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**Dental Amalgam -- To Be or Not To Be**

Since the 1800’s, dental amalgam has been the most commonly used restorative material in dentistry. However, the mercury content of dental amalgam continues to create a controversy regarding its safety for patients and dental personnel. Each year, dentistry in the United States uses over 100 tons of mercury.

A recent review of the literature by Langan¹ clearly states the position of the ADA Council on Dental Materials, Instruments, and Equipment:

"Occupational exposure to mercury is a potential hazard for dental personnel, but is completely preventable with the implementation of proper mercury hygiene practices. There is no evidence in the scientific literature that the minute amounts of mercury vapor that may be released from amalgam restorations cause mercury poisoning. Allergic reactions to mercury and other constituents of amalgam have been documented, but are exceedingly rare. The association between allergies and oral lichen planus requires further investigation. Finally, dental amalgam, which has been used extensively for more than 100 years, has an exemplary record of safety and benefit to the dental patient."

The position does not reflect the concern generated by recent additions to the literature on dental amalgam.

The following editorial is a critical review of the literature regarding:

1. the risk of toxic accumulation of inorganic mercury in dental patients.
2. the risk of hypersensitivity to inorganic mercury in dental patients.
3. the risk of toxic accumulation of inorganic mercury in dental personnel.
4. the risk of hypersensitivity to inorganic mercury in dental personnel.
5. the hazardous waste disposal of mercury.

**The risk of toxic accumulation of inorganic mercury in dental patients:**

Relatively recent research has demonstrated, beyond reasonable doubt, that dental amalgam contributes to the patient’s systemic burden of mercury.

Svare et al.² demonstrated that minute amounts of mercury are continuously released from dental amalgam...
restorations in humans and that the release is accelerated 15-fold in expired air immediately after mastication.

Abraham et al.\textsuperscript{3} reported a correlation of inorganic mercury levels in the blood of humans to the total surface area of occlusal dental amalgam. Subjects with dental amalgam had twice the inorganic mercury content in the blood as controls without dental amalgam and the amount of inorganic mercury in the blood in the subjects was enhanced eight-fold immediately after mastication.

"The World Health Organization has recommended that the exposure of women of child-bearing age to mercury vapor should be as low as possible because elemental mercury readily passes the placental barrier."\textsuperscript{4}

The placement of dental amalgam in women of childbearing age is a legitimate concern because of a possible unknown pregnancy. The question of the teratogenic effect of elevated inorganic mercury in the blood for many years after the placement of dental amalgam has been raised by Abraham et al.\textsuperscript{3}.

Macdonald\textsuperscript{5} stated that "mercury is a teratogenic agent and should be avoided by pregnant women. Since damage to the fetus is most likely to occur early in pregnancy, particularly during the first 8 to 10 weeks, consideration should be given to eliminating all exposure prior to conception."

Schiele et al.\textsuperscript{6}, Friberg et al.\textsuperscript{7}, Eggleston et al.\textsuperscript{8}, and Nylander et al.\textsuperscript{9} have reported separate studies demonstrating a positive correlation between the dental amalgam restorations and mercury levels in human brain tissue. On the average, dental amalgam contributes approximately 10 ng of mercury per gram of brain tissue, resulting in approximately a two to three fold increase in total mercury over controls without dental amalgam.

The toxic levels of mercury in human tissues have not been sufficiently investigated and the amount of mercury in human brain tissue from dental amalgam may or may not be clinically significant.

There are two sides to this issue:

1) it is safe for dentistry to double or triple the mercury level in the brains of patients until it is scientifically established that this practice is harmful; or

2) the elevation of mercury in systemic tissues among dental patients with dental amalgam restorations represents a legitimate concern until the safe levels of mercury in human tissues are scientifically established well above the levels attributed to dental exposure.

The risk of hypersensitivity of inorganic mercury in dental patients:

Clinical observations of overt allergic reactions (hives, edema, urticaria, etc.) to dental amalgam are reported in the literature,\textsuperscript{10-35} but are relatively rare. The incidences of these types of reports are diminishing, possibly because of the reluctance of editors to publish "case reports" and possibly because the decrease of mercury from mercurochrome and merthiolate has diminished these products as sensitizing doses of mercury to adolescents.

