

# IX Fremdsprachige Beiträge

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## Fremdsprachige Beiträge

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## Englisch

## Mercury poisoning from amalgam. Major symptom: Headache

## Toxicology Section

Dentist are confronted with the symptoms of patients who have had amalgam fillings for many years. Since the mercury levels were only elevated to 5–40 µg/l in urine in the first few days after placement and then clearly dropped, the thought was that it could be a hypersensitive reaction.

However, a number of patients reported in the anamnesis very clearly that months to years after the placement of several amalgam fillings, their sufferings began. After placement of more fillings or after less than 10 years the symptoms clearly got worse. This happened not only to patients with additional gold- or metal prosthetic work; because of electrochemical reactions one must expect increased metal absorption together with unpleasant local reactions in this case. Even after the removal of the amalgam fillings, the problems reduced not until several years. In this paper we will report on 200 patients who had 1–22 fillings for a mean of 8 years. Clear problems started after about 6 years.

## Amalgam

Amalgams are produced by mixing about equal amounts of alloy powder and mercury.

## Composition of the alloy powder:

Ag:	min	40%
Sn:	max	32%
Cu:	max	30%
Hg:	max	3%
Zn:	max	2%

(Values in % weight)

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The hardened amalgam mainly consists of the original phases  $\text{Ag}_3\text{Sn}$ ,  $\text{Cu}_3\text{Sn}$  and often  $\text{Cu/Ag}$  Eutecticum and the reaction phases of  $\text{Ag}_2\text{Hg}$ ,  $\text{Hg}_2$  and  $\text{Cu}_2\text{Sn}_2$  and little or none of the  $\text{Sn-Hg}$  phase.

Through placement and as well as removal of amalgam fillings there will be increased release and organ accumulation of all metals and consequently a chronic poisoning.

**Main Symptoms:**

Headaches (migraine-like) the most common (85%)

Sleep disturbances

Memory disturbances

Nervousness

Tremor

Depression

Gastritis

Colitis

Infection prone

Allergy

Especially hard affected will be nervous patients since they react especially strongly on nerve poisons. The same is valid for patients with vasoactive headaches.

In contrast to acute mercury poisoning there will be no increased nerve conduction and no kidney insufficiency in the first 10 years. There are no studies on increased risk of cancer, embryotoxic and teratogenic effects.

For differential diagnostic purposes, other sources of mercury poisoning must be investigated, e.g. extreme consumption of sea-food (tuna fish, crab, mussels).

**Effects**

Absorbed mercury will mainly be bound to sulphhydryl groups of proteins. The uptake in brain takes place more slowly than in other tissues. However, in brain it reaches the highest values and has a half-life of 18 years in contrast to 70 days for other tissues. Only a minor fraction of absorbed mercury is excreted in the kidney and intestines. The rest goes into depots where the CNS is especially important for clinical aspects.

Even after removal of the amalgam fillings and without chelation, the symptoms of poisoning reduce very slowly. In treated cases migraines, for instance, completely stopped in about 4 months.

**Effects of DMPS**

DMPS, in contrast to all earlier chelators, reduces the accumulated mercury in the brain, also when the starting blood- and urine values are with normal limits (normal up to  $4 \mu\text{g/l}$  in urine). The effect is only extracellular. Therefore, detoxification is only possible by diffusion.

It is a definite proof of the accumulation of mercury in organs and brain, if the measured urine value increases to more than  $50 \mu\text{g/l}$  after giving DMPS (1 ampule 250 mg i.v.). The elimination can be carried out successively, e.g. every 4th week. The therapy also can be necessary several years after amalgam removal. After repeated DMPS administration, the substitution with zinc aspartate is necessary.

**Procedure**

1. Spontaneous urine for Hg-measurement.
2.  $4 \text{ mg/kg}$  DMPS i.v. (Dimaval) – Children  $10 \text{ mg/kg}$  capsule orally about 20 ml of next urination.

## 3. Measurement of Hg, Cu and Sn.

## 4. Repeating the procedure:

- Every fourth week if Hg 100 µg/l.
- Every third month if Hg 50 µg/l.
- Otherwise after 6 months.
- If Hg 1000 µg/l one capsule every week.

## Toxicity

More dangerous than narrow, deep fillings are occlusal fillings with a large surface area. After chelation of one large amalgam filling we can find levels of 40 µg/l for each year of presence. Our experience is that values over 50 µg/l give neurological disturbances like headaches and neurasthenia.

More than 10 fillings (→ up to 2565 µg/l after DMP5) generally lead to tremendous problems.

## Environmental toxicology

At least 13% of the mercury in sewage originates from dental clinics (Berlin). Every year 100 dental clinics in Hamburg dump 0.4 tons of mercury into the water.

Bio-probe comment: with the ever increasing contamination of rivers and lakes in the U.S. recipients of this news letter should make every effort to bring the data on Berlin and Hamburg to the attention of investigative reporters and responsible state environmental officials.

## Summary

In contrast to not decisive determinations in blood or urine which give no information, the evaluation of values after giving DMP5 once, gives an estimate of accumulated mercury depending on number and age of amalgam fillings. Values up to 2565 µg/l were found in urine. With urine values over 50 µg/l, typically neurological problems might occur, the most common ones are migraine-like headaches. The symptoms will disappear after repeated administration of chelator. The insurance system will pay for the exchange of gold inlays after mobilization test and character-like poisoning symptoms. Since mercury is toxic during handling and occupational processes, avoiding exposure is preferred.

## Conclusion

Amalgam fillings should not be placed today. When a number of old fillings are present together with headaches and nervous symptoms, a DMP5 chelation should be carried out again after amalgam removal. If needed, additional therapy should be used.

## Amalgam fillings – malpractice

### Case reports.

1. A 9 year old girl had 5 amalgam fillings one year. Within one year and a fall on her head severe encephalopathy developed. The EEG became flat. She was extremely agitated, had rhythmic seizures and lost contact with her surroundings. She had to be fed parenterally. Hg in spontaneous urine 18,5 µg/l. After 3,5 mg/kg DMP5 orally, urine Hg 213,5 µg/l.

In her hair Hg-levels were elevated 6 times. During a seven month treatment with DMP5 (100 mg/week

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orally) the poisoning symptoms slowly reversed. Substantial improvement did not occur until the amalgam fillings had been removed.

2. A 30 year old female patient had constant migraine attacks for several years and pain in the lower abdominal region. Since her dentist blamed the amalgam fillings they were exchanged for 11 new amalgam fillings. The problems became immediately worse. Hg in spontaneous urine was 11 µg/l. After 3 mg/kg i.v. DMPS it came to 2565 µg/l. Headache and abdominal pains disappeared rapidly after the chelation and reappeared in milder form after 6 weeks. After removal of the fillings and repeated chelation the patients symptoms disappeared.

3. A 33 year old female patient had 14 amalgam fillings for 25 years (partially renewed). Since the fillings have been 3 1/2 years old, she felt very tired, depressed and had constant vertigo, sick-feeling, headaches, concentration- and capability disturbances, paresthesia and increased infection sensitivity. She complained about bad smell or taste, abdominal pain, diarrhea, paroxysmal arrhythmias and tachycardia. She developed a candida bronchitis, asthma bronchiale and nickel allergy. Hg in spontaneous urine 7 µg/l. After 250 mg DMPS i.v.: Creatinine 1.36 g/l, Pb 121 µg/l, Cu 2493 µg/l, Hg 2794,3 µg/l (limit after chelation up to 50 µg/l according to our experience). After detoxification by chelation and removal of amalgam fillings, considerable improvement.

4. In a 64 year old patient with 21 amalgam fillings we found Hg in urine to be more than 5000 µg/l after zinc therapy. It normalized two years later after continuous zinc supply. Then the patient felt healthy. After an implantation in the lower jaw 8 years later there was a renewed excretion of mercury from depots in the jaw. Spontaneous level 20 µg/l. After 250 mg DMPS i.v. 22,579 µg/l. Titr. demonstrated the high levels of mercury in bone tissue.

## Own Observations

During studies of the question whether amalgam carriers with neurological problems always show a mercury dumping after chelation with DMPS we found up to now 800 patients:

- Patients who never have had amalgam fillings showed values to a maximum of 20 µg/l after 3 mg/kg DMPS i.v. and no special increase in chelated copper.
- Patients who regularly ate sea food, especially tuna fish and crab, had a maximum of 50 µg Hg/l urine 30 minutes after 3 mg/kg DMPS i.v.
- 98% of patients with amalgam fillings or recently removed fillings had over 50 µg/l Hg in urine after chelation and also a significantly elevated copper excretion with values over 500 µg/l Cu in urine. Rapidly after removal of fillings and chelation the symptoms reduces considerably.
- Patients with the most severe symptoms generally showed considerable zinc deficiency, elevated copper excretion and also cadmium and lead excretion. This load, in addition to exposure to wood preservative (pentachlorophenol, Lindane) to an enhancement of the neurological organ damage.
- The degree of poisoning symptoms is not only determined by the number of fillings but primarily also by the zinc status which is in the first line of detoxification of heavy metals. Copper is antagonistic to zinc and potentiate, as do other toxic substances, the neurological problems.
- Primary symptom of amalgam poisoning: Apathy, tiredness, headache, abdominal pain, muscle and joint problems, memory disturbances, depression, sleep disturbances, susceptibility to infection.
- A sudden onset of symptoms during an infection (zinc deficiency!) after a latency of years.
- Zinc supplementation enhances the excretion of extracellular mercury. However, not from depots.
- We have seldom diagnosed selenium deficiency.
- Many cases of colitis (ulcerative) and some with multiple sclerosis improved considerably after removal of the amalgam fillings and detoxification with DMPS.

- The type of amalgam does not seem to have any relation to the severity of symptoms.
- Because of the amalgam-induced mercury poisoning and the ensuing zinc-deficiency other poisonous heavy metals like lead and cadmium and also arsenic will be retained in the body to a higher degree.
- As long as the amalgam fillings remain in the mouth, the symptoms reduced only temporarily after chelation therapy. Final recovery was achieved only after amalgam removal.
- Mercury from amalgam can be differentiated from mercury from other sources by a fair degree of certainty by:
  1. Determination of other amalgam components in addition to Hg (Cu, Ag, Sn).
  2. The appearance of symptoms in connection with the placement of amalgam.
  3. The extremely high levels in depots which only occur after continuous poison release.
  4. The rapid improvement of the clinical picture and the T-helper reduction after amalgam removal.

Other cofactors for increased Hg-release from amalgam fillings:

Frequent hot drinks, acid food, fluorine-containing toothpastes, chewing gum use, bruxism, other metals and the already mentioned zinc deficiency.

In severe cases we also found a clear T-cell depression which disappeared after amalgam removal.

Up to 20 years after removal of amalgam and persistent symptoms, a depot can be found and treated.

After a number of tests, a practical procedure for a insurance-connected physician has been worked out.

1. Spontaneous urine I: measurement of zinc and possibly nickel (in addition to Hg).
2. Slow i.v. injection of 4 mg/kg of DMPS.
3. After 30 minutes, urine II for mercury and copper (+ lead if blood pressure is high and cadmium for osteoporosis).

Orally the chelator is unreliably absorbed. 10 mg/kg in one single dose after fasting.

The capsules have been available and allowed for 13 years as Dimaval. Our first case was treated successfully in 1976 (Arsenic poisoning).

Any physician should be able to use the treatment before and after the removal of amalgam and record the symptomatology. We send our urine samples to a respectable laboratory.

#### Evaluation:

Environmental poisonings must always be evaluated with consideration to interactions with other poisons like copper, lead, cadmium, wood preservatives, dioxin, zinc deficiency, length of exposure, underlying diseases like allergies etc.

After 3 mg/kg DMPS at more than 50 µg/l Hg there was always an improvement in neurological symptoms. This should therefore be considered a MAC-Value unless a too high copper excretion complicates the evaluation.

After high copper excretion and a re-chelation there will be still 50% of the Cu values after 6 weeks and therefore also a higher excretion of the other heavy metals. Chromium and nickel will be retained if the copper excretion is high. Cadmium is excreted in large amounts during zinc deficiency.

#### Legal Consequences:

1. Even if the observed cases were isolated cases (everything contradicts that) the severity of the observed side effects and the impossibility of preventing them, prohibits any further use of dental amalgam which contains mercury.
2. Gold as an insurance-paid alternative must be acknowledged for poisoned patients as well as for allergic patients.
3. Compensation for severe demonstrated cases must be possible (funds?).

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4. All dental materials must be tested for long-time effects according to the drug-laws (non-noble alloys, indium, gallium, vanadium, nickel, beryllium, synthetic materials, formaldehyde in root-fillings, etc.).

5. Dentists, family doctors, neurologists must as soon as possible be informed about the various poisoning symptoms and possible treatments.

## Open Scientific Questions:

- Immunodeficiency caused by amalgam are fungus infections, virus diseases, multiple sclerosis, ulcerative colitis, rheumatic problems, etc increased or induced?
- Mercury is teratogenic. Can these concentrations cause fetal damage?
- Mercury is mutagenic. To what degree at these concentrations?
- Mercury in bone under amalgam fillings in autopsy material?
- Irreversible damage (malformation, neoplasms, M.S.).

## Resume:

The prohibition demanded in the USA in 1840 for the use of Hg-amalgam for dental fillings was clearly well founded and was unfortunately in 1855 taken back because of industrial pressure since the scientific demonstration of depots was lacking. Amalgam fillings should immediately be considered malpractice.

## Demand to forbid Amalgam-Toothfillings – Review after one Year

## Cause

We made a demand to forbid amalgam toothfillings in march 1989 in 'Forum des Praktischen Arztes' because of severe cases of poisoning symptoms in some patients. A series of medical papers had refused the article before, but reprinted it later. It was taken up by public press in July.

After this, a time of quarreling started. Bavarian Dentist's Council told, that they had recently paid 1 mio German Marks each, for two expert opinions, which said amalgam to be harmless.

## Casuistic

Our demand was caused by the case of a female patient from Passau who had proved allergy to amalgam and suffered from yearlong migraine attacks, which have instantly ceased after removing the amalgam fillings. Health insurance only would pay, if poisoning was evident. We were surprised ourselves, when we still found and eliminated large organ depots of mercury after several months of mobilising with DMPS (We use it with excellent results in heavy metal poisoning since 1981).

There was a London scientist who suspected amalgam poisoning in himself and asked for DMPS-treatment. In fact his urin level of mercury was 650 microgram per liter.

In January 1989 we traced 5 recently made amalgam fillings in a nine year old girl as the only cause of her proved mercury poisoning, which had made her stay in coma for nearly half a year in a Munich children's hospital. DMPS woke her from the coma after 2 months of treatment, but she only recovered fully after removal of the fillings.

Volunteers with several amalgam toothfillings (members of the family, assistants) showed mercury levels of 1250 µg, 850 µg, 650 µg per liter in urine after mobilisation. Remobilisation brought the same high results until fillings were removed.



## Extreme levels

A patient from Vienna, who once had 21 teeth filled with amalgam excreted 22570 µg/l mercury in urine, a bavarian housewife 42000 µg mercury/l after mobilisation. Both suffered from severe symptoms of mercury poisoning, which nearly had made them commit suicide (pain attacks like in prolaps of discus vertebralis, receptively frightening fits of vertigo). Both patients were non fish eaters, and other causes of mercury intoxication could not be found. Their symptoms vanished slowly under repeated mobilisation.

## Observations

During this time following findings were made: Amalgam free patients usually had mercury levels after DMPS of about 5 µg/g Creatinin, passionate fish eaters among them up to 20 µg/g (maximum). Recent fillings seem to cause more mercury absorption, older ones more tin. If the mercury levels were high, there also was found a large amount of copper. Organic compounds of mercury and tin made 80%. Organic tin is about 100fold more toxic than mercury. Anorganic tin and mercury are transformed to organic compounds by oral bacterial flora. Symptoms on chronic poisoning of these two heavy metals are much alike. Copper increases tremor and often causes changes in liver enzymes.

