Effect of placing, removing and polishing of amalgam restorations on 24-h urinary mercury concentration

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Twenty-four-hour urinary mercury concentration were assessed in 43 patients before and after removing old amalgam fillings (8 pts), placing (13 pts), and polishing of amalgam (22 pts). Baseline analyses 8 days before the treatments showed on average $18.5 \pm 7.2 \,\mu g$ mercury mass excreted per 24-h urine samples. The removal of old fillings caused a total excreted mass of $56.3 \pm 32.3 \,\mu g$ Hg, the placing of amalgam $45.9 \pm 26.2 \,\mu g$ Hg, and the polishing $56.25 \pm 33.77 \,\mu g$ Hg, respectively, one day after the treatments. When compared with the baseline values, the urinary mass excreted remained significantly elevated during the 8-day follow-up. However, all Hg values measured were below the WHO recommandations for the threshold limits for urinary mercury.

1. Introduction

Amalgam is the most frequently used material for dental restorations in posterior teeth. Dental amalgam releases mercury as vapour [1–3], as ions [4, 5], and as particulate matter [6]. In groups of people with no other known exposure to mercury than their amalgam restorations, there is a statistically significant correlation between urinary mercury concentration and the number of amalgam restorations [7, 8]. Because mercury from amalgam restorations, no doubt, contributes to the body burden, its possible side effects have been extensively discussed throughout the history of use of dental amalgam [9, 10].

Mercury vapour is released, especially during the insertion, condensation and carving of amalgam [7,11–13], during the removal of old amalgam restorations [13–17], and during polishing [13]. This mercury can be measured in the expired air and saliva. The amount is in direct proportion to the free surface areas of amalgam restorations [7, 8, 11, 13]. It is estimated that at least 75–80% of inhaled mercury vapour is rapidly absorbed by pulmonary epithelium into the blood stream [18].

The purpose of the present study was to determine the mercury exposure related to insertion, removal and polishing of amalgam restorations. This was done by measuring the urinary mercury concentration collected over 48 h from individuals, before and after a single session of restorative treatment.

2. Materials and methods

2.1. Subjects

The non-randomized study group consisted of 43 persons who came for dental treatment to the University Dental Clinic in Strasbourg, France, and permitted the analysis after the following procedure: removing old amalgam (8 patients), placing of amalgam (13 patients), and polishing of amalgam (22 patients). The patients mean age was 23 years (range 22 to 24). None of the patients had been occupationally exposed to mercury. The treatment procedures and materials were those normally applied in the clinic. The duration of the treatments ranged from 20 to 30 min. To adjust for the size differences of the amalgam restorations, which varied depending on the cavity type and the extent of the caries lesions, each restoration was scored according to the method of Olstad et al. [7]. Score 1 denoted Class I and V cavities, grooves and pits; score 2 described Class II restorations and large occlusal amalgams; and score 3 concerned only molars and large restorations including at least three tooth surfaces. The total number of amalgam surfaces was registered. The mean number was 4.27 ± 2.16 surfaces in the setting group, 4.82 ± 1.07 in the removing group and 13.75 ± 4.23 in the polishing dental restorations group.

2.2. Restorative procedures

The amalgam used for the restorations was nongamma 2-amalgam (Dispersalloy from Johnson and Johnson, DPC, New Jersey, USA, batch no. 2921), placed manually into the cavities. Old restorations were removed with a cylindrical carbide drill (Komet 558-012) fitted on a high-speed rotary instrument. All amalgam fillings were removed by means of water spray and by using a vacuum evacuator. Polishing

TABLE I Effect of placing removing and polishing amlagam restorations on urinary Hg expressed in μ g Hg/24 h and in μ g Hg/g creatinine, and statistical analyses by Behrens–Fisher test (t_{BF}). d - 8 = baseline measurement 8 days before treatment, d + 1 = measurement 1 day, and d + 8 = measurement 8 days after treatment

