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The dental amalgam controversy: a review

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In spite of the long history of mercury amalgam as a dental restorative material, its use continues to be controversial.

Mercury vapour is continuously released from dental amalgam and is ultimately absorbed into a variety of tissues. Experimental data have demonstrated that the uptake, tissue retention and excretion of mercury from dental amalgam is significant. Evidence has accumulated indicating a relationship between tissue mercury levels and a multitude of clinical manifestations. However, the clinical significance of mercury toxicity from dental amalgams is a matter for debate. The literature is devoid of randomized clinical trials that are rigorously designed to address this issue. Thus, although research data renders the notion of amalgam safety questionable, the dental community appears determined to continue its use as long as unequivocal evidence correlating amalgam mercury toxicity to specific clinical conditions is lacking.
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En dépit de l'historique étendu de l'amalgame au mercure à titre de matériel de restauration dentaire, son utilisation demeure controversée. L'amalgame dentaire rejette continuellement des vapeurs de mercure qui, en bout de ligne, sont absorbées par une variété de tissus. Les données expérimentales ont démontré que l'absorption, la rétention par les tissus et l'excrétion du mercure à partir des amalgames dentaires étaient significatives. Des preuves ont été accumulées indiquant une relation entre le niveau de mercure des tissus et une multitude de manifestations cliniques. Cependant, la signification clinique de la toxicité du mercure à partir des amalgames dentaires reste un sujet de débat. La documentation à ce sujet est dépourvue d'études cliniques réparties au hasard qui sont rigoureusement conçues pour évaluer ce problème. Donc, bien que les données des chercheurs remettent en question la sécurité des amalgames, la communauté dentaire semble déterminée à continuer d'utiliser ces derniers tant qu'aucune preuve catégorique mettant en corrélation la toxicité de l'amalgame au mercure et certaines conditions cliniques ne sera fournie.
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MOTS CLÉS : amalgame dentaire, toxicité du mercure.

Introduction

The safety of dental amalgam as the primary material applied in dental restoration treatments has been debated since its introduction in the early 1800's in France.[1] Many side effects have been associated with the uses of mercury amalgam in dentistry and the controversy was sparked again in the 1990's.[2–8] The renewal of the century old debate was mainly due to three factors: (a) A great number of studies have been published describing the adverse effects of dental amalgam on physiological processes, (b) the rise of public awareness to any type of environmental contaminants representing health hazards was expanded to mercury poisoning, and (c) with the great advances of analytical chemistry extremely low amounts of mercury could be detected in blood, urine or other tissue extracts.

As primary health care practitioners, chiropractors are

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often asked for advice on health issues which may not be directly within their scope of practice. However, with the patient's overall well-being in mind, it is important that their knowledge is well-rounded and, in particular, they are up to date with issues that are of general public health interest. One such issue is the question of dental amalgams and whether or not they are contributing factors to the development of disease.

In order to review this subject comprehensively, a search was conducted utilizing the Medline and Chirolars databases for the period 1985–1995. Articles were selected for review if they contained information relevant to the topic of dental amalgam composition, mercury toxicity and clinical consequences of dental amalgam use. Review articles and textbooks were also used as a source to retrieve much valuable information.

In this review we provide an overall picture of the documented associations between mercury applied in dental amalgams and various possible neurological, immunological and other disorders. In addition, in order to facilitate a better understanding of the effects of mercury, we give a brief account of the physical properties of dental amalgams and a synopsis of other sources of mercury and its half-life in humans. The literature is very wide[2,9,10] and it is not possible to discuss every detail of this issue which concerns dentists, toxicologists, neurologists, immunologists and in a broad sense all health professionals. A recent article by Lorschider and coauthors[11] provides a review of the evidence for mercury toxicity and questions the traditional dental paradigm that continues to embrace the use of mercury amalgam. The present article assesses the status of dental amalgam effects and provides the reader with current educational information in the hopes of enhancing the clinician's appreciation of the issues surrounding the dental amalgam controversy. (See addendum)

