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Inhalation of Mercury-Contaminated Particulate Matter by Dentists: An Overlooked Occupational Risk

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ABSTRACT

Dentists are exposed to mercury (Hg) during the removal of amalgam fillings. Recent research on dentists and other occupational groups report neurological impairment at Hg exposure levels below the current occupational TLV® (25 $\mu g/m^3$) and/or the occupational BEI® (35 μg Hg/g creatinine in urine). Surveys of Hg^0 in the general office air of dental offices fail to measure the high levels of Hg-laden respirable amalgam particulate matter sprayed into the dentist's breathing zone during the removal of old amalgam fillings. This respirable particulate matter represents the vast majority of daily Hg exposure in practicing dentists. Despite this, no research is available on the pharmacokinetic fate of inhaled particulate amalgam Hg in humans. What indirect data does exist demonstrates that absorption from the lung occurs but that fecal excretion may predominate. As a result, urine analysis for Hg may be ineffective as a means of occupational monitoring. Various countries are moving to limit the use of amalgam as a dental restorative material in order to protect dental patients from Hg exposure. However, dentists' occupational exposure should also be considered as a justification for reduced amalgam use.

Key Words: mercury, amalgam, particulate, occupational, dentist, exposure.

INTRODUCTION

The removal of old amalgam fillings is probably the single largest source of mercury exposure to the dental practitioner. Dental amalgam contains 43 to 50.5% mercury (Hg) by weight in combination with silver, tin, copper, and other metals in lesser amounts (Berry *et al.* 1994). The process of removing an amalgam filling for subsequent repair and replacement generates high levels of Hg vapour (Hg^0) in the breathing zone of the dentist (Ritchie *et al.* 2002; Richards and Warren 1985; Buckwald 1972). Short-term levels (lasting throughout and only slightly longer than the filling removal procedure; measured in minutes) routinely exceed 100 $\mu g/m^3$ (Richards and Warren 1985) and can reach 2,500 $\mu g/m^3$ depending on the practices employed during the removal process (Buckwald 1972).

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Levels of respirable amalgam particulate matter (that is approximately 50% Hg by weight) are also high in the dentist's breathing zone during an amalgam removal procedure (Nimmo *et al.* 1988, 1989a, 1990; Werley *et al.* 1990). The subsequent potential for deposition of significant quantities of micron-sized and sub-micron sized particles to the lungs and alveoli is significant. Using manikin models, the removal of a single small 100 mg amalgam filling led to the collection of an average of 19 mg of amalgam particulate in the dentist model 'lung', with the average particle size being 2 μm in aerodynamic diameter (Nimmo *et al.* 1990). However, the average or typical amalgam filling is on the order of 1,000 mg in mass (Reinhardt *et al.* 1983), approximately 10 times greater than that used in the Nimmo *et al.* (1990) study. As a result, it is likely that the deposition of amalgam particulate to the dentist's lungs is much greater, on a filling by filling basis, than indicated by Nimmo *et al.* (1990).

The deposition of respirable dust to the lungs of persons in the dental industry is not just a hypothetical problem. Using magnetopneumographic techniques, Stahlhofen and Moller (1993) reported an average of 22 mg of metallic dust(s) in the lungs of dental technicians. Le Gros *et al.* (1988), using similar techniques, reported respirable dust deposition to the lungs of 4 dental technicians ranging from 725 mg to as much as 3,065 mg. Therefore, the problem of particulate inhalation within the dental industry is a real phenomenon.

The purpose of this paper is to review this occupational health issue and to provide a relative quantification of particulate inhalation as a source of Hg to dentists' occupational exposure.

