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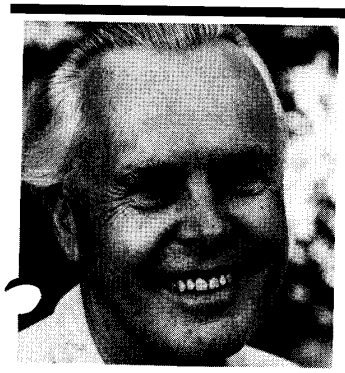

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Direct Transport of Mercury from the Oro-nasal Cavity to the Cranial Cavity as a Cause of Dental Amalgam Poisoning

Introduction

The toxicity of mercury from dental amalgam is the subject of vigorous debate in many countries. In the USA, as well as in some Western European countries, particularly in Sweden, various official statements, with information to the lay public, have been broadcasted on TV and published in daily newspapers and in scientific journals.

In the USA for example, representatives of the American Dental Association and the National Institute of Dental Research have officially declared that "amalgam is safe" and that "the small amount of mercury released from amalgam dental fillings does not represent a health hazard except in those few individuals who may be hypersensitive to mercury". They have even stated that "when mercury is combined with the metals used in dental amalgam, its toxic properties are made harmless".

In Sweden, several highly distinguished persons, as representatives from the National Board of Health, directors, university professors and chiefs from schools of dentistry and medicine, have taken part in the amalgam debate. Many individuals claiming to be amalgam experts have made statements on TV such as, dental amalgam is a "stabile" and absolutely "harmless' compound and that "out of our present scientific knowledge, no reason exists to warn dentists against using mercury dental amalgam in their practice".

Furthermore, in Sweden an expert committee from the National Board of Health has published a report on low-dosage exposure to mercury from amalgam[43] (1987), where the following highly remarkable declaration is made: "As mercury exposure from amalgam is low, the number of patients with symptoms caused by mercury should be very low. In fact, we do not know whether any such cases *exist*, except for a few local allergic conditions".

The Swedish Parliament, too, in hearings[44] (1988/89) has discussed the toxicity of dental amalgam, when a lay politician i.a. postulated that "there has been no scientific evidence hitherto published that mercury from dental amalgam should produce symptoms of disease".

In my view it is impossible to decide about the amalgam toxicity unless we consider the main question: *"How does the mercury from dental amalgam reach the brain?"*

Regrettably, the majority of discussants, including the pertinent Swedish expert committee[43], have totally neglected this key issue – namely, the *direct* pathway for transport of mercury from the oro-nasal to the cranial cavity with the brain and pituitary gland.

Mercury Release from Dental Amalgam

Dental amalgam, containing about 50 percent mercury, is not a "stabile" compound. Most people, patients as well as dentists, are fully aware of the simple fact that their own dental amalgam fillings will, sooner or later, undergo corrosion, necessitating restorative work, possibly years later. With regard to the

From a lecture given at Tufts University, Dental School, Boston, USA. Sept. 20, 1988.

Summary

The toxicity of mercury from dental amalgam is the subject of vigorous debate in many countries. In Sweden an expert committee from the National Board of Health has published a report on low-dosage exposure to mercury from amalgam[43].

In my view amalgam toxicity should not be discussed without addressing the matter of how mercury from amalgam can reach the brain. Regrettably the majority of discussants, including the Swedish expert committee[43], have neglected this key issue — namely, the *direct* pathway for transport of mercury from the oro-nasal to the cranial cavity.

Dental amalgam, containing 50 percent mercury, is not stable. Already 60 years ago Stock[29] demonstrated that amalgam steadily gives off mercury, that may be inhaled by the lungs and so reach the circulation and pass into the body, including the brain.

However, far more dangerous are mercurial fumes, which settle on the mucous membranes in the upper region of the *nasal cavity*, from where the mercury is transported *directly* to the brain and pituitary gland.

The pathways are the *olfactory nerves* or the valve-less *cranial venous* system that provides an open communication between the oro-nasal cavity and the intracranial cavity according to the "Principle of the Shortest Pathway" (Störtebecker[32] 1961), by-passing the general arterial bloodstream and the liver and its detoxifying processes.

The direct *nose-brain* pathway is valid for other materials (e.g. microorganisms, toxins, and aminoacids) besides metals such as aluminum[24], cadmium[47], and mercury.

Moreover, neuronal transport along trigeminal nerves to the brain has long been recognized for herpes virus[17, 45], and horseradish peroxidase inserted into a tooth-pulp readily spreads to the brain stem[2].

Surprisingly high concentrations of mercury have been found post-mortem in the pituitary glands of dentists (Nylander 1986) — concentrations out of all proportion to the mercury found in other parts of the brain, as e.g. the occipital lobes (see table 1).

rate of *amalgam corrosion*, basic scientific work was done already 60 years ago by Stock[29] (1926). Today unfortunately, there are very few dentists and physicians who are aware of the following facts:

In vitro experiments, performed at a temperature of 30°C, demonstrated that mercury *amalgam* pieces with a weight of 1.0 gram, sealed in a glass tube, during a period of less than a month, gave off such a big amount of *mercury vapor* as up to 30 milligrams, i.e. about 1 milligram mercury per day, or *1,000 micrograms* Hg *daily* (Stock[29] 1926).

A continued amalgam corrosion rate of a similar magnitude should imply that all mercury had vanished from the amalgam in less than a 2-year period.

Let us take another example: As mercury-silver amalgam usually contains 50 percent mercury, a few amalgam fillings, with an initial total weight of 2 grams, should originally contain approximately 1 gram mercury, or 1,000,000 micrograms of mercury.

In case of a 50 percent amalgam corrosion over a 10-year period, that is 3,650 days, half of the mercury content in these amalgam fillings has vanished, thus 0.5 gram or 500,000 micrograms Hg. This total amount is equivalent to a release of *137 micrograms* of mercury *per day*.

On the surface of corroding amalgam fillings a highly characteristic finding is the liberation of minute droplets of metallic mercury, lying like small microscopic pearls, particularly in the neighbourhood of small cracks and fissures in the amalgam fillings (cf Fredin[10] 1988).

The setting free of these small mercury *droplets* on the surfaces of amalgam fillings is highly increased by mechanical stimuli, in particular by the heavy force of chewing, as might be measured by the amount of *mercury vapor* in the mouth before and after chewing gum for 10 minutes.

During rest, without any stimulation, one usually finds a value of 3—4 micrograms mercury per 1.0 m^3 respiratory air within the oral cavity of amalgam-bearers. This resting value increases at least *10-fold* by chewing, or through chemical or physical effects, like an augmented temperature in the oral cavity caused by intake of *hot* drinks, as scalding warm coffee or tea, and comsumption of *acid* food, containing vinegar or citrus fruits, and in particular by the co existence of the two metallic compounds *amalgam/gold*, simultaneously within the oral cavity.

