

## The amalgam controversy: An evidence-based analysis

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*J Am Dent Assoc* 2001;132;348-356

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# The amalgam controversy

## An evidence-based analysis

JOHN E. DODES, D.D.S.

**E**vidence-based care, or EBC, is the name of a clinical decision-making paradigm first described in 1993.<sup>1</sup> To provide EBC, clinicians must develop appropriate skills to evaluate research literature and clinical data. These skills require an understanding of the rules of evidence (box, "Evidence-Based Approach Rules of Evidence for Evaluating Reports of Treatment Efficacy") and the realization of the limitations of clinical experience. The skill of critically evaluating research literature and clinical data is barely touched on in dental school. Enid Neidle, former director of the ADA Council on Scientific Affairs, commented that dental education is too authoritarian, leaving many students "susceptible to the experiences of others" and willing to accept the views of an authority figure without demanding to know the science supporting those views.<sup>2</sup>

**I attempt to follow the evidence-based approach rules in evaluating data on the possible dangers of amalgam restorations.**

To apply an evidence-based approach to the dental amalgam controversy requires studying articles on the subject that have been published in peer-reviewed and non-peer-reviewed publications and evaluating them as to their relevance, research design and statistical analysis, as well as to whether the conclusions follow from the data.<sup>1,3,4</sup>

In this article, I attempt to follow the evidence-based approach rules in the evaluating data on the possible dangers of amalgam restorations.

### HISTORY OF DENTAL AMALGAM

The history of dental amalgam restorations containing mercury is a long one. Tin-mercury dental restorations are reported to have been used in China in A.D. 600.<sup>5</sup> Silver-mercury restorations were introduced to the

**Background.** There are a number of patients and health care professionals who believe dental amalgam restorations are a factor in a host of diseases and conditions. They have been influenced by anecdotal case reports in the medical and dental literature, research published in the refereed literature and media stories concerning the alleged dangers of amalgam restorations.

**Methods.** The author uses an evidence-based approach in analyzing the data both supporting and condemning the continued use of amalgam restorations. He reviewed the articles from both peer-reviewed and non-peer-reviewed sources and evaluated their relevance, research design and statistical analysis, as well as whether the conclusions follow from the data.

**Conclusions.** There are numerous logical and methodological errors in the anti-amalgam literature. The author concludes that the evidence supporting the safety of amalgam restorations is compelling.

**Clinical Implications.** Amalgam restorations remain safe and effective. Dentists should educate patients and other health care professionals who may be mistakenly concerned about amalgam safety.

Western world in France in the 1830s. In the 1850s, American dentists who used amalgam were threatened with malpractice actions by dentists who did not. This became known as the "amalgam wars." In 1896, Dr. G.V. Black published a detailed scientific report advocating the use of amalgam,<sup>6</sup> but it still took many years for Dr. Black's conclusions to be universally accepted by the dental profession.

In 1926, Alfred Stock, Ph.D., a German chemist, published an article condemning amalgam restorations.<sup>7</sup> Dr. Stock had been exposed to high mercury levels while working in his chemical laboratory. He recognized the danger posed by the type of amalgam that was in use at that time; a tablet had to be heated in a spoon until the beads of mercury appeared, and then it was transferred to a mortar and pestle for trituration.

## EVIDENCE-BASED APPROACH RULES OF EVIDENCE FOR EVALUATING REPORTS OF TREATMENT EFFICACY.\*

Questions to ask when using an evidence-based approach to evaluate research literature and clinical data:

- Are the results applicable to a particular patient?
- Were the study patients randomly and properly assigned?
- Were all of the patients in the study followed up completely or was there an excessive dropout rate?
- Were the study populations analyzed in their randomized groups?
- How blinded was the study?
- Except for the experimental intervention, were the groups treated equally?
- Was the statistical analysis done properly?
- Did the authors perform so many statistical tests that a mistaken “significant” finding was found?
- Did the article report on the participants’ compliance with the treatment?
- Were all the clinically significant outcomes discussed?
- Were the side effects and negative effects of the treatment reported and discussed?
- Do the benefits of the treatment outweigh any potential negative effects and costs?

