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CERAMIC RESINS AND TOOTH SENSITIVITY
PART II
by Murray J. Vimy, D.M.D., F.A.G.D.

INTRODUCTION:

In the previous newsletter we addressed some of the possible etiological factors of pulpal pathosis following posterior resin placement. In this report we would like to review one specific approach to this problem. This approach and the research substantiating it have been developed by Dr. Martin Brannstrom, associate professor of Oral Pathology and Docent in Experimental Histopathology at Karolinska Institute, School of Dentistry, Sweden.

Dr. Brannstrom is one of the major proponents of the "Theory of Hydrodynamics". According to this theory the pulp has a gradient of fluid pressure directed from the pulp outwards through the dentin and the enamel. This gradient is due to the higher fluid pressure in the pulp. According to Johnson et al. 1973, an open odontoblastic tubule if left unprotected would theoretically empty itself 10 times a day.

Other research has shown that fluid flow from the pulp may be outward or inward, depending upon environmental factors. Dr. Ralph Steinman, now professor emeritus at Loma Linda School of Dentistry, demonstrated that pulpal fluid under normal physiological conditions flows outwards from the pulp, through the dentin, and through micro-pores in the enamel. His studies, which have not received the attention they are due, utilized a dye tracer injected into rats. Various nutritional factors were altered and the teeth were sectioned and the dye patterns observed. According to the research there is a hypothalamic/paratoid endocrine axis. This hormonal axis triggered by the nutritional factors causes the parotid gland to release a hormone which reverses the natural direction of pulpal fluid flow. Interestingly, one of these nutritional factors is sugar.

Nature has given the tooth a natural protective mechanism.

The outward fluid flow is especially evident in tooth areas where secondary dentin has not been formed. According to Brannstrom's research, sensitivity is created when mechanical stimulation removes fluid from the tubule ends which are exposed.

This removal increases capillary forces and the once slow fluid movement increases. Further, this increased fluid flow activates mechanoreceptors at the end of the dentinal tubules and results in pain. Sensitive dentin indicates tubules that are open to the pulp and which are exposed to mechanical irritation. If the exposed dentine is kept wet, the flow decreases and the pain dissipates.

The autocure ceramic resins set slowly. Consequently, droplets of pulpal fluid can collect under the restoration. This is especially true if the dentin has been exposed to acid or strong chelating agents which remove the smear layer and open up the ends of the odontoblastic tubules.
Further, most of these types of resins contract during setting and create a gap (5-20 micrometers) between the tooth and the material. If saliva or other fluids are present they get pulled into the gap by capillary forces. Once the gap becomes filled with pulpal fluid, the slow outward movement continues.

With time, other factors slow the fluid flow. These are:

1) accumulation of solid materials,

2) delayed expansion of the filling and

3) a calcified pellicle which may form.

This outward fluid flow does not, in Brannstrom’s opinion, inhibit chemical gradients which may be directed inward. Thus, sugar molecules on the outside could diffuse through the gap and reach bacteria under the restoration.

He makes the following points:

"Leakage, that is, the diffusion of particles inward, does not, except for sugar and possibly acid, seem to have much clinical relevance.....I have not seen any experimental evidence that bacterial toxin.....may exist at the tooth surface and at the margin in concentrations great enough to reach the pulp....Nor can I imagine how variations of intraoral temperature, which are usually of short duration and within the normal range, can have a significant influence on the outward flow of fluid around the filling."

Brannstrom’s opinion based on his research and review of the literature is that the most significant factor causing pulpal irritation is INFECTION IN THE GAP. Even a few bacteria may multiply in the gap and reach thousands within 10 days. Consequently toxins are concentrated in the gap and irritate the pulp. These bacteria can enter dentinal tubules.

Further, they may multiply from the smear layer even if the communication with the oral cavity is sealed.

He concludes very emphatically:

"The results (of all the research) have pointed to the same conclusion, namely: when infection is avoided there is no appreciable irritation to the pulp; pulpal damage is caused by infection and not by the filling material or the pretreatment procedures."

This conclusion is a dramatic departure from the notions of most practicing dentists. However his research evidence is compelling.

Other conclusions of Brannstrom’s research:

1) acid etching even near the pulp does not irritate the pulp.
2) irrespective of the materials used there is no pulpal inflammation as long as there is no infection under the fillings. This is true even for dental amalgam. 

