FDA DOES NOT APPROVE THE USE OF AMALGAM!

The Dental Products Panel of the Medical Devices Advisory Committee met at the Food and Drug Administration building in Rockville, Maryland on 15 March 1991. After a full day of testimony from numerous speakers, the Panel voted unanimously on two resolutions:

1. There was not sufficient evidence presented to warrant a position that dental amalgam is unsafe, but unanswered questions were raised that warrant further investigation.

2. A committee should be formed to define the required areas of investigation and the appropriate research to resolve the question of amalgam safety.

WHAT THE FDA PANEL DIDN'T DO!

This panel will be remembered more for what it didn’t do than for what it did do. Although it was only an advisory committee with no authority to determine policy or actions, it did have an obligation to recommend a classification for dental amalgam to the Food and Drug Administration.

The law is clear on medical and dental devices. The Medical Device Amendment to the Federal Food, Drug, and Cosmetic Act was signed into law on 28 May 1976. This Amendment (Sections 513 through 521) requires the Food and Drug Administration to classify all medical (including dental) devices accepted for use in the United States. If a material is not classified under the law, it cannot be considered a device approved for dental use.

Having personally witnessed the testimony presented at the meeting, it is easy for this writer to understand why the Panel did not make a recommendation to classify dental amalgam. There was no scientific documentation presented that demonstrated amalgam to be harmless. Claims of safety are still based on the strictly anecdotal position that over 150 years of use demonstrates the safety of dental amalgam.

In view of the lack of scientific data demonstrating the safety of dental amalgam, the Panel could not recommend acceptance of the device under FDA rules. Recommended acceptance into Class II or Class III of the FDA mandate would have placed amalgam manufacturers, as well as amalgam-using practicing dentists, in an untenable position. These categories require valid scientific documentation demonstrating the safety and effectiveness of devices containing materials of potential health risk. Class I is reserved for those devices determined to not contain materials of potential health risk.

REMOVAL OF AMALGAM FILLINGS IS NOT A PUNISHABLE OFFENSE!

The significance of the actions (or rather the lack
there-of) of the FDA Advisory Panel is truly astounding. The FDA is the only body with legal authority over the use of dental devices. Since the FDA does not consider dental amalgam to be an accepted dental device, its removal from patients cannot possibly be considered illegal! Dentists who have been previously punished may yet have recourse in this matter.

This brings us to the question of public statements that the FDA "grandfathered" approval of dental amalgam. These statements are absolutely false!

**DENTAL DEVICE PANEL VIOLATES FDA MANDATE!**

The Final Rule of the FDA on Classification of Dental Devices is found in the Federal Register, Volume 52, Number 155, 12 August 1987. There is no mention in this document of dental amalgam as a dental device. Rather the FDA accepted and classified the following: "Dental mercury" (Section 872.3700) as a Class I device. "Amalgam Alloy" (Section 872.3050) as a Class II device.

The same is found in the 1980 Proposed Rules (with the exception that dental mercury was recommended to be in Class II) and all previous actions of the Dental Device Panel as documented in the Federal Register. Dental amalgam was never addressed or considered as a dental device.

The acceptance and classification of these two materials was a clear violation of FDA rules, which mandate that classified medical devices must be both "safe" and "effective".

Neither "dental mercury" nor "amalgam alloy" can be used alone as a dental device. Either of these "devices" would rapidly wash out of a dental cavity preparation. Both materials fail the clear, and often repeated, FDA rule requirement for "effectiveness". The Dental Device Panel erred in classifying them in the first place, and the FDA is obligated to correct that error by immediately withdrawing their classification.

**CONSIDERATION OF UNITED STATES TORT LAW**

Should the FDA consider future classification of dental amalgam as an accepted dental device, the effectiveness of this device would not be challenged. However, one extremely important factor concerning the "safety" of the device has been totally overlooked. United States Tort Law [Restatement of Torts] defines "harm" as: "Any change in the structure or function of the body." It is further stated that nothing beyond that definition need be proven to establish tort.

