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MERCURY AMALGAM FILLINGS AND PERIODONTAL DISEASE
Michael F. Ziff, D.D.S.

The International Academy of Oral Medicine and Toxicology has conducted an in-depth review and analysis of the Transcript of the July, 1984 NIDR/ADA "Workshop on Biocompatibility of Metals in Dentistry". (1) This review revealed some intriguing, if not disturbing aspects of the mercury amalgam controversy. One consideration brought forth is a relationship between mercury amalgam fillings and periodontal disease.

THE NIDR/ADA WORKSHOP ON BIOCOMPATIBILITY OF METALS IN DENTISTRY: TRANSCRIPT.

It should first be emphasized that the NIDR/ADA Workshop (2) determined that "studies have demonstrated that patients are exposed to mercury vapor when amalgams are placed as a restoration, when existing amalgams are removed, and during chewing" (3) and further stated that "practitioners are encouraged to become familiar with the symptoms of metal exposure". (4) These two statements were also printed in the Journal of the American Dental Association in volume 109, September, 1984.

Dr. Nelson W. Rupp states in his workshop presentation that GINGIVITIS is a DISCRIMINATING symptom for micromercurialism and that "it is essential that dentists and physicians be alert to the possibilities of mercury toxicity, because even if there are only a few hypersensitive individuals, for each one of them the condition is serious." (5)

Further evidence of a relationship between periodontal disease and mercury amalgam fillings can be found in the Workshop presentation of Dr. Jack E. Lemons. (6) Dr. Lemons states that "selected metallic elements have been shown to be very toxic at low concentrations in tissue culture experiments. Examples of toxic profiles at concentrations below 10 ppm include copper, silver, mercury, chromium (plus 6 oxidation state), and vanadium. Another aspect of this type characterization is the synergistics of several elements being present simultaneously, which is most common in the in-vivo situation under conditions of biocorrosion. In general, however, the most toxic elements are thought to cause significant tissue reactions both locally and systemically." Dr. Lemons also states that "another tissue reaction sequence is associated with electrical phenomena. If a metallic surface is the anode (in) an in-vivo circuit (e.g., two electrochemically different alloys) the local tissue response can cause rapid and significant resorption of bone."

Dr. Robert L. Cooley adds further evidence in his workshop presentation. (7) He states that "the oral mucosa may respond to both primary irritation and allergic sensitization from metals used in restorations or a prosthesis. An example of allergic sensitization from metals is contact stomatitis. When this occurs, the patient may present with any of the following symptoms and signs:
SYMPTOMS
Loss of Taste
Numbness
 Burning Sensation
Soreness
Possible Itching

SIGNS
Mild to Marked Erythema (redness)
Edema may be Present
Absence of Lingual Papillae
Vesicles (rarely)

TEXTBOOKS AND REFERENCES:
The standard, most widely used and respected textbooks and references clearly establish an unassailable verification of the effect of mercury on the oral tissues.

A Textbook of Oral Pathology, by Shafer, Hine and Levy(8) states the following: "The oral cavity suffers seriously in mercurialism and evidences numerous characteristic but not necessarily pathognomonic signs and symptoms. There is an increased flow of saliva and a metallic taste in the mouth. The salivary glands may be swollen, and the tongue is sometimes enlarged and painful. Hyperemia and swelling of the gingiva are occasionally seen. The oral mucosa is prone to ulcerations on the gingiva, palate, and tongue. In severe cases, pigmentation of the gingiva may occur. Loosening of the teeth, even leading to exfoliation, has been reported. A toxic reaction from absorption of mercury in dental amalgam has been reported on a number of occasions. Patients with ACRODYNIA (a mercury induced syndrome) exhibit profuse salivation and often much dribbling. The gingiva becomes extremely sensitive or painful and may exhibit ulceration. Bruxism is a common finding, and loosening and premature shedding of the teeth often occur."

