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THE PRACTITIONER'S DILEMMA
Robert E. Reeves, J.D.

The choice of amalgam as a filling material rather than gold, composite, etc., presented no possible legal exposure to the average practicing dentist until recently. Recent statements both orally and in writing would appear to be changing the situation. By acknowledging that some individuals have a hypersensitive allergic reaction to amalgam and that the dentist should become familiar with the symptoms of metal exposure and that patient health histories should include documentation of any sensitivity to metals, the American Dental Association may have taken themselves off the hook to some extent but they placed the average practitioner in a real dilemma, at least from a legal standpoint, whether he or she was aware of it or not.

Given the statements of the ADA, it would seem that practitioners, in order to protect themselves, would have to:

1. Inform all dental patients who have amalgam in their mouth that some people may be hypersensitive and their reactions may be systemic.

2. In any patients in which there is any reason to believe the patient might be hypersensitive to refer them to a physician for determination on that issue before placing any amalgam in the patient's mouth.

The latter would be complying with the ADA's suggestions which specifically state that the dentist should not be doing any kind of patch testing. The former while not specifically contained in any ADA statements is necessary to comply with the legal doctrine of "informed consent". Normally that would only apply when a practitioner is placing additional amalgam in a patient's mouth. But suppose for a moment, a patient comes to a dentist with a considerable amount of amalgam in their mouth and at the same time has an undiagnosed medical problem. The dentist by choice places composite in their mouth and they remain his patient over a period of years. During this time, the patient is suffering from an undiagnosed medical problem, perhaps even unknown to the dentist. If the practitioner does not inform them of the "newly discovered evidence" of hypersensitivity, he could well face a lawsuit and find himself in a very difficult position if the medical problem is later found to be a hypersensitive reaction to mercury from their old amalgam fillings.

Another possible pitfall of the practitioner would be in placing gold or some other metal in the mouth of a patient who has amalgams. There appears to be sufficient statements in the dental literature about the incompatibility of dissimilar metals in the mouth to make this a potentially hazardous situation. Because of this it probably would be more advisable to refer the patient to a physician before proceeding assuming there are no other options, i.e., the placement of braces.
While informed consent can be accomplished simply by an oral statement to the patient, legally there are still pitfalls. It is likely the patient won’t remember and therefore it would probably be best to not just place something in your own records but to actually have the patient read and sign a form.

Even a practitioner placing a composite filling faces some risk because of the tendency of composites to necessitate more root canals if placed improperly. If the practitioner explains to the patient the differences in cost, life span and risks of side effects of the composite and of comparable material such as amalgam and gold then he has met his duty to the patient. Again, it would be advisable to do this by written form.

CERAMIC RESINS AND TOOTH SENSITIVITY
Murray J. Vimy, B.A., D.M.D., F.A.G.D.

Many dentists who have become mercury free have found a great deal of difficulty with tooth sensitivity and pulpal pathology as a result of the alternative materials. The purpose of this article is to address some of the factors which may be causing these problems and to identify some techniques to overcome them.

Elimination of dental amalgam leaves one with few therapeutic alternatives of reasonable cost. Indeed, a quality gold alloy is by far the best. However, the patient’s budget will often stand in the way of total rehabilitation. Our next materials of choice are the posterior composites. These materials have been proliferating like rabbits with each new material hitting the market claiming superiority.

The smart practitioner does not jump from one material to the next. Rather he chooses one material out of a new generation and works with it until he is adept at placing it successfully. When a new generation of materials comes available he will again make another choice.

Rule #1: Keep it simple.

The major problem that we hear today is that of tooth sensitivity and pathology following the placement of a posterior composite. Such difficulty results from two major areas:
1) technique failures and
2) material sensitivity and toxicity.

Technique failure for the most part has resulted from the notion of physical dentin bonding. Recently I have attended a continuing education course by Dr’s. Ralph Phillips and Sheldon Newman. The discussion centered on the possibility of actually developing
materials which will bond to dentin. If I understood the discussion physical bonding to dentin is contra-indicated. Indeed hours were spent discussing the hot new topic, the smear layer. This is the remnants of crushed material left after cavity preparation. The discussion revolved around whether we should keep it or not before bonding.