Titled disease states are nothing more than names given to eventual body responses to pre-existing harm. The scientific literature is clear. Mercury causes damage within the body long before clinically observable signs and symptoms appear. Recognized authorities on mercury toxicology have publicly declared that no amount of exposure to mercury vapor can be considered totally harmless.

Currently, defenders of amalgam rely on the position that patient exposure to mercury from dental amalgam fillings has not been conclusively connected to any disease state. This position ignores U.S. Tort Law and places the amalgam-using practicing dentist in an absolutely indefensible position.

**BIO-PROBE NOTE:** We should all be grateful to dentist/attorney Joel Berger for discovering the two preceding issues. Unfortunately, Joel came down with the flu and could not get to the FDA meeting. They were presented in his stead by Michael F. Ziff, D.D.S.

**WORLD HEALTH ORGANIZATION CONTRADICTS DENTISTS!**

Two renowned speakers at the FDA meeting were Dr. Lars Friberg and Dr. Thomas W. Clarkson. They are the world’s foremost authorities on mercury toxicology and members of the World Health Organization’s committee on mercury. They stated that W.H.O.’S new document on mercury would soon be available and that they had permission to release data from that document.

The most significant information they presented was the W.H.O. conclusion on estimated intake of mercury subjects receive from dental amalgam fillings. The major contributions to human intake of mercury were: Dental amalgam fillings = 3.0-17.5 micrograms/ day (mercury vapor); fish = 2.4 micrograms/day (methylmercury); non-fish food = 0.3 micrograms/day (inorganic mercury). Other sources (air and water) were found to be negligible.

It was pointed out that these data contrast sharply with estimates derived by dental authorities (Mackert, Olson, Bergman, et al) who have decided that patient intake of mercury from dental amalgam fillings is
1-2 micrograms per day and claim this amount to be minimal compared to intake from fish. It would seem that the medical scientific community is not in agreement with dental authorities on this issue.

**MERCURY SENSITIVITY**

Alfred V. Zamm, M.D. another of the invited speakers offered a totally new approach in looking at the effects of dental amalgam mercury on the human body. Dr. Zamm presented data and clinical histories to support the observation that there is a subset of the population who are intolerant to chemicals and to mercury as xenobiotic substances. Xenobiotic (pronounced xenobiotic) is defined as substances that are foreign to the body, such as drugs, poisons, or metals which serve no metabolic purpose. Mercury from dental amalgam induces symptoms in a sensitive subset of the population who have been observed to be chemically intolerant. Dr. Zamm has clinically observed that the chemically intolerant and the mercury intolerant, are in fact one group. More importantly, this sensitive subset of the population serves as a marker that warns of the potential danger of dental mercury to the rest of the population who are at risk but may not yet exhibit symptoms. Dr. Zamm also pointed out that harm cannot be determined only by the concentration of mercury to which the patient is exposed. One must also determine the magnitude of the sensitivity of the patient. The magnitude of disease is proportional to both of these factors. The measurement of either one alone provides no information concerning the outcome in terms of induced disease. This relationship can be mathematically summarized as follows: \((Hg) \times (MS) \propto MD\). (Formula codes: \((Hg) = \text{concentration}; (MS) = \text{Magnitude of Sensitivity}; \text{proportional to Magnitude of the Disease}).

**OTHER SPEAKERS**

Dr. K. Sune Larsson of Sweden presented data demonstrating the transfer of mercury through the placenta to the fetus; Robert Pinco Esq. and John Osbourne, D.D.S. spoke representing the dental amalgam manufacturers; and representing the ADA was Dr. Robert Baratz who presented his personal (scientifically unsubstantiated) views on the lack of validity of the published research by Drs. Vimy and Lorscheider and even went so far as to question the brain autopsy data of Drs. Frieberg and Nylander. Dr. Baratz prefaced his remarks by stating that "The ADA endorsed his presentation." (Dr. Rodway Makert, Jr. although not an invited presenter, did present his pro-amalgam views during the open public hearing).