## Symptoms

Leading symptoms in amalgam poisoning are: lethargy, headaches and/or abdominal pain, vertigo, impaired vision or hearing, tremor, pain in muscles and increased tendency to infections.

## Contraindication to amalgam fillings

Until now, official contraindications for mercury containing amalgam are not noted, though its toxic effects like carcinogenity, mutagenity and teratogenity are well proved. Generally it is to be postulated, that amalgam fillings should not be used in any affection of the nervous system, multiple sclerosis, M. Alzheimer, Myositis, sudden blindness or deafness, amyotrophic lateral sklerosis, AIDS, Colitis ulcerosa, anorexia, repeated diarrhea, M. Crohn, repeated sinusitis, bronchitis or other repeated infections, depressive moods, M. Parkinson, paraesthesies, atactic movements and other. We found good effects after mobilisation in such cases. In problematic cases, amalgam removing only should be started after mobilising, in order not to get too high levels after the procedure.

## Test with chewing gum

A difference between mercury and tin levels in saliva before and after 10 minutes chewing, can give a hint for the rubbing off from amalgam fillings. We saw levels up to 405 µg/l saliva of mercury and 82 µg tin. Levels in urine after mobilisation were in relation. They usually had about 10fold the saliva concentration for both heavy metals. This is a very simple information test, that can be done also by not medical personel. It is a faint hint for bad amalgam fillings or badly set ones. If this is too expensive, pre-chewing saliva may be left out to investigate.

## DMPS test

DMPS is produced by the Leningrad Chem. Pharmaceutic plant "October" as Unithiol® since 1957 (ampullas à 5 ml containing 500 mg DMPS). For 10 years they have no longer been producing tablets because of the uncertain resorption rates (up to 30%). In the FRG both application forms are produced under the name of "Dimaval®" by Heyl, Berlin. The ampulla form is licensed since 8/91. Chronic mercury intoxication is the main indication for Unithiol.

## Prohibition of amalgam

1000 publications on the danger of amalgam preceded prohibition of amalgam fillings in Soviet Russia in 1985. While in 1988 a forensic specialist in Markredwitz still voted, mercury poisoning was only sufficiently to be verified in carcass, nowadays, even in Germany, the 33 year old russian experiences come to knowledge.

## Threshold levels

Not only since Chernobyl did we have to learn, that high levels of a damaging substance often makes politicians and opportunistic experts lifting up limiting levels, instead of trying to diminish danger. So here. There is a number of dentists who now try to get threshold levels for mercury raised.

## 24 hours collected urine

It is well known that the effect of DMPS starts 17 minutes after injection, it decreases after 30 minutes and there is no more effect after 4 hours. In cases of acute mercury poisoning, DMPS injection has therefore to be repeated constantly after 3 hours. But still occupational physicians do stick to their method of "highly careless". Of course such urine levels will show much lower quantities of mercury (about 20fold less), being a solution with poisonfree urine. The method of occupational medicine is of course valuable in factory workers, where nobody knows, where the intoxication took place, and when excretion started. But in surveyed patients and the exact time of mobilisation. The average mercury level is interesting in exposed workers, but as a mean of absolute diagnosis, the easier way of dated investigation is of more importance to a clinical toxicologist.

## General practitioner

Measuring spontaneous levels and 30 minutes after mobilisation with DMPS is a method easy to cope with in general practicioning. Thousands of investigations by different colleagues proved it. The amount of patients suffering from amalgam damage (about 2 mio in FRG) makes general practitioners look for a practicable and quick testing method.

## Legal consequences

If chewing test shows toxic levels of tin and/or mercury, amalgam fillings should be taken in responsibility and take the risk of paying compensation, as correctly composed and set amalgam fillings do not cause relevant uptake of mercury – according to Dentist's Council. The exact amount of mercury and tin, a patient has to endure is not fixed though, even though amalgam is used since 150 years. Concerning the not yet licensed DMPS, we were confirmed by Prof. Spann (emer. Professor of Forensic Medicine in Munich), that it is at any time allowed to be given in acute danger of poisoning. In uncertain cases you should rather chose the unsufficient form of capsules.

## History

Rarely in medical fields, such old, nearly unchanged methods as amalgam fillings are used. Recently even the old copper amalgamates are discussed again. 150 years ago, amalgam was forbidden in the USA for the first time, because of intoxications with mercury vapor. 14 years before, it had been invented, and a lot of damages have been seen during these 14 years. After 15 more years it was allowed again under a number of reservations. In 1925 Prof. Stock, Berlin, desperately demanded a prohibition of amalgam. Suffering from severe mercury poisoning himself, which later caused his death. War, inflation and currency reform made forget these warnings. Hanson (Sweden) collected over 8000 publications about amalgam intoxi-

cations, we ourselves have seen 1000. The most impressing of which is a report on depression of T- and B-lymphocytes, followed consequently by immune deficiencies, which are reversible after removal of amalgam fillings, as well as are neurological alterations. Yet patients are extremely worried about other long year damages that often do not vanish again.

### View

After having pointed out a coincidence with thalidomide and deformations, the paediatric Dr. Lenz was regarded a boaster and outsider. During the following two and a half years, another 2 500 children were born crippled, meagerly paid off with 10 000 DM each.

How long will it take until poisoning by amalgam fillings will be banned?

## Tests in Relation with Amalgam

The metals in amalgam intensify each other in their effects. These are the most important ones:

**Tin** increasing weakness, apathy, neuralgia, pain sensitivity, paresis, undulating pain in the gastro intestinal tract, headache, hoarseness, cough, sensibility to chill and changing weather, paleness.

**Copper:** clonic cramps, gripes, impaired vision, difficulty in breathing, paraesthesia, tremor, weakness, anal cramps, constipation, grinding of teeth, allergy, liver damage.

**Silver:** anxiety, forgetfulness, blocking of thought processes, encephalomalacia, headache, vertigo, defectiveness to stress, mental weakness, impaired ligaments muscles and joints, chondrogenesis, vertebral pain, rheumatism.

**Mercury:** apathy, headache, gastro intestinal disorders, vertigo, tremor, forgetfulness, sleeping trouble, muscular asthenia, vertebral pain, allergy, nervousness, changing moods of apathy and irritability depressive mood, ataxia, paresis, paraesthesia, impaired vision and hearing, susceptibility to infections, anaemia, disorder of cardiac rhythm.

### Test with chewing gum

#### Indication:

to prove the amount of toxic metals from amalgam tooth fillings in normal saliva (saliva I), respectively what is abraded by chewing (saliva II) – in relation to acute toxic status in the body (urine I).

#### Realization:

saliva I: collect half a test tube (about 5 ml without foam)

saliva II: after 21 hours without chewing – intensive chewing of gum on the hilled teeth, collecting saliva in a second tube (5–10 minutes, same amount)

Send to laboratory: saliva I for Hg, Sn, Ag  
saliva II for Hg, Sn, Ag, pH  
urine I for Hg, Sn, Zn

**Valuation:** the amalgam fillings are of inferior quality or poisonous if saliva II (after chewing) contains much more than urine or saliva I (norm.:  $ul < salI < salII < 5 \mu g/l$ ). Severe toxic effects occur at differences over  $100 \mu g/l$  Hg.

### DMPS-test (Dimaval)

**Indication:** in cases of severe nerval or immune deficiencies the depots of heavy metals in the organs should be treated and at the same time measured in urine by giving an antidot. The test is harmless and it is paid

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for by health insurances if the symptoms indicate poisoning, because healing or amelioration of the chronic intoxication is to be expected. The test can be extended to prove sufferings from environmental poisons as lead and cadmium.

Before removal of fillings: AIDS, generalized allergy, amyotrophic lateral sclerosis, (partial) loss of vision or hearing, rhythmic disorders of the heart, melanoma, multiple sclerosis (also not typical forms), paraesthesia, high levels in chewing gum test ( $Hg > 100$ ,  $Sn > 30 \mu g/l$ ).

After removal of the fillings: Allergy, apathy, asthma, abdominal pain, colitis ulcerosa, eczema, gastritis, susceptibility to infections, ischialgia, cancer, headache, morb. Crohn, and muscular asthenia, neuritis, depression of T-cells, trifacial neuralgia, infertility.

**Realization:**

Urine I: after drinking a fair amount of water – Fill test tube with urine.

DMP5: Injection: the exactest way of uptake which shows best results in elimination rate (available T. 030/8176052).

Dosage: 3 mg/kg b.w. (about 1 ampulla Dimaval) intravenous capsules: (without prescription at the chemist's) to be taken on empty stomach – then further fasting for 2 hours. Dosage: Test: 10 mg/kg b.w. (has about the same results as 1 ampulla). Therapy: 3 mg/kg b.w. once every fortnight.

Urine II: With ampullas: after 30–45 minutes.

With capsules: after 2 hours – in test tube.

**Precautionary measures:**

1. Not in acute viral infections (which can be lengthened because of zinc elimination)
2. No alcohol (including beer) for 2 days (increases tiredness)
3. Only to be repeated in longer intervals (6–12 weeks with injection, 2 weeks with capsules)

Side effects: extremely seldom allergic skin reactions, which pass over without therapy. Hyperventilation tetany during the injection can be prevented by superficial breathing.

Send to laboratory: Urine I for: Zn, creatinin

Urine II for: Hg, Cu, Sn, (Pb, Cd), creatinin

Valuation: Hg (after 3 mg DMP5/kg b.w.) up to  $20 \mu g/g$  creatinin: load by food. Over  $50 \mu g/g$  creat.: chronic intoxication. Copper as a sign of poisoning with heavy metals varies between  $500$ – $4500 \mu g/g$  creatinin.

Therapy: After removal of amalgam fillings DMP5 should be given until symptoms vanish.

>  $100 \mu g Hg/g$  creat – every 3 months

>  $500 \mu g Hg/g$  creat – every 6 weeks

Removal of amalgam in cases of above named failure under precaution of rubberdam and not with high speed turbine!

Alternatives: in cases of severe heavy metal poisoning, amalgam should intermediately (until final inlays can be given) be replaced by non metallic inlays. Ceramic fillings may later be a good alternative.

Zinc: When zinc in urine is less than  $140 \mu g/g$  creatinin (spontaneously), lack of zinc is obvious. In cases of Hg-, Pb-, Cu-, and Cd-poisoning it should be substituted until a level of  $400$ – $600 \mu g/g$  creat. is reached. Therapy: weekly 1 ampulla of Unizinc (i.v.) or 3 times a day 1–2 dragees Unizinc two hours before the meals.

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These levels are only relevant for the measurement after the first mobilization, not if the substance has to be given again after assessment.

There is also a heavy metal depot if the measured value after mobilization is more than ten times higher than the first value before. In such cases the mobilization to reduce the levels should be administered monthly to every third month, according to the symptoms for each poison.

In cases of chronic poisoning, the antidote should be given repeatedly until the levels in urine have become normal.

### Other Antidotes

1. Dimercaprol (Sulfactin): elevates arsenic and mercury in brain (contraindicated). [Bio-Probe Note: Dimercaprol was previously called British antilewisite (BAL).]
2. D-Penicillamine (Metalcaptase, Trolovol): caution by penicillin allergy. Possibility for granulocytopenia, severe kidney insufficiency.
3. Na-Ca Edetat (Calciumedatat): Cases of nephrosis with anuria observed.
4. Ca-Trisodiumpentate (Ditripentat): Fever, exanthema, thrombocytopenia, nerve- and kidney damage observed.
5. Desferoxamin (Desferal): Reduced blood pressure.

#### Substitution

To enhance elimination of poisons and to compensate for losses during chelation, zinc should be given (zinc aspartate).

## Mobilization test for environmental metal poisonings

### Medical Laboratory Section:

Earlier depots of heavy metals in organs could only be measured in autopsies. Now, there is a practical method to verify these by giving a heavy metal antidote and measure the urinary concentration. Normally levels in blood and urine only give information on recent uptake of poisons. Mercury from amalgam fillings, for instance, with a half-time of 50 days, will be accumulated in the brain and cause migraine-like headaches. Lead, with a half-time of 30 days, will accumulate in bone-tissue and lead to anemia.

Measurements of levels of environmental toxic compounds in blood or spontaneous urine will give no proof of the existence of a poisoning and organ damage after long-time uptake.

The Desferal-test has therefore earlier been used to mobilize iron in cases of iron-storage disease.

#### Examples:

1. 30 years old patient, with 6 years of severe migraine, abdominal pains, nervousness. Hg in spontaneous urine 11.2 µg/l (maximum allowable concentration 4 µg/l). After 250 mg DMPS-Heyl i.v. urine Hg 2565 µg/l.
2. 45 years old patient with 3 years of extreme tremor and inability to eat. Cu in spontaneous urine 61 µg/l (max 50 µg/l), after 250 mg DMPS urine Cu 8426 µg/l.
3. Patient with arsenic symptoms. Spontaneous urine 5 µg/l. After 250 mg DMPS-Heyl i.v. urine arsenic 140 µg/l.

After treating about 1 000 cases, the following procedure has proved useful:

Translated by Mats Hanson Ph.D.

M. Daunerer, M.D. Forum des Praktischen und Allgemein-Arztes 28 (3): 88, 1989.

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**Dimaval test:**

1. A sample of spontaneously voided urine (50 ml)
2. Immediately afterwards injection of 4 mg/kg body weight (250 mg for 70 kg) DMPS (Heyl) intravenously.
3. After 30 minutes a second urine sample.

**DMPS**

DMPS ("Heyl," since 1957 in the USSR "Unithiol") is a weak toxic substance (300 times less toxic than Sulfactin (Dimercaprol)) and has so far shown almost no side effects. The effect, after i.v. injection, takes place after 17 minutes. Orally the absorption is about 30% and the effect is after 30–45 minutes. Up to now there have only been a few cases of allergic skin reactions.

Maximum allowable concentrations:

Poison	µg/l in urine after DMPS i.v. (= µg/g Creatinine)
Arsenic	25
Lead	50
Copper	300
Manganese	10
Mercury	50

**DMPS-test ist indicated for amalgam bearers**

Before removing of the fillings in cases of:

**AIDS**

allergy, generalized (1 ml)  
 amyotrophic lateralsclerosis  
 disturbance of cardiac rhythm  
 loss of hearing  
 loss of sight (or partly)  
 melanoma  
 multiple sclerosis (also untypical)  
 paralysis  
 perception disorders

**Chewing gum test:**

If mercury is over 100 µg/l, Sn over 30 µg/l in saliva after chewing test. Remark: filling should be removed under rubberdam and not with high speed turbine. 6–12 weeks after removing next remobilisation with DMPS (earlier when removed without named precaution).

Because: removal of amalgam fillings can cause irreversible damage in severe cases by unpreventable uptake of more poison.

**After removing the fillings in cases of:**

abdominal pain  
 allergy  
 apathy  
 bronchial asthma  
 carcinoma  
 colitis ulcerosa  
 eczema

gastritis  
 headache (migraine)  
 infertility  
 ischialgia  
 malignoma  
 Morb. Crohn  
 muscular pain



myatrophie  
neuritis  
reduced defence against infections  
T-cell depression  
trigeminal neuralgia  
chewing gum test:  
If mercury below 100 µg Sn blow 30 µg/l in saliva.

