

Effect of dental amalgam restorations on the mercury content of nerve tissues

Krister Nilner, Sigvard Åkerman and Björn Klinge

Department of Prosthetic Dentistry, Department of Stomatognathic Physiology, and Department of Periodontology, Faculty of Odontology, University of Lund, Malmö, Sweden

Nilner K, Åkerman S, Klinge B. Effect of dental amalgam restorations on the mercury content of nerve tissues. *Acta Odontol Scand* 1985;43:303-307. Oslo. ISSN 0001-6357.

In an autopsy study in two men and an experimental study performed on three female beagles the mercury burden of nerve tissues was determined. Nerve tissues from the head and face region and from three peripheral nerves were analyzed for mercury content with the aid of atomic absorption spectroscopy. In the dogs dental amalgam restorations were placed so as to investigate the possible influence from the amalgam on the mercury content of the tissues under study. The mercury content in man and dogs differed widely from one nerve to another, with no apparent relation to the number, type, or location of tooth restorations. □ *Atomic absorption spectroscopy; dental amalgams; dogs; human autopsy; mercury; nerve tissue*

Krister Nilner, Department of Prosthetic Dentistry, Faculty of Odontology, University of Lund, S-214 21 Malmö, Sweden

The possibility of toxic effects of the mercury contained in dental amalgams has for decades been extensively discussed in the dental literature, with reference to both dental professional personnel and patients (1-3). By and large, the conclusions of these discussions imply that the risk to the patient from mercury in dental amalgam is negligible (4), even though it is indisputable that mercury is released in minute amounts from dental amalgams.

In vitro studies have clearly shown the presence of mercury in different electrolytes as a result of corrosion processes in dental amalgams and the influence of, for example, the pH value of the test solution on the mercury release (5, 6).

One objective of earlier studies has therefore been to trace in vivo the mercury released from dental amalgam restorations. Some investigators (7, 8) were not able to find detectable amounts of mercury in saliva with their respective analytical methods, but Möller (9) and Svare et al. (10) demonstrated mercury from dental restorations in the dental pulp and expired air, respectively.

The finding of mercury in the pulp is of particular interest, since in this location mercury might be bound to nerve tissue and hence subjected to retrograde axonal trans-

port, as suggested by Hansson (11) and shown for other heavy metals in other nerves (12).

In the present work we therefore wanted to study whether the amount of mercury was higher in human trigeminal nerves than in other nerve tissues in humans with dental amalgam fillings (aim of part I).

It was also considered of interest to compare the findings from the study of human nerve tissues with corresponding values from a study in beagles in which silver amalgam restorations had been experimentally placed (aim of part II).

Materials and Methods

Part I

In two 74-year-old men who before death had donated their bodies to research the dental status, including location and extent of the metallic dental restorations, was recorded. During the autopsy procedure nerve tissues were dissected and placed in 10% formalin solution in glass bottles. The nerve tissues dissected are presented in Table 1. The tissues were transferred to the laboratory, where they were dried and weighed.

Table 1. Nerve tissues dissected from two persons (A and B) at autopsy for detection of mercury content

Tissue	Person	
	A	B
Chiasma opticum	X	X
Left radial nerve	X	X
Right lower alveolar nerve		X
Left lower alveolar nerve	X	X
Right trigeminal ganglion		X
Left trigeminal ganglion	X	X

The mercury content was assayed by flameless atomic absorption spectroscopy (Warian AA 6) and expressed as $\mu\text{g Hg/g}$ dry weight (equals ppm).

Part II

In two of three female beagles, 36–48 months old and weighing 9–15 kg, silver amalgam restorations were placed in the cuspid(s) and first molar(s). The third dog served as an untreated control.

After administration of atropine sulfate to reduce salivation, the operative procedures were carried out under balanced anesthesia consisting of petidine, apozepam, and pentobarbital sodium. Approximately 2-mm-deep cavities were prepared on the buccal surface of the mandibular cuspid and first molar by using a diamond bur at high speed with water cooling. The cavity pulpal walls were based

with a thin layer of calcium hydroxide (Dycal®, Caulk, Del., USA), and silver amalgam restorations (Amalcap®, Ivoclar, Liechtenstein) were placed with hand instruments and condensed in a routine manner.

In an attempt to study the effect of time, in one of the dogs dental amalgams were placed at two sessions. The locations of the restorations and time of exposure are presented in Table 2. During the entire experimental period all the three dogs were fed an identical soft canned diet (Vov®, Tre Kök, Sweden).