All this may seem trivial and indeed in my opinion it is! Dentin is a live tissue. We all know that to get a physical bond to dentin we must have a rough surface. To accomplish this some authorities are recommending etching the dentin. Common sense tells one that if you etch open odontoblastic tubules you will most likely bring about the termination of the pulp. 1,2,3,4,5,6

Rule # 2: DON'T ETCH THE DENTIN!! Only etch the enamel.

It is well known in scuba diving circles that air pockets trapped in the fillings or between the fillings and the tooth can be a great source of pain when diving. Similarly such entrapments will respond to the application of hot and cold to the tooth. This is simply the application of the principles of hydraulics. The composite materials by their very physical properties encourage entrapment of a thin layer of air between the material and the tooth. This is one of the reasons for tooth sensitivity and why if you replace the filling with the same material again the sensitivity often vanishes. The second time you eliminate the air entrapments.

Rule # 3: Place copious amounts of bonding material on the tooth prior to placing the bulk material. This helps eliminate the air entrapments.

It has also been suggested that some of the curing lights available on the market today produce sufficient heat that when placed close to the freshly prepared tooth can result in irreversible pulpal damage.

The second source of tooth sensitivity is from reactions to the materials themselves. These reactions can result from some of the following:

1) Reaction to the etching (discussed previously),
2) Histamine reaction,
3) Toxicity or trauma due to material component and
4) low pH of some of the bonding materials.

The composite materials themselves are composed of chemicals many of which are petroleum based. It is very likely that individuals who have sensitivities to petroleum will develop chronic tooth hypersensitivity due to a a pulpal histamine reaction.

Moreover, many of the materials in use have components that are cytotoxic when placed on open odontoblastic tubules. For example, Scotchbond which is a two component system contains phosphoric esters and benzoyl peroxide in Resin A and an alcoholic solution of a tertiary amine and a sulfonic acid salt in the Resin B.
While taking a course given by the 3M Corp. on the use of Scotchbond the instructor said to blow the material with air for 15-20 seconds after applying it to the tooth and then reapply the Scotchbond and reblow it. When questioned as to why this procedure he indicated that this was to remove the excess ethanol. It stands to reason that any alcohol applied to live tissue will have cytotoxic effects and pain. Just try to place some alcohol on an open cut! Moreover, what if some of the ethanol gets trapped under the restoration? What effect will this have on the pulp?

Rule # 4: If you use a material like Scotchbond, mix it in the dish and then blow it before you place it on the tooth. This may decrease the bond strength, we're not sure, but it has cut down on tooth sensitivity in our practice.

Finally, some of the bonding materials may be on the acidic side. Thus, placing these materials on the exposed dentin can cause irreversible pulpititis.

As you can see the use of ceramic resins has many complicating factors. However, the use has to be considered far superior to dental amalgam. In the final analysis the loss of a few pulps is still preferable to the systemic toxicity of mercury released from dental amalgam.

SOME HELPFUL TECHNIQUE HINTS:

1) Always place copious amounts of base under your restoration prior to etching. We use Dycaill which has further reduced the incidence of sensitivity.

2) We have found that the old amalgam condensers make excellent base applicators. They allow you to not only paint the pulpal floor but the axial walls.

3) Let the base dry before you etch.

4) Apply the etch only to the enamel. Bonding is important! If amalgam has a useful characteristic it is that it is self sealing. Composites don't have this advantage. Research indicates considerable leakage. Therefore you must bond.

5) Acid can be carefully applied if it is placed in a tuberculin syringe. This way you can control the placement. Sterilize the tip and reuse it again.

6) Use light curing materials whenever possible. They set faster with greater control, there is less chance of air entrapment, and they are radiopaque.