The statement "When contacting dissimilar base metal alloys are present in the oral cavity, spectacular examples of corrosion and soft and hard tissue destruction have been reported" is found in Skinner's Science of Dental Materials, edited by Dr. Ralph W. Phillips.(9)

The Pharmacological Basis of Therapeutics, by Goodman and Gillman's offers the following signs and symptoms of mercury poisoning: ACUTE MERCURY POISONING: "There is first a strong metallic taste; with 24 to 36 hours, a stomatitis develops, characterized by foul breath, sore gums and excessive salivation. Discoloration of the gingival margins, similar to the lead line, appears later, and local infection, loosening of the teeth, and necrosis of the alveolar processes may follow". CHRONIC MERCURY POISONING: "Chronic poisoning with inorganic mercury causes gingivitis, stomatitis, and excessive salivation."

Even the prestigious Merck Manual(11) lists gingivitis as one of the three characteristics of chronic mercury poisoning.

The document A Recommended Standard for Occupational Exposure to Inorganic Mercury presented in 1973 by the National Institute of Occupational Safety and Health(12) (NIOSH is a branch of the U.S. Public Health Service under the Secretary of Health and Human Services) states that "the appearance of gingivitis and stomatitis accompanied by excess salivation or a metallic taste, erythema, and
tremor are the classical signs of poisoning by mercury vapor and inorganic forms of mercury."

In 1968, the Report of an International Committee for the Establishment of Maximum Allowable Concentrations for Mercury Compounds(13) declared that "symptoms related to the mouth such as gingivitis, stomatitis, and excessive salivation may occur from chronic exposure to mercury vapor."

Smith and Williams, in their 1982 textbook Biocompatibility of Dental Materials,(14) declare that "the most common signs and symptoms of chronic mercury exposure described are erethism, tremor, disturbances of kidney and gastrointestinal function, salivation, metallic taste, sore throat, speech disturbances, changes in handwriting, motor and sensory disturbances, vision impairment, ORAL PATHOSIS, hypersensitivity, loss of appetite and weight, and insomnia. They also point out that "Restorations are placed close to the gingiva or subgingivally. The epithelial attachment may, therefore, be indirectly or directly effected. The leaching out of toxic elements from a restoration may also have an influence on the hemidesmosomal attachment mechanism found in this location, and it may affect the normal cell proliferation of the gingival epithelium. Local gingival inflammation has been reported adjacent to amalgam restorations. Such gingivitis may be due to chemicals leaching out of the restorations or bacteria adhering to the amalgam. Biopsies from gingivae in contact with amalgam restorations have higher mercury content than control biopsies. The tendency for bacterial adhesion to amalgam is relatively low; indicating that any local gingivitis adjacent to amalgam restorations may be due to toxic elements from the restoration. However, the use of amalgam and gold in close approximation may lead to increasing plaque accumulation due to galvanic action."

Although the preceding comments represent only a partial listing of the documentation found in the textbooks and scientific references (much more is available), it represents undeniable definition of the pathological effects of mercury on the tissues of the oral cavity.

SCIENTIFIC DOCUMENTATION:

In 1933, Hyams and Ballon(15) stated that "dissimilar metals in the oral cavity may be responsible for local objective changes such as acute and chronic inflammation, blanched or grayish patches, erosions, ulcers, areas of leukoplakia and pigmentation. Sufficient evidence has now accumulated to indicate definitely that dissimilar metals in the oral cavity can be responsible for local lesions, and local and general symptoms."

Freden and associates(16), in 1974, reported that Zander (1957) and App (1961) had found local inflammatory reactions in the gingival tissues, apparently induced by amalgam fillings, in their studies. Freden's group measured the mercury content in control gingival tissue and gingival tissue in contact with silver amalgam fillings. The results showed that biopsies from gingival tissues in contact with amalgam restorations had a markedly higher mercury content than
control biopsies. The range was 19-380 micrograms per gram (Mean=147) in the test biopsies and 0-10 micrograms per gram (Mean=3) in the control biopsies.

In a 1975 study, Goldschmidt and associates(17) reported that "Dental alloy restorations have been associated with chronic inflammatory changes in gingival tissues (App 1961, Sanches Sotres, Van Huyzen & Gilmore 1969, Trivedi & Talim 1973, Trott & Sherkat 1964, Turgeon, LeMay & Cleroux 1972, Zander 1987). Using three independent criteria, they found that 10(-4) to 10(-6) M. concentrations of ions liberated from the corrosion of dental amalgam produced injurious effects on human gingival fibroblasts. They concluded that "These results suggest that corrosion products of amalgam are capable of causing cellular injury or destruction."