SOURCES OF Hg EXPOSURE IN THE DENTAL OFFICE

Chronic sources of Hg contamination in the dental operatory include:

- leakage of Hg from dental amalgam capsules during storage (Burgess and Makinson 1983);
- leakage of Hg from capsules during trituration (mixing of mercury with the alloy powder to create the 'amalgam') (Anderson 1988; Anderson *et al.* 1988; Blitzer and Pollack 1981; Brown *et al.* 1984; Burgess and Makinson 1982; Capdeboscq and von der Lehr 1979, 1984; Carter and Marier 1982; Castagnola *et al.* 1973; Castagnola and Wirz 1974a,b; Cooley and Lubow 1984, 1985; Cooley and Barkmeler 1984; Cooley *et al.* 1985; Eames 1972; Gough *et al.* 1977; Jorgensen and Okuda 1970, 1971; Von der Lehr and Capdeboscq 1979; Newman 1979; Nicholson *et al.* 1968; Reinhardt and Taylor 1983; Schoenmakers 1979; Wilson and Wilson 1982; Donahue and Cooley 1987; Anderson *et al.* 1988; de Clerk 1983; Dünninger and Klaiber 1991; Oilo 1973; Vesely and Wirz 1984; Perotti and Corteletti 1987; Wirz and Schmidli 1993; Glockmann *et al.* 1990; Riccio *et al.* 1990);
- leakage of Hg from the inappropriate disposal of spent amalgam capsules to the operatory trash (Burgess and Makinson 1983; Cooley and Lubow 1985);
- emissions of Hg^0 from contaminated amalgamators (triturators) (Brooks Air Force Base 2000; Warfvinge 1995; Hooper 1980; Roberts *et al.* 2001);
- emissions of Hg^0 due to the heat-sterilization of contaminated dental instruments (Cooley *et al.* 1985; Rothwell *et al.* 1977);

- emissions of Hg^0 from amalgam particulate contamination within and throughout the dental operatory (Schneider 1974; Warfvinge 1995);
- emissions of Hg^0 from chair-side saliva extractors/aspirators that collect amalgam particulate matter during a filling removal procedure and vent contaminated air, often within the dental office (Rubin and Yu 1996; Smart 1990; Stonehouse and Newman 2001);
- inhalation of elevated Hg^0 in the dentist's breathing zone during the removal of old amalgam fillings and the placement of new amalgam fillings (Ritchie *et al.* 2002; Richards and Warren 1985; Haikel *et al.* 1990; Powell *et al.* 1994); and perhaps most significantly,
- the inhalation of amalgam particulate matter (approximately 50% Hg by weight) during routine amalgam filling removal procedures (Nimmo *et al.* 1988, 1989a, 1990; Werley *et al.* 1990).

A recent investigation in Scotland (Ritchie *et al.* 2002) found that the dentists studied had urine Hg levels more than 4 times greater than controls, and had increased kidney disorders, increased memory disturbances, and detriments in certain psychomotor performance measures relative to unexposed controls. That investigation complemented numerous other studies (Echeverria *et al.* 1995, 1998, 1999; Bittner *et al.* 1998; Foo *et al.* 1993; Gonzalez-Ramirez *et al.* 1995; Langworth *et al.* 1997; Ngim *et al.* 1992; Ritchie *et al.* 1995; Shapiro *et al.* 1982; Uzzell and Oler 1986) that have identified sub-clinical (pre-clinical) neurological, renal, and psychomotor effects in dentists and dental personnel with mercury exposures below current occupational exposure guidelines.

The inhalation of Hg-laden amalgam particulate matter by dentists removing old amalgam fillings has been largely overlooked as a source of occupational Hg exposure. Much of the particulate matter generated when removing fillings is trapped by water spray and removed to the clinic wastewater stream (Trip 2001). This source of Hg to the sewage system is now considered the single largest industrial contribution, and a Canada Wide Standard has been introduced to capture and remove this particulate from the dental clinic wastewater stream (Trip 2001). However, much of the particulate generated during an amalgam removal still escapes into the air, particularly into the breathing zone of the dentist, despite using water spray during the removal procedure (Nimmo *et al.* 1990). Effective means of monitoring and preventing the inhalation of this Hg-laden particulate matter by dentists have not been developed or promoted by the broad dental profession.

Although general room air Hg contamination within the dental office appears to have declined over the past 20 years (Brown and Sherriff 2002), this reflects, at least in part, the simple reduction in amalgam use. For example, in Canada the use of dental amalgam has declined approximately 23% between 1995 and 1999 (Richardson 2000). In Sweden, between 1991 and 1995 amalgam use declined in children from 30% to 1.5% of fillings placed, and in adults from 32% to 15% of fillings placed (Forssell *et al.* 2001). However, room air monitoring is ineffective in quantifying breathing zone Hg vapour levels and personal exposure in dentists.