REFERENCES


*ABSTRACTS/REVIEWS*

One hundred and eighty children with obesity and diabetes, aged 2-15 years, were studied over time for sulfhydryl groups (total, reduced and oxidized glutathione). Deep changes were observed in ratios between the reduced and oxidized forms towards the latter's increase which correlated with the degree of diabetes severity. However, children with carbohydrate metabolism disorders showed no deficit of sulfhydryl groups due to the compensatory stabilization of the synthesis of thiol compounds in childhood. The multiple modality therapy contributed to a relative recovery of the disrupted ratios. It is proposed that the kinetics of sulfhydryl compounds be considered in evaluating the severity of diabetes mellitus. A. M. Yusubova. Blood glutathione levels in children with obesity and diabetes mellitus. Pediatriia, 5:54-55, May, 1984. (English Summary).

*The absorption of inorganic mercury in rats was studied by using ligated gastrointestinal segments and perfusion of small intestine. Poorly soluble mercuric oxide (HgO) as well as mercuric chloride (HgCl₂) was absorbed from the ligated segments in the following order: duodeenum > stomach = jejunum = ileum. The ligation of bile duct decreased the duodenal absorption of HgCl₂, while no change was observed in that of HgO. In the bile duct-ligated rats, the coadministration of bile increased the absorption of HgCl₂ compared to that in rats without the ligation. The absorption of HgCl₂ was increased with an increase of pH of the solution perfused into small intestine. These results suggest that the alkalinity of bile promotes the absorption of HgCl₂. The authors conclude with the following statement: "Gastric acid did not promote the absorption of orally*
administered HgO, although it was expected to promote the absorption by raising its solubility. On the other hand, bile appeared to increase the absorption of HgCl₂, possibly by converting it to the hydroxide forms. Thus, alkaline fluids such as bile may influence the absorption of inorganic mercury from the alimentary canal." Endo, T. et al. Gastrointestinal absorption of inorganic mercuric compounds in vivo and in situ. Toxicology and Applied Pharmacology, 74 (2): 223-229, 1984.

Mercuric chloride stimulates phospholipid hydrolysis and prostaglandin release in 3T3 mouse fibroblasts. This response is distinctly different from that stimulated by other sulfhydryl-reactive agents, but it exhibits a variety of characteristics similar to the phospholipid hydrolysis response stimulated by Ca²⁺ plus inophore A23187. Also, the additivity of phospholipid hydrolytic responses stimulated by Hg²⁺, Ca²⁺ and A23187 is consistent with Hg²⁺ interacting with a Ca²⁺-dependent enzyme(s). These results are consistent with Hg²⁺ acting by a novel, Ca²⁺-mimetic mechanism; i.e., with it entering cells and activating cell processes that are activated by Ca²⁺ in calcium-dependent cell death. Shier, W.T. and DuBourdieu, D.J. Stimulation of phospholipid hydrolysis and cell death by mercuric chloride: Evidence for mercuric ion acting as a calcium-mimetic agent. Biochem Biophys Res Commun, 110 (3):758-765, 1983.

