SPECIAL ARTICLE

DOES MERCURY AND DENTAL AMALGAM AFFECT THE IMMUNE SYSTEM?

Sam Ziff

There is a growing collection of scientific research indicating the answer to the title question is an unqualified yes. Some of the following papers are considered pertinent.

Dr. Koller in a 1973 paper titled "Immunosuppression produced by lead, cadmium, and mercury" describes an experiment utilizing rabbits to determine the effect that lead, cadmium and mercury had on the humoral antibody response when exposed to a viral agent. Dr. Koller concludes with the statement "The significant aspect of the present study is that chronic exposure to lead, cadmium or mercury produces immunosuppression to a viral agent."

In a follow-up study published in 1975 Dr. Koller describes the results of an experiment where mice were fed methylmercury chloride at doses of 1 or 10 ppm for 84 days. The mice fed methylmercury chloride had significantly higher mortality rates when inoculated with encephalomyocarditis virus (EMCV) than did nonmethylmercury-treated mice. One ppm methylmercury produced a significant increase in the mortality rate of mice inoculated with EMCV. Dr. Koller concludes "The present study illustrated that prolonged exposure to subclinical concentrations (1 ppm) of methylmercury increased susceptibility of a host to a nononcogenic virus, but did not affect an oncogenic virus. Results emphasize that methylmercury not only poses a threat to public health as a toxic agent, but at subclinical concentrations it may augment infectious agents to cause disease."

Some of you, at this point, probably think that discussing methylmercury and dentistry is inappropriate because dentistry is only concerned with mercury vapor. That thought process would have been valid a few years ago but I am afraid it is slightly outdated today.

Today we have documented scientific evidence that mercury is released from amalgams under various conditions: (1) In certain aqueous solutions (Brune D., 1981); (2) Through migration from restorations into the enamel, dentin, pulp tissue and adjacent gingival tissue where it may accumulate or migrate elsewhere in the body (Soremark, 1962); (3) The normal act of chewing can cause a 15 fold increase in the release of mercury vapor (Gay et al., 1979). We also know now that microorganisms from various sources including the human intestine, are capable of methylating mercuric ions both under aerobic and anaerobic conditions.

In April 1983 Heintze and his associates presented evidence that methylmercury could be formed, in vitro, by common oral streptococci. The experiment utilized mercuric chloride and pulverized dental amalgam in distilled water, as the sources of mercury. Using the oral bacteria Streptococcus mitior, S. mutans and S. sanguis, the researchers found methylmercury in the bacterial cells of all three tested strains. The results indicated that organic mercury compounds may be formed in the oral cavity.
In a study published in 1981, D.A. Lawrence investigated the ability of heavy metals to modulate in vitro primary humoral immune responses. The heavy metals which significantly inhibited antibody production included (relative order of inhibitory activity): Hg²⁺ > Cu²⁺ > Cd²⁺ > Co²⁺ > Cr³⁺ > Mn²⁺ > Zn²⁺ > Sn²⁺.

The immunosuppressive activities of the heavy metals usually correlated with their toxicity and their inhibition of lymphocyte proliferation. Hg²⁺ inhibited the mixed lymphocyte culture response, was extremely toxic and inhibited all in vitro lymphocyte proliferation. The study concluded with the following statement "Heavy metal effects on the various components of the immune system need to be assessed so that their potential role in the development of immunologically related diseases can be determined."

P. Druet, et al, 1982, discuss some interesting studies that had recently been completed in France. The researchers were investigating immune dysregulation and auto-immunity induced by toxic agents. The mechanisms of how different drugs can induce auto-immune disorders including immunologically mediated nephropathies in humans is poorly understood. The accepted theory was that drugs act as haptons or by modifying auto-antigens. However, the mechanism of action to support the theory has not been established. Recent experiments with rats utilizing mercury to induce auto-immune disease suggest that some of these toxic agents modify the fine balance between subpopulations of lymphocytes leading to immune dysregulation and to production of auto-antibodies.

Some of the data from the experiments with the rats strongly suggest that mercury modifies immune homeostasis resulting in immune dysregulation and the production of auto antibodies some of which are of pathogenic significance. It is postulated that this dysregulation may result from the interaction of Hg with the cell membrane of lymphocytes. Other possibilities are: (1) that a modification of B-cell surface determinants could lead to nonspecific B cell stimulation through an enhancement of T helper function and (2) that Hg decreases suppressor T cell functions.

Sporadic cases of the nephrotic syndrome (massive proteinuria) following exposure to mercury imply a direct pathogenesis than occurs in acute tubular necrosis. Renal biopsies of such patients show a variety of histologic types of glomerulonephritis. "Glomerular changes associated with mercury occur independently of acute proximal tubule damage and in the absence of neurologic manifestations of mercurialism." (Wedeen, 1983)

Wedeen also reproduced the results reported by Druet, et al, utilizing Lewis/Brown-Norway F-1 hybrid rats. "These animals showed granular IgG deposits in glomeruli and proteinuria 2 to 6 months after injection." (mercuric chloride 0.1 mg/100 gm intra-peritoneally three times weekly for four weeks) "The biphasic HgCl₂-induced immune disease in rat kidneys appears to be under direct effects on B and/or T suppressor cells rather than a result of modification of autologous antigens."

Wedeen concludes his findings with this statement: "Genetically controlled immunologic variability in rats suggest that immunologically mediated glomerulonephritis may occur as a genetically determined "idio-
syncratic" response in man. The sporadic occurrence of proteinuria in individuals subjected to occupational exposure to mercury is consistent with this view."