**Symptoms:**

allergy  
anaemia  
apathy  
bleeding gingiva  
bronchitis  
cancer  
cardial arrhythmia  
changing moods  
chronic cold  
copper coloured gingiva  
coughs  
defective hearing  
defective memory  
defective vision  
delusion of persecution  
depression  
diarrhea  
easily frightened  
ekzema  
facial twitches  
fear of suffocation  
fear of unknown  
feeling of numbness  
feeling of weakness  
flatulence  
gingiva blueish/-violet  
gingivitis  
hallucinations  
headaches (migraine)  
impairment of learning faculty  
increase of cholesterol  
increased perceptibility to pain  
increased salivation  
indecisiveness  
infertility  
insomnia

irritability  
kidney defects  
lack of energy  
lack of iron  
liver dysfunctions  
loss of hair  
loss of teeth  
loss of weight  
metallic sensations in mouth  
multiple sclerosis  
muscular weakness  
mycosis  
nervousness  
oral herpes  
oral pain  
pain of joints  
pain of ligaments and tendines  
pain of meniscus  
palsy  
paranasal sinusitis  
parapharyngeal pain  
paresthesy  
psoriasis  
restlessness  
rheumatic symptoms  
self consciousness  
sensitivity to electrostimulation  
shaky handwriting  
shortness of breath  
shyness  
slowed down reactions  
slurring speech  
stomach ache  
sutter  
tendency to infections  
teratogenity  
tiredness, persistent  
tremor (increased at intention)  
trigeminal neuralgia  
unawareness  
unsteadiness  
urinary disorders  
vertebral pain  
vertigo

## DMSA AND DMPS

Two drugs are now being used to treat Chronic Mercury Toxicity in Europe with considerable success.

Use of DMPS (sodium salt of 2,3-Dimercapto-1-Propanesulfonic Acid) results in a 25 times higher loss of mercury in the urine just one hour after taking the drug for those who had amalgams. This is considered a good test for measuring the body burden of mercury in addition to being a chelation treatment.

The objectives are to shorten the time an individual is sick with mercury and to help those who do not improve on their own. After amalgams are removed, mercury can change to different compounds and relocate to other parts of the body, causing new problems.

DMSA (Meso-Dimercaptosuccinic Acid) is even less toxic than DMPS, though both are tolerated well. People may experience headaches, tiredness and a metal taste in the mouth, all from the mercury being unbound from tissue. DMSA is more effective in removing mercury from the brain, while DMPS is better at removing mercury from the kidneys. DMSA also chelates zinc, so it is administered in cycles several days apart with zinc supplements given before and after. DMSA is preferred over DMPS for people suffering from neurological problems.

Neither drug is approved by the FDA, but U.S. citizens are permitted to import these and other experimental drugs by mail, or carry them through customs. In either case the FDA requires that:

1. the product be purchased for personal use only,
2. the amount not be excessive (i.e. a 3-month supply or less) and not for commercial distribution, and
3. the intended use be appropriately identified, and
4. the patient/importer affirms in writing that it is for his or her own use and provides the name and address of the licensed physician in the U.S. responsible for treatment with the product. Under no circumstances can a U.S. physician legally purchase and distribute the drug to his own patients.

Typical urine mercury content for those who have never had amalgams is 10 µg/l, 50 for regular fish eaters. Levels obtained with these drugs for those who had amalgams reach into the thousands of µg, with the record level of 42 000 µg/l. DMSA also is a good chelator of the heavy metal lead.

DMSA and DMPS can be purchased from Kripp's Pharmacy, P.O. Box 5198, Vancouver, BC, V6B 4B3, Canada, (604) 687-2564 or Jay's Pharmacy Ltd., 100-1940 Lonsdale Ave., North Vancouver, B.C. V7M 2K2 Canada, (604) 988-7128.

## Heavy Metal Removal with DMPS-Patient Protocol and Rationale

Apart from a possible allergy which is not dealt with here, amalgam, containing as it does mercury, copper, zinc together perhaps cadmium and nickel can lead to chronic poisoning. The use of DMPS as an antidote results in the elimination of heavy metals in the following preferential order: copper, arsenic, mercury, lead, iron, cadmium, nickel and chromium.

A high copper level will preferentially prevent excretion of other metals i.e. they will only be removed after elimination of the copper deposit. The extent of the accumulated copper in fact provides a yardstick for a zinc deficiency and zinc provides a relative antidote of the other heavy metals and works in opposition to copper. A zinc deficiency can only be measured in the urine before administering the antidote and a relative zinc deficiency is required before a relative increase in heavy metals is found. DMPS causes considerable copper elimination the toxicity of which is due to its interaction with other heavy metals. As this elimination concerns an emptying of the reservoir by mainly normal excretion methods, a higher level of urinary copper after treatment does not indicate copper storage disease (morb. Wilson) but rather a chronic imbalance. After the second treatment the copper level will generally lie below half of the original value and will be normal following a third treatment. After which the other heavy metals will be available for complete elimination. In case of further treatments zinc must be supplemented (amp. zinc aspartate or Unizinc) and in the case of prolonged therapy, iron as well.



Amalgam fillings need to be removed without the use of high speed drills and disposed of immediately in a sealed container. There should be at least a 4 week interval between the first and the second DMPS treatments, the latter of which could be in the form of capsules, 10 mg/kg with urine levels of heavy metals being sampled 2 hours after treatment.

Zinc requirements:

Children 10 mg/day. Adults 15–25 mg/day.

Zinc content of food (mg/100 g)

Wheat bran	10.0	Meat	3.5	Fish roe	1.0
Oats	7.0	Eggs	3.5	Milk	0.5
Whole wheat	3.0	Cheese	2.0	Veg/fruit	0.1
Wheat flour	0.5	Chicken	2.0	Fats/oils	0.1

Other indications and management tips:

Renewed treatments after approx. 6 weeks.

Further treatment after amalgam removal.

Zinc supplementation (weekly 1 amp zinc aspartate of daily capsule).

To assess whether further therapy is needed.

Control after elimination of the possible causes.

No controls necessary.

## Improvement of Nerve and Immunological Damages After Amalgam Removal

M. Dauderer

**Summary:** Dental amalgams cause neurological and immunological damages. Besides mercury, silver and copper, the metal tin is greatly responsible. Mouth bacteria convert organic salts into highly toxic organic compounds. The poisonous effects can be recognized by measuring the saliva before and after the so-called chewing gum test, which can cause abrasion test values up to 100 000 times higher than normal. High abrasion values correlate with high values in the mobilization test with the DMPS antidote. Only after amalgam sanitation and detoxification will damaged organs improve. It was found that high poison concentration corresponds with psychistic symptoms, while low concentrations relate to allergic symptoms. Even after outlawing amalgams, dentists and personnel would still be at great risk. This report describes the results and treatment procedures of one clinic having treated 2 500 patients with over 15 000 measurement values. Specific detoxification procedures are recommended.

Physicians as well as dentists are coming to the realization that dental amalgam fillings are much more dangerous than had been commonly believed. After placement in the mouth, these fillings not only release large amounts of heavy metals for several weeks, but during strong chewing or when drinking hot and/or sour liquid they are responsible for the release of up to 100 000 times the amount of mercury legally allowed in drinking water. Additionally, for instance, in the case of a 21-year-old, 740 micrograms of silver and up to 450 micrograms of tin are released via the saliva into the body. Mouth bacteria are responsible for changing mercury and tin into highly toxic organic compounds which are stored in the body's brain and immune system. Further, the largest amount of mercury (80 percent) is absorbed in gaseous form by the lung.

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**The Chewing Gum Test**

Since many dentists still claim that good amalgam fillings do not release toxic metals, the following test reveals whether we are dealing with inferior amalgam:

1. Have patient refrain from chewing for at least 2 hours.
2. Obtain spontaneous urine specimen (100 milliliter).
3. Collect 5 milliliter of saliva.
4. Have patient chew intensively on amalgam fillings with sugar-free chewing gum, and collect in a second container another 5 milliliter of saliva.

Tests are to be performed in a special laboratory for mercury and tin and possibly for silver and copper as well (a competent laboratory in Germany, Tel. 04 21/34 96 40). The amalgam fillings are considered inferior while 40 percent of the cases had to be treated over one year. In 5 percent of the patients we had to break off therapy after 5 to 10 injections due to a DMPS allergy (mucous tissue and skin symptoms). If the preparation is used in capsule form, at the most it must be taken every two weeks as a single dose of 3 µg/kg on an empty stomach.

Since zinc is also strongly excreted and additionally promotes the elimination of mercury, it should always be determined in spontaneous urine. If lacking, it needs to be given. Selenium and iron need to be supplied only if necessary.

**Border Values of Occupational Medicine**

Lately it has been found that practitioners of occupational medicine utilize this mobilization test although they previously strictly refused to see any value in our method of recognizing and eliminating poison deposits which we discovered ten years ago.

Now they would like to extend their border values, which are valid for healthy workers, to severe ill patients, allergics, and children. It should be obvious, however, that an amalgam-poisoned patient with multiple sclerosis or sudden deafness, spontaneous visual disturbance, lameness or AIDS cannot tolerate an additional nerve or immune poison in whatever concentration.

Since we know in a mobilization when and how the elimination begins, we do not need a 24-hour urine, which is only indicated when one does not know when the poison was received or the elimination begins.

Our method was developed in a toxicological practice for the general physician and has of course been compared with clinical methods and is standardized. An evaluation of mercury elimination by DMPS makes only sense with a co-determination of copper, proving the amalgam is made possible by the additional tin determination. Comparisons with the chewing gum test verifies the formation of deposits from the amalgam components. This clinically verifiable toxicological diagnosis is still lacking up to now in occupational medicine examinations.

**Error of the Dentists**

Dentists are in error when they evaluate only the mercury in amalgam, while not considering the poisonous effects of the other components such as tin, copper and silver. If they were to do so, they would not have accepted the erroneous view that mercury deposits come from food but not from the fillings. The fact that the chewing of gum on amalgam fillings releases up to 197.8 micrograms of mercury into the saliva was already described by the dentist Prof. KRÖNKE and the occupational physician Prof VALENTIN (OTT, 1984), who therefore knew that amalgam poisons the saliva. This makes the denials of many dentists difficult to understand.

## A Brief History

Amalgam was developed in 1826 by Taveau, but was prohibited in 1840 in the U.S. because of mercury vapor poisoning problems, although it was reintroduced by the dentists in 1855. In more recent times, its use was prohibited in January 1985 in the USSR due to the many chronic cases of amalgam intoxications. Prof. Stock of Berlin warned already in 1926 of the mercury vapor poison danger by amalgam. He himself died of chronic mercury poisoning, although his warnings did not succeed in an amalgam prohibition.

## Symptoms of Amalgam Poisoning

The following symptoms of illness were found by us in lessening frequency in 3 000 afflicted cases: No drive or initiative for work, stomach pain, susceptibility to infections, memory disturbances, sleep problems, depressions, dizziness, tremors, muscular weakness, visual disturbances, hearing problems, malignant tumors, and multiple sclerosis.

## Indications for the Amalgam Test

Before the sanitation in: AIDS, allergy, generalized amyotrophy, lateral sclerosis, loss of sight (partial), hearing loss, heart rhythm disturbances, paralyses of other types, malign melanoma, multiple sclerosis (also nontypical), sensibility disturbances (loss of feeling).

Chewing gum test: If Hg values are over 100, Sn over 30 µg/l saliva. Comment: Amalgam removal after giving the antidote should be performed with a rubber dam and without fast turbine. Six to twelve weeks after removal, repeat mobilization (shorter interval without above protection). Reason: Amalgam sanitation with the unavoidable additional poison ingestion can lead to irreversible damage in the patient's condition.

After the sanitation: Allergies, loss of drive, bronchial asthma, stomach pains, ulcerative colitis, eczema, gastritis, susceptibility to infections, infertility, ischialgia, carcinomas, headaches, cancer, Crohn's disease, muscle pains, neuritis, T-helper cell depression, trigeminal neuralgia.

Chewing gum test: When Hg under 100, Sn under 30 µg/l saliva.

## Alternatives

Approximately 800 dentists in Germany, who have joined together in the International Association for Total Dental Medicine, have had a policy for many years of not utilizing amalgam. Their patients do not suffer from allergies nor do they show symptoms of poisoning due to their dental sanitation. We have to thank this association for the following information:

1. After the exchange of many amalgam fillings and in severe chronic poisonings, gold or ceramics must be used for at least six months (or preferably one year) until detoxification is completed, since the amalgam depots in the jaw bone (which can be observed in panorama views) may still act as a disturbance.
2. The dental gold must not contain palladium, indium, gallium and vanadium and should contain as little copper as possible.
3. Allergic patients should be tested for a gold allergy.
4. Light-hardening plastics or glass ionomer cements are suitable for longer temporaries.
5. At least in serious organ damages by amalgam poisoning, removal should be made with rubber dam and without fast turbine. Deaths have occurred in cases of grave organ damages without protection and prior and accompanying detoxification.
6. The National Health Insurance Service will pay readily for the cheaper alternatives.

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7. The official dental spokesmen limited themselves early on by choosing the amalgam side and did not show flexibility to investigate new measurement results, respectively to work with physicians regarding the subsequent diseases. As in all modern environmental problems, the afflicted have to discover their own protection.
8. Old amalgam fillings must be removed as quickly as possible.

## Should Amalgam be Prohibited?

In Switzerland, dental treatment is not paid by the National Health Service. Because of unfavourable amalgam publicity, amalgam sales went down by 99 percent. Herein can be seen the wishes of the patients. In July of 1990 the Swedish government released preliminary information about prohibiting amalgams. Final rules are said to be forthcoming in late 1990.

In the U.S., Canada and Australia, the amalgam question is being hotly debated. Informed patients in Germany will also demand an early amalgam prohibition. Even when passed, we will have to suffer from the amalgam consequences for many decades. Compensation claims for damages can be expected in positive amalgam test cases when typical organ damages are involved, since a dentist must not cause iatrogenic harm to his patients.

## Amalgam Fillings: An Error in the State of the Art

## Case Reports

A nine-year-old girl received five amalgam fillings within one year. After having hit her head in a fall, she developed at grave encephalopathy during the same year. The EEG became flat, she was extremely agitated, jerked rhythmically and stopped interrelating with her environment, and finally needed force feeding. Hg spontaneous 18.5 µg/l, after 3.5 mg/kg KG DMPS orally, 213.5 µg/l in urine (!), although 80 percent of the elimination was in the urine.

The hair showed a mercury level six times higher than normal.

During a seven-month DMPS therapy (100 mg/week orally), the severe poisoning symptoms gradually reversed themselves. A considerable improvement, however, occurred only after removal of the amalgams.

A thirty-year-old female patient suffered for years with permanent migraine and pains in the lower abdomen. Since her dentist suspected her amalgam fillings as the cause, he exchanged them for 11 new ones. Immediately after the exchange, her symptoms became worse. Hg in spontaneous urine was 11 µg/l, after 3 mg DMPS/kg i.v. 256 µg/l. The headaches and stomach pains disappeared after this mobilization spontaneously and only returned in milder form six weeks later. After removal of her amalgam fillings and remobilization, the patient finally became free of symptoms.

A 33-year-old female patient with 14 amalgam fillings, which had been in place for 25 years (and were occasionally renewed), felt very tired, exhausted and suffered from constant feelings of dizziness, nausea, headaches, could not concentrate or remember, formication, and paresthesias on the extremities as well as a pronounced decrease in her ability to resist infections. She complained about a bitter taste in her mouth and unpleasant body smell, abdominal pains, gas diarrhea, cardiac palpitation, tachycardia and paroxysmal arrhythmias. In addition, she developed a candida bronchitis, bronchial asthma and nickel allergy. Hg in spontaneous urine was 7 µg/l, after 250 mg DMPS i.v.: Creatinine 1.36 g/l, Pb 121 µg/l, Cu 2,493 µg/l, Hg 2,794.3 µg/l (border value according to our experiences after mobilization free of symptoms up to 50 µg/l). After detoxification by mobilization and removal of amalgams, there was substantial improvement of all complaints.