Distribution of inhaled radioactive metallic mercury vapour (²⁰³Hg⁰ in rats and Marmoset monkeys (Callithrix jacchus), with or without pretreatment by ethyl alcohol or aminotrizole (rat), was studied by means of whole-body autoradiography, microautoradiography and scintillation counting of excised organs. Metallic mercury is oxidized by the catalase-H₂O₂ complex (Complex1) to the ionic form (Hg⁴⁺) and is known to be accumulated and retained in organs such as lungs, liver, myocardium, and brain, apparently after local oxidation in these organs. To this list of organs can be added the whole respiratory tract (nasal mucosa, trachea, and bronchi), a number of endocrine organs such as adrenal cortex, thyroid, corpora lutea of the ovaries, and interstitial tissues of the testes, the uvea and retina of the eye, and the salivary glands. In the liver, a regionalized pattern of distribution corresponding to the periportal hepatocytes was observed. Similarly, the subcapsular parts of the adrenal cortex (mainly the zona glomerulosa) were responsible for most of the adrenal mercury oxidation and retention. These organs (liver, adrenal) thus have a reserve capacity to oxidize Hg⁴⁺. This is apparent also by the fact that ethyl alcohol and aminotrizole (known catalase inhibitors) — which depress oxidation and retention in most organs and whole body and thus increase blood concentrations of Hg⁰ — cause an increased retention in most liver and adrenal cells. Khayat A. and Dencker L. Organ and cellular distribution of inhaled metallic mercury in the rat and marmoset monkey (callithrix jacchus): Influence of ethyl alcohol pretreatment. Acta Pharmacol et Toxicol (Copenh), 55 (2):145-152, 1984.
Silver accumulations in the anterior pituitary of argyric rats were demonstrated using a histochemical method that visualizes minute traces of the metal. Silver was localized intra- and extracellularly throughout the anterior pituitary. Intracellular deposits were found within the lysosomes of somatotrophs and gonadotrophs. Extracellularly the grains were located in basal laminae of portal veins and sinusoidal capillaries and in the membrane separating the anterior pituitary and part intermedia. The amount of deposited silver was dependent upon the dose of silver administered. Increasing the dose of silver lactate from 10 to 30 mg resulted in increased deposition, whereas a further increase to 60 mg did not alter the amount of silver deposited. The author's conclude their article by stating: Silver is a potent inhibitor of adenylate cyclase and cyclic AMP phosphodiesterase activity in many organs (Nathanson and Bloom, 1976). The secretory processes of endocrine cells are dependent upon respiratory energy (Jamieson and Palade, 1968, 1971). Changing the ability of the cells to produce cyclic AMP may change their secretory rate. Thus silver may interfere with the level of activity in anterior pituitary cells in at least two different ways. This might have functional significance." Ole-Thorlacius-Ussing and Jorgen Rungby. Ultrastructural localization of exogenous silver in the anterior pituitary gland of the rat. Experimental and Molecular Pathology, 41 (1): 58-66, 1984.

The effects of neonatal CH$_3$-Hg exposure on development and function of peripheral catecholaminergic synapses were examined by measuring tissue norepinephrine (NE) levels and turnover rates and cardiac biochemical responses to sympathetic reflex stimulation. In the rat, cardiac sympathetic neurotransmission normally develops towards the end of the first week postnatally; however, pups given CH$_3$-Hg showed responses to sympathetic reflex stimulations as early as 3 days of age. The accelerated maturation of cardiac sympathetic effect was accompanied by initial enhancement of NE levels and turnover. This effect appeared to be specific to the heart, as kidney displayed subnormal NE levels in CH$_3$-Hg treated animals. Since neonatal CH$_3$-Hg produces heart and kidney overgrowth, we examined the potential role of sympathetic input in altered tissue growth, utilizing chemical sympathectomy with 6-hydroxydopamine (6-OHDA). Sympathectomy inhibited the early phase of renal overgrowth, suggesting that sympathetic nerves participate in the initial effect of CH$_3$-Hg on this tissue; however, 6-OHDA did not influence later phases of renal enlargement nor did it alter the CH$_3$-Hg-induced car dia overgrowth. These results indicate that neonatal exposure to CH$_3$-Hg alters the synaptic development of peripheral catecholamine neurons, which may play a role in some of the subsequent effects on tissue development. Bartolome J. et al. Neonatal methylmercury poisoning in the rat: Effects on development of peripheral sympathetic nervous system. Neuronal participation in methylmercury-induced cardiac and renal overgrowth. NeuroToxicology, 5 (4):45-54, 1984.
Following the outbreak of mercury poisoning in Minimata, Japan (1953–60), much work has been done on the toxicology of mercury—in particular methyl mercury. In this paper, we derive two compartmental models for the metabolism of methyl mercury and inorganic mercury based upon the data which have been collected since 1960....For both methyl mercury and inorganic mercury a long-term compartment needs to be included in accordance with ICRP Publication 30, a 10,000-day half-life is appropriate for this compartment (ICRP80).


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EDITORIAL

I have been receiving an ever increasing number of telephone calls from patients and practitioners alike concerning the requirement to measure electrical currents in the mouth in order to diagnose the correct sequence for removal and replacement of amalgam fillings.