In 1978, Professor Till in Vienna, Austria found that the amounts of mercury released from dental amalgam produced periodontitis in germ free animals. (Bio-Probe is currently attempting to obtain an English translation of the German-language study.)

SUMMARY:

The current position of the dental profession is that the etiology of periodontal disease is multifactorial. Traditional periodontal therapy has relied upon the elimination or limitation of subgingival plaque formations, augmented by correction of occlusal disharmony and surgical elimination of excessive pocket depth when necessary.

Stimulated by frequent failures, poor patient response, and clinical inconsistencies numerous dentists began seeking other answers to the perplexing problem. Attention was directed to body chemistry (nutritional supplementation). dietary influences (i.e. control of blood sugar levels), and the newly popular aggressive attack on specific microorganisms.

The documentation presented herein provides more than ample scientific foundation for a formal position that mercury released from dental amalgam fillings is a potential etiologic factor in periodontal disease.

CAN THIS POSITION BE REFUTED?

1. The textbooks, scientific references, and even the NIDR/ADA Workshop clearly establish that mercury and/or amalgam fillings can pathologically damage periodontal tissues.

2. The ADA and NIDR have publicly acknowledged that mercury is released from amalgam fillings during chewing.

3. Amalgam advocates claim that not enough mercury is released from dental amalgam to harm patients, unless the individual is "hypersensitive" to mercury. Yet, they cannot produce one single scientific study to support this position. Textbooks and world experts on mercury acknowledge that TLV's are based only on the appearance of "tremor" in subjects and the toxic threshold for pathological damage from mercury is unknown.
4. The scientific literature contains a few case histories of periodontal disease corrected by removal of amalgam fillings. There are no case history reports of periodontal disease NOT being corrected by the removal of amalgam fillings.

5. There have been no scientific studies conducted that refute a possible correlation between periodontal disease and mercury amalgam fillings. The documentation found in the textbooks and other references stands absolutely unchallenged on a scientific basis.

6. The partial success of other periodontal therapies can be scientifically explained. In 1983 Heintze and associates(18) demonstrated that oral streptococci found in dental plaque were capable of methylating the mercury released from dental amalgam. Since methylmercury is lipid soluble, it is capable of being absorbed more readily by the contacting tissues. Plaque removal would therefore result in LESS mercury absorption by the contacting gingival tissue.

Antibiotic therapy and use of other antimicrobial agents would also dramatically reduce the methylation of released mercury in the gingival sulcus.

Nutritional supplementation invariably includes numerous agents that are capable of chelating or otherwise detoxifying mercury in humans.

CONCLUSIONS:

A formal position that mercury released from dental amalgam fillings is capable of causing pathological damage to periodontal tissues can be substantiated by referral to documentation found in textbooks, scientific references, case reports, and the NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry Transcript.

No SCIENTIFIC documentation or evidence can be found to refute this position. If formally or informally challenged, opposition to this position cannot be scientifically defended.

The significance of this position presents consideration of its influence on three distinct aspects of dental practice:

1. AMALGAM REMOVAL:

If the patient exhibits any evidence at all of periodontal pathology, amalgam removal can be justified on that basis alone. Obviously, radiographic evidence of periodontal bone loss, particularly in locations containing inter-proximal amalgams, would be ideal. In the absence of this evidence, thorough and accurate description and charting of the condition of gingival tissues, particularly if initiated by doctor and auxiliary, would be uncontestable.

The advantages of utilizing this approach for justifying amalgam removal are obvious:

A. Periodontal disease is a universally acknowledged pathological entity.
B. It is readily and undeniably demonstrable.
C. The pathological influence of mercury and/or dental amalgam can be scientifically substantiated.
D. The need for additional testing or evaluation, much of which is controversial or contestable, is reduced.
E. It undeniably falls within the purvue of dental therapy.
F. Dental insurance companies would not be able to justify denial of amalgam removal claims.
G. The position augments the systemic effects of mercury from
dental amalgam, rather than precluding them.