Euthanasia was performed by rapid intravenous injection of a saturated solution of pentobarbitone, and nerve tissues were dissected (Table 2). To remove the inferior alveolar nerve, a full-thickness flap was raised distally from the first molar on the lingual aspect. The nerve was separated from the surrounding tissues, and a 10-mm piece was cut with a scalpel. After the parietal bone had been removed, the brain was brought down, and the trigeminal ganglion was dissected with a chisel and cut with a scalpel. By means of blunt dissection the ischiadic nerve in the hind leg was reached and also cut. All biopsy specimens were directly transferred to mercury-free plastic test tubes (Vacutainer®, Becton Dickinson, France) and immediately frozen at -78°C , later to undergo the same analytical process as presented in part I. One piece of tissue was collected from each biopsy site and fixed in formalin. These specimens were embed-

Table 2. Locations of dental amalgam fillings, time of exposure, and mercury content ($\mu\text{g Hg/g}$ dry weight) of the nerve tissues analyzed in the three beagles (— = analyses not performed)

	Dog I		Dog II	Dog III (untreated control)
	Mandibular cuspid and 1st molar			None
Side	Left	Right	Left	
Exposure time	25 weeks	1 week	1 week	
Mercury content				
Right lower alveolar nerve	—	25	12	—
Left lower alveolar nerve	9	—	1	13
Right trigeminal ganglion	—	23	—	—
Left trigeminal ganglion	1	—	—	240
Ischiadic nerve	—	<0.5	—	15

ded in paraffin, sectioned, and stained with Gridley's selective nerve staining.

Results

All biopsy specimens submitted to selective nerve staining techniques were shown to consist of nerve tissues.

Part I

The results of mercury analyses of the different nerve tissues are illustrated in Fig. 1 (person A) and Fig. 2 (person B) together with the dental status.

The highest mercury level, 130 $\mu\text{g/g}$, was found in the left trigeminal ganglion of person B, who had dental restorations of conventional gold alloy and of amalgam. The second highest values were found in the left radial nerve, 70 $\mu\text{g/g}$, of person A, who had 66 $\mu\text{g/g}$ in his left trigeminal ganglion. The dental restorations in person A were mainly made of amalgam, but some porcelain fused to metal crowns was also present. Other analyzed tissues showed only small amounts of mercury, ranging from 0.03 $\mu\text{g/g}$ to 3.3 $\mu\text{g/g}$.

Part II

The results of the analyses of the mercury content in nerve tissues from the beagles are given in Table 2. The largest amount of mercury, 240 $\mu\text{g/g}$, was found in the untreated control animal's left trigeminal ganglion.

In dog I more mercury was found in the lower alveolar nerve and the trigeminal ganglion from the side that had been exposed to dental amalgams for 1 week than on the side exposed for 25 weeks.

Dog II had more mercury on the non-treated side in the lower alveolar nerve than on the side with the restorations.

With regard to the mercury content in the ischiadic nerves the analyses showed that the untreated control held 15 $\mu\text{g/g}$, whereas the dog that had dental amalgams placed both at 25 weeks and at 1 week before the dissection had a burden of less than 0.5 $\mu\text{g/g}$.

Discussion

Little is known about the 'normal' levels of mercury content in human tissues. Some attempts to survey different organs have been made (13-15), but the vast majority of

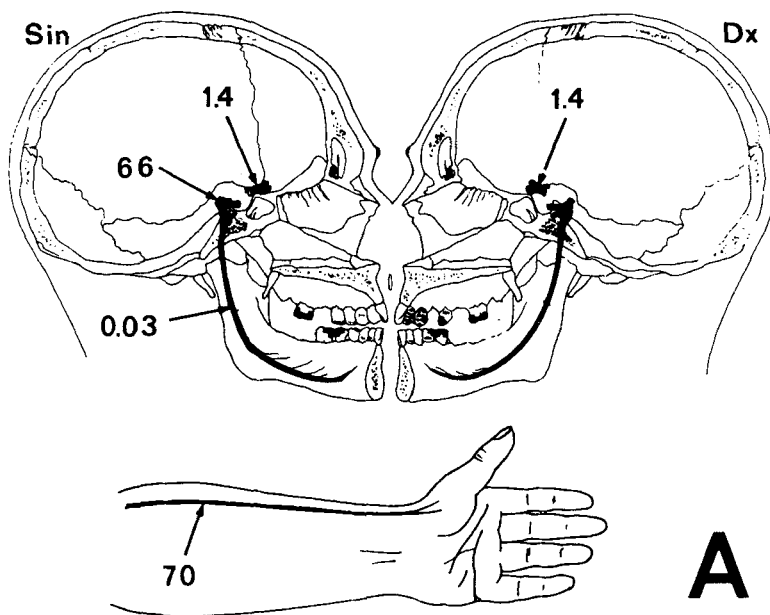


Fig. 1. Mercury content ($\mu\text{g Hg/g}$ dry wt) in different nerve tissues from person A, indicated by arrows and explained in Table 1. Dental status: black areas = amalgam; dotted areas = porcelain fused to metal.

