RESEARCH INVALIDATES

RECENT REVIEW AND

OPINION ARTICLES

THE MERCURY/CHLORINE CONNECTION

by

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The April 1983 issue of the JADA contained the official ADA policy statement on the safety of dental amalgam. (1) The underlying scientific foundation of the ADA position on the safety of amalgam was based on the Ph.D. dissertation of K.O. Frykholm published in 1957 (2) and the urine mercury/symptom studies of mercury cell chlor-alkali workers published in 1976, 1978 and 1979. (3-5)
Dr. Frykholm's research protocols used for his dissertation experiments have been questioned by scientists all over the world and subsequent scientific studies have invalidated most of his dissertation conclusions. The urine mercury excretion levels of chlor-alkali workers remains however as a cornerstone of the ADA position that amalgam is safe.

To quote from the April 1983 ADA position paper: "Studies in humans have repeatedly demonstrated and documented that biological effects, particularly central nervous system effects, are not manifested by the most sensitive neurological measurements until the human urinary mercury level reaches 500 ug/l. This level is 170 times the average urinary mercury excretion (3ug/l) of the general public."(1)

This particular position on the diagnostic value of urine mercury measurements was restated in a December 1983 ADA News Release entitled "Dentists, staff members healthy, despite daily contact with mercury", and quoted here in its entirety. "Chicago -- Dentists and their assistants, people who are regularly exposed to pure mercury as part of their daily work, are a generally healthy group of individuals.

'If the mercury used in dentistry posed any real health threat, it stands to reason that the professionals who handle it each day would suffer adverse effects,' said Gordon Schrotenboer, Ph.D., Chief of the American Dental Association's Division of Scientific Affairs. 'The fact is, dental professionals are a generally healthy group of individuals who show no ill effects from their contact with mercury.'

In support of that statement, Dr. Schrotenboer cited results of the ADA's own mercury testing program, a service the Association offers its members and their office aides as an outgrowth of a health assessment program started in 1975.

The Mercury Testing Service has been in operation since January 1982. To date, 776 dentists and dental staff members have enrolled in the program, which employs urinalysis to measure levels of mercury in the human system.

As of November 1983, 2575 urine samples have been tested and an average urinary mercury level of 14.6 micrograms (one-millionth of a gram) per liter has been found. This figure is understandably higher than the level found in the general population (about three micrograms per liter) but well below the level at which neurological symptoms are normally first detected -- about 500 micrograms per liter of urine.

'The Mercury Testing Service has shown that dentists and their staff members have mercury levels well within the safety zone of acceptability,' said Dr. Schrotenboer. 'And the mercury levels we detect in dental patients are considerably lower than those found in patients. To put it bluntly, patients are in no danger.'" (Ed note: We believe the last paragraph was meant to compare dentists to patients)

If one examines the 1978 study, for example, by Langolf et al.(4) upon which the ADA position on urine mercury levels is based, some rather startling information is revealed:

The study involved 130 male subjects from three chloralkali plants. Of the 130, 79 were male workers exposed to mercury and 51 were controls working in other areas of the chloralkali plants. The average urine mercury levels for the exposed workers was 240 micrograms per liter and for the controls 30 micrograms per liter.

Of the 79 exposed workers, only 10 showed urinary mercury histories which exceeded 500 micrograms per liter. It was also from this group that the conclusion was drawn that the first subclinical effects may appear when urinary mercury exceeds 500 micrograms per liter.

Within the dental profession in the United States there are well over 500,000 dentists and staff members who annually provide dental care to millions of patients and it must be reassuring to all of these innocents that their safety and protection from mercury intoxication has been assured on the basis of urine mercury measurements of 10 mercury-cell chloralkali workers.
There is one major defect in the underlying scientific position of the ADA and the studies of the chloralkali workers upon which it is based. That defect is the total lack of consideration to the metabolic effect associated with inhaling from an atmosphere containing both chlorine and mercury. In the initial major study of 642 chloralkali workers done by Smith et al in 1970(6) only one aspect of the effect of chlorine on mercury, that of measurement, was discussed: "A gas phase reaction between chlorine and mercury was shown to occur by chemists from one of the participating plants and the reported reaction kinetics were confirmed by Roggenbaum. Although low concentrations of chlorine are capable of removing mercury vapor from the air, and hence can cause a vapor meter to give low results, the ambient levels of chlorine in most cell rooms were in general, sufficiently low that the effect appeared to be of minor consequence."

