Acknowledges the intimate relationship of the teeth and the mouth to the rest of the body.

This booklet is an attempt at providing the most up to date and relevant information with which