A 64-year-old patient with 21 amalgam fillings experienced in the area of an old spinal fracture lancinating pains similar to a prolapse of an intervertebral disk without organic substrate. After removal of all his teeth and supplementary zinc, the Hg excretion increased from normal values to over 5 000 µg/l in urine and normalized itself under continuation of the zinc substitute over two years.

cations, we ourselves have seen 1000. The most impressing of which is a report on depression of T- and B-lymphocytes, followed consequently by immune deficiencies, which are reversible after removal of amalgam fillings, as well as are neurological alterations. Yet patients are extremely worried about other long year damages that often do not vanish again.

## View

After having pointed out a coincidence with thalidomide and deformations, the pediatricist Dr. Lenz was regarded a booster and outsider. During the following two and a half years, another 2 500 children were born crippled, meagerly paid off with 10 000 DM each.

How long will it take until poisoning by amalgam fillings will be banned?

## Tests in Relation with Amalgam

The metals in amalgam intensify each other in their effects. These are the most important ones:

Tin increasing weakness, apathy, neuralgia, pain sensitivity, paresis, undulating pain in the gastro intestinal tract, headache, hoarseness, cough, sensibility to chill and changing weather, paleness.

Copper: clonic cramps, gripes, impaired vision, difficulty in breathing, paraesthesia, tremor, weakness, anal cramps, constipation, grinding of teeth, allergy, liver damage.

Silver: anxiety, forgetfulness, blocking of thought processes, encephalomalacia, headache, vertigo, defectiveness to stress, mental weakness, impaired ligaments muscles and joints, chondrogenesis, vertebral pain, rheumatism.

Mercury: apathy, headache, gastro intestinal disorders, vertigo, tremor, forgetfulness, sleeping trouble, muscular asthenia, vertebral pain, allergy, nervousness, changing moods of apathy and irritability depressive mood, ataxia, paresis, paraesthesia, impaired vision and hearing, susceptibility to infections, anaemia, disorder of cardiac rhythm.

## Test with chewing gum

### Indication:

to prove the amount of toxic metals from amalgam tooth fillings in normal saliva (saliva I), respectively what is abraded by chewing (saliva II) – in relation to acute toxic status in the body (urine I).

### Realization:

salvia I: collect half a test tube (about 5 ml without foam)

salvia II: after 21 hours without chewing – intensive chewing of gum on the hilled teeth, collecting salvia in a second tube (5–10 minutes, same amount)

Send to laboratory: saliva I for Hg, Sn, Ag  
salvia II for Hg, Sn, Ag, pH  
urine I for Hg, Sn, Zn

Valuation: the amalgam fillings are of inferior quality or poisonous if saliva II (after chewing) contains much more than urine or saliva I (norm.:  $uI < sII < sIII < 5 \mu g/l$ ). Severe toxic effects occur at differences over  $100 \mu g/l$  Hg.

## DMPS-test (Dimaval)

Indication: in cases of severe nerval or immune deficiencies the depots of heavy metals in the organs should be treated and at the same time measured in urine by giving an antidot. The test is harmless and it is paid

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## Conclusion

Prohibiting Hg amalgams in 1840 in the U.S. was clearly justified, but was reversed because of industry and dentist pressure since, due to inadequate scientific methods at that time, it was not possible to verify deposit formation. However, as of now, the use of amalgams must be seen as a serious error in the state of the dental art.

## Literatur

DAUNDERER, M.: Klinische Toxikologie, volume 13. Umweltgifte. Amalgam. Ecomed, München-Landsberg-Zürich, 1989.

## Acknowledgement

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Co-determining factors of increased release of Hg from amalgam fillings are:

- Frequent hot drinks, sour foods, fluorcontaining toothpaste,
- Chewing of gum, grinding of teeth, other metals in the mouth and the already mentioned lack of zinc.
- In severe cases of intoxication there was a clear T-helper cell depression, which disappeared after therapy.
- A deposit could be recognized and treated in cases of continuing symptomatic complaints up to 20 years after amalgam fillings were removed.

## How to Proceed

After a series of varying methods, the following procedure has shown itself most practical for the day-today routine of a general practitioner:

1. Spontaneous urine I: Testing for zinc and possible nickel
2. Injection of 3 mg/kg KG DMPS slowly i.v.
3. After 45 minutes, urine II for mercury and copper (plus lead in high pressure, plus cadmium in osteoporosis).

If given orally, the antidote is resorbed only uncertainly, this is why the test must be given on an empty stomach. Since only about 30 percent is orally resorbed, the needed dose is 10 mg/kg KG as bolus. The capsules have been on the market for 13 years under the name of Dimaval®. Our first grave intoxication case (arsenical poisoning) in 1976 was treated with it successfully.

Each physician should himself conduct the treatment before and after removal of the amalgam fillings for the appropriate clinical symptomatology. We send the urine samples to an experienced laboratory physician (Tel. 04 21/34 96 40 in Germany).



## Legal Consequences

1. Even if the observed cases had been singular events – although everything speaks against it – the severity of the side effects and the impossibility to prevent them, makes it mandatory to immediately stop all further use of dental amalgams containing mercury.
2. Gold is a good alternative, routinely approved by the National Health Service and is to be employed for allergies (after careful testing for compatibility) as well as for intoxicated patients.
3. Filing of claims for damages in severe documented disease involving private and job-related losses must be allowed (common fund?).
4. All dental materials should be meticulously tested as regards their long-term effects according to current drug tolerance laws before they are permitted on the market (economic gold: indium, gallium, vanadium, nickel, beryllium, plastics, formaldehyde in root canals, etc.).
5. Dentists, general practitioners, neurologists and others need to be informed post haste about the diverse intoxication symptoms and their therapeutic possibilities.

## An Open Scientific Question

- Immune deficiency caused through amalgams: Can mycetogenic and virus diseases, multiple sclerosis, ulcerative colitis, rheumatic forms, etc., be induced or intensified?
- Mercury is teratogenic: Can these concentrations cause fetal malformations?
- Mercury is mutagenic: In which frequency in this concentration?
- Mercury concentration in bone under amalgam fillings in dissection material?
- Are there irreversible injuries (misshaped, neoplasms, MS)?

American Journal of Probiotic Dentistry and Medicine, January-March 1991

## DMSA

### 2,3 – Dimercapto Succinique Acid ( $C_4H_6O_4S_2$ )

Category: Heavy metal chelator.

Activity: DMSA is the best derivative for chelating organic mercury which is sequestered in the brain and spinal cord (chronic exposure). DMSA is the least toxic dimercapto chelator.

Indications: Chronic heavy metal toxicity especially for patients exposed to cadmium, mercury, lead, arsenic, and copper – in the order of effectiveness. The literature indicates that DMSA is also effective for nickel. (Ni forms a strong disulfide-organometallic membered heterocyclic compound with DMSA in vitro.)

Contraindications: Do not take during pregnancy (DMSA can mobilize long term sequestered stores of mercury from brain into the blood stream. Such organic mercury is easily transported into the fetus).

- Sulphide sensitivity
- Overdosing\*

Precautions: Gradually increase dosage over 3 day period to 100 mg daily. Do not increase over 100 mg daily without physicians approval.

Overdosage: Overdosage gives rise to same symptoms as for acute organomercury poisoning such as irritability, ringing in the ears, fatigue, nausea, malaise, skin rash, joint pain, muscle pain, liver pain, kidney pain, agitation, personality changes and very seldom seizure.

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Dosage: Adults, one 100 mg capsule # a week

Supplied: 100 Hypoallergenic and hyposensitizing 100 mg capsules

\* A wide range of tolerance exists due to many factors involved. Initiate therapy in chronic mercury toxicity.

Compiled by: Edward THORPE Ph.D. Consultant (604) 325-0020.

## Non-existence of a "social indication" for the continued use of amalgam

These research results obtained from the annual experiments in Calgary have definitely confirmed the statements presented by scientists in the field of holistic medicine regarding test results and the toxic dangers of dental repair using amalgam.

Comments from experts, such as those made by the toxicologist R. Shulte Hermann, have also been identified as misleading and emotional statements. The results of the practical work of dentists and industrial physicians are unfortunately also not able to conceal the statistical evidence for its inability to maintain teeth: a rate of illness of over 90% tooth decay and tooth loosening is said to have been caused.

Whether the main causes of this misdevelopment lies in the overly commercial attitudes, ignorance or mental retardation (through the presence of mercury depots in the limbic system of our dental practitioners) is at the moment of no consequence – it is, at this point, however, irresponsible towards patients if they continue to be harmed iatrogenically with this toxic material.

There exists no "social indication" for the use of amalgam!!! Just as there is no longer any treatment of syphilis today with mercury preparations!!!

Till T.

## Holistic Medicinal Consequences

### A. Nutritional modification – Outline

From all benevolent sources, it is to be deduced that our nutrition has been modified and made unhealthy by many manipulations in the course of which, our natural instincts have been negatively influenced by the great variety of tricks used to deceive our taste.

This type of false programming can only be slowly and intentionally corrected and put into order again.

Accordingly, it is important to outline the advice presented in the first chapter in more detail: The use of kitchen utensils and crockery made of aluminium or nickel alloys is to be absolutely avoided and to be eliminated from the household.

Under all circumstances, the avoidance of pork and all products prepared from it, milk and cheese products (only in the rarest cases can exceptions be made), alcohol and nicotine abuse, commercially prepared sugar and white flour, wheat (crop disinfectants). The consumption of all innards is to be prohibited!

Preverence is to be given to natural products which have not been modified: Fruit, vegetables, lettuce, nuts as fresh as possible, fresh rolled grains (oats, spelt, buckwheat, corn, etc.) and a little honey. Recommended beverages are freshly brewed teas and mineral water. The consumption of meals with animal protein must be absolutely reduced to a maximum of 2–3 times weekly.



If it is necessary to eat guest house food once daily, then one must strive to choose freshly acquired and prepared meals, those which have not been cooked in microwave ovens. Foods that have been repeatedly re-warmed, or pre-cooked commercial preparations, and economising and substitute products of the postwar cuisine respectively, should be avoided, together with minced meat, sausages and canned foods as sources of human nutrition.

If we were compelled to consume animal proteins as a form of nutrition in times of distress (Ice Age, etc.) – it is not by any means a reason to equate the Homo sapiens with the pig. This poor creature is unfortunately compelled to feed on everything – but we are definitely not!!

## B. Environmental Protection – Conditions

Finally, legislative fuling of environmental protection should ensure that the further use of mercury-containing crop disinfectants in grain cultivation be prohibited, which has been carried out in Sweden for many years. Besides, it is surely possible to gradually exclude mercury-contaminated feed used in stockbreeding, and to introduce appropriate measures for stricter controls of industrial waste water.

## C. Dental Protection Measures – Outline

1. For a purposeful consultation, it is of primary importance that the patient provides the complete and latest radiological status of his teeth.
2. Oral inspection and planning of the necessary and often long-term rehabilitation and detoxification measures.
3. Detection of metal depôts of the various trace elements contained in amalgam, such as Hg, Cu, Sn, Cd, and Se, during the "mobilisation test" in an individual manner in specially-equipped laboratories – in Germany: Tox Center, Weinstraße 11, 80333 München (Dr. Dr. M. Daunerer), in Austria: Labor für Bio-Analytik, Schwarzschanerstr. 15, 1090 Wien (Prof. Dr. Dr. J. Birkmayer).
4. After the detection of the metal depôts, planning of the actual rehabilitation and detoxification programme, beginning with the fractionated removal of amalgam (maximally 2–3 teeth per sitting) and provisional filling together with accompanying medical treatment if necessary.
5. After complete removal of the amalgam (also from below the crowns), renewed testing for the presence of metal depôts to allow for subsequent metal-detoxifying measures, when required. For this, there are various instructions available at the special laboratories mentioned above. In addition, the patient must be instructed to minimize extra burdens caused by heavy metal-containing foodstuffs such as preserved foods, stockbred fish, pork products and all innards.
6. Definitive dental care can only be undertaken when the heavy metal detoxification has been completed and verified by tests.

On the basis of holistic medicinal facts and experiences the dietary adjustments made by the patient are to be accompanied by long-lasting dental care of the molar area in the form of protection with crowns as opposed to other forms of repair. This is especially the case when multiple defects are to be rectified.

At present, a wide range of faultless materials are available: 22- and 20-carat gold, laboratory-prepared synthetic materials, Dicor or porcelain for the front teeth. Principally, only one type of metal should be used for dental repair, exceptions are the cases in which removable dentures made from chromium-cobalt alloys. In this case, the possibility of the so-called "passivity" (neutralisation) of such alloys in the mouth is put to use. This special tolerance can be tested. Also, the possibility of considering the presence of existing hip joint implants must be included.

7. Dental care should be intensified in many cases and carried out after every meal. Taking fluoride products and the use of fluoride toothpaste is expressively forbidden.

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8. In all cases, the patient is to be impressed with information on his health situation. At the same time, he should be encouraged to consciously correct various unhealthy habits.

If the capacity to understand has been decreased by the presence of mercury depots in the limbic system, further indication that the detoxification has been insufficient is evident. This can be tested.

Should it turn out that the patient consciously tends to keep up his unhealthy habits, it is then possible for every respectable physician to decline further responsibility for the treatment and leave the patient to his own devices.

Till T. Techn Univ. of Linköping

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## Diffusive Precipitation of Mercury from Dental Amalgams.

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## Diffusive Precipitation of Mercury from Dental Amalgam

Dental amalgam releases mercury. This leakage takes place in several different ways, e.g. by corrosion. JORGENSEN (4) suggested that mercury, released by corrosion, is taken up by the  $\gamma$ -phase and produced "mercurioscopic expansion." However, he admits that during severe corrosion, free mercury is present among the corrosion products from conventional amalgams. FREDIN (1) confirms in his study the presence of free mercury droplets on the surface of old amalgam fillings.

SCHNEIDER & SARKAR (6) have observed free mercury droplets on the surface of polished Dispersalloy. The drops are concentrated to the surface of unreacted silver/copper-eutectic grains. HERO & JORGENSEN (3) have observed small, elongated drops of  $\gamma$ -phase on the polished surface of condensed ANA 2000. The drops were considered to be concentrated along the phase boundaries. The appearance of drops on the surface of polished or ground amalgam will subsequently be called DIFFUSIVE PRECIPITATION. The expression diffusive precipitation is not yet an established expression.

## Materials and methods

The following amalgams have been studied: Culk Ease from Culk Inc., Dispersalloy from Johnson & Johnson and ANA 2000 from Nordiska Dental. The two first amalgams have been prepared from alloy powder and ANA 2000 was triturated in a capsule. Six cylindrical test specimens, 5 mm height and 3 mm diam., were prepared from each amalgam. Condensation was made in DELRIN, a polycrystalline plastic of homopolymer type (POM). All samples were mixed and prepared by practicing dental personnel according to daily practice.

All test samples were treated with wet abrasive paper down to wize 4000. This corresponds to a maximal profile depth of about 4  $\mu$ m. The finishing was done under excessive water flow to avoid a temperature rise.

Ulf Bengtson 1986, British Journal of Industrial Medicine 1990, 47:105-109. Translated by M. Hansen.

After the treatment the test samples were washed in water and then in 99.5 alcohol. Drying was done in cold air. No polishing was done. The chosen finishing method is a reasonable compromise between a surface sufficient for allowing microscopy and the real surface finish of the *in vivo* situation. The studies were mainly done by microscopy. The following microscopes were used:

- 1/ JEOL JSM-25 III Scanning electron microscope (SEM)
- 2/ Reichert MeF2 light microscopy for metallographic studies
- 3/ Reichert Metapan 25 light microscope for metallographic studies
- 4/ Swift 84M1027 light microscope for metallographic studies.

