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MERCURY TOXICITY FROM DENTAL AMALGAMS

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ABSTRACT

This article reviews the scientific literature and presents the studies and articles relating to mercury toxicity generally and from dental amalgams specifically. Mercury is lost from dental amalgams in many ways ranging from chewing and eating to toothbrushing, and enters the patient's body. The mercury vapour levels of mouths of patients who have amalgams have been measured and have consistently been shown to be much higher than the levels of patients without amalgams. The absorption of mercury is rapid and widespread. The potential for transformation of mercury into methylmercury by oral bacteria has been shown and the transformation into methylmercury in biological systems generally has been known since the early 1970's. Increased levels of methylmercury in dentists and increased levels of mercury in patients with amalgams has been reported. The toxicity of mercury and methylmercury in small doses is extensively documented in the scientific literature. Disturbing evidence exists regarding the health of dental practitioners and their staff. Further intensive research into all aspects of mercury toxicity from dental amalgams is needed as a matter of urgency.

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MERCURY TOXICITY FROM DENTAL AMALGAMS

There has been questioning of the safety of dental amalgams for several decades. In the last few years, this questioning has become more vocal and has prompted a controversy that continues to grow. This review discusses studies and articles which readers should be aware of so that they may assess this controversy in a more informed manner.

Amalgam has been around for a long time. In 1818, a Monsieur Regnart published 'A memoir of a new method of filling teeth.' Presumably then, amalgam was in use for a few years before that.(97) It was introduced into the U.S.A. in about 1833 and criticism began two years later and has continued since. Initially it was deemed unethical and a malpractice for dentists in the U.S.A. to use amalgams because it contained the poison, mercury. In 1855, with the demise of the American Association of Dental Surgeons and the formation, by the supporters of amalgam, of the American Dental Association, this view of what constituted unethical behaviour was deleted. For some years only intermittent reports and criticism continued until 60 - 65 years later, when a significant amount of criticism was directed against amalgam in the 1920's by a Professor Stock, director of the Emperor William Academy in Germany,(1) and a widely respected researcher. He was adamant in his criticisms but was unsuccessful in bringing about a change primarily because of the lack of a suitable alternate restorative material. The next wave of concern started in the 1970's and its momentum continues to this day. It is increasingly supported by evidence that suggests that amalgams are not as innocuous as was previously thought.

To establish Amalgam as a problem it should satisfy some criteria. It seems reasonable to show that

1. mercury is lost from the filling.
2. the lost mercury enters the patient's body.
3. this is toxic or can form a toxic compound
4. enough is released to cause damage
5. remission of problems occurs upon its removal
6. there is evidence of health problems among those who work with it, ie dentists and staff.

MERCURY LOSS FROM AMALGAMS

There is little doubt now that mercury is not retained within an amalgam in a stable form. The American Dental Association has admitted as such in July '84.(2)

It has been stated that placing a resin lining under the filling would prevent this migration but O'Brien and Ryge,(4) editors of 'An outline of dental materials' suggest that it is merely delayed.

Another way of investigating the loss of Hg from amalgam is to analyse the Hg content of old amalgams. If no Hg is lost then the mercury ratio should be much the same as in a fresh amalgam. Pleva reported in 1983,(7) that 5 Y.O amalgams had an average mercury content of only 27%. Radics in 1970 (8) calculated the loss of Hg over a 10 year period, in a mouth with many restorations, to be about 560 mgms...this is a daily loss of 150 mcgms. Phillips and Schwartz (9) tested 100 amalgam restorations. No attempt was made to separate them on the basis of length of service, type of alloy, packing technique etc. Their results showed that the Hg content varied from 28.6% to 61.0% with 25% of the amalgams having a mercury content of 39% or less. This was at a time when, to quote Phillips and Schwarz, "The recommended mercury ratio, usually 60% Hg by weight, always calls for more Hg than is required in the finished restoration"

Rao et al (111) recently published a report about the dissolution of mercury from dental amalgams in saliva. They incubated a number of different brands of amalgam in human saliva at 37 °C. for 24 hours, and found that the amount of Hg released (total) into saliva from all the amalgams tested increased with time during the 24-hour incubation period. the amounts ranged from 0.5 - 1.1 mcgms/ml and 2.5 - 5.0 mcgms/ml at 1 hour and 24 hour respectively.

Svare et al in 1981 (11) showed that Hg vapour levels of up to 87.5 mcgms/m³ after chewing and also showed that the levels were higher in people with more amalgams than in those with fewer amalgams. This point was raised in 1979 by Gay et al (12) who reported in the Lancet that Hg was released from amalgams during chewing.

Most recently we have further confirmation that Hg is lost from amalgams, in the research of Vimy and Lorscheider (109). They demonstrated a 54 times increase in the mercury vapor levels, after chewing, of subjects with amalgams, compared with controls who had no amalgams. They also showed that in unstimulated mouths, i.e. mouths at rest where no chewing took place, the mercury vapour levels in the mouths of subjects with amalgams were nine times higher than in the mouths of subjects with no amalgams. In another report by Vimy and Lorscheider (102), they

calculated these elevated mercury vapour levels and concluded that they constituted a major and significant addition to the body burden of mercury.

Patterson et al. in 1985 (105) demonstrated highly significant increases in the levels of elemental mercury in the breath of subjects after toothbrushing. Their conclusions echoed Vimy and Lorscheider's.

In 1985 Emler (110) measured mercury vapour off amalgam fillings. He concluded ... 'Dental amalgam restorations and mercury vapour exposure was shown to be causally related. Chewing increased the evaporation of mercury from one week old amalgam.'