Unfortunately, the metabolic effect of chlorine on mercury vapor toxicity was not considered, although the scientific data indicating such a phenomenon occurred was published in 1968(7) two years before the 1970 Smith et al study.

In 1968 Viola and Cassano(7) published a paper entitled "The effect of chlorine on mercury vapor intoxication: Autoradiographic study". The authors investigated changes in the airborne concentrations of mercury induced by adding chlorine vapors to the atmosphere and studied the effect this had on the intake and distribution of mercury. Mice and rats were exposed to mercury vapors alone and to equal amounts of mercury vapors in the presence of chlorine. Concentrations of mercury in the air and in the bodies and organs of the animals were determined by three different techniques in three separate experiments. The study resulted in the revelation of vital information and conclusions:

1. The addition of chlorine vapors resulted in a decrease of the mercury vapor concentration in the atmosphere. This decrease occurred proportionally to the formation of a fine particulate precipitate in the form of the relatively insoluble mercurous chloride.

2. A strikingly higher toxicity after exposure to an atmosphere containing only mercury vapors as compared to one with both mercury and chlorine vapors was found. This observation was supported by the finding that mice exposed to mercury vapors in the presence of chlorine absorbed 40% less mercury than the mice exposed to mercury vapors alone.

3. The animals exhibited markedly different intoxication profiles. Animals exposed to only mercury vapors exhibited severe neurological symptoms, while those exposed to mercury and chlorine vapors demonstrated mild gastroenteric disorders. The different type of poisoning shown by animals exposed only to mercury vapors was clearly explained by the much higher levels of mercury found in the brain, which were ten times higher that in the dual exposed animals. In contrast, kidneys of the animals exposed to both chlorine and mercury vapor showed almost a double concentration of mercury.

4. The results not only demonstrated variations in the mercury intake but also a clear-cut difference in the distribution patterns. The findings were in agreement with data previously reported by Berlin et al. (1966) and by Cassano et al. (1966) showing a selective and very high accumulation of mercury in the brain and heart muscle after exposure to mercury vapor alone. In contrast, very low levels of mercury were observed in the same organs of animals exposed to both vapors. "The reason for this appears to be the transformation of mercury vapors into mercurous chloride."(7) This would also appear to be the reason for the doubling of the concentration of mercury found in the kidneys of the animals exposed to both chlorine and mercury vapor.

Why is this information so important? The answer to that question may be found by examining the documents of organizations establishing standards for exposure to mercury vapor. In the United States, the most widely referenced mercury vapor exposure standard is that of the National Institute of Occupational Safety and Health (NIOSH), whose recommended Threshold Limit Value (TLV) is 50 mcg Hg/cubic meter.
as a Time Weighted Average (TWA).(8) This standard is based on the 1970 Smith et al study evaluating the appearance of clinically observable signs of neurological damage (tremor) in occupationally exposed workers in chlor-alkali plants.

As stated earlier, the study by Smith and associates, which was concerned only with the effects of chlorine on the measurement of mercury and not on any metabolic effect it might precipitate, has mistakenly been utilized as the basis for establishing standards of exposure to mercury vapor alone. The Viola group clearly showed the differences in exposure, intake, distribution, toxic effects, and symptomatology resulting from mercury vapor exposure with and without the presence of atmospheric chlorine vapors. It is therefore obvious and indisputable that these standards are applicable only to mercury-exposed workers in chlor-alkali plants, and cannot be considered valid for exposures to mercury vapors alone.

The same can be said for guidelines for the levels of mercury in blood and urine. Viola and associates found that the two types of exposure resulted in differences in the intake and distribution of mercury vapor. This would obviously influence the blood and urine mercury levels relative to the appearance and profile of observed symptomatology.