1. Physical-chemical properties of dental amalgams

Amalgams are alloys of mercury with other metals and they have many advantages for use in dentistry.[9,10,12,13] The literature provides wide ranging data on the composition of the amalgam. Accordingly, the conventional silver amalgam contains in approximate percentages by weight, 48–60 parts/g of Hg, 15–37 Ag, 12–13 Sn, 0–3 Cu and 0–1 Zn.[10,12] Newer brands used almost exclusively in North America and Europe utilize higher concentrations of Cu (up to 26 parts/g).[9] The higher copper composition results in a stronger, less corrosive amalgam with improved clinical performance.[14] The amalgamation reaction does not proceed stochiometrically and unreacted silver-alloy particles and free liquid mercury are present after the settling reactions.[13]

Various types of amalgams can deteriorate by electrochemical corrosion.[13] Chewing and abrasion, brushing and increased temperature strongly stimulate amalgam corrosion and evaporation of mercury. Cracking due to stress and mechanical wear can also contribute to deterioration. Every amalgam restoration is attacked by corrosion to some extent resulting in a surface layer of corrosion products and slow and continuous release of mercury.[15]

It has been demonstrated that the released mercury vapour from the amalgam filling is present in the air, and absorbed by the lungs.[2,16–18] It has been estimated that the daily average intake of mercury from dental amalgam ranges between 2–20 μg.[2,19–21] Furthermore, due to corrosion, mercuric ions and microscopic amalgam particles are also released into the saliva.[22]

2. Mercury Toxicity

a) Environmental occurrence and dietary intake

On the basis of toxicological properties, there are three forms of mercury: metallic (elemental), inorganic and organic compounds.[9] Metallic mercury volatilizes at room temperature, and exposure can occur by inhalation. It is lipid soluble and readily diffuses across alveolar membranes. The gastrointestinal tract does not effectively absorb elemental mercury. Metallic mercury has an affinity for the central nervous system and erythrocytes.

Inorganic mercury usually occurs as mercurous or mercuric salts. Gastrointestinal absorption of these compounds show large variations.[23] Following exposure to inorganic mercury compounds, the kidney contains the greatest concentration. Unlike elemental mercury, inorganic mercury compounds do not have a special affinity for red blood cells or the central nervous system.

Organic mercury, which occurs mainly in the alkylated form (e.g. methyl mercury), is primarily produced by microorganisms including plankton and the gastrointestinal flora. Thus, humans acquire alkyl mercury primarily from the consumption of fish and to some extent from inorganic mercury compounds converted by the bacterial flora of the mouth and intestine.[2] Alkyl mercury is lipid

soluble, can be easily absorbed and partially transformed to inorganic mercuric salts or oxides.

Excretion of mercury from the body occurs primarily by urine and feces. Unabsorbed elemental mercury is eliminated in the feces. The fecal route is also predominant in the excretion of inorganic mercury compounds.[2,21] In time, however, urinary excretion of inorganic mercury is increased. Almost 90% of alkyl mercury is excreted in the feces.

Excluding occupational exposure, dietary intake is the most important source of mercury next to dental amalgam restorations.[6,9,24] Thus, when the contribution of amalgam fillings to body mercury is discussed it is essential to consider the absorption of mercury salts contained in food. Methyl mercury found in fish is the most important dietary form of mercury. Other food sources of mercury contamination appear to be scarce. In addition, mercury may be present in drugs, contact lens solutions, cosmetics and vaccines.[9] According to a Swedish study, the average daily mercury intake was 5.5μg, and 12.4μg with diets containing more than 75g fish per day. Considering 5g mercury in the teeth (10g amalgam) the daily release is about 10μg mercury.[9] However, this value will probably vary not only with the weight of amalgam, but also with the number and size of restoration surfaces. It is probably more meaningful to express the amount of mercury released in terms of μg Hg/restoration/day. Nevertheless, according to WHO Environmental Health Criteria,[25] the estimated average release of elemental mercury vapour from dental amalgams in the general population is between 3.8–21 μg/day, and its retention in the body is 3–17 μg/day. The release and retention of inorganic mercury is 4.3 and 0.3 μg/day respectively, and that of methyl mercury from food 2–4 μg/day of which 2–3 μg/day is retained.[25]