Out of the vast literature we may notice a report from the Iowa University (Svare et al[42] 1981), showing that the content of mercury within the oral cavity increased *15 times* after chewing, with values up to about 90 micrograms Hg per 1.0 m^3 air.

Moreover, Harold Utt[48] (1984) measured post-chewing concentrations up to 400 micrograms Hg per 1.0 m^3 air within the oral cavity. The last-mentioned concentration of mercury, 400 micrograms Hg per 1.0 m^3, measured in the respiratory air of the oral cavity, exceeds by more than *1,000 times* the permissible Treshold Limit Value (TLV) of mercury in the respiratory air, *0.3 microgram Hg per 1.0 m^3 air, which the Soviet Union considers as acceptable for its own people to breathe within their domiciles!*

In this connection it is noteworthy that the World Health Organization (WHO) has recommended a maximum allowable concentration (MAC) in working premises of up to 25 micrograms Hg per 1.0 m^3 air, that is based on a daily exposure of 8 hours, during a 5-day-week.

The afore-mentioned MAC of 25 micrograms Hg per 1.0 m^3 air would correspond to an *exposure* of *100 micrograms Hg* for each work-day, as the daily respiratory volume is about 4 m^3 during an 8-hour working period.

Level of Mercury in Air Causing Toxicity

More than 50 years ago the German chemist Stock[30] (1936) stressed that it

The disparity in mercury concentration between the pituitary gland and the occipital cortex canonly be explained by different routes by which the metal arrived at these sites. Through the general arterial circulation both the pituitary gland and the occipital cortex receive a small amount of mercury, but the pituitary has an "extra" dose by direct transport from the nasal cavity.

Certainly, the *direct* nose-brain transport *toxins*, like mercury, has a much bigger implication in the pathomechanism of nervous and mental disorders than most people of today can imagine (cf Störtebecker[40] 1988).

was time to settle the highly controversial question of which *concentration* of mercury in the air would be injurious to health.

He reported that concentrations of *10—20 micrograms* Hg per 1.0 m^3 air in the work-rooms, and a daily stay for several hours during some weeks, produced among the majority of workers obvious symptoms of *ill-effects*, both physically and mentally.

Later on, Stock[31] (1943) sharpened his criteria concerning the levels of mercury in *air* causing intoxication, and he stressed that even minute amounts of mercury might be harmful if they acted *directly* on the *pituitary* region (see later). Thus Stock[31] (1943) had observed that the inhalation of air with the slight content of only *1.0 microgram* Hg per 1.0 m^3 respiratory air gave symptoms of intoxication, like *dizziness*, urge to *micturition*, and increased *salivation.*

According to Stock[31] this enigmatic fact, signs of intoxication present at a low mercury air-level of 1.0 microgram Hg per 1.0 m^3 air, would be very easily understood, if we only consider that the mercury has *by-passed* the general blood circulation, and instead spread from the nasal cavity *directly* to the pituitary region at the base of the brain.

Direct Transport of Mercury from Oro-nasal Cavity to Brain.

In order to elucidate the "amalgam problem" of today it is essential to recapitulate some older investigations. Already 50 years ago Stock[30] (1936) made the undeniable observation that inhaled mercury vapor spread from the mucosa of the upper nasal cavity *directly* to the brain.

CASE 1 — NOSE-BREATHING versus MOUTH-BREATHING (Stock[30])
On himself, Stock (1936) tested the inhalation of air containing 8 micrograms mercury per 1.0 liter air.

After inhaling through his *nose* only a few liters, i.e. some breaths of this air, he rather soon got dizziness, headache, and a nasal catarrh, all symptoms that again disappeared after some days.

Calculating with about 3 liters of air, each containing 8 micrograms Hg, Stock thus inhaled a *total* amount of approximately *25 micrograms* mercury.

"How could these small amounts of mercury produce symptoms of poisoning?": asked Stock (1936).

His explanation was that the mercury *vapor* settled down on the mucous membranes of the ethmoid region in the upper *nasal* cavity, from where mercury was absorbed and passed further to *olfactory bulbs*, and then *directly* into the *brain* (see fig 1).

Some weeks later, Stock performed similar tests, though instead he inhaled the mercury vapors through his *mouth*.

When inhaling through his mouth the same kind of mercury contaminated air, containing 8 micrograms Hg per liter air, Stock could *orally* inhale more than *10 times* the afore-mentioned quantity of nasally inhaled air, still *without* getting any symptoms of mercury intoxication.

Consequently Stock could *orally* inhale about *250 micrograms* mercury, and in spite of this 10 times larger quantity not even experience subjective symptoms of poisoning!

From experiments on *dogs*, which had been sacrificed soon after having inhaled a low-grade Hg-containing air during some hours, Stock[30] (1936) obtained the ensuing values:

Blood:	100 nanograms Hg per 1.0 gram blood
Nasal mucosa:	1,500 nanograms Hg per 1.0 gram tissue
Olfactory bulbs:	500 nanograms Hg per 1.0 gram brain tissue
Frontal brain:	250 nanograms Hg per 1.0 gram brain tissue
Remaining brain:	30 nanograms Hg per 1.0 gram brain tissue

Of interest is the high concentration of mercury in the *nasal* mucosa, and moreover in the *olfactory* bulbs a 5-fold higher Hg-concentration compared to the blood-Hg.

These findings confirm the fact that mercury *vapor* settles down on the nasal mucosa, from where mercury then is transported by a *direct* passage to the cranial cavity.

Morevoer, Stock displayed 63 autopsy cases, in whom he had examined the mercury content of various organs. Among the "Amalgam-bearers" the fol-

lowing case might be of interest:

CASE 2 —MERCURY IN BRAIN & PITUITARY GLAND (from Stock)

A 60-year-old manager died from over-consumption of soporific drugs. POST-MORTEM EXAMINATION with analysis of mercury contents:

(a) Blood:	only 1.0 nanogram Hg per 1.0 gram blood
(b) Pituitary gland:	*1,580 nanograms* Hg per 1.0 gram tissue
(c) Cerebral cortex, basal ganglia & cerebellum:	Rather equal, about 20 nanograms Hg per 1.0 gram fresh brain tissue

Highly remarkable in the afore-mentioned case is the *1,500 times higher* Hg-concentration in the pituitary gland than in the blood, and moreover the *75 times higher* value in the pituitary gland than in the brain.

A similar *discrepancy* in the Hg-concentrations can only be explained by the way of a *direct transport* of mercury from the nasal cavity to the pituitary gland.