**THE OPEN PUBLIC HEARING**

The FDA allotted the first 90 minutes of the meeting to 5-minute presentations from interested parties from the floor. We can be proud of the dignified, factual presentations made by three members of D.A.M.S.; Pat Link, Louise Herbeck, and Glenda Smith. They were truly outstanding and gave the FDA a new perspective on the people who have been effected by dental amalgam.

Excellent presentations of information and documentation pertinent to the issue were presented to the FDA by members of the International Academy of Oral Medicine and Toxicology (I.A.O.M.T.); Dr. Michael F. Ziff, Dr. Sandra Denton, Dr. W. Wayne King, Sam Queen, Dr. Phillip Sukel, Dr. Richard Fischer, and Dr. Michael Pawk. These presentations are now on public record and will hopefully have an impact on future actions of the Food and Drug Administration.

**ADA RETREATS ON MERCURY AMALGAM "ETHICS"**

In January of 1991 the American Dental Association published the following statement from the ADA Division of Legal Affairs regarding its Advisory Opinion (NO. 7) on ADA Resolution 42H-1986 in the ADA Principles of Ethics and Code of Professional Conduct: "The important point to remember regarding this Advisory Opinion is that it refers only to the dentist who removes amalgam solely on his or her own recommendation. The dentist who removes amalgam based on a request from a patient or the patient’s physician is not acting unethically." (ADA News. 7 January 1991 -and- the insert "Special Report" with the January 1991 J.A.D.A.)

The astounding alteration of position in this statement is signified by the declaration that removal of amalgam fillings upon "request from a patient" is not unethical.

Resolution 42H-1986 was passed by the House of Delegates at the 1986 Annual Session of the American Dental Association and is found as Transcript 1986:536. Advisory Opinion No. 7 was inserted into the ADA Principles of Ethics and Code of Professional Conduct in 1987 and includes the following: "Based
on available scientific data the ADA has determined that the removal of amalgam restorations from the non-allergic patient for the alleged purpose of removing toxic substances from the body, when such treatment is performed solely at the recommendation or suggestion of the dentist, is improper and unethical."

The following statement was published in the Journal of the American Dental Association (Vol. 114:23. Jan 1987.): "Advocating the removal of clinically serviceable dental amalgam restorations solely to substitute a nonmercury-containing material is unwarranted and violates the ADA Principles of Ethics and Code of Professional Conduct. In cases in which state dental boards initiate proceedings on this question, the Association will cooperate by making available scientific personnel as expert witnesses."

It is clear from this latter statement that, in 1986-87, even "advocating" the removal of amalgam fillings, let alone actually performing the procedure was considered a punishable offense by the ADA. It is further clear that the ADA, by virtue of its statement printed in the J.A.D.A., was encouraging state Dental Boards to take action against anti-amalgam dentists.

The January 1991 alteration is a clear retreat from the former position albeit not, however, without raising some very interesting questions.

The most obvious question is "why the sudden change?" Has the newly published research raising questions on the safety of dental amalgam made an impression on the ADA? Has the pronounced media attention on the issue and the resulting public interest forced the ADA to reevaluate the foundation for their public statements that dental amalgam is totally harmless to patients not allergic to mercury? Offering "over 150 years of use" as a counter to controlled research published in the foremost scientific journals can be a frightening position. Have the responsible individuals finally realized the legal jeopardy they face?

What about the anti-amalgam dentists who have already been punished? There are many of these. Some have had their licenses suspended or revoked; others have been punished by virtue of financial and emotional losses in defense of the harassment. How about anti-amalgam dentists who have been ostracized and openly vilified by their peers? Being labelled "quacks" or "frauds" or accused of "ripping off patients" isn't easy to live with. Will all of these dentists now be exonerated or, perhaps, receive apologies and be welcomed back into the fold with open arms?

Are anti-amalgam dentists now suddenly not "unethical" simply because the patient has "requested" amalgam removal? Are we now, by ADA edict, suddenly once again esteemed and valued members of the dental community?