The stated position of the ADA is that far less than 1\% of the population is hypersensitive to mercury. Yet, an exhaustive research of the literature and requests directed to the ADA have failed to find published research documenting this figure.

Djerassi et al.\textsuperscript{36} patch tested 240 people with dental amalgam. Of the 180 subjects with amalgam restorations, 16.1\% exhibited a positive patch test and of the 60 controls with no amalgam restorations, there were no positive patch tests. Time may be a factor, as positive tests were found in 5.8\% of people with up to 5-years-old restorations, while the percentage in persons whose restorations were older was 22.5.

Miller at al.\textsuperscript{37} found a significant correlation between the number of amalgam restorations and the incidence of positive patch tests to mercuric chloride:

1. 10 of 39 dental students with 0 to 4 amalgam restorations ha a positive patch test (25.6\%).
2. 17 of 71 dental students with 5 to 9 amalgam restorations had a positive patch test (23.9%).
3. 27 of 61 dental students with 10 or more amalgam restorations had a positive patch test (44.3%).

The presence of 10 or more amalgam restorations doubled the incidence of positive patch tests. Statistically, the chi-square value showed the results to be significant (p<0.050).

Freshman dental students prior to mercury exposure as dental personnel, were considered representative of the general population. Overall, 51 freshman dental students showed 31.4% positive patch tests to mercuric chloride.

Newman considers a positive patch test with mercuric chloride to be a chemical burn of the skin and not a true hypersensitivity. However, this theory does not explain the correlation of dental amalgam and positive reactions. Either dental amalgam creates hypersensitivity to mercuric chloride patch tests or dental amalgam makes people more susceptible to chemical burns by mercuric chloride. In either event, research by Djerassi and Miller concur that amalgam is doing something to the human immune system. To my knowledge, there are no published reports of research contradicting this.

The possibility of dental amalgam adversely affecting T-lymphocytes is still under investigation.

The risk of toxic accumulation of inorganic mercury in dental personnel:

Langan and others have stated that occupational exposure to mercury in dental personnel is "completely preventable". In reality, occupational exposure to mercury is "completely inevitable". Mercury vaporizes at room temperature, therefore, any dentist that places or removes dental amalgam will inhale mercury vapor.

Occupational exposure has occurred and continues to occur. Approximately 14% of dental offices in the United States have mercury vapor levels above 50 µg/m³, some as high as 180 µg/m³.²⁻⁰,⁴⁻¹,⁴² Fifty µgHg/m³ is the threshold value limit (TLV) recommended by the National Institute for Occupational Safety and Health (NIOSH). The Occupational Safety and Health Administration (OSHA) could close 14% of the dental offices in this country. Some dental offices are so heavily contaminated with mercury that shoes and clothing worn home by the dentist have elevated entire homes up to 30 µg/m³ of mercury vapor.⁴⁰

"Recent studies with pregnant women indicate that elemental mercury does cross the hemochorial placenta and incorporates into the fetus. Thus, the placenta, the chorioallanoic membrane, the amniotic membrane, and the neonatal blood of women who were exposed to mercury while working in dental offices were found to contain significantly higher mercury levels than in control women with no occupational exposure to mercury".

Bloch et al., reported that epidemiological data collected in the United Kingdom suggested that, when compared with control groups, female dentists have a higher abortion rate, a raised incidence of premature labor, and possibly an elevated perinatal mortality.⁴⁴

Shapiro, et al.⁴⁵ and Ship II, et al.⁴⁶ reported the relation between cumulative exposure to mercury and chronic health impairment. "298 dentists had their mercury levels measured by an X-ray fluorescence technique. Electrodiagnostic and neuropsychological findings in the dentists with no detectable mercury levels. 30% of the 23 high mercury dentists had polynepathies. No polynepathies were detected in the control group. The high mercury group had mild visuographic dysfunction; they also had more symptom-distress than did the control group. These findings suggest that the use of mercury as a restorative material is a health risk for dentists."