In considering mercury toxicity, the contribution of contamination through food consumption cannot be ignored. In a publication from an Austrian investigation, based on the mercury concentration of the average raw food product, the estimated weekly intake of mercury compounds is about 42.1 μg, excluding drinks and fish.[6] When fish is included with the food, this rises to about 53 μg per week.[24] According to a FAO/WHO report, the maximum weekly mercury intake with food regarded as having no adverse effect on health, is 300 μg in the form of inorganic mercury compounds and 200 μg per week in the form of methyl mercury.[6]

b) Biological half-life

The biological half-life of mercury varies depending on its chemical status and the tissue(s) in which it is stored. It is generally accepted that the average half-life of methyl mercury in human adults is 70 days.[26] In oxidized form the biological half life of mercury in human volunteers was about 58 days in the whole body, 64 days in kidneys, 21 days in the head region[27] and 3.3 days in the blood.[19,28] The biological half-life of ingested mercuric salts was 29–41 days in women and 32–60 days in men suggesting a sex related difference. It is likely that the half life of mercury compounds is longer in the kidney than in the whole body due to the presence of mercury binding proteins.[23]

The biological half-life of methyl mercury was 71 days in the whole body of volunteers,[23] and 50 days in erythrocytes.[23,29] Segmental analysis of hair samples from methyl mercury intoxicated patients also indicated an average half-life of 70 days.[30]

Mercury converted to mercury sulfide or mercury selenide *in vivo* persists significantly longer in the body than other mercuric salts.[20]

Thus, in view of the relatively long half-life of mercury in human tissues, and the potentially steady release of mercury from dental amalgam over prolonged periods (often lifetime), it is theoretically possible that tissue mercury may increase to clinically significant levels.

c) Tissue mercury levels

Autopsy investigations have demonstrated mercury in a variety of tissues including brain, kidney, thyroid, pituitary, skin and blood.[31–34] Tissue specificity appears to be related to the specific chemical form of mercury. Thus, the central nervous system appears to be the primary target of the lipid-soluble forms namely, elemental mercury and short-chain alkylmercurials,[35] while the renal proximal tubular cells are the primary target of the water-soluble forms such as mercuric mercury.[36]

Tissue concentrations of mercury appear to depend on a number of variables not the least of which are duration of exposure and the dose of mercury involved. Thus, higher tissue levels of mercury have been demonstrated four weeks and one year after amalgam placement.[37] As well, tissue concentrations of mercury have been shown to correlate quantitatively with the number of amalgam fillings in human autopsy studies.[29,32] Nylander[34] demon-

strated significantly higher concentrations of mercury in necropsy preparations of pituitary glands of dentists. He explained the increase by the proximity of the pituitary to the nasal mucosa through which mercury could be directly transported. It is equally possible, however, that glandular tissues such as the pituitary may accumulate mercury at a higher rate due to their highly vascularized nature and cellular activity.

Increased tissue mercury levels in amalgam-bearing subjects has been also suggested from urinary mercury measurements. In amalgam-free subjects, the average urinary mercury excretion varies between 0–25 μg/l/24h. This is more than doubled in amalgam-bearing subjects, being as high as 150–180 μg/l/24h in subjects with several amalgam fillings.[38] This is also borne out by demonstration of a correlation of elevated mercury levels in the blood, urine and hair samples of patients suffering from environmental mercury intoxication.[39]

d) Mechanism of mercury toxicity

Mercury and mercury salts are cell and protoplasm toxicants. They appear to exert their effect by binding to sulfhydryl groups leading to protein precipitation and diminished enzyme activity.[40] This in turn can lead to nonspecific cellular damage due to impaired metabolism including cell membrane alterations and changes to nucleic acid synthesis and structure. A second mechanism of subcellular damage by mercury is via the formation of free-radicals. A recent investigation on human granulocytes has shown that mercury, in 10^{-17} to 10^{-13}M concentrations, increased oxygen-mediated free radical production[41] and DNA breakage in hamster ovary cells.[42] The release of free radicals has been suggested to be the mediator in the development of adverse hypersensitivity reactions.[43]

e) General toxic effects of mercury

The primary target organ for elemental mercury and methyl mercury or short-chain alkylmercurials is the central nervous system which is more accessible to these lipid soluble compounds than to other forms of mercury compounds such as salts and oxides.[2,44,45] Inhalation leads to a slowly developing and insidious poisoning which produces psychic effects that are difficult to recognize until more objective symptoms become apparent.[46] Neuropsychological tests indicate cortical dysfunction.[47–49] The primary effect of highly neurotoxic methyl mercury mainly manifests in the sensory part of the nervous system.[49] Ethyl mercury also has neurotoxic effects but in addition causes some renal damage.[2]

