SPECIAL ARTICLE

Amalgam - The New Fish Bait
Sam Ziff

The ADA News, December 1, 1986 contained an article titled "mercury - from fish to fillings". The article was apparently written to "quiet" the growing concern of the public and practicing dentists regarding the "true" safety of mercury/amalgam as a dental material.

The objectivity of this article on a scale of 1-10 rates 0. In the face of overwhelming scientific evidence raising serious questions on the propriety of the dental profession continuing to use mercury amalgam, the ADA has once again demonstrated it's impotence to provide any corresponding scientific research supporting it's pro-amalgam public statements.

As a trade organization collecting millions of dollars annually from it's dues-paying dentist membership as well as exercising the major influence on how and what the U.S. Government grant funding in dental research is expended upon, the ADA certainly has the means and influence to insure that suitable scientific research is done to verify their currently "unscientific" position that the small amount of mercury vapor released from dental amalgam fillings does not pose a health hazard except in those few individuals hypersensitive to mercury. If I were a dues-paying member of the ADA I would demand to know why action has not been taken to fund the research required to once and for all answer the question of mercury amalgam filling biocompatibility in humans, especially in view of the inherent liability related to each dentist's continued use of amalgam if not scientifically proven to be harmless.

I would find the motives of the ADA much more in line with their chartered responsibilities if they had set out to replicate the scientific studies of Dr Kuntz et al. and Dr. Abraham et al., rather than attempting to denigrate them. It is incomprehensible to me that the only ADA response elicited by an article that was published in the premier refereed research journal of the dental profession was to wait three years after publication and then attempt to attack and impugn the entire research project and its conclusions.

For those of you who have never read the Abraham et al. article I would like to quote the last paragraph: "The health hazard of blood mercury levels associated with dental amalgams has not been documented. It is however, of interest that the average blood mercury level for the amalgam group reported here (0.7 ng/ml) was similar to that reported by Kuntz et al. (1982) for a group of 57 pre-natal patients (0.79 ng/ml). In the latter group, a significant correlation was found between the history of
stillbirths and mercury levels in both maternal and cord blood. Further, the occurrence of malformed infants in previous births correlated significantly with pre-natal mercury blood levels. None of the histories obtained from that group reflected possible exposure to mercury except for that related to the presence of dental amalgams. It is important to note that the blood mercury levels were measured with the same type of apparatus for both studies. Given these facts, the small increase in blood mercury levels that is statistically associated with dental amalgam restorations should be a matter of concern for dentists as well as for the recipients of these restorations." Those conclusions should have raised immediate concerns by responsible personnel within the U.S. Government and the ADA. To do less was, and is, totally irresponsible.

Back to our "fish story" that started all of this. Dr. P.L. Fan (associate secretary, ADA Council on Dental Materials, Instruments, and Equipment), and Dr. Dan Langan (director of the ADA Mercury Testing Service) were given "creative license" by Dr. Enid A. Neidle, (Director of the ADA Division of Scientific Affairs) to further the pro-amalgam cause. Drs. Fan and Langan singled out the article by Abraham et al. as the sole reference on which to exercise their "creative license".

The first order of business seemed to be to call into question the statistical significance of the blood mercury levels reflected in the Abraham et al. article: "the mean blood mercury concentration in a group of subjects with dental amalgams was found to be 0.7 nanograms per milliliter (ng/mL) compared to 0.3 ng/mL for subjects with no amalgam. 'Taking into account the density of blood, this comes to about 0.7 ng per gram of blood (ng/g)-but how much is that' Dr. Langan asked. A nanogram is 1 x 10^-9 g or 1 billionth of a gram, so 0.7 ng/g means that there is 0.7 of one billionth of a gram of mercury in each gram of blood. 'This is unarguably an extremely minute quantity,' Dr. Langan said. 'It should be noted that the group with no amalgams had a blood level of 0.3 ng/g and the difference between groups was only 0.4 ng/g. To assume the difference has biological significance is questionable'."

It would seem from the inference drawn by Dr. Langan that the entire research findings of Abraham et al., were without merit and by association Kuntz et al., were placed in the same category. It would have been extremely useful if Dr. Langan had cited some basis or accepted authority for his statement claiming that higher mercury blood levels in individuals with amalgams is only of questionable biological significance.