Where does all of this lead us to in relation to dentistry? Dr. David W. Eggleston in a 1984 article gives us a partial answer. In his experiments, Dr. Eggleston measured the T-lymphocyte percent of the total lymphocyte count in his patients before and after the insertion and removal of dental amalgam and nickle-base alloys.

The ability of dental amalgam to modify immune homeostasis is evident in this study that produced some dramatic changes in the three patients who participated in it. For example, in patient #1, removing 6 amalgam restorations increased the T-lymphocyte percentage from 47% (before removal) to 73% (after removal), an increase of 55.3%. Reinsertion of only 4 new amalgam restorations decreased the T-lymphocyte percent down to 55%. The other two patients in the study presented data almost as significant. Doctor Eggleston concludes "Preliminary data suggests that dental amalgam and dental nickel alloys can adversely affect the quantity of T-lymphocytes."

"Human T-lymphocytes can recognize specific antigens, execute effector functions, and regulate the type and intensity of virtually all cellular and humoral immune responses. Normal immune function depends on a proper quantity, quality, and ratio of T-lymphocyte helper and suppressor subsets."

"Further research may determine the frequency and magnitude of T-lymphocyte reduction and alteration by dental materials."

This next paper published in December 1983, does not bring mercury into the research protocol. However, I personally believe that there is a highly probable relationship with mercury amalgam as brought out in Dr. Eggleston's paper.

A. Kilpi and his colleagues investigated the T and B lymphocyte (sub)populations in the periductal lymphocyte-rich infiltrates in the labial salivary glands of 8 patients with Sjogren's syndrome (2 SS) associated with systemic lupus erythematosus (SLE). Some of the results of their work follow: "59 + or - 7% (range 29-82) of all inflammatory round cells in the periductal infiltrates in the labial salivary glands of patients with 2 SS with SLE were T3- positive. The number of T4- positive cells exceeded the number of T8-positive cells giving a local T4/T8 ratio of 3.5 + or - 0.8."

The T4/T8 ratio to maintain immune homeostasis is normally 1.8:1 to 2:1. A ratio above or below the norm can predispose to auto-immune type diseases such as SLE and MS (Chatenoud, 1981; Traugott, 1983; Kohler, 1982).

Kilpi and his associates, in the discussion of their work, draw some very interesting correlations with their findings and the multiple serological abnormalities of both SLE and Rheumatoid Arthritis (RA) related to Sjogren's syndrome. They conclude with the following statement: "In the present study the local T4/T8 ratio varied greatly between patients.
This finding extends the earlier observations about the variation in local T/B lymphocyte proportions. Thus, local infiltration of T4-positive and B cells may lead to local autoantibody production."

Low serum sulphydryl (SH) levels may be a useful early prognostic indicator of persistent rheumatoid synovitis. The increased numbers of proliferating mononuclear cells in the peripheral blood may be an indicator of synovial inflammation in early rheumatoid arthritis (RA). The source of these immunoblasts in RA patients is likely from lymphoid tissue within rheumatoid synovial. Immunoblasts in peripheral blood is also a characteristic of several hematological autoimmune disorders. (Hall, et al, 1982). Although the authors make no mention of mercury in their article, the fact that mercuric ions are known to combine with SH-groups of biologically active molecules could be one answer to the low serum sulphydryl levels.

Schopf, et al, 1967, reported that mercuric chloride caused the in vitro blast transformation of normal human peripheral small lymphocytes from individuals who showed no evidence of allergy to mercury. While performing lymphocyte culture studies on a patient with allergic contact dermatitis from mercury, it was determined that blast transformation by the antigen mercuric chloride occurred not only in cultures from the allergic patient, but also in cultures from normal control subjects who were not allergic to mercury.

Caron, et al, 1970, confirmed that mercuric chloride caused nonspecific blast transformation on purified lymphocyte suspensions and also on human fetal lymphocytes. They also tested the effect of other mercury containing drugs Mercurochrome and Merthiolate. The organic mercurial compound merbromin (Mercurochrome) also caused blast transformation whereas thimersol (Merthiolate) was cytotoxic but did not induce transformation. Both mercuric chloride and merbromin function as non-specific mitogens. The authors concluded that the mercuric ion is a non-specific stimulant to lymphocyte transformation in vitro, that it acts in the absence of granulocytes, induces blast cells of normal karyotype and can cause transformation of fetal cord lymphocytes.

If the results of Eggleston's work are applied to the findings of the other authors I have cited, drawing a possible causal relationship between mercury-amalgam and the presently unknown etiology of SLE and RA does not seem totally unreasonable. The possibility of mercury-amalgam being involved in the etiology of SLE and RA as well as other devastating autoimmune diseases demands that the responsible U.S. Government agencies conduct and/or fund the research required to prove or disprove the hypotheses. It would also appear most appropriate at this time for the ADA to reexamine its position on continued unconditional support of amalgam as the restoration material of choice.

As is readily apparent from the different research data presented, the multiplicity of factors affecting homeostasis of the immune system presents problems of tremendous complexities that can no longer be viewed or evaluated in isolation. For example, let's take a person that has amalgam fillings, who develops a cardiac arrhythmia condition and is placed on the drug procainamide to control the arrhythmia. There is scientific evidence that procainamide will decrease suppressor function
in humans. Now we have two factors acting to suppress immunity. Is this person at greater risk than the patient who doesn’t have amalgam fillings?

Then we have the problem of some evidence being presented by researchers that provide conflicting data, such as nickel and lead acting as immunopotentiating metals in some instances. (Lawrence, 1981)

Perhaps Wedeen said it best in the closing paragraph of his excellent paper: "It should be emphasized that the significance of the information I have reviewed remains controversial. The nature of these diseases is likely to remain the subject of controversy until the mechanisms of toxic damage are better understood. In meeting these challenges, the pooling of resources from different disciplines may best serve the interest of both the public by protecting health and of scientists by unraveling some mysteries of nature."