3) dentine is a good insulator. Therefore, there is no need to place insulating cements under amalgam. 

4) Zinc oxide and eugenol is the only material that appears to cause pulpal inflammation.

TECHNIQUE RECOMMENDATIONS ACCORDING TO BRANNSTROM

To avoid infections under the filling:

1) the cavity must be clean

2) a lining must be placed on all walls to prevent bacterial invasion.

3) a lining must be attached to all dentin so that the bacteria can not get a place to multiply especially in the open odontoblastic tubules.

To eliminate sensitivity:

1) where possible caries should be removed without anesthetic. Sensitivity when excavating indicates tubules open to the pulp.

2) when sensitive teeth are encountered while removing the caries the teeth should be medicated with a temporary dressing to stimulate secondary dentin formation and this reduces sensitivity.

3) a temporary dressing containing calcium hydroxide is excellent in reducing tubule infections.

However, the use of thick bases is discouraged because:

1) They don’t always adhere,

2) They can not be placed on all walls,

3) They, are difficult to use on small shallow cavities,

4) They can impair the retention of the final restoration and

5) If there is leakage they can dissolve leaving a significant gap.

PROCEDURES:

Before the tooth is lined all the superficial smear layer must be removed. The superficial smear layer is everything except the plugs in the dentinal tubules. Further, an antiseptic is needed to remove all the microbes present. Strong solutions such as acid or 15% EDTA are not good because they remove the tubular plug and open up the tubules.
To facilitate this procedure a solution was developed which only removes the superficial smear layer. (a surface active component, 0.2% EDTA, and benzalkonium chloride).

To desiccate (dry) the tooth it is recommended that the air be blown at right angles to the direction of the dentinal tubules. This should be done for 10–15 seconds continuously.

Brannstrom recommends a primer of shellac resin mixed in alcohol with some benzalkonium chloride to act as an antibacterial. This is blown dry to a thin film. On this film is applied a polyesterene lining (Tubulitec: Dental Therapeutics AB, Nacka Sweden). This acts to seal the open tubules and unlike Copolite is compatible with resins.

The cavity design should follow those previously recommended (Bio-Probe 1(3):9, 1984).

As we can see the results of this discussion conflict considerably with the recommendations given in the previous article. In the final analysis ceramic resin restorations are difficult to do. They demand great care, time and attention. From a productivity stand point one would be well advised to use castings of acceptable gold alloys where possible both because of biocompatibility and time considerations.

It is no wonder many endodontists are finding more terminal pulps under ceramic resins. Although a biologically safer material than amalgam, the demands of the technique necessitate our constant consideration.

REFERENCES

2. Abstracts of Steinman: Compiled by the International Academy of Microendocrinology.
CORROSION OF DENTAL AMALGAM AS CAUSE OF MERCURY POISONING

by Jaro Pleva Ph.D.*

INTRODUCTION

The external environmental sources of potential poisoning hazard are subject to considerable attention. However, the possible danger from the internal poisoning source in the body, dental amalgam, is mostly overlooked.

Amalgam is still by far the most extensively used material for dental restorations, estimated to be 80% of all tooth restorations(2). The common type of amalgam is an alloy containing typically in wt % : 50 Hg, 35 ag, 10 Sn, Cu, Zn. Reported types of amalgam degradation are crevice corrosion (1,2,3,4), selective corrosion (5), galvanic corrosion in contact with dissimilar alloys (6,7) and mechanical wear (8). Besides selective corrosion, stress corrosion was proposed to be responsible for the marginal breakdown of an amalgam restoration (9). Further, cyclic loading strongly promoted corrosion of the amalgam surface (10).

Mercury released by corrosion can principally be taken up by the body in three different ways: as mercury salts or complexes in gastrointestinal tract and through mucous membranes (11), as mercury vapour in lungs (8), and finally uptake was reported on the bottom of fillings through dental tissue and nerve ends. Mercury contents up to 1200 ppm in teeth roots have been found (12).

Amalgam bearers have increased mercury contents in many organs; Hg accumulates especially in the pituitary gland, kidneys, liver and brain (13).
In spite of this knowledge, amalgam is still postulated to be a "stable" alloy, which cannot give poisoning. No investigation about possible long term effects of amalgam has ever been made. With a questionnaire, the author easily gathered over 200 cases with both certain and assumed serious mercury poisoning. Even if individuals with only amalgam restorations may be severely ill, the most flagrant cases are these with permanent or intermitent galvanic cells gold - amalgam in the oral cavity (14). Until the day of writing this, 20 of these patients had removed all amalgam fillings. All but one reported total or partial recovery in a few months.