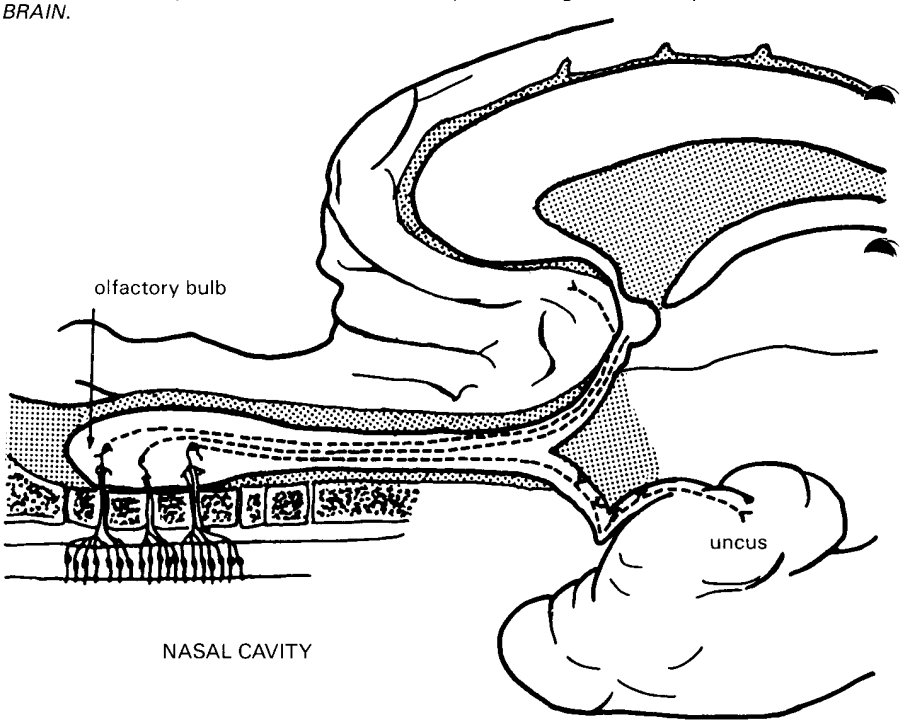
Such disparate Hg-values in various organs can *never* be caused by a spread of mercury through the general *arterial* blood-circulation.

Furthermore, we should notice that these findings tell us that we can *never* rely on *blood-tests* for mercury in order *diagnose* a Hg-intoxication; hence similar blood-tests are hardly of any practical value!

In order to obtain a final proof of his working hypothesis Stock[31] (1943) performed some, ethically rather questionable, investigations.

On moribund cancer patients, a few hours before death, he applied a *mercurial ointment* to the mucosa of the *nasal* cavity, and found at post-mortem examination very high contents of Hg in

Fig 1 *OLFACTORY ORGAN — PATHWAYS FROM NASAL CAVITY TO THE BRAIN.*
From mucosa of the upper nasal cavity the fila olfactoria go through the lamina cribrosa of skull base to OLFACTORY BULBS.
Further fibers from olfactory bulbs pass in olfactory tract to the UNCUS in the temporal region of the brain.
Similarly, MERCURY VAPOR, being released from dental amalgam and settled down on the nasal mucosa, may be absorbed and then transported along the olfactory nerve fibers to the BRAIN.



the pituitary gland, as in the ensuing case.

CASE 3 — DIRECT TRANSPORT OF MERCURY FROM NASAL CAVITY (Stock[31])

A 35-year-old woman was dying of an intractable carcinoma. She had been staying at the hospital for the last three months, and had not received any mercury-containing drugs.

Between 14 and 4 hours prior to death, altogether five times, a mercurial *ointment*, containing 30 percent Hg, was applied to the mucous membranes in the nasal cavity.

POST-MORTEM EXAMINATION with analysis of mercury contents:

(1) Blood:	260 nanograms Hg per 1.0 gram blood
(2) *Pituitary gland:*	*1,370 nanograms* Hg per 1.0 gram tissue
(3) Olfactory bulbs:	510 nanograms Hg per 1.0 gram tissue
(4) Brain (cortex):	60 nanograms Hg per 1.0 gram tissue
(5) Medulla oblongata:	270 nanograms Hg per 1.0 gram tissue

Stock[31] concluded that his observations were in favour of the assumption that the mercury, present in the *pituitary* gland, originated not only from the general blood stream, but *mainly* from a direct transport from the *nasal* cavity, probably via the olfactory bulbs, as they showed a high mercury content, too.

For nearly half a century, Stock's observations remained rather unnoticed until Nylander[20, 21] (1986) published his remarkable findings concerning high concentrations of mercury in *pituitary* glands of deceased dentists (cf later on).

Transport of Toxins via Cranial Venous System

The cranial venous system presents a valve-less open venous pathway from the *oro-nasal* cavity to the *intracranial* cavity. The flow in the system is unimpeded, as there are *no valves* directing the circulation.

This fact implies that a transport of blood, including all its constituents, even various toxins, microbes, and metastases, can freely take place in every direction[6, 8, 27].

In former days, before the "Antibiotic Era", when no remedies existed against the highly feared hemolytic streptococci, many dreadful deaths occurred through a spread of bacteria from facial infections, as a lip furuncle, or from a periapical tooth-infection, all taking place via this *valve-less* cranial venous system directly to the brain.

Radiographically it has been demonstrated that a *direct* venous communication exists between the *teeth*, their surroundings, and the *cranial* cavity, including the brain (cf Störtebecker[33, 34] 1965, 1967).

The intracranial venous system was filled by injecting water-soluble radiographic contrast into the *bone-marrow* of the *mandible* in cadavers (see fig 2 a-b).

Similarly, contrast medium, injected into the *tooth-pulp* of the canine in the upper jaw, passed directly into the cranial venous system.

Through a rich venous net of anastomoses on the base of the skull the contrast medium passed into the *intracranial* venous system, the big venous sinuses, as well as central veins. Radiographic studies on live rabbits, during injection of contrast medium into the *tooth-pulp*, demonstrated that the contrast passed directly into the venous system of the face skeleton, orbits, and skull base.

Consequently these investigations illustrated that conditions are present for a transport from the oral cavity, the jaws with their teeth, and up to the cranial cavity, a fact that might be applicable to all toxic compounds, as microbial neurotoxins[32, 36, 37], cancer-causing agents[35, 40], and why not mercury[38, 39, 41], too.

Experiments on rats, with ligatured oro-pharyngeal cavity, have displayed a transport of radioactively labeled glucose and sodium chloride from the *mouth* by a direct passage to the *brain* (Maller et al[16] 1967). This direct transport might take place via the valve-less cranial venous system, as well as along cranial nerves (cf later on).

By means of whole-body autoradiography in mice Khayat & Dencker[14, 15] (1983—1985) studied the distribution of mercury after *inhalation*, as well as after intravenous injection.

After inhalation high Hg-concentrations were encountered i.a. in the oro-nasal cavity and in the *brain*. But after intravenous injection it is highly noticeable that *no uptake* of mercury occurred in the *brain!*

Their findings[14, 15] cannot be explained in any other way than by a *direct* transport of mercury from the mucosa of the oro-nasal cavity to the brain. The pathways are either by the olfactory nerves or by the valve-less cranial venous system that presents an open venous communication between the oro-nasal and the cranial cavity, all according to the "Principle of the Shortest Pathway" (Störtebecker[32] 1961). In the last-mentioned circumstance the mercury can act in a much stronger concentration on the brain cells and the pituitary gland (cf later), as it by-passes the general arterial blood stream, where the mercury would be diluted in several liters of blood, as well as exposed to the liver and its detoxifying processes.