2. PERIODONTAL THERAPY:
The revelation of the scientific documentation defining the
pathological influence of mercury and/or dental amalgam on periodontal
tissues introduces a new parameter to periodontal therapy. The omis-
sion of its consideration from formal education and "usual and custom-
ary practice" does NOT release the dental profession or individual
practitioners from their obligation of responsibility to the scien-
tific documentation.

If, as it has been clearly demonstrated herein, that even the
textbooks define the potential influence, then the practitioner is
clearly responsible for the information. If formally challenged, the
practitioner would find it difficult to defend omission of this
documented etiologic factor.

3. AMALGAM PLACEMENT:
Contemporary emphasis of the doctrine of "Informed Consent"
dictates consideration of the pathological influence of dental amalgam
on periodontal tissues.

Routinely informing patients of the possible consequences of
the placement of materials in operative dentistry is not "Usual and
Customary Practice". So, if practitioners merely place amalgams with-
out comment, they are still protected by this doctrine.

However, if discussion of the "Amalgam Controversy" is
initiated by either the dentist or the patient, an entirely different
situation exists. The doctrine of "Negligent Misrepresentation"
maintains that a professional is responsible for information related
to the scientific documentation. A practitioner who declares, or even
worse, puts into writing that "amalgam fillings are not harmful to
patients" may very well be in jeopardy, since that position is
contrary to what is found even in dental textbooks. The same holds
ture for amalgam defenders who are on record for that position in
public or professional media.
The prudent course of action would seem to be that of openly
declaring only that which can be scientifically defended and
maintaining a totally neutral and open-minded posture on all else.

REFERENCES

1. Vimy, M.J.; Ziff, M.F.; Ziff, S. (on behalf of The International
Academy of Oral Medicine and Toxicology) A Critical Evaluation of the
NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry. 1985.

2. Transcript: Workshop on Biocompatibility of Metals in Dentistry.
3. Ibid. pg. 418.
4. Ibid. pg. 422.
5. Ibid. pg. 277.
6. Ibid. pg. 171, 173.
7. Ibid. pg. 180.

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REVIEWS/ABSTRACTS

In man the clinical patterns of inorganic and organic mercury toxicity are different. Inorganic mercuric chloride mainly affects the renal and gastrointestinal systems. The characteristic neurological feature is a fine tremor, particularly of the hands and fingers. In contrast organic methyl mercury toxicity results in an exclusively neurological disorder, the characteristic features being ataxia, dysarthria, parasthesia and tunnel vision. To study the action of these classes of mercury compounds on neurones small amounts of mercuric chloride or methyl mercuric acetate were injected or iontophoresed into the rat cerebrum. The ultrastructural changes which followed were identical. Progressive and often pronounced cytoplasmic swelling of neurones suggested a defect at the cell membrane level. Thus in spite of their distinctive clinical syndromes these 2 classes of mercury compounds are capable of inducing neuronal necrosis. Gallagher P.J., Mitchell J., and Wheal H.V. Identity of ultrastructural effects of mercuric chloride and methyl mercury after intracerebral injection. Toxicology 23: 261-266, 1982.

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Methylmercury dicyandiamide (MMD) when given intraperitoneally at a dosage of 4 mg/kg of body weight at weekly intervals for 3 weeks
resumed in death of guinea pigs fed an ascorbic acid deficient diet. Controls fed an ascorbic acid deficient diet survived during this period as did guinea pigs given MMD and fed an ascorbic acid adequate diet. In a second experiment, guinea pigs fed an ascorbic deficient diet containing 22 ppm of MMD died with 26 days and had severe hemorrhagic and ulcerative gastroenteritis and coagulative necrosis of the liver. Ascorbic acid deficient controls died at 34 days. The MMD-containing ascorbic adequate diet killed guinea pigs in 150 days. Guinea pigs fed an ascorbic acid deficient diet with 44 ppm of MMD died within 20 days with acute neurologic signs. Pathologic changes were mostly in the gray matter. Guinea pigs fed MMD and a diet with adequate ascorbic acid survived for 38 days whereas the ascorbic acid deficient controls survived for 47 days. Results indicate that ascorbic acid deficiency can be a factor in the location and severity of clinical signs and lesions of MMD. Yamini B. and Sleight S.D. Effects of ascorbic acid deficiency on methyl mercury dicyandiamide toxicosis in guinea pigs. J Environ Pathol Toxicol Oncol (JEPTO), 5 (4/5) 139-150, 1984.