CASE STORY AND SYMPTOMS

A documented case history typical for the above mentioned group, has recently been reported about the syndrome of chronic mercury poisoning from corroding dental amalgam (15). It has been shown on the same person, that mercury released from corroding amalgam caused serious poisoning. An amalgam filling in a gold bridge restoration that caused increasing symptoms of both psychical, emotional and physical are described below. No dysfunction could be found by common diagnostic methods on heart muscle (EKG) or on gastrointestinal tract (X-ray) or elsewhere.

Being a corrosion engineer, the patient pointed at the black and rough amalgam filling in the galvanic cell gold - amalgam, but no physician took the idea of poisoning from corroding amalgam seriously. First a look into a toxicology handbook identified the symptoms as chronic mercury poisoning. Removal of amalgam in the galvanic cell broke the trend and the strongest symptoms went partly back, but four years later many troublesome symptoms were still present, some of them increasing in severity.

First, after all amalgam fillings had been removed, all symptoms successively disappeared. All the recorded symptoms are known as typical for chronic mercury poisoning: irregular heart beat, pains in chest and heart region, headaches, vision and hearing disturbances, vertigo, chronic cold, loss of memory, fatigue, physical disturbances, anxiety, stress, joint and back pains and stiffness, asthma, burning mouth, increased salivation, metallic taste, eczema, paralysis. The change in subjective feeling and perception capability after amalgam removal has been described as coming from a period of mere sick physical existence to real living again (15,19).

INVESTIGATION

Parts of removed amalgam fillings have been spared and examined with respect to corrosion attack in Scanning Electron Microscope (SEM), equipped with Energy Dispersive X-ray Analysis device (EDAX).

1. Morphology of attack
The fillings showed blackening of the crevice surfaces, adjacent to the tooth cavity. The crevice surfaces were corroded, at some locations cracks have been observed, Fig. 1,2. Some external surfaces of the fillings were attacked by a type of selective corrosion, Fig.3.
possibly at the gamma-2 phase Sn$_{7.8}$Hg, reported to be the least corrosion resistant phase in the complicated metallographic system of silver amalgam (5,16).

2. EDAX - analysis
The microanalysis of the black crevice surfaces in Fig. 4 showed a definite decrease in the content of both mercury and silver from about 40% to very low, in some cases zero contents. On the other hand, tin concentrations increased up to 80%. According to published investigations (1,11,16) corrosion products of silver amalgam left on the surface are a mixture of tin oxide and tin oxichloride. In all attacked surface areas of the fillings the mercury content was lower compared to the bulk content.
(NOTE: Figures 1-4, have been omitted)

DISCUSSION

The mechanical performance of amalgams such as initial high strength in combination with comfortable insertion properties seems to be deciding for continuing use of amalgams. No proof of their compatibility with the human body in long term applications in the oral cavity has ever been presented.

In some years, the classical silver-tin amalgam filling often fails due to crevice or selective corrosion of gamma-2 phase (1,2,5). The new high copper amalgams (s.k. non gamma-2 type) seem to be less prone to these types of attack. However, the metallographic system is very unstable and releases free mercury, which has been identified on the surface of "Dispersalloy" amalgam in the form of droplets (17). Undoubtedly a part of the volatile mercury droplets will evaporate and become absorbed in lungs.

Also the silver based amalgam gives off mercury, as shown by Stock (18). In two weeks as much as 15 mg Hg has been released from 0.8 grams of hardened amalgam at maximum temperatures of 35°C, in the absence of mechanical wear and a corrosive environment.

There is no doubt that mercury from amalgam fillings represents an undesirable heavy metal contribution to the body, in addition to other possible environmental hazards. For example, the acidification of the environment may increase heavy metals contents in food and waters, but also decrease contents of essential trace elements such as selenium, in soils. Selenium has been shown to be an essential element, effective in preventing the s.c. "metal syndrome" disease (20).

CONCLUSION
It is worthy to draw attention to the fact that amalgams are extensively used in spite of the knowledge that they corrode and that no clear evidence of their long term biocompatibility has ever been presented. Acceptance of amalgam has been the cause of slow development rate of alternative dental materials.

