AMALGAM CLASS ACTION SUIT IN CANADA

The class action lawsuit against mercury/silver amalgam dental fillings has survived its first legal challenge. Lawyers for the defendants (Health Canada and several amalgam manufacturers) filed for dismissal of the lawsuit on 23 November 1998, on the claim of “failure to demonstrate a reasonable cause of action.”

At the beginning of the day, the lawyers for the amalgam manufacturers withdrew from the motion, leaving Health Canada lawyers to argue the case, vigorously for over three hours. The argument was that Health Canada’s actions on amalgam constituted a “Policy Decision” rather than an “Operational Decision” and, therefore, no duty of care is obliged to the public!

The judge was unconvinced! He dismissed the motion. The case is now expected to go before the courts for Certification under the “Class Proceedings Act” early in 1999.

A representative for Canadians For Mercury Relief, an organization actively promoting public education on mercury fillings, questioned the argument of the Health Canada lawyers, stating: “Why is our Health Minister, an obvious policy maker, making promises through the media if he has no duty or obligation to protect the public. Why doesn’t he get up and tell the media and the Canadian public that government policy provides that he has no duty to protect us?”

For more information, contact: Wayne Obie, Media & Public Relations, Talk International; T: (416) 410-6314; Email: communications@talkinternational.com.

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DOES IT HAVE HARMFUL SIDE EFFECTS??

** Does it Work?? ** Does it have harmful side effects??

These are the two questions that must be asked when recommending the use of any medical therapy! Biological doctors have asked these questions about mercury/silver amalgam dental fillings. Do they work? Yes they do! However, the scientific research provides sufficient evidence of potential for harmful side effects to argue against their use. Efficacy, economy, and ease of use are not valid reasons to ignore the possibility of adverse health effects. Consideration of suitable available alternatives demonstrating fewer, or preferably no potential adverse effects is not only essential, but is the foundation of Biological Dentistry.

Physicians normally practice and prescribe medication under the protection of FDA approval, with
research and known side effects readily available in the PDR. Dentists also operate under this protective umbrella. Even under this protection there were over 100,000 prescription drug related deaths one year, as reported in a recent JAMA article. Therapy still remains judgmental and a risk assessment call on which products to use and prescribe for patients.

Health care providers must be aware of the potential risks, as well as the documented benefits of therapy provided. For example, British Anti-Lewisite (BAL) is a very effective agent for removing mercury from the body, but has severe side effects. DMPS and DMSA are less effective and do have potential side effects, but these effects are less frequent and less severe than those from BAL. Risk-benefit comparisons, based on valid documentation, favor the use of DMPS or DMSA over BAL.

The point being addressed is the recommendation (and/or sale) of products that may or may not have any valid documentation supporting their use. The two vital questions MUST be asked for any therapy utilized by biological doctors. The potential for trouble was underscored by a recent article in a Florida newspaper. Scientists have been searching for, and finally found, the cause of numerous deaths of turtles in Florida. The cause is severe liver damage caused by a species of Blue-Green Algae! Many doctors are now prescribing or recommending the use of different algae. There are a myriad of different algae. Some are more toxic than others; some have adequate research supporting their use in humans; many have no valid documentation supporting safety or efficacy. The fact that there are many species of algae, and not all have the potential for severe harm, does not relieve doctors of the responsibility for knowledge on materials that they do use. Ask the two vital questions: Is there valid, independent (not by manufacturer or seller) documentation demonstrating that it works in humans? Is there valid independent documentation on its potential for adverse effects in humans? Health care of patients demands special care, morally and legally.

To illustrate the point, consider a hypothetical example of the use of a hypothetical algae, Asparagustus Schmidlapia ("AS"). You attend a seminar where a very impressive lecturer advocates the use of "AS". You like what you hear, so much so that you do not even ask for documentation supporting the efficacy and SAFETY of "AS." You are so impressed that you may even purchase a stock of the product right at the seminar, so that it is readily available for use or sale to your patients. You administer, or recommend, "AS" to a patient to remove mercury from the body. Patient dies. An autopsy is performed. Cause of death is determined to be severe liver damage caused by "AS". You have no documentation to support the safety of "AS". Where are you? If sued or prosecuted, your professional career is now over. How many more patients can you now help?