Perhaps we can shed a little light on the subject for Dr. Langan and tell him why he should be concerned about the "insignificant" statistical difference between people with amalgams and those without amalgams. In normal individuals there is almost
complete absorption of mercury vapor from the alveolar parts of the lungs (F. Nielsen Kudsk 1965). Other researchers have indicated an 80% absorption rate or stated another way 80% of inhaled mercury vapor enters the bloodstream (Gerstner and Huff 1977). Gerstner and Huff go on to state: "Elemental mercury, after entering the bloodstream is oxidized through the mercurious into the mercuric ion according to: Hg\(^0\) -- Hg\(_2\)\(^{2+}\) -- Hg\(^{2+}\). Completion of these reactions requires several minutes; because of the delay, elemental mercury exists in the blood for a sufficiently long time to reach all tissues and organs. This fact has serious consequences. In its elemental form, mercury easily penetrates the blood-brain barrier and infiltrates nerve cells, where final oxidation proceeds. Such an ease of penetration—which is well documented for the blood-brain barrier—also applies presumably to the placental barrier, as indicated by a few observations during human pregnancies. By overcoming the two critical barriers, elemental mercury is particularly dangerous during long-term or chronic exposures, representing a potentially serious hazard in many occupations."......"Elemental mercury, after tissue incorporation, swiftly converts into the mercuric form and thus behaves essentially like inorganic compounds. Nevertheless, two characteristic aspects require special attention. First, before its conversion elemental mercury strongly accumulates within the central nervous system. And second, the subsequently existing mercuric form returns back into the bloodstream only at a very low rate of speed."

It would appear from the above information that only a small percentage of inhaled elemental mercury vapor would be in the blood and then mostly in its inorganic form. There are two other aspects of how the body handles mercury that must also be considered. 1) The half-life of mercury has a slow component that represents about 15% of each dose. This slow component will continue to accumulate over the lifetime of an individual under chronic exposure conditions (Rothstein and Hayes 1960, Sugita 1978). 2) Each dose of mercury vapor in multiple exposures behaves independently of the other. The kinetics are such that small chronic doses are more completely absorbed than large acute doses (Rothstein & Hayes 1960).

Consequently, when all of the above factors are considered in relationship to the following indisputable scientific facts a 4 ng/ml difference between amalgam and no amalgam blood chemistries is certainly not biologically insignificant as Dr. Langan would have everyone believe: elemental mercury vapor is released from amalgam fillings; there is a positive correlation between numbers and surfaces of amalgam fillings and the amount of mercury vapor released; Friberg et al., (1986) have shown a positive correlation to numbers and surfaces of amalgam fillings and mercury content of the brain; Kuntz et al., (1982) drew some positive correlations between stillbirths/malformations and prenatal maternal blood mercury levels; Abraham et al., showed a
positive correlation between numbers and amalgam surface areas to increased blood mercury levels; and Snapp et al., 1986 who confirmed the causal relationship between the presence of dental amalgam fillings and elevated blood mercury levels.

More of our "fishy" article. Dr. Fan, not to be outdone by Dr. Langan, now provides us with a little slight of hand magic by immediately jumping into methylmercury blood levels that will produce toxic symptoms. However, I am at a loss to see what that has to do with the article by Abraham et al., as not once in the entire article do they use the word toxic or toxic symptoms or toxic blood levels or toxic anything. The whole purpose of their research project was to determine if the transfer to the blood of amalgam-originated mercury actually occurred and if it did, what the relationship between mercury levels in blood and in mouth air and amalgam numbers and surface area might be. What the Abraham et al. paper showed was, in fact, that there was such a relationship. So I can only conclude that Dr. Fan was attempting to attack the last paragraph of their article (previously quoted above) by citing data from the Japanese Minamata and Niigata poisoning episodes (methylmercury in fish) by stating "The lowest whole blood concentrations of mercury at which toxic symptoms occurred in these episodes was 200 ng/g. "Even with this more toxic form of mercury," Dr. Fan observed. "the lowest blood mercury concentration that produced symptoms of toxicity was 285 times the level found in the group of subjects with amalgams."

The ADA News article then goes on to elaborate showing that because of the Japanese incidents the FDA, after applying a safety factor of 10, established 20 ng/g as an acceptable mercury level for whole blood from the consumption of fish contaminated with methyl mercury. Further, that the FDA set a limit of 500 ng/g (0.5 parts per million-ppm) of mercury in fish and seafood. To set the frame work for their subsequent calculations, the authors state: "Swedish studies have shown that a blood concentration of 20 ng/g corresponds to a daily intake of 30,000 ng (0.03 milligrams) of mercury by a man weighing 150 lbs." Two ounces (60g) of fish containing 500 ng/g of mercury would provide 30,000 ng of mercury. Using the intake to blood level ratio just stated, Dr. Neidle points out that the 0.7 ng/g mercury blood levels reported in the Abraham et al. subjects with dental amalgams could be reached by the daily consumption of only 2.1 g of tuna fish.