In dental praxis, first of all galvanic contact between gold and amalgam should be avoided, even in remote position, where intermittent contact is possible (6).
Careless continuing use of amalgams should be considered very seriously, as mercury is reported to be a factor in diseases such as metal syndrome (20), multiple sclerosis, genetic alterations, rheumatism, skin diseases, mental diseases and cancer (21).

Acknowledgement:
The author is grateful for the possibility to use instrumentation at Uddeholm Research AB.

REFERENCES

REVIEW/ABSTRACTS

The effects of methylmercury (MeHg) on cytoplasmic microtubules in cultured neuroblastoma cells, glioma cells, and fibroblasts were compared. Neuroblastoma cells appeared to be more sensitive to disruption of microtubules by MeHg than the glioma cells or fibroblasts; cellular concentrations of mercury after MeHg were also higher in neuroblastoma cells. Recovery of microtubule structure was monitored in cells after removal of MeHg; addition of the chelating dimercaptosuccinic acid (DMSA) increased reassembly of microtubules. During MeHg treatment and early recovery, microtubule integrity was dependent on cellular mercury concentrations. However, after prolonged DMSA exposure, mercury appeared to reenter the cell, without causing dissociation of microtubules. Sager P.R., Syversen T.L. Differential responses to methylmercury exposure and recovery in neuroblastoma and glioma cells and fibroblasts. Exp Neurol, 85(2):371-382, 1984.

An IgG anti-D prozone is produced by progressive inactivation of the D antigen following red cell exposure to increasing concentrations of thimerosal and phenol present as antibody excess is achieved. Partial inactivation of the D antigen by routinely added thimerosal, an organic mercurial, and phenol is associated with an unstable D antigen-antibody complex resulting in an increased rate of dissociation of anti-D and with a decreased reactivity of the cell-bound anti-D in the antiglobulin reaction. Complete D inactivation occurs with concentrations in excess of 0.43 microM thimerosal. If attributable to inactivation of a surface-exposed sulfhydryl group, it suggests that less than 5 percent of these are involved in D-antigen activity. The data do not exclude the possibility that D inactivation may result from alteration of sulfhydryl groups other than those exposed at the surface. Jameson J.T. et al. Anti-D prozone and membrane sulfhydryl modification. Transfusion, 24 (2): 130-135, 1984.

The effect of zinc on mercuric chloride-induced lipid peroxidation in the rat kidney was investigated. The rats received zinc acetate (2.0 mmol/kg, po) for 2 days before being given mercuric chloride (15 mmol/kg, sc) and were killed 6, 12, and 24 hr after the last injection. Lipid peroxidation occurred in the rat kidney 12 hr after mercury administration, and this mercury-induced lipid peroxidation was significantly reduced by zinc pretreatment. A decrease in vitamin C and E contents in the kidney was observed 12 hr after the
administration of mercury, and this decrease was prevented by zinc pretreatment. In the kidney of rats pretreated with zinc, the activities of the protective enzymes, glutathione peroxidase and glucose-6-phosphate dehydrogenase, were increased after mercury injection. Non-protein sulphydryl content (mostly glutathione) also rose markedly. The results indicate that zinc not only induces metallothionein, but also increases protective enzyme activities and glutathione content, which would tend to inhibit lipid peroxidation and suppress mercury toxicity. Fukino H. et al. Effect of Zinc pretreatment on mercuric chloride-induced lipid peroxidation in the rat kidney. Toxicol Apply Pharmacol, 73(3):395-401, 1984.