All pictures were taken with JEOL-25 III and Reichert MeF2. Occasionally Reichert Metapan 25 and Swift 84M1027 were used during these studies. The temperature elevations from the light sources of the microscopes are diagrammed in fig. 2. A possible temperature in SEM is difficult to estimate. Experience operators find a slightly elevated temperature during so called spot-measurements possible. Some of the pictures with Reichert MeF2 have been taken with the help of interference contrast according to Nomarski. This type of examination which uses polarized light, is used to trace extremely small variations in height in metallographic samples. In the studied samples the diffusive precipitations are so large that interference contrast is unmotivated. However, with a certain adjustment of the interference equipment, the precipitations appear golden whereas the background material is light blue. Most probably this is caused by the presence and absence of an oxide layer, respectively. It is known that mercury forms oxide layers with difficulty in dry air. In the SEM pictures the precipitations appear as strongly bright droplets. This is caused partly by so called point effects and partly by atom number of the ingredients in amalgam. For some samples Energy Dispersive x-ray Spectrometry was used. In these samples qualitative measurements with line-sweep were preferentially used. This test method gives the Hg-content of the diffusive precipitations in relation to the other surfaces and thus not the exact mercury content of the precipitations. To exactly measure the mercury concentration in small droplets on the surface of amalgam is a very difficult task. It is very difficult to avoid penetration of the droplets with the electron beam and "contamination" by the subsurface. A micromanipulator was constructed and built to be able to touch the diffusive precipitations within the microscope viewing field. A mechanical loading apparatus was constructed and built to be able to study the effect of mechanical loading in SEM. To study the diffusive precipitations in sequences of pictures, a special computer program, KAMSTYR, suitable for studies of non-linear events, was developed.

## Hypothesis, Results, and Discussion

The mechanisms of the diffusive precipitations is explained in fig. 1a and 1b. Fig. 1a shows Caulk Ease or Dispersalloy and fig. 1b ANA 2000. All amalgams contains powder grains from the alloy which together with mercury are the ingredients of the final metal. Caulk Ease and Dispersalloy are related and both contain grains of two types. Fig. 1a shows the type of grain. The shell is a mixture of  $\eta$ -phase and  $\gamma$ -phase. ANA 2000 consists of one type of powder only. The alloy contains two phases,  $\gamma$  and  $\epsilon$ . The  $\gamma$ -phase contains silver and tin and the  $\epsilon$ -phase copper and tin. Remains of unreacted alloy the surface or slightly more slowly by abrasion (chewing). The strong concentration gradient between the nucleus of the grains and surrounding metal constitutes the force behind a strong surface diffusion of mercury. This is particularly strong on a newly ground surface which is rapidly exhausted in free mercury atoms. Then a more slow process takes over and free mercury atoms migrate through the metal by volume diffusion to the surface and further into the exposed grain surfaces. In ANA 2000 the liberation of mercury seems to be so profound that the grain surfaces are rapidly filled with mercury. Then free mercury will be precipitated in the form of drops also outside the grain rests. In Caulk Ease and Dispersalloy it is mainly the grains of silver/copper eutecticum which are involved in the precipitations. In ANA 2000 it appears as if it is one of the alloy phases which initially participates in the precipitation. Some EDS studies suggest that it is the  $\gamma$ -phase which is involved in the diffusive precipitation. Normally the mercury which accumulates on the grain surfaces should react with the rest of the alloy. Apparently this does not happen to any appreciable extent. A possible explanation is that the formation of an oxide layer on the surface of the exposed grains,

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This will act as a diffusion barrier which prevents or strongly reduces diffusion between the mercury and the alloy. It is known that untreated, copper-rich alloys react with difficulty with mercury because of oxidation around the grains. Some manufacturers use surface treatment of the grains to avoid this oxidation. The powder grains which are exposed because of abrasion or polishing are for obvious reasons not surface-treated inside.

It should be noted that both surface and volume diffusion rates increase exponentially with increasing temperature. A rise in this has considerable effects on diffusion rates. Also the vapor pressure of mercury increases exponentially with temperature. A rise in temperature will thus both increase transport of mercury atoms within the metal and release of these in the vapor state. The tested amalgams are unstable alloys. Diffusion phenomena which occur at temperatures of several hundred degrees centigrade for our common metals can for dental amalgam be studied as a continuous process at room temperature. Picture 1 shows a test specimen of Caulk Ease. The sample was triturated 1985-12-02, grinding was done 1985-12-10 at 10.30 h and photographed in SEM 1985-12-10 at 12.30 h. Already a few hours after abrasion the powder grain rests started to be invaded by drop-like particles. The drops are at the beginning in rings somewhat towards the center of the grain surfaces. Picture 2 shows the same sample 24 h later. The ringlike orientation of the drops within the grains has now disappeared and the whole surface is covered with drops. Observe that the drops mainly are concentrated to alloy grains with a reaction shell of  $\eta$ -phase and  $\gamma$ -phase, i.e. grains of silver/copper eutectic grains.

Picture 3. Dispersalloy triturated 1985-12-18, grinding 1986-01-27 and photographed in SEM 1986-01-29. Two days after grinding also this amalgam shows the typical concentration of mercury droplets in unreacted rests of silver/copper eutectic grains.

Picture 4 shows a specimen of ANA 2000 triturated 1986-01-27, grinding 1986-01-27 and photographed in SEM 1986-01-29. Also here drop-like structures are precipitated on the surface of unreacted grains. However, only part of the grains have this ability, most likely the  $\gamma$ -phase.

Picture 5. Caulk Ease triturated 1985-12-01, grinding 1985-12-10 and photographed the same day. A drop on the surface of a silver/copper eutectic grain. A line-sweep was done across the drop along the black line. The curve shows the mercury signal  $Hg\ L\alpha = 9987\ keV$  viewed through the window 9.87–10.06 keV.

Picture 6 shows the corresponding test on ANA 2000 triturated 1986-01-15, grinding 1986-01-27 and measured in SEM 1986-01-29.  $Hg\ L\alpha$  was here studied in the window 9.82–10.16 keV.

Some of the samples in this study were studied in SEM. This means that the samples were exposed to vacuum. To study if this could stimulate the diffusive precipitation one sample was left in vacuum of the scanning electron microscope for one hour. Pictures of the same area were taken at the start and at the end of the experiment. No difference between the pictures could be seen. To study if mechanical pressure could stimulate the formation of the diffusive precipitations, loading experiments were done in SEM. Four test specimens, all Caulk Ease, have been subjected to pressure in an apparatus which gives pressure loading at right angles to the ground surface. The four specimens were subjected to increasing pressure up to breakage. No changes in the diffusive precipitations were found, neither in rate nor in extent.

Just 10–30 min after grinding the mentioned changes were seen. To further illuminate the mechanisms involved, picture sequences of grain rests were taken after grinding. These studies were done on Dispersalloy and ANA 2000 with Nomarski interference contrast microscopy. To catch the relatively fast process immediately after grinding – especially for ANA 2000 – the following exposure interval was chosen:

- 10 pictures, 10 min between exposures.
- 20 pictures with 20 min between exposures and
- 40 pictures with 40 min between exposures.

A photo sequence will thus consist of 34 h 50 min. After completed photo sequence, additional pictures were taken with oblique illumination, ordinary light. The picture sequence of Dispersalloy shows that the diffusive precipitations start immediately inside the previously mentioned reaction shell and continue towards the center until the whole surface is covered. For ANA 2000 some parts of the grain remains are

relatively quickly covered. Careful studies of the pictures show, however, that the precipitations appear to have the character of a film. From this film drops grow up. Drops which initially are small coalesce to larger ones. The test specimens, used in the photo sequences were then left untouched for a month. Liberated mercury should have had plenty of time to react with the grain rests. The diffusive precipitations have apparently continued, especially ANA 2000 shows now considerable presence of drops also outside the grain rests. Both test specimens were placed under the microscope and the diffusive precipitations were touched with the micromanipulator. A large number of pictures have been taken, both before and after touching the precipitations. With Dispersalloy these can be moved and pushed together to larger drops. With ANA 2000 the precipitations were relatively dark. After touching with the micromanipulator the precipitations immediately became silvery. Many drops were smeared out, some moved and some coalesced. The subjective impression was that they had a porridge-like consistence. A few EDS studies show that the precipitations in mercury and silver. The binary phase diagram shows that this alloy will be a solid phase for a few percent silver content. No scratches from the needle of the micromanipulator were seen either in the powder grains, the metal outside or the precipitations. The observations indicate liquid or porridge-like consistency of the diffusive precipitations.

Ground, polished, abraded or in other ways worked metal surfaces are known to contain many dislocations in the gitter structure (5). RUOFF (5) mentions dislocations as enhancing factors for diffusion. Approximately the double diffusion rate can be expected along these gitter defects compared to the diffusion rate in a perfect gitter. The same is also true for grain boundaries. GUY (2) states that the surface diffusion coefficient  $D_s$  is "several magnitudes larger" than the usual volume diffusion coefficient  $D$ . JØRGENSEN (4) states that grain rests from the powdery alloy always remain in dental amalgam. In contrast to common metals, a strong diffusion occurs in dental amalgam already at room temperature. In fact, this is a prerequisite for the hardening process, the amalgamation. JØRGENSEN (4) states that amalgam flows at room temperature. Since diffusion is one of the mechanisms of flow (RUOFF, 5), this confirms the conclusion that diffusion occurs in hardened amalgam at room temperature. Since grain rests continuously are exposed by abrasion, the process will continue. JØRGENSEN states that the  $\gamma$ -phase initially contains 70.3% Hg but is transformed within months and years to a new phase,  $\beta$  1, which contains about 50% Hg. The process is thus continuously fuelled with new free mercury atoms.

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To illustrate my findings I enclose 4 slides of microscopical images (s. slide 25–28). The approximate length of the picture shown is 0.14 mm. The samples have been ground under water (WTRUERS 4000 grindingpaper). After grinding the samples were placed under a microscope and 70 photos were taken during 34 hours and 50 minutes. The first 10 pictures were taken at 10 minutes intervals, the next 20 pictures at 20 minutes intervals and the last 40 pictures at 40 minutes intervals.

The two green slides are visualized with a special technique utilizing polarized light (Interferenzkontrast nach Nomarski). The pictures show two Ag-Cu eutectic particles of Dispersalloy® amalgam (Johnson & Johnson Dental Products Co, MJ, USA). In the Nomarski technique the droplets appear in a yellow colour. The slide with few yellow droplets was taken 1 hour and 50 minutes after grinding and the next one after 4 hours and 10 minutes. At the end of the test 34 hours and 50 minutes after grinding (and 70 pictures later) the same area was photographed in ordinary light and as you can see the Ag-Cu eutectic particles are completely covered with droplets.



The fourth slide shows the surface of the Swedish amalgam ANA 2000® (Nordiska Affineriet AB, Helsingborg, Sweden).

When I first found these droplets I made a computerized information retrieval trying to find if this phenomenon had been dealt with in the scientific literature. To my surprise almost nothing had been written about it. The only paper dealing with the problem was SNEIDER PE, SARKAR NK: Mercury release from Dispersalloy amalgam, sent to me by Jaro Pleva, HERO H, JØRGENSEN RB: Flow stress and deformation hardening of triturated amalgam, mentioned the phenomenon in one sentence in a paper dealing with a completely different subject.

I found these droplets alarming since:

1. They visualized the instability of amalgam in a dramatic way.
2. If the droplets are loosely bound to the amalgam surface there is an obvious risk that they are transported to the gastrointestinal tract. The uptake of mercury from particles one  $\mu\text{m}$  wide and less is as far as I know unknown.
3. Only the modern, copper-rich amalgams introduced on the market some 15 years ago showed these droplets to a great extent.

I informed Socialstyrelsen, SoS, a government agency dealing with health care. As a result of this they asked the Swedish Institute for Metals Research to measure the Hg-content of these droplets. They succeeded in stripping of five droplets out of hundreds possible from a two weeks old sample. All droplets were from the same area of the sample. The Hg-contents measured was 44.1%, 45.2%, 47.7%, 49.6%, and 85.4%. In a letter to SoS I told them that I suspected that the method used only stripped solid particles and could not handle liquid mercury. In my own studies I have been touching the fresh droplets with a micro-manipulator being able to smear them out and sometimes they have simply disappeared – may be amalgamating with the micro-manipulator needle. Since there is no definite proof for the droplets being pure mercury, I prefer to speak about mercury-rich droplets. I find the question of their exact content less interesting. The important lesson from the experiment is that the mercury in modern, copper-rich amalgam subjected to grinding and wear is very unstable and moveable and exhibits an extreme surface diffusion at room temperature.

The Socialstyrelsen did not do anything to further investigate this matter. They twice told me that if I wanted research done I had to do it myself. This was very surprising since I spoke to them as a concerned individual who never before had been working in the field of materials science. My contacts with NIOM revealed the same attitude.

## Effects of low exposure to inorganic mercury on psychological performance

L. SOLEO, M.L. URBANO, V. PETRERA, L. AMBROSI

### Abstract

The effects of low exposure to inorganic mercury on psychological performance was investigated: the study groups included eight chronically exposed workers and 20 who were only occasionally exposed. These were compared with a control group of 22 subjects from the same plant who were not exposed to mercury. All subjects were administered the WHO test battery to detect preclinical signs of central nervous system impairment: the battery includes the Santa Ana (Helsinki version) test, simple reaction time, the Benton test, and the Wechsler digit span and digit symbol. In addition, the Gordon test was used to study personality profiles and the clinical depression questionnaire. Urinary mercury was used as indicator for

internal dose. To this effect urinary mercury observed in workers examined from 1979 to 1987 was evaluated. Of the psychic functions exposed by behavioural tests, only short term auditory memory was found to be impaired in the chronically exposed workers ( $p < 0.05$  compared with the controls). The chronically exposed workers were also found to be more depressed than those in the two other groups. No changes of visual motor functions were observed. The personality of the occupationally exposed workers was found to be considerably changed compared with that of the control group. On the basis of the results obtained and in view of urinary mercury mean concentrations in the exposed group which were  $30\text{--}40\text{ }\mu\text{g/l}$  over the years, it is suggested that the TLV-TWA for mercury should be lowered to  $0.025\text{ }\mu\text{g/m}^3$  and that the biological urinary exposure indicator for biological monitoring should be  $25\text{ }\mu\text{g/l}$ .

## The Dental Amalgam Issue – Review

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**Keywords:** Dental amalgam, corrosion, mercury release, threshold values, toxicity, neurology, immunology.

### Abstract

Using an interdisciplinary approach, the current position in the dental amalgam controversy and the potential impact of amalgam mercury on human health are reviewed. Aspects of materials science, corrosion, mercury exposure, toxicology, neurology and immunology are included.

New data on mercury exposure from corroded amalgam fillings *in vivo* are presented. The exposure can reach levels considerably over known threshold limit values. Also, measurements of mercury absorption from intraoral air are presented. The vital importance of avoiding a galvanic amalgam-gold coupling is emphasized. The symptomatology of a disabled patient, who recovered after amalgam removal, has been included.

It is concluded that discussion of the dental amalgam issue has been suffering from the lack of an interdisciplinary approach. It would be wise to learn from the lesson of acrodynia, and consider amalgam mercury among other possible factors in neurological and immunological diseases of unclear etiology.

### 1. Introduction

Amalgams are, in general, alloys of mercury with other metals. At the beginning of the 19th century, the idea of filling carious teeth cheaply and simply with mercury alloys spread from France and England to the U.S.A. and other countries. The first dental amalgams (ca. 1818) were based on an alloy of bismuth, lead and tin, which had a melting point around  $100\text{ }^{\circ}\text{C}$ . Addition of a small amount of mercury decreased the melting temperature to  $68\text{ }^{\circ}\text{C}$  and thus also the suffering of the patient during the filling of the cavity.