This is not to say that any pharmaceutical or non-pharmaceutical agent is free of risk. The intention of this article is not to condemn any one product or material. Algae is only used as an example of potential for problems, as brought to light by the Florida newspaper article. The International Academy of Oral Medicine and Toxicology (IAOMT) is now developing “Standards of Care” for Anti-Toxic Therapy. The goal is to determine which materials and procedures have documentation supporting their use and which ones do not. Some may be given approval by IAOMT and some may be designated as not approved. Through this program, practitioners will at least know the status of particular agents. This will provide a foundation of valid knowledge upon which to base anti-toxic therapy.

The safe and prudent course for health care providers is to always seek valid documentation supporting the efficacy and safety of any treatment modality. If documentation is absent - be cautious!

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DENTAL BOARD UPDATE

As of this writing, Bio-Probe is aware of only two mercury-free dentists under attack by state dental boards. Not that long ago, there were over twenty under attack in a dozen states. The present situation is a tribute to the efforts of many people, not the least of which are attorneys Charles Brown and James Turner with the Washington, D.C. law firm of Swankin & Turner, and attorneys James Love and Robert Reeves. The efforts of these attorneys, and others, have led to victories in many states. At least a dozen cases against mercury-free dentists have been dismissed by state dental boards in the
past year or so. Most, if not all, of these complaints were filed by dentists, not patients.

The relief from dental board attack is a result of many factors; legal, scientific, public support, and changing attitude within the rank and file of the dental profession. If we can secure these last two cases, and emerge victorious, attacks by state dental boards against dentists for simply being mercury-free may well be over. We may all, then, be free to treat our patients according to our moral dictates.

The two remaining mercury-free dentists under attack are fighting serious battles. These two are Dr. Mark A. Breiner in Connecticut and Dr. Terry J. Lee in Arizona. These two dentists have been engaged in long, hard struggles against determined dental boards (usually only one or two members of the board are hostile). They have endured tremendous hardships, emotionally and financially. They are valiantly fighting for all of us! They deserve our fullest support. Please send a contribution to help defray the tremendous legal costs and help to end the unwarranted attacks against all of us: *Mark A. Breiner, D.D.S. 325 Post Road, Suite 3A, Orange, Connecticut, 06477 [T: (203) 799-6353]. *Terry Lee Defense Fund, c/o NISLAPP, 1424 16th Street, NW, Suite 105, Washington, DC, 20036 [F: (202) 265-6564].

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**MERCURY FREE BUMPER STICKER!**

DAMS, Inc. (Dental Amalgam Mercury Survivors) has produced an excellent new bumper sticker, in 3" by 12" dimension. It contains the "No Hg" red circular logo surrounded by a tooth, toothbrush and dental drill. In blue, are the words: "Mercury Free and Healthy" and below that in black the web page address: "www.amalgam.org."

The DAMS web page describes the dental amalgam issue and presents abstracts of pertinent scientific documents. A bundle of 100 of these bumper stickers can be purchased for $50 US, plus shipping cost, at: Northern Sun Merchandising; 2916 East Lake Street, Minneapolis, MN 55406. T: (800) 258-8579 or (612) 729-2001. F: (612) 729-0149. Email: nsm@scc.net.

Support of this DAMS project will help spread the word to the public, with profits from the sale of the bumper stickers going to support the activities of DAMS, Inc.

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**SCIENCE**

Mercury Vapor Release From Dental Amalgam After Laser Treatment.

Pioch, T; Matthias, J.


ABSTRACT: The aim of this in vitro study was to determine whether the treatment of amalgam with different lasers leads to an increased release of mercury (Hg) vapor. In the case of CO2 lasers in pulse and continuous wave mode, there was no effect visible on the amalgam surface and no Hg vapor could be detected. Using an Nd:YAG, Er:YAG or Nd:YLF laser, crater formation could be observed on the amalgam surfaces. With the solid state lasers tested, however, the Hg vapor measurements taken indicated that pulses applied to amalgam cause a substantially increased release of Hg vapor. This vapor may contribute to the patient's total mercury exposure.