There is no simplistic answer. As professional health care providers it is imperative today, more than ever before, that you stay abreast of current research that is beginning to invalidate many of the "textbook facts" you were previously taught and that you have been applying in your practice.

REFERENCES


REVIEW/ABSTRACTS

Blood plasma measurements were conducted on a population of workers occupationally exposed to mercury in the chlor-alkali industry. An increased incidence of circulating immune complexes was found in the mercury-exposed workers compared to controls. (Stonard et al., Int Arch Occup Environ Health, 52:177-189, 1983).

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T-lymphocyte levels were measured in three patients before and after restorative procedures. In patient No.1, the percentage of T-lymphocytes increased from 47% to 73% after six amalgams were replaced with temporary ethyl methacrylate fillings. After four amalgams were reinserted, T-lymphocyte percentage decreased from 73% to 55%. The T-cell level increased from 55% to 72% when all amalgam and temporary fillings were replaced with cast gold restorations.

Patient No.2 had only one pin retained anterior composite. The T-cell level fell from 63% to 56% when this was replaced with a porcelain-fused-to-nickel-base alloy crown and rose to 77% when this was exchanged for a porcelain-fused-to-ceramic (Cerestore) crown.

Patient No.3 was a multiple sclerosis victim with nine amalgam fillings. The T-cell level increased from 60% to 71% after the amalgams were replaced with cast gold restorations.

The author stated: "Preliminary data suggest that dental amalgam and dental nickel alloys can adversely affect the quantity of T-lymphocytes. An abnormal T-lymphocyte percent of lymphocytes or a malfunction of T-lymphocytes can increase the risk of cancer, infectious disease, and autoimmune diseases." (Eggleston, D.W., D.D.S. The J of Prosthet Dent, 51 (5):617-623, 1984).

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The ability of heavy metals to alter the in vitro primary humoral immune response was investigated. The relative immunosuppressive activities of the heavy metals tested were Hg$^{2+}$, Cu$^{2+}$, Mn$^{2+}$, Co$^{2+}$, Cr$^{3+}$, Sn$^{2+}$, Zn$^{2+}$, Fe$^{2+}$ had no significant effect. Pb$^{2+}$ and Ni$^{2+}$ enhanced the PFC response. The immunosuppressive activities of the heavy metals usually correlated with their toxicity and their inhibition of lymphocyte proliferation. Hg$^{2+}(10^{-6}M)$ and Zn$^{2+}(10^{-4}M)$ inhibited the PFC (B cell) response to a greater extent than they reduced viability, which indicates...
that a functional assay expresses a greater sensitivity to toxic factors. Nickel (10⁻³M) was more toxic than lead (10⁻³M). Mercury was extremely toxic and inhibited all proliferation. Heavy metals may have more significant effects on B-cells than T-cells.

Mercury and copper were the most toxic metals tested. At concentrations greater than or equal to 10⁻⁵M, mercury and copper were toxic to lymphocytes; whereas, 10⁻⁵M of the other heavy metals did not significantly alter cell viability.

Mercury, at 10⁻⁶M concentration, produced a 50% reduction in cell viability, but produced greater than 90% inhibition of the PFC (B-cell) response and 60-90% inhibition of mitogen-induced proliferation. Mercury was shown to be inhibitory as a result of its toxicity.

According to Kazantzis (1978) and Vos (1977), heavy metals induce immunopotentiating or immunosuppressive effects which could result in immunopathologic effects on numerous tissues. Alterations of immune responsiveness has been related to the toxicity of heavy metals. For example, mercury (Hg²⁺) produces renal disease, and some of the pathologic conditions result from the formation of immune complexes or autoantibodies, as demonstrated by Roman-Franco et al., (1978), Kibukamukos, et al. (1974) and Sapin et al., (1977). It has become apparent that heavy metals can alter the humoral and cell-mediated portions of the immune system and, in effect, play a role in the development of immunopathologic disorders. (Lawrence, David A. Heavy Metal Modulation of Lymphocyte Activities. Toxicology and Applied Pharmacology, 57:439-451, 1981).

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Sporadic cases of the nephrotic syndrome (massive proteinuria) following both medicinal and occupational exposure to mercury suggest a more indirect pathogenesis than occurs in acute tubular necrosis. The finding of a variety of histologic types of glomerulonephritis in renal biopsies of such patients implies immunologic mediation of the glomerular disease.

Animal studies have provided support for immunologic mechanisms in mercury-induced glomerular disease. In one study inbred Brown-Norway rats, given 0.1mg/100gm of mercury for two weeks, developed linear deposits of immunoglobulins in glomerular basement membranes without proteinuria. At a later stage of the glomerular disease, granular IgG deposits appeared in the glomeruli and proteinuria 2-6 months after injection. The hypophase mercuric chloride-induced immune disease in rat kidneys appears to be under highly specific genetic control mediated by direct effects on B and/or T suppressor cells rather than a result of modifications of autologous antigens.

Genetically controlled immunologic variability in rats suggest that immunologically mediated glomerulonephritis may occur as a genetically determined "idiosyncratic" response in man. (Wedeen, R.P. Neurotoxicology, 4 (3):134-146, 1983).

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Recent evidence suggests that a number of drugs modify the fine balance between subpopulations of lymphocytes, leading to immune dysregulation and production of auto-antibodies. The evidence for this comes partly from studies in the rat of mercury-induced auto-immune disease.