Methylmercury is toxic to both the mature and the developing nervous system. One mechanism of its effects on the developing neonatal cerebellum is its interference with cell production by mitotic arrest. To investigate whether this mechanism is active in the prenatal CNS, fetuses exposed to methylmercury were compared to control fetuses 24 hours or 48 hours after an 80 mg/kg dose to their dams. By the first sacrifice time, levels of Hg in fetuses approached the level in the dam and by the second sacrifice time methylmercury-exposed fetuses weighed significantly less than controls. Four regions of the developing brain were studied to evaluate methylmercury effects on mitotic activity. General measure such as mitotic index, number of proliferative cells, and thickness of the proliferative zone were not reduced by treatment in any region at either sacrifice time. In contrast, each region showed evidence of methylmercury effects on the pattern of mitosis. Exposed fetuses had increased numbers of early mitotic figures, decreased numbers of late mitotic figures, or a decrease in the proportion of cells reaching late mitosis. Thus, neurons produced during gestation, like those produced postnataally, appear to be sensitive to methylmercury's antimitotic action. Whether the arrest of these cells leads to a permanent reduction in neuron number, as it does in neonates, remains to be investigated. Rodier P.M., Aschner M., and Sager P.R. Mitotic arrest in the developing CNS after prenatal exposure to methylmercury. Neurobehav Toxicol Teratol 6 (5): 379-385, 1984.

Concentrations of eight trace elements viz. Fe, Zn, Co, Cr, Se, Rb, Sc, and Hg were determined in the normal and leukaemic human whole blood. Neutron activation technique incorporating a Ge (Li) detector having 60 cm³ active volume and a 4096 channel pulse height analyser was used for this purpose. There was a significant decrease in the concentrations of Co, Cr, Rb, and Fe in leukaemic blood. The concentrations of Zn and Sc were decreased by small amounts which were not
statistically significant. The concentrations of Se and Hg were increased in leukaemia. When concentrations of these trace elements in leukaemic blood were compared with the leukocyte cell counts in each subject, it was found that the concentrations of Fe, Zn, Co, Cr and Sc decreased and those of Se and Rb increased monotonically with increase in the leukocyte cell counts. The increase in the level of Rb and the decrease in the level of Hg as a function of leukocyte cell counts was in contradiction to the changes in the concentrations of these elements in leukaemia, as compared to those in the normal blood samples. The study thus suggests some special role of Rb and Hg—both non-essential elements, in the initiation and progress of leukaemia. Mangal P.C. and Sharma P. Effect of leukaemia on the concentration of some trace elements in human whole blood. Indian J Med Res, 74:559–564, 1981.

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A number of studies have already shown that mercury is an allergen. Since the dental community is constantly being exposed to mercury through the preparation of silver amalgams, the objective of this study was to see if the rate of hypersensitivity to mercury increased as students progressed through the dental curriculum.

A total of 171 students (51 freshmen, 52 sophomores, 37 juniors, and 31 seniors) volunteered for the project. Each student filled out a questionnaire and information was collected on all previous exposures to mercury and the presence of other allergies. Two telfa pads were placed on a piece of waterproof adhesive tape. One of the pads had been treated with 0.5 ml of a 0.1% solution of HgCl₂. The other pad, which served as a negative control, was treated with 0.5 ml of water. The tape containing the pads was placed on the student’s forearm. After 48 hours, the patch was removed.