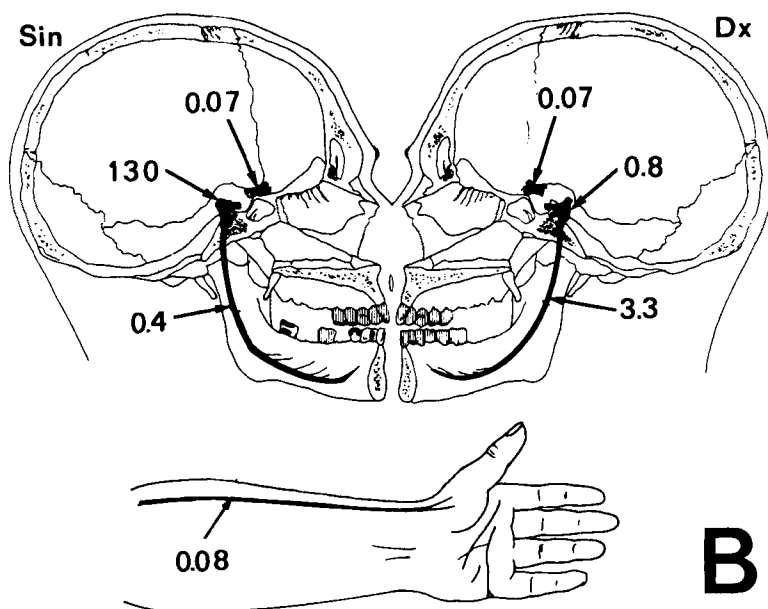


Fig. 2. Mercury content ($\mu\text{g/g Hg/g dry wt}$) in different nerve tissues from person B, indicated by arrows and explained in Table 1. Dental status: black areas = amalgam; hatched areas = conventional cast gold alloy.

studies have concentrated on blood and urine contents (4, 16). Because only a few reports are available on the mercury burden of human brains (13, 14) and because information on corresponding values from experimental studies in animals are even more scarce (17), the present mapping of mercury in nerve tissues was considered justifiable, especially when bearing in mind the enigma of mercury toxicity and dental treatment with amalgam reconstructions.

The method used for detection of mercury has been applied and proved appropriate in several other investigations (9, 13, 18). An autopsy study combined with an experimental study is advisable, since any findings in such an autopsy study cannot entirely be correctly evaluated. In this study it was not possible to obtain sufficient information on, for example, therapeutic occupational or environmental data and exposure to dental treatments because of local restrictions.

The minor change in the methods between part I and part II, of storing the tissues deep frozen instead of in formalin, represents an improvement. Formalin may act as a reducing agent (oxidizer), and to avoid any loss of mercury due to chemical reactions in the medium, it was decided to freeze the tissues in the experimental part of the study.

The findings reported are disparate. From the autopsy part it could be concluded that the trigeminal ganglion is loaded with more mercury when the patient has dental restorations of different metal alloys. Such an assumption is, however, contradicted when taking into consideration that in the experimental study most mercury in the trigeminal ganglion was found in the untreated control animal. Thus it is apparently not appropriate to discuss possible effects of electrochemically corrosive processes and ion release in different metallic reconstructions (8, 20), even though low values of mercury content were found in the trigeminal ganglion innervating a jaw with merely one kind of alloy—that is, in the right side of person B.

Möller (9) found large amounts of mercury in the dental pulp under silver amalgam restorations than in dental pulps from teeth without restorations, but the present study gives no evidence of augmented amounts of mercury in nerve tissues beneath calcium hydroxide-lined amalgam restorations. Möller's figures reflect his experimental design, which did not include the use of a cement base in deep cavities. Beneath our experimentally placed amalgams the cavity pulpal walls were based as in clinical work, indicating a reduced access of mercury to the

dental pulp through the dentinal tubules. Less mercury in the dental pulp probably means less mercury to be transported along the nerve tissue. However, dog II exhibited less mercury in the lower alveolar nerve from the side with amalgam restorations than in corresponding tissue from the contralateral untreated side. Consequently, such findings may contradict potential retrograde axonal transport of mercury from dental amalgams (11).