Evidence that mercury vapour is released through the surface of water is available. Barkowski in 1979 (14) reported levels of 110 mcgms/m³ at the surface of water covering scrap amalgams and Hal Huggins,(15) has recorded up to 200 mcgms/m³.

We are right to be concerned about Mercury vapour. Cutright et al's. study (10) showed that Mercury vapour is rapidly absorbed into the blood stream and accumulates in the lungs, heart, brain, kidneys and liver.

Awareness of the dangers of mercury has changed with time but it is interesting to note that in June 1976 (13) Dr. Eames, a prominent critic of amalgam critics, and not averse to being personally offensive in his criticism (101), stated that not only did scrap amalgam not need to have a lid on the container that it was kept in, but that it didn't even need to be under water. This was in direct contradiction to the recommendations of the American Dental Association that, five years earlier in 1971, (69) wrote that scrap amalgam should be kept in a tightly covered container. This view has been reinforced by the National Health and Medical Research Council of Australia, (100) which in recommendation no.13 of 16 recommendations to do with dental mercury hygiene, said

'salvage all amalgam scrap and store in a tightly closed container. STORAGE UNDER WATER OFFERS NO PROTECTION' (emphasis by author.)

The American Dental Association further recommended in 1982 (108), that amalgam scraps be stored under photographic fixer solution in a tightly closed container.

When the N.H.M.R. Council of Australia tells us that storage under water offers no protection from mercury contamination, and the American Dental Association suggests that scrap amalgam be stored under photographic fixer solution then any thinking dentist must ask himself or herself..... 'what is the status of amalgam fillings in the human mouth and what protective steps can be taken to stop the mercury contamination from what is essentially scrap amalgam stored in teeth.' Perhaps a vacuum sealed mouthguard containing a solution of photographic fixer bathing the teeth could be devised. Aesthetics might be a problem and feeding would have to be intravenous of course, but at least this solution would conform with the recommendations listed above.

It is to be hoped that in light of the N.H.M.R.C. recommendations and the available evidence, that the dental profession will no longer be told that although a small amount of mercury is released from the surface of an amalgam immediately after placement, once it is covered by saliva, then no more is released. Such a position is, quite clearly, totally untenable and unscientific.

MERCURY FROM AMALGAMS ENTERS THE PATIENT'S BODY

Moller in 1978 (3) showed a 40X increase in Hg levels in the pulp one day after an amalgam filling was placed compared with a control where no filling was placed. Freden in 1974 (5) measured Hg levels in gingival tissues which were in contact with amalgams and recorded average levels of Hg which were 49X higher in the test tissues than in the control tissues which were in the same mouth which were not in contact with amalgam.

In 1978, Till and Maly (6) reported levels of mercury of 200-300 mcgms/gm in the root and bone around teeth that contained amalgam, and levels of 1200 mcgms where there was amalgam under gold crowns.

Abrahams et al. (50) demonstrated that blood mercury levels correlated with the number and size of amalgams present in the subject's mouth. Not only that but they also showed that when the amalgams were removed, the blood mercury levels dropped.(51)

A recent study done has shown that the brain and kidney mercury levels clearly correlated with the number and size of amalgam fillings present in the mouths of subjects. This study by Schiele et al. in March 1984 (52) was done on accident victims and the only other correlation that was able to be made was that of increasing levels of brain mercury levels and age, a finding that confirms the fact that mercury continues to be lost from amalgam fillings over the life of the amalgam. The results of this study were confirmed by another researcher, Nylander (53), shortly after, who also showed that the mercury levels were not derived from food.

THRESHOLD LEVELS OF MERCURY.

Are all or any of the previous figures significant? To make some assessment of this, we need to consider evidence which details current threshold limits and physiological changes at low levels of exposure. It is also important to be aware that Threshold Limit Values are arbitrary levels that are set so that no obvious clinical damage is apparent after exposure at those levels. It is also assumed that these levels offer an adequate safety margin. It is also not unknown for levels to be influenced by political and economic considerations. It may be of interest to readers to know that the National Research Council of the U.S.A. has determined in its 1978 report (104) that it is not possible to determine a safe threshold limit. Nevertheless, true to the tradition of governments and bureaucracies everywhere, such an unequivocal statement is not allowed to deter regulatory authorities from setting arbitrary threshold limits, which, unfortunately, are then regarded as safe and acceptable.

The National Institute for Occupational Safety and Health Administration in the U.S.A. has a Threshold Limit Value (TLV) of 50 mcgms/m³, but requires that medical records be kept and reviewed for five years if vapour levels in work areas exceed 40% of that value...in fact, a nonreportable limit of 20 mcgms/m³. Switzerland and the USSR have a maximum exposure limit of one fifth of the USA TLV, ie 10 mcgms/m³. This is for a place of work, presumably eight hour day, five day week. For continuous exposure, the USSR has set a limit of 0.3 mcgms/m³. (16) The TLV in West Germany is set at only one mcgms/m³ (102) Please recall the levels reported by Svare following chewing (11)...up to 87.5 mcgms/m³. This was for chewing gum and not for something hot and/or acidic, where it is reasonable to suggest that higher levels could be expected.

Readers who subscribe to the view that threshold limits are safe levels, are invited to ponder the contradictions inherent in the fact that different levels are prescribed by different countries. Does this mean that citizens of one country are hardier than those of another and able to take a greater level of exposure, or could it mean that nobody really knows what level to set.

CAN LOW LEVELS CAUSE PROBLEMS?