One week after starting mercuric chloride injections, Brown-Norway
(BN) rats developed circulating antglomerular basement membrane antibodies. At the same time, circulating immune complexes were also detected. Two to three weeks after the first HgCl₂ injection, granular IgG deposits were found in the glomeruli and most other organs tested. A similar disease has been described in mercury-treated rabbits.

Several observations suggest that mercury acts, at least in part, by modifying T-B cell cooperation and induces a polyclonal activation. (1) A lymphocyte proliferation, evidenced by spleen and lymph node enlargement, is observed from day 6 in intoxicated BN rats. (2) Spleen cells from HgCl₂ injected BN rats produce significantly higher anti-hapten trinitrophenyl (TPN) and anti-sheep red blood cells (SRBC) plaque forming cells (PFC) than control rats. Anti-SS DNA antibodies are also produced. (3) HgCl₂ increases the production of nonspecific IgE and is also able to potentiate the specific anti-ovalbumin IgE response.

Data presented strongly suggest that mercury modifies immune homeostasis in BN rats and that, as a consequence of immune dysregulation, autoantibodies are produced, some of which are of pathogenic significance. This dysregulation may result from an interaction of mercury with the cell membranes of lymphocytes.

Another aspect of the disease observed in BN rats is the self-limited character of the auto-immune abnormalities. This suggests that, after polyclonal activation, specific (suppressive cells, auto-anti-idiotypic ab) or non-specific immune suppression occur.

Mercury could also act in different ways: by inducing polyclonal activation, by interaction with cell membranes of lymphocytes, and by enhancing immunogenicity of self-antigens such as nuclear or GBM antigens. (Druet et al., Transplantation Proceedings, XIV (3):482-484, 1982).

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**EDITORIAL**

**WHAT IS BIO-PROBE? WHY ANOTHER NEWSLETTER?**

Those of you who have ever researched a particular scientific subject know how difficult it is to search for, and obtain, information. Computerized data bases have assumed "life saving" proportions to any researcher. With the NIH Mediars data base you can save months of effort in screening and selecting articles you think are pertinent. Once you have identified the articles or books you want to review, the fun really begins. The abstract you initially read gave you a clue but how do you get access to the actual article to review it in its entirety?

Therein lies the problem, and also the genesis, of Bio-Probe. I started out three years ago agreeing to help my son Michael Ziff, a practicing dentist in Orlando, Florida, gather some data on the subjects of dental amalgam and the toxicity of mercury.

At the time my wife and I were living in Alexandria, Virginia within easy access distance to the medical library at NIH in Bethesda. In fact, I was making frequent trips to the library doing research on my own area of interest, nutrition.

Quite frankly, I didn’t have my heart into doing any research on
mercury as I was very skeptical of the basic premise that amalgam fillings (of which I had a mouth full) could have any adverse health effects. About 50 articles later, and no more amalgam fillings, I was no longer a skeptic. In fact I was so excited that my first love, which is nutrition, suffered grievously because of mercury.

The thing that fascinated me from the outset was the paucity of data on the safety of dental amalgam and the utter bewildering scope of the biochemical, pharmacological, pathological, and neurological information on mercury. 700 articles and books later my wife and Michael's wife are "Mercury Widows", but my opinion hasn't changed. There is almost a total void of "primary scientific research" on the safety of dental amalgam, although millions of pounds of this toxic substance have been installed in the mouths of people all over the world. Perhaps more importantly is the fact that scientists and researchers, world-wide, are coming to grips with the insidious nature of mercury and its possible involvement in the many pathological conditions currently of unknown etiology.

So what does all of this have to do with Bio-Probe and another newsletter? I think my son put it in perspective when he stated "Dad, we have to get information on this problem but I just don't have the time. I can't run a dental practice and have the necessary time left to re-search the hundreds of articles we need to review. I know it isn't fair, but you're retired and you're the only one with the time to get it done."

During the last two years I have seen that principle in full op-eration. Phone calls from as far away as Australia, several a day from dentists and other health care providers wanting information on the potential toxicity of dental amalgam. Not opinions, but copies of re-search papers providing scientific data. These calls didn't come to me, they came to my son. How do you run a practice while spending two hours a day on the phone talking and wearing out your poor xerox machine running 30,000 sheets of paper through it (in 9 months) to make copies of articles to send to health care providers desperate for information?

The answer is you can't and still do justice to either discipline, or to the respondents seeking information. Admittedly, there are others trying to fill the information gap: Dr. Hal Huggins to whom we all owe a debt of gratitude for his zeal in pursuing the battle, with his media efforts, seminars, lectures, and newsletter. Dr's. Jerry Mittleman, Joel Berger, Victor Penzer, and Roy Kupsinel for their newsletters and/ or public speaking engagements and Tom and David Mays of Toxic Testing, Inc. with their unceasing efforts to get the "word" out.

The need for scientific information was real so, in an effort to satisfy this need, a group of professionals embarked on a program to put on seminars. These were the PACE seminars in which 8 hours of scientific presentations were made by five speakers and the attendees were provided hard copies of all of the information covered. The speakers were Dr's. Kenneth Goljan, Don Vollmer, Bob Wolf, J.E. Hardy, and Michael Ziff. Unfortunately, the PACE seminars did not prove financially viable and the Doctors elected to withdraw their participation and, instead, publish a Manual embodying all of the research information that had been collected, and for which each had obtained copyright registration. Enter Bio-
Probe. The first copies of the new manual titled "Mercury and Dental Amalgam - The Scientific Facts and Alternatives" were shipped in late June. The manual is approximately 300 pages and is in loose leaf form to permit easy updating. It represents a distillation of over 500 scientific articles and books on the subject of mercury and dental amalgam. There are also sections on immunology, alternative filling materials, and clinical applications and testing protocol.