There are numerous case studies of dental personnel and industrial personnel with high mercury measurements and associated polynepathies.⁵⁻⁰,⁴⁻¹,⁴⁻⁴⁻⁵⁻⁸

As an example, Mantyla's⁴¹ report of 2 dentists with mercurial toxication. "The basic symptoms were numbness, pain in the limbs and neck, a heavy depressing fatigue, diminished reflexes, metallic taste, and
tremors. The dental office was contaminated with initial ambient air levels as high as 0.150 mg Hg/m³. After decontamination of the office, many of the symptoms subsided, although complete regeneration generally does not take place."

Also typical is Smith's²³ report. "Three dentists suffered symptoms of mercury poisoning from the use of this substance in the production of amalgam for dental fillings.

Patient 1, symptoms included marked tremor of the hands, loss of motor control, indifference toward family and friends, and some visual disturbance. Urine mercury level was 300 ug/L. Penicillamine treatment reduced urine Hg to 38 ug/L and patient became symptom free.

Patient 2, symptoms included weakness, digestive disturbance, diarrhea, nervous tension, red palms, increased salivation, metallic taste, and marked tremor of his hands. Urine level was 48 ug/L. Treatment with dimercaprol (BAL) increased the urine level to 211 ug/L, which was taken as evidence of the rapid excretion of mercury. Subsequent mercury levels were normal and his symptoms cleared.

Patient 3, symptoms included a maculopapular rash on the lower legs, muscular weakness, slurred speech, ptosis of the left eyelid, redness of the palms, obesity, extreme tremor of the hands which prevented legible writing. Urine test showed 86 ug/24 hours, which is above the upper limit of normal (30 ug/24 hour specimen). This patient was given no drugs, but the environment of the office was cleaned. The maculopapular rash cleared in two months, and by that time the level of mercury in the urine had dropped by half. After nine months no mercury was found in the urine, and tingling of fingers and toes was the only symptom present."

The most common symptoms of poisoning are: excitability, especially when criticized; inability to concentrate; fearfulness; depression; headache; fatigue; weakness; loss of memory; drowsiness or insomnia; symptoms of kidney disease; and tremors of the hands, head, lips, tongue, or jaw. The tremors may affect handwriting and, as poisoning progresses, the writing becomes illegible.⁴⁰

Diagnosis of mercury intoxication is extremely difficult because of the insidious nature of the onset of symptoms. Macdonald⁵ stated, "Since symptoms vary greatly, improper diagnosis may result. Failure to consider mercury as a causative factor in digital numbness resulted in two exploratory surgical procedures for a 40-year old dentist. He was treated in several prestigious medical facilities for 16 years before a 'long shot' test for urine mercury was taken."

Cook et al.⁵⁸ reported that a 42-year old dental assistant with at least a 20-year history of exposure to mercury developed a rapidly fatal nephrotic syndrome. The high levels of mercury in the kidney, estimated by neutron activation analysis and demonstrated histologically, indicate that this was the result of mercury intoxication. The warning is given that the risks of exposure to mercury in the dental operatory are not trivial.

"The amount of mercury present in the kidney was considerable. This was probably accumulated over twenty years. Most dental assistants do not practice their profession for such an interval of time. This is the first reported case of fatal mercury intoxication in a dental assistant. Possibly the relative brevity of the dental surgery assistant's career has effected this, but more probably evidence of mercury intoxication has not been sought. Because of the wide range of normal behavior, irritability, excess salivation or tremor might well pass unnoticed."

Urinary mercury levels for 4,272 U.S. dentists who participated in the Health Assessment Programs held at the ADA annual sessions (1975-1983) had a mean level of 14.2 ug/L with a range of 0 to 556 ug/L. Urinary levels were statistically related to the following variables: geographic location; type of practice; years in practice, in office, and in specialty; hours of practice per week; number of restorations placed; method of amalgam preparation and mercury expression; type of amalgam capsule used; and type of heating and cooling system.