\* Source: Guyatt and colleagues.<sup>1,2</sup>

This procedure produced a significant release of mercury vapor. Dr. Stock’s concerns led to a commission’s being established to investigate his allegations. In 1930, the commission issued a report that validated the safety of the newer dental amalgam formulation, which no longer required heating and rapidly was replacing the older formulations.<sup>8</sup>

In the 1970s, Dr. Hal Huggins began promoting the theory that amalgam restorations caused a wide variety of diseases. In 1985, he published a book that detailed his beliefs about mercury toxicity.<sup>9</sup> Dr. Huggins contends that amalgam restorations release enough mercury to cause neurological, cardiovascular, immunological, collagen, emotional and allergic diseases and disorders. The resulting conditions are said to include multiple sclerosis, depression, high or low blood pressure, tachycardia, arthritis, lupus, scleroderma, leukemia, Hodgkin’s disease, mononucleosis, fatigue, and Crohn’s disease, ulcers and other digestive problems. Dr. Huggins has

attracted many followers, and his writings and media appearances have led some dentists to question the safety of amalgam restorations.

A 1995 survey reported that 8.7 percent of dentists wanted to ban amalgam use and that 14.3 percent were undecided about its safety.<sup>10</sup> Much of the opposition to amalgam has been fueled by the media, particularly the “60 Minutes” segment that was broadcast in 1990.<sup>11</sup> Physicians with large public audiences, such as Robert Atkins, M.D., and Andrew Weil, M.D., also have warned the public about the potential danger of amalgam restorations. Both Drs. Atkins and Weil have written best-selling books on health. Dr. Atkins hosts a nationally syndicated radio program, and Dr. Weil has hosted various programs about holistic health on public television.

Most lay people and many dentists are unfamiliar with the peer-reviewed dental literature and, therefore, are more easily convinced by media stories that amalgam is dangerous. The problem is so serious that the American Council on Science and Health, a consumer education and advocacy group, has determined that the allegations against amalgam restorations constitute one of the “greatest unfounded health scares of recent times.”<sup>12</sup>

### MERCURY AND ITS COMPOUNDS

Mercury and its compounds are everywhere in our environment. Between 2,700 and 6,000 tons of mercury are released annually from the oceans and the Earth’s crust into the atmosphere.<sup>13</sup> Another 2,000 to 3,000 tons are released from human activities, primarily burning household and industrial waste and, especially, from burning fossil fuels such as coal.<sup>13</sup> Hippocrates was aware of mercury’s toxicity.<sup>14</sup> Yet mercury still has a long history of use in medicaments; for example, calomel (mercurous chloride) was used well into the 20th century for the treatment of syphilis. Inorganic mercury still is used widely in electrical applications, chlorine production and dental restorations.

In 1969, a report written by a committee of international toxicology experts classified mercury and its compounds according to their order of decreasing toxicity: methyl and ethyl mercury compounds (organomercury), mercury vapor (elemental mercury), inorganic salts and a number of additional organic forms such as phenyl mercury salts.<sup>15</sup>

**Methyl Mercury.** Certain bacteria present in seawater are capable of transforming elemental mercury into methyl mercury. It then concentrates in the tissues of fish and other sea creatures and moves up the food chain, which includes seafood-consuming humans. For example, industrial waste containing high concentrations of elemental mercury was released into the waters around Minamata, Japan, for many years. Fish from these waters were contaminated with methyl mercury and were responsible for both acute poisonings that resulted in death and chronic poisonings that resulted in central nervous system disturbances now known as Minamata disease. There also was a teratogenic effect called congenital Minamata disease.<sup>16</sup> It is estimated that the minimum dose needed to develop symptoms of Minamata disease was 5 milligrams per day of methyl mercury.<sup>17</sup>