Treatment	Hg concentration	Time of measurement			Statistical analysis					
				$(2) \\ d + 8$	(0)–(1)			(0)-(2)		
					t _{BF}	5%	1%	t _{BF}	5%	1%
Removing	μg Hg/24 h	19.24 ± 6.62	56.32 ± 32.33	29.13 ± 12.00	3.18	+	_	2.04	-	_
<i>n</i> = 8	µg Hg/g creatinine	17.42 ± 7.50	63.40 ± 36.15	30.86 ± 18.88	3.52	+	±	1.87		_
Setting	μg Hg/24 h	16.49 ± 7.07	45.93 ± 26.16	27.56 ± 14.16	3.92	+	+	2.52	+	_
<i>n</i> = 13	µg Hg/g creatinine	12.54 ± 7.71	39.26 ± 30.17	28.03 ± 25.06	3.09	+	+	2.13	—	-
Polishing	µg Hg/24 h	19.83 ± 7.82	56.25 ± 33.77	38.92 ± 26.11	4.92	+	+	3.29	+	+
<i>n</i> = 22	µg Hg/g creatinine	18.17 ± 14.71	44.24 ± 40.70	34.25 ± 27.10	2.83	+	+	2.45	+	



Figure 1 Urinary mercury values in relation to creatinine ($\blacksquare d - 8$; $\blacksquare d + 1$; $\blacksquare d + 8$). See Table I caption for explanation.

was done with a succession of instruments fitted on a handpiece [13].

2.3. Sampling

From each patient, three samples of urine were collected over 24 h periods at 8 days before, 1 day after and 8 days after dental treatment. The subjects were instructed to urinate directly into the sampler container over 24 h.

2.4. Analyses

The samples were analysed by atomic absorption spectrometry in cold vapours (IL 15, Instrumentation Laboratory Incorporated, Lexington, MA) [19]. This method has a detection limit of 10 µg/l. Samples of 200 µl were treated either directly or in cold mineralization. This latter technique used a mixture of 2 ml H_2SO_4 and 3 ml HNO₃. Mercury was reduced in stannous chloride according to the equation $Hg^{++} + Sn^{++} \rightarrow Sn^{4+} + Hg^0$. The forming of metallic mercury was obtained in a reaction vessel by the cold vapour method [20]. The signal obtained by spectrometer was then converted to µg/l based on previous calibration.



Figure 2 Urinary mercury in relation to total excreted mass of mercury per 24 h ($\boxdot d - 8$; $\boxtimes d + 1$; $\boxtimes d + 8$). See Table I caption for explanation.

For each sample, a blank reference was prepared and analysed in the same conditions but without urine samples and this signal was deducted from the urine mercury value obtained by spectrometer. A standard reference (Titrisol Merck) of 1 g/l of mercury was used for calibration and for quality control. The results of the analyses were expressed in total excreted mass of Hg per 24 h (μ g Hg/24 h) and in μ g of mercury per gram of creatinine (μ g Hg/g creatinine).

The creatinine was determined by Technicon Method. (Technicon Instruments Corporation, R.A. 1000 System, New York).

A Behrens-Fisher test was used for statistics and significance was accepted at the 5 and 1% levels.

3. Results

The removal of old fillings with dental amalgam leads to a significant increase of Hg concentrations in the 24 h urine samples. The difference was statistically significant when compared with baseline determination (p < 0.05) (Table I). Urinary excretion of Hg still continued 8 days later, but this difference was statistically not significant when compared with the Hg concentrations observed before the removal procedure (Table I; Figs 1 and 2).

The setting of dental amalgam leads to a highly significant Hg increase in the urine samples collected during the 24 h after the procedure (p < 0.01). Urinary excretion of Hg remained statistically significant in the samples collected 8 days after the restorative procedure, when expressed in μ g Hg/24 h (p < 0.05) (Table I; Fig. 2).

The polishing of dental amalgams in the oral cavity leads to greater excretion of mercury in urine, statistically significant 24 h (p < 0.01) and 8 days (p < 0.05) following the procedure, when compared with baseline values (Table I; Figs 1 and 2).

No statistical correlation was found between scored amalgam surfaces and urine mercury concentrations.