Here again, Neidle et al. are comparing apples to oranges in that Abraham et al. were referring to the findings and conclusions of Kuntz et al. which drew a positive correlation between maternal mercury blood levels and stillbirths and malformed infants. What Neidle et al. also did not tell you was that in the Japanese epidemics as well as those in Russia and Iraq, there were incidents of organic mercury poisoning that caused "brain damage in infants in spite of little or no evidence of maternal
At this point the ADA News article gets real fishy as the "researchers" are now going to go from fish to amalgam fillings and see "how many amalgams would be needed to bring the blood mercury concentration to the FDA's maximum acceptable level of 20 ng/g". After going through a number of mathematical machinations Neidle et al. end their display of scientific wizardry by stating "Since the lowest blood concentration at which symptoms of toxicity appear is 200 ng/g," concluded Dr. Fan, "one would need 1000 occlusal amalgam restorations before any signs of toxicity would be observed. All one can really say about the measurement of mercury vapor from amalgams is that it can be measured. There's just not enough vapor to link it to toxicity."

There are several things wrong with the analogy and their terminology. Firstly, the question is not toxicity, but rather the clinically observable symptoms of toxicity. Secondly, based on the existing published scientific research documenting the amount of mercury vapor released from amalgam fillings, and dose-response relationships, death would occur long before significant blood/mercury levels were obtained. The reason for this is the manner in which the body handles inhaled mercury vapor, which is covered in detail in previous paragraphs. Most of each inhaled dose of mercury vapor would be deposited in tissues and organs. So what the blood would show is mostly that portion undeposited in the tissues and organs that was subsequently ionized. In this context it is important to remember that the site of toxic action of mercury compounds in the body is closely related to the distribution pattern (Passow et al. 1961). Third and lastly, most competent researchers working in this field have concluded from the available scientific data that no threshold below which symptoms will not occur, can be established at this time. Even the U.S. EPA Report in discussing clinical and sub-clinical effects of mercury vapor state that no threshold has clearly been established.

I am extremely interested in determining how Neidle et al. were able to totally disregard the transport, biotransformation, distribution and deposition as well as blood levels of inhaled mercury vapor originating from dental amalgam fillings, to focus only on the blood mercury levels associated with the ingestion of methylmercury in fish. Somehow or other, the parallel escapes me. While we are speaking of mercury blood levels I think it important to point that a certain degree of confusion exists on exactly what is meant by mercury blood levels. For example, on page 1625, Goodman & Gillmans The Pharmacological Basis of Therapeutics states under the paragraph titled Diagnosis of Mercury Poisoning: "Since methylmercury is concentrated in erythrocytes and inorganic mercury is not, the distribution of total mercury between red blood cells and plasma indicates
whether the patient has been poisoned with inorganic or organic mercury. Measurement of total mercury in red blood cells gives a better estimate of the body burden of methylmercury .... Concentrations of mercury in plasma provide a better index of the body burden of inorganic mercury. However, the relationship between body burden and the concentration of inorganic mercury in plasma is not well documented. For example, exposure to vapor results in concentrations in brain about ten times higher than those that follow an equivalent dose of inorganic mercury salts."

While we are speaking of mercury vapor released from dental amalgam I would like to point out that it was mentioned in the EPA report. Citing the work of Gay et al on page 4-11 the paragraph concludes with the following sentence: "The significance of these findings to potential human health effects is wholly unknown." (It's too bad Dr. Clarkson didn't check with Neidle et al. for the answer).

Although not related to the article in the ADA News, in view of the fact that Dr. Langan is director of the ADA Mercury Testing Service (which is responsible for all the urine mercury determinations of dentists participating in the program) I thought it would be appropriate to touch on the subject of intake and excretion.

Tompsett and Smith (1959) published a very interesting report on the subject. Using 24 subjects without a history of abnormal exposure to mercury, urine mercury determinations were made. The authors found that the mercury content was so low as to be indeterminable with the procedures used and it was concluded that the daily urinary excretion of mercury by the normal is less than 5 ug/day. They also did feces mercury determinations. All were collections over a period of four or five days. The mercury content of the feces was assessed at 55 to 180 (average 90) ug/day. The authors conclude from their results that the average normal intake of mercury in the diet is of the order of 100 ug/day.

An intake of 100 ug/day from dietary sources does not seem at all possible on the basis of data available on mercury content of various foods. Most scientific papers have been using 10 or 20 ug/day as a normal mercury intake from dietary sources. This is not too far off from the most recent report in the FDA Total Diet Study series (Podrebarac D.S. 1978) which showed the following dietary intakes of mercury for FY 1977 & 78: 1977 6.3 ug/day, and 1978 3.4 ug day. The adult market basket samples used in the study represented the basic 2-week diet of a 16- to 19-year old male and were collected with 4 different geographical areas of the U.S. I would have to assume this group would have low fish consumption.
Now the problem with all of this is that if we have a mercury intake of approximately 10 ug a day from our food, water and atmosphere, that only gives us a total daily mercury intake of 10 ug. Yet scientific data is telling us we excrete about 100 ug/day. So the question is, where is the additional 90 ug/day of mercury we are excreting coming from? Unfortunately, the only other source of mercury I can think of that might contribute to the deficit is those good ole mercury amalgam fillings that keep working away at releasing mercury 24 hours a day. Moreover, I think it important to emphasize that the EPA has stated that the daily absorption of both forms of mercury should not exceed 30 ug Hg/70 kg body weight.