One suspects that these ethical and legal questions will be answered in the not too distant future. It has been noted that everyone has their day; ours is coming!

**RESEARCH ADVISORY!**

A report by Jim Warren in the Lexington Herald-Leader on March 15, 1991 carried the headline "Possible Clue Discovered In Infant Death Syndrome." The article stated that researchers Dr. D. Larry Sparks and Dr. John Hunsacker III of the University of Kentucky, Sanders-Brown Center on Aging had discovered high levels of nerve-cell degeneration in the brains of babies who had died of sudden infant death syndrome (SIDS). The Drs. believe that if cell degeneration in SIDS infants is confirmed in areas of the brain that control respiration it might explain the breathing stoppages that kill SIDS victims.

The SIDS discovery is related to the on-going studies on Alzheimer's disease being done at the Sanders-Brown Center. The brains of infants who had died of various causes were being used as controls in one of the Alzheimer's disease experiments. As reported in the November 1990 issue of Bio-Probe, Dr. Markesbery and his group at the Sanders-Brown Center had found high levels of mercury in the brains of 180 Kentuckians who had died of Alzheimer's disease over the last five years. Hopefully, the research team will investigate whether the neuron degeneration tissue from SIDS infants also contains mercury.
SCIENTIFIC REVIEW

Du Preez, IC; Rosslee, D; Van Der Merwe, CA; Hugo, L.
Mercury Release During Polishing of Amalgam Restorations.

ABSTRACT: Polishing of amalgams is a process which generates friction and heat. The present study was conducted to investigate the effect of polishing on the mercury release of amalgam restorations.

Amalgam restorations of 25 patients were polished with amalgam polishing cones (Shofu) and oral mercury level was directly measured with a gold film mercury analyzer (Jerome). The measurements were taken directly before and after polishing, after 24 hours, 7 days, 1 month and 3 months after polishing. A few readings were also taken during the polishing procedure.

According to the measurements directly before and after polishing, it appears that polishing drastically increases the mercury levels (from 0.005 mg Hg/m$^3$ before polishing to 0.124 mg Hg/m$^3$ after polishing). According to the measurements taken during polishing, the effect of polishing was even more dramatic (0.914 mg Hg/m$^3$). Within 24 hours the mercury levels were found to return to the readings obtained before polishing and these values remained constant at the 7 day, 1 month and 3 month follow-up investigations. Although the number of restorations, the restoration surfaces and polishable surfaces have no statistical significant effect on the level of the mercury readings before polishing, the polishing process did however influence the mercury level significantly.

According to the above mentioned results, it can be concluded that the polishing of amalgams causes a brief, but sharp rise in the oral mercury vapor levels.

BIO-PROBE COMMENT: One can not help but wonder the influence of this information on probable mercury vapor exposures encountered by dental hygienists. Mercury vapor levels found in this study far exceed OSHA and NIOSH Standards.

Leary, R; Kilgus, G; Leinfelder, KP.
In-vitro Microleakage of Glass Ionomers and Dentin Bonding Agents.

ABSTRACT: The purpose of this study was to measure the microleakage of glass ionomer as a liner and restorative material, a new polyamide sealing agent (Barrier) and a new dentin bonding agent (Scotchbond II). Also evaluated was the effect of insertion technique on microleakage. Both hydroxyl ion detection and basic fuchsin techniques were used to monitor microleakage. Only Class V cavity preparations were employed. The results of the study demonstrated that of all variables included, glass ionomer was the most effective in reducing microleakage. Using Retief's method of scoring the mean value for glass ionomer as a liner or restorative material was 0.5. When used as a liner, any microleakage initiated at the gingival margin progressed no further that the restoration/glass ionomer interface. The polyamide liner was effective in reducing microleakage in the occlusal area only. Finally, the microleakage (OH-) of Silux (3M Co.) was significantly less than Durafile (Kulzer, Inc.). The use of Scotchbond II had only a limited effect (25% reduction) on the microleakage of Durafile. The difference in leakage rates can be attributed to water sorption. On the basis of this study, the most effective method of reducing microleakage was on incorporation of glass ionomers as a liner or base.