Some of the patients were demanding the name of a dentist in their area who had the necessary equipment to measure the electrical currents in their oral cavity so that they could get their fillings replaced in the proper sequence. Their own dentists were unaware of the need to do sequential removal, although they were routinely using the newer posterior composites. One patient was even willing to pay for the rental of a machine that he could take to his own dentist.

As was stated in the lead article of our last issue "The Medico-Legal Question" "No scientific, physiologic or biochemical justification can be found to substantiate claims that mercury can be permanently "frozen" into a patient's body as a result of improper sequential removal of amalgams." The use of any of the available electric measuring devices to do anything other than demonstrate to the patient that electric currents are present in the oral cavity is not scientifically/legally defensible at this time. If at some future date the hypothesis of sequential removal is proven to have some basis in scientific fact, then and only then should it be incorporated into diagnostic protocols.

The following information has been provided by Dr. Mats Hanson of Lund University, Lund Sweden. In corresponding with Dr. Hanson I asked him if he would give me his thoughts on the measurement of galvanic currents in the mouth and their application to sequential removal.

"You have here two metals immersed in an electrolyte. There is also a small potential over the soft tissue saliva boundary but that can be disregarded since other errors in measurements are much larger.

The two electrodes will be the amalgam electrode and the saliva electrode. The only factor which determines the direction of the
current will be the electrochemical potentials of the electrodes. The magnitude of the current will depend on the contact between probe and amalgam and the degree of oxidation of the under surface of the amalgam and the oxidation of the saliva electrode. The current will rapidly reduce due to the build up of polarization of the surfaces.

The capacity of the battery can be deduced from repeated measurements. If the battery soon can produce current again it is a fresh battery; if it remains discharged it is a weak battery. A fresh battery will every time the surface is scratched by chewing, produce current and thus release metal ions. The direction of current during chewing will not necessarily be the same as that measured with the amalgameter; that’s an artificial situation with an electrode which is not normally present.

If the saliva electrode is the most noble electrode, the current will be negative. This probably means that the amalgam electrode has a lot of easily dissolvable tin or zinc, producing constant migration of metals into saliva and teeth (positive metal ions migrate with the current). When tin is released mercury will also be set free. Since the venous blood vessels in the pulp have direct contact with the vasculature of the brain without the usual venous valves, there is a direct route to the brain. It is sufficient to raise up rapidly to have a back flow.

If positive ions move out from the fillings, electrons will move in the other direction. These will mainly come from chloride ions but can in principle come from anything. The substance which loses electrons will be oxidized. Glutathione and other sulfhydryls are easy targets. Also ascorbic acid, producing dehydroascorbate. Low ascorbic acid produces loose teeth.

If the amalgameter gives a positive current, the amalgam electrode will be the more noble, indicating that the surface layers are low in metallic tin. The release of metal will then probably be more dependent on scratching of the exposed surface, releasing mercury vapor. Old fillings seem to have very much loosely bound or liquid mercury.

The high-copper amalgams are more problematic. They are undoubtedly more noble (in the electrochemical sense) than the ordinary amalgam. On the other hand, the corrosion products from copper are very easily soluble and give no protective surface layer. Copper is also highly toxic. Whether they will give a positive or negative reading depends on the composition of the saliva electrode, whether there is good contact between the probe and the filling etc. It is not possible to say without knowing more about the electrical properties.

However, one important factor is that since the surface remains clean (because of the solubility of copper salts) they should evaporate more Hg. The difference between these "new" fillings and the old binary copper amalgams is not very large. The latter contained about 33% Cu and new one 15-20. The copper amalgams evaporated massive amounts of Hg.
The high-copper, amalgams corrode (according to Jaro Pleva) by general surface corrosion and not especially by oxygen-dependent, crevice corrosion. Hence their good marginal resistance and clean look (electropolishing).

One more point: The normal corrosion process for ordinary amalgam will be crevice corrosion, i.e., the surfaces with lowest \( \text{O}_2 \) tension will dissolve. That can easily be seen on the bottom surface and just beneath the margins.