An examination of the position paper of the American Dental Association on the safety of dental amalgam (1) reveals the significance of this information. The ADA Joint Councils based their position on three studies correlating the levels of mercury found in the urine to the appearance of clinically observable symptoms in workers in chlor-alkali plants. These three studies were Miller et al. (1976), Langolf et al. (1978), and Langolf et al. (1979). (3-5)

A recent review of the dental amalgam controversy was published in the Journal of the American Dental Association. (9) The authors utilized 134 references, but were apparently unaware of the vital research conducted by Viola and Cassano. Their conclusions were based on the comparison of exposure to mercury vapor from dental amalgam and the associated elevations of mercury in blood and urine to the appearance of symptoms related to those parameters in workers in chlor-alkali plants (Table 2. Page 873. Reference 9).

The obvious conclusion resulting from the evidence provided by Viola and Cassano’s work is that use of the existing standards for exposure to mercury vapor are invalid because they are all based on worker exposure in chloralkali plants. Standards for exposure to pure elemental mercury vapor must be established through validation by proper scientific experiment. The use of erroneous data to politically validate the safety of dental amalgam should not be tolerated any longer. Certainly, the use of all-male standards cannot be tolerated when there is scientific evidence showing a difference in the metabolic effect and clearance of mercury in women and knowing that women comprise the major portion of the total dental personnel pool. There is also the question of what is the real significance of urine mercury values? We feel Dr. T.W. Clarkson makes the point succinctly: "Urinary excretion of mercury is used widely in monitoring workers exposed to mercury vapor (see USEPA, 1984). However, the relationship between urinary excretion and absorbed dose is not well understood; urinary excretion may be directly related to the kidney burden of mercury unless renal damage has occurred." (10) This point was also made by Lamm & Pratt in their 1985 study when they discovered a clear, negative and significant correlation between time on the job and the level of mercury in the urine. These researchers found that the longer a worker was on the job, the less mercury is excreted into his urine. (11)

There is an ever increasing body of literature detailing the release of mercury from amalgam dental fillings and showing positive correlations to the numbers and surfaces of amalgam fillings to accumulations of mercury in the brain and kidneys. There are also recent studies showing a direct correlation between the number of amalgam surfaces and urine mercury levels and also blood mercury levels. Recognized world authorities on mercury toxicity have recently stated that "The release of mercury from dental amalgams
makes the predominant contribution to human exposure to inorganic mercury including mercury vapor in the general population."(12) and "It is concluded that the cause of the association between amalgam load and accumulation of mercury in tissues is the release of mercury vapor from amalgam fillings."(13)

The significance of the information contained in the preceding paragraph has not as yet registered on the scientific community. Think about it for a moment. The phrase "NO KNOWN EXPOSURE TO MERCURY" is reflected in every scientific paper establishing "NORMAL" mercury blood and urine levels. However, most of the scientific data collected reflects population samples from industrialized nations where dental services are readily available. Consequently it would appear reasonable to also conclude that at least 75% of individuals sampled had from one to 50 amalgam surfaces in their teeth. Therefore, if the release of mercury from dental amalgam makes the predominant contribution to human exposure, what then are the true NORMAL VALUES for mercury in the biological fluids of humans WITH NO KNOWN EXPOSURE TO MERCURY?

This is only the tip of the iceberg. Consider the following paragraph quoted from a major toxicology text and reference book: "With chronic exposure to mercury vapor the major effects are on the central nervous system (Friberg & Vostal, 1972). Early signs are nonspecific and have been termed the 'asthenic-vegetative syndrome' or 'micromercurialism.' Identification of the syndrome requires neuroasthenic symptoms and three or more of the following clinical findings: tremor, enlargement of the thyroid, increased uptake of radiiodine in the thyroid, labile pulse, tachycardia, dermographism, gingivitis, hematologic changes, or increased excretion of mercury in urine. With increasing exposure the symptoms become more characteristic beginning with intentional tremors of muscles that perform fine-motor functions (highly innervated), such as fingers, eyelids, and lips, and may progress to generalized trembling of the entire body and violent chronic spasms of the extremities. This is accompanied by changes in personality and behavior, with loss of memory, increased excitability (erethism), severe depression, and even delirium and hallucination. Another characteristic feature of mercury toxicity is severe salivation and gingivitis."(14)

From a purely medical and toxicological viewpoint the major diagnostic criteria reflected above relates primarily to signs and symptoms and not solely to urine mercury measurements. The first criteria for diagnoses requires neuroasthenic symptoms. Without a medical dictionary most of us would not know what neuroasthenic symptoms are. Dorland's defines it as: "a neurosis marked by chronic abnormal fatigability (sometimes exhaustion), lack of energy, feelings of inadequacy, moderate depression, inability to concentrate, loss of appetite, insomnia, etc."(15) What is so fascinating about the definition of neuroasthenia is that you could easily substitute the current medical phenomenon identified as Chronic Fatigue Syndrome or CFS; a debilitating condition that appears almost pandemic.