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MR Findings of Minamata Disease: Organic Mercury Poisoning.

Korogi, Y; Takahashi, M; Okajima, T; Eto, K.


ABSTRACT: We describe MR findings in patients with Minamata disease who have been followed for a long time. All patients examined were affected after daily eating of a large quantity of methyl mercury contaminated seafood, from 1955 to 1958, and showed typical neurological findings.

On MR images, the visual cortex, the cerebellar vermis and hemispheres, and the postcentral cortex are significantly atrophic in Minamata disease. The visual cortex is slightly hypointense on T1-weighted images and hyperintense on T2-weighted images, probably representing the pathologic changes of status spongiosus. MRI can demonstrate the lesions located in the calcarine area, cerebellum, and postcentral gyri, which are probably related to three of the characteristic manifestations of this disease: the constriction of the visual fields, ataxia, and sensory disturbance, respectively.

**BIO-PROBE COMMENT:** The diagnosis of mercury poisoning is an acknowledged problem, as so much of the damage is not specific. Although this study investigated organic mercury damage, it will hopefully point the way to diagnosing damage
from elemental mercury vapor, which is so similar to that from methyl mercury.

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Effects of Micronutrients on Metal Toxicity.
Peraza, MA; Ayala-Fierro, F; Barber, DS; Casarez, E; Rael, LT.

ABSTRACT: There is growing evidence that micronutrient intake has a significant effect on the toxicity and carcinogenesis caused by various chemicals. This paper examines the effect of micronutrient status on the toxicity of four nonessential metals: cadmium, lead, mercury, and arsenic. Unfortunately, few studies have directly examined the effect of dietary deficiency or supplementation on metal toxicity. More commonly, the effect of dietary alteration must be deduced from the results of mechanistic studies. We have chosen to separate the effects of micronutrients on toxic metals into three classes: interaction between essential micronutrients and toxic metals during uptake, binding, and excretion; influence of micronutrients on the metabolism of toxic metals; and effect of micronutrients on secondary toxic effects of metals.

Based on data from mechanistic studies, the ability of micronutrients to modulate the toxicity of metals is indisputable. Micronutrients interact with toxic metals at several points in the body: absorption and excretion of toxic metals; transport of metals in the body; binding to target proteins; metabolism and sequestration of toxic metals; and finally, in secondary mechanisms of toxicity such as oxidative stress. Therefore, people eating a diet deficient in micronutrients will be predisposed to toxicity from nonessential metals.

BIO-PROBE COMMENT: This paper supports the approach of the International Academy of Oral Medicine and Toxicology (IAOMT). IAOMT has established a Standard of Care called “Anti-Toxic Therapy.” This addresses the subject from five perspectives: 1) Prevention (exposure, absorption, damage); 2) Elimination (pharmaceutical and non-pharmaceutical “chelation”); 3) Neutralization (chemically producing non- or less toxic compounds); 4) Restoration (repairing damage); and 5) Verification (testing). The subject of addressing exposure to mercury is very complex; it should not be addressed simplistically.

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Degree of Peroxidative Status in Neuronal Tissues by Different Routes of Inorganic Mercury Administration.
Anuradha, B; Rajeswari, M; Varalakshmi, P.

ABSTRACT: Mercuric chloride was administered by three routes; subcutaneous, intramuscular and intraperitoneal to adult female Wistar rats. The peroxidative status of the cerebral cortex, cerebellum and the sciatic nerves were studied. Enhanced levels of lipid peroxides indicate progressing cellular injury.

All the experimental groups show high levels of reduced glutathione and increased activities of glutathione peroxidase, superoxide dismutase and catalase. Neurotoxic status was more pronounced in intramuscularly administered mercuric chloride, followed subsequently by intraperitoneal and subcutaneous routes of administration.

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Reversible Color Vision Loss in Occupational Exposure to Metallic Mercury.
Cavalleri, A; Gobba, F.