Morphological lesions have been shown to be present in the precentral regions and in calcarine tissues of the brain and in the granular layer of the cerebellum.[50] Swedish register studies have reported a significantly high risk of intracranial gliomas in dentists and dental assistants.[46,49] Mercury crosses the placenta and may be found in fetal blood at higher concentrations than the mother's. The fetal brain is more sensitive to mercury exposure than the maternal brain and this may account for the possibility of neurological and psychological anomalies developed later in life.[51] A recent study has demonstrated a direct correlation between the number of maternal amalgam fillings and the level of mercury found in newborns and between mothers and children aged from one to five years.[52] This brings into question the whole idea of using amalgam restorations in women of child-bearing age. However, the results should be interpreted carefully as the paper[52] provides no information about the contribution of dietary consumption of mercury by either the mother or child, and also, possible occupational exposure to mercury was not reported.

The alimentary tract and the proximal tubular cells in the kidney are also primary targets for mercury compounds. Inorganic mercury salts cause a corrosive action on the alimentary tract and through cardiovascular shock they can also indirectly potentiate kidney effects. Inorganic mercurous salts are less nephrotoxic than mercuric derivatives. Organomercurials show less nephrotoxic effects than inorganic mercuric compounds. Direct effects of inorganic mercury compounds on other organs have also been documented.[36]

Mercury affects the immune system. Exposure to mercuric chloride or methyl mercury results in systemic autoimmune reactions. This reaction is characterized by antibodies to a variety of proteins, mainly of endothelial origin.[53] Skin is affected by all forms of mercury; repeated exposure is followed by increased dermal reactions indicating a connection with the immune system.[54] Exposure to elemental mercury, mercuric and mercurous salts can elicit hypersensitivity reactions in the form of nephrotic syndrome.[51,53]

3. Dental amalgam toxicity

The adverse health effects of amalgam is dependent on the number and size of the fillings, the occlusal geometrical surface area, corrosion factors and the sensitivity of the mercury-exposed individual.[2] The published literature suggests a relationship between dental amalgam and a variety of clinical symptoms. However, an unequivocal correlation has not been established. Several case studies have attempted to look at the reversal of symptoms following removal of amalgam fillings. In one case, a patient with 11 fillings showed various clinical symptoms which were improved after the removal of the fillings.[55] In another case, having ten amalgam fillings, anorexia hydrargyria developed.[56] Removal of the fillings and treatment with dimercaptopropane sulfonate brought about complete recovery. On the other hand, a study on 1,024 women aged 38–72 years with 1–4 amalgam fillings, did not show any relationship between the number of fillings and impairment of health.[57] No correlation between the occurrence of mercury-related symptoms and the number of amalgam surfaces was found in another study.[58]

Thus, the current position on amalgam toxicity is controversial. Several studies have shown that subjects with amalgams have significantly more problems of physical health than subjects without amalgams.[2,9,59] On the other hand, several studies did not find any correlation between the amalgam restorations and the apparent health of the selected subjects.[4,5] The general symptoms associated with amalgam fillings include headaches, fatigue, apathy, abdominal pain, muscle, back and joint complaints, disturbed sleep, insomnia, impaired memory, stress, depression and other psychic effects, diarrhea with strong bleeding, irregular heart beat, heart and chest pains, proneness to infections as evidenced by frequent inflammations in the eyes and upper respiratory passages, sinusitis and asthma.[2,9,59]

The maximum acceptable limits for elemental mercury has been established.[3,10,11] However, critical thresholds of appropriate toxicological responses need sensitive and accurate biological or biochemical parameters that are clearly associated with the clinical symptoms. The major problems of the amalgam controversy are the great variations in individual sensitivity and the lack of biological and biochemical indicators and accurate quantitative diagnostic tests.[60] In some studies, the correlation between blood and urine mercury levels, or concentrations in brain and other tissues and the clinical symptoms, is mainly qualitative.[19,34,61,62] However, other investigations have established a quantitative dose/response relationship between the toxicity symptoms and mercury level in the blood, brain and other tissues.[10,28,44,63–65]

The following is a systematic documentation of clinical manifestations that are thought to be associated with dental amalgam toxicity.