Selenium deficiency and vitamin E deficiency deficiency both affect xenobiotic metabolism and toxicity. In addition, selenium deficiency causes changes in the activity of some glutathione-requiring enzymes. We have studied glutathione metabolism in isolated hepatocytes from selenium-deficient, vitamin E-deficient, and control rats. Cell viability, as measured by trypan blue exclusion, was comparable for all groups during the 5-h incubation. Freshly isolated hepatocytes had the same glutathione concentration regardless of diet group. During the incubation, however, the glutathione concentration in selenium-deficient hepatocytes rose to 1.4 times that in control hepatocytes. The selenium-deficient cells also released twice as much glutathione into the incubation medium as did the control cells. Total glutathione (intracellular plus extracellular) in the incubation flask increased from 47.7 +/- 8.9 to 152 +/- 16.5 nmol/10(6) selenium-deficient cells over 9h compared with an increase from 46.7 +/- 7.1 to 92 +/- 17.4 nmol/10(6) control cells and from 47.7 +/- 11.7 to 79.5 +/- 24.9 nmol/10(6) vitamin E-deficient cells. This overall increase in glutathione concentration suggested that glutathione synthesis was accelerated by selenium deficiency. The activity of gamma-gluamylycysteine synthetase was twice as great in selenium-deficient liver supernatant (105,000 X g) as in vitamin E-deficient or control liver supernatant (105,000 X g). Hemoglobin-free perfused livers were used to determine the form of glutathione released and its route. Selenium-deficient livers released 4 times as much GSH into the caval perfusate as did control livers. Plasma glutathione concentration in selenium-deficient rats was found to be 2-fold that in control rats, suggesting that increased GSH biosynthesis and release is an in vivo phenomenon associated with selenium deficiency. Hill K.E., Burk R.F. Effect of selenium deficiency and vitamin E deficiency on glutathione metabolism in isolated rat hepatocytes. J Biol Chem, 257(18): 10668-10672, 1982.

The placental transport of mercury in pregnant mice and its localization in the embryo and fetus from early organogenesis through the whole fetal period was studied by whole-body autoradiography and gamma counting. Metallic mercury (203Hg0) (after inhalation) was compared to inorganic 203HgCl2 (after i.v. injection). Hg0 appears to be oxidized to Hg2+ in the fetal tissues and Hg0 inhalation results in about 4-fold higher fetal mercury concentration than Hg2+ injection
(9.9 versus 2.4% gram dose per gram tissue). Preadmission to the dams with ethanol or aminotriazole resulted in higher fetal concentrations (especially in the liver) of mercury after inhalation of Hg0 but not after injection of Hg2+. A high placental concentration and accumulation in the corpora lutea of mercury after Hg0 inhalation should also be noted. Khayat A. and Dencker L. Fetal uptake and distribution of metallic mercury vapor in the mouse: influence of ethanol and aminotriazole. Int J Biol Res Pregnancy, 3 (1): 38-46, 1982.

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The molecular mechanism of methylmercury and mercuric chloride inhibition of brain muscarinic acetylcholine receptor is investigated. Both mercuric cations strongly inhibit L[3H]quinuclidinyl benzilate (L[3H]QNB) binding to rat brain lysed synaptosomes. Mercuric chloride is 350 times more potent as an inhibitor of L[3H]QNB binding than is methylmercury. Inhibition of the agonist binding site by methylmercury is demonstrated by the competitive action of carbamylcholine chloride on L[3H]QNB binding. D-Penicillamine is found to chelate mercuric cations from the receptor binding site and regenerate the L[3H]QNB binding in a concentration-dependent manner. The tightness of mercuric chloride interaction with the receptor binding site is demonstrated by measuring L[3H]QNB before and after extensive washing. The correlation between mercuric chloride inhibition and D-penicillamine regeneration of L[3H]QNB binding emphasizes the involvement of sulfhydryl groups in muscarinic receptor binding site. These essential sulfhydryl groups may have a significant role in the proper functional configuration of the receptor binding site. Blocking these essential sulfhydryl groups is suggested to be the molecular mechanism of inhibition of brain muscarinic receptors by these mercurials.

The above summary of this paper does not bring out information contained in the discussion portion of the report that I consider to be extremely significant: "These results provide evidence supporting the suggestion by Clarkson (24) that inorganic mercury could be the mediator of the neurotoxic effects of organomercurials. Earlier studies (25,26) have shown that inorganic mercury detected in rat brain was about 4-7% of total methylmercury in the brain tissue after a single injection (i.p.) of methylmercury. Accordingly, if 1-5% of methylmercury is being biotransformed into inorganic mercury, it may cause 1-5 times more inhibition of muscarinic receptors than that caused by the total methylmercury concentration in the brain. It is important to emphasize that methylmercury can penetrate the blood-brain barrier more readily than can mercuric chloride. Demethylation of methylmercury may occur slowly in the brain, enhancing the inhibitory potential of methylmercury in the brain. This may be the reason that the neurological symptoms of methylmercury intoxication always appear after a latent period. Furthermore, similar pathological lesion in the central and peripheral nervous systems have been observed after injection with either mercuric chloride or methylmercury (27)."  