ABSTRACT: Color vision was evaluated in twenty one mercury exposed workers and referents matched for sex, age, tobacco smoking, and alcohol habits. The Lanthony 15 Hue desaturated panel (D-15 d) was applied. In the workers, mean urinary Hg (HgU) was 115 +/- 61.5 microg/g creatinine; in all but one the values exceeded the biological limit (BEI) proposed by the American Conference of Governmental Industrial Hygienists.

A dose related subclinical color vision impairment was observed in Hg-exposed workers compared to the referents. Just after the survey, working conditions were improved. Twelve months later the workers were reexamined. Mean HgU was 10.0 microg/g creatinine and in no subjects was the BEI exceeded. Color perception was significantly improved compared to the first examination and, furthermore, no differences were observed between exposed workers and referents.

The results add evidence that the color vision loss observed during the first part of the study was related to Hg exposure and, moreover, show that this effect is reversible. These data indicate that metallic Hg can induce a reversible impairment in color
perception. This suggests that color vision testing should be included in studies on the early effects of Hg. The possibility of applying the D-15 d as an early effect index in the biological monitoring of Hg exposed workers should also be entertained.

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Mercury Content in Amalgam Tattoos of Human Oral Mucosa and Its Relation to Local Tissue Reactions.

Forsell, M; Larsson, B; Ljungqvist, A; Carlmark, B; Johansson, O.


**ABSTRACT:** Mucosal biopsies from 48 patients with and 9 without amalgam tattoos were analyzed with respect to their mercury content, distribution of mercury in the tissue, and histological tissue reactions. The distribution of mercury was assessed by autometallography (AMG), a silver amplification technique. The mercury content was determined by energy dispersive X-ray fluorescence (EDXRF), a multielemental analysis.

Mercury was observed in connective tissue where it was confined to fibroblasts and macrophages, in vessel walls and in structures with the histological character of nerve fibers. A correlation was found between the histopathological tissue reaction, the type of mercury deposition, the intensity of the AMG reaction, and the mercury content. Mercury was also found in patients with amalgam dental fillings but without amalgam tattoos.

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Influence of Chronic Mercury Poisoning Upon the Connective Tissue in Rats: I: Effect of Mercuric Chloride on Glycosaminoglycan Levels in Tissues, Serum and Urine.

Olczyk, K; Kucharcz, EG; Gowacki, A.


**ABSTRACT:** Rats were intoxicated with mercuric chloride (1 mg/kg b.w.) daily, for 12 weeks. A decrease in total glycosaminoglycan content was shown in the skin, the lungs, the liver and the heart muscle. These changes were accompanied by a slight alteration of the glycosaminoglycan pattern, a decrease in hyaluronic acid in the skin, the lungs and the heart muscle and an enhancement of heparan sulphate level in the kidneys. In serum of mercury intoxicated rats, an increase in total glycosaminoglycan levels was shown. This enhancement was caused by elevation of almost all fractions. Urine output of glycosaminoglycans was higher in mercury treated animals as compared to the controls.

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Influence of Chronic Mercury Poisoning Upon the Connective Tissue in Rats: II: Effect of Mercuric Chloride on Collagen and Elastin.

Kucharcz, EJ; Olczyk, K.


**ABSTRACT:** Rats were intoxicated with mercuric chloride (1 mg/kg b.w.) daily, for 12 weeks. An increase of total collagen and elastin content was found in the skin, the lungs, the liver, the kidneys and the heart muscle. The increase resulted from the elevated level of soluble collagen. These changes were accompanied by elevated hydroxyproline level in serum and urine. It is concluded that chronic intoxication with mercury leads to disturbed composition of the connective tissue matrix.

**BIO-PROBE COMMENT:** The firm diagnosis of chronic mercury intoxication is an acknowledged problem. These two studies point to factors that should be investigated as possible diagnostic indicators. In particular, if the urine levels are valid indicators of chronic mercury intoxication, the tests would be non-invasive.

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Effect of Mercuric Chloride Poisoning on Iron Distribution in Rats.