The manual sells for $125.00 and is available exclusively through Bio-Probe. The manual satisfies a major need of the health care provider by providing a basic reference document that includes a bibliography of over 500 scientific articles and books on the subjects covered. More importantly, it eliminates the need for hundreds of hours of research to get basic data on the subjects.

However, the manual only satisfies one aspect of the information gap. The other aspect in the battle for information was the need for a vehicle that would appraise those interested individuals of the new information on the subject in a timely manner. There was also the need for someone to take the time and effort to do in-depth reviews of several articles, tying them together in a framework that permitted the reader to draw his own conclusions. It was felt that these needs could only be satisfied with the routine publication of scientific data presented in an objective manner. Enter the Bio-Probe Newsletter.

Although I readily admit being biased against the continued use of amalgam in dentistry, the purpose of the newsletter is not to have a forum for me to preach this bias. On the contrary, the purpose of the Newsletter is to provide information that will permit any subscriber to make his own "informed" judgement on the merits of amalgam and mercury. Each health care provider must make that decision individually, and can only do so intelligently if he or she is in possession of the facts.

The Bio-Probe Newsletter is being published eight-times a year at a subscription price of $95.00. The newsletter will be 3 hole punched to permit inclusion in the Manual binder.

To order the Manual or subscribe to the Newsletter, please send your check to Bio-Probe, Inc., 4401 Real Ct., Orlando FL 32808.

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TECHNIQUE TIPS

One goal of Bio-Probe is to gather and disseminate information that will assist practitioners in providing the highest possible quality of posterior composite restorations. Information sources will be from scientific literature as well as clinical experience. Contributions of ideas and suggestions from readers will be welcomed and appreciated.

Our first Technique Tip comes from Michael Ziff, DDS of Orlando, Florida. One possible source of postoperative sensitivity from posterior ceramic restorations is the effect of the acid etchant (conditioner) on the dentin and/or base material.
Dr. Ziff offers the following suggestion:
1. First pack the cavity preparation tightly with dense cotton pellets.
2. Apply gel-type conditioner over desired enamel surfaces with sable brush, paper point or cotton pellet for proper time period.
3. Rinse thoroughly.
4. Remove cotton pellets, rinse again and dry preparation.
5. Apply desired base material.
6. Place bonding agent and restorative material of choice.
This sequence will insure that conditioner will not be flowed onto the dentin and/or base material during the rinsing process, thus avoiding acid deterioration of the base material and/or damage to the dentinal tubules.

Our next two Technique Tips come from Don Vollmer, DDS, of Denver, Colorado. The first one came from the June, 1984 meeting of the Denver Academy of Restorative Dentistry.

Hints from Dr. Gordon Christensen as to restoring two types of Class V lesions.

Large cervical erosion:
1. Roughen sclerotic dentin with a diamond.
2. Gingival retention (helps during 1st 24 hours until bonding agent is effective on dentin).
3. Acid etch enamel.
5. Resin.

Class V Carious Lesion:
1. Remove decay and place undercuts.
2. Liner on entire axial wall.
3. Acid etch enamel.
4. Dry bonding agent eg., Adhesit.
5. Place material in 2 segments: First placement will fill bulk of prep and allow it to draw to the enamel bond. Place second increment which will attach to initial resin and give a tighter gingival seal. By using this second step procedure you will avoid a gingival marginal opening.

Dr. Vollmer thinks that G.C. Lining Cement is the greatest thing he has used for crown buildups. It is ideal for small and medium buildups and he doesn't know what he would do without it.

Procedure:
1. Reduce occlusal surface.
2. Remove old silver filling and decay.
3. Line deep layers with liner eg., Life, Improved Dycal.
4. Mix desired amount of G.C. Lining Cement and apply to undercut areas and build up to desired form.
5. The G.C. Lining Cement sets in one or two minutes and you can proceed with prep, as the lining material adheres well to the dentin.

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MATERIALS

In 1983 (around June) Roel J. Wyman, DDS published an excellent book entitled "The Posterior Composite Resin Restoration - Winning at Restorative Dentistry without Mercury". In it he listed the available choices of composite resins. They were:

- [cc=chemically cured, lc=light cured]
- Estilux Posterior (Kulzer): lc
- Profile (S.S. White): lc
- Santay (Healthco Canada): lc
- Class II (Denmat): lc or cc
- P10 (3M): cc
- Fulfill (Caulk): lc

Since then, two additional materials may be added to that list:

- P30 (3M): lc
- Marathon (Denmat): lc

Bio-Probe will be reporting information, scientific and clinical, on these materials as well as new products, base materials and bonding agents as the information becomes available. Personal preferences and clinical experiences of practitioners are welcomed by Bio-Probe and will be included in subsequent issues.

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Speaking of materials, a dentist recently told me to stop calling amalgam fillings "restorations". He said they were not restorations because they didn't restore anything. They are fillings, he said, because that is all that amalgam can do, fill a cavity. Makes sense to me.

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CASE HISTORIES

We need your help. Marvelous things have been happening out in the "real world". We solicit your help in providing us with documented anecdotal evidence that removal and replacement of amalgam fillings does make a difference in the health condition of some patients. It is significant that the ADA is now recommending that you send case histories to them. If you do elect to send any to the ADA, send us a copy also. We intend to build a repository of case histories that will overwhelm the basic scientific objection to the use of anecdotal evidence demonstrating the toxicity of dental amalgam fillings. Each issue will have one or more case histories that should be of extreme interest to all health care providers.