BIO-PROBE COMMENT: This study provides strong support for the position that all dentin should be covered with glass ionomer, especially at the gingival areas.

Kidd, EAM; O'Hara, JW.
The Caries Status of Occlusal Amalgam Restorations with Marginal Defects.

ABSTRACT: The purpose of this study was to histologically examine occlusal amalgam restorations with both defective and sound margins for comparison of their caries status. Thirty extracted teeth with occlusal amalgam restorations were sectioned so that a ditched and a clinically sound margin could be
examined on the same tooth in the mouth. Samples were randomly selected from a large pool and the clinical ages were unknown.

Histological examination showed a low prevalence of caries lesions in the outer enamel. However, lesions were present in the enamel of the cavity wall in 54% of specimens, whether the margin was defective or sound. The occurrence of caries in dentin surrounding the amalgams was 17%. The authors stated that if the results of this study are applicable to all occlusal amalgams with defective margins, it would appear that a defective margin alone is not an indication that a restoration needs to be replaced.

**BIO-PROBE COMMENT:** The dental profession widely regards the performance of dental amalgam, even though few controlled studies have been conducted that actually evaluate clinical performance. This is another of the recently published studies indicating that empirical opinions of dental amalgam are not necessarily valid.

Danielsson, BR; Khayat, A; Dencker, L.
Foetal and Maternal Distribution of Inhaled Mercury Vapour in Pregnant Mice: Influence of Selenite and Dithiocarbamates.

**ABSTRACT:** The distribution of mercury after inhalation of metallic mercury vapour (6-8 mumols $^{203}\text{Hg}^0$/kg b. wt.) was studied in pregnant mice (day 17 of gestation) after pretreatment with selenite (10 mumols Se/kg b. wt. intraperitoneally 1 hr before inhalation), thiram, disulfiram or diethylidithiocarbamate (1 mmol/kg orally 2 hr before inhalation of $^{203}\text{Hg}^0$). For comparison, the effects of thiram, disulfiram or diethylidithiocarbamate on the distribution of mercury after administration of ionic mercury (7 mumols $^{203}\text{HgCl}_2$/Kg b. Wt. intravenously) were also studied.

Selenite pretreatment caused a longer retention of mercury in maternal tissues but decreased the foetal concentrations after $^{203}\text{Hg}^0$ inhalation, similarly to what has been shown previously after administration of ionic mercury ($\text{Hg}^{2+}$). Pretreatment with the three dithiocarbamates markedly increased the uptake in maternal brain and fat and decreased the foetal concentrations after intravenous injection of $^{203}\text{HgCl}_2$. In contrast, no change in foetal uptake and only slight changes in maternal tissue concentration of mercury were observed after treatment with the dithiocarbamates followed by inhalation of $^{203}\text{Hg}^0$, compared with $^{203}\text{Hg}^0$ inhalation alone.

The results are in favor of a firmer binding of mercury after $\text{Hg}^0$ inhalation, when oxidation of $\text{Hg}^0$ to $\text{Hg}^{2+}$ occurs intracellularly, than after $\text{Hg}^{2+}$ injection.

**BIO-PROBE COMMENT:** This study provides confirmation of two very important features of exposure to mercury vapor ($\text{Hg}^0$). Researchers at the University of Calgary have demonstrated that mercury from amalgam dental fillings in the teeth of pregnant mothers transfers to their unborn babies. This study confirms the finding that exposure to mercury can pass from pregnant females to fetal tissues. The second feature is that exposure to mercury in vapor form is more serious than exposure to inorganic mercury (even when it is injected directly into the body).

Friberg, L; Mottet, NK.
Accumulation of Methylmercury and Inorganic Mercury in the Brain.