Grosicki, A; Kossakowski, S.


**ABSTRACT:** The aim of our work was to establish the influence of the HgCl2 poisoning (various doses) on the Fe distribution in the rat organism. 175 Wistar rats were divided into 5 groups: I: control group; II: intoxicated with HgCl2 in the dose of 1 mg/kg of body weight; in the III group the HgCl2 dose was 6 mg/kg; in the IV group, 12 mg/kg; in the V, 12 mg/kg, but rats of this group were given afterwards BAL (2,3-dimercaptopropanol).

All rats were given 0.2 ml of 59FeCl3, radioactivity 32.7 kBq. The animals were put to sleep with chloroform after 3 and 6 hours, and 1, 2, 4, 8, 14 days after the isotope administration. For the radiometric assays the following organs or tissues were taken: stomach, small and large intestine, liver, kidneys, lungs, heart, muscles, spleen, blood, brain, testicles. The results are given in the percent of Fe
dose in 1 g of wet tissue. The statistical analysis (Students’ t test) was performed.

The results indicate that in poisoned animals the Fe distribution was different than in controls. Increased concentration of 59Fe was noted after 3 and 6 hours in stomach and after 1 day in large intestine, whereas in other organs the Fe concentration was lower, correlating usually with the Hg dose. One interesting thing was noted: the Fe concentration in almost all organs was higher in rats intoxicated with the 12 mg/kg dose of HgCl2 and given afterwards BAL (IV group), than in rats that were not given BAL. Present results can have some practical significance in therapeutic procedure in mercury poisoning and during the convalescence.

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Antibiotic Resistance in Oral/Respiratory Bacteria.
Roberts, MC.

ABSTRACT: In the last 20 years, changes in world technology have occurred which have allowed for the rapid transport of people, food, and goods. Unfortunately, antibiotic residues and antibiotic resistant bacteria have been transported as well. Over the past 20 years, the rise in antibiotic resistant gene carriage in virtually every species of bacteria, not just oral/respiratory bacteria, has been documented.

In this review, the main mechanisms of resistance to the important antibiotics used for treatment of disease caused by oral/respiratory bacteria - including beta-lactams, tetracycline, and metronidazole - are discussed detail. Mechanisms of resistance for macrolides, lincosamides, streptogramins, trimethoprim, sulfonamides, aminoglycosides, and chloramphenicol are also discussed, along with the possible role that mercury resistance may play in the bacterial ecology.

BIO-PROBE COMMENT: The role of amalgam derived mercury in engendering bacterial resistance to mercury and to antibiotics is well documented. Although the research has been ignored by the dental profession, this article emphasizes the importance of that factor in treating dental disease!

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Participation of Mercuric Conjugates of Cysteine, Homocysteine, and N-Acetylcysteine in Mechanisms Involved in the Renal Tubular Uptake of Inorganic Mercury.
Zalups, RK; Barfuss, DW.

ABSTRACT: Mechanisms involved in the renal uptake of inorganic mercury were studied in rats administered a nontoxic 0.5 mmol/kg intravenous dose of inorganic mercury with or without 2.0 mmol/kg cysteine, homocysteine, or N-acetylcysteine. The renal disposition of mercury was studied 1 hr after treatment in normal rats and rats that had undergone bilateral ligation. In addition, the disposition of mercury (including the urinary and fecal excretion of mercury) was evaluated 24 h after treatment.

In normal rats, co-administering inorganic mercury plus cysteine or homocysteine caused a significant increase in the renal uptake of mercury 1 h after treatment. The enhanced renal uptake of mercury was due to increased uptake of mercury in the renal outer stripe of the outer medulla and/or renal cortex. Ureteral ligation caused reductions in the renal uptake of mercury in all groups except for the one treated with inorganic mercury plus N-acetylcysteine. Thus, it appears that virtually all of the mercury taken up by the kidneys of the normal rats treated with inorganic mercury plus N-acetylcysteine occurred at the basolateral membrane. Urinary excretory data also support this notion, in that the rate of excretion of inorganic mercury was greatest in the rats treated with inorganic mercury plus N-acetylcysteine.