CASE 1

The individual is a white male who at the time of treatment was 61 years old. He is an engineer who had been exposed to mercury containing instruments most of his working life, i.e., manometers, thermometers, etc., and also frequently had elemental mercury stored in his home.

He had previously been the patient of another dentist who did not believe anything about the potential toxicity of mercury from dental
amalgam. Wanting to explore the problem further, he sought out his present dentist (who practices mercury-free dentistry) to get an evaluation as to whether mercury could possibly be involved in any of his varied health problems.

On November 11, 1983 he had his first appointment with the new dentist. The examination revealed that the patient had 6 amalgam surfaces (2 upper right and 4 upper left). He also had a 4 unit nickel/porcelain bridge on the lower left and a 3 unit nickel/porcelain bridge on the lower right. A complete medical history was taken and he was then evaluated for possible mercury toxicity/hypersensitivity. His diagnosis was possible mercury intoxication and the dentist recommended removal and replacement of the amalgam fillings with a composite. It was also recommended that the nickel bridges be replaced because of their sensitizing potential.

The patient’s health history, taken at the time of the initial visit revealed the following:

First amalgam fillings installed in 1943. Suffered incident of convulsions shortly thereafter. Also developed an allergic reaction to scallops shortly after placement of the fillings.

In 1945, while overseas with the armed forces, had 21 amalgam filling surfaces installed. Developed eczema right after the dental work was completed and was hospitalized for six months during which time he almost had one of his legs amputated because of the severity of the eczema.

In 1960, a gold bridge was installed on the upper right. He was hospitalized 1-2 months later with nausea and vomiting. He was diagnosed as having an ulcer, but did not respond to treatment. His immune system was found to be depressed and he was given antibiotics by IV.

During 1980-1981, patient was hospitalized 7 times. Diagnosed as having an inflamed pancreas that should be removed. Patient refused surgery. Was also diagnosed as having some degeneration of the right kidney, gall bladder, and liver.

Patient has had painful ear infections for 34 years. Chronic headaches, that would start in the occipital region, all of his adult life. For the past 15 years has had joint pains, especially in his wrists and knees. Has had abdominal pains for 30 years. Paresthesia in his feet the last three years. He has had tremors in his right arm and left leg. Occasional uncontrollable hiccups. Occasional chest pains and constipation. Occasional cardiac bradycardia and missed beats. Loss of visual accomodation, farsighted in the early 1960’s. Patient says he is irritable, experiences anxiety, is unable to concentrate and feels he has had a decrease in memory, has suffered a loss of libido, is more introverted (shy), and has had an erythematous rash, with itching and burning, especially on his legs, sternum and right facial cheek.

No depression, no fits of anger or loss of self control, no nightmares, and hearing is ok.

Patient was referred to an M.D. for a coordinated protocol on Jan 11, 1984. On Feb 6, 1984, M.D. advised dentist that patient had recovered from a bleeding ulcer and that he would administer Vitamin C by IV the morning of his first dental appointment which was scheduled for March 7, 1984.

March 7, 1984: 6 amalgam surfaces replaced with composite (2 upper right and 4 upper left).

March 15, 1984: Removed 4 unit nickel/porcelain bridge from lower left. There were three amalgam surfaces under the bridge which were then replaced with composite.
March 22, 1984: Removed 3 unit nickel/porcelain bridge from lower right.

March 29, 1984: Delivered new porcelain fused to gold 4 unit bridge to lower left. Patient stated he is "feeling much better every week. Back pain is now totally gone".

April 5, 1984: Delivered new 3 unit porcelain fused to gold bridge to lower right. Patient stated "I am feeling great now". The patient also presented the dentist with a list of his symptoms and their chronology since he had started treatment. Changes in patient symptomatology started to occur on March 11, 1984: sneezing and headaches decreased but back pain and eczema increased. By March 27: back pain, neck stiffness, sneezing, coughing, eczema had all noticeably decreased. Headaches, dry throat and tooth aches had all ceased. By April 15, 1984: all symptoms had ceased with the exception of the eczema and earaches which had both decreased from moderate to light. Follow-up on June 26, 1984, patient stated that the eczema and earaches had cleared entirely.

NOTE: When you send in your case histories, whenever possible, please send complete documentation, i.e., copies of patient's health history, dental records, etc. Records should include patient's name and phone number. Names will never be used for any purpose without your's and the patient's written permission. If we are going to publish a case history in the Newsletter we legally have to have adequate proof as back up in our files.

EVENTS

We intend that our 'Events' section do more than just report events that are traditionally reported in other publications. Ours' will be more personal. In addition to major events we will be telling you about individual events in which our subscribers are participating, such as one-on-one debates with pro-amalgam advocates, appearances on local radio and TV, presentations being made to auxiliary dental personnel, etc. We also welcome our readers to report their observations of major events.

Our first event is being reported by Dr. Michael Ziff who attended the "Workshop on Biocompatibility of Metals in Dentistry".

The workshop was sponsored by the National Institute of Dental Research and hosted by the American Dental Association (ADA). It was held in Chicago on July 11-13, 1984. According to the ADA, "more than 200 dental researchers, practicing dentists and physicians from around the world participated in this workshop. Eleven presentations were made on a wide range of topics related to the biocompatibility of dental metals."

John W. Stanford, PhD, Chairman of the Workshop and Secretary of the ADA Council on Dental Materials, Instruments, and Equipment (CDMIE) opened the conference by declaring: "This workshop is not a 'Consensus Committee'. Recommendations for the use or discontinuance of use of dental materials will not be made. The purpose of the workshop is to present information on the subject matter and identify areas for recommendations for research where needed. This is the first of a series of
workshops to be held over the next 12-18 months".

