Dental amalgam and mercury poisoning

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Introduction

Dental "silver" amalgam, a mixture of approximately 50% mercury with a powder of copper, silver, tin and zinc, has been in widespread use since the late 19th century. After considerable initial toxicity concerns by the professions it was soon uncritically endorsed by all western dental associations with successive generations of dental students graduating with a firm belief in amalgam being a safe, effective and durable restoration.

As a restoration for dental decay, it has some useful properties being cheap, durable and easy to use. The tooth does not even need to be dry or the surfaces etched as with the more technically demanding microcrystalline composites. It is therefore a pity that it poisons people as mercury is an accumulative toxic element. The time taken, the degree of harm and the presentation will merely depend on the individual's genetic makeup and the dose.

In 1991, the World Health Organisation (WHO) in an up-date on inorganic mercury, listed dental amalgam as being the largest source of mercury vapour in the non-industrial populations at up to six times the amount from all other combined sources.[1] The WHO based their conclusions on an average of 6-8 fillings. In 1992, the Swedish Government passed legislation to start a 5-year phase-out of dental amalgam that would also allow dentists to be properly trained in safe amalgam removal procedures. In the following decade, the legislation was amended to approve State funded dental treatment when a doctor diagnosed mercury toxicity. However, following escalating complaints that doctors were not helping, the legislation was finally changed so that from 2005 any Swedish citizen could get subsidised amalgam replacement if they thought that they were suffering from adverse mercury effects. This remarkable and so far unique legislation was based on the findings of the Swedish Dental Materials Commission that included representatives from the Swedish Dental Association, the dental schools, the Swedish National Board of Health and Welfare, and the Swedish Association of Dental Mercury Patients. The Commission had tasked Professor Emeritus Maths Berlin with giving an updated risk analysis in environmental medical terms on mercury in dental fillings, based on an overview of scientific literature published between 1997 and 2002 and current knowledge. Berlin had previously led two World Health Organization Task Groups with one on inorganic mercury and the other on methyl-mercury.

The U.S. EPA mercury health standard[2] for elemental mercury exposure (as vapour) is 0.3 micrograms per cubic meter of air (0.3 ug/m^3). For the average adult breathing 20 m^3 of air per day[3], this amounts to an exposure of 6 micrograms (ug) per day. The corresponding tolerable daily exposure developed in a report for the Canadian Health Agency, Health Canada, is .014 ug/kg body weight or 1 ug/day for average adult.[3] The U.S. Agency for Toxic Substances and Disease Registry (ASTDR) standard Minimum Risk Level (MRL) for chronic- duration inhalation exposure (365 days or more) to mercury vapour is 0.2 ug Hg/m^3, which translates to approximately 4 ug/day for the average adult.[4] The range of mercury exposure levels found in people with amalgam fillings by the World Health Organization Scientific Panel on Mercury was 3 to 70 ug per day[3], with other medical studies finding up to 200 ug/day in gum chewers or people who grind their teeth.[6,11,16,17,18] The average exposure was above 10 ug/day[3-.18] The average mercury exposure for a Canadian adult with amalgam fillings was found in the Health Canada study to be 9 ug/day.[2] In a large German University

study with 20,000 tested subjects, the average exposure from fillings was over 10 ug/day and over 50% of all those with six or more amalgam fillings had daily exposure exceeding the EPA health guideline.[17]

Studies have consistently found modern high copper non gamma-two amalgams have greater release of mercury vapour than conventional silver amalgams.[21-23] Recent studies have concluded that because of the high mercury release levels of modern amalgams, mercury poisoning from amalgam fillings is widespread throughout the population.[17,18,22]

Levels found in persons with amalgam fillings can be over 10 times the Health Canada TDI, and more than the EPA health standard for mercury vapour. Thus persons with amalgam fillings have levels of intraoral mercury vapour and body exposure levels higher than the level considered to have significant health risk and proportional to the number and extent of amalgam surfaces, but other factors such as chewing gum and drinking hot liquids influence the intake. Swedish research by Skare et al concluded that the average citizen was conservatively estimated to have 32 micrograms of mercury in faeces daily with an uptake of 12 micrograms of mercury into their bloodstream and tissues every 24 hours.[6] The worst case individual in the study was measured at 190 micrograms per day of mercury in faeces, with an estimated bloodstream and tissue uptake of 70 micrograms per day. Thus the average citizen could have absorbed up to or over 100mg of mercury after 25 years. Skare's findings need to be compared with the EPA limit of 2 parts per billion for safe drinking water. Notably, the WHO Scientific Panel concluded that a safe level of mercury exposure below which no adverse effects occur has never been established.[3]

