area encompassing northern Wisconsin and northern and central Minnesota, and because "the deposition rates are relatively uniform" across the seven lakes, the authors concluded that the mercury sources were "regional if not global." Data for six of the seven lakes indicated a substantial increase in deposition rates around 1850 and another increase around 1920. Subsequent research indicates mercury deposition rates may have peaked in the 1970s.

Amalgam is the dentist's stock in trade. Today a typical adult carries ten amalgams weighing a total of about ten grams, of which five grams is mercury. What little research there is on the rate at which mercury escapes amalgam suggests about half a gram of mercury will escape from these ten fillings over the ten-year life of these fillings, and most of this mercury will be absorbed by the bearer of the amalgams. To put a half-gram in context, consider these facts: Half a gram of mercury dropped into a ten-acre lake warrants the promulgation of a fish advisory for the lake in Minnesota; the tennis shoes with mercury in them that were banned by the Minnesota legislature in 1994 contained half a gram of mercury per shoe.

Mammals have been evolving on this earth for 70 million years. A permanent tripling or quadrupling of environmental mercury levels over a mere century may well have had some impact on human health even if amalgams had never been invented. If in fact amalgam accounts for at least half of all mercury absorbed today, then we may say with some accuracy that the introduction of amalgam coupled with the increase in atmospheric mercury exposed humanity to at least a six-fold increase in mercury over approximately 100 years. A century is a very short period of time for any organism to develop new defense mechanisms against unprecedented levels of something as toxic as mercury.

Blacks have far fewer amalgams in their heads than whites; they also die far less frequently from AD and MS. According to a 1996 report from the Centers for Disease Control, white people are nearly two times more likely to die from AD than blacks. According to a 1978 study of MS, "The . . . US Army suggests that the risk of MS for white males is 2.5 times the risk for black males." Blacks in the US and England have long had much lower rates of caries than whites. According to the latest health survey by the National Center for Health Statistics (which was conducted over the 1988-91 period), adult blacks had only one-third the number of "filled surfaces" as whites (8.5 versus 22.8). A small part of this difference was due to more untreated decay among blacks, but most of it was accounted for by less decay in blacks. The small difference in longevity in blacks and whites is too small to account for a two-fold difference in AD mortality rates.

AD as an inflammatory response

The hypothesis that AD is caused by mercury from amalgam and the environment is consistent with the theory that AD is an inflammatory disease caused by the body's reaction to an infection or environmental insult. The evidence that AD "fits the paradigm of the idiopathic rheumatic disorders" was recently presented by Aisen and Davis. "According to this model," wrote the authors, "an unknown set of circumstances results in an initial insult triggering an inflammatory reaction in the

brain. The inflammation becomes self-propagating, or it continues because the obscure inciting factors persist." They argue that the acute phase response may augment production of beta-amyloid, the protein found among the plaques in AD brains. They conclude that "cytokines, acute phase proteins, activated microglia, and complement," all mechanisms which can be triggered by the acute phase response of the immune system, are "involved" with AD, either as causes or consequences, and that anti-inflammatory drugs may "alter" the progression of AD. Mercury may well be one of the "obscure inciting factors" that triggers the inflammatory response.

Some direct evidence that anti-inflammatory drugs may delay the onset of AD already exists. McGeer et al. reported data suggesting that "the prevalence of Alzheimer disease in patients with rheumatoid arthritis is unexpectedly low and that [the use of] anti-inflammatory therapy might be the explanation." Aisen and Davis cite two other studies that reach the same conclusion.

ApoE status

Researchers at Duke University have recently shown an association between AD and the presence of a blood protein called apolipoprotein E type-four (apoE4). People with two apoE4 genes have eight times the risk of developing late-onset AD as those with two apoE3 genes, and those with two apoE2 genes have an even lower risk. The Duke researchers subsequently found that microtubules are more likely to break down in people with apoE4 than E3 genes. They surmise that this breakdown is caused by the inability of a protein called tau to participate in the construction of microtubules. I reviewed above the research by Haley et al. indicating the breakdown may be caused by the AD brain's reduced ability to utilize another protein -- tubulin -- in the construction of microtubules, an inability triggered by the presence of mercury.