The following information, taken from a report by Lamm and Pratt (1985) should give all devotees of urine mercury measurements a little extra heartburn: "Clear, negative and significant correlation was found between time on the job and the level of mercury in the urine; $R = -0.83$, $p < 0.0005$. In other words, the longer a worker is on the job, the less mercury is excreted into his urine, as measured during the year prior to the neurological examination in our laboratory." 

Similarly, it is not at all surprising that no significant correlations were found between the concentration of mercury in the urine and measures of neural conduction. Since this measure indicates the efficiency with which the kidneys flush out mercury and not necessarily the level of mercury in the body or blood, it can be used neither as a reliable measure of subclinical damage nor as a correlate of such a measure."

Finally, to end this whole "fishy" story I would like to state that the ADA and NIDR have publically admitted that measurements of the mercury in blood and urine do not relate to the toxic effects of mercury. Unless they were coerced into making those statements I have to wonder why Dr. Langan is still testing urine and more importantly, why the ADA continues to spend your money on a project that they have stated cannot inform you if you are being harmed by your continued use of amalgam.

REFERENCES


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ABSTRACTS/REVIEWS


SUMMARY: Mercuric chloride (HgCl₂) induces in Brown-Norway rats (BN) a B cell polyclonal activation resulting in autoimmune disease. Spleen cells from BN rats injected with HgCl₂ were fused with IR983F, a nonsecreting rat myeloma cell line, in order to obtain monoclonal antibodies reacting with autoantigens or IgE-producing hybridomas. After screening for immunoglobulin-producing clones, we found 5% clones with anti-tissue activity, 8% with anti-TNP activity, and 41% secreting IgE. Among the anti-tissue monoclonal antibodies, one recognizes both TNP and mesangial structures of rat normal glomeruli, which could be an as yet
unrecognized mechanism of nephrotoxicity. These experiments 1) confirm that HgCl₂ induces polyclonal activation, 2) show that the mercury model is of interest to obtain monoclonal IgE and various autoantibodies, and 3) suggest a new possible mechanism of antibody-mediated renal injury.


SUMMARY: Biotransformation of methylmercury in rats was studied by enhancing or inhibiting its biotransformation with various procedures. A new sensitive method developed to determine specifically inorganic mercury in the presence of organic mercury was used. Biotransformation was enhanced by treating the rat with phenylhydrazine. The increase of inorganic mercury was highest (four to five times) and rapid in the spleen. Inhibited biotransformation of methyl mercury was observed in splenectomized rats. The inorganic portion of total mercury in the macrophage-rich fraction of spleen cells was clearly higher than that in unfractionated spleen cells. The biotransformation of methyl mercury was inhibited by treating the rat with carrageenan, a well-known substance blocking macrophage function. These results suggest that the spleen is an important site for the formation of inorganic mercury, and that the macrophage participates in this biotransformation.


SUMMARY: This paper discusses metal exposure in the male, the nonpregnant female, and the maternal-offspring unit. In the first two situations, the primary targets are the gonads. In the mother-offspring unit, consideration must be give to effects on the fertilized ovum, the growth of the embryo, and finally, to the fetal and perinatal stages. The central nervous system may be especially vulnerable during development. The placenta also undergoes development, and either the placenta or the fetus may be the primary target. In humans, certain metals may cause abortion or other effects on the conceptus. Effects may also be produced by metal exposure both in utero and in the suckling infant. For example, methylmercury gives rise to a range of effects on the central nervous system at doses lower than those producing damage to the mature nervous system. Effects of lead and arsenic are associated mainly with postnatal exposures during infancy and early childhood, but there is reason to believe from animal experiments that some effects may occur from prenatal exposure to certain metal compounds. In the actual report, the authors make some important statements: "It is of interest that, although there is now substantial evidence from animals that inhaled mercury vapor can produce higher levels of mercury in the fetus than in the mother, very little, if any, work on animals or humans has been carried out to look for prenatal effects of
mercury vapor."....."There is anecdotal and rather brief clinical
information suggesting that, indeed, inorganic mercury can affect
the human infant, but to date no careful study has been carried
out, either on animals or on man, using inorganic mercury at this
stage of the life cycle."....."Surprisingly, mercury vapor and
inorganic mercury, the two forms of mercury to which people are
occupationally exposed, have received very little attention
despite the fact that animal work suggests that effects might be
expected at least in certain times in the life cycle, such as the
suckling stage."