All these levels are very small, and the question is asked, 'Can this low level really be in any way harmful'. The answer is yes. Stock, in 1926 reported symptoms at levels as low as one mcgm/m³, and changes in conditioned reflex activity were reported by Kurnosov in mice at five mcgms/m³, and in cats at eight mcgms/m³. (17) Lauwerys and Buchet reported in 1973 (18) that laboratory technicians exposed to an average level of 28 mcgms/m³ could expect significant changes in enzyme activity, specifically, a decrease in RBC cholinesterase activity and an increase in plasma galactosidase and catalase activity.

Malamud et al. (103) have found that very low levels of mercury inhibit the respiratory burst in human polymorphonuclear leukocytes. The significance of this relates to the fact that the 'respiratory burst' results in the formation of a number of reactive oxygen species that participate in the destruction of microorganisms. The authors state 'In the present investigation, we demonstrate that low levels of Hg(II), (10 - 100 nanograms/ml) profoundly inhibit the PMN respiratory burst. The observation that inhibition occurs rapidly even at low mercury concentration suggests that environmental exposure to this cation may compromise the normal protective function of human PMN's.' (1 nanogram = 1/1,000,000,000th of a gram)

This observation is supported by statements in an article by Ahlrot-Westerland in 1985 (122). In it was said '...in metal syndrome patients, cellular findings of Mercury are distinctive features. The origin of the mercury is unclear but it is known that this and other metals leak from amalgam fillings. In granulocytes, glucose is used at "oxygen burst" in killing of intracellular microorganisms. Since it is known that mercury impairs glucose transport into the erythrocyte, bacterial killing and hence neutrophil defenses might be influenced.'

In view of reports such as the two above, it should not seem too radical to propose that the inhibition of a process which is central to our ability to survive in our environment, would seem to be less than desirable, and that any contamination with the inhibiting agent should be minimised as much as possible.

Langolf et al., in 1981, (19) in their work which examined mercury exposed chlor-alkali workers, conclude their report with..... "Guidelines for permissible exposure levels for many neurotoxic substances were based on prevention of clinically observable effects. Behavioural studies, however, have shown that subclinical effects can occur at levels of exposure which were previously considered acceptable." This view is reinforced by the work of Spyker (59) who demonstrated significant behavioural differences in test animals not obviously clinically affected by mercury.

Varshaeve et al., in 1976 (107) found that lymphocytes from the blood of subjects with amalgams had significantly more chromosomal aberrations than controls. Is it more likely or less likely that in such cases, that an adverse effect on the immune system is present? Are we justified in exposing our patients to such known risks?

Consider the findings of preliminary research showing that mercury in a concentration as low as 20 parts per billion (ppb) was sufficient to stop osteoblastic activity. (Personal communication in Oct 1985 regarding preliminary studies

at the Department of Biology, University of Colorado, Colorado Springs) Nickel needed a concentration of 200 ppb to achieve the same result.(106) (20 ppb = 20 nanograms/gm; 1 nanogram = 1/1,000,000,000th gram)

Symington in 1980, (20) reported two dentists being diagnosed as having Hg poisoning and exhibiting classic symptoms. The environmental Hg measurements revealed vapour levels of five mcgms/m³ and 2-8 mcgms/m³. Both these measurements were well within the TLV's mentioned earlier, except West Germany's.

According to gynaecologists, Koos and Longo, in 1979, (21) fertile women should not be exposed to levels higher than 10 mcgms/m³ and pregnant women should not be exposed to any methylmercury vapour at all. They write.. " while the placenta may protect the fetus during acute mercury exposure it does not protect fetal blood mercury accumulation during chronic maternal exposure." They also state that the guidelines for mercury exposure also should allow for subclinical effects as mercury can cause cellular, subcellular and biochemical alterations not sufficient to produce overt poisoning.

The limits of our understanding of toxicity levels are evident when we consider that based on studies of lethal doses in mice, of thalidomide, a safety factor of 100 was proposed as representing a safe level. It turned out that humans were 150 times more sensitive than mice. (Eyl. T.B.). (22)

As far as the body burden of mercury derived from amalgam is concerned, we have two very recent studies which both conclude that the mercury released from dental amalgams makes a significant and major contribution to man's body burden of mercury. Vimy and Lorscheider in August 1985 (102), measured the amount of mercury vapour released from the surfaces of the amalgams and calculated the daily levels of mercury to which their patients were exposed. They concluded...'Our calculations strongly suggest that dental amalgam Hg makes a major contribution to total daily dose, and that this warrants further quantitative examination'.

Patterson et al.in 1985 (105) measured the amount of mercury released following toothbrushing and calculated daily exposures. and wrote..'we therefore conclude that the levels of elemental mercury in breath derived from silver-tin amalgam fillings represent a significant and undesirable contribution to man's "normal" body burden of mercury'.

Brune and Evje, in 1984,(23) reported that mercury released from the amalgams studied could essentially exceed food and drink intake values. Using the results of previous researchers (24,25), they calculated that a mean mercury excess of about 10 times the food and drink intake would occur during the first day following the insertion of a filling. It is important to note that they also say that man may be frequently exposed to mercury levels that are substantial parts of food and drink intake, from old amalgams during chewing.

It should be quite apparent by now that mercury is a very toxic substance and that it has significant effects on the living organism in very small amounts. It has been said, in an attempt to defend the use of amalgams, that we are exposed to environmental mercury in the form of fish, pollution, smoking etc. and that the mercury from amalgams is not a significant amount. Even if that last comment was true, which it is not, we need to consider, as a profession and as individuals, whether it is acceptable to expose our patients to a known highly toxic agent because they are getting much more from other sources, or, in those circumstances, is it even more imperative to reduce their exposure, not to contribute to it.