The half-life of methyl mercury is about 70 days in adults and slightly longer in fetuses.<sup>18</sup> Approximately 15 percent of the body burden of methyl mercury is in the brain.<sup>18</sup> In 1983, Heintze and colleagues<sup>19</sup> reported the methylation of mercury in vitro by oral streptococci. Their technique, which has not been replicated, yielded 0.029 mg of methyl mercury per gram of powdered amalgam after 35 days of a complicated procedure. Although it does not appear possible to recreate this process in vivo,<sup>20</sup> their study often is cited as proof that mercury is converted to methyl mercury in the human gastrointestinal tract. A close look at their article, however, shows that the methyl mercury was intracellular and that the bacteria would have to be digested before the methyl mercury would be released. If this did occur, the amount of 0.029 mg/g is a fraction of the minimum safe level.<sup>21</sup> Birke and colleagues<sup>22</sup> reported no symptoms of poisoning with levels of 0.8 mg of methyl mercury per day for five years through the consumption of contaminated fish.

**Mercury vapor.** Mercury vapor (elemental mercury) is the major source of concern to dentists and patients. Mercury has a high vapor pressure (.005 mg of mercury at 37 C), and approximately 75 percent of inhaled inorganic mercury vapor will be absorbed through the lungs.<sup>23</sup> Gastrointestinal absorption is low, with estimates ranging from .01 to 10 percent.<sup>24</sup> Absorption also is minimal through the skin, although the precise level has not been determined.<sup>18</sup> Elemental mer-

cury accumulates in the kidneys and brain and is excreted in the urine, secreted in bile and exhaled from the lungs.<sup>18</sup> On an individual basis, there is little correlation between sampling of hair, blood and urine and toxic effects at target organs.<sup>18</sup> Elemental mercury's toxicity probably is a result of its affinity for sulfhydryl groups on proteins, but the results of studies in vitro do not relate well to conditions in vivo, in which distribution and accumulation of elemental mercury ions vary immensely from one type of tissue to another.<sup>25</sup> Acute toxic exposures are rare, and there have been cases of elemental mercury accidentally being released into the bloodstream, such as when a rectal thermometer breaks, or when several grams of mercury were swallowed intentionally,<sup>26</sup> without any reported adverse effects from the mercury. Chronic toxicity leads to a condition called erethism, characterized by insomnia, irritability, loss of memory, lack of self-control, timidity, drowsiness, depression and eventual tremors.<sup>18</sup> The renal effects lead to proteinuria, and a diagnostic discoloration of the lens of the eye also may develop.

Both the Occupational Safety and Health Administration<sup>27</sup> and the National Institute for Occupational Safety and Health<sup>28</sup> give a threshold limit value, or TLV, of 50 micrograms per cubic meter of mercury vapor as a time-weighted average based on constant exposure of 40 hours per week. The World Health Organization, or WHO, has adopted a recommended limit of 25  $\mu\text{g}/\text{m}^3$ .<sup>29</sup>

Clinically significant effects (erethism, intention tremor, gingivitis) have not been reported below air concentrations of 100  $\mu\text{g}$  mercury/ $\text{m}^3$ .<sup>30</sup> Slowed nerve conduction and short-term memory loss have been observed in and special instrumental tests for tremor (preclinical effects) have been conducted on people exposed to mercury levels below 100  $\mu\text{g}$  Hg/ $\text{m}^3$ .<sup>30</sup> But no clinical deficiency in kidney function has been discovered in this same population. The range of mercury in urine for populations with no identifiable source of mercury exposure is up to 20  $\mu\text{g}/\text{liter}$ .<sup>30</sup> Clarkson and colleagues<sup>31</sup> estimate the total daily absorption for all forms of mercury to be 2.3  $\mu\text{g}/\text{day}$ , compared with the 5.8  $\mu\text{g}/\text{day}$  estimated by the Environmental Protection Agency, or EPA.<sup>32</sup> Two-thirds of this difference in estimates stems from the EPA's higher allocation of ingesting inorganic mercury

Mercury and its compounds are everywhere in our environment.

from nonfish food, while the other one-third comes from the larger EPA estimate of methyl mercury from fish consumption.