a) Muscle and joint complaints

Chronic mercury poisoning caused by amalgam fillings is often associated with muscle and joint complaints.[9] Pain usually affects the extremities (acrodynia)[36] and this condition appears to be due to stimulation of the sympathetic nervous system by mercury.[66] However, motor signs also occur at the early stages including fine muscle tremors and impaired coordination of finger movement. More severe toxicity may affect larger segments of the motor system resulting in muscular dysfunction and impairment of gait.[5] In most cases the joint pains are just one of several symptoms of micromercurialism brought about by dental amalgam.[66,67]

b) Fatigue

One of the characteristic symptoms of dental amalgam poisoning is fatigue.[68] A study has shown that women with amalgams have significantly lower average red blood cell count and lower hemoglobin content than women without dental fillings. A direct correlation was found between lower values of erythrocytes, hemoglobin and urinary mercury content.[2,29,69] It has been suggested that mercury toxicity probably compromises the hemoglobin molecule in carrying oxygen to the tissue, and the relative lack of oxygen may be one of the mechanisms related to fatigue.[68,70]

c) Anxiety and anger

Psychometric examination has provided evidence that mercury from dental fillings may be an etiological factor in anxiety and excessive anger.[71] Mercury toxicity may have been the causative factor in sudden anger reactions.[72] In a study in which 47 subjects with an average of 13 amalgam fillings were compared with 48 age and sex-matched subjects without amalgam, it was found that the amalgam-bearing subjects had a significantly higher incidence of sudden anger and depression than the controls.[73]

The scores on anger and depression were decreased in subjects whose fillings had been removed.[74]

Experimental data suggests that psychological conditions may be associated with transmitter abnormalities. Changes in serotonin levels or metabolism can be the causative factors in aggressive behaviour.[75] It has also been shown that the magnitude of aggression may be directly related to the decrease of norepinephrine.[76] Lower levels of dopamine can evoke irritative aggression in laboratory animals.[77] This is related to mercury salts or methylmercury in the brain of experimental animals suggesting that irritability and anger might occur in people in association with mercury toxicity.[78]

Amalgam-bearing individuals have been shown to smoke more cigarettes than their non-amalgam bearing counterparts. It has been suggested that the biochemical basis for this appears to be related to a decrease in neurotransmitter uptake due to amalgam which can be reversed by nicotine.[79,80] It has also been reported that people with dental amalgam smoke more to relieve their anxiety.[81]

e) Depression

The presence of amalgam fillings is also connected with a significantly higher incidence of depression, mercury being suggested as an etiological factor of this condition.[72] Depressed subjects with amalgam have significantly higher levels of mercury vapour in their mouth than their counterparts without amalgam. It was postulated, that with more mercury in the oral cavity, these patients probably had higher levels of mercury in the brain. Post mortem analysis confirmed a direct correlation between brain mercury content and occlusal surfaces of dental amalgam.[71]

There is some evidence indicating that one of the main etiological factors in depression is an imbalance of neurotransmitters.[77,78] Animal experiments have shown that mercury affects some neurotransmitters including the uptake of dopamine and norepinephrine by brain synaptosomes, and inhibits the binding of serotonin to high affinity receptors in the brain.

f) Cardiovascular effects

Mercury poisoning from dental amalgam may play a role in the development of diseases of the cardiovascular system. A comparative study between subjects with and without amalgam fillings has shown that cases with amalgam have significantly lower heart rate, lower hemoglobin, lower red blood cell count, and higher blood pressure.[20,70] These changes were correlated with increased levels of mercury in the urine. The amalgam bearing cases exhibited tachycardia, anemia, a greater incidence of chest pain, fatigue, and easy tiredness especially in the morning.

g) Skin, acrodynia and the immune system

Dental amalgam has been shown to cause skin sensitivity both in dentists and patients.[74] Skin is the target for all forms of mercury causing erythema, burning sensation, blisters and hypersensitivity reactions.[41] Skin hypersensitivity to mercury has been reported to occur up to 26.6% of the population.[82,83] The increase of dermal reactions to repeated exposure might indicate that probably the immune system is the primary target.[9,54,82]

In children below the age of 5 years prolonged exposure to mercury can cause an idiosyncratic disease, characterized by irritability, rashes, photophobia, loss of body weight and pain in legs and arms. The development of this condition called acrodynia is probably a combination of neurotoxic and immune system related actions of mercury.[84] The sequence of events in acrodynia was suggested to be an initial attack by mercury on the endothelial cells in the brain, followed by a secondary immune reaction to components of the brain.[20]

h) Miscellaneous conditions

There are many other cases suggesting mercury poisoning. The types of symptoms largely depend on the structure of the mercury compound and on the mode of entry.