THE ROLE OF METHYLMERCURY

In a report by the American Dental Association Council of Dental Materials and Devices, (69) Dr Rupp reported that methyl mercury has no direct significance to dentistry. This was in 1971 and the majority of studies showing methylation of mercury in biologic systems were not published until afterwards. These showed that mercury could be transformed into methylmercury not only within the sediments in rivers and lakes, but also within rat intestines (in vivo) and in human faeces (in vitro). (115, 116, 117, 118, 119) While these studies showed that mercury was not the stable element it had been thought to be, the study which is most significant and the most relevant to Dentistry, is the work by Heintze et al. in 1983 (70) which showed that oral bacteria were capable of transforming the mercury present in dental amalgam into methylmercury. The potential consequences are of concern. Methylmercury is many times more toxic than mercury, by some estimates, up to a 100 times more. Levels as low as 0.1 ppm have been reported to inhibit mitosis and cause chromosome breakages. (22) (N.B. 0.1 ppm = 100 parts per billion = 100 nanograms/gm 1 nanogram = 1/1,000,000,000th of a gram)

Lest it be thought that the evidence linking Dentistry and methylmercury is just theoretical, consider the findings of Cross et al. (79) who found that dentists had significantly higher levels of methylmercury compared to controls. They also found the symptoms of chronic mercurialism. This study is discussed again later in this article.

There is ample evidence of methylmercury's extreme toxicity! Racz and Vanderwater, in a report in 1982,(71) state...'Methylmercury is highly toxic. Chronic methylmercury poisoning is characterised by peripheral and CNS damage. The rate of absorption and distribution of this organomercurial into neural tissue determines the rate of development and the severity of the neural lesion. Furthermore the rate of metabolism and excretion of an organomercurial will greatly influence its neural toxicity. There are differences in the accumulation of the mercury

in different regions of the brain, as well as by the different cell types in these regions. The significance of this variable accumulation of MeHg is not known. MeHg influences a large number of neurocellular functions ranging from inhibition of membrane integrity to alteration in the synthesis and release of transmitter substances. There appears to be a latent period between exposure and development of symptoms.'

Rabenstein, in 1978 (72) states that the first clinically apparent signs are parasthesia of the extremities of the hands, and feet and areas around the mouth. He ends his article with the statement ... 'Because damaged cells in the central nervous system are not replaced by new ones produced by cell division, damage to the central nervous system is irreversible and apparently cumulative.'

Charlebois (73) reports that as symptoms worsen they include constriction of the visual field, deafness, ataxia, muscle spasms, general paralysis as well as damage to the other organs (liver, kidneys, pancreas, bone marrow) which may be masked by the neurological disturbances. While there are clinical signs and symptoms it is important to realize that damage is occurring on a cellular level. Vostal and Clarkson in 1970 (74) reported that although only 1-4% of brain mercury was inorganic, that latent development of structural brain damage and neurological symptoms appeared to coincide with the accumulation of inorganic Hg in the brain.

Ware et al. 1974,(75) reported that all mercury compounds cause severe damage to the blood brain barrier, impairing membrane functions and increasing permeability, and this is thought to play a significant role in allowing methylmercury to reach the CNS in high concentrations. Chang, in 1977 (76) reported that a single injection of methylmercury results in a slow and even accumulation of mercury in the brain, the most concentrated areas being the cerebral cortex and the cerebellum.

The mean biological half-life of methylmercury is 70 days and ranged from 38-189 days (77). Electron microscope studies have shown structural changes at the cellular level. The first observable sign of methylmercury induced damage in neurons is the disorganisation of the rough endoplasmic reticulum and Golgi apparatus (78). Other changes reported include cell shrinkage, plasma membrane distortion, increased size and number of vacuoles, thickened nuclear envelope and affected nuclei or nuclear fragments. The most commonly affected cells were the small neurons of the cerebral and cerebellar cortex.

A study by Koller in 1975, (80) reported that prolonged exposure to subclinical concentrations of methylmercury increased the host susceptibility to a nononcogenic virus but not to an oncogenic virus. He made the point that the methylmercury at no stage produced clinical signs of mercury poisoning or death. Dr. Koller concluded his article with the statement 'Results emphasise that methylmercury not only poses a threat to public health as a toxic agent, but at subclinical concentrations, it may augment infectious agents to produce disease.' This study was preceded by another by Koller, published in 1973 (81) which showed that marked immunosuppression occurred in experimental animals which were given lead, cadmium or mercury but that overt signs of toxicity were not observed.

Pitkin et al. in 1976 (57) found that in the studies which had looked at mercury and pregnancy, that the total concentration of mercury in the placenta ranged from 4.4 times to 2.3 times that of the concentration of mercury in the maternal blood. The authors also note that there is considerable evidence that the principal form of mercury in the fetus is methylmercury. The toxicity of methylmercury is well known and Eyl has reported it as being up to 100 times more toxic than mercury.(22)

Mansour et al. in 1973 (58) reported the results of their work which showed that methylmercury is very easily transmitted across the placenta as well as through the milk. They cited Japanese reports where pregnant women had been exposed to mercury and had no clinically observable effects from this exposure, but whose infants had cerebral palsy.