In 1819, the chemist Bell started production of a silver-based amalgam. Around 1900, Black recommended an "improved" highsilver alloy, which continued to be used as a basis for the most extensively used conventional filling material (130). A pure copper amalgam was also in use until recently, often to fill children's teeth, in spite of its strong propensity to corrosion and related unfavourable performance and toxicity. In the 1970's, new copper-rich amalgams, called the non-gamma-2 type, have been introduced with the purpose of increasing the corrosion resistance of silver amalgam.

The use of amalgam for dental implant has been a subject of controversy throughout the 150 years of its extensive use. During the "Amalgam War I" (ca. 1830–1856) no dentist using amalgam was accepted as a

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member of the Society of Dental Surgeons in the USA. Due to lack of alternative cheap filling materials the use of amalgam continued and the "Amalgam War II" of the 1920's and 1930's reserved the debate.

In Germany the distinguished chemist Alfred Stock contributed to the debate with many works on amalgam and mercury toxicology and analytical determination, and also with a very informative description of his own chronic mercury poisoning (107, 111). In spite of its scientific background, Stock's warning that "the thoughtless introduction of amalgam as a substance for filling teeth was a grave sin against humanity" has not influenced the dental profession. Surprisingly, no epidemiological evaluation of health status, for example of diseases such as neurasthenia, has been made with respect to amalgam introduction 150 years ago.

The medical use of mercury also initiated frequent debates. The insidious toxic effects often were very difficult to differentiate from the symptoms of the disease for which the metal was administered. One of the first careful descriptions of the symptoms of mercury poisoning was an attempt by KUSSMAUL (64). The question was whether mercury poisoning produced symptoms distinctly different from those of syphilis for which mercury was the preferred treatment. The study was carried out in mirror factories, where exposures were generally high, but already Kussmaul noted that the sensitivity towards the metal was highly individual and unpredictable.

Today, extreme exposures as in the mirror factories are rare but they do sometimes occur because of ignorance or accidents. Instead, most persons in the western world are chronically exposed to lower levels from dental amalgam fillings, often from childhood. In addition, anyone may be exposed to mercury in vaccines, drugs, contact lens solutions, cosmetics and, of course, food. Thus no single source can simply be dismissed as "not more than a given level", but the sum of all sources has to be considered as a total load.

The degree of mercury exposure from amalgam has apparently been underestimated. A simple consideration of the amount of mercury in the teeth, compared to the daily intake from food, makes it apparent that amalgam would have to be an exceedingly stable alloy in order to contribute less mercury than the daily amount ingested with food. With 5 g mercury in the teeth (10 amalgam), the fillings would last 1370 years if the release is a maximum of 10 µg/Hg a day. 10 µg Hg is about what most people take in with food, if they do not eat too much fish. In southern Sweden the average daily intake was found to be 5.5 µg, but was 12.4 µg with food containing more than 75 g fish a day (27). Amalgam fillings will sometimes remain in the teeth for 2-3 decades, but the average lifetime is considerably shorter. The 10-year survival rate for fillings in adults ranged from 13 to 74%, in one study (78), others reported 50% replacement within 5 years and an average life span of 4-8 years (13). In 6 year old children the average survival time for occlusal amalgam fillings was 2 years and 2 months (132).

The terms "silver fillings" or "amalgam" do not give a layman or non-chemist the important information about the 50% mercury content. Inorganic mercury has insidious effects, not readily recognized unless one is aware of the symptoms of chronic mercury exposure. Proper diagnosis will be even more difficult if the exposure is from amalgam fillings. To quote again from STOCK (108), "The dentists are seldom in a position to recognize general effects of amalgam fillings or even learn about them. Patients suffering from nervousness, intellectual exhaustion catarrh etc. usually do not complain to the dentist, in addition they are prevented from talking during the treatment. They will rather discuss their problems with the family physician, neurologists, laryngologists and internists." The physician in turn is completely unaware of dental treatments, and does not suspect mercury from amalgam. Thus it is not surprising that reports on mercury poisoning from amalgam are relatively rare in the medical literature. However, in the daily press and in magazines there have been numerous descriptions of health changes, following amalgam removal.

There are many descriptions of the symptomatology of inorganic mercury intoxication, most of them written before interest was focused on methyl-mercury. Biochemists have also provided many studies on the cellular and molecular effects of mercury, providing explanations for many symptoms observed in clinical practice. Recently, the immunotoxic effects of mercury have attracted considerable attention. Nowadays, mercury is the best studied of the substances able to cause autoimmune disease. Immune reactions were also considered to be a factor in acrodynia of children observed after calomel (mercurous chloride) exposure. The acrodynia epidemic illustrates the puzzling nature of mercury intoxications. Even more puzzling is mankind's short memory after the discovery of acrodynia's Hg-etiology. "Man is



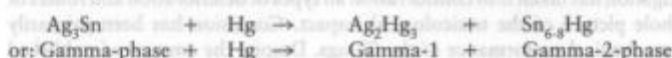
apparently a poor student of history" as the CASARETT & DOLL'S "Toxicology" says, referring to the use of mercury as a medicine.

In this review we will try to give an overall picture of the documented connections between mercury, especially in dental amalgams, and various possible neurological, immunological and orthomolecular effects. The fate of amalgam components other than mercury is not within the scope of this review. It is not possible to discuss every detail of this cross-scientific issue which touches not only odontology, but also materials science, toxicology, neurology, immunology, physical biology, analytical chemistry and diagnostics. The pertinent literature which we found to be available includes more than 8 000 titles. This comprehensive bibliography is available from the authors. As well as reviewing the literature, we include new data of our own observations on mercury exposure, related to symptomatology, similar to that of chronic mercury poisoning, as known from occupational and accidental exposure. Further, measurements of mercury vapour absorption in the oral cavity are presented.

## 2. Structure and Deterioration of Dental Amalgams

### 2.1 Structure

The conventional silver amalgam contains typically, in weight-%, 50 parts of Hg, 35 Ag, 10 Sn, Cu, Zn. The amalgamation reactions have been studied by ESPEVIK (35) and can be described by the equation



The reaction between the alloy powder (gamma) and mercury does not proceed stoichiometrically. Hence, both the composition and the amount of the structural phases in fillings varies depending on the overall composition, trituration time and insertion technique of the particular dentist. This is illustrated by the varying formulas reported for the gamma-1-phase:



The corrosion sensitive phase gamma-2 has been described as  $\text{Sn}_7\text{Hg}$ ,  $\text{Sn}_8\text{Hg}$ ,  $\text{Sn}_{10}\text{Hg}$  (35) and  $\text{Sn}_6\text{Hg}$  (74). The content of the gamma-2 may vary between zero and 59% (14), but 15–20% seems to be frequent (89). Normally, unreacted particles of the  $\text{Ag}_3\text{Sn}$  alloy are found after setting reactions. However, due to complicated and unpredictable setting reactions, free liquid mercury can also appear on polished surfaces of silver amalgam (99).

The preferential corrosion of the gamma-2-phase is partly responsible for the deterioration of conventional amalgam restorations. Therefore, attempts have been made to eliminate this phase by adding copper in concentrations 6–15% Cu (36). These high copper amalgams are called "non-gamma-2." The corrosion-prone phases in this system are  $\text{CuSn}$  (36) and the  $\text{Ag-Hg-Sn}$  phase (73).

### 2.2 Corrosion

Deterioration of various types of amalgam by electrochemical corrosion has been described in a large number of publications. The documented types of deterioration are:

- crevice corrosion (35)
- selective corrosion (56, 57)
- galvanic corrosion in contact with dissimilar metals (40, 65, 66)
- stress corrosion cracking (37, 75)
- general corrosion (75)
- mechanical wear (26)

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MATEER (75) investigated fifty extracted teeth containing amalgam restorations. Every restoration was attacked by corrosion, resulting in a surface layer of corrosion products. These observations have been consistently confirmed in the published literature and in clinical practice (36, 47, 100, 62, 120, 97, 89). Corrosion may have advanced so that the amalgam has lost most of its strength (56). For both mechanical and biological performance, avoidance of galvanic coupling to gold and other noble metals is of the utmost importance. Galvanic corrosion is also the only corrosion type which is possible to avoid. The other types of attack, listed above, seem always to be present to some extent. The corrosion mechanisms of the nongamma-2 amalgams differ from those of silver amalgam, but no clear model of their corrosion behaviours has been presented. The corrosion products of silver amalgam consist mainly of insoluble hydroxy-chlorides and oxides of tin (97). As the products have a larger specific volume than the original alloy, they partly substitute for the released mercury and help to maintain the mechanical function of the filling. The device between tissue and filling may occasionally become sealed (75, 131), but the voluminous corrosion products may also cause tooth cracking due to the high internal pressure (71).

### 3. Mercury Exposure

A reliable knowledge of the exposure level is a prerequisite for any meaningful conclusion about the health hazard of amalgam in vivo. The traditional opinion of the dental profession during 150 years has been that amalgam becomes biologically inert as soon as it is set; i.e. in a few days (95). A survey of available data on metal release from dental restorations was published in 1986 by BRUNE (16). In the rather sparse information available, no investigation has taken into consideration all types of deterioration and routes of uptake, in order to get the whole picture of the toxicological impact. Corrosion has been primarily considered with respect to the mechanical performance of the fillings. Despite the amount of published information about corrosion of amalgam, the toxicological impact of the released mercury has been discussed very little. Therefore, the section on mercury exposure has been devoted comparatively large attention.

The lack of an interdisciplinary approach has tended to lead to underestimates of the mercury released from amalgam fillings in vivo. The work by MAYER and DIEHL (76) provides an example. The investigators used oxygen-free distilled water for measurements of mercury released from amalgam. The elimination of any cathodic depolarizer, such as oxygen from the air, effectively removed the possibility of any corrosion process and led to a wrong conclusion about the high corrosion resistance of amalgam under conditions where oxygen is present.

The dissertation by FRYKHOLM (43) has been used by dental professionals as a proof of the insignificant contribution of amalgam to the body burden of mercury. Frykholm did find increased urine mercury, and migration into dental pulp, days and weeks after insertion of amalgam, but he classified the amounts as insignificant. Frykholm's anticipation, that the exposure to mercury released in mouth can be related to mercury found in the excreted urine does not find sufficient support in the literature. Until now, no defined partition of mercury between that excreted and that deposited in various organs has been observed, so it is not possible to judge the amount of dental mercury released from measuring the urine concentrations (see section on mercury in blood and urine). Therefore diagnosis cannot be based on urinary mercury determination. Frykholm's investigation of exposure during and a short time after insertion may not be relevant to long term mercury release after the onset of corrosion in vivo.

MAREK (72) observed up to 56-fold (average 23-fold) increase of electrical corrosion currents during abrasion of five brands of fresh amalgams. According to Faraday's law, the measured corrosion currents corresponded a daily release of 60 to 735 micrograms mercury from 1 cm<sup>2</sup> of surface, assuming a total daily chewing time of 2 hours (89).

In vivo, the two processes of corrosion and wear are interdependent (15), corrosion being strongly promoted by mechanical wear. To illustrate the possible toxicological impact of the volume of worn amalgam, it is of interest to consider the role of the wear process alone. DELONG (26) studied the wear rates in an artificial mouth, and found a correlation with clinical observations. At points of contact with enamel,

a layer about 65  $\mu\text{m}$  thick was worn away after 250 000 masticatory cycles per year. An assumption of a total contact surface in the mouth of 1  $\text{cm}^2$  then gives 6.5  $\text{mm}^3$  of worn amalgam, containing 37 000  $\mu\text{g}$  mercury. As the tiny particles will be exposed to dissolution in the acidic stomach environment, the individual might be exposed to 101  $\mu\text{g}$  Hg a day.

The most complete documentation of total mercury exposure and uptake up to now is that recently published by HAHN (48) and VIMY (127). The radioactive labeled mercury from 12 fillings in sheep was determined by whole-body scanning and measurement in specific tissues. Mercury appeared within 29 days in various organs and tissues and in fetuses. The investigation confirms the three main ways of Hg-uptake; in the lung, the gastrointestinal tract and through jaw tissue absorption.

After one month, the total Hg content of the kidney was 1.86 mg and that of the liver 0.77 mg. Also the brain, pituitary, thyroid pancreas and ovary glands showed evidence of Hg accumulation from dental amalgam. In addition to the deposited mercury, the sheep eliminated daily over 9 milligram of Hg in 2 000 grams of feces. The order of magnitude is in reasonable agreement with the released amounts of mercury computed from Faraday's law and in vitro current measurements (89, 72). With 2 hours chewing a day, 500–700  $\mu\text{g}/\text{cm}^2$  will be released from fresh conventional amalgam, i.e. 5–7 milligrams from 10  $\text{cm}^2$  surface.

Hahn's findings of dental mercury in tissues are consistent with analyses of mercury in autopsied human brains and kidneys by NYLANDER (82, 83). Subjects with amalgam fillings showed significantly increased Hg-contents in the pituitary gland and the concentrations were related to the number of amalgam surfaces.

### 3.1 Mercury vapour

An amalgam bearer is exposed to a daily amount of mercury released from the fillings in the form of vapour, liquid metal and ions. A part of the released amount will be taken up by the body. The main ways for uptake are:

- as metal and ions from the bottoms of fillings (120) and through the mucous membranes,
- as vapour in the lungs (86, 51),
- in all forms in the gastrointestinal tract,
- by direct transport from the oro-nasal cavity to the brain (110, 117).

The assay of mercury vapour  $\text{Hg}^0$  in the expired air of amalgam bearers is experimentally feasible, and it has been measured in a number of investigations. Stock demonstrated in 1926 that dental amalgam fillings generated mercury vapour in the mouth (107). Chewing and abrasion (41, 44, 118, 125), brushing (86) and increased temperature (121) strongly stimulated corrosion of amalgam and evaporation of mercury. SVARE (118) measured on average a 15.6-fold increase of mercury in the expired air after chewing, compared to amalgam-free controls. The mercury content in exhaled air or in the oral cavity after chewing can exceed permissible industrial levels (86, 118, 125). Stock's results inspired BRECHT-BERGEN (14) to measure the Hg-vapour pressure over amalgam:

Ag/Sn/Hg-alloy with 45% Hg:	10.7% compared to pure Hg
Ag/Sn/Hg-alloy with 54% Hg:	25.7% compared to pure Hg
Sn/Hg-alloy with 45% Hg:	54.7% compared to pure Hg

Measuring the concentration of Hg in expired air or in the oral cavity leads to difficulties in calculation how much Hg is actually inhaled. ABRAHAM (1) introduced flushing of the mouth for 15 sec, measuring the evaporation rate from the fillings. A pre-chewing evaporation during 15 sec was found to be 1.0–11.8 nanogram (ng), i.e. 0.007–0.8 ng/sec with a mean of 2.24 ng (0.15 ng/sec). After 3 min chewing, the emission was 1.2–162.7 ng (0.08–10.8 ng/sec) with a mean of 18.97 ng (1.27 ng/sec). BERGLUND (9) found 0.16 mg/sec (range 0.04–0.34) for unstimulated Hg-evaporation. The actual values after chewing might be even higher than those by Abraham since the vapour levels continue to increase during 30 min of chewing (126). Thus the evaporation rates from amalgam fillings in the oral cavity can reach 11 ng/sec $\cdot\text{cm}^2$  at room temperature and maximum air flow (113). This will correspond to about 6 ng/sec $\cdot\text{cm}^2$  at

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the oral temperature. Assuming an amalgam surface of  $10 \text{ cm}^2$ , the highest values recorded by Abraham will correspond to the vapour pressures measured by BRECHT-BERGEN (14).