4. Discussion

Dental amalgam releases mercury, especially during placing, polishing and removing retorations [13]. The present study was performed to explore whether release of Hg in these situations would measurably influence the individual urinary Hg concentration of the patients due to a single-session treatment with dental amalgam.

The release of Hg from an amalgam restoration is at its peak following amalgam procedures, and then declines to a much lower, steady-state level 10 to 15 days after treatment [4, 21, 22]. The results of the present study are in agreement with this, indicating that exposure to Hg vapour affects urinary Hg level. The urinary mercury elimination was at its peak the first day after amalgam procedures and decreased significantly after 8 days. In recent publications no change in the mercury level of urine could be detected 9 days or longer after placement of amalgam in the oral cavity [7, 23, 24]. This result, however, may be due to the small number of subjects and the small amount of amalgam used in their studies. In the study by Frykholm [25], the urinary Hg concentrations came down to the pre-removal level after 2 weeks. However, in a study by Molin [26], the mercury values came down to pre-removal levels after 3 months.

The Institute of Occupational Health in Oslo, Norway, regards 10 nmol Hg/mmol creatinine (18 μ g/g creatinine) as an upper limit for urinary Hg concentration in a Norwegian reference population without occupational exposure to mercury [27]. Following an international survey [28] 20 μ g/l was adopted as the upper limit for normal concentrations in the urine. The World Health Organization recommends the following threshold limits for urinary mercury: 50 nmol/l (10 μ g/l) in unexposed people, 250 nmol/l (50 μ g/l) in mercury exposed [29] people. In the present study, all concentrations before amalgam procedures were well below these limits.

In all of the published data, the urinary concentration of Hg can fluctuate considerably: this can, to some extent, be reduced by relating the urinary concentration to creatinine, thus obtaining a more reliable measure of Hg exposure in spot urine samples [7]. However, in the present study the Hg determination was made in total urinary samples collected over 24 h, which is a more reliable measure in reflecting the exposure to mercury than spot sampling. Our analyses expressed in μ g Hg/24 h seem to be more reliable than those expressed in μ g Hg/g creatinine. The theoretical translation of the data expressed in μ g Hg/24 h does not correspond to the experimental values of μ g Hg/g creatinine.

Another typical feature of all the published data including the present one is the marked individual variation, which can be seen in the high standard deviations [1, 13, 30, 31]. This concerns Hg levels in blood, urine, intra-oral air and expired air due to mercury exposure used in dentistry. This may be partly because of analytical problems, but may also reflect the fact that ingestion, retention, metabolism, and toxicity of any toxic chemical are markedly influenced by such confounding factors as heredity, race, age, sex, dietary habits, presence of various diseases and even social habits [32].

The inhaled Hg vapour in man, its accumulation in the various organs, and the elimination processes are complex, and are not only dose dependent but also influenced by the duration of exposure [32–34]. It is estimated that at least 75–80% of inhaled Hg vapour is rapidly absorbed across the pulmonary epithelium into the blood stream where rapid oxidation to divalent ionic Hg takes place, catalysed by the enzyme catalase-hydrogen peroxidase.

Evidence from occupational health studies indicates a correlation between workers exposed to an average air mercury level of $50 \ \mu\text{g/m}^3$ and urinary mercury concentrations of approximately $150 \ \mu\text{g/l}$ [35]. However, in dentistry the patients are exposed to Hg vapour only for a short time during a single treatment amalgam procedure. Haikel *et al.* [13] showed that mercury vapour was released during all amalgam procedures: removing, setting, and polishing, and that the mean levels were between 85 and 326 $\mu\text{g/m}^3$.

Roels *et al.* [36] noted that following mercury exposure, neurological and renal changes, supposedly reversible, begin to occur at a urinary mercury level of about 50 μ g/g creatinine. However, this value holds only for chronic exposure to Hg vapour and there is no evidence that the amount of mercury vapour released from amalgam restorations causes harm to the patients [37]. To conclude, the results of the present study show that mercury from amalgam procedures significantly contribute to the Hg concentrations in urine for only a short time.

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