Based on the information presented here it would seem that all of the government agencies, NIOSH, NIDR, OSHA, FDA and those non-government agencies such as the ADA should make it their highest priority to fund research projects that will scientifically establish standards for exposure to elemental mercury vapor from dental amalgam fillings, and determine what relationship exists between the symptomatology of chronic mercury toxicity and this source of mercury vapor exposure.

REFERENCES
Nylander M., Friberg L., and Lind B. Mercury concentrations in the human brain and kidneys in relation to exposure from dental amalgam fillings.

ABSTRACT: Samples from the central nervous system (occipital lobe cortex, cerebellar cortex and ganglia semilunare) and kidney cortex were collected from autopsies and analyzed for total mercury content using neutron activation analyses. Results from 34 individuals showed a statistically significant regression between the number of tooth surfaces containing amalgam and concentration of mercury in the occipital lobe cortex (mean 10.9, range 2.4-28.7 ng Hg/g wet weight). The regression equation \( y = 7.2 + 0.24x \) has a 95% confidence interval for the regression coefficient of 0.11-0.37. In 9 cases with suspected alcohol abuse, mercury levels in the occipital lobe were, in most cases, somewhat lower than expected based on the regression line. The observations may be explained by an inhibition of oxidation of mercury vapor. The regression between amalgams and mercury levels remained after exclusion of these cases. The kidney cortex from 7 amalgam carriers (mean 433, range 48-810 ng Hg/g wet weight) showed on average
a significantly higher mercury level than those of 5 amalgam-free individuals (mean 49, range 21-105 ng Hg/g wet weight). In 6 cases analysis of both inorganic and total mercury was carried out. A high proportion (mean 77% SD 17%) of inorganic mercury was found. It is concluded that the cause of the association between amalgam load and accumulation of mercury in tissues is the release of mercury vapor from amalgam fillings.

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Horsted-Bindslev, P. Rungeby J. and Danscher G. Mercury release from silver amalgam implants in monkey bone. Toxicology Unit, Royal Dental College, Aarhus, Denmark; Inst. of Anat. B, Neurobiology, Univ of Aarhus, Denmark.

ABSTRACT: Silver amalgam is used for various dental restorative purposes. Mercury originating from amalgam fillings has been demonstrated by autometallography in a variety of organs on monkeys (first meeting of ISTERM, abstract p. 101, 1986). Silver amalgam is also used for reverse fillings after apicoectomy. The aim of the present investigation was to study the possible distribution of mercury from silver amalgam implanted in bone. Three adult vervet monkeys received maxillary implants of 0.1 - 0.3 g silver amalgam each. Three monkeys without implants served as controls. After one year, the monkeys were anesthetized and killed by transectional perfusion with buffered gluteraldehyde. Tissue from various organs were removed and processed for light and electron microscopic autometallography (J. Histochem. Cytochem. vol 33, 219-28, 1985; Histochem J. vol. 18, 109-14, 1986) in order to demonstrate mercury. Until now we have examined spinal ganglia, liver, kidney and pituitary glands. Deposits of mercury were demonstrated in all of these organs except the liver. Tissues from the control animals were devoid of mercury. In general the findings were in accordance with our previous observations on mercury release from silver amalgam fillings. The amount of mercury in the organs was less that the amount found after amalgam fillings. It was concluded that bone implants produce mercury accumulations in various organs.