When we assess the evidence we have to bear in mind that we are dealing with a substance which is very toxic in extremely small doses, and we also have to understand that a diagnosis which relies on the presence of clinical signs and symptoms is characterised by an extreme lack of sensitivity and subtlety. We must be mindful of Spyker et al's. report (59) of significant behavioural differences in apparently clinically unaffected animals. Their report concluded with the comment.. "we believe there is a need to examine subtle parameters when assessing the risks from methylmercury exposure. It seems likely that many people with minor symptoms or subclinical damage have gone undetected."

The A.D.A. in the U.S.A. has warned of the extreme toxicity of methylmercury. It wrote in 1983 (123) '...An international committee classified mercury and its compounds in the order of decreasing toxicity: first is methyl and ethyl mercury compounds, next, mercury vapours and least, the inorganic salts as well as a number of organic forms.' Unfortunately, the article went on to say '..the toxic potential of these forms of vapour is limited to mercury vapour. The other compounds are not used.' In light of Cross et al's finding in 1979, Heintze et al's. study in 1983, and all the other evidence showing that methylation of mercury takes place in biologic systems, a clarification of the above statement would seem to be necessary.

ASSOCIATION OF AMALGAMS WITH TISSUE DAMAGE AND HEALTH PROBLEMS

There is no question of mercury's toxicity. Bleeding gums and alveolar bone loss are classic signs of mercury toxicity. Unquestionably, these same symptoms can be caused by other factors but consider the periodontal implications suggested by the following eight studies.

Till (1978),(26) used germ-free animals and found that mercury in amounts released from dental amalgams could produce the same signs (bleeding and bone loss) as well as inflammation.

Goldschmidt et al. 1976 (27), postulated that amalgam corrosion, leading to the deposition of ions into the tissues could produce concentrations capable of causing cellular death or injury. They suggested that only a small amount of these agents might be necessary for damage to occur. They further commented that they had observed in vitro cytotoxicity and release of histamine from rat mast cells under conditions where insoluble products would be expected to form. They concluded by suggesting that fibroblasts exposed to amalgam corrosion products might synthesize diminished amounts of collagen, and this, together with various physical and chemical insults, could cause, in part, the inflammation and destruction seen in tissue adjacent to amalgams, to products released from the restorations themselves.

The results of this work were supported by Ellender, Ham and Harcourt (106) who studied the effect of an amalgam implant within rat subcutaneous tissue. They showed that amalgam breaks down and that the products become widespread throughout the tissue. They found that a dental porcelain control led to normal wound healing with a mature capsule within 28 days but that amalgam took 100 days or so and even then they observed continuing metabolic interference to susceptible fibroblasts resulting in deranged collagen formation. One of the comments made in the article was as follows '...the changes caused by the dental amalgam are probably attributable to silver and mercury, BOTH OF WHICH ARE KNOWN TO BE CYTOTOXIC AS WELL AS CARCINOGENIC.' (author's emphasis). Their report concluded '...clinical use of dental amalgam results in release of corrosive products which may retard resolution and modify repair in inflamed healing gingival tissues and along with other insults may compound the problem and complicate management of periodontal disease.'

The above study did not consider mercury levels or its distribution throughout the body but based on other research we could expect increased levels of mercury in various organs throughout the body. The effects of such contamination on the immune system are discussed in a later part of this article.

More interesting results come from a study reported by Catsakis and Sulica, from the Georgetown University School of dentistry in Washington.(29) They reported a case of persistent periodontitis which did not clear up, despite constant periodontal therapy up to and including periodontal surgery, until all the amalgams were removed. The periodontal problem had persisted for seven years but after the amalgams were removed, the periodontal condition healed quickly and the tissues remained healthy for a period of more than two years up to the time of publication of that report.

Fisher et al. (30) reported a study where 54 amalgams were placed in 43 patients and followed up to four years. Yearly measurements were made between the alveolar crest and the apical margin of the fillings in the experimental group and the cemento-enamel junction and the alveolar crest in the control group. They found that in the experimental group the level of alveolar crest resorption was almost twice that of the control group, i.e. 0.8 mm vs 0.45 mm.

Consider the above in the light of the work of Freden in 1974. He measured the amount of mercury in tissues in contact with amalgams and found average levels 49 times higher than control tissues from the same mouth.(5) Is it reasonable to postulate, in light of our knowledge of the extreme toxicity of mercury, that some deleterious effect could be expected in tissues that have 49 times more mercury compared with tissues which have no mercury?

Consider also the findings from some preliminary research mentioned earlier, that mercury in a concentration as low as 20 parts per billion (ppb) was sufficient to stop osteoblastic activity. (20 ppb = 20 nanograms per gm = 20/1,000,000,000th of a gram.) Nickel needed a concentration of 200 parts per billion (ppb). to achieve the same result.(106).

Finally, consider the findings of Koivumma & Makila (28) who reported that, of a variety of materials, amalgam, in a human mouth, attracted more plaque than any other material.

Other physical responses to amalgams have been reported frequently over the last few decades. Bergenholtz in 1965,(31) described the disappearance of multiple polypoid hyperplasia in 2 patients following the removal of their amalgams. He postulated that high levels of Copper and Zinc in the tissues were responsible. Unfortunately, he omitted measuring the mercury levels. Fernstrom in 1962, (32) reported a case of oozing eczema, Frykholm in 1957,(33) observed along with Fernstrom, that the dermatitis was most marked on the side of the body where the amalgams had been placed. Fisher in 1974,(34) reported a case of stomatitis, urticaria and an eczematous flare of a facial eruption after a mercury amalgam was placed. These symptoms persisted until the amalgam was replaced.