Isn't it unbelievable that most authors of today totally neglect this direct *"Nose-Brain"* pathway, and still hold the belief that the minute quantity of mercury released from dental amalgam fillings is predominantly either exhaled through the mouth or absorbed to 80 percent by the lungs, and thereupon via the general arterial blood-stream reaching the brain!

Fig 2a *CRANIAL VENOUS SYSTEM (post-mortem studies)*
Water-soluble radiographic contrast medium, injected into the bone-marrow of the mandible, spread by a direct venous communication to the cranial cavity and the brain.
(a) Side-projection (b) Frontal projection
(cf Störtebecker[33, 34] 1965, 1967)

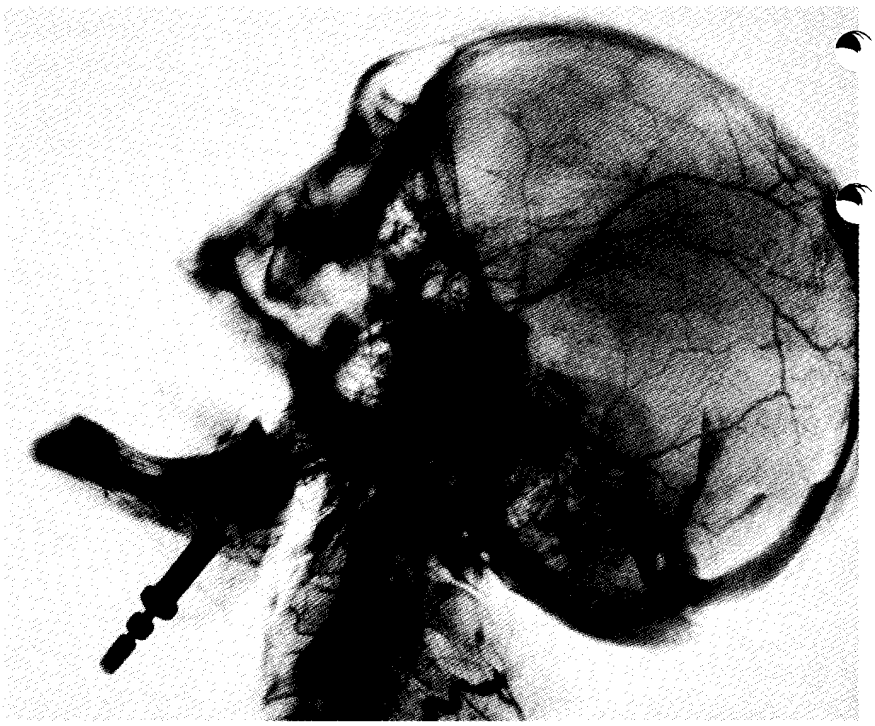
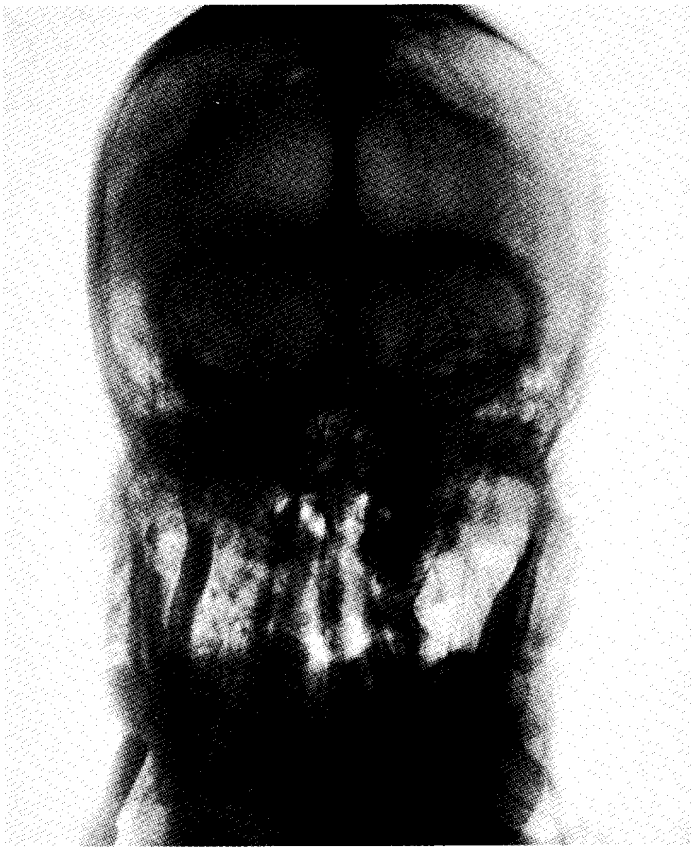


Fig 2 b *CRANIAL VENOUS SYSTEM (post-mortem studies)*
Frontal projection reveals the big intracranial venous sinuses.



Transport of Toxins along Cranial Nerves

In man there is good evidence that toxins and viruses can extend along nerves. This mode of spread is applicable to several viruses, such as rabies, poliomyelitis, and herpes simplex, as well as bacterial toxins, e.g. from tetanus, diphtheria, and leprosy.

More than 60 years ago Marinesco & Draganesco[17] (1923), and simultaneously Teague & Goodpasture[45], performed ingenious animal experiments with inoculation of *herpes simples* virus into the cornea of a rabbit's eye, thus demonstrating the spread.

The virus followed the nerve fibers from the cornea to the ophthalmic branch of the *trigeminal* nerve, with spread via the ganglion on the base of the skull and further downwards to the brain stem (see fig 3).

Unfortunately, these remarkable discoveries from 1923, concerning spread along nerves, were not given the attention that they really should have been paid. Even today their great clinical implication is alien to many medical students and to physicians, as well.

Over 30 years later, Payling Wright[23] demonstrated that virus particles in experimental monkey poliomyelitis were transported along nerves at a speed of 2—3 millimeters per hour, thus traversing *5—7 centimeters* in 24 hours.

Experimental animal research has clarified that a *direct* transport of various

compounds to the brain is applicable to *olfactory* nerve fibers. For example, after soaking the olfactory mucosa of the garfish with labeled ^{3}H-leucine the spread along the olfactory nerve fibers was mapped out by measuring the radioactivity (Weiss & Holland[51] 1967, Gross & Beidler[13] 1973).

The time of spread to the brain varied from a few hours and up to two days.