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ABSTRACT: The nervous system is the principal target for a number of metals. Inorganic compounds of aluminum, arsenic, lead, lithium, manganese, mercury, and thallium are well known for their neurological and behavioral effects in humans. The alkyl derivatives of certain metals--lead, mercury and tin--are specially neurotoxic. Concern over human exposure and in some cases, outbreaks of poisoning, have stimulated research into the toxic action of these metals. A number of interesting hypotheses have been proposed for the mechanism of lead toxicity on the nervous system. Lead is known to be a potent inhibitor of heme synthesis. A reduction in heme-containing enzymes could compromise energy metabolism. Lead may affect brain function by interference with neurotransmitters such as gamma-aminobutyric acid. There is mounting evidence that lead interferes with membrane transport and binding of calcium ions. Methylmercury produces focal damage to specific areas in the adult brain. One hypothesis proposes that certain cells are susceptible because they cannot repair the initial damage to the protein synthesis machinery. The developing nervous system is especially susceptible to damage by methylmercury. It has been discovered that microtubules are destroyed by this form of mercury and this effect may explain the inhibition of cell division and cell migration, processes that occur only in the developmental stages. These and other hypotheses will stimulate considerable experimental challenges in the future.

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ABSTRACT: Lichen planus is a common disorder of unknown etiology. It has been proposed that in some cases it represents a form of allergic reaction to the metals contained in dental amalgam, particularly
mercury. Twenty-nine consecutive dentate patients who had lichen planus of the oral mucosa were patch-tested to the range of metals contained in dental amalgam. Ten out of 29 (34%) showed an allergic reaction to mercury and all of these patients had amalgams greater than 5 years old. The amalgams had corroded, resulting in continued release of mercury ions. Six patients had their amalgams replaced with composite or glass ionomer materials resulting in resolution of ulcerated lesions. In a follow-up of 3-24 months, one patient had a recurrence of ulcerated areas and another, despite resolution of the oral lesions had persistent discomfort.

[Bio-Probe Comment: The two cases of recurrence and discomfort could possibly be a continued reaction to mercury from other sources in a sensitized individual, or mercury amalgam cores under crowns which was not disclosed in the article.]


ABSTRACT: It is common practice for lighthouses with large Fresnel lenses to use mercury baths as a low-friction rotation mechanism. Some recent acute mercury poisonings and incidents of abnormal behavior in lighthouse keepers have drawn attention to the potential for chronic mercury poisoning in these workplaces. This study evaluated the distribution of mercury in a lighthouse on the Canadian west coast, and the exposure of its keepers and their spouses under two weather conditions. The urine mercury levels found in the lighthouse personnel were all less than would be expected in an occupationally exposed group (less than 4 micrograms/24 hr urine). Air concentrations in the lighthouse ranged from 4.4 to 26.3 micrograms/m³. Swabbing showed considerable accumulation of mercury on surfaces in the area of the light rotation mechanism, as well as transport throughout the lighthouse. The mercury levels in this lighthouse appeared to be under control through effective convective ventilation and employee awareness. The study signals potential problems where precautions have not been taken, especially in situations where the keepers and their families live in the lighthouse.

[Bio-Probe Comment: Hopefully the managers of the ADA mercury testing program for dental personnel are aware of this research and its implications.]


ABSTRACT: It has been previously shown that autoreactive T cells appear during mercury-induced autoimmunity in Brown-Norway (BN) rats. In the present work, it is shown that: 1) T cells and T helper cells from HgCl₂-injected BN rats are able to actively transfer autoimmunity in normal BN rats; the disease transferred is exacerbated when recipients are treated with the antisuppressor/cytotoxic T cell monoclonal antibody (OX8); 2) normal T cells preincubated with HgCl₂ are also able to transfer the disease in OX8-treated but not in T cell-depleted rats; and 3) T cells from HgCl₂-injected BN rats also transferred the disease in both normal and T cell depleted rats. It is concluded that: 1) autoreactive T cells, and presumably anti-Ia T cells are involved in the pathogenesis of mercury-induced autoimmunity; 2) these autoreactive T cells induce suppressor/cytotoxic T cells to proliferate in normal syngeneic recipients; the fact that this T cell subset did not proliferate in HgCl₂-injected BN rats suggests that HgCl₂ also affects T suppressor cells; and 3) mercury-induced autoimmunity could result from the additive effect of the emergence of autoreactive T cells and of a defect at the T suppressor level.