**ABSTRACT:** Differences in metabolism between different mercury species are well recognized. Conclusions that only a minor demethylation of methylmercury takes place in the brain are based primarily on results from short term studies. Studies on animals and humans indicate that the one-compartment model for methylmercury cannot be used without reservations. Inorganic mercury has a complicated metabolism. Studies on rats and monkeys indicate that inorganic mercury penetrates the blood-brain barrier only to a very limited extent. After exposure to metallic mercury vapor, inorganic mercury, probably bound to selenium, accumulates in the brain. A fraction of the mercury is excreted, with a long biological half time.

**BIO-PROBE COMMENT:** The authors are widely respected authorities on mercury toxicology. Their
remarks indicate that mercury vapor is at least as serious a poison as is methylmercury, contrary to public statements issued by dental amalgam advocates (who obviously are not expert on mercury toxicology).

Sharma, GP; Soti, RC; Chaudry, A; Ahluwalia, KK.  
Chromosome Aberrations and Dominant Lethals in Culex Fatigans due to Mercuric Chloride.  
ABSTRACT: The mosquito, Culex fatigans, was used for determining possible mutagenic potential of mercuric chloride, using chromosome aberrations and frequency of dominant lethals as the parameters. As many as 27.75 ± 0.85% aberrations against 2.75 ± 0.35% (P less than 0.001) in the controls were observed in the chromosomes of the animals treated with 0.001 microgram/ml of mercuric chloride. Similarly the frequency of dominant lethals was statistically significant in the treated lot. The results indicate that mercuric chloride is genotoxic.

Lu,KP; Zhao, SH; Wang, DS.  
The Stimulatory Effect of Heavy Metal Cations on Proliferation of Aortic Smooth Muscle Cells.  
ABSTRACT: Heavy metal cations Cd²⁺, Pb²⁺, and Hg²⁺ were added to substitute for Ca²⁺ in culture media to study their effect on the relationship between CaM and the proliferation of cultured rabbit aortic smooth muscle cells (ASMC). It was found that all the heavy metal cations studied stimulated the proliferation of ASMC in varying degrees, increased the CaM content in cells at late G1 stage and decreased the activity of cAMP PDE. These results suggest that the adverse effect of heavy metals may be related to the pathogenesis of atherosclerosis and hypertensive disease.

Siblerud, RL.  
The Relationship Between Mercury From Dental Amalgam and the Cardiovascular System.  
ABSTRACT: The findings presented here suggest that mercury poisoning from dental amalgam may play a role in the etiology of cardiovascular disorders. Comparisons between subjects with and without amalgam showed amalgam-bearing subjects had significantly higher blood pressure, lower heart rate, lower hemoglobin, and lower hematocrit. Hemoglobin, hematocrit, and red blood cells were significantly lower when correlated to increased levels of urine mercury. The amalgam subjects had a greater incidence of chest pains, tachycardia, anemia, fatigue, tiring easily, and being tired in the morning. The data suggest that inorganic mercury poisoning from dental amalgam does affect the cardiovascular system.

BIO-PROBE COMMENT: These three studies are examples of what we may look forward to in the near future. Scientists are devoting increased effort to the investigation of the pathologic effects of exposure to heavy metals. The recent University of Calgary research, published in top scientific journals, has now alerted the medical scientific community of the chronic exposure to mercury in subjects with amalgam dental fillings.

Arvidson, B.  
Accumulation of Mercury in Brainstem Nuclei of Mice after Retrograde Axonal Transport.  
ABSTRACT: Adult mice were injected intramuscularly in the region of the vibrissae muscles on the left side of the nose with a small volume of mercuric chloride dissolved in distilled water. The animals were sacrificed after 1-6 weeks and fixed by whole-body perfusion. Frozen sections were taken from different levels of the brain stem and from the kidney. The sections were subjected to silver acetate autometallography for visualization of mercury. Mercury was found to accumulate in neurons of the facial nerve nuclei, of the motor trigeminal nuclei and of the trigeminal mesencephalic nuclei of the brain stem, after retrograde axonal transport. Mercury was also demonstrated in proximal tubular cells of the kidney. The mechanism for uptake of mercury at the neuromuscular junctions, and the fate of mercury within neurons are analyzed. The possible significance of retrograde metal transport for the development of motor neuron disease is discussed.
Arvidson, B; Arvidson, J.
Retrograde Axonal Transport of Mercury in Primary Sensory Neurons Innervating the Tooth Pulp in the Rat.