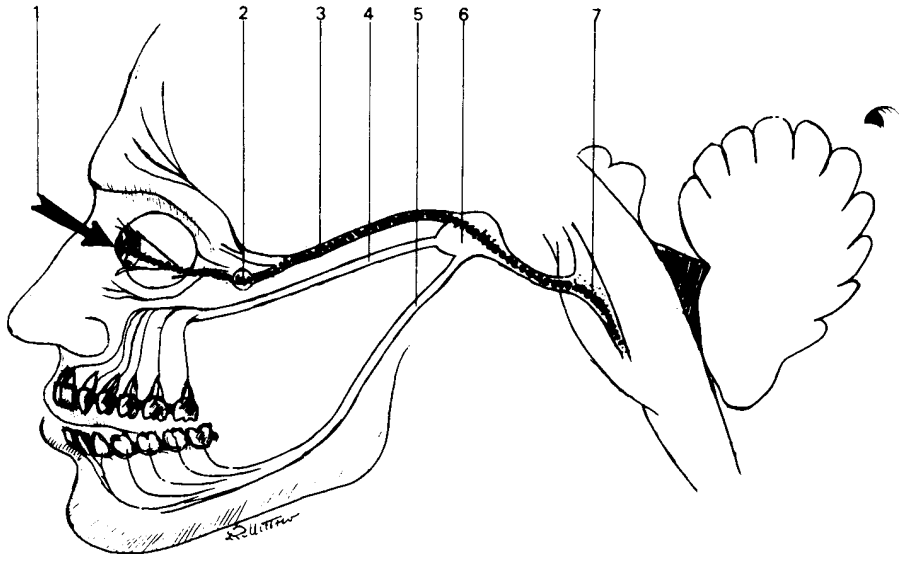
In white mice a spread by the olfactory route to the brain has been commented upon by Martinez et al[18] (1975), who instilled live *ameba* intranasally and thus induced encephalitis. In animals various tracers, injected into the tongue, are transported from the oral cavity along the hypoglossal nerves to the nerve nuclei in the lower brain stem (Sjöstrand[28] 1970, Frizell[11] 1974).

Transport of the enzyme horseradish peroxidase (HRP), after insertion into the *tooth-pulp* on rats and cats, could be followed along the trigeminal nerve to the big ganglion on the skull base[2, 12], and further down to the brain stem (Arvidsson & Gobel[3] 1981), in the same way as it was done already 60 years ago, in the 1920's with regard to herpes simplex virus[17, 45] (cf fig 3).

The highly significant clinical implication of these experimental studies was given by the neurosurgeon Perna[25] (1981), who found a big actinomycotic granuloma of the trigeminal ganglion on the skull base, originated through spread from a similarly infected same-sided "wisdom tooth"!

Spread along nerves, of metals, like lead[5], cadmium[47], and aluminum[24], is

Fig 3 *SPREAD OF HERPES SIMPLEX VIRUS ALONG TRIGEMINAL NERVE*
Inoculation into the cornea of the eye — modified from rabbit experiments by Marinesco & Draganesco[17] in 1923.
(1) Cornea of the eye, as portal of entry for virus
(2) Ganglion ciliare, behind the eye
(3) Trigeminal ophthalmic branch, from the eye
(4) Trigeminal maxillary branch, from teeth of upper jaw
(5) Trigeminal mandibular branch, from teeth of lower jaw
(6) Trigeminal ganglion, on base of the skull
(7) Trigeminal spinal tract, in the brain stem
Possibilities exist for a similar spread of TOXINS, harbored in a TOOTH-PULP, along trigeminal maxillary and mandibular branches.



experimentally well established.

The impportant transport of molecules from *nose to brain* has been discussed i.a. by Shipley[26] (1985), who in the nasal cavity of rats had implanted gelfoam soaked with a horseradish peroxidase (HRP) compound and found that a transneuronal transport took place, i.a. to several neuronal cell groups in the *olfactory bulbs*, basal *fore-brain* and *brain-stem*, and other targets as well.

Shipley[26] commented that the *inhaled* molecules, i.a. various pharmacological agents and substances of abuse, may be transported from the *nasal cavity* to *brain neurons*, and influence the function of neurons in the brain, which have extensive projections to wide areas of the central nervous system, even encompassing non-olfactory areas of the brain.

Balin et al[4] (1986) studied the "nose-brain" pathway and found that in mice, rats and monkeys, native horseradish peroxidase (HRP), administered *intranasally*, passed freely through intercellular junctions of the olfactory epithelium to reach the *olfactory* bulbs within *45—90 minutes!*

They[4] concluded that the nose-brain pathway offers a potential route for passage of *air-borne* noxious, carcinogenic, infectious, and neurotoxic agents and addictive drugs, and also for the delivery of chemotherapeutic agents to combat infectious and deficiency states of the central nervous system.

Furthermore, it might be of interest to take part of a study concerning uptake of *aluminum* into the brain along the *nasal-olfactory* pathways in rabbits (Perl & Good[24] 1987). Strips of gelfoam, saturated with a solution of aluminum lactate, were inserted in the *left* nasal recess of rabits.

The animals were killed 4 weeks later, and granulomas, consisting of accumulations of macrophages, lymphocytes, and plasma cells, were encountered in the *left* olfactory bulb and cerebral cortex. The cortical involvement was bilateral, but more severe on the left. Lesions were also found in the hippocampus of the temporal region.

Perl & Good[24] proposed that *aluminum* can enter the *human* nervous system through a similar route and that this may explain the accumulation of this element in neurofibrillary tangles in *presenile dementia* and the Guamanian form of amyotrophic lateral sclerosis and parkinsonism dementia.

With regard to a possible metal intoxication in the etiology of *presenile dementia* we should notify that Ehmann and coworkers[9, 46] (1986, 1988) found an elevation of *mercury* in some *basal nuclei* of the brain in patients suffering from dementia of the *Alzheimer* type.

A first-rate research project should be to clarify whether any correlation exists to poisoning from *dental amalgam!*

High Content of Mercury in Pituitary Gland of Dentists

In post-mortem studies of the mercury content in brains, the Swedish dental scientist Magnus Nylander, who works at the Department of Environmental Hygiene, Karolinska Institute in Stockholm, discovered that dentists had a surprisingly high Hg-concentration in their *pituitary* glands (see table), being totally out of proportion to the Hg-content in other parts of the brain, as e.g. the occipital lobes[20, 21].

On the one hand, 7 dentists and 1 dental nurse had very high concentrations of mercury within the pituitary gland, ranging from a value of 135 and up to *4,000 nanograms* Hg per 1.0 gram pituitary substance.

On the other hand, 2 edentulous women aged 70 years showed a value of only *5—10 nanograms* Hg per 1.0 gram pituitary substance, thus a content *800 times less* than in a dentist. Most intriguing was Nylander's observation about the disparity between the high concentrations of mercury in the pituitary gland and simultaneously a rather low content of mercury in the brain's occipital cortex

among the dental personnel (see table).

How should we explain this discrepancy between the uptake of mercury in these various regions of the brain?

As its pertinent kind was established as being predominantly inorganic mercury, it was probably derived from dental amalgam, present in the dentist's own office, and inhaled in the shape of Hg-vapor.

The following two cases, namely case (1) and case (3) from the table will be commented upon more in detail.