Haley hypothesizes that the apoE4-AD correlation is due to the inability of apoE4 protein to transport mercury out of the brain. The following statement by David Kennedy, a dentist and researcher on amalgam toxicity, summarizes Haley's theory:

> *The function of [the apoE] protein is to transport cholesterol out of the brain... The difference between apoE2, E3 and E4 is that E2 has two cysteines, in E3 one cysteine is replaced by an arginine, [and] in E4 both cysteines are replaced by arginine. [Haley] explained that unlike cysteine the arginine does not pick up mercury since it contains no sulfur. Sulfur is called a mercaptan (Latin for mercury capture). Mercury loves sulfur more than other molecules. It will drop whatever it is attached to and bind with sulfur.... E2 people, who are less likely to get AD, have a protein that carries mercury as well as cholesterol out of the brain by binding mercury with sulfur seats.*

Haley's explanation of the relationship between apoE status and AD is consistent with a 1988 study of trace element levels in hair and nails of AD patients done at the University of Kentucky by Vance et al. The study found no difference in hair mercury levels of patients with and without AD; it found that AD patients had lower mercury levels in their nails than did the non-AD controls. In his review of the literature on amalgam toxicity, Richardson cited the study by Vance et al. as evidence against the conclusion that amalgams cause AD. If Haley's description of apoE is correct, or if research demonstrates some day that for other reasons some humans are have diminished capacity to scavenge and excrete mercury, we should expect to find that AD victims have the same or lower mercury levels in hair and nails despite having above-average levels in their brains.

If Haley's thesis is correct, the following hypothesis seems quite plausible: because of a reduced capacity to excrete mercury, mercury (from amalgam and nonamalgam sources) builds up more rapidly in the brains of people with the epoE4 gene; rising mercury levels in the brain eventually cause the destruction of microtubules which in turn leads to neuron death; rising mercury levels trigger an inflammatory response that accompanies, and may contribute to, neuron death.

AD afflicts many who do not carry the apoE4 gene. What that signifies is that an intolerable level of mercury can build up in people regardless of their apoE status. This intolerable level may be reached (1) because of exposure to high levels of mercury, (2) because of exposure to other toxins and

stressors that reduce the ability to cope with mercury, (3) because of genes other than those controlling apoE status, or (4) some combination of these three conditions.

Other theories: estrogen, education, head trauma, electromagnetic fields, and smoking

The hypothesis that mercury causes AD is consistent as well with other theories of AD etiology.

Evidence supports the claim that estrogen supplements reduce the risk of AD in women. Animal experiments indicate that estrogen stimulates nerve growth, perhaps indirectly by its effect on nerve growth factor. Higher education levels may also protect against AD. Like estrogen, education (or perhaps the habits of mind that higher education encourages) may help the brain maintain or construct neurons. These findings are consistent with the mercury hypothesis. Estrogen and education may protect against AD by minimizing or compensating for the neuronal destruction caused by mercury.

Trauma to the head is occasionally mentioned in the literature as a factor associated with a higher incidence of AD. At least one expert believes head trauma is a risk only for people with the apoE4 gene. A blow to the head may aggravate the toxic effect of mercury in at least two ways: it might stress the body and thereby weaken the body's ability to withstand the presence of mercury; the trauma may fracture fillings and increase the victim's exposure to mercury.

I base the latter explanation on my familiarity with the health history of June Varner, a Little Falls woman who overcame nearly paralyzing confusion by getting her amalgams removed. Her inability to concentrate and make decisions set in after a 1978 car accident. The problem was severe. June stated in a letter to me, "I can remember looking down at my shoelaces months after the accident; I could not remember how to tie them. I knew that I knew how to do it but I could not remember." Other symptoms that appeared after the accident included headaches, vertigo, nausea, extreme fatigue, and memory loss. These symptoms persisted until 1992 when her dentist discovered that several teeth in the upper right side of her mouth with amalgams in them were cracked. The replacement of these amalgams with crowns eliminated the mental confusion and the other symptoms that came with the confusion. June speculates that the blow to her head suffered during her auto accident allowed mercury from her fillings greater access to her brain.

Golden mentions a study done by Eugene Sobel at the University of Southern California which found "that the onset of Alzheimer's is unusually high in dressmakers and tailors," possibly because sewing machines create large electromagnetic fields (EMFs). EMFs could augment the damage that mercury does to the brain in two ways. They may render the blood-brain barrier more permeable to toxic material, including mercury; they may accelerate the release of mercury from amalgams (see discussion of the role of electricity in releasing amalgam mercury in attachment).

Finally, smoking seems to play a protective role against AD. The reason for this may be that nicotine has the opposite effect on neurotransmitters that mercury has. Whereas mercury inhibits the uptake of, or otherwise reduces the effect of, dopamine, norepinephrine, serotonin and acetycholine, nicotine increases the levels of these neurotransmitters. This may explain why people with amalgams tend to smoke more than people without amalgams.

ANECDOTAL EVIDENCE

Mary and Monica are, like me, patients of a Bloomington dentist under attack by the Minnesota Board of Dentistry for his stance on amalgam (he won't use them, and he takes them out). Mary, Monica and I have come to know each other well in the course of working together to defend him and other mercury-free dentists. Monica and Mary both recovered from Alzheimer's-like symptoms after the dentist took their amalgams out.