We have not compared our findings directly with those from other reports on mercury in different human organs and tissues. Difficulties will arise because the results are not given in the same units. For example, Joselow et al. (14) have presented their results in ppm wet weight, whereas Mottet & Body (13) and Guccione et al. (17) present theirs in $\mu\text{g Hg/g}$ wet tissues. These values are interchangeable but supposedly somewhat lower than the concentrations in $\mu\text{g Hg/g}$ dry weight reported in this study.

Finally, the mercury content in nerve tissues from the hind legs of the experimental animals and from the forearms of the cadavers lends support to the opinion put forward by Hoover & Goldwater (16) and Kröncke et al. (21) that the mercury burden of the body is not dependent on or affected by dental amalgam restorations.

References

1. Bauer JG, First HA. The toxicity of mercury in dental amalgam. *J Calif Dent Assoc* 1982;10:47-61.
2. Kallus T. Kvicksilver från dentala amalgam. En toxikologisk riskvärdering. *Tandlakartidningen* 1981;73:1226-38.
3. Rupp NW, Paffenbarger GC. Significance to health of mercury used in dental practice: a review. *J Am Dent Assoc* 1971;82:1401-7.
4. Rao GS, Hefferren JJ. Toxicity of mercury. In: Smith DC, Williams DF, eds. *Biocompatibility of dental materials*. Vol. III. Boca Raton: CRC Press, 1982;19-40.
5. Brune D. Corrosion of amalgams. *Scand J Dent Res* 1981;89:506-14.
6. Dérand T, Johansson B. Corrosion of non- γ_2 -amalgams. *Scand J Dent Res* 1983;91:55-60.
7. Frykholm KO. On mercury from dental amalgam. Its toxic and allergic effects and some comments on occupational hygiene. *Acta Odontol Scand* 1957;15(suppl 22).
8. Nilner K, Glantz P-O. The prevalence of copper-, silver-, tin-, mercury-, and zinc ions in human saliva. *Swed Dent J* 1982;6:71-7.
9. Möller B. Reactions of human dental pulp to silver amalgam restorations. Mercury determination in the pulp by flameless atomic absorption spectrophotometry. *Swed Dent J* 1978;2:93-7.
10. Svare CW, Peterson LC, Reinhardt JW, Boyer DB, Frank CW, Gay DD, Cox RD. The effect of dental amalgams on mercury levels in expired air. *J Dent Res* 1981;60:1668-71.
11. Hansson M. Oral galvanism—ett neurotoxiskt fenomen? *Lakartidningen* 1981;78:4240-1.
12. Baruah JK, Rasool CG, Bradley WG, Munsat TL. Retrograde axonal transport of lead in rat sciatic nerve. *Neurology* 1981;31:612-6.
13. Mottet NK, Body RL. Mercury burden of human autopsy organs and tissues. *Arch Environ Health* 1974;29:18-24.
14. Joselow MM, Goldwater LJ, Weinberg SB. Absorption and excretion of mercury in man. *Arch Environ Health* 1967;15:64-6.
15. Stein PC, Campbell EE, Moss WD, Trujillo P. Mercury in man. *Arch Environ Health* 1974;29:25-7.
16. Hoover AW, Goldwater LJ. Absorption and excretion of mercury in man. *Arch Environ Health* 1966;12:506-8.
17. Guccione P, Frank CW, Svare CW, Karlsson U, Chan KC. Distribution of mercury in the rat after long-term exposure to vapors. *J Dent Res* 1975;54:1235.
18. von Schneider V. Untersuchungen zur Quecksilberabgabe aus Silberamalgam-Füllungen mit Hilfe flammenloser Atomabsorption. *Dtsch Zahnärztl Z* 1977;32:475-6.
19. Holland RI. Galvanic currents between gold and amalgam. *Scand J Dent Res* 1980;88:269-72.
20. Nilner K, Glantz P-O, Zöger B. On intraoral potential- and polarization-measurements of metallic restorations. *Acta Odontol Scand* 1982;40:275-81.
21. von Kröncke A, Ott K, Petschelt A, Schaller K-H, Szécsi M, Valentin H. Über die Quecksilberkonzentrationen in Blut und Urin von Personen mit und ohne Amalgamfüllungen. *Dtsch Zahnärztl Z* 1980;23:803-8.