CASE 4 — OLD DENTIST WITH EXTREMELY HIGH MERCURY IN PITUITARY GLAND[21]

A 78-year-old male dentist suffered from a "Shaking Palsy" with a severe tremor.

Moreover he had a diabetes mellitus, rapidly deteriorating during his last years, and he succumbed in a cachexia.

POST-MORTEM EXAMINATION with analysis of mercury contents:

(a) *Pituitary gland:* *4,040 nanograms* Hg per 1.0 gram tissue

(b) Brain cortex occipital lobe: 300 nanograms Hg per 1.0 gram tissue

Noticeable is the very high Hg-concentration in the pituitary gland, being *14 times higher* than in the occipital lobe which also contained an unusually big amount of mercury.

In the next case the content of mercury in the *occipital* lobe is rather normal, in spite of a considerably augmented Hg-concentration in the pituitary gland of nearby 3,000 nanograms Hg per 1.0 gram tissue. Remarkable is the big difference between the *Hg-storage* in the pituitary gland and the brain, at a ratio of up to 169:1 (cf table).

CASE 5 — INCONGRUITY BETWEEN MERCURY IN PITUITARY GLAND AND BRAIN[21]

A 50-year-old dentist died of renal failure, a long-lasting chronic glomerulo-nephritis.

POST-MORTEM EXAMINATION with analysis of mercury contents:

(a) *Pituitary gland:* *2,700 nanograms* Hg per 1.0 gram tissue

(b) Brain cortex occipital lobe: 16 nanograms Hg per 1.0 gram tissue

One might ask why the Hg-concentration was *169 times higher* in the pituitary gland than in the occipital lobe?

Even if we always have to take into account the existence of a kind of "specific" *Hg-receptors*, like some "mercury-seizing" cells, situated in the pituitary gland, the observed disparity in the Hg-concentrations between the two re-

Table 1 *MERCURY CONCENTRATION IN PITUITARY GLAND AND BRAIN*
25 autopsy cases — Values in nanograms Hg per 1.0 gram tissue

	Pituitary Gland	Occipital Cortex	Ratio
A: Occupationally Exposed			
Case (1) Dentist	4,040	300	14:1
Case (2) Dentist	3,650	84	43:1
Case (3) Dentist	2,700	16	169:1
Case (4) Dentist	350	40	9:1
Case (5) Dentist	350	5	70:1
Case (6) Dentist	300	17	18:1
Case (7) Dentist	135	19	7:1
Case (8) Dental Nurse	1,300	18	72:1
B: Not Occupationally Exposed			
a) Amalgam-bearers			
Cases (9—23) Range	7—77	3—23	
Mean Value	28	11	2.5:1
b) No Amalgam			
Case (24) Edentulous	10	6	2:1
Case (25) Edentulous	5	6	1:1

(By courtesy of Magnus Nylander[20, 21] 1986, 1987)

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gions, the *pituitary* gland and the *occipital* brain, can best be explained in the manner that mercury has arrived to these organs by *two different* ways of transport.

(a) Through the *general* arterial blood circulation, both the pituitary gland and the occipital brain cortex have received a certain, but minor amount of mercury.
(b) Moreover, the pituitary gland has got an "extra" supply of mercury by means of a *direct* transport from the nasal cavity to the cranial cavity.

Unfortunately, the majority of physicians and dentists of today seem to be totally unaware of this *direct* transport of mercury to the pituitary gland!

Why is it that a Swedish expert committee[43] (1987), as well as many other authors[22, 49, 50], in calculating the contribution from *dental amalgam* to man's body burden of mercury totally neglect the direct "Nose-Brain" transport, but mainly consider the uptake via the lungs to the general arterial blood stream and further elimination of mercury through the kidneys to the urine.

"The Heart of the Matter" — Hypothalamic-Pituitary System

A steady feed-back takes place between the pituitary gland and the hypothalamic region at the base of the brain, in order to serve the regulation of all intricate hormonal and neural functions, necessary for maintenance of life.

As a whole, the *hypothalamic* region functions as a vital autonomic center, that regulates our "Struggle for Life" by means of influencing e.g. intake of FOOD and DRINK, HUNGER, THIRST, our body TEMPERATURE, ALERTNESS or SOMNOLENCE, INSOMNIA or SLEEP, moreover our SEXUAL behaviour, et cetera. Hypothalamic lesions may manifest themselves as vegetative attacks with symptoms of irrational FATIGUE, heart PALPITATION, AIR HUNGER, SWEATING, highly increased URINARY OUTPUT, moreover diverse *mental* symptoms, as sensation of UNREALILTY, MOODINESS, ANXIETY, DEPRESSION, mixed with outbursts of ANGER, like RAGE.

This clinical picture may fully be explained by release of mercury from amalgam fillings, and its further spread through a *direct venous passage* to the base of the brain (see Störtebecker[38, 39, 40, 41] 1985–1989).

In view of the recent findings of high contents of mercury in the pituitary gland (Nylander[20, 21]), probably emanating from dental mercury, the dental status of all patients suffering from various *endocrine* and *mental* symptoms should be carefully scrutinized.

Malignant Brain Tumors Twice as Common among Dentists

The principle of the "Shortest Pathway" holds true not only for mercury , but for a magnitude of *poisonous* compounds, as i.a. bacterial toxins and viruses, which might spread from the oro-nasal cavity directly to the brain and the pituitary gland, both along cranial nerves and via the valve-less cranial venous system (cf Störtebecker[32, 35, 37, 40] 1961, 1978, 1982, 1988).

Noteworthy in this connection is a study by Ahlbom and co-workers[1] (1986), who investigated the *cancer-indicence* among *9,000 dental personnel*, half dentists and half dental nurses. They found that the encountered number of *malignant gliomas* of the brain corresponded to a *two-fold* increased risk for each of male dentists, female dentists, and dental nurses, seen in comparison to the general population.

Certainly, one might ask if *cancer-causing* agents can spread to the cranial cavity according to the principle of the "Shortest Pathway", i.e. directly from the oro-nasal cavity to the brain.

This in particular, as the malignant gliomas are *10 times* more frequently occurring in the *anterior* regions of the

15. *Khayat A, & Dencker L:* Organ and Cellular Distribution of Inhaled Metallic Mercury in the Rat and Marmoset Monkey (Callitrix jacchus): Influence of Ethyl Alcohol Pretreatment. Acta Pharmacol Toxicol 1984, 55: 145–152.
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brain, seen in comparison to their occurrence in the posterior *occipital* lobes (cf Morley[19], Störtebecker[37, 40]).

Moreover, it is well known that mercury *suppresses* the *immune* defense mechanisms, a happening that confers an increased sensitivity to all kinds of infections, as viruses, bacteria, and molds, in its turn *enhancing* tumor growth.

Störtebecker[32–41] (1961–1989) has discussed the implication of the valveless cranial venous system in the etiopathogenesis of various nervous disorders, and as his opinion claimed that a direct spread of *microbial carcinogens* from *teeth* with periapical abscesses in the jaws should be considered as an origin to *malignant gliomas*[35].