The only positive reactions that were seen were simple contact dermatitis and these cutaneous reactions were only seen under the pad containing HgCl₂. Fifty-four students had a positive reaction, and the rates of mercury hypersensitivity from freshmen to seniors were 31%, 27%, 32%, and 39%, respectively. Through the use of chi-square analysis, it was found that these differences were not significant. From the questionnaires it was found that the average number of alloy restorations in the students with positive reactions was 9.5. The average for the negative reacting group was 7.7. By using the chi-square test it was found that there was a significant correlation between the number of amalgams and the incidence of mercury hypersensitivity. A similar analysis showed that there was no significant relationship between a previous history of allergies and a positive reaction of HgCl₂. Miller E.G., Perry W.L. and Wagner M.J. Prevalence of mercury hypersensitivity in dental students. J Dent Res, 64:338, Special Issue Abstracts, March 1985.
EDITORIAL

We all spend a great deal of time talking about the qualities and properties of various composite materials. I suppose in part this is a direct result of the ADA position on amalgam viz a viz composites for posterior use. There is a great preoccupation in research and the profession about the strength and durability of composites in comparison to amalgam. There is also a great deal of research being done on the biocompatibility aspects of the new composite resins.

Studies to date have indicated compressive strengths of the new materials and wear resistance are coming close to or exceeding the comparative values of amalgam. Further, studies to date, at least those that I have been able to find through literature searches, show that biocompatibility is not a viable issue either.

In fact, as stated in prior issues of Bio-Probe, it appears that composite resins are becoming the material of choice. The market share of composites exceeding that of amalgam by 4% in 1983. I feel certain that 1985 data will show a further erosion of the amalgam share of the market.

There is an issue though, related to the use of composites, that is not receiving the attention it deserves. That issue is post-operative sensitivity and pain. As pointed out by Dr. Vimy in his articles in Bio-Probe, the use of composites as a restorative material is very technique sensitive. Perhaps more importantly, because so little positive information and guidance is being provided by those agencies and associations normally responsible for funding research and disseminating such information, most practicing dentists find themselves in the unenviable position of having to experiment with various products and techniques. In essence, supply-house or product detail people are the primary source of information.

Outside of Roel J. Wyman D.D.S. and his group of participating dentists, who are not formally organized, The International Academy of Oral Medicine and Toxicology is the only organization I know of that openly exchanges information and research on this subject between members, presents research findings on the subject at every meeting, and solicits member participation in resolving questions or problems brought out by any member. However, that is not enough. If the movement to replace amalgam is to maintain credibility the resolution of any patient problems related to post-operative pain or sensitivity must become the overriding consideration.

"It works for me" doesn't solve the problem unless we are able to pass the information along. Nor does that statement bear up under scrutiny in many cases. For example, many dentists I have talked to maintain that they are not having any patient problems with the materials or techniques they are using. However, upon further discussion, it is apparent that this is a subjective judgement because there is absolutely no post-operative follow up. In other words, the patient is never called that evening or during the next few days to determine if they are experiencing any pain or sensitivity. There is also the statement "You will have some pain and sensitivity to hot and
cold, but that will dissipate in a few days or weeks. If it bothers you too much, use one of the desensitizing tooth pastes and that should take care of the problem.

It is also apparent from my discussions with those dentists who have essentially solved the post-operative pain and sensitivity problems that they have effective follow-up protocols; have an ongoing dialogue with their colleges on techniques and materials; and apply and change accordingly to the benefit of their patients and their practice.

I am also the recipient of phone calls and letters from patients all over the country who have experienced these problems and are requesting information about what they should do to get rid of the pain and/or extreme sensitivity. Or, where they can go to have the problem resolved because their own dentist has not been able to help. Unfortunately, in some instances, the cure is an endo procedure and a replacement restoration of gold.

There appears to be certain basics involved in successful placements of composites:
1. A well ventilated office and operatories where established effective mercury hygiene protocols are continually practiced.
2. Proper technique for amalgam removal. Minimum drilling, high water volume through the hand piece and by the assistant (cold water only); high vacuum evacuation; patient breathing through a nose piece where nitrous has been turned off, unless the patient has opted for nitrous; proper mask and surgical gloves for dentist and staff.
3. Dry field and immediate use of a suitable base to seal the dentin. At the present time experience is showing the use of glass ionomer to be the most effective. Etching of the glass ionomer can then be done without damage to the dentin.
4. A curing light that does not exceed tooth damage temperatures. Testing of the light to insure curing depth of penetration and proper technique for curing.
5. Patient follow-up to confirm post operative success.
6. Recall of patients experiencing any unusual difficulty to resolve the problem. Discussion with knowledgable colleagues when experiencing intractable problems or when successful resolution of these problems has been effected. In this regard, Bio-Probe would welcome and disseminate this kind of information.