Juhlin and Ohman in 1968,(35) reported a patient who presented with an inflammatory reaction to a tattoo and with erosions of the oral mucosa that was in contact with old amalgam fillings. Further cases of urticaria, weeping eruptions, edema and fever have been reported (36,37,38,39,40). Janet Bauer and Howard First (41), in their 1982 review, cite 30 references, some of which have been mentioned, which describe physical reactions to dental amalgams. Most recently Mobachen et al. (42) reported 67 patients with oral lichen planus. 64 of these had dental amalgams in contact with the mucosal lesions. 11 of these (16%), reacted positively to at least one of the mercury compounds, compared with only four people (8%), in the control group. This group is currently evaluating the results of amalgam elimination in patients with and without allergy to mercury.

Eversole and Ringer (43) reported a study examining the incidence of oral lichen planus. They concluded that oral lichen planus subjects show a higher correlation with delayed hypersensitivity to dental materials than a control population. Unfortunately, the patients with positive reactions refused to have their restorations replaced and the researchers were unable to show a cause and effect relationship. They did however, cite a number of studies which did.

Finne et al. (44) patchtested 29 patients with Oral Lichen Planus. 18 of these (62%) had a contact allergy to mercury. In a control group, the frequency of mercury allergy was only 3.2%. 4 of the patients had their amalgams removed and of these, three experienced complete resolution of their symptoms and the 4th showed considerable improvement. The authors state... " It would seem possible to draw the conclusion that contact allergy to mercury in amalgams may be an etiological factor in oral lichen planus." They go on to state that all patients are now tested for mercury allergy and when positive, the amalgams are removed and in the author's words..." this has proved to be a most effective treatment, especially in erosive lichen planus since the erosions and also the subjective symptoms such as burning and soreness of the affected mucosa disappear within a couple of weeks." It should be noted at this point that the lack of a reaction to a patch test is not necessarily indicative that there is no effect on the body by mercury. Bergenholtz reported the regression of polypous hyperplasia after removal of amalgam fillings yet in that report there were negative results to the patch tests of all the components of amalgam, including mercury.

Banoczy et al. (45) have reported similar findings to those of Finne et al., except that they attribute the problem to oral galvanism.

Reaction to mercury can be latent and can be triggered any time and does not have to be triggered by the form to which the person was originally sensitized. Nakayama (46) studied patients who presented with allergic rashes diagnosed as mercury exanthema. He concluded that this contact dermatitis was triggered by exposure to mercury vapour. He found however that the original sensitization was brought about by other forms of mercury and not mercury vapour.

As far as the incidence of allergic reaction to mercury is concerned, we have consistent evidence of a substantial percentage of people being allergic. Djerassi and Berova's work in 1969 (47) reported up to 27% of allergy prone people reacted to mercury. White and Brandt in 1976 (48) reported an increase of sensitivity to mercury among dental students from 2% in their first year to 10.8% in their final year. Most recently, Miller et al. (49) found a significant correlation between the number of amalgams and the incidence of mercury hypersensitivity. The number of subjects in the four groups tested who demonstrated mercury hypersensitivity were 31%, 27%, 32% and 39%.

Kuntz et al. in June 1982 (55) reported... 'Stillbirths as well as history of blood defects, exhibited significant positive correlation with background mercury levels. Patients with large numbers of dental fillings exhibited a tendency to higher maternal blood mercury levels.

HEALTH OF DENTISTS AND DENTAL STAFF

When we look at studies discussing the health of dentists and their staffs, there are some disturbing aspects.

1. the studies which exist do show that being a dentist or a dental staff member is a health risk.
2. the studies are not enough in number.
3. there seems to be little awareness within the dental profession that their health is at risk, and no obvious demand that more studies be done.
4. some of the studies raise more questions than they supply answers. No follow up studies appear to have been done to answer those questions.

Vandenburg in the JADA June 1977,(54) reported a clinical blood serum Hg test of dentists and auxiliaries and showed that >50% have above normal serum Hg levels.

Shapiro et al. (60) reported the results of a study which showed that at least 50% of the dentists who had high mercury levels had peripheral nerve dysfunction. None had any of the classic signs of mercurialism. There was also a high incidence of Carpal Tunnel Syndrome. The description of these dentists agreed generally with previous descriptions of low level chronic Hg exposure in human populations.

Symington in 1980, (20) reported two dentists being diagnosed as having Hg poisoning and exhibiting classic symptoms. The environmental Hg measurements revealed vapour levels of five mcgms/m³ and 2-8 mcgms/m³. Both these measurements were well within the TLV's mentioned earlier, except for West Germany's.

Cross et al., in 1978,(79) reported a study which measured the levels of Mercury, Methylmercury, and the MeHg/Hg ratio and found highly significant differences between dentists and controls. They found that dentists had an average of almost five times higher levels of methylmercury than controls. They noted the symptoms of chronic mercurialism... depression, irritability, memory failure, difficulty concentrating, hand tremor. None of these are startling symptoms but they are real nevertheless.

Smith in 1978 (61) reported the case of three dentists suffering with classical signs of mercury poisoning.

The difficulty in establishing a diagnosis of mercury toxicity is illustrated by the report from Iyler et al. (62) who described the case of a dentist in whom a variety of symptoms were caused by exposure to mercury. The researchers also demonstrated the changes which took place in the conduction along sensory nerve fibres.

Nixon et al. in 1979 (56) reported a study in which 1615 female dentists were questioned about the pregnancy experience. Results showed female dentists had a significantly higher spontaneous abortion rate. No reason was suggested but research to determine the reason should be done as a matter of urgency. A good starting point would be the studies by Pitkin and Mansour.(57, 58).