### AMALGAM CORROSION

Amalgam corrosion is an oxidation-reduction reaction in which the metals in the amalgam react with nonmetallic elements in the environment to produce chemical compounds.<sup>33</sup> This is important because corrosion is a major factor in determining the amount of mercury that is released into the oral cavity. Amalgam corrosion is influenced by factors that disrupt the surface layer of the restoration such as toothbrushing and chewing, which can cause an increase in mercury release. The mercury released in this fashion can be in two forms: mercury vapor or mercuric ions. The mercury vapor can be inhaled or exhaled, depending on the subject's breathing pattern, while mercuric ions can pass into the saliva and enter the gastrointestinal tract.<sup>24</sup> The corrosion of amalgam restorations is complex and actually decreases the baseline release of mercury.<sup>24</sup>

### AMALGAM TOXICITY

A minority of dentists and physicians allege that the amount of mercury that "leaks" from amalgam restorations is sufficient to be a factor in developing or directly causing a host of diseases including, but not limited to, Alzheimer's disease, multiple sclerosis and immune system dysfunction.<sup>9</sup> This measurable leakage can enter the body through breathing mercury vapor or swallowing the mercury that dissolves in the saliva. The oral cavity constantly is wet owing to the continuous secretion of saliva and the high humidity of exhaled air. Since the absorption of mercury through the gastrointestinal tract is minimal, the mercury from amalgam that is swallowed adds very little to the total body burden of mercury.

Investigators have demonstrated that people with amalgam restorations have higher oral levels of mercury vapor than do people who do not have amalgam restorations.<sup>34</sup> Yet determining the amount of mercury released and absorbed from amalgam is difficult and complex. Olsson and Bergman<sup>35</sup> have listed the following factors as variables affecting the amount of mercury released from amalgam restorations: number of teeth, number of surfaces, baseline mercury re-

lease, magnification factors such as eating or toothbrushing, eating habits, toothbrushing habits, oral breathing habits, nose-mouth breathing ratio, inspiration-expiration ratio, swallowing, inhalation absorption, ingestion absorption and body weight.

These confounding variables have caused large variations in the estimates of daily mercury release and absorption. Several researchers<sup>36-41</sup> have arrived at figures higher than 10  $\mu\text{g Hg}/\text{m}^3$ , but other researchers<sup>34,42-46</sup> consistently have reported a much lower dose of mercury of around 1 to 2  $\mu\text{g}/\text{day}$ . In 1992, Olsson and Bergman<sup>35</sup> arrived at an amount of 1 to 2  $\mu\text{g}/\text{day}$  of mercury uptake for subjects with more than eight amalgam restorations.

Analysis of the data concerning daily mercury release and absorption leads me to conclude that mathematical errors led to serious miscalculations in arriving at the total amount of mercury vapor exposure. These computational errors led many investigators to overestimate the amount of mercury that is released and absorbed during daily life. The International Committee on Maximum Allowable Concentration of Mercury Compounds gives a TLV of 50  $\mu\text{g}/\text{m}^3$  of mercury vapor.<sup>18</sup>

There also are two levels that are used in determining industrial and other thresholds for mercury concentrations in the air. One is the lowest observed adverse effect level, or LOAEL, and the other is the no observed adverse effect level, or NOAEL. These thresholds are based on the levels at which adverse effects appear or fail to appear. The LOAEL is 100  $\mu\text{g}/\text{m}^3$  for clinical mercurism and 50  $\mu\text{g}/\text{m}^3$  for nephrotoxicity. Both of these levels relate to constant mercury exposure during a 40-hour work week. The NOAEL is 25  $\mu\text{g}/\text{m}^3$  for WHO industrial threshold, 5  $\mu\text{g}/\text{m}^3$  for the general public threshold, and 1  $\mu\text{g}/\text{m}^3$  for children, pregnant women and ill people (the last two levels relate to continuous mercury exposure).<sup>47,48</sup>