In one case anorexia hydrargyria was described in a 15-year-old girl with multiple amalgam fillings who developed headaches, fatigue, joint pains, vertigo, loss of memory, sleep disturbances and hair loss. Lack of appetite led to weight loss and symptoms of anorexia nervosa.[56] Several cases of an amyotrophic lateral sclerosis type syndrome have been reported after exposure to mercury vapour[85] inhalation of mercuric oxide containing dust[86] and ethyl mercury[87] or due to amalgam fillings.[9] In these cases removal of the amalgam has resulted in complete recovery. Mercury neurasthenia, various forms of paralysis affecting different parts of the nervous system have been described including Guillian-Barré syndrome and polyradiculoneuritis.[9] Micromercuralism is also

postulated as a causative factor in the development of Parkinsonism.[88]

8. Views against amalgam toxicity

The prevalent view of the dental community has been that the evidence presented against the use of mercury amalgam as a dental restorative material is insufficient to warrant its discontinuation.[3]

One of the major arguments against amalgam toxicity is the uncertainty whether mercury is released from dental fillings in sufficient quantities to pose a health hazard. Reported values of mercury vapour entering the body are widely variable.[4,89] Although it is acknowledged that people with dental amalgam restorations have generally higher mercury levels in their blood, tissues, and urine, it is argued that the health consequences of these findings are not well understood.[63,64,90–92] Moreover, the contribution of dental amalgam to the total body mercury burden, including mercury from various food products has not been well established.[7,4] According to Henderson,[93] an attempt to define this relationship has been undertaken by the federal government of Canada and a more in depth research effort is anticipated by the Canadian Dental Association, pending funding.

It has been suggested that pathological changes resulting from low levels of mercury toxicity are often absent or difficult to demonstrate at the tissue/cellular level.[61] Clinical data are often used in order to make correlations between mercury amalgam toxicity and neurological and behavioural changes in patients.[62,94,95] However, these changes are typically subtle and non-specific, rendering the interpretation of results difficult.

It is also acknowledged that hypersensitivity reactions to mercury amalgam do exist. However, it has been argued that this should not discourage the use of mercury amalgam in the general population as the rate of allergy occurrence is about 3% and that symptoms actually disappear within a day or two after amalgam placement.[7]

According to Brown[5] attempts to correlate clinical symptoms to mercury amalgam toxicity should take into account the critical matter of the latency period. This may be particularly difficult to establish in the case of chronic low grade toxicity in which symptoms develop over a long time, making the establishment of a cause and effect relationship difficult.

Finally, reviews of the literature have pointed out that the evidence cited by anti-amalgam forces are drawn from studies that are weak in research design[5,7,60,96,97] making their interpretation difficult. Brown[5] has discussed the issue of research design for studying dental amalgam toxicity in human populations in some detail, pointing out that controlled randomized intervention trials are required in order to establish correlations between dental amalgam and adverse clinical effects.

Conclusion

Surveying the literature we can state that several independent reports have established that amalgam restorations may represent certain adverse health effects through mercury toxicity. However, to date, threshold mercury levels responsible for the clinical signs of toxicity have not yet been clearly established. Furthermore, studies investigating dental amalgam toxicity have failed to consider the contribution of dietary mercury contamination. These weaknesses not withstanding, the occurrence of toxicity signs and clinical manifestations in some amalgam bearing patients, should make the clinician question the safety of dental amalgam.

The dental community has traditionally considered the use of dental amalgam safe, citing its advantages of ease of use and inexpensiveness and arguing that the evidence against its use is insufficient and anectodal in nature. However, until the definitive study is done to show unequivocal evidence that amalgam fillings have no long-term side effects, the issue of potential amalgam toxicity will remain unresolved.

Alternative dental filling materials are available.[98] However, these have their shortcomings. It is hoped that these materials will be subjected to rigorous scientific scrutiny before their use becomes widely popular, so as controversies similar to those surrounding the use of mercury amalgam might be avoided.

In our view, it is incumbent on all health care providers to be cognizant of all the issues surrounding the use of dental amalgams. The Health Canada study released recently, *(see Addendum)* will serve as an excellent source of information for the clinician, research scientist, as well as the interested public. The recommendations contained in the Health Canada Position Statement reflect an awareness of the side-effects of dental amalgam and provide a measure of increased protection for the public.

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