The majority of New Zealand children born in the 50-60s have lived with dental amalgam fillings since primary school when the school dental nurses in the "murder houses" drilled and filled any tooth that had the slightest surface defect or even a deep natural groove. This went by the euphemistic name of "filling the valleys" or essentially prophylactic odontotomy, i.e. making a hole and filling it to prevent a hole. Regarding this, a Health Department paper revealed that in 1968 21-year-old adults had an average of 16 amalgam fillings with 15-year-old teenagers averaging 13 fillings.[24]

In 1976, the New Zealand Health Department sent out an unusual notice to all dental personnel that essentially requested that they only drill into teeth showing decay. The underlying reason for this has remained mysterious but there are some interesting and plausible reasons. The first fluoride experiment was carried out in Hastings in the preceding years and had been a complete failure with the control town of Napier actually showing the same or even less dental "decay". Estimation of decay was based on the numbers of decayed, missing or filled teeth (the DMF score). However, most of the results related to the F or filled teeth. The inspectors thus counted the number of fillings but it was then realised that the dental nurses had been instructed to treat all teeth that had even minor surface defects as being decayed but were essentially non-carious. The research protocol was therefore covertly altered and concealed in the published document.

Some 20 years later a revealing paper appeared in the New Zealand Dental journal where the author euphemistically stated that "changes in dental practice" resulted in a decrease in dental fillings. That decrease was identified as 30% in 1977 and an astounding 64% by 1981.[25] This would have dropped the average from 16 to between 7 and 8 or what is generally accepted as the norm by the WHO. Obviously that decrease in "decay" had far less to do with fluoride than the fact that dental nurses and dentists were no longer making the holes or "filling the

valleys". However, the 1996 Health Canada report on dental amalgam revealed that the maximum tolerable daily intake (TDI) of mercury vapour would be reached from 4 average sized amalgam fillings (or 8 tooth surfaces) for a 70kg adult when based on industrial safety levels[3] and this needs to be additionally considered in view of the current amalgam loading.

The symptoms and signs of mercury toxicity listed by amalgam manufacturers Dentsply-Caulk and Ivoclar-Vivadent in their 1997 Manufacturer's Safety Data Sheets (MSDS) included the following adverse health effects from chronic inhalation and/or ingestion: tremor, fatigue, headaches, irritability, excitability, depression, insomnia, loss of memory, hallucinations, psychiatric disorders, mental deterioration and resentment of criticism.[26] However, the covert underlying common denominator is usually missed as all of these symptoms and signs can occur with other more readily diagnosable conditions, and as medical students let alone doctors have not been instructed to look at teeth. Mercury release from amalgam has to occur when dissimilar metals are placed in the hot, salty and frequently acidic saliva due to oral galvanism and electrolysis. This is fundamental school chemistry.

Alzheimer's Senile Dementia (AD)

The genetic factor in AD is well known. There is a blood test for AD called Apo-lipoprotein E genotyping or apo-E for short. Two papers on this appeared between '96 and '98 when Dr. Alan Roses from Duke University revealed that Apo-E genotyping was related to the risk of early onset AD.[27] There are 3 genotypes E2, E3 and E4 with 6 possible combinations as we inherit from both parents, i.e. E2/2, E2/3, E2/4, E3/3, E3/4, E4/4. The last of these has the greatest risk at 70% chance of early onset AD before age 70. The onset of AD in those with E3/4 comes about 10 years later and the E3/3 again about 10 years later. Those with the E2 have to live to a very old age before any signs develop. Research subsequently revealed that about 1-2% of the population has the E4/4, 15-20% the E3/4 and 50-60% E3/3. However, the underlying reason remained a mystery and further research only complicated matters. For instance, one paper revealed that Africans in Africa with E4/4 did not get AD but Africans in the USA did and the authors wondered about different diets.