In general those dental practices associated with higher exposure risk were related to increased urinary
mercury excretion levels. There is an increasing trend in the mean urinary mercury levels as the age of the office, age of the practice, and age of the dentist increase. Participants in general practice had the highest urinary mercury levels (15.3 ug) and orthodontists showed the lowest levels (3.9 ug)\(^{59}\).

The average range of mercury in the urine of the general population is 0 to 5 ug/L with a level greater than 20 ug/L considered abnormal. The Centers for Disease Control has published an opinion that 30 ugHg/L is the maximum acceptable level.\(^{60}\) Berlin\(^{61}\) found that 50 ugHg/L of urine is the level associated with induced tremors (tremors with a 15 lb weight on the forearm). 100 ugHg/L of urine is the level generally considered to cause outright tremors.\(^1\)

Using the results of the ADA of 4,272 dentists:

1. 19.1% are over the maximum normal measurement of 20 ugHg/L (extrapolates to approximately 29,500 dentists).
2. 10.9% are over the C.D.C. maximum acceptable level of 30 ugHg/L (extrapolates to approximately 16,500 dentists).
3. 4.9% are over 50 ugHg/L, the level found to cause induced tremors (extrapolates to approximately 7,500 dentists).
4. 1.3% are over 100 ugHg/L, the level found to cause tremors (extrapolates to approximately 2,000 dentists).

The Centers for Disease Control recently reported that 22 students and a teacher in a Connecticut high school chemistry laboratory were accidentally exposed to mercury vapor during an experiment on December 8, 1986.\(^6\) The maximum concentration of mercury in the air was 50 mg/m\(^3\). "On December 11, urine samples were obtained from the 23 persons who were in the classroom during the experiment. Eight persons had urine levels of mercury at or above 30 ug/L, the maximum level considered acceptable. On January 20, 1987, repeat tests showed that six of the eight students still had urine mercury levels above 30 ug/L. School officials decided to have follow-up testing performed on the remaining 15 persons in the class. The urine mercury level for all but one of these 15 persons had increased from the original values, and some had risen to 30 ug/L or above. The highest level was 72 ug/L. Testing of a control group to determine the normal average urine mercury level for unexposed students at the school was also requested. However, school officials did not allow control samples to be obtained. Additional follow-up testing was conducted on February 24, 1987 and again on March 31, 1987. On February 24, 1987, everyone in the class, including the teacher, had a mercury level either at or below 30 ug/L. On March 31, 1987, one student had a mercury level of 37 ug/L; all others remained at or below 30 ug/L.

The Centers for Disease Control found it necessary to follow these 22 students and teacher until the urine level of mercury as at or below 30 ug/L. However, data from the ADA indicates that 16,500 dentists (and undoubtedly a proportionate number of dental assistants) have urine levels of mercury above 30 ug/L.

The comment is often made that the use of mercury in dentistry is safe because of the normal health of dentists who obviously get more exposure than patients. However, it is common knowledge in the insurance industry that dentists have one of the highest utilization rates of medical insurance. This may have nothing to do with mercury. However, to state that dentists have a normal level of health is contrary to Shapiro’s research, the ADA’s urine sample project, case reports of mercury poisoning of dentists, and the dentist utilization of medical insurance.

### The risk of hypersensitivity to inorganic mercury in dental personnel:

White\(^{62}\) patch tested dental students to mercuric chloride and silver amalgam to determine if the rate of hypersensitivity to mercury increased as they were exposed to silver amalgam during dental school. A statistically significant increase was found in the rate of mercury hypersensitivity from the prefreshman to
the senior class. Prefreshmen were 2.0% positive, sophomore were 4.1% positive, juniors were 10.3% positive, and the seniors were 10.8% positive.

Miller\textsuperscript{37} did not find a statistically different level of hypersensitivity to mercury between freshman and senior dental students. He states that the use of amalgam capsules has reduced the skin contact with mercury among dental students and the incidence of sensitization has dropped since White's study.

The risk of hypersensitivity to inorganic mercury in dentist personnel with additional years of clinical exposure to dental amalgam has not been sufficiently investigated.