ABSTRACT: Several methods of silver staining have been employed to localize mercury in tissue, under the assumption that the techniques represent total Hg, but recent reports have suggested that these stains are specific for a limited fraction of the Hg present in some samples. Magos et al. (1985. Arch Toxic. 57:260-267) hypothesized that the stains actually vary with inorganic mercury content. The purpose of the present study was to compare localization by radiolabeling to localization by silver stain, the photoemulsion histological technique, in tissues prepared to contain a range of levels of total Hg and a range of levels of inorganic Hg. Mice dosed with 8 mg Hg/kg as MeHg were killed 24 hrs, 1 week, or 2 weeks after exposure, to allow a decrease in total Hg and an increase in the proportion of demethylated Hg over time. Mice dosed with 4 mg Hg/kg as HgCl₂ provided samples in which all the Hg present was in the inorganic form. Atomic absorption of kidneys of mice dosed with MeHg showed that total Hg fell from 55 micrograms/g to 39 to 25 over 2 weeks, while the inorganic fraction climbed from about 2 to 27 to 35%. Grain counts from autoradiographs of 203Hg-labeled sections correlated with total Hg content at +0.88, but silver staining was correlated with inorganic Hg content, appearing only at late termination times in MeHg-exposed animals, but soon after dosing in mice exposed to inorganic Hg. The photoemulsion histochemical technique revealed a substance strictly localized in the proximal tubules, while autoradiographs and grain counts showed total Hg to be present throughout the kidney tissue. These results support the contention that silver stains are selective for inorganic Hg and suggest that the distribution of inorganic Hg, whether introduced experimentally or by gradual demethylation, is different from the distribution of MeHg. If subsequent studies support the association of silver stains with inorganic Hg, it should be possible to localize Hg in histologic sections, distinguishing between organic and inorganic forms which differ in toxicity.

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ABSTRACT: Concentration of metallic mercury in the arterial blood was higher in acatalasemic mice after exposure to 3.45 mg/m³ for 10 minutes in comparison with normal mice, whereas concentration of mercuric ion was lower in acatalasemic mice than in normal mice. (Ed note: Catalase is an enzyme found in all cells that catalyzes the decomposition of hydrogen peroxide and acatalasemia is a deficiency of catalase in the blood.) Thus, the ratio of metallic mercury to total mercury in the arterial blood of acatalasemic mice was 5.86 3.61% which was statistically significantly higher than the value (1.36 0.65%) of corresponding normal mice. Data indicate that the concentration of metallic mercury in the arterial blood of acatalasemic mice was higher than that of normal mice and that metallic mercury soluble in lipids is likely transferred to the brain and liver from the blood. Conclusively, metallic mercury in the arterial blood is the biologically active form for transferring mercury from blood to organs.

[Bio-Probe Comment: Eggleston et al. have recently demonstrated the difference in the analytical determination of mercury tissue levels between Neutron Activation Analysis and Atomic Absorption Spectrophotometry. (See Bio-Probe Newsletter. Vol 4, No. 5, Nov 1987) The two studies above present further evidence of the complexity of mercury in the body and the validity of selective research. Moreover, it should be re-emphasized that methylmercury and elemental mercury vapor are highly toxic because of chemical properties which facilitate their entry into and passage through the body. Actual pathology occurs only after their conversion to ionic inorganic mercury at the action site. Authors of review papers attempting to establish or influence professional practices that have a bearing on the public health should be held responsible for knowledge of this documented research.]

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ABSTRACT: A series of cytologic imprints obtained from periapical granulomas were studied with the conventional light microscope, the scanning electron microscope, and the electron microprobe to analyze the chemical composition of several black deposits that were randomly observed near or in the cytoplasm of macrophages and multinucleated giant cells. These granulomas had been removed from endodontically treated human teeth in which the root canals had been obturated with silver cones and Grossman’s sealer 3 to 5 years previously. To investigate whether corrosion had occurred on the silver cones, the cones were also examined with the scanning electron microscope and the electron microprobe. Our observations revealed that all the examined silver cones showed different degrees of corrosion on their surfaces, whereas different concentrations of silver, sulfur, and chlorine were detected at the same sites in the cytologic imprints. However, it is impossible to determine from this study whether the presence of corrosive by-products in the periapical tissues is responsible for the development of a pathologic reaction at these sites.