Cook and Yates (63) reported the case of fatal mercury intoxication in a dental surgery assistant. There had been no previous evidence of chronic mercury poisoning in the victim, although it was pointed out that some of the symptoms may have been overlooked. In discussing this case, the authors speculate that one of the explanations could be that she suddenly developed a sensitivity to mercury without there being any change in tissue mercury levels. This is an interesting comment when you consider Mantyla's report (64) that mercury poisoning can occur after prolonged sensitisation.

Dentists might care to alert their staff to the report by Cooley et al. who, in 1985 (113), found that high levels of Hg vapour were produced when amalgam contaminated instruments were sterilized. Their study also confirmed a previous study (114) which found that once a sterilizer was contaminated with mercury, it continued to emit mercury vapour each time it was used. The average levels reported by Cooley et al. were 270 mcgms/m³. This is more than 13 times the nonreportable U.S.A. TLV of 20 mcgms/m³, and 270 times the West German TLV. Paper bags reduced this to 30 mcgms/m³ which is still above the nonreportable U.S.A. tlv of 20 mcgms/m³. Cooley et al. also note that the heat expelled small droplets of mercury with the resultant contamination of the sterilizer and the continued emission of Hg vapour. Whoever then opens the sterilizer door is exposed to the sudden rush of mercury vapour. We are reminded by Cooley et al. that inhaled mercury vapour is rapidly absorbed into the blood stream.

Fox and James (65) report that dentists aged 45-54 have the highest deathrate in this age group and the 2nd highest deathrate during ages 55-64, and Ship and Shapiro (66) expressed concern regarding the high incidence of suicide, anxiety and depression and related problems in dentists.

When we look at 2 studies from the ADA (67,68) on the deaths of American dentists we see that all deaths from nervous disorders, senility, and illdefined conditions in the periods 1961-66 and 1968-72 were 13.17% and 10.77%. This compares with the population at large of 11.37% and 10.11% for the same period. These figures were used in this review because they would reflect possible effects of mercury on practicing dentists. The main problem with these two studies is that we don't know what proportion of the dentists represents clinical restorative dentists who would be in contact with amalgams. For example, what would the figures be if only dentists who were in general practice were considered, and groups such as orthodontists, periodontists, academics etc. were excluded. There is also no attempt to differentiate between dentists who had amalgams in their mouths and those who didn't. Another problem is that the cause of death doesn't necessarily reflect the health history. What needs to be done, is that in future studies, these figures should be sorted as suggested, and detailed health histories obtained.

Such a study should be one of the highest priorities!

One's sense of apprehension is not eased by a report presented at a symposium on Occupational Health in September 1985 in Italy. Ahlbom et al. (120) looked at the incidence of brain tumours among dental personnel. They studied 9,241 personnel.. 3,454 male dentists, 1,125 female dentists and 4,662 female dental nurses. They found that each dentist and dental nurse had a risk of having brain cancer which was TWICE that expected of the population at large. They concluded....'it seems most likely that the origin is some occupational factor common for dentists and dental nurses.' They did not state that mercury was responsible but prior to this finding, others have

suggested a possible mechanism. Arrhenius (121) hypothesized that methyl mercury might enhance the tumour inducing effect of certain amines, in vivo, by inhibition of enzymes involved in detoxification, thereby leading to an accumulation of carcinogenic intermediates.

After reading the above articles, can readers still accept, with complete equanimity, reassurances that dentists' health profile is as good as, or better than, the average,

THE IMMUNE SYSTEM AND MULTIPLE SCLEROSIS

The most recent work linking amalgams and immunosuppression, is that reported by Eggleston in May 1984 (82). Dr Eggleston measured the T-lymphocyte count of three patients before treatment and then after treatment. The first patient had six amalgams replaced, resulting in a 53% increase of T-lymphocytes count. Reinsertion of four amalgams within temporary fillings resulted in a decrease of 24.7%, and subsequent removal of these four amalgams resulted in an increase of 30%. The other two patients had similar results. One of these had amalgams replaced, the other had a nickel based crown replaced with an all porcelain crown. To put this into perspective, Dr Eggleston points out firstly that the T-lymphocyte count does not vary by more than 10% and rarely more than 5% in an eight week period. Secondly, T-cell lymphocytes are a very important component of the immune system. Human T-cell lymphocytes can recognise specific antigens, execute effector functions and regulate the type and intensity of virtually all cellular (T-cell, B-cell) and humoral (antigen) responses. Thirdly, an abnormal T-lymphocyte count can increase the risk of cancer, infectious diseases, and autoimmune diseases. (83-92)

We had some indication of the effects of mercury on the immune system when Koller's (80, 81) work was published. He showed, in animal studies, that marked immunosuppression occurred but, and this is an important but, it occurred with no overt signs of toxicity. i.e. there were no clinical signs or symptoms. We need to keep reminding ourselves of this as so much of our thinking about the presence or absence of disease is predicated on the presence or absence of clinical signs and symptoms.

Readers may be surprised to learn that there are a number of researchers who believe there is a clear connection between amalgam, dental caries, mercury and multiple sclerosis.

2 separate studies have been shown how mercurialism (mercury poisoning) brought about signs and symptoms which resembled Amyotrophic Lateral Sclerosis. (94, 95).

Craelius reported a study in 1978 (93) in which he found that rates of death from M.S. in Australian states related linearly to the numbers of decayed, missing and filled teeth. He also found positive correlations between MS death rates and the DMF in the USA and 45 other countries. He commented ... 'the possibility that the close correlations found between MS and dental disease are due to chance is extremely unlikely, and it has been shown that MS prevalence varies with actual rates of dental disease and not simply with dental indices which are influenced by levels of dental care. Increases in the prevalence of dental caries both in Sth Africa and in the Scottish Islands have been followed by increases in the prevalence of M.S.'