In 1999, Professor Boyd Haley, Chair of Chemistry, at Kentucky University, Lexington, and a leading researcher into mercury, revealed to a group of doctors that there was no mystery about the underlying reason for apo-E genotyping. It lay in the biochemistry. Apo-E has 299 amino acids with different ratios of cysteine and arginine at position 112 and 158. Apo-E2 has 2 cysteines, apo-E3 one cysteine and one arginine, and apo-E4 two arginines. As arginine, unlike cysteine, lacks the sulphydryl (SH) groups to potentially bind bivalent metals such as mercury, lead, copper or zinc, it would be logical to suspect the possibility of increased metal accumulation in those chronically exposed individuals who had not inherited apo-E2. Notably, mercury has been proven to cause all the unique microscopic brain lesions that are found in the AD brain at autopsy. Rats exposed to mercury vapour at levels found in people's mouths with dental amalgam for a few hours a day, developed AD lesions within 2 weeks[28] and a remarkable research paper came out of Calgary University in 2001 showing how in a nerve cell culture the nerve sheath "melted away" when minute amounts of mercury were placed in the culture solution.[29]

Apo-E genotyping was then performed on hundreds of New Zealand patients considered to be suffering from adverse health effects from their dental amalgam to see if there was a statistically valid association and in 2003, the journal of Alzheimer's Disease published the first paper showing the association between amalgam and the risk of developing AD.[30] This

was followed 3 years later with a second paper showing that chronic fatigue, depression and memory loss were also markedly increased in those with the apo-E4 and amalgam fillings, together with evidence that removal of amalgam combined with proper protection and detoxification resulted in a significant reduction of the symptoms.[31] This research has been independently confirmed by the findings of another research group at Uppsala University, Finland, who also investigated the effects of protected amalgam removal.[32]

It would seem appropriate that all patients with memory loss and the other symptoms listed above need to be evaluated for mercury toxicity from dental amalgam. AD could be either preventable or at least prevented from deteriorating in those with dental amalgam. Proper advice and where to get the best treatment in the USA is available[33] and it is of paramount importance that the process is done correctly.

Good science is now therefore proving that mercury is related to the onset of these common problems and, as the WHO had confirmed, that amalgam is the biggest source of mercury vapour in the non-industrially exposed populations[1], we need to seriously look at this dental material.

Mercury and the blood

It is an established fact that approximately 80% of inhaled mercury is retained to then travel to the main target organs, namely, brain, heart and kidneys. However, although levels in the blood may not be significantly elevated, mercury covertly does two highly relevant things. Firstly, it binds to haemoglobin and reduces that red cell's oxygen binding capacity and secondly, it kills lymphocytes. Unfortunately, standard laboratory tests will detect neither. The laboratory checks total haemoglobin and an assumption is made that the oxyhaemoglobin (OxHb) proportion was normal. However, research at Colorado University with a Co-oximeter differentiating total Hb, OxHb and CarboxyHb has confirmed in some hundreds of symptomatic patients with amalgam that their OxHb levels were at least 20 per cent lower confirming what other research has shown.[34] This would equate to having a litre of blood missing as far as oxygen carrying capacity and would quite likely result in being "tired all the time" or chronic fatigue syndrome, a diagnosis that does nothing to help the patient.

The lymphocyte effect was discovered when fresh blood was centrifuged and the white cell layer removed. A propidium iodide viability stain then identified the viable vs. the non-viable proportion with a significant incidence of the latter being found. Subsequently, a culture of lymphocytes was exposed to a level of mercury usually found in the blood of people with amalgam fillings and considered safe.[35] After 4 days incubation over 80 per cent had become non-viable compared to only 3 per cent in the control culture. This could be a reason why the white blood cell reference range has progressively widened over the past century as the body's internal monitoring systems may be detecting a lymphocyte "deficiency" as the lymphocyte "policemen" are being eliminated as soon as they leave the academy and go on the beat.

Two papers on cancer with one on breast[36] and another on colon[37] have studied natural Killer (NK) lymphocyte activity and outcome. Both found that those patients with "active" lymphocytes had a much better outcome than those with "inactive" NK cells. There was a 47% 5-year mortality in the breast cancer patients with non-reactive NK cells vs. 4% in those with active NK cells. Neither of the papers' authors discussed what, if anything, could influence activity but a definitive cause is there if this description is changed from active to

viable and inactive to dead as non-viable mercury contaminated NK cells would certainly be inactive.

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