Diagnosis of Mercury Poisoning — Clinical Symptoms and Signs

Already in the antiquity the highly toxic effects of mercury were well known from mercury ores in ancient Roman Idria and in Almaden in Spain, where the slave-workers already after the *first day's* work suffered the following acute symptoms: *Fatigue, dyspnea,* and *epigastric pain*. Rather soon, other symptoms, especially from the mucous membranes and skin added to the intoxication picture, such as *"Stomatitis Mercurialis"* with painful, swollen and bleeding gums, leading to a severe *paradentosis* with loosening of the teeth. *"Ptyalismus Mercurialis"* with enormously increased salivation, and *"Hydrargyria"*, a vesiculopustular excema appeared rather early.

Somewhat later came a magnitude of highly characteristic symptoms of mercury poisoning, in particular from lesions of the *nervous* sysatem, like *"Erethismus Mercurialis"* with mental disturbances, especially "Moodiness", as well as *"Tremor Mercurialis"* with involuntary shaking movements of a choreatic type, so called intention tremor.

We should notice that *tremor* is rather *rare* in a "low-dosage" mercury intoxication, emanating from dental amalgam fillings! Sooner or later, these unhappy prisoners, condemned to death in the mercury mines, wasted away in the terminal stage of poisoning, *"Cachexia Mercurialis"*, propably due to the damaging action of mercury on the hypothalamic-pituitary endocrine balance.

On the other hand, the more *slowly* developing chronic poisoning, due to *small* doses of mercury, such as releas[illegible] from *dental amalgam fillings*, is predominantly characterized by *mental* symptoms; hence often difficult to diagnose and therefore put in the shade.

"Low-Dosage" Mercury Poisonin[illegible] — "Subjective" Symptoms

In all people, who harbor dental amalgam fillings, *mercury vapor* is daily released, especially during hard chewing, and we make a big mistake if we consider mercury amalgam as being a "stabile" compound (cf earlier).

An *insidiously* occurring mercury intoxication from dental amalgam gives both "subjective" symptoms, which might be rather difficult to verify, and "objective" signs of poisoning, which are fairly characteristic and should be easy to diagnose.

1: "FATIGUE"

The paramount symptoms is an extrem[illegible] inexplicable lassitude, an enormou[illegible] weakness with complete depletion of strength, that both *physically* and *mentally* engulfs the patient.

The "tiredness" may be so enormou[illegible] that the patient cannot any longer stay up, but is mostly confined to his bed, except for visiting the lavatory. Neither can the patient manage to take his meals at the table with his family, nor to look at TV!

2: MENTAL SYMPTOMS

A preponderant symptom is the *psychasthenia*, with incapacity to resolve doubts and uncertainties or to resist obsessions, compulsions, or *phobias*, that one knows are irrational.

There is a conspicuous *loss of memory*, especially to "close" events, while

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remote happenings are better memorized. "Bad memory", combined with a total *lack of concentration*, leads to inaptness to perform even the slightest intellectual work, and to make decisions, even concerning trivial matters of "daily life".

To all these mental symptoms comes an undue lack of self-confidence, manifested in the behaviour as a non-rational and unreasonable *timidity*, further an insane *self-effacement*, which may lead to a grave *depression*.

HIghly characteristic is the "Moodiness", the unpredictable and rapid changeableness of mood, i.a. with "Rage", especially uttered as sudden outbursts of *anger*.

Unmotivated "panic" *anxiety*, lacking all reality, "Pavor Mortis", an inexplicable fear of death, are very typical symptoms of acute, as well as chronic mercury poisoning, and even *hallucinations* may sometimes occur.

How many people of the "Western civilization" do not suffer from this whole multi-flora of *mental* symptoms, which may be caused by a "low-grade" mercury poisoning from dental amalgam fillings.

3: "HEAVY-HEADEDNESS"
As a symptom of mercury intoxication a *severe* headache is *rare*. But the Hg-intoxicated persons often have a feeling of being in a "mist", sometimes commented upon as "my head is covered by a globe of glass!"

4: DIZZINESS & NAUSEA
Usually the patients experience a "seasickness" with nausea, and the balance is impaired, with a slight side-stepping, that makes it impossible to be up and around.

5: "FITS OF SHIVERING"
The Hg-intoxicated persons often feel "chilly" and "shivery". like the beginning of an infection or onset of a "common cold", why they prefer to stay in bed.

Apparently, on account of all these nasty symptoms the mercury poisoned persons must, least to say, feel rather "ill". However, all these distressing symptoms are *only* "subjective" sensations, which for a surrounding world, as family, neighbours, and fellow-workers, are impossible to register!

Consequently, the whole picture is very often misinterpreted, and the Hg-intoxicated persons are being looked upon as suffering from "imaginary illness", like *hysteria*.

"Low-Dosage" Mercury Poisoning – "Objective" Signs

Dental amalgam gives off metallic mercury in the shape of an easily volatile gas, totally *odorless*, why impossible to detect by means of smell; hence persons are mostly unaware of being poisoned.

In any case, if physicians really penetrate the case history carefully, and even examine the patients, they will find conspicious and "objective" signs indicating a mercury intoxication.

1: "STUFFED NOSE"
Among people suffering from chronic mercurialism a generally shared ailment is a pertinacious "Stuffed Nose", due to a chronic rhinitis, usually manifesting as a *bloody* nasal discharge and dry crusts.

Mercury is a strong cell-poison and the loss of smell is not uncommon. Mercury may also spread to the para-nasal sinuses, the inner ear-tubes, and the middle ear, inducing a chronic inflammation, giving clinical symptoms as *hearing impairment, tinnitus,* and *vertigo*.

What concerns the "stuffed nose", most patients finally get the *diagnosis* of something "allergic", but unknown to its real origin, namely mercury released from dental amalgam fillings, a most poisonous compound harbored in the mouth!

2: "IRREGULAR HEART ACTIVITY"
Very typical complaints among people, who suffer from mercury intoxication, are *attacks* of "Chest Pain", often occurring about 15–30 minutes after a meal.

Sometimes the precordial pain is accompanied by a rather regular, but highly

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increased heart frequency, a "paroxysmal tachycardia", up to the double of normal frequency.

The patients may also suffer from a so called "Arrhythmia perpetua", when at auscultation we find totally irregular heart-beats.

These attacks of cardiac arrhythmia are probably elicited by irritative phenomena from the hypothalamic region due to mercury intoxication.

In animal experiments it is well established that a part of the posterior *hypothalamus*, when being stimulated, causes an increase of heart rate, rise of blood pressure, etc.

The circumstance that these attacks usually occur about half an hour after a meal, is probably due to an *extra release* of dental Hg, caused by heavy chewing, and hot drinks (cf earlier).

3: "AIR HUNGER"

The mercury-intoxicated patient may feel "sick", why he starts to breathe heavily, so called "Hyperventilation", usually in order to avoid fainting.