Our data also indicate that uptake of inorganic mercury in the kidneys of rats treated with inorganic mercury plus cysteine occurred equally at both luminal and basolateral membranes. In addition, the renal uptake of mercury in rats treated with inorganic mercury plus homocysteine occurred predominantly at the basolateral membrane with some component of luminal uptake.

The findings of the present study confirm that there are at least two distinct mechanisms involved in the renal uptake of inorganic mercury, with one mechanism located on the luminal membrane and the other located on the basolateral membrane. Our findings also show that cysteine and homologs of cysteine, when co-administered with inorganic mer-
cury, greatly influence the magnitude and/or site of uptake of mercuric ions in the kidney.

HgCl2-induced acute renal failure and its pathophysiology
Yanagisawa H.
Nippon Eiseigaku Zasshi, 52(4):618-623, Jan 1998

ABSTRACT: Mercury chloride (HgCl2) has a potent nephrotoxic effect. Most of Hg2+ existing in plasma following HgCl2 exposure forms a complex with sulfhydryl-containing ligands such as albumin and glutathione (GSH). The Hg(2+)-GSH complex is filtered in the glomeruli of the kidney and degraded into Hg(2+)-cysteine in the proximal tubules by the combined action of gamma-glutamyl transpeptidase and dipeptidase present in the epithelial cells. The degradation product is then incorporated and accumulated into the proximal tubule epithelial cells. The accumulated Hg2+ in the epithelial cells finally causes acute tubular necrosis (ATN) by its cytotoxic effect. At present, it is believed that tubular obstruction resulting from ATN triggers the onset of HgCl2-induced acute renal failure (ARF). A progressive fall in glomerular filtration rate (GFR) contributes to the progression of HgCl2-induced ARF. The fall in GFR may be caused by an increment in afferent arteriole resistance (RA) and a decrement in the ultrafiltration coefficient (Kf) due to mesangial cell contraction. These changes in RA and Kf may be attributed to the increased action of the vasoconstrictors, angiotensin II and endothelin-1 and to the decreased action of the vasodilator, nitric oxide observed at the glomerulus level of HgCl2-induced ARF. Accordingly, the imbalance between these vasoactive substances appears to play an important role in the progression of HgCl2-induced ARF due to reducing GFR. Further studies, however, remain to elucidate the mechanisms involved.

Hattis, D; Silver, K.

ABSTRACT: For noncancer effects, the degree of human interindividual variability plays a central role in determining the risk that can be expected at low exposures. This discussion reviews available data on observations of interindividual variability in (a) breathing rates, based on observations in British coal miners; (b) systemic pharmacokinetic parameters, based on studies of a number of drugs; (c) susceptibility to neurological effects from fetal exposure to methyl mercury, based on observations of the incidence of effects in relation to hair mercury levels; and (d) chronic lung function changes in relation to long term exposure to cigarette smoke. The quantitative ranges of predictions that follow from uncertainties in estimates of interindividual variability in susceptibility are illustrated.

BIO-PROBE COMMENT: The possible adverse health effects of human exposure to lead, cadmium and most other toxic agents are based on “Risk Assessment” principles. These principles are manifold, some of which are detailed in this study.

For some reason, human exposure to amalgam mercury is viewed differently; i.e., in relation to causing named syndromes (such as Multiple Sclerosis and Alzheimer’s Disease). In the absence of epidemiological studies investigating a relation between human exposure to amalgam mercury and these syndromes, the exposure is declared to be harmless. Such a position arrogantly contradicts the principles of valid and respected science.

There have been two formal risk assessments that address patient exposure to amalgam mercury, conducted by the governments of the United States and Canada. Both of these determined that the exposure exceeds minimal risk levels for the general population exposure to mercury vapor. Rather than acknowledge these valid risk assessments, the defenders of mercury fillings continue to relate patient exposure to standards for occupational exposure to mercury vapor. The workroom standards are limited to 40 hours of exposure per week, require regular medical examination of the workers, and are limited to otherwise healthy adult workers. Undeniably, the workroom standards can NOT be applied to the general population. Yet, the supposedly “scientific” dental profession continues to adamantly do so!