**The hazardous material disposal of mercury:**

Dental amalgam is classified as a hazardous material by OSHA\textsuperscript{63}, and excess dental amalgam must be disposed of according to its Material Safety Data Sheet. If the exact amount of dental amalgam could be mixed for each restoration, all of the amalgam could be placed in the patient's tooth. Invariably though, there is excess to be rid of. The ADA recommends the following:\textsuperscript{64}

"All amalgam scraps should be salvaged and stored in a tightly closed container. The scrap should be covered by a sulfide solution such as X-ray or photographic fixer solution."

"A no touch technique of handling amalgam should be used. Skin that is exposed to mercury should be cleaned. Precapsulated alloy should be used. Water spray and high-volume evacuation should be used when removing old or finishing new dental restorations. Evacuation systems should be passed through filters, strainers or traps. A face mask should be used to avoid breathing amalgam dust."

Proper disposal of scrap amalgam is essential. Recently the ADA News\textsuperscript{65} reported a case of a metal recycler using a heat distillation process. "This extraction process, the EPA said later, caused some of the mercury to vaporize and settle downstream from the recycling site. Children playing with a metal detector later discovered the mercury in the ground and reported it to authorities. The EPA then conducted the $710,000 cleanup of the two sites."

"Under existing environmental laws, the EPA can charge anyone who 'arranges for' the disposal of 'hazardous' material for payment of costs associated with any cleanup required, said Mary K. Logan, ADA associate general counsel."

"On this basis, in June 1987, the EPA launched its investigation, later deciding to pursue claims against the dentists, the metal recycler, and the four dental suppliers. The agency claimed that the dentists and dental suppliers could be held responsible under federal environmental laws simply because mercury the metal recycler purposefully extracted from their scrap amalgam was found at the two sites."

"The 58 dentists together are to pay $69,812 in damages to cover about 10% of the cleanup."

Dental amalgam contains elemental metallic mercury while mercury in the food chain is in the more toxic organic form (elemental mercury combined with a methyl or ethyl radical). The vast majority of mercury in blood is organic. However, organic mercury has a shorter transit time in the body and therefore, 75-80% of the mercury accumulated in organ tissues such as the brain is inorganic.\textsuperscript{9} Even though organic mercury is more toxic, the pathogenetic potential of inorganic mercury must be considered.

A few months ago I was faced with a moment of truth. My 15 year old daughter, Jaime, presented with the need of occlusal restorations on eight molars. Should I use dental amalgam and

1) expose myself and staff to mercury vapor knowing that approximately 7,500 U.S. dentists have urine levels of mercury capable of producing tremors;\textsuperscript{59,61}

2) create the need for disposal of excess amalgam knowing that amalgam is classified as a hazardous material by OSHA and improper disposal is a risk to the community;\textsuperscript{63,65}

3) turn the occlusal surfaces of her molars black from corrosion of a base metal alloy:
4) likely alter her immune system knowing that 10 amalgam restorations increase the incidence of positive patch tests to 44%;

5) probably triple, and possibly quadruple or more the total mercury level in her brain and kidneys; and

6) likely double the level of a known teratogenic substance in her blood for decades?

I think not.

David W. Eggleston, D.D.S.
Editor (Pacific Coast Society of Prosthodontists Newsletter)

References:


43. Editor: This is only aest... J Calif Dent Assoc 12:37, 1984.


**REVIEW/ABSTRACTS**


**SUMMARY:**

We will all be hearing more about risk assessment because of the public demand for safety and health policies that are based on clearly understandable reasoning. Dr. Reinhardt in this paper has attempted to bring together the quantitative methodologies used to evaluate environmental risks and to utilize these objectively to estimate the safety of dental amalgam restorative therapy. This evaluation is based on existing knowledge of mercury exposure from dental amalgams and the potential of mercury to cause adverse health effects. The need for attempting a safety assessment of dental amalgam is aptly stated by the author "This controversy has grown beyond the confines of the dental profession itself and is becoming an emotional public health issue. In hope of regaining good health, many dental patients with chronic systemic diseases are considering replacement of their amalgams. Dentists are increasingly being challenged to prove the safety of amalgams."