In an effort to provide a more scientific approach to the overall problem of determining to what degree this problem actually exists I would like to solicit your cooperation in a research project.

Each dentist willing to participate in this effort, please forward to Bio-Probe the names and addresses of 20 patients who have had one or more amalgams replaced with a composite. In addition, please indicate the material involved ie. base, cement/bonding agent, composite used.

Bio-Probe will send each individual a simple no-name response (name optional at patients discretion) questionnaire eliciting the basic information concerning post-operative pain or sensitivity and
the office protocols employed. We will then provide a statistical analysis of the results in a subsequent issue of Bio-Probe.

PLEASE ASSIST IN THIS EFFORT. THE INFORMATION DERIVED WILL ASSIST THE PROFESSION IN COPING WITH A VERY SERIOUS PROBLEM.

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MATERIALS
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In the Dec 1984 issue of the Journal of Prosthetic Dentistry, Hembree and Taylor published the results of their study on the marginal leakage of visible light-cured composites.

Two microfilled composite resins: Heliosit (Vivadent USA) and Visio-Dispers (ESPE/Premier); one small-particle conventional composite resin (Prisma-Fil (L.D.Caulk); and one composite resin with both conventional and microfilled particles Visar-Fil (Den-Mat) were evaluated. The authors also evaluated two chemically cured composites: A small-particle conventional composite Profile (S.S. White) and a microfilled composite resin Isopast (Vivadent USA). The chemically cured composites essentially showed the same results as the light-cured composites.

The authors concluded: "The results of the study indicate that the use of visible light to polymerize the composite resin has little effect on the marginal leakage of the restoration. More important is the type of composite resin. The results indicate that marginal leakage is reduced significantly or eliminated in conventional composite restorations when the cavosurface angle and the enamel are etched and a layer of unfilled resin is placed before and after insertion of the restoration. Apparently, the same technique of placement is not as effective when a microfilled composite resin is used. The study indicated that marginal leakage persists in the microfilled composite resins after 1 year whether an acid-etch technique is used or not. This is thought to be due to the higher coefficient of thermal expansion of the microfilled composite resins.

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EVENTS
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The Holistic Dental Study Club is sponsoring a one-day seminar on "Dental Amalgam Toxicity" Fact or Fiction? The presentors will be Murray J. Viny, D.D.S., F.A.G.D. and Dr. Michael F. Ziff, D.D.S. The seminar will be held at the Amfac Hotel, San Francisco Internatinal Airport on August 24, 1985. Tuition: Registration prior to 7/24, Doctors $150.00, Staff $100.00. On Site Registration: Doctors $160.00, Staff $110.00. Payment by check of Visa/Mastercard only to: Holistic Dental Study Club, c/o Frederick Aquaviva, D.D.S., One Mirada Road., Half Moon Bay, CA 94019. Telephone registrations may be made by calling 415-726-5514. The Amfac Hotel has established a
special rate of $60.00 single or double. Call the Amfac Hotel at 415-347-5444 to make your reservations.

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The first annual meeting of the International Academy of Oral Medicine and Toxicology will be held in Denver, Colorado, Friday-Sunday September 20-22, 1985. The Academies policy of allowing all members who so desire to be present during Board meetings will provide all attendees with some added insights into this outstanding group of clinicians, researchers and practitioners. The event is being billed as:

MERCURY
DENTAL, MEDICAL AND LEGAL IMPLICATIONS
A symposium sponsored by
The International Academy of Oral Medicine and Toxicology

AGENDA:
Friday, 20 September 1985

8:00 A.M. - 12:30 P.M.: I.A.O.M.T. Board Meeting/Annual Meeting.
12:30 P.M. - 2:00 P.M.: Lunch Break
2:00 P.M. - 6:00 P.M.: Scientific presentations by Academy members:
"Contribution of daily dose of mercury vapor from dental amalgams"
Murry J. Vimy, D.M.D., F.A.G.D.

"Signs and symptoms of the mercury-thyroid relationship"
Harold Utt, D.D.S.

"Type 2 diabetes mellitus and dental mercury: A possible correlation"
Laurence L. Barsh, D.M.D.

"Mercury amalgam fillings and periodontal disease"
Michael F. Ziff, D.D.S.

"Mercury amalgam fillings and the immune system response"
Robert O. Wolf, D.D.S.

Saturday, 21 September 1985

8:00-10:00 A.M.: "Overview and update of the mercury amalgam controversy" Murray J. Vimy, D.M.D., F.A.G.D. and Michael F. Ziff, D.D.S.

10:00-10:15 A.M.: Break

10:15 A.M.-12:15 P.M.: "Mercury vapor release from dental amalgam"
Carl W. Svare, D.D.S., Ph.D.

12:15-2:00 P.M.: Lunch Break

2:00-3:45 P.M.: "Overview of heavy metal toxicity" Earl Snyder, Ph.D.

3:45-4:00 P.M. "Chelation and detoxification" Willam E. Doell, D.O.
Sunday, 22 September 1985

8:00-10:00 A.M.: "Medico-legal aspects of the amalgam controversy" Robert E. Reeves, J.D., Aaron J. Rynd, B.A., Ph.D.

10:00-10:15 A.M.: Break


11:45 A.M.-12:00 P.M.: Break

12:00 P.M.-2:00 P.M.: Panel Forum to discuss questions from the floor.

REGISTRATION

No registration required for Friday session.
Saturday and Sunday sessions:

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FORUM

In December 1984 I sent a letter to Secretary Margaret Heckler, Health and Human Services, bringing the mercury/amalgam issue to her attention. In February, 1985 I received a response indicating that the July 1984 NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry had concluded that dental amalgam was a safe dental material. On April 18, 1985 I sent a copy of the I.A.O.M.T. Critique to the Asst Secretary for Health and Human Services. The following reply was received:

"Your letter of April 18 addressed to Dr. James O. Mason expressing concern that there is no single primary pathological study proving the safety of amalgam, has been forwarded to the National Institute of Dental Research (NIDR) for reply.

It is not surprising that you do not find studies cited in the literature. As you know, scientists and clinicians do not customarily conduct studies to prove the safety of a substance. Their approach is usually the converse; they conduct studies to screen for any deleterious effects of a substance. Your are aware, I am sure, that there are isolated reports in the literature about deleterious effects of amalgam mercury, but none of the reports, to my knowledge, have
received scientific substantiation outside of those related to hypersensitivity.

The NIDR encourages scientists and clinicians to submit applications for support of research on this subject. Indeed, the National Institutes of Health's Guide to Grants and Contracts soon will publish and announcement to remind scientists of the NIDR's continuing interest in research in this area.

Thank you for providing me with the copy of the International Academy of Oral Medicine and Toxicology report.

Sincerely yours,

Signed Harald Loe, D.D.S., Dr. Odont.
Director
National Institute of Dental Research

Roel J. Wyman, D.D.S., author of the book "The Posterior Composite Resin Restoration" is now publishing a newsletter on the subject called "The PCR Reporter". Rather than continually revise and update his book Dr. Wyman has elected to publish a regular letter that will keep all subscribers up to date with developments in the field of posterior composite restorations and dentin bonding, as they happen.

It looks like a real winner. The first issue which I have just read addresses the same issue covered in the Bio-Probe Editorial and additionally provides some outstanding technical advise. Dr. Wyman is also doing a sensitivity survey.

2 year charter subscriptions to The PCR Reporter are available until July 15, 1985 at the reduced price of $110.00. Normal one year subscriptions will be $68.00. You may pay for your subscription by check or master card/visa. Mail orders to Maxplax Limited, 7084 Airport Road, Mississauga, Ontario, Canada. Or they will accept telephone orders at 416-677-9362. I don't see how any practitioner can get by without the kind of information being provided.

Copies of the I.A.O.M.T. Critique "A critical evaluation of the NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry" are available through Bio-Probe. The price is $7.00 for non-I.A.O.M.T. members and $5.00 for I.A.O.M.T. members. Please make checks payable to I.A.O.M.T.