Monica's symptoms were mild; she would say one word when she meant another, and her memory deteriorated. She overcame these symptoms with a combination of amalgam removal and

supplements. Because Monica's mother had AD for many years, Monica has little doubt she has the genes that make her susceptible to AD. Upon her mother's death, Monica and her sister asked the Mayo Clinic to determine the levels of aluminum, nickle and mercury in their mother's brain. The results: aluminum, normal; nickle and mercury, extremely high.

Mary's symptoms were severe, especially for a woman in her thirties which is when Mary's health deteriorated. She suffered from memory loss so severe she could not remember people she had known for years or how to drive to places she had driven to for years. She suffered fits of rage so intense she had to lock herself in the bathroom to avoid hurting her children. She recovered quickly after having her amalgams removed. We can only speculate whether Mary and Monica had the plaques and tangles of fully developed AD.

I am told dozens, perhaps hundreds, of other Americans could tell similar stories. I have the phone numbers of several of them.

Overview of the evidence linking amalgam mercury to ALS

The evidence supporting the hypothesis that amalgam mercury is a cause of ALS is as strong as the evidence implicating amalgam in the onset of AD. Like AD, ALS was not described until well after the Industrial Revolution had begun. According to Felmus et al., "[T]he original description of amyotrophic lateral sclerosis" appeared in 1869. Like the evidence linking amalgam with AD, the evidence linking amalgam to ALS includes research showing elevated mercury in ALS patients and anecdotal evidence of recovery from ALS after amalgam removal. Unlike the AD literature, the literature on ALS includes at least five articles describing the appearance of ALS symptoms in people exposed to organic mercury and mercury vapor, and, for some victims, the disappearance of these symptoms after exposure to mercury ceased. Like the literature on AD, the literature on ALS contains speculation that ALS may be an inflammatory disease caused by a toxin. I find it intriguing that familial ALS and AD "have [both] been mapped to chromosome 21."

The scientific literature on mercury, amalgam and ALS

The first of several reports on ALS-like symptoms triggered by exposure to mercury appeared in 1954. The article described an ALS-like syndrome in a 39-year-old farmer who absorbed organic mercury from a fungicide he used on oats. At least three similar articles have been published since. One described ALS symptoms in 11 Iranians who ingested bread made with wheat treated with a fungicide containing ethyl mercury; another reported ALS symptoms in two men exposed to mercuric oxide and mercury vapor in a factory that manufactured mercuric oxide; the third described a 54-year-old man who developed ALS symptoms three-and-a-half months after he spent two days gathering liquid mercury from old thermometers.

A number of people who have had amalgams removed have recovered from ALS. In a 1994 article, Redhe and Pleva described such a recovery by a 29-year-old Swedish woman. She had been diagnosed with ALS by the neurology department at the University Hospital in Umea, Sweden. This same department pronounced her free of ALS in August 1984, five months after her amalgams were removed. Nine years later the woman was still free of ALS symptoms.

Mercury has also been implicated by studies examining health histories of groups of people diagnosed with ALS. Felmus et al. found that 25 ALS patients were more likely to be exposed to mercury and lead than a control group of sick people with non-ALS diagnoses (although the two groups did not differ in number of amalgams). Sienko et al. sought to explain the sudden appearance of ALS in six residents of Two Rivers, Wisconsin over the 1975-1983 period. They found that the ALS victims suffered more instances of physical truama, reported more cancer in their families, and had eaten more fish from Lake Michigan than had 12 controls.

Some of the University of Kentucky scholars who examined mercury levels in AD victims were among the authors of a similar study of ALS patients. They found that seven deceased ALS victims had more mercury in their brains than did nine deceased controls who did not have ALS, and that blood cells of

40 living ALS patients contained more mercury than the blood cells taken from 31 living controls. Interestingly, ALS victims had lower levels of selenium in blood serum. Selenium has been shown to "protect experimental animals against the toxcity of heavy metals, such as mercury . . . ."

Unpublished anecdotal evidence of recovery from ALS after amalgam removal

The Redhe-Pleva article on the Swedish woman who recovered from ALS after amaglam removal is the only such report in the peer-reviewed literature I know of. However, similar but unpublished stories are numerous. I recount one here. Cynthia Hughes is a Nevada woman who recovered from ALS after her amalgams were removed by Dr. Hal Huggins, a Colorado dentist who practiced mercury-free for 23 years until the Colorado Board of Dental Examiners took his license because of his public criticism of amalgam. Cynthia and the neurologist who diagnosed her appeared on a four-part television report entitled "Toxic Teeth" which aired on a Las Vegas TV station in the early 1990s. The reporter stated: "Cynthia could not walk or talk until she had her mercury fillings removed. Even her doctor was amazed by her sudden improvement." At this point the camera showed a doctor sitting at his desk with his name and specialty shown on the screen -- "Dr. Hal Griffith, neurologist." Dr. Griffith stated, Cynthia "had a dramatic . . . complete recovery."