KNAPPWOST (60) measured the evaporation rate of mercury from  $0.5 \text{ cm}^2$  amalgam in artificial saliva under static conditions. The evaporation levels were  $0.66 \times 10^{-3} \mu\text{g/sec}$  without, and ten times as much with contact to gold. From amalgam alone, the evaporation rate corresponds to an average exposure of  $57 \mu\text{g Hg}$  a day. From 10 fillings of  $0.5 \text{ cm}^2$  surface the exposure will be several hundreds micrograms a day. This is in good agreement with our own evaluation based on depth of corrosion (89, this paper).

The evaporation depends on the vapor pressure, the flow of air over the surfaces, abrasion etc. The actual inhaled and absorbed amount of Hg is difficult to determine. However, nose versus mouth breathing does not seem to have a central role. To test for a possible direct oral absorption, we infected known amounts of mercury vapour into the closed oral cavity of amalgam, and gold-free subject with no detectable mercury emission from lungs or oral cavity. After 0-3 min, air with the remaining mercury was sucked out and the mouth flushed with 30 ml of Hg-free air. An immediate extraction - in reality 3-5 sec - showed an absorption of  $5.5 \pm 4.5 \text{ S.D. ng Hg}$  ( $n = 6$ ). Delayed extraction (1.3 min) resulted in steadily less Hg recovered. In one series of experiments, 2.4 ml samples of Hg vapour from the mercury-saturated air over metallic Hg were injected. Mean absorption was  $10.2 \pm 2.1 \text{ S.D. ng/min}$  ( $n = 12$ ). To obtain information on absorption kinetics, increasing amounts of Hg from 32 to 120 ng were injected and extracted after 1, 2 and 3 min (a total of 10 injections). The results indicate exponential absorption kinetics (Table 1) and that most of the mercury vapour, generated from fillings in the mouth might be absorbed even when chewing is done with closed mouth and nose breathing. The further fate of the absorbed mercury is unknown. FREDIN (41), using a somewhat different approach, placed an inverted cup on the buccal oral mucosa and introduced known amounts of mercury vapour. The results were similar to those described above. Also HAHN (48) reported high mercury content in the gum mucosa ( $323 \text{ ng Hg/g}$ ) of sheep with amalgam fillings.

### 3.2 The present authors' data on exposure

Twenty used restorations were investigated using a Scanning Electron Microscope (SEM, JEOL 940), equipped with an Energy Dispersive X-Ray Analytical device (EDX). The donors of the restorations belonged to a group of 250 patients with chronic health problems of unknown etiology. Information on age, sex, profession, medical history and dental treatments was obtained by a questionnaire. About 50 of these patients who had removed all amalgam fillings (with initial aggravation of symptoms and after months up to a year their alleviation) constituted a test group for possible mercury etiology. Patients who did not remove amalgam fillings served as one control group and 10 persons who had never had any such fillings as a second one. Persons with possible occupational exposure to mercury and those consuming fish from inland lakes more than twice a week were excluded from the investigation. A detailed evaluation of the parities will be published elsewhere.

Eighteen patients succeeded to recover pieces of large amalgam fillings, whole teeth with amalgam or gold restorations in direct contact with amalgam. All were microscopically examined. Although the type of corrosion attack was similar in all investigated amalgam fillings, the severity of corrosion varied between specimens, probably depending on filling quality, age of filling, saliva composition and eating and chewing habits.

In one case a general corrosion of the amalgam surface was found. This caused total release of mercury from a layer of  $100 \mu\text{g}$ , that is 68 milligrams in 9 years, i.e.  $29 \mu\text{g Hg}$  a day. Additionally, selective corrosion throughout the amalgam is estimated to have released at least another  $30 \mu\text{g Hg}$  a day.

### 3.3 Case history

The bearer of the restorations before mentioned was a Swedish woman, 58 years old, a telephone operator, retired at age 40 because of disabling chronic health problems. The syndrome included 24 symptoms of

mainly somatic character: diarrhoea with strong bleeding, joint-, muscle-, and back pains, frequent inflammations in eyes and upper respiratory passages, sinusitis, severe fatigue, stress, loss of memory, vertigo, impaired hearing, increased salivation, bleeding gums, asthma, irregular heart-beat, heart and chest pains, disturbed sleep, eczema.

The official diagnosis was Morbus Crohn with rheumatic and allergic affliction of intestines (organic enteritis), joints, eyes and upper respiratory passages. The etiology was not known. Dental status: since childhood amalgam fillings in most teeth; a short time before the severe disease manifestations she was given gold restoration which were in contact with amalgam.

When interviewed in 1990, four years after amalgam removal, she reported amazing recovery with only 4 weak symptoms left: fatigue, eczema, occasional diarrhoea and impaired concentration ability. The woman was one of the 250 patients (see 3.2) with similar symptoms. Three other cases are described in (89).

### 3.4 Comparison to literature data

Comparison of the above results with published investigations confirms the considerably high mercury release under realistic conditions (15, 48, 60). The common denominator of many published investigations is the use of fresh amalgam, i.e. amalgam which is only at the start of its deterioration process (15, 36, 40, 62, 72, 76, 99). The daily release of 9 milligrams Hg, found by HAHN (48) in feces of the sheep one month after insertion of 12 fillings would have consumed all the mercury in less than 2 years. As this rate of loss has not been observed in practice, either the release must decline after some months, or the filling will be loosened and be replaced by a new one in a relatively short time.

Considering the strong variations of the Hg-content in the faeces of amalgam bearers (12, 112), mercury excretion due to oral corrosion may vary appreciably. STOCK (112) reported daily amounts between 10 and 56 µg Hg in feces and urine, compared to the average 3.5 µg Hg of controls. No data about number and size of the fillings are given.

BORINSKI (12) also states that excretion (urine+feces) of 5–10 µg Hg a day is frequent in amalgam-free subjects not exposed to mercury except from food. Borinski discovered the ubiquitous occurrence of mercury. Excretion over 10 µg indicates an exposure level considered to be potentially harmful. After insertion of an amalgam, strongly increased excretion of Hg has been observed during some months, whereafter the excretion decreased. During the first three months, about half of the test children showed a total daily excretion in the region 10–100 µg, the other half over 100 µg Hg. From Borinski's text it is apparent that most of the test subjects were treated with one filling only. From the above, and from KOZONOS (62) investigations, it is apparent that the mercury release is highest in the first months after insertion and decreases with time. The decrease of the decay depends on the build up of corrosion products, impeding the transport of mercury and other species between the inner parts of fillings and a non-abraded surface. Also the decreasing concentration of mercury in the filling will lower its diffusion rate to the surface. However, after a longer time the metal release may increase again (62).

The Swedish "standard man" for discussion of dental restorations has been defined as an adult age 40–50 years, with sixteen amalgam restorations with normalized occlusal geometrical surface area of 10 cm<sup>2</sup> (84). A part of the hidden surfaces (bottoms of fillings) is subject to crevice corrosion. Therefore, to obtain the total surface releasing mercury, the occlusal surfaces need to be multiplied by a factor larger than 1.

### 3.5 Mercury in blood and urine

"Normal" values for mercury in blood and urine (B+U) were found in the sheep with amalgam fillings, whereas the tissue and feces levels were high (48). The first reliable measurements of Hg in B+U demonstrated that blood values remained low until the exposure level became high. Little increase was found in the urine, but much higher levels in the feces (112). Some studies show that amalgam fillings do have a certain effect on blood Hg-levels (105) and that removal of amalgam fillings reduces the Hg-level (105). Other studies found no difference (63). Amalgam placement causes a transient peak of mercury in



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the urine (43, 98, 114). Human experiments with single doses of swallowed radioactive ionic or inhaled elemental mercury show that 1–2% of the absorbed dose is eliminated in the urine in the course of a week after the exposure (21, 91). The observed urinary excretions after amalgam placement thus corresponds to several milligrams of total absorption. Recent studies show, that the number of amalgam fillings is positively correlated with urinary mercury (69). Few measurements of fecal mercury levels have been made. TOMPSETT and SMITH (122) found levels of 50–180 micrograms a day, without considering amalgam fillings as a source. LAMM and PRATT (67) found a significant negative correlation between exposure time of workers and urinary excretion of mercury, indicating impaired urinary excretion of the metal. The investigators found a positive relation between time at work and subclinical neurological damage. Administration of dimercaptopropane sulphonate (DMPS), a heavy metal chelator, produced a massive increase of mercury excretion in amalgam exposed patients with low, "normal" Hg levels in urine (25). In the Soviet Union, the substance is used for diagnostic purposes. An eight times increased urinary Hg-excretion after chelation, is considered to be indicative of adverse Hg-effects when symptoms of "micromercurialism" are present (42).

There are several uncertainties regarding the use of B+U Hg-levels as diagnostic tools. Most industrial studies relate the B+U-levels to the percentage of affected workers, irrespective of the severity of symptoms. The adverse effects of long term exposure to low levels of mercury – "micromercurialism" – can be completely devastating for the affected individual, as was vividly described by Stock in 1926 (107).

Detailed investigations on single blood cells with proton-induced x-ray emission equipment demonstrated that only some of the thrombocytes and red and white cells in patients with suspected amalgam poisoning contained mercury (4). In a healthy control population the mercury levels in all cells were below the detection limit. Only one controlled study on animals has assessed blood-Hg levels in relation to mercury levels in inhaled air (34). Blood Hg was found to relate exponentially to the exposure level. Assuming that a similar situation exists in humans, blood Hg levels would be expected to show moderate changes within a broad range of levels of exposure.

Chlorine gas reacts spontaneously with Hg<sup>0</sup> vapour in air (46). Already in 1904 it was recognized that the best method of protecting workers in mercury mirror factories was to maintain a low chlorine content in the air (24). The elemental mercury is then oxidized to the much less toxic mercurous chloride (calomel). VIOLA (128) and VIOLA and CASSANO (129) found precipitated calomel in mercury-using chlorine factories. They also studied rats exposed to mercury and vapour in comparison to exposure to air with a mixture of mercury and chlorine. Exposure to the mixture in the same proportions as in the chlorine factories but at higher levels, was strikingly less toxic than exposure to Hg alone. Whereas the rats exposed to Hg only suffered severe neurological symptoms, those exposed to Hg + chlorine only showed mild gastrointestinal disorders. The difference in symptomatology can be explained by the fact that the brains and hearts of the rats exposed to both species had only 8–10% of the levels found in rats exposed to Hg only. Total body uptake in the doubly exposed rats was 60% compared to Hg-only rats.

#### 4. Amalgam Poisoning

Mercury poisoning from amalgam fillings have been described several times. STOCK (109) relates cases with devastating psychic effects, and also aggravated symptoms when fillings were drilled out without suction. Further cases have been described by STOCK (109, 111). FLEISCHMANN (39) reported that conditions for poisoning were present in carriers of copper amalgam fillings (as judged from the Hg-values in urine and feces), whereas no conclusion could be reached for silver amalgam. Fleischmann found that the disappearance of symptoms after the removal of silver amalgam indicated that poisoning could occur. HARNDT (49), studying the same patients, considered patients with gold in contact to amalgam as self-evident poisoning cases since the enhanced corrosion could clearly release enough Hg. Other cases have been presented by WESSELHAFT (136), HYAMS (52), STEFFENSEN (106), LAIN & CAUGHNAN (65), STRUNTZ (115), ROST (94), ZAMM (137), PLEVA (88) and DAUNDERER (25).

Effects of amalgam removal or placement on T-cells in 3 patients (one with multiple sclerosis, MS) were measured by EGGLESTON (33) and the acute exacerbation of JS-symptoms during removal (pulverization) of one old filling was reported by INGALLS (53). TASKINEN (119) followed a patient who had fillings ground to

form a bar to support a bridge. In addition, eleven fillings had about 1 mm ground away to improve occlusion, and three fillings were replaced during the following session. After a week, the patient developed stomatitis, a sore throat, a rancid taste in the mouth, loss of the sense of smell, dizziness and headache, and later pains in the thorax, fever, elevated sedimentation rate, weakened sense of touch in her left hand, sensitivity to cold in fingers, and weakened hand grip. The patient felt rather bad, lost 9 kg of weight and became anxious and depressed. The fillings have been removed with extreme caution. The authors find that the symptoms correspond to those of micromercurialism.

Anorexia hydrargyria was described in a 15 year old girl (31), who developed headache, joint pains, vertigo, loss of memory, fatigue, sleep disturbances and hair loss. Lack of appetite led to loss of weight and symptoms of anorexia nervosa. There were, however, no psychic problems. The physician noticed that the patient's mouth contained 10 glittering amalgam fillings. At an early school age, the patient had received 6-8 fillings with no effects on her health. In 1986 the fillings were all replaced by new ones, and new ones added. The girl was treated with dimercaptopropane sulphonate and the fillings removed. This treatment brought about a complete recovery. The author of the report considers the current toxicity evaluation by the dental authorities as insufficient. Diffusion of mercury through the pulp, the number and quality of the fillings and the toxicity of amalgam for pregnant women, children and adolescents have not been taken into consideration.

Studies of the relationship between the number of amalgam fillings and impairment of health do not show any difference. AHLQWIST (3) studied 1 024 women, aged 38-72 years. No difference was found between persons with 1-4 amalgam fillings and persons with more, when the number of teeth was used as a corrective variable. Time factors were not studied, neither was there any information on gold-amalgam combinations or other types of dental restorations. It is not likely that middle-aged persons in Sweden will have many intact teeth. LAVSTEDT and SUNDBERG (70) could not find any correlation between the prevalence of mercury-related symptoms and the number of amalgam surfaces. HUGOSON (50) found only a few cases among 100 patients whose symptoms could be attributed to reactions to dental restorative materials.

In a study by JONTILL (54), 62 patients with "oral galvanism" were studied. The authors conclude that most symptoms could be ascribed to psychosocial factors. Blood mercury measurements showed no differences between the patient and control groups. MÜLLER-FAHLEBUSCH & WÄHNING (80) also ascribed the symptoms of 50 patients, who suspected amalgam poisoning, to psychogenic factors. Removal of amalgam led to an aggravation of the symptoms in 28 of 29 cases. Three of the patients had new, allegedly improved, amalgam fillings. There is no information on protective measures during the amalgam removal, whether the aggravation was immediate or on later changes in health. In one county in Sweden, 248 patients were referred to the specialist dental clinic for possible amalgam related problems. They have been questioned after 1/2 to 3 years about treatment and possible health insults. Most of the patients had exchanged the amalgam fillings. Total amalgam removal gave significantly better results than other types of treatment or no treatment, the former having hardly any effect at all. Improvements ranged from 85% for oral symptoms, 75% for headache to 66% for psychic problems. The authors consider the improvements real. However, they refer them to placebo effects, since so many symptoms disappeared (59).

## 5. Mercury Toxicity and Symptomatology

The toxicity of inorganic mercury has been described several times in the course of centuries. The most common form of exposure is inhalation of vapour. There is general agreement that inhalation leads to a slowly developing and insidious poisoning which primarily produces psychic effects, and is very difficult to recognize until more objective symptoms appear. Numerous more or less extensive descriptions can be found in the literature. BAADER (5) has described the major symptoms:

Stomatitis, gingivitis, loose teeth, salivation or dry mouth, foul breath, metallic taste, redness in the throat, black line along the teeth, diarrhea, speech problems, nephritis, anemia, relative lymphocytosis, pressure over chest, irregular heart, circulation disturbances, low blood pressure, increased sweating, disturbed sleeping, tremor, shaky handwriting, dull pains in lumbs and joints, fatigue, headache, anxious seclusion, uncertainty, shyness, labile mood, agony, forgetfulness, feeling of intellectual exhaustion, sensory



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disturbances of skin, irregular menstruation, thyroid disorder, eye pigmentation, eczema. Common misdiagnoses: neurasthenia, hysteria, schizophrenia. Other symptom descriptions can be found in SCHULZ (101), BURGNER (17), OETTINGEN (85), NORDIN (81), MOESCHLIN (79) and POULSON (90).