The equation for risk determination involves calculating an estimated margin of safety (MOS) by dividing the experimental no-observable-effect level (NOEL) by the estimated daily human dose. Applying the data available to him the author concludes that the margin of safety for mercury toxicity in humans from dental amalgams is approximately 8- to 30-fold.

The article concludes that the assessment of the safety of dental amalgams is hampered by several limitations in current knowledge. Among them he brings up the point that most existing studies of industrial exposure did not consider dietary intake, intake from air away from the workplace or intake from dental amalgam and that for some individuals who have above-average background exposures, "mercury from dental restorations may add the critical margin of exposure that leads to toxicity. More complete measures of total mercury exposure are needed." Another limitation is that the dosage of mercury related to the number of amalgams is not clearly defined and that "until more precise data are available, including blood levels related to number of amalgams, it will not be possible to tell whether actual exposures are higher or lower than estimated here." A further limitation is that the proportion of elemental mercury that becomes methylated in humans is unknown which is a factor that could lower the margin of safety. Dr. Reinhardt concludes "The answer to the question of dental amalgam safety is neither simple nor clear, and further study is warranted to address this important issue."

**BIO-PROBE COMMENT:** We think Dr. Reinhardt is to be congratulated for attempting to bring some scientific methodology into the amalgam safety controversy. It is certainly a far cry from the calculated and carefully crafted establishment statements concerning the safety of dental amalgam, which cannot be supported by one basic scientific study. We would hope that future use of the "risk assessment" model would include data from major studies that were excluded from consideration or have been published subsequent to the 7/29/87 acceptance for publication date of this study. For example, the brain mercury/amalgam filling correlation work of Friberg, Nylander and Eggleston; Clarkson's work showing mercury vapor passes the placental membrane; Malamud et al. showing low levels of mercury inhibit the
respiratory burst of human polymorphonuclear leukocytes; the Clarkson and Friberg work on the predicted intake of mercury from dental amalgam and the work of Britt Ahlrot-Westerlund showing greatly increased mercury levels in the spinal fluid of MS victims. Correspondence and print requests to the author, currently Robert Wood Johnson Research Scholar, c/o Dept of Dental Care Administration, Harvard School of Dental Medicine, 188 Longwood Ave, Boston, MA 02115.


ABSTRACT:

The intracellular free thiol, GSH, functions in numerous cytoprotective roles that include: detoxification of xenobiotics, removal of reactive oxygen species, maintenance of protein thiol status, and modulation of enzyme activity via disulfide interchange. Further, GSH has been hypothesized to serve as an intracellular mediator in cell cycle progression, with reported levels of GSH varying during the cell cycle. The purpose of the study was to further clarify the role of GSH in cell cycle kinetics (i.e., % of populations in G1, S, G2(M) were assessed via flow cytometric evaluation of propidium iodide stained nuclei. In addition, the dual beam spectrophotometric kinetic assay of Eyer et al was used to determine total GSH & % as the disulfide, GSSG. Proteins were assessed via the Lowry method. Concurrent determination of GSH levels with cell cycle kinetics showed that GSH levels ranged from 13.29-51.62 nmol GSH/mg protein, and correlated with cell cycle distribution, i.e., highest GSH levels were noted when a majority of cells were in S phase (n=20). Further, GSH depletion (via a combined protocol of buthionine sulfoximine, an inhibitor of GSH synthesis, and 2-cyclohexene-1-one, a specific depletor of GSH) was shown to significantly prevent cell cycle progression from G1 to S, the DNA synthesis phase (n=8, p, Mann Whitney U). These results imply that GSH may play an integral role in cell cycle progression, perhaps via maintenance of function of thiol dependent DNA synthesizing enzymes, such as polymerase and ribonucleotide reductase.

BIO-PROBE COMMENT: More and more research is indicating that glutathione is critical to a myriad of functions in the human body. The fact that mercury and other heavy metals deplete glutathione should receive more attention from researchers and physicians.