### ESTIMATES OF TOXIC MERCURY LEVELS

Using the lowest established value—the NOAEL for children, pregnant women and ill people of 1  $\mu\text{g Hg}/\text{m}^3$ —as a safe threshold for continuous mercury vapor exposure for the general public and assuming a respiration rate of 22  $\text{m}^3$  per day, a safe threshold for mercury vapor absorption by

Corrosion is a major factor in determining the amount of mercury that is released into the oral cavity.

the lungs is 20 µg/day.<sup>29</sup> Eley<sup>29</sup> also estimated the safe level for intestinal absorption of mercury from amalgam by multiplying the lowest NOAEL figure by a factor of 10 to reflect the low absorption by the gastrointestinal tract and then another factor of 2 to account for the reduced toxicity of mercuric compounds. This yielded a safe threshold for gastrointestinal absorption of salivary mercury of 400 µg/day.

### ADVERSE HEALTH CLAIMS

Eggleston<sup>49</sup> claimed that the mercury from amalgam reduced lymphocyte responses, thereby compromising immune function. Mackert and colleagues<sup>50</sup> criticized Eggleston for not blinding his study and not giving a thorough review of his methodology. Mackert and colleagues<sup>50</sup> measured the levels of three major populations of lymphocytes in 37 subjects, 21 who had amalgam restorations and 16 who did not. The results of this study showed no indication that amalgam affects the human immune system.

Mercury from amalgam also has been implicated in the development of Alzheimer's disease.<sup>51</sup> However, two studies on patients with Alzheimer's disease and on a population of nuns strongly suggest that this is not true.<sup>52,53</sup> Saxe and colleagues' study,<sup>53</sup> in particular, was compelling because the participants were Roman Catholic nuns who were 75 to 102 years old and who had lived together in a relatively homogeneous environment for many years. The nuns with amalgam restorations did not score lower than the nuns who did not have amalgam restorations on eight different tests of cognitive function.<sup>53</sup>

A number of studies have contrasted the general health of subjects who had and who did not have amalgam restorations. Mackert and Berglund<sup>54</sup> concluded that the extremely low dosage of mercury attributable to amalgam restorations was insufficient to produce any detectable negative effect on general health. Ahlqwist and colleagues<sup>55</sup> conducted a survey of more than 1,000 Swedish women, asking them about 30 specific symptoms and complaints. The researchers attempted to relate the answers to the size and number of amalgam restorations but could find no correlation. Berglund and Molin<sup>56</sup> measured the blood and urine mercury levels of people who had and who did not have complaints about amalgam toxicity. The researchers found the daily dose of mercury from the patient's amalgam

restorations was low in both groups and did not differ significantly between groups. These studies are compelling from an EBC viewpoint, as dose-response curves exist for all known environmental toxins, with subjects with more severe symptoms having higher exposure and higher body levels of the toxin in question. Indeed, in Ahlqwist and colleagues' study,<sup>55</sup> the women who had amalgam restorations actually exhibited better general health than did the women who did not have them. The authors said this probably reflected a greater concern for health matters among those women who received routine dental care.

It would seem logical and prudent to search for any evidence of disease among dentists, as they have been shown to have a much higher and consistent exposure to mercury vapors than the general public.<sup>57</sup> This is because dentists inhale dispersed mercury vapors every time they place or remove amalgam restorations.<sup>58,59</sup> Naleway and colleagues<sup>60</sup> reported findings from onsite screenings at the ADA annual sessions in 1985 and 1986. Measurements of concentrations of β<sub>2</sub>-microglobulin in serum and urine, of creatinine concentration in serum and of creatinine clearance were used to evaluate kidney dysfunction. The mean urinary values in the 1985 and 1986 surveys were 5.8 µg Hg/L and 7.6 µg Hg/L, respectively.<sup>60</sup> Approximately 10 percent of the subjects had urinary mercury concentrations higher than 20 µg Hg/L. No clear relationship was demonstrated between elevated urinary mercury concentrations and kidney dysfunction.<sup>60</sup> The general population has a mean urinary value of 1 to 3 µg Hg/L.<sup>61</sup> Although urine mercury levels can vary greatly from day to day and person to person, on a group basis, urine concentrations have been found to show good correlation with exposure to mercury vapor.<sup>62</sup> Dentists have a much higher mean urinary mercury value and yet exhibit no higher levels of morbidity or mortality.<sup>63</sup>