"Deacetylation of N-acetyl-DL-penicillamine leads to the release of the toxic DL-penicillamine, which may eliminate the clinical use of this drug in cases of mercury poisoning. On
the other hand, D-penicillamine is nontoxic (11) and stable in situ (10) and, because of its solubility in water, it can cross the blood-brain barrier (16)......The clinical use of D-penicillamine as a drug in mercury intoxication is quite feasible for short-term treatment, if the kidney is in a functioning condition."

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The antagonistic action of sodium selenite against dental amalgam-induced cytolytic in the L cells was studied using the Cr-release assay and photomicroscopic observations. Three pieces of dental amalgams (2.12 +/- 0.04 cm²) were immersed in 1 ml of culture medium composed of Eagle's minimum essential medium, 10% (v/v) calf serum, and 10 mM Hepes buffer (pH 7.4), at 37°C for 96 hr. 51Cr-labeled L cells were incubated in culture medium containing 39.4% (v/v) amalgam-dissolved solution (ADS) obtained abovye, at 37°C for 24 hr. Compared to the release of 29.1 +/- 0.7% of 51Cr in the control, ADS-treated cells released 73.1 +/- 1.5% of 51Cr which corresponded to the lysis of all cells. Morphologically, the ADS-treated cells underwent lytic changes. A simultaneous administration of 25, 50, and 100 mcg sodium selenite produce a marked decrease in the 51Cr release of the ADS-treated cells. Sodium selenite (50 mcg) suppressed the elevated Cr release caused by ADS and prevented all of the morphological changes induced by ADS. Kumei Y., and Sato A. Toxicol Appl Pharmacol. 59:257-261, 1981.

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EDITORIAL

The information presented by Dr. Plevo and the abstracts contained in this issue of Bio-Probe reinforce the insidious nature of the problems related to clearly defining the clinical or subclinical manifestations of mercury toxicity related to dental amalgam.

Khyat and Dencker's article has a direct relationship to dental amalgam because the findings implicate elemental mercury vapor. Further, I believe it has a positive correlation to the work of Kuntz et al and Abramson et al, who also suggested a relationship with their findings and dental amalgam.

Although not addressed in the study, the biotransformation of MeHg to HgCl brought out in the paper by Abd-Elfattah and Shamoo should apply equally to the brain of a fetus. This possibility, coupled with the potential of oral and gut bacteria to biotransform elemental mercury vapor to methylmercury, presents an added dimension to the potential damage that could be caused by dental amalgam fillings in a pregnant woman.
The paper of Jameson et al presents some intriguing and thought provoking immunological considerations with regard to mercury. Dorland's Medical Dictionary defines prozone as: "the phenomenon exhibited by some sera, which can give effective agglutination reactions when diluted several hundred- or thousand fold but do not visibly react with the antigen particles when undiluted or only slightly diluted. The phenomenon is not due simply to antibody excess, but often involves a special class of antibodies, blocking or incomplete antibodies, which react with the corresponding particulate antigen in an anomalous manner. The bound antibody not only fails to elicit agglutination but actively inhibits it."

Stites et al. in Basic & Clinical Immunology talk to IgG Anti-D: Hemolytic disease of the newborn results from mother's antibodies crossing the placenta and destroying fetal red cells. This leads to hemolytic anemia and hyperbilirubinemia in the newborn infant. Formation of Rh antibodies is the most common form of alloimmunization to give rise to clinically important disease. Over 90% of Rh-negative women having Rh-positive offspring do not form anti-D-antibodies. The mechanism of inhibition of antibody synthesis is unclear, but rapid destruction and clearance of Rh-positive cells from the circulation seem to play a role. In experimental conditions, Rh-positive cells coated with blood group antibodies other than anti-D are quickly destroyed and anti-D-antibodies are not formed.

If I comprehend correctly what Jameson and his associates are saying then it would appear that a possible relationship could exist between the mercury being released from dental amalgam during function and the sulfhydryl modification the authors are talking in hemolytic disease of the newborn. It is also within rational logic to relate it to the work of Kuntz et al., Abraham et al., Svare et al., Eggleston, Khyat & Dencker.

A disturbing aspect of Sager and Syversen’s article was their finding that after prolonged use of DMSA, mercury once again entered the cells. This poses some very interesting questions regarding detox protocols and begs for additional research.

It becomes more apparent, with each research article published on the toxicity of all forms of mercury, that the current position of the American Dental Association that the small amount of elemental mercury vapor released from dental amalgam fillings during function does not constitute a health hazard is totally a subjective judgement without any basis in fact.