Rajanna B. and Hobson M. Influence of mercury on uptake of
$[^3\text{H}]$dopamine and $[^3\text{H}]$norepinephrine by rat brain synaptosomes.
The authors bring out some very important points in the
discussion portion of this report. Traditionally, in any compari-
son of the toxicity of inorganic versus organic mercury, the
scientific literature, until recently, has usually concluded that
the organomercurials were more toxic. Recently, however, more and
more research is being published that indicates inorganic mercury
may in fact be more toxic than organic. Such is the case in this
report: "The results of this study suggest that mercury compounds
depress the ATP-utilizing and ATP-synthesizing systems by
inhibiting the Na$^+$$, \text{K}^+$$-$and Mg$^{2+}$-oligomycin-sensitive (mitochon-
drial) ATPases respectively in brain synaptosomes."....."Our
results indicate that both inorganic and organic forms of mercury
inhibit the membrane-bound Na$^+$, K$^+$-ATPase and the uptake of $^3$HDA
and $^3$HNE by brain synaptosomes in vitro. Inorganic mercury
inhibits ATPase and the uptake system more severely than organic
mercury. One possibility is that inorganic mercury might have
acted as a poison by destroying the entire protein content rather
than inhibiting a single enzyme system."

Pach J. et al. The hepatotoxicity of mercury vapours in the light
of biochemical, scintigraphic and morphological data. Mater Med.
SUMMARY: In a group of 83 workers with occupational exposure
to mercury vapour (mean Hg in air 0.064 mg/m$^3$) the following
biochemical tests were performed: aspartate aminotransferase
(AspAt), alanine aminotransferase (AlAt), sorbitol dehydrogenase,
alcaline phosphatase (AP), cholinesterase (ChE), prothrombin
index and bilirubin level determination.
Abnormal SDH, AlAt, AspAt, AP and prothrombin values were
significantly more frequent in the exposed group, than in the
control group. Scintigraphy of the liver gave pathological
results in 57 cases. In a group of 17 subjects, exhibiting
abnormalities in biochemistry or scintigraphy, the liver biopsy
was performed. In 15 cases fatty degeneration and inflammatory
reaction were found in liver tissue. No history of alcoholism or
hepatitis was elicited in both groups.

SUMMARY: The removal of mercury (silver/amalgam) dental fillings can provide great benefit to patients suffering from the yeast sensitivity syndrome by providing a greater tolerance to inhalant, ingestant, and endogenous loads. Moreover, therapy with nystatin and other anti-yeast medicaments can be reduced and even, in some cases, eliminated as a result of the removal of dental mercury.


"Mercury binds with blood proteins, but whether this binding affects their electrophoretic mobilities was not known. We injected mercuric chloride (5 mg/kg of body wt) into male albino rats, intraperitoneally, and collected their blood after killing them at various times after injection. We then performed paper electrophoresis of the plasma proteins, using Whatman no. 1 chromatography paper and barbital buffer (75 mmol/L, pH 8.6), and electrophoresis of hemoglobin, using Tris-EDTA-boric acid buffer (pH 8.6). To resolve the amino acids in plasma, we used descending chromatography paper with butanol/acetic acid/water (12/3/5, by vol) mobile phase and ninhydrin for color development."

"The anodic mobility of albumin and hemoglobin from mercury-treated rats increased within the first hour after injection, peaked in the third hour, and returned to normal by the sixth hour. Results for hemoglobin are shown in Figure 1. These changes may be consequences of binding and subsequent release of mercury. We propose that the increased anodic mobility of hemoglobin and albumin, observed as long as 5 h after mercury administration, could serve as a simple and early diagnostic test of mercury poisoning."

"The chromatographic separation of amino acids revealed the absence of the spots for cystine, lysine, and arginine in plasma collected 1, 2, 3, 4, and 5 h after the mercuric chloride administration (data not shown). These spots reappeared in plasma collected after 6 h and persisted thereafter. The absence of these amino acid spots may be due to interference with the ninhydrin reaction by mercury-binding of these amino acids."


ABSTRACT 81. In a foregoing study concerning the cytogenetic effects of mercury after occupationally low-level exposure we
obtained data suggesting that a variety of mercurials have at first an important effect upon aneuploidy but may also induce chromosomal aberrations. Amalgams were also included in that study. These findings and the problem of mercury toxicity in relation to dentistry as discussed in the scientific literature inclined us to look at possible cytogenetic damage in dental personnel.

10 Persons involved in the dental practice and 10 age-related controls were investigated cytogenetically (100 cells/person). A statistically significant increase of aneuploidy (and hypoploidy) was observed in the "dental group" in comparison with the controls. Chromosomal aberrations however were within normal values. The increase in aneuploidy which may be explained by the action of the mercury-amalgam upon the SH-groups of the spindle apparatus thus cannot rule out a possible health hazard to the dental personnel. Other factors however (like X-ray exposure) may be responsible for the observed data. A more thorough investigation seems to be necessary in order to come to more definite conclusions. (BIO-PROBE COMMENT: WHY HASN'T NIEDLE ET AL. FOLLOWED UP ON THIS ONE?)