As a means of monitoring occupational exposure to Hg, monitoring Hg^0 levels in the general dental office environment has been conducted (*e.g.*, Ritchie *et al.* 2002; Brown and Sherriff 2002). However, monitoring of general room air fails to

adequately reflect personal exposures in dentists and dental personnel. General room air concentrations of Hg^0 correlate poorly with urine Hg levels in exposed workers, whereas personal dosimetry of the breathing zone is far more effective as an indicator of exposure (Stopford *et al.* 1978). Data of Ritchie *et al.* (2002) show that the maximum personal dosimeter measurement (of breathing zone) for the dentists studied was 452 $\mu g/m^3$, whereas the maximum general room air Hg^0 concentration was only 24 $\mu g/m^3$. As previously mentioned, levels of Hg^0 in the dentist's breathing zone can routinely exceed 100 $\mu g/m^3$ (Richards and Warren 1985) and can reach 2,500 $\mu g/m^3$ depending on the practices employed during the removal process (Buckwald 1972).

The method of room air Hg^0 monitoring can also be important. Brown and Sherriff (2002), for example, report that only 5.05% of 1070 dental operatories monitored between 1996 and 2001 had a room air Hg level >25 $\mu g/m^3$. However, passive Hg sampling devices were used that were left in place for 24 hours per day over the course of 7 consecutive days. Therefore, the data collected identified dental offices where the average Hg^0 concentration exceeded 25 $\mu g/m^3$ (the occupational threshold limit value) for the 168 consecutive hours that the sampling device was exposed. Unfortunately, this 168 hours would include 128 hours beyond a typical work week of 40 hours (8 hours per day, 5 days per week), the period when active occupational exposure would most likely occur, and to which the occupational limit of 25 $\mu g/m^3$ applies. Many of those 128 hours would have been periods during which the dental office was vacant and inactive. As a result, such data would significantly underestimate the number of dental offices (and number of dentists) for which the occupational limit was exceeded for the critical exposure period.

Surveys of the concentrations of mercury in the urine of dentists attending dental conferences (Naleway *et al.* 1991, 1985) suggest that Hg exposure in this profession is generally within acceptable occupational limits. However, it is also apparent from those same studies that approximately 8% to 10% of the participating dentists had urine mercury levels greater than 35 $\mu g/g$ creatinine (assuming that, on average, 1 litre of urine contains on average 1 g of creatinine (Echeverria *et al.* 1995)), the current biological exposure index for mercury in urine (ACGIH 2002). A more recent, smaller scale survey (Ritchie *et al.* 2002) determined that the urine of dental practitioners ranged up to approximately 37 $\mu g/g$ creatinine (20.9 nmol Hg/mmol creatinine) and averaged 4 to 5 times the levels found in unexposed controls. However, urinary excretion may not be the principal excretory route for inhaled particulate Hg.

THE PHARMACOKINETIC FATE OF INHALED AMALGAM PARTICULATE

The pharmacokinetic and metabolic fate of inhaled amalgam particles is unknown. There are no published studies on the toxicokinetics of amalgam particulate administered via inhalation, nor are there studies on the fate of Hg that is inhaled in particulate form, such as in amalgam particulate (Clarkson 2002). Therefore, the kinetics of tissue distribution, metabolic transformation and excretion are unknown. It is impossible to know if excretion will occur predominantly via the urine, as with exposure to Hg vapour (WHO 1991), or if it will happen rapidly or gradually. Investigations of the absorption of inhaled inert, insoluble particulate matter indicate that

fecal excretion may dominate (Langenback *et al.* 1990). Therefore, urinary monitoring may be ineffective in identifying or quantifying particulate Hg inhalation exposures.

The amalgam particulate generated during a filling removal procedure is in the respirable range. The vast majority of dental-generated particulate is less than 3 μm in aerodynamic diameter (Nimmo *et al.* 1990; Nimmo *et al* 1989a; Brune *et al.* 1980), and at least 65% of the particulate generated by dental drilling is less than 1 μm in aerodynamic diameter (Brune *et al.* 1980). One study determined that more than 63% of the amalgam particulate generated in a dental office passed through a 0.7 μm filter (Cupelin *et al.* 1986). Therefore, particles generated during an amalgam removal procedure are well within the size range for full respirability.