Craelius hypothesised that dietary factors were involved and didn't mention amalgams. We should, in response to his statement that increases in the prevalence of dental caries have been followed by increases in the prevalence of M.S., ask the question..."what is the consequence of increased caries?" The answer would reasonably be expected to be...more fillings (usually mercury silver amalgams).

The most direct report stating a clear connection between Multiple Sclerosis and amalgams comes from Dr Theodore Ingalls, Director of the Epidemiology Study Centre, Framingham Union Hospital in Massachusetts. (96) He states....' Slow, retrograde seepage of ionic mercury from rootcanal or class five amalgam fillings inserted many years previously, recurrent caries and corrosion around filling edges and the oxidising effects of purulent response may lead to M.S. in middle age.'

Dr Baasch, in 1966 (97) put forward the proposition that a great number of the unexplained observations concerning M.S. would be explained by mercury leakage from dental amalgam fillings. The most interesting of these include..

1. No known cause.
2. No comparable spontaneous animal disease.
3. Onset characteristically after puberty. Onset after 45-50 years of age comparatively rare.
4. Course consists mostly of attacks and remissions, rarely of gradual progression.
5. Onset frequently in the head region.
6. Unusual geographic distribution.
7. A disease of civilization.
8. Social preference for well-to-do populations.
9. Familial and local aggregation.

10. Apparent connection with diet.
11. First certain appearance in France in 1818.

This proposition has not been widely accepted. Unfortunately it has also not been disproved or even put to the test.

Stortebecker, a Norwegian neurologist published an extensive study in 1961 (98) in which he discussed the effects of toxins from dental infections on the central nervous system. He described a number of cases where epilepsy, vertigo, headache, optic neuritis and Multiple Sclerosis were successfully treated by the removal of some or all of the teeth. In this study Stortebecker was concerned only with the toxins being produced by infected and nonvital teeth and he did not consider the possibility of mercury toxicity or response to the amalgams. It is quite apparent from the photographs and X-rays accompanying his study that the teeth which were removed were very heavily filled with mercury based silver amalgams. It is interesting to read some of the symptoms listed and compare them with the symptoms of mercurialism.

- impaired memory
- depression
- smell and taste distortion
- photopsies of the visual field
- fatigue.

In his article, Currier (99) looked at M.S. and the dental connection and said in his summary.... 'a series of patients with multiple sclerosis was personally interviewed for historical factors over a 10 year period. It is thought that the amount of dental work they had before the onset may be unusual and might bear further investigation'. He also commented earlier in his paper... 'It is our understanding that in China and India, where M.S. is virtually unknown, both dental care and dental caries are infrequent, teeth being lost from periodontal disease.

SUMMARY

It has been asserted that amalgam is safe because it has been in use for 150 years. It should not need to be pointed out that length of time of use is not proof of safety. It has also been asserted that there are over 100 years of studies showing that amalgam is safe. The fact is that any reader who asks for a bibliography of these studies will find a remarkable and, in view of the intensity of the debate in the U.S.A., a quite disturbing paucity of material. Readers are invited to try this for themselves.

It can be argued that restorative dentistry is the science of specific surgical implants. The teeth and the mouth are, after all, part of the human body and to think that we can place restorative materials into human tissue with scant regard to the overall consequences is a luxury we can no longer afford.

There is no valid reason why our materials should not be subject to continuing reviews of their suitability and biocompatibility as new techniques to measure these parameters become available.

There is also no valid reason why our materials should not be tested against standards of safety which change as our knowledge and techniques improve, and discarded if they fail the new tests.

Standards change, and our profession's guides must be increased knowledge, facts and objectivitynot sentiment! We have at least three precedents in recent times, of complete about-turns in attitudes to the health consequences of materials which were previously used extensively, and for a very long time. They are..the use of asbestos, lead in paints and lead in petrol.

Readers are invited to give serious thought to the next two statements which conclude this review.

First, the view of Wolff et al., who in 1983, in the journal 'Neurotoxicology', (112) wrote...

'There is adequate evidence that dental amalgam restorations during and after placement, result in the release of Hg into the patient's body. Whether the Hg is released from amalgam is due to placement procedures, surface abrasion, or later corrosion breakdown, there is evidence that a low level Hg release continues for years. IT IS GENERALLY AGREED THAT IF AMALGAM WAS INTRODUCED TODAY AS A RESTORATIVE MATERIAL, IT WOULD NEVER PASS F.D.A. APPROVAL' (author's emphasis)

and secondly, the general finding of the National Research Council of the U.S.A., in their 1978 report titled 'An assessment of Mercury in the Environment' (104)

'MERCURY COMPOUNDS HAVE NO KNOWN NORMAL METABOLIC FUNCTION AND THEIR PRESENCE IN THE CELLS OF LIVING ORGANISMS, INCLUDING HUMAN BEINGS, REPRESENTS CONTAMINATION FROM

NATURAL AND ANTHROPOGENIC SOURCES. IN VIEW OF THE TOXICITY OF MERCURY AND THE INABILITY OF RESEARCHERS TO SPECIFY THE THRESHOLD LEVELS OF TOXIC EFFECTS ON THE BASIS OF PRESENT KNOWLEDGE, ALL SUCH CONTAMINATION MUST BE REGARDED AS UNDESIRABLE AND POTENTIALLY HAZARDOUS.'

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