Regrettably, in Medicine hyperventilation is often regarded as being a "psychogenic" phenomenon, instead of representing a *classic sign* of mercury poisoning!

4: HIGHLY INCREASED URINARY OUTPUT

Moreover, commonly occuring among Hg-intoxicated persons, but diagnostically often overlooked, are attacks of a highly increased *urinary* output, up to *2 liters* during the period of *a few hours*, as compared to a normal amount of about 1.5 liter urine during 24 hours.

How can we explain that the relatively small quantities of mercury, being released from dental amalgam fillings, may cause this type of periodically increased urinary output?

Through the direct "Nose-Brain" pathway mercury vapor is also affecting the posterior *pituitary* gland, where a lesion causes *lack of the anti-diuretic hormone* vasopressin, and consequently a highly increased diuresis.

Erroneous diagnoses cost billion of dollars

Numerous patients, who themselves have questioned if their symptoms of illness might originate from their *teeth* and be caused by a *mercury poisoning from dental amalgam*, have by their dentists and physicians been gravely misdiagnosed, and as a consequence also been subject to a highly erroneous treatment, and even dealt with as suffering from "psychic" and "imaginary" illnesses!

The social and economic consequences to the individual and the community are enormous.

The annual direct cost for treating these mercury poisoned patients, as suffering from "mental" disease, may be estimated to *billions* of dollar, only for the faulty medical care concerning incorrectly established diagnoses and treatment.

Moreover, caused by this dental and medical wrongdoing, comes an approximately 10 times higher indirect cost, from loss to the gross national product and loss to federal taxes!

At last, but not least, added to this national burden is the personal tragedy, which is hardly measurable in monetary terms.

Certainly, it is high time that medical and dental authorities realize the sheer madness of deliberately poisoning mankind with mercury from dental amalgam and consequently ban all further use of mercury amalgam. As a self-protection all dental patients should hereafter refuse to have any mercury amalgam implanted into their teeth!

* * * * *

Patrick Störtebecker

Sammandrag på svenska

Direkt transport av kvicksilver från mun-näshålan till kraniehålan som orsak till förgiftning från tandamalgam

Kvicksilverförgiftning från tandamalgam är föremål för en häftig debatt i många länder, bl a i Sverige där en offentlig rapport om "lågdos-exponering för kvicksilver"[43] 1987 föreligger, den så kallade "LEK-utredningen".

Enligt min åsikt kan det aldrig bli någon rätsida på frågan om tandamalgamets giftighet såvida icke den väsentligaste frågan tas upp ur forskningssynpunkt: **"Hur kommer kvicksilvret till hjärnan?"**

I amalgamdebatten har tyvärr så gott som samtliga deltagare, i synnerhet Socialstyrelsens "experter" i LEK-utredningen[43] helt förtigit pudelns kärna, nämligen den *direkta spridningsvägen* från mun-näshålan till kraniehålan med hjärnan och hypofysen.

Tandamalgam är ingen "stabil" substans, utan avger hela tiden kvicksilverånga, som med andningsluften kommer ner i lungorna och sedan över i allmänna blodcirkulationen för att på så sätt spridas till alla organ i hela kroppen, inkl en smärre del även till hjärnan.

Likväl, långt farligare är den kvicksilverånga, frigjord från tandamalgam, som slår sig ner på slemhinnorna i munnäshålan, varifrån kvicksilvret sedan *sprids direkt* till skallhålan med hjärnan och hypofysen!

Transportvägar för kvicksilver direkt från näshålan till hjärnan finns dels längs med *luktnervernas* fibrer upp genom skallbasen, dels via det klafflösa *kraniala venösa systemet* med öppna kommunikationer, allt enligt "Regeln om kortaste spridningsvägen" (Störtebecker[32] 1961).

Sistnämnda *direkta* spridningsvägar medför att kvicksilvret kan verka i starkare koncentration, enär det ej kommer ut i det allmänna blodomloppet där det skulle spädas ut. Även smärre doser blir på detta sätt farliga för såväl hjärnan som hypofysen!

De *direkta* spridningsvägarna från mun-näshålan till hjärnan är väl dokumenterade både i kliniken och djurexperimentellt sedan många år tillbaka, men tyvärr är flertalet av dagens läkare och tandläkare mycket okunniga härvidlag.

Nästan alla tänkbara substanser kan spridas direkt från näshålan till kraniehålan med hjärnan och hypofysen, sålunda mikrober, virus, bakterietoxiner, aminosyror, enzymer, flera droger, bl a "crack", läkemedel, vidare metaller som aluminium, kadmium och kvicksilver.

Det är verkligen högst besynnerligt att alla dessa fynd och vetenskapliga fakta helt förtigs av de flesta deltagare i amalgamdebatten.

Ytterst oroande torde även vara den upptäckt som gjordes häromåret av Magnus Nylander[20, 21] (1986), nämligen att avdöda tandläkare hade mycket höga halter av kvicksilver i hypofysen (se tabell 1), vilket endast kan förklaras genom en *direkt* transport av kvicksilverånga via näs-slemhinnan till hypofysen.

Transport av olika gifter, som bl a kvicksilver, vilket sker via de *direkta spridningsvägarna* från mun-näshålan till kraniehålan med hjärnan och hypofysen, är säkerligen av långt större betydelse för uppkomsten av *neurologiska sjukdomar* än de flesta idag kan föreställa sig (Störtebecker[40] 1988).

Kvicksilvrets giftverkan på *hjärnan* ger sig uttryck i mångahanda symptom, men

framförallt i en outsäglig och för personen ifråga helt obegriplig *trötthet*, både fysiskt och psykiskt.

En "lågdos"-kvicksilverförgiftning ger främst *psykiska* symptom: Ökad irritabilitet, lynnighet med plötsliga och omotiverade vredesutbrott, bristande koncentration, nedsatt minne, sänkt intellektuell arbetskapacitet, självutplånande med total avsaknad av självförtroende, vidare initiativlöshet och skygghet, fjärmande från socialt umgänge, och inte sällan svåra depressioner, "panik-ångest", och i flera fall suicid.

Vem i västerlandet lider icke, i högre eller mindre grad, av dylika *psykiska* rubbningar, så typiska för en kronisk kvicksilverförgiftning?

Enär halten av kvicksilver i blod inte är korrelerad till Hg-koncentrationen i hjärnan eller hypofysen, ger tyvärr *blodprov* med Hg-analys vanligtvis *intet* av värde ur praktisk diagnostisk synvinkel.

Diagnosen kan dock oftast säkerställas genom iakttagande av "objektiva" tecken, såsom "nästäppa", ofta blodig snuva, ojämn hjärtverksamhet, luftbrist, andnöd och starkt ökade urinmängder, ett mycket karakteristiskt tecken, men vanligen förbisett av läkarna.

Det är nu hög tid att vi alla, både tandläkare, läkare och lekmän äntligen fatta hur farligt kvicksilver, frigjort från tandamalgam, är för vår hälsa och vår hjärna!

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