Boyd and colleagues<sup>64</sup> claimed that sheep kidney function was damaged dramatically by mercury from amalgam restorations. EBC analysis concludes that there was no damage because there was neither a pathological change in the kidney nor an increase in the blood urea nitrogen, which ordinarily will increase when there is an impaired glomerular filtration rate.<sup>65</sup> In addition, Sandborgh-Englund and colleagues<sup>66</sup> were unable to confirm Boyd and colleagues' findings.

Ekstrand and colleagues<sup>65</sup> found no effects on various parameters of kidney function in humans and concluded that sheep may not be appropriate models for testing the toxic effects of dental restorative materials.

Summers and colleagues<sup>67</sup> reported a significant increase in the proportion of mercury-resistant bacteria present in the intestines of six monkeys after amalgam restorations were inserted and removed. They concluded that amalgam may contribute to the emergence of drug-resistant bacteria. Edlund and colleagues<sup>68</sup> retested this hypothesis with human subjects. They found that analysis of the cohort with amalgam restorations gave significant results, but when they compared these results with the normal variations from a control group, the results no longer were statistically significant.<sup>68</sup>

Allergies to components of amalgam do exist. The allergic reaction to amalgam may be local or more widespread. The skin is the most common site, and the reaction often is self-limiting and subsides within two or three weeks even without the removal of the restoration.<sup>69</sup> The percentage of people who are allergic to mercury has been shown to be less than 1 percent.<sup>70</sup>

## DIAGNOSTIC METHODS

Antiamalgam advocates often use a number of scientifically unsupported diagnostic methods. One is the electrical reading of restorations that is done with a device similar to a common volt meter. This device is purported to provide the data necessary to determine the sequence of removal of the amalgam restorations.<sup>9</sup> Marek<sup>71</sup> stated that this device actually records the “difference between the corrosion rate without that contact of two materials [the electrical probe and the amalgam] and with the contact of two materials. It is not the corrosion rate, and there is no way by simple measurement to determine the corrosion rate or the release rate of ions from a metal in the mouth.” Marek further stated that because mercury is a more noble metal than the other components in amalgam, its long-term dissolution rate in saliva “is not high enough to be reason for concern.”<sup>72</sup>

A symptom questionnaire routinely is given to patients by dentists who believe that amalgam is

toxic. It often asks for a general history and includes specific questions concerning skin problems, nervous disorders, digestion, blood diseases, cancer, endocrine problems and emotional problems, as well as feelings of malaise, tiredness, restlessness, boredom or excitability that occur now or have in the past.<sup>9</sup> The list is so inclusive that any healthy person would find it hard not to confirm the presence of at least some of the telltale symptoms. These wide-ranging questionnaires neglect a cardinal rule of toxicology: the specificity of symptoms to a poison. Forensic pathologists often depend on a patient’s symptoms to determine what kinds of diagnostic tests should be performed to arrive at a proper diagnosis

and to begin proper treatment. In the case of amalgam, the diagnostic symptoms are so varied that it would be impossible to attribute all these responses to a single toxin.

Dr. Huggins<sup>9</sup> recommends using hair analysis to determine the patient’s calcium, manganese, mercury, zinc and potassium levels. Yet an EBC analysis of the literature demonstrates that “hair grows very slowly, so even samples taken close to the scalp may not reflect present

bodily conditions.”<sup>73</sup> Moreover, different laboratories reach different conclusions about the same hair samples,<sup>74</sup> and a normal range for minerals in the hair has not been established.<sup>75</sup> Nor is it clearly understood how mineral content of the hair relates to mineral concentration in the blood and tissues. Hair analysis may be of value in determining if a person was exposed to a toxic element such as arsenic, chromium or lead. But even then, shampoos and hair dyes can distort the test results.<sup>73</sup>