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ABSTRACT: With the results of 2 independent comparative clinical studies, both with a follow-up of 5 years, the survival and the causes of failure of amalgam restorations was compared with those of composite restorations.

For the first study 720 class 1 and 2 restorations were made in 106 adult patients by 3 operators. As amalgams 2 conventional and 2 high copper alloys were used. For the second study 175 class 1 and 2 restorations were made in 42 patients by 3 operators (2 of them participated also in the first study). As composite material a microfilled, a strontium glass filled and a quartz filled (conventional) composite was used. Encountered failures during the yearly examination of the restorations, that is when a replacement was necessary, were divided into 3 causative classes: 1 = related to the restoration. 2 = related to the restorative process and 3 = related to external factors.

At the 5 year recall 14% of the amalgam and 10% of the composite restorations was lost due to patient drop out. Of the remaining restorations 16% of the amalgam and 11% of the composite restorations had failed. The leading cause of failure in the amalgam and composite study is bulk fracture (11 resp. 7%). 0.2% of the amalgam restorations and 0.6% of the composite restorations had failed because of recurrent caries. For all other reasons 5.5% of the amalgams and 3.2% of the composites failed.

In the amalgam study the material has a significant influence on class 1 failures. However, in the composite study such an influence does not exist. The 5-year survival of amalgam restorations and composite restorations is 84 and 89% respectively.

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ABSTRACT: The usage of base metal (primarily Ni-Cr-Be) dental casting alloys for fabrication of fixed prosthetic devices is common in the United States. This has resulted in increasing numbers of patients presenting with Ni allergy to these prostheses. Since the usage of these base metal alloys is not as common in Germany, the purpose of this presentation will be to acquaint members of the IADR-CED with various
aspects of Ni allergy. This will be done through a summary of a five year investigation into the incidence of this problem supplemented with clinical photographs.

Our findings have enabled us to develop a profile of the "typical" Ni allergy patient, which is as follows:

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>INCIDENCE:</th>
<th>RANGE:</th>
</tr>
</thead>
<tbody>
<tr>
<td>female</td>
<td>&gt; 90%</td>
<td>-</td>
</tr>
<tr>
<td>pierced ears</td>
<td>&gt;90%</td>
<td>-</td>
</tr>
<tr>
<td>Caucasian race</td>
<td>= 80%</td>
<td>Asian 10%, Black 10%</td>
</tr>
<tr>
<td>25 - 40 years old</td>
<td>= 65%</td>
<td>18-58</td>
</tr>
<tr>
<td>rashes to jewelry</td>
<td>&gt;60%</td>
<td>-</td>
</tr>
<tr>
<td>prosthesis in place</td>
<td>= 60%</td>
<td>6 weeks to 4 years</td>
</tr>
<tr>
<td>one year or longer</td>
<td></td>
<td>70% no pain</td>
</tr>
<tr>
<td>Painful type of</td>
<td></td>
<td>10% moderate to severe</td>
</tr>
<tr>
<td>allergic reaction</td>
<td>= 20%</td>
<td></td>
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</tbody>
</table>

Clinically, the "typical" appearance is best described as a bright red, well-circumscribed line approximating the free gingiva. Often the condition is mistaken for a non-resolving mild periodontal disease. Due to its appearance, Ni allergy can mimic poor oral hygiene, rough prosthetic margin, or cement remaining in the sulcus. Widespread stomatitis type reactions, while possible, are somewhat rare. Since the condition is usually not painful, the dental professional is often the first person aware of the condition.

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ABSTRACT: The release of corrosion products during long-term immersion in vitro of dental alloys, with particular reference to dissimilar alloys in contact has been studied. The effects of Hg$^{2+}$ and Cu$^{2+}$ in low concentrations on the guinea-pig ileum have also been studied. The following conclusions were drawn: In an aggressive solution the release of elements from amalgams could be continuous and subsurface corrosion could cause a considerable increase in the corrosion products released. The change in microstructure observed in cross-sections of the corroded specimens was related to the amounts of corrosion products released into the saline solution. In an aggressive solution the corrosion products could increase when amalgams, Co-Cr, and Ni-Cr alloys are in contact with gold alloys, a high-Cu amalgam is in contact with a conventional amalgam, a type III gold alloy is in contact with gold alloys for metallo-ceramic purposes. The high-Cu amalgams released more corrosion products into the saline solution than a conventional one. Greater quantities of corrosion products were released from amalgams at pH 4 than at pH 6. Hg$^{2+}$ and Cu$^{2+}$ both had diverse and dose-dependent effects on the guinea-pig ileum. In low concentrations, 10nM, both ions exerted effects, probably on the muscle cell membrane.