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FORUM

INTERNATIONAL ACADEMY OF ORAL MEDICINE AND TOXICOLOGY

The IAOMT has initiated the first formal Accreditation Program for biocompatible dentistry. This program is designed to be the “gateway to the
future of dentistry”, hopefully leading to eventual board certification. It will also satisfy the increasing demands of the public for “qualified” or “specially trained” biological dentists. The program is comprehensive, including six required Core Curriculum Courses with a written examination, elective courses at IAOMT meetings, interview of two case presentations, and submission of a Standard of Care on a material, procedure or product. IAOMT members interested in enrolling in this program may do so through the office in Orlando [see below].

The rapid growth of the IAOMT has provided the resources for addressing four additional areas of dental therapy. Committees have now been formed to develop procedures and policies for a biocompatible approach to periodontal therapy, endodontic therapy, anti-toxic therapy, and cavitations.

IAOMT MID-YEAR MEETING

DATE: Friday-Saturday, 19-20 March 1999.
SITE: Las Vegas, Nevada.
HOTEL: Riviera Hotel, 2901 Las Vegas Boulevard, South; Las Vegas, NV. 89109. T: (702) 734-5110; F: (702) 794-9410. IAOMT rate: $95.00/night, single/double (plus 9% tax); $20.00 each additional.
MEETING REGISTRATION: IAOMT, P.O. Box 608531, Orlando, FL. 32860-8531. T: (407) 298-2450; F: (407) 298-3075. Members= $395.00; non-members= $495.00. Lunches on Friday and Saturday included for registrant and one additional (spouse/staff); $100.00 for each additional.

PROGRAM:

Friday: Clinical Applications.
○ 8:30am-12:00pm: “Periodontal Standard Procedures.” IAOMT Periodontal Therapy Committee; Dr. Thomas Baldwin, Chairman.
○ 4:00-5:00pm: “Clinical Practice Orientation.”

Chairman Richard Chalin, DMD and IAOMT committee.
• 1:30-5:00pm: IAOMT Accreditation Program case history interviews: David Regiani, DDS and IAOMT Education Committee. [By appointment; candidates contact IAOMT Executive Director.]
• 5:00-6:30pm: IAOMT Business Meeting.
  Saturday: Speakers.
  Murray J. Viny, DMD: “Never Have So Few Done So Much Harm to So Many.”
  J. C. Pendergrass, Ph.D.: “Gingival Crevicular Fluid: Components and Analysis.”
  David Quig, Ph.D.: “Enhancement of Mercury Elimination Via the Biliary/Fecal Route.”
  David C. Kennedy, DDS: “Health Effects of Ingested Fluoride.”
  James M. Love, JD: “Medico-Legal Considerations for Biological Dentistry.”

IAOMT 1999 ANNUAL MEETING

DATE: Friday-Saturday, 8-9 October 1999.
SITE: Atlanta, Georgia.
HOTEL: Sheraton Perimeter Center Hotel and Suites Atlanta, 111 Perimeter Center West, Atlanta, GA 30346. T: (770) 396-6800; F: (770) 394-4805.
MEETING REGISTRATION: IAOMT, P.O. Box 608531, Orlando, FL. 32860-8531. T: (407) 298-2450; F: (407) 298-3075.
MEETING HOST: Dr. Ronald Dressler.
PROGRAM: To be announced.

If you are a mercury-free dentist or are contemplating going mercury-free, you need to join the IAOMT. The IAOMT has helped fund or has been the catalyst for much of the current scientific research demonstrating that dental amalgam is not the benign dental material that 150 years of use and the ADA would like you to believe. Furthermore, the IAOMT is doing something about Standards of Care and Protocols that protect you, your staff and the patient. For membership information contact Dr. Michael F. Ziff, DDS, P.O. Box 608531, Orlando, FL 32860-8531. Phone 407-298-2450, Fax 407-298-3075.