Mercury and the other metals inhaled in amalgam particulate matter are systemically absorbed in a relatively short period of time. The World Health Organization (WHO 1991) concluded that, for inhaled particulate mercury, "significant absorption must take place directly from the lung." A study by Cutright *et al.* (1973) of rats exposed for inhalation only to a combination of amalgam particulate and Hg vapour (termed 'amalgam mist' by the authors) demonstrated increased blood mercury levels immediately following the onset of inhalation exposure, and the vast majority of the mercury deposited to the lungs was absorbed within 3 days. Since mercury vapor is rapidly absorbed immediately upon inhalation, the more slowly absorbed mercury contained in the lungs of the rats investigated by Cutright *et al.* (1973) must have been associated with amalgam particulate.

Mercury readily dissolves from amalgam. Chew *et al.* (1991) reported that mercury dissolved from set amalgam into distilled water at a rate of 43 μg per cm^2 of amalgam surface area per day. Gross and Harrison (1989) determined the loss of mercury from amalgam to be 37.5 μg per cm^2 of amalgam per day into Ringers solution. Hg^0 is lipid soluble (Lorscheider *et al.* 1995) and that alveolar fluid contains lipids (Hamm *et al.* 1992; Guthmann *et al.* 1995). Therefore, the solubility of mercury from amalgam, combined with the lipids contained in lung fluid, would combine to dissolve amalgam Hg within the alveoli in a relatively short period of time.

Absorption within a few days is also supported by other calculations. Based on the removal of 4 amalgam fillings per day (see below), the resulting intake of amalgam particles was 76 mg. Assuming an average aerodynamic diameter of 2 μm (2×10^{-4} cm) per particle (Nimmo *et al.* 1990), and with amalgam having a density of 11.5 g/cm^3 (Letzel *et al.* 1997), then the resulting number of deposited particles would be 1.578×10^9. The total surface area of all of those deposited amalgam particles would be 198.2 cm^2. Assuming a dissolution rate for mercury from set amalgam of 40 μg/cm^2-day (based on Chew *et al.* 1991 and Gross and Harrison 1989), and ignoring the influence on Hg solubility of the lipid content of the lung, then the 38 mg of Hg associated with that deposited amalgam particulate would dissolve out of that particulate matter within 4.8 days, an estimate close to that observed by Cutright *et al.* (1973).

Further evidence of the likelihood that amalgam particulate will be absorbed is offered by a study by Takenaka *et al.* (2001) who investigated the fate of metallic silver particles deposited to rat lungs. Although amalgam is approximately 50% mercury by weight, it is also approximately 35% metallic silver by weight in the set amalgam (Berry *et al.* 1994). Metallic silver is not volatile and is considered

to be insoluble (ATSDR 1990). Takenaka *et al.* (2001) reported that 96% of deposited silver particulate was removed from the lungs of rats within 7 days, not greatly dissimilar to the 3 days for elimination of the amalgam Hg from the lungs of rats in the Cutright *et al.* (1973) study. Takenaka *et al.* reported that levels of silver in the blood increased immediately after deposition of the silver particles to the lungs, clearly indicating that systemic absorption of that insoluble metal was occurring.

Interestingly, when amalgam is placed in saline solution, silver is not detected (Kozono *et al.* 1982), whereas Hg is detectable (Gross and Harrison 1989). Therefore, it is probable that all of the metals contained in amalgam (silver, copper, tin, zinc, indium, palladium, and possibly others) are absorbed from the lungs, irrespective of their water solubility or lack of volatility.