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ABSTRACT: Titanium's inert behavior might be counteracted by galvanic coupling to metal restorations. This work was made to study such reactions in vitro.

A Ti rod was placed in 1% NaCl in a corrosion cell, simulating a dental implant in the jaw, and an amalgam rod was placed in 0.1% NaCl in another cell, simulating a dental restoration in saliva. The cells were fitted with pH electrodes, connected by an agar bridge, and aerated. The rods were connected by 10 ohms for current monitoring.

From 13 uA/cm$^2$ at the start, the current fell steeply to a minimum after 5 hours and then rose more slowly to a maximum of 20-30 uA/cm$^2$ after 2-6 days, finally leveling off at 15-20 uA/cm$^2$. The Ti was
always positive. From 6 initially, pH steadily rose to 9-10 in the Ti cell and fell to 2-3 in the amalgam cell, all within 2.5 days.

The reactions are driven by the anodic corrosion of amalgam (tin), summarized as $\text{Sn} + 4\text{OH}^- \rightarrow \text{SnO}_2(\text{s}) + 2\text{H}_2\text{O} + 4\text{e}^-$, accounting for the pH drop and producing electrons for the cathode reaction on the Ti: $4\text{e}^- + \text{O}_2 + 2\text{H}_2\text{O} \rightarrow 4\text{OH}^-$, which accounts for the pH rise. The current fall after start can be ascribed to the build-up of a resistive Ti oxide layer. The electric charge can be correlated to the weight changes due to the corrosion.

In conclusion, a corrosion current and, in particular, a disconcerting pH rise could be demonstrated around a specimen of titanium - normally considered inert in a biological context - by coupling it galvanically with a metallic dental restoration material.

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ABSTRACT: Dentists are constantly faced with the task of eliminating cross-infection and dental burs are one of the weak links in this chain of sterility. Previous studies have shown that most methods are time consuming and result in damage to burs. The purpose of this study was to determine the most efficient method of sterilization of burs.

Rosehead latch-grip burs were contaminated by the removal of debris from carious lesions. Adherent debris was removed by either brushing with a bur brush or ultrasonication in ultrasonic cleaning solution for 2, 3, 4 and 5 minutes. Ultrasonic cleaning, the most efficient method, was combined with disinfectants and tested. Burs were placed in a solution of either Borex Lugol’s iodine or 70% alcohol and ultrasonicated for 2, 3, 4 and 5 minutes. The burs were removed using aseptic techniques and cultured in thioglycollate broth for 7 days at 37°C. Each method was tested in triplicate. The bur brush did not remove all debris. Ultrasonic cleaning removed all visible debris, but not viable organisms. After 2 minutes, burs unultrasonicated with iodine and Borex were sterile. All the disinfectants tested, rendered the burs sterile after 3 minutes. The most efficient method of sterilization of dental burs was ultrasonication with a disinfectant for 3 minutes.

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FORUM

- Anyone needing a Jerome #411 mercury vapor analyzer, please contact Dr. Bruce Andrews at 312-940-0296. Dr. Andrews is asking $2500.00. If you are interested, make him an offer.

- International Academy of Oral Medicine and Toxicology will hold its annual meeting in Chicago, at the Oak Brook Hills Hotel & Conference Center September 16-18, 1988. Meeting chairperson is Marcia Basciano, D.D.S. (312) 629-6299.

- The National Center for Homeopathy presents a seminar on Dental Homeopathy July 29-31, 1988 at Endicott College, Beverly, Massachusetts. For information call (202) 223-6182.

- The 11th Annual National Dental Seminar in Homeopathy will be held in Chicago, October 21-23, 1988. There will be a basic introductory and advanced course running concurrently. For additional information write to: National Seminar in Homeopathy, P.O. Box 123, Marengo, IL 60152.

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