ABSTRACT: The content of calcium, copper, iron, magnesium and zinc in the plasma, liver and kidney of control female and male, as well as pregnant rats on days 12 and 19 after a i.p. injection of 4 mg/kg of nickel was studied. The content of 19-day conceptuses was also measured. The injection of nickel provoked significant alterations in the essential metal homeostasis, more marked in the case of pregnant rats, with additional differences between male and female animals. In general, nickel provoked increases in metal concentrations in tissues, with diverse changes in plasma. In a number of tissues and metals, the effects lasted up to 48 hours after the single injections, long after the nickel being washed off the animal. The results suggest some sort of long-lasting nickel effect upon metal homeostasis, which is postulated not to be directly related to acute effects and which is enhanced by pregnancy.


SUMMARY: The levels of inorganic and organic mercury in human amniotic fluid were examined in 57 Japanese pregnant women with a gestational age of 4 months to term. Inorganic mercury was detected in all but two samples, while organic mercury was found in only 30 women. The level of inorganic mercury was higher than organic mercury. The highest levels of both inorganic and organic mercury were observed in the seventh month of gestation.
EDITORIAL

THE NEW A.D.A. POLICY - SECURITY OR SUICIDE?

Volume 114, the January 1987 edition of the Journal of the American Dental Association (JADA), contains a synopsis of the 1986 Annual Session of the American Dental Association. The current position of the A.D.A. on the dental amalgam controversy is contained on page 23 of that JADA edition:

"the Association states that, based on a wide consensus of current documented scientific research, the continued use of dental amalgam as a restorative material does not pose a health hazard to the nonallergic patient. It also provides that advocating the removal of clinically serviceable dental amalgam restorations solely to substitute a nonmercury-containing material is unwarranted and violates the `ADA Principles of Ethics and Code of Professional Conduct'. In cases in which state dental boards initiate proceedings on this question, the Association will cooperate by making available scientific personnel as expert witnesses;"

It is indeed puzzling that the sentence immediately preceding this position states: "Societies are also urged to support legislation requiring courts in appropriate cases to instruct juries on the availability of alternative treatments and the role of patients in their own care;". On the one hand, the ADA proposes legislation requiring courts to specifically consider the differences in opinion held by dentists regarding dental treatment; then follows with an official position clearly calling for disciplinary action against dentists who disagree with their position on dental amalgam. On the one hand, the ADA officially acknowledges the role of patients in their own care; then follows with an official position that denies the patient the choice of having mercury-containing fillings replaced if they so desire.

The ADA does not designate the section of the `ADA Principles of Ethics and Code of Professional Conduct' that is violated by advocating the removal of clinically serviceable dental amalgam restorations solely because of their mercury content (or more correctly, their scientifically demonstrable release of mercury vapor). A thorough review of the ADA document fails to reveal any section of that document that can even be remotely considered to be applicable to the subject. However, in view of the 'current documented scientific research', there are two sections of the document that are violated by amalgam advocates or if patients are not informed of the amalgam controversy: Section 2-EDUCATION states "The privilege of dentists to be accorded professional status rests primarily in the knowledge, skill, and experience with which they serve their patients and society. All dentists, therefore, have the obligation of keeping
their knowledge and skill current". Section 4- RESEARCH AND DEVELOPMENT states "Dentists have the obligation of making the results and benefits of their investigative efforts available to all when they are useful in safeguarding or promoting the health of the public". Therefore, it is a matter of factual record that, according to the ADA document itself, a dentist must remain currently informed of the valid scientific documentation and must make this information available to `all'. On the other hand, additional specific wording would have to be added to the document to justifiably prosecute those who supposedly violate the document by advocating the removal of clinically serviceable dental amalgam restorations solely to substitute a nonmercury-containing material. Moreover, the ADA does not define "clinically serviceable". What are the criteria and who determines the condition? Are amalgams that exhibit corrosion, open margins, or surface defects considered clinically serviceable? Is the replacement of an amalgam with a nonmercury-containing gold inlay a violation? If the release of mercury vapor from the ostensibly stable dental amalgam filling is demonstrated is the filling no longer clinically serviceable?