The fact that many, if not most, dentists now wear face masks may have little impact on mercury exposures during filling removal procedures. Obviously, such masks offer no protection against Hg vapour exposures. However, little protection is likely offered against particulate exposures either. Commonly used dental face masks have a stated filtering efficiency of at least 95%, but this stated filtering efficiency is for particles greater than or equal to 3 μm in aerodynamic diameter (Christensen *et al.* 1991). The efficiency for filtering out particles of less than 3 μm is not readily available, and available data indicate that their efficiency at filtering out particles that are less than or equal to 1 μm in diameter is very poor. A study undertaken by Nimmo *et al.* (1989b) showed that, although larger particles were stopped, particles of 1 μm or less readily passed through common dental masks. With the majority of amalgam particles being $\leq$1 μm, the respiratory protection offered by typical dental face masks is questionable.

THE RELATIVE CONTRIBUTIONS OF PARTICULATE AND VAPOUR INHALATION TO Hg EXPOSURE IN DENTISTS

To provide a relative comparison between amalgam particulate and Hg vapour as a source of mercury exposure in dentists, a simple assessment of exposures was undertaken. The three sources of mercury considered were inhalation of amalgam particulate during the removal of old fillings, inhalation of Hg vapour during the removal process, and the inhalation of Hg vapour contained in the general office environment when removals were not being conducted.

A recent survey (Martin *et al.* 1995) suggests that the removal of 4 amalgam fillings per day is not unusual for a practicing dentist. Assuming that the removal of one filling results in the inhalation of 19 mg of amalgam particulate (Nimmo *et al.* 1990) then 4 removals per day would contribute 76 mg of amalgam particulate and approximately 38 mg of Hg (based on amalgam being 50% Hg by weight).

During a filling removal procedure, which lasts approximately 10 minutes (Powell *et al.* 1994), the Hg^0 levels in the breathing zone of the dentist rise rapidly and significantly, remaining elevated for the duration of the procedure. Although levels of Hg^0 in the dentist's breathing zone can, in some cases, exceed 2.5 mg/m^3 (Buckwald 1972), it is more likely that levels average approximately 0.1 mg/m^3 following currently accepted dental practice techniques (Richards and Warren 1985). Assuming

an inhalation rate for the dentist of 1 m^3/hour (value prescribed for light occupational activity; USEPA 1997), then 4 amalgam removals at 10 minutes each (Powell *et al.* 1994) would result in the inhalation of 0.067 mg of Hg^0.

Finally, it can be assumed that the dentist inhales Hg^0 from the general office environment for those periods when he/she is not involved in amalgam removals. Assuming a typical 8 hour day, of which 7.33 hours are spent not performing removals, assuming an average or typical Hg^0 concentration in dental office air of 0.007 mg/m^3 (Ritchie *et al.* 2002), and assuming the same 1 m^3/hour inhalation rate, then the daily intake of Hg^0 at times other than during amalgam removals would be 0.05 mg of Hg^0.

Based on the simple assumptions and calculations presented above, it is obvious that the inhalation of amalgam particulate provides the greatest potential source of Hg intake; >99% of total intake per day.

DISCUSSION AND CONCLUSIONS

Considerable attention has been paid over the past two decades to the potential risks posed to dental patients by the mercury contained in dental amalgam (reviewed by Richardson and Allan 1996). Dental patient exposure to Hg is now well quantified, with at least 13 different authors having offered analyses of the chronic Hg exposures to dental patients with amalgam fillings contained in their teeth (see Figure 1; Mackert and Berglund 1997; Richardson and Allan 1996; Halbach 1995; Weiner and Nylander 1995; CCEHRP 1993; Olsson and Bergman 1992; WHO 1991; Vimy and Lorscheider 1990; Berglund 1990; Aronsson *et al.* 1989; Snapp *et al.* 1989; Clarkson *et al.* 1988; Patterson *et al.* 1985; Mackert 1991, 1987).

Concerns for this Hg exposure in dental patients have lead a variety of countries, including Canada (HC 1996), Norway (NDHSS 2002), Germany and Austria (USPHS 1997), to publish or propose guidelines aimed at limiting mercury exposure in the general population, at least to those individuals or groups who may be predisposed to mercury toxicity. Sweden has taken the further step of eliminating the placement of dental amalgam fillings as a reimbursable procedure under their national health care program (Forssell *et al.* 2001).