CASE HISTORIES

The following letter was received from Ms Louise Reddell of San Diego, CA and she has given me permission to print it in Bio-Probe.

"Your book, "The Toxic Time Bomb", has helped me to make the decision to have the amalgam removed from my mouth."
I am a vibrantly healthy recovered cancer patient, thanks to the alternative methods of Dr. Virginia Livingston. I have had a lifelong thyroid deficiency. I also have hypoglycemia. I am 63 years old.

In October, 1983 I was in intensive care for two days due to "allergic reaction to thyroid" -- my heart was fibrillating (following an hour's lesson windsurfing) and was placed at that time on synthroid. Then I read Broda Barnes' book Hypothyroidism and subsequently found my basal metabolism to be a constant 2 degrees below normal. When I tried very gradually to increase my thyroid intake my heart beat became irregular.

So there had to be something else.

In February of this year I read an article in Let's Live Magazine written by a dentist who listed a variety of symptoms caused by MERCURY. One symptom was conjunctivitis, which caught my attention as I have been trying unsuccessfully for the past year to find the cause of my one red eye.

I read your book, along with other material, and found that a low body temperature is one symptom of mercury poisoning.

I read that mercury is greatly attracted to the thyroid gland.

I am now in the process of replacing a lifetime collection of various metals in my mouth. With the amalgam entirely gone now for five weeks, my basal metabolism is only a half degree below normal. My red eye is now history. My need for frequent naps is gone, as are the occasional attacks of narcolepsy. Though my research indicated that the damage done to the thyroid gland was irreversible, I am led to believe by the improvement in my basal metabolism that in my case it is reversible. However, I have been on the Livingston regimen of megavitamin therapy since 1981 and am saturated with the highest quality nutrition known to man. I'm sure that my recuperative powers are in top form and can accomplish more than those of people on the SAD (Standard American Diet).

To fill in a little more detail on myself, I have a long list of allergies. Though my garden all around my house is full of flowers, this year I may have sneezed four times. No more itching eyes and running nose. I expect to know in the next few weeks if my chronically low white blood count has returned to normal. My energy has doubled, and I am on an emotional high plane. My mind is working like a fine new watch, and I have lost ten pounds.

How much of my good fortune I owe to the removal of the mercury from my system, I don't know. But the sequence of events leads me to believe that there is a direct cause and effect.

I want to thank you for your fine book. And thank you, again, for your help in returning me to a state of health which I have always yearned for. (I don't recall ever being all in one piece like this before!)
FORUM

On February 9, 1985 Dr. Michael Ziff sent the following letter request to Senator Lawton Chiles:

I am vitally interested in obtaining the following information:

1. The amount of funds expended on dental research by the National Institute of Dental Research (NIDR) and the Dental Division of the National Bureau of Standards.
2. The total amount of funds expended on dental research investigating the biocompatibility (potential health hazards) of dental silver amalgam fillings of patients by these two government agencies.

On June 12, 1985 Senator Chiles forwarded the following letter Dated May 28, 1985 which had been received from the Department of Health and Human Services:

Dear Senator Chiles:

Thank you for your letter of April 2 to Mr. Thomas Donnelly, Jr., on behalf of Dr. Michael F. Ziff who is interested in knowing the amount of funds expended on dental research by the National Institute of Dental Research (NIDR) and the Dental Division of the National Bureau of Standards (NBS).

The total amount of funds expended on dental research by NIDR during FY 1984 was $88,380,000. This amount includes $3,051,000 for research training grants. In FY 1984, the Dental Division of the NBS expended approximately $1,611,000 for dental research. Of that amount, $1,203,655 was received from NIDR.

In FY 1984, NIDR expended $48,598 on research investigating the biocompatibility of dental silver amalgam fillings. So far in FY 1985, the Institute has expended $83,494 in this area. It is expected that this amount will increase significantly during FY 1986.

The NBS Dental Division is not currently conducting research on the biocompatibility of dental amalgams. Instead, it has successfully concentrated its efforts on the development of alternate filling materials to amalgams and is studying the biocompatibility of the new materials that have been developed.

I hope this information will be of assistance to Dr. Ziff.

Sincerely yours,

Signed, James O. Mason, M.D., Dr.P.H.
Acting Assistant Secretary for Health

Although the response does not provide the "total" figures requested it does provide evidence refuting the ADA claim that they are continuously researching the safety of dental amalgam. No money/No Research.