An industrial-grade mercury detector also often is used to diagnose mercury toxicity. This device multiplies the amount of mercury it actually measures by a large factor so that the reading will give the amount of mercury vapor in a cubic meter of air. Normal tidal volume—the amount of air entering the lungs during one normal breath—is 0.5 L<sup>24</sup> (human inspiratory capacity is 2.8 to 4.3 L), a volume far less than a cubic meter (1,000 L). As I mentioned previously, mercury release is inconsistent, and total daily dose is difficult to determine accurately. Taking a reading after a patient chews vigorously and then

.....  
**The list of supposed symptoms of amalgam toxicity is so inclusive that any healthy person would find it hard not to confirm the presence of at least some of the telltale symptoms.**  
 .....

extrapolating this value to represent daily dose can be frightening to a patient who is unaware of these methodological complexities.

Some physicians and dentists also use a skin patch test to determine “mercury allergy” or “hypersensitivity.” The reactions of the skin and the oral mucosa often are different. It is possible for the skin to be sensitized but not the oral mucosa, there may be concurrent sensitization of both skin and mucosa, or the mucosa may be sensitized but not the skin (a rare occurrence).<sup>62</sup> Interpretation of patch test results is difficult and requires the expertise of specially trained allergists. And even in cases in which these allergists are consulted, there are numerous situations that can lead to false positive or false negative reactions.<sup>76</sup> This makes patch testing for mercury allergy highly subjective and of little value.

**CONCLUSIONS**

The cardinal rule of toxicology is that “only the dose makes a poison.” Mercury can be toxic, for example, when high exposures occur in occupational settings. In these cases, the severity of response correlates well with the amount and duration of exposure. The relationship of dose (number and size of amalgam restorations), exposure time and symptoms has not been established.<sup>77</sup>

The call to ban amalgam is unusual among potential environmental toxins. In the past, a specific disease or condition has been recognized in a population that has been compared with a symptom-free population and possible causative agents are sought. The best example is the methods employed to epidemiologically establish the strong cause-and-effect nature of smoking and both lung cancer and heart disease. With amalgam, a wide range of diseases and conditions have been attributed to it based solely on self-reported improvements in symptoms when the amalgam restorations were removed. The already collected data on the morbidity and mortality of dentists who have a proven higher body burden of mercury than do the general public was ignored. Today, we also have compelling data from groups with and without restorations who lived under similar environmental conditions and these, too, refute the claims of amalgam toxicity.

Data strongly suggest that mercury levels many times higher than those associated with a



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mouth full of amalgam pose no risk of adverse health effects. There is evidence that the body’s mercury burden is highest immediately after placement or removal of amalgam restorations.<sup>29</sup> This information casts a critical light on those dentists, physicians and patients who have claimed improvement of symptoms immediately after amalgam removal.

EBC requires an “acceptance of an uncharacteristically high level of uncertainty concerning the impact of one’s clinical interventions.”<sup>78</sup> In contrast to this, Dr. Huggins has proposed that “in

order for mercury to be a problem, it would have to ... demonstrate remission of the symptoms on amalgam removal.”<sup>79</sup> Thus, he and those who are similarly opposed to amalgam base their conclusions on clinical judgments of symptom improvement. In EBC, the following are seen as potentially leading to incorrect conclusions about treatment efficacy when one relies on clinical observation:

- placebo effects of treatment;
- statistical regression toward the mean;
- spontaneous remission;
- natural variability of signs and symptoms;
- failure to consider treatment dropout;
- bias in self-reports of symptom remission.<sup>79</sup>

The logical and methodological errors of the leading opponents of amalgam restorations are clearly evident when analyzed using the EBC paradigm.

In 1993, James Mason, M.D., the Assistant Secretary for Health, reaffirmed the U.S. Public Health Service’s position that “there are no data to compel a change in the current use of dental amalgam.”<sup>80</sup>

This review supports Dr. Mason’s conclusion, and I propose that EBC be used by clinicians as a way to more accurately evaluate health care interventions. ■

The author would like to thank Dr. Wilmer Eames for his friendship and encouragement.

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