Mercury exposures in practicing dentists, on the other hand, have received little or no regulatory control. Although a large number of mercury sources to the dental operatory have been identified, there has been relatively little effort to directly control or eliminate those potential exposures, relying instead on education and information dissemination within the profession.

Despite advances over the past two decades in occupational hygiene practices and the handling and storage of mercury-contaminated wastes within dental clinics, dental practitioners still have significantly elevated urinary mercury levels relative to unexposed controls. Despite the fact that the vast majority of those urinary levels appear to be below currently accepted occupational limits, those limits are under increasing scrutiny as not providing adequate protection from the neurotoxicological effects of Hg exposure (Echeverria 2002).

It is apparent that the single largest source of mercury exposure to the practicing dentist is the removal of old amalgam fillings. The dose of mercury contained in the

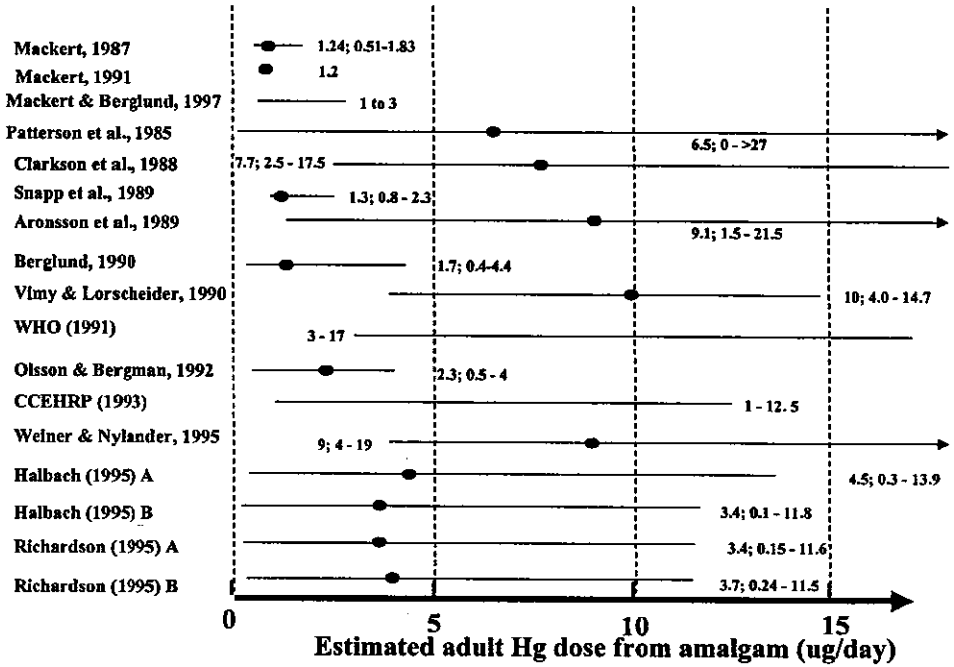

Figure 1. Published estimates of mercury exposure arising in dental patients from amalgam fillings. Estimated exposures exclude short-term doses resulting from removal and placement procedures. Horizontal lines indicate range of exposure reported. Circles indicate mean value if reported by authors. For CCEHRP (1993), range corrected for up to 25 filled teeth, the upper limit of fillings found in adults.

amalgam particulate inhaled with the removal of just a single small filling far exceeds that associated with the inhalation of Hg vapour contained within the general dental operatory air over the course of an entire week.

With the lack of data on the pharmacokinetics of Hg contained in inhaled particulate, it is impossible at present to know if urinary excretion is the dominant excretion pathway and subsequently, if urinary Hg concentration is an adequate and effective bio-monitor for particulate Hg exposure. Available data for particulate matter in general suggests that it is not. Therefore, the relevance of urinary Hg monitoring as a biological index for particulate Hg exposure is uncertain.

The use of dental amalgam is declining in industrialized countries. With the advent of modern dental materials with an aesthetically pleasing colour, there is increasing consumer preference to avoid 'silver' fillings. Therefore, it is apparent that the use of amalgam as a dental material in Canada and other industrialized countries will eventually cease. However, it seems apparent that occupational exposure issues should also be considered, both with respect to the continued use of amalgam and in the setting of any time line for its elimination from the dental practice.

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