Evidence linking amalgam mercury with multiple sclerosis and Parkinson's

Like AD and ALS, MS and Parkinson's are adult-onset diseases that either did not exist or were rare prior to 1800. Like the AD and ALS literature, the MS and Parkinson's literature offers some evidence that toxins in general and mercury in particular plays a critical role in the etiology of these diseases. Finally, there is substantial anecdotal evidence that amalgam removal reduces MS symptoms dramatically in many MS patients. I know only two Parkinson's patients who have had their amalgams removed, and one of them improved.

Evidence that mercury is swallowed and absorbed into mouth tissue: the corrosion studies

Although experts think most amalgam mercury enters the body is mercury that escaped via evaporation and inhaled, I start with a review of the evidence that mercury is released via corrosion because that evidence is the oldest. Electrical currents, created by the amalgams themselves, liberate mercury from the filling and allow it to travel into the saliva and mouth tissue, including gums and pulp. The liberation of mercury and other metals from the amalgam by electrical currents is called corrosion and, sometimes, "oral galvanism."

It has been known since at least 1878 that amalgams create these electrical currents. It has been known since at least 1881 that amalgams discolor and soften the dentin (the soft material between the enamel and the pulp), and since at least 1953 that one phenomenon causes the other, that is, that the electrical currents in the mouth cause fillings to corrode and release metals that then travel into the dentin causing discoloration. The 1953 study examined 300 freshly extracted teeth containing amalgams and found a "greenish to grayish black discoloration" in the dentin of 85% of the teeth. In this discolored dentin the authors found "relatively large amounts of mercury . . . with smaller amounts of silver, zinc, tin and copper. . . ." The authors recreated this same greenish-black color in the dentin by running electric currents through the amalgam, leading the authors to conclude that the migration of mercury and other materials from the amalgam was precipitated by "intermittent galvanic action arising from within the amalgam filling itself." Other studies have confirmed this finding. By the 1970s it was established that mercury was migrating into the gums, pulp, and, by the 1980s, the jawbone.

Evidence that mercury is inhaled: the vapor studies

A report in 1979 that fillings give off mercury vapor led to the revival of the amalgam debate in this country. Prior to 1979 the position of the ADA and most dentists was that a newly inserted filling would give off mercury vapor for a few hours but after that mercury vaporization ceased. The 1979 study was important because it established that chewing released mercury vapor even from old fillings. The study was done by three researchers at the University of Iowa. They announced preliminary results in a letter to Lancet, a widely read British medical journal. They reported that chewing gum for 15 minutes caused mercury vapor levels in expired air to rise by as much as 17 times in five individuals with amalgams whereas gum chewing by two subjects without amalgams had no effect on the amount of mercury in their breath. Although other research was also published that year linking amalgams to mercury levels in the blood, the Lancet announcement is the one cited throughout the scientific literature as the first study in recent times demonstrating that mercury escapes from fillings. The final report on the Iowa research published two years later concluded that chewing increased the amount of mercury vapor in the breath of subjects with amalgams by an average of 15.6-fold and that, even before chewing, subjects with amalgams had three times as much mercury in their breath as the non-amalgam subjects.

Patterson and two other New Zealand researchers reported in 1985 that brushing one's teeth with a soft tooth brush for one minute also stimulates mercury vapor release. Mercury vapor levels rose from an average of 3.1 ng/L of expired air before brushing to 8.2 ng/L after brushing. Even eating musli, a soft cereal, raised mercury vapor levels.

In 1988, Langworth et al. published a study of "intratracheal mercury levels" (that is, mercury levels in the air in the windpipe) of ten subjects (all, apparently, with amalgams). Prior to brushing their teeth, tracheal mercury vapor levels were below the instrumental detection limit of 1 ug/m3 for five subjects and ranged from 1-6 ug/m3 for the other five. After brushing, the average level for all ten subjects rose to 56.4 ug/m3.

The last study of oral mercury vapor levels was published in 1994 by Siblerud et al. They reported that people with amalgams had twice as much mercury vapor in their mouth air as people without amalgams prior to chewing and four times as much after chewing.

The reader should note that the breath levels just reported are averages among the people volunteering for the various studies and therefore do not reveal the high levels of mercury vapor reached in some people's mouths. Dr. Wayne King, a Georgia dentist, in testimony before the FDA Dental Panel in 1991, made this remark indicating that oral mercury vapor levels can reach very high levels in some of his patients: "I have been absolutely horrified to see some of the numbers that I have measured coming out of some of the mouths of my patients; for instance, 200 micrograms per cubic meter in a suicidally depressed patient."

That mercury vapor is released by amalgams is no longer debatable. As one expert who defends amalgams put it at the 1991 National Institute of Dental Research conference, "The question is not if but how much mercury vapor is released."

Kip Sullivan - August 1996