In addition to the general symptoms of mercury poisoning there are numerous reports on individual cases of less common forms of poisoning, which are likely to pass unnoticed in industry. The largest such study involved only about 600 workers in the chlorine industry (104). The types of symptoms depend on the mercury compound and the mode of administration. An amyotrophic lateral sclerosis-like (ALS) syndrome has been reported after exposure to ethylmercury (58), mercury vapour (2) and inhaled mercuric oxide (8). REDHE (92) described the complete recovery of a 29 year old woman with an official ALS-diagnosis, after all amalgam fillings had been removed.

Mercury-neurasthenia has been known for a long time. Various forms of paralysis, affecting different parts of the nervous system and diagnosed under different names have been reported: polyradiculoneuritis, Guillain-Barré and multiple sclerosis (6, 11, 64, 138). If the mercury exposure is recognized and interrupted, most cases recover, some slowly, but many often surprisingly rapidly. When the mercury etiology of acrodynia was clarified, the possibility that MS was an adult form of acrodynia, and a neuroallergic reaction, was considered. BAASCH (7) recognized the possibility that amalgam fillings could be the responsible source of Hg. He concluded that the amalgam mercury etiology could explain the known facts about MS. Baasch noted the presence or absence of amalgam fillings in 500 consecutive MS patients in Zürich. All but one (or possibly two) had amalgam fillings. However, amalgam fillings are common, so this finding proved nothing. On the other hand, there are also other sources of mercury. Three cases were described by Baasch. Two of these had their amalgam fillings removed and the patients improved. The third one showed a rapidly developing disease, starting a few months after she got her first amalgam fillings at 19 years age. When 8 years old she had been treated with mercury for congenital syphilis.

KNOLLE and GÜNTHER (61) described the mercury/amalgam status of 100 MS patients. Eleven of these had previously been treated with mercury ointments. Seven had no teeth, and the percentage with amalgam did not differ from the general population. The high percentage of the mercury-treated patients seems to be remarkable. STUTTE and GROH (116) discuss an acrodynia case with paralysis, caused by the rectal administration of metallic mercury in vaseline, and the suspected mechanisms behind this kind of "idiosyncrasy." The proposed sequence of events in acrodynia was suggested to be an initial attack by mercury on the blood-brain-nerve endothelial cells with a secondary immune reaction to brain components.

Oral or subcutaneous mercuric chloride in rats was found to cause a long-lasting but not permanent impairment of the blood-brain barrier with extravasation of plasma components (19), demonstrating that acute or subacute exposures could expose the immune system to neuronal antigens.

STOCK (107) published one of the few descriptions of how it feels to be poisoned by mercury. He emphasized the psychic effects which were especially troublesome for an intellectual. In addition to a number of somatic symptoms, Stock mentions: "Intellectual exhaustion and depression, lack of energy and working ability, especially for intellectual work, increased need for sleep. For a person with intellectual work, the loss of memory was the most severe burden. Especially the ability to calculate and to perform mathematical thinking, also to play chess, was severely affected. The lowered ability to remember and the difficulties in calculating seem to be a special sign of insidious mercury vapour poisoning. The intellectual capacity was depressed in other ways as well although not as much as the memory. In addition there was psychic depression and painful inner restlessness, with time causing disturbed sleep. By nature fond of company, and enjoying life, I withdrew depressed into myself, avoiding public relations, people and social contacts. I lost the love for art and nature. Humor became rusty. Difficulties, previously cleared with ease, (as they are again today) appeared insurmountable.

Scientific work required considerable effort. I forced myself into my laboratory but could not produce anything of value despite all efforts. My thoughts were heavy and pedantic. I had to give up participating in matters of no immediate importance. The lectures, previously something I liked, became tormenting. The preparation of a lecture, the writing of a paper, even a simple letter required immense efforts in handling the contents and language. Not seldom I happened to write words in the wrong way or forgot letters. To

be aware of these shortcomings, without knowing their cause, seeing no way to get rid of them, expecting further aggravation – that was not nice!”

Three cases among dentists have been described by SMITH (103). The first dentist had hand tremor, impaired motor control, indifference towards family and friends and a visual disturbance. The subjects experienced irritability, critical excitability, fearfulness, restlessness, melancholy, depression, weakness, timidity, fatigue, indecisiveness and headache. The dentists emphasized that the mental effects of mercury poisoning were most distressing and frightening. Being deeply affected by the feeling of hopeless situation, depression and futility, they urged the physician to bring the cases to the attention of the medical profession.

## 6. Mercury and Immunology

Mercury is well known to be immunotoxic, inhalation or swallowing mercuric chloride or methylmercury in doses comparable to industrial exposures or intake in food, results in the same systemic autoimmune reactions which occur after subcutaneous administration in susceptible rat strains (10). The autoimmune disease is characterized by antibodies to a variety of proteins, mainly of endothelial origin (96). Outbred animals show a more complicated response (282, 93). The effects on the immune system are thought to be mediated by interaction between mercury and T-cells. The result is a genetically determined, polyclonal activation of B-cells (87). The Hg-induces autoimmune disease shows the same types of autoantibodies as in a number of human diseases, graft vs. host disease and reactions to some drugs (32). Long-term exposure of rats and rabbits to low levels of Hg-vapour, 6–10 µg/m<sup>3</sup>, for 6 hours a day, caused first a stimulation of the immune response and a decline after months (123). TRACHTENBERG finds that the immunological changes occur significantly earlier than other signs of latent toxic effects.

A recent study on human granulocytes demonstrated that exceedingly small amounts of Hg are required to stimulate oxygen free radical production from neutrophils (PMNS) in vitro and to depress various other PMNS functions (23). The radical production was in at 10–17 µ, reached a maximum at 10–13 µ and almost disappeared at 10–17 µ concentration. In vivo, the presence of sulfhydryl groups in blood proteins might change the figures considerably by non-specific binding of Hg. Also, free radical production and DNA single strand breaks in hamster ovary cells, induced by mercury, showed a distinctly nonlinear response (18). Release of oxygen free radicals is suspected of mediating the pathological effects of adverse drug hypersensitivity reactions (124).

Skin hypersensitivity to mercury has been reported to occur in up to 26.6% of the population (29). There is a limited number of described cases of skin reactions caused by amalgam fillings. There is no indication that skin tests for mercury would give any information on systemic or immunotoxic effects of mercury. Stock, who was hypersensitive to traces of inhaled mercury, showed no positive skin reaction. Children with acrodynia seldom gave a positive reaction (134). FANCONI (38) observed that in children who were exposed to calomel parenterally, a positive skin reaction could sometimes be shown when the calomel-disease was at its peak. A few weeks later, a new test was negative. Children with a negative patch-test could develop a flare-up at the site of the previously negative Hg application when they accidentally swallowed another dose of calomel. Some children developed systemic hypersensitivity after calomel treatment, but some became more resistant to a second dose.

BAADER & HOLSTEIN (6) report that occupational exposure leads to increased sensitivity toward further exposure. The acquired “idiosyncrasy” is often the only permanent effect of mercury poisoning. STOCK (110) describes the slow development of mercury sensibilisation: “To provoke a first reaction to mercury vapour, a stronger and longer exposure is required than what is needed for following exposures. Later on, symptoms can appear within an hour after exposure to much lower levels. A further exposure is avoided, the sensitivity slowly disappears, more so if the poisoning has been severe and prolonged. Recovery can take years.”

Mercury has a strong affinity for sulfhydryl groups and it is generally assumed that inhaled mercury is rapidly ionized in the blood and binds to SH-groups of enzymes. Exposure of animals or humans will lead to a measurable reduction of free tissue and blood sulfhydryl groups. A simple estimate, however, will lead to the conclusion that even with fatal kidney levels of Hg there will not be enough metal to bind to more than 10% of available sulfhydryl groups (22). Glutathione levels alone are in the millimolar range. A

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catalytic oxidation of sulfhydryl groups by one mercury ion seems to be a plausible explanation. Mercury is known to release calcium from mitochondrial stores (20), an effect of free radical chemistry known to involve oxidation of thiol groups (102). Glutathione reductase is inhibited up 15% by 13 ng Hg/ml ( $67 \cdot 10^{-9}$  M) and myelin phosphodiesterase up to 40% by 100 ng Hg/ml (30, 135). The levels of mercury in the brains of humans with amalgam fillings is proportional to the number of fillings, and is within the range mentioned (82).

## 7. The Acrodynia Lesson

The best studied cases of human mercury poisonings come from the now almost forgotten disease acrodynia. This disease, mostly affecting children, was first recognized in France in 1828. It reached epidemic forms in certain parts of the world. The skin on children's fingers and toes was particularly affected and peeled off. The skin appearance gave the illness the name "pink disease." The patients suffered from pains in the limbs and circulation troubles, and in extreme cases fingers and toes could be lost. Extreme weakness of the muscles, weight loss, sleep and gastrointestinal disturbances, tremor, chorea, sometimes fever and conjunctivitis belonged to the clinical picture. Some patients developed salivation, loose teeth and necrosis of the jaw bones. Thousands of children died. Many causes have been suggested: lack of vitamins, endocrine disorders, allergy, hysteria, neurosis, mould poisons and viruses. Mercury etiology was first suggested in 1846. Again in 1922 a physician pointed out the similarities with mercury poisoning. In 1945 WARKANY (133, 134) found that in almost every case of acrodynia, mercury exposure could be demonstrated. The most common exposure sources were teething powders containing calomel and calomel-containing preparations against gastrointestinal parasites.

WARKANY and HUBBARD, in 1953 (134), and WARKANY in 1966 (133), pointed out that although the administration of these mercury preparations was frequent, only a fraction of the exposed children developed acrodynia. They calculated the incidence to be about one in 500. Mercury excretion was often found in controls as well, although no symptoms were present. Despite the exposure, mercury was not always detected in the urine samples of acrodynia children. However, careful studies showed mercury exposure in every case. WARKANY notes: "It seems rather odd that one could not detect the injurious mercury at the entrance to the alimentary canal, whereas it could be demonstrated at the end of the urinary tract."

Pseudo-acrodynia with some features of the real disease was produced by different treatments in animals. However, "a subtle, complicated, and no doubt molecular disease was eradicated by such a prosaic measure as removing calomel from old-fashioned teething powders and worm medicines. There were data on electrolyte changes, explaining the symptoms of acrodynia and their alleviation by subtle saline treatments. But these data did not take into account the one electrolyte that mattered, namely mercury" (133).

## 8. Threshold Limit Values – How Much Mercury is Too Much?

Safety evaluations for inorganic mercury are based on industrial observations, and for amalgam specifically, on estimates of the exposure levels from amalgam compared to the industrial MAC-values (Maximum Allowable Concentration, comparable with the concept of Threshold Limit Values, TLV, used in the U.S.A.). In the U.S.A., most government agencies recommend a maximum of  $50 \mu\text{g Hg/m}^3$  in industry, based on the study of SMITH et al. (104). A review of the subject (139) shows that the U.S. EPA (Environmental Protection Agency) National Emission Standard of  $1 \mu\text{g Hg/m}^3$  for mercury as a hazardous air pollutant is the only air exposure standard based on studies of the effects of mercury on the general population, and not limited to a 40-hour week. In the Soviet Union, the corresponding value is  $0.3 \mu\text{g Hg/m}^3$ , based on thorough studies of chronic mercury exposures by TRACHTENBERG (123).

As shown in Table 2, estimates of safe industrial exposure levels vary between different countries, the safety limits for the general population are 30–50 times lower than the industrial limits.

The MAC-values are defined as: that average concentration in the air which causes no signs or symptoms of illness or physical impairment in all but hypersensitive workers during their working day, on a continuous basis, as judged by the most sensitive internationally accepted tests (77). Hypersensitivity is not

stated to be due only to skin reactions. No regulation for any mercury compound considers additional exposures or possibly reduced protection by low dietary levels of selenium, zinc and other essential substances, which are known to counteract mercury effects. The occupational TLV's are not relevant for the general population for the following main reasons; There is (or should be) regular medical control of occupationally exposed workers and suspected mercury-related problems can be alleviated by changing the working conditions. TLV's for mercury are based on studies in the electrolytic chlorine industry where the workers are to some extent protected by the oxidative effect of chlorine on mercury vapour (see section on Hg in blood and urine). The TLV's may therefore be too high for continuous exposures in chlorine-free air. For the general population, it is also necessary to consider individual sensitivity, chronic diseases, and effects on children, pregnant women and elderly people.

## 9. Conclusion

The puzzling insidious biological effects of various forms of mercury are often difficult to recognize when correct data on mercury exposure are not considered. Besides the diagnostic difficulties, the dental amalgam debate has been affected by the lack of an interdisciplinary approach. Despite a large number of specialized papers on amalgam, very few attempts have been made to relate clinically observed deterioration and wear of amalgam restorations with metal release estimated from such studies. Corrosion currents, measured in vitro, have not led to the obvious use of Faraday's law to calculate the corresponding metal release but instead to a strange discussion on "oral galvanism" and possible biological effects of the generated currents. With known facts and available scientific and technical methods, estimated exposure levels for mercury will be higher than previously thought. The conclusion is that such levels could have considerable toxicological and immunological consequences. A review of the controversy indicates a serious deficiency in the efforts of the dental profession to test the published warnings, for example those of Professor Stock, and to bring clarity into the issue.

Recent investigations on the fate of amalgam mercury, using whole body image scans (48) show, that in sheep mercury release of up to one milligram a day from one filling is possible during the first months after insertion. Sheep were chosen as experimental animals since they use moar chewing as do humans (48). Some humans use chewing-gum or exhibit bruxism and might abrade their fillings as much as sheep fed two meals a day. Further, investigations of amalgam restorations after a known time in vivo show that the magnitude of mercury release can reach levels several times higher than accepted threshold values. The corrosion process may not be apparent to the eye, as it is a matter of amalgam conversion rather than dissolution. The extensive published knowledge about mercury toxicology indicates that toxic effects from amalgam mercury cannot be excluded. Immunological disorders can appear at considerably lower exposures than those from dental amalgam. Comparison with occupational threshold limits for mercury suggests that amalgam bearers should be under regular medical control in the same way as mercury-exposed workers.

The comprehensive bibliography with over 8 000 titles on mercury and its health effects can be obtained from the authors.

## Tables

Table 1: a/ Absorption of mercury vapour in the oral cavity.

b/ Absorption kinetics with increasing doses of mercury vapours (Measurements by M. H.). Mercury determined with a Jerome Gold Film Mercury Analyzer, Modell 511. For experimental details, see 3.1.

Injected amount Hg g +/- S.D.	Mean absorption/min ng +/- S.D.
a/ 32.9 +/- 1.5	10.2 +/- 2.1 n=12

## IX-1 English

## b/ Non-absorbed Hg, extracted

after 1-3 min ng Hg

	1	2	3 min
32	14	8	5
42	21	13	10
79	34	31	22
120	-	-	39

Table 2: Examples of Threshold Limit Values (TLV or MAC) for mercury in air and food. Exposure amounts of mercury are given in each case. Presumption: respiration volume [rate 0.75 liter/15 per minute]. About 80% of the respired mercury is taken up in the lungs (51)

Occupational exposure 8 h/day, 40 h/week	TLV/MAC $\mu\text{g Hg}/\text{m}^3$	Exposure calculated from respired air and Hg level; $\mu\text{g Hg}/\text{day}$
Germany	100	540
Sweden	50	270
World Hlth Org (WHO)	25	135
Soviet Union	10	54
General Population, 24 h/day EPA, U.S.A.	1	16.2
Soviet Union	0.3	4.8
Exposure from Food		
Sweden	30 $\mu\text{g}/\text{day}$	30

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