There were actually thirteen 45 minute presentations made. They were:

2. "Dental amalgam related mercury vapor exposure". Carl W. Svare, D.D.S., Ph.D.
3. "Epidemiologic aspects of assessing the effects of metals used in the practice of dentistry". Herman Lehman, D.D.S., M.S.
4. "Allergic potential or hypersensitivity – methods of determining and significance". Desmond Burrows, M.D., FRCP.
5. "Allergic reactions of nickel and cobalt in the mouth". Torkil Menne', M.D.
7. "Carcinogenic potential of metals". John Autian, Ph.D.
8. "Corrosion, galvanic cell production, and release of metal ions". Miroslav Marek, Ph.D.
9. "Toxicity considerations for metallic products". Jack Lemons, Ph.D.

A fifteen minute question and discussion period followed each presentation, as well as one hour periods Wednesday and Thursday afternoons and a two hour period Friday morning.

On Friday morning an additional presentation was made by Dr. Rob Herber, representing a group of toxicologists and chemists in Holland. This group, concerned with the effect of dental amalgam mercury on patients, has been conducting research on the subject. They are prepared for publication and recommendations to the government of Netherlands regarding the risk to patients, especially fetuses. They especially decried the reliance on TLV and MAC values, as well as urine and blood Hg levels, to predict the safety of the levels of released mercury.

The Workshop Summary and Recommendations were provided by Edgar W. Mitchell, Ph.D., Secretary for the ADA Council on Dental Therapeutics. A brief synopsis of his remarks follow:

The summary and recommendations of this workshop are a result of meetings of the Planning Committee (Dr. Mitchell, Dr. Stanford, Dr. Rupp, Dr. Moffa, Dr. Autian, Carl W. Fairhurst, Ph.D., James Miller, D.D.S. and Joyce Reese, D.D.S., M.S.). The results will be published in a future edition of the ADA Journal.

Questions have been raised about the safety and efficacy of various metals used in dentistry. There is a need to continue to assess the safety of the use of nickel alloys. The prevalence of nickel hypersensitivity is approximately 10% in females and less than 1% in males. Lung cancer has been reported from exposure to beryllium and some chromium products, but there is no evidence of any risk to dental patients. The prevalence of cobalt sensitivity is less than 1%.
As a result of recent technological developments, we are now able to measure extremely small amounts of mercury released from dental amalgams in the form of mercury vapor. Evidence was presented to the workshop showing that mercury vapor is released from the surface of dental amalgams, particularly during chewing.

There is limited information available on the pathology resulting from this released mercury. Some information is available on the effect on the developing fetus, but it is controversial. Additional studies are required to determine the actual risks.

Galvanic currents do result from the use of metal alloys in the mouth, but no adverse effect has been demonstrated.

Measurements of mercury levels in the hair, blood or urine do not correlate to the toxic effects of mercury.

The **Consensus** of the workshop was that the prevalence of mercury hypersensitivity is less than 1%. ("Consensus" means that no scientific documentation was presented at the workshop to support that estimation!). Patch testing to determine hypersensitivity to dental materials should be referred to a qualified medical specialist.

**Special Note:** Although Dr. Stanford in his opening remarks stressed the fact that the workshop was not a Consensus Committee and would make no recommendations for the use or discontinuance of use of dental materials, the prepared ADA Press Release presented to the media did contain recommendations on continued use of amalgam, and are summarized as follows:

- On the basis of the information presented in this workshop, there is no documented evidence for recommending the discontinuation of the use of dental amalgams as a restorative material in dentistry. Additionally, the removal of dental amalgam can only be recommended in those patients who have a true hypersensitivity to mercury or other constituents.

The media representatives present at the press conference, following the workshop, made a specific point of questioning Dr. Mitchell on this apparent discrepancy. **It will be interesting to see how the ADA treats this when they publish the workshop results in the JADA.**

Dr. Mitchell listed 15 recommendations for future research:

1. Diagnostic and analytical procedures should be investigated for documenting exposure to metals in alloys.
2. An evaluation should be made of nickel salts or other nickel compounds which may be formed during the fabrication and use of base-metal alloys.
3. Investigate the role of nickel, beryllium and chromium as potential carcinogens in dental laboratory technicians.
4. An assessment should be made of mercury loss from chewing on dental amalgams of different alloy compositions.
5. Studies should be initiated to determine whether methyl mercury can be formed in vivo.
6. Epidemiologic studies should be initiated to assess the prevalence of mercury allergy in the United States population.
7. Biological sampling procedures should be investigated to determine a reliable means of estimating body burden of mercury.
8. Studies should be initiated to assess blood levels of mercury which may result from dental amalgam.
9. Research should be initiated to determine whether the effects of mercury on T-lymphocytes may be a means of early detection of sub-clinical manifestations of mercury toxicity.
10. Studies should be initiated to develop more definitive tests for determining the hypersensitivity to metals used in dentistry.
11. Studies should examine the potential that thyroid gland enlargement may be an early predictor of mercury intoxication.
12. Studies are encouraged to determine whether a relationship exists between maternal exposure to mercury and teratogenesis.
13. The effects of conditions which accelerate corrosion of dental materials on the release of metal ions should be studied in more detail.
14. The composition of corrosion products should be identified as well as the effect they may have on oral tissues.
15. Continued research is recommended on the development of alternative restorative materials.