Corrosion according to the situation created by the amalgameter will be when an upper and a lower filling come into contact or worse, when a gold filling or crown comes into contact with amalgam. Since gold is nobler than anything in the amalgam, such a contact will corrode continuously at a high rate.

If fillings are measured they should be dry since otherwise the saliva will partly short-circuit the amalgam to the electrode."

FORUM

The ADA, NIDR, and Asst Secretary for Health and Human Services have all cited the NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry, July 11-13, 1984 as their basis for concluding that there is not enough documented evidence to warrant discontinuing the use of amalgam as a restorative material in dentistry.

The reliance of the ADA and U.S. Governmental agencies on the conclusions of the NIDR/ADA Workshop prompted the International Academy of Oral Medicine and Toxicology to commission an analysis of the transcript of the Workshop proceedings. The purpose of the analysis was to determine the validity of the Workshop Conclusions based on the scientific documentation presented.

The final document published by the IAOMT contains 35 pages and is titled "A Critical Evaluation of The NIDR/ADA Workshop on Biocompatibility of Metals in Dentistry. I would just like to quote the first paragraph of the Summary "Of the 13 speakers offering presentations at the workshop 4 did not address the subjects of amalgam, mercury, mercury toxicity, or mercury hypersensitivity. Of the nine presenters who did address these topics only three (Dr's. Marek, Newman and Rupp) concluded that dental amalgams do not constitute a health hazard to patients. None of the three offered any primary scientific documentation demonstrating the biocompatibility of dental amalgam fillings in patients to support their conclusions."

If any of you wish to purchase a copy of the IAOMT analysis, it is available from Bio-Probe at $7.00 per copy and $5.00 per copy for IAOMT members. Please make your checks payable to IAOMT.

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The California Dental Society has approved the following new position on mercury toxicity:

"After reviewing recent articles regarding mercury toxicity and claims of some holistic dentists, the Committee recommended that CDS make an allowance for a corresponding amalgam restoration toward the cost of composite restorations used to replace existing amalgams in patients who exhibit toxicity or allergy to mercury. This allowance is available only when the Attending Dentist's Statement is submitted with documented verification by a medical specialist (allergist). Benefits for amalgam restoration replacement are otherwise available only for failure of the existing restoration due to fracture or carious pathology." A teenie crack in the dike???

The next Board Meeting of the International Academy of Oral Medicine and Toxicology will be held at the Hyatt Regency Woodfield, in Schaumberg, Illinois (Suburban Chicago) on Saturday and Sunday, June 22-23, 1985. The Board Meetings of the I.A.O.M.T. are open to the membership and to any qualified individuals contemplating joining. So if you are considering a membership in the Academy and you wish to attend, you are welcome. Call (312) 885-1234 for room reservations. Specify Academy rates ($53.00 per night, single or double). Deadline for room reservations is June 7, 1985. You might also give Dr. Phillip Sukel a call at (312) 253-2240 and let him know you will be attending.

Concerned Dentists For Public Health. That's the name (at least for now) of the new organization that was formed at the April 27-28 meeting that was held in California. Jim Stefan, D.D.S. of Seattle, Washington was elected President. The basic purpose of the new group was defined as follows:

A. The purpose is to establish an ongoing inquiry into the scope of the practice of dentistry for the purpose of promoting the health, welfare and education of the public by involving the following: 1. Health care professionals; 2. Professional organizations; 3. Academic institutions; 4. Governmental agencies and offices; 5. Media; 6. Research scientists; 7. Industry.

B. To safeguard the individual freedom to practice the art and science of dentistry and to employ any method that may be beneficial to the diagnosis, treatment, or prevention of any oral or perioral disease or condition, when the use of such methods are within the competence of the dentist.

Anyone desiring more information on the new organization contact John K. Char, D.D.S., 98-1801 Kileka Place, Aiea, HI 96071, (808) 456-1022 or David Regianni, D.D.S., P.O. Box 458, 101 South Street., Ortonville, MI 48462, (313) 627-4934.

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