This new ADA position has ominous overtones, indeed. The ADA has quite apparently suggested, if not instructed, that the State Boards of Dentistry should suspend the licenses or otherwise discipline dentists who disagree with the official ADA position regarding the harmlessness of dental mercury-containing amalgam fillings. At first glance, this action may seem to jeopardize anti-mercury dentists and, indeed, the ADA has apparently made a deliberate move to transfer the controversy from the scientific and professional arenas into the legal arena. Up to now, responsible anti-mercury amalgam professionals have attempted to gather valid documentation and expose the pro-amalgam professional establishment to this documentation. However, the new ADA attack will obviously force a change in these tactics. Faced with a loss of livelihood from license suspension or other disciplinary action, anti-mercury amalgam dentists must either compromise their morality or fight back with every available weapon. It is highly unlikely that all of the anti-amalgam dentists will be willing to compromise the health of their patients by meekly submitting to the ADA onslaught, in spite of the economic and personal sacrifices encountered. This means that the new ADA attack will inevitably force the controversy into the courts, which will eventually have to settle the issue based on scientific evidence, not unsubstantiated personal opinions. In spite of the burden placed on anti-amalgam dentists, the ADA has now placed itself, the State Boards of Dentistry, and the entire pro-amalgam dental establishment in a position of the most extreme jeopardy for the following reasons:

I. DOCUMENTED SCIENTIFIC RESEARCH:

The ADA position statement specifies that it is based on "a
wide consensus of current documented research". Once in the courts, the content of the pro-amalgam 'research' will be revealed. The total lack of scientific validity of unreferenced personal opinion papers and articles in such periodicals as Consumer Reports will finally be revealed, as will those papers whose conclusions were derived from measurements of mercury in the blood or urine. Even the American Dental Association and the National Institute of Dental Research have publically admitted that the toxic effects of mercury do not correlate to measurements of mercury in the blood, urine, or hair. Any opinion papers based on these 'studies' will be found to be scientifically, and legally, invalid, as will opinions based on claiming that the mercury vapor exposures are harmless by relating these exposures to occupational TLV's.

The ominous implications to the ADA and other public advocates of dental amalgam is the issue of 'negligent misrepresentation'. The ADA and its constituents, the State Dental Boards, the governmental dental agencies, and the dental schools influence the direction and the actions of the entire profession, and therefore possess an undeniable major impact on the health of the public. If a dental procedure is even potentially harmful to the patient, the officials and spokesmen of these groups have an obligation to investigate and determine the SCIENTIFICALLY DEFENSIBLE validity of their position. If their public position is based on invalid personal opinions they are in jeopardy, because the actions and policies of the profession and the resultant effects on the public health are based on their statements. The responsibility of the positions held by these individuals dictates that they devote the time and effort to reading and learning the scientific basis of the controversy, not simply the personal opinions of some of their peers. The ominous import of this point may be established by examining the 'current documented scientific research' mentioned by the ADA. Since unreferenced personal opinions do not qualify as valid research, we may only address valid research studies. The following information has now been irrefutably established scientifically:

1. Mercury is not locked into the set amalgam filling. Mercury escapes from dental amalgam fillings throughout their lifetime in the forms of vapor, ions, and abraded particles. The release of mercury vapor from dental amalgam fillings is stimulated by exposure to chewing, brushing, and hot fluids.

2. The absorption rate of inhaled mercury vapor is extremely high, averaging approximately 80% of the inhaled dose.

3. The half-time for the elimination of a single dose of mercury is extremely long, certainly at least 30 days for the whole body and perhaps as long as 10,000 days for the brain. Multiple small daily doses will therefore result in body accumulation.
4. Autopsy studies have confirmed that the mercury released from dental amalgam fillings does enter and accumulate in the patient.

5. The extreme toxicity of mercury is well established. Current research is clearly demonstrating that inorganic mercury is just as toxic as organic mercury under various physiologic conditions.

6. The toxic threshold for mercury vapor has never been found. Even the U.S. Environmental Protection Agency has so stated. The existing occupational standards are all specifically declared to be estimates based only on the appearance of clinically observable signs and symptoms. Statements that the amount of mercury exposure encountered by patients from dental amalgam fillings is too small to be harmful are contradicted by the scientific literature and are totally indefensible.

7. Controlled, broad-scale studies investigating the effects on the health of patients of mercury released from dental amalgam fillings have never been conducted. The true nature and extent of effects are therefore unknown. Claims that the effects are non-existant or harmless are totally indefensible. Claims that the effects are harmful are based on an accumulation of clinical case reports.

This synopsis of scientific documentation has been thoroughly referenced in previous Bio-Probe issues and can be fully substantiated. It is painfully obvious that, based on scientific validity alone, the ADA has now placed itself and other amalgam advocates in a hazardous position. The major problem is that those individuals who should have been carefully reading and digesting the scientific documentation have not done so. What happens now? Let's assume that the State Dental Boards follow the 'suggestion' of the ADA that anti-amalgam dentists be disciplined. Can these State Dental Boards proceed merely on the word of the ADA, or will they be held liable for their actions based on the valid scientific documentation? What will be the position of liability of the 'expert witnesses' provided by the ADA to the State Dental Boards if their testimony is not scientifically thorough and valid?