ABSTRACT: The pulp cavity of the first upper molar was exposed unilaterally in adult rats with a dental drill and about 1 microliter of mercuric chloride was injected into the coronal pulp. The rats were sacrificed after 1-24 days and frozen sections from the trigeminal ganglia were subjected to silver acetate autometallography for demonstration of mercury. Mercury was found to have accumulated in neurons of the ipsilateral trigeminal ganglion by retrograde axonal transport. The possible implications of this finding are discussed.

BIO-PROBE COMMENT: These two studies confirm the earlier findings of Dr. Alfred Stock and, more recently, Dr. Patrick Stordebecker. Most consideration of mercury exposure from amalgam dental fillings has been directed to the inhalation of mercury vapor. This exposure route involves the passage of mercury vapor into the body through the lung alveoli, into the blood, then into body tissues. Part of this mercury will be oxidized before it can penetrate the blood-brain barrier and enter the tissues of the central nervous system. This confirmation of the axonal transport system for mercury means that mercury in the oral and nasal cavities can pass directly into CNS tissues without encountering the blood-brain barrier, a very significant finding.

CASE HISTORY
We are beginning to receive reports of patient health recoveries after removal of mercury amalgam fillings resulting from the "60 Minutes" program. One of the more dramatic cases is now being documented. A young woman in Las Vegas, Nevada had a longstanding affliction with motor neuron disease. It was finally diagnosed as bulbous lateral sclerosis, a syndrome akin to amyotrophic lateral sclerosis (Lou Gehrig’s Disease). The extreme seriousness of the condition is well acknowledged and the cause is unknown. After viewing the "60 Minutes" program, the patient decided that she had nothing to lose by having the mercury amalgam fillings removed. Although it is still too early to determine final results, her initial health improvement after amalgam removal has been dramatic and encouraging.

FORUM
IAOMT MAY MEETING
The International Academy of Oral Medicine and Toxicology will hold its Regional Meeting in Pittsburgh, Pennsylvania on 18-19 May 1991. The one day seminar on Saturday, 18 May, will feature presentations by Murray J. Vinoy, D.D.S., Michael F. Ziff, D.D.S., William L. Marcus, Ph.D., Robert Carton, Ph.D., and Janette D. Sherman, M.D. Doctors Marcus and Carton are scientists with the U.S. Environmental Protection Agency and will address the health effects of fluoride. There is no registration fee for IAOMT members. Registration is $75.00 for non-member doctors or $125.00 for staff plus doctor. Add $15.00 for late registration (at the door). The Board meeting of the I.A.O.M.T. will be held on Sunday morning, 19 May 1991. Meeting chairman is Michael Pawk, D.D.S. 210 N. Washington St. Butler, PA, 16001. (412) 285-3305.

The meeting will be held at the Sheraton Hotel at Station Square. 7 Station Square Dr. Pittsburgh, PA, 15219. IAOMT special room rate is $82.00 per night. Call (412)261-2000.

IAOMT ANNUAL SCIENTIFIC SESSION
The Annual Scientific Session of the International Academy of Oral Medicine and Toxicology will be held at the Sheraton Inn in Seattle, Washington on 13-15 September 1991. Presentations will be on Friday and Saturday, 13-14 September with a public forum to be held on Friday evening. The 1991 Annual Meeting of the I.A.O.M.T. will be on Sunday morning, 15 September. Meeting chairman is Paul G. Rubin, D.D.S. (206)328-0221. Special room rates at the Sheraton Inn are available by specifying "IAOMT", and may be made by phoning (206)621-9000.