The Workshop Planning Committee suggested immediate clinical implementation of the following:
1. There is a need by dentists and physicians to recognize nickel as a common allergen.
2. Manufacturers, laboratories and dentists should be encouraged to identify alloys used in the fabrication of prosthetic devices in terms of contents which may affect a patient’s health (nickel, chromium, cobalt, etc.).
3. Dentists and administrators of dental laboratories should be encouraged to inform employees who work as technicians regarding the need to avoid inhalation exposure to dusts from alloys.
4. Practitioners are encouraged to document in patient records content of alloys used in restorative materials.
5. Health histories should include documentation of individuals sensitive to metals.
6. Patch testing for sensitivity to metals is the responsibility of professionals trained in the administration and interpretation of the tests.
7. Practitioners are encouraged to become familiar with the symptoms of metal exposure.
8. Practitioners are encouraged to report case histories of adverse reactions to or effects from biomaterials to the American Dental Association.

ADDITIONAL EVENTS

2. The Dental School of The University of Texas at San Antonio, has invited Dr. Michael Thurmond and Dr. Kenneth W. Goljan to participate with Dr. Nelson W. Rupp in a seminar to be held August 17-18, 1984. I think it significant that this teaching institution recognizes that a potential problem may exist and is willing to expose its faculty and student body to an open debate on the potential toxicity of amalgam fillings.

3. The establishment of The Academy of Biological Dentistry. Murray J. Vimy, B.A., D.M.D., F.A.G.D. of Calgary, Alberta is founding the Academy. One of the primary purposes of the Academy will be to help promote greater awareness in the dental profession regarding the possible toxic and allergic effects of dental materials. If you want to know more, contact Dr. Vimy at 615 Gulf Square, 401 9th Avenue S.W., Calgary, Alberta T2P3C5. Or call him at (403) 266-2251. The founding meeting is presently scheduled to be held October 11-14, 1984. (Note: The academy is
not intended to supplant the existing political structure of dentistry. It would work within it to encourage and stimulate change within the dental profession itself.

4. The Ohio State Dental Assistants Association has invited Dr. Michael Ziff and Dr. Nelson W. Rupp to debate the subject of mercury/dental amalgam at their annual meeting in Columbus, Ohio (Hyatt Regency) on September 15, 1984.

5. Dr. Michael Ziff and Dr. J.E. Hardy will put on a 6 hour seminar at the semi-annual meeting of the Southern Academy of Clinical Nutrition scheduled to be held in St Petersburg Beach, Florida on September 22-23, 1984.

6. Something you should all be aware of has happened in the State of Massachusetts. The Attorney General of that state has summoned Dr. Victor Penzer to turn over all records, all correspondence, names of correspondents (and anything else he could think of to put in the summons) in his work related to mercury toxicity. The basis of the summons? The Attorney General has stated that it is a violation of the law of the State of Massachusetts to assert that mercury from dental amalgam fillings can be toxic and calls such action deceptive and fraudulent. If you want to assist Dr. Penzer in his fight or desire more information, contact him at 197 Grant Avenue, Newton, MA 02159, (617-332-1234).

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**FORUM**

One of our subscribers is presently working with an MS patient and has written us requesting information on possible treatment protocols. The literature is replete with many therapies, some of which have been effective in a great many patients, none of which are effective for everyone diagnosed as having the disease. Although it is supposedly a demyelinating disease of the CNS, medical researchers are suspecting and finding some substantiation for the hypotheses of an immunologic abnormality being the underlying cause.

Many of the symptoms and signs identified with MS are similar to those of micromercurialism. As we have indicated in our lead article on the immune system, there is growing scientific evidence that mercury can cause significant variations in one’s immunocompetence.

There are also a number of anecdotal case histories that reflect amelioration or elimination of MS symptomatology upon removal of amalgam fillings. Of course, there are also case histories that show little or no effect or improvement with amalgam removal and replacement. Which of course is symptomatic of the disease itself, i.e., spontaneous remissions or exacerbations of symptoms. This last aspect confounds the researchers because if it is truly a demyelinating disease, how can there be spontaneous remissions that can last for months or years?

The following information has been gleaned from the literature and anecdotal case histories:

1. Do not perform or recommend a mercury patch test because of the potential for exacerbation of symptoms.

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2. Do utilize sequential removal of the amalgam fillings based on galvanic readings in accordance with the protocol established by Dr. Hal Huggins. Sequential removal is indicated because of possible CNS and motor/muscle involvement.

3. Utilize oral Detoxification protocols embodying as a minimum Vitamin C, Cysteine, Selenium, Magnesium, Zinc, and B-6.

4. From a dietary standpoint, there is substantial evidence that a great many MS patients are unable to tolerate gluten and that an imbalance of essential fatty acids or dysfunction of the enzymes responsible for conversion of linoleic acid to GLA may be involved. Dietary modification should include avoidance of gluten, a reduction of saturated fatty acids, supplementation of eicosapentaenoic acid (Max EPA), or increased intake of fish rich in oils such as mackerel, trout, salmon and sardines) Octacosanol therapy, Evening Primrose Oil (rich in GLA) and lastly a reduction in sugar and refined carbohydrate intake.

You may wish to consult with Dr. Carlton Fredericks or Dr. Jeffrey Bland. Both have extensive clinical experience with MS patients and have specific protocols that have achieved excellent results in many cases.

NOTE: The FORUM section will be devoted to providing answers to readers questions (if we can) and publishing letters submitted by our readers.

A Patient's Guide to Mercury Amalgam Toxicity by Roy Kupsinel, M.D. This is an excellent booklet with original artwork (16 pages 5 1/2x8 1/2) that really gets the message across. The back cover has blocked area for insertion of your name and address. They can be mailed or used as patient hand-outs. Cost is $44.00/100, postage and handling included. Single copy is $1.00. Write to Dr. Kupsinel at P.O. Box 550, Oviedo, Fl 32765. Or give him a call at (305) 365-6681.

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If you have any questions or if we can be of assistance, give us a call at 305-299-4149