Looking beyond the strictly scientific issues of the dental amalgam controversy, the new ADA position raises several other frightening possibilities:

II. ANTI-TRUST:

The American Dental Association is basically a trade union (presented to read 'professional organization'). The organization is funded by dues from member dentists (not all U.S. dentists are members). Employee salaries are paid from these dues. All officers are elected solely by the membership. Technically, the American Dental Association represents its
membership and only its membership. Neither the consumer nor non-member dentists have representation in the organization.

On the other hand, the State Dental Boards are legally established by state law to determine who shall be allowed to pursue the practice of dentistry in that state. Should the State Dental Boards act based on the direction of the ADA, would this be in violation of anti-trust laws?

III. RESTRAINT OF TRADE:

Should the State Dental Boards take action based on the direction of the ADA, would this endeavor be considered collusive restraint of trade? Would then the Federal Trade Commission be obliged to take action against the ADA and State Dental Boards?

IV. RESTRICTION OF FREEDOM OF CHOICE:

The freedom of choice is a basic and unalienable right of citizens of the United States. If the ADA and the State Dental Boards prevent dentists from removing mercury-containing dental fillings from patients who so desire, the rights of these patients have been denied. This issue is very ominous, indeed!

In view of these delineated issues, it is obvious that the new, publically stated position of the American Dental Association is very dangerous and ill-advised, to say the least. It is sad and unfortunate that the ADA has chosen to take this course of action. In dental therapy, intra-professional differences of opinion exist in virtually every area. There is hardly an area of dental practice that enjoys unanimous acceptance. Why has the ADA decided to eliminate the voices raised against the use of mercury (an acknowledged serious poison) in dental fillings? One can only speculate over the motivation for such a drastic and potentially dangerous act. Certainly fair, judicious, and prudent minds reside in the hallowed halls of the American Dental Association! It is time for these minds to intervene and reverse this explosive act, before it is too late! The fate of the American Dental Association and the good name and future of dentistry hangs in the balance. Rapid action is imperative!
FORUM

International Academy of Oral Medicine and Toxicology calendar of 1987 meetings:

   Embassy Suite Hotel, 2630 E. Camelback Rd. Phoenix, AZ 85016, (602) 955-3992. $110/night, includes Continental Breakfast. Limousine available to and from airport. Specify IAOMT member when making reservations.
   Sat., March 7: 8:30 A.M.-12:30 P.M.
   Speakers: Dr. Murray Vimy and Dr. Michael Ziff
   2:00 P.M. - 5:00 P.M. - Panel Forum
   ($50.00 fee for Non-IAOMT members for this program)
   Sun., March 8: 9:00 A.M. - 1:00 P.M. - Board Meeting.
   (Non-members are welcome to attend)

    King Edward Hotel, 37 King Street, East, Toronto, Ontario Canada M5C 1E9, (416) 863-9700. $135 (Canadian)/night. Details and program to be announced.

III. October 16-18, Friday - Sunday: San Diego, California.
    Chairman: David C. Kennedy, D.D.S., (619) 231-1624. This will be the Annual Meeting and the program will be announced when arranged.

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The first World Congress on Oral Prevention will be held in Paris France, July 6-8th, 1987. A registration form has been included. However, the dates on the enclosure are incorrect. The correct dates are July 6-8, 1987.

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American Academy of Biological Dentistry is hosting a 4 day seminar, May 6-9, 1987 on Dental Ecology, Dental Electro Acupuncture and Oral Facial Focal Disturbances, Diagnosis and Therapy. The presenters are Dr. Fritz Kramer and Dr. Ralf Turk.

Course will be held at Carmel Mission Inn, Highway One & Rio Road, P.O. Box 221550, Carmel, CA 93922, (408) 624-1841. $56 single occupancy and $61 double occupancy.

Full Seminar fees are payable in advance. Write American Academy
of Biological Dentistry, P.O. Box 856, Carmel Valley, CA 93924 for a program and registration information.

A beginners pre-seminar ond day course will be taught by Ed Arana, D.D.S. and Gary Verigin, D.D.S., the Co-Founders of the American Academy of Biological Dentistry. The course will be held on May 5, 1987, 10:00 A.M. until 6:00 P.M. Tuition is $80.00. Write for registration information to the Academy address given above.

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The International Association of E.A.V., Hawaii Chapter, Presents Dr. Joachim Thomsen, D.D.S. March 2-6, 1987 in Phoenix Arizona. "E.A.V. - An Alternative Dental/Medical Program. For registration information write: International Association of EAV, 1441 Kapiolani Blvd., Suite 721, Honolulu, Hawaii 96814. There is an unconfirmed report as we go to press that the seminar may be switched to Las Vegas. Please call Dr. Sam Wong at (808) 948-2876

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* As this is in effect another "Rebuttal Issue", extra copies * * are available for those who desire to provide them to their * * colleagues, patients, media, or put them in your reception * * room. Prices including postage are: single copies $2.00 each; * * 2-10 copies $1.75 each; 11-25 copies $1.50 each and 26 or * * copies $1.25 each.

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