ABSTRACT:

Pocket depth bleeding on controlled-force (40g) probing and attachment level were measured at baseline and at 30 and 60 days in 113 subjects from 5 centers who had 4 nonadjacent teeth with 6-10 mm pockets that bled on probing. The four teeth in each subject were randomly assigned as follows: TC fiber, control fiber, untreated, and scaling. TC fiber therapy resulted in a reduction in pocket depth (1.04 + 0.09 mm at 60 days) and bleeding on probing (51% at 60 days, as compared to 95% at baseline). These changes were significantly greater than in other test groups. Attachment gain in the TC fiber treated teeth was 0.64 mm + 0.12 mm at both 30 and 60 days. Scaling resulted in a 0.76 + 0.09 mm reduction of pocket depth; 75% of sites bled on probing at 60 days. Pocket depth reduction by scaling was significantly greater than at untreated sites at 60 days but significantly less than that achieved by TC fiber. Bleeding on probing was not significantly affected by scaling. Attachment gain by scaling was 0.35 + 0.12 mm at 30 days and 0.43 + 0.12 mm at 60 days. A slight tendency toward added clinical improvement was noted at control fiber test sites where pocket depth reduction was 0.61 + 0.10 mm at 60 days. Untreated sites also significantly improved with a pocket depth reduction of 0.48 + 0.09 mm at 60 days, which was interpreted as the
response to prophylaxis and improved homecare. The mean plaque index of subjects prior to prophylaxis was 0.83 and remained constant at approximately 0.5 throughout the experimental period. These results indicate that compared with scaling and control groups, TC fiber therapy resulted in greater pocket depth reduction, attachment level gain and reduction in bleeding on controlled-force probing.

Tanner A., McArdle S., and Goodson JM (Forsyth Dental Center, Boston, MA) Multi-center evaluation of tetracycline fiber therapy. V. Microbial effects.

ABSTRACT:

This study examined the effect of local periodontal treatments on the subgingival presence of 6 suspected periodontal pathogens: B. gingivalis (Bg), B. intermedii (Bi), W. recta (Wr), F. nucleatum (Fn), E. corrodens (Ec), and A. actinomycetemcomitans (Aa).

113 adult subjects were selected from 5 different states in the U.S. (CA, MO, GA, NY, MA). Plaque samples were taken from deep periodontal pockets before and following therapy, in a study of 4 test groups: TC fiber, control fiber, untreated and scaling. Samples were stored frozen prior to assay using oligonucleotide DNA probes.

Pre-treatment, 96% subjects were infected by Fn, 92% by Bg, 86% by Bi, 86% by Ec, 74% by Wr, and 27% by Aa. There was a very similar distribution of species at each of the treatment centers. The average detection limit by the assay was 2.9 x 104 organisms representing approximately 0.3% of the total microbiota. Percent reduction of sites with detectable infection following TC fiber therapy was for Aa 55%, Bg 42%, Bi 71% Ec 55%, Fn 68%, and Wr 83%. Comparable reductions were obtained after scaling. Pocket depth reduction at sites where species were successfully reduced to undetectable levels was greater following TC fiber therapy than with scaling.

Monitored species were isolated from all 5 geographic locations of the U.S. TC fiber therapy and scaling reduced levels of monitored species. Pocket depth reduction was greater when the monitored species were suppressed by TC fiber therapy.


ABSTRACT:

This pilot study examined possible synergistic effects of combining scaling with tetracycline (TC) fiber therapy (Goodson et al. 1983) for treatment of adult periodontitis. Eight subjects (5 male and 3 female, 30-63 years old) were randomly assigned to either fiber therapy alone (full mouth) or half-mouth combined therapy or scaling, forming 3 treatment groups. Sites selected for treatment exhibited bleeding on probing and attachment loss -3 mm. Attachment level change was the principal clinical response variable. Data were analyzed on a site basis. One randomly selected site in each patient received no therapy. All patients were monitored at 1 and 3 months following therapy; 5 patients were also monitored at 6 and 12 months. No treatment was given during the monitoring period.

Both TC fiber therapy and combined therapy produced significant attachment gain for 6 months (0.7 mm and 0.9 mm respectively). Scaling alone resulted in a nonsignificant attachment gain for 6 months. The principal difference between combined therapy and either TC fiber or scaling alone was in persistence of the clinical response. At 12 months combined therapy produced 0.9 mm attachment gain. TC fibers alone produced 0.2 mm attachment gain, and the scaling response had deteriorated to pre-treatment levels. These results suggest that the combination of TC fiber therapy and scaling may result in prolonged attachment gain as compared to either use alone.
NOTE: All of the above TC fiber evaluations utilized an intrapocket tetracycline (TC) fiber delivery system manufactured by ALZA Corp., Palo Alto, CA.

FORUM

The American Academy of Biological Dentistry is presenting the first United States Seminar on: Neural Therapy, Reflex Zones and Somatotopies: A key to the diagnostic and therapeutic understanding of man's ills. Presenters are Jochen Gleditsch, M.D., D.D.S. and Franz Hopfer, M.D., D.D.S. The seminar will be given June 27-30, 1989 at the Carmel Mission Inn, Carmel, California. For more information call (408) 659-5385 or (209) 838-3522.

The program for the 1989 Annual Scientific Meeting of the International Academy of Oral Medicine and Toxicology has had some exciting additions since our initial announcement in the March issue of Bio-Prob. The Academy is proud to announce that Dr. Mats Hanson and Dr. Magnus Nylander will both be making presentations. Dr. Hanson will be providing us with the latest research on free radicals and Dr. Nylander will be talking about his latest research projects regarding the relationships of selenium and mercury. The meeting will be held in Detroit, Michigan September 15-17, 1989 at the Somerset Inn. IAOMT special room rates are $70.00 (single or double occupancy) and $90.00 for an Executive room. In addition, contractual arrangements have been made with Northwest Airlines for a 40% reduction in normal coach fares and a 5% reduction on Supersaver fares. As usual, the meeting promises to provide the latest scientific data available on many different subjects: Trevor Lyons, LDS, Fungi & protozoan and their role in periodontal disease; James Masi, Ph.D. Bio-engineering factors in dentistry; Bob McMahon D.D.S., M.S. Atypical facial pain caused by residual bony defects "Alveolar bony cavitational osteopathisis"; David Kennedy D.D.S. OSHA Guidelines in relation to the dental environment; Michael F. Ziff, D.D.S. Update on the scientific literature; and last but not least Murray J. Vimy, D.M.D. will update on the tremendously important research on the safety of mercury amalgam being done by he and his colleagues at the University of Calgary Medical School. For more information please write to IAOMT, P.O. Box 458, Ortonville, MI 48462, Attn: Sandy, or call her at Dr. Regiani's office (313) 627-4934.

The 12th Annual National Dental Seminar in Homeopathy will be held October 20-22, 1989 at the Oak Brook Hills Hotel and Conference Center in Oakbrook, IL. For further information please write National Dental Seminar, P.O. Box 123, Marengo, IL 60152.

There are a number of Hyperthermia Detoxification Clinics around the country. Most will test your sweat and fat cells for the presence of toxic chemicals and use hyperthermia techniques to remove the toxins from your body. Lupus patients with known or suspected exposures to toxic substances who have undergone this type of detoxification have had good results. There are some cautions for the use of Hyperthermia Detoxification i.e., Individuals with type I (juvenile onset) diabetes, active kidney disease, acute hepatitis, anemia, uncontrolled hypertension, or severe heart disease should not undergo hyperthermia detoxification.

Center for Environmental Medicine. Allan Lieberman, M.D. 7510 North Forest Dr., Charleston, SC 29405-4297. (803) 572-1600.


Human Environmental Medicine, Inc. 6836 Alvarado Court, #326, San Diego, CA 92120. (619) 583-5865.

dba Environmental Health Center. 8345 Walnut Hill Lane, #205. Dallas, TX 75231. (214) 368-4132.

Theron Randolph, M.D., 505 N. Lakeshore Dr. #6506, Chicago, IL 60611. (312) 828-9480. (The above information on Hyperthermia Detoxification was taken from the L.E. Beacon Vol 5(2), Jan/Feb 1989. published by the L.E. Support Club, 8039 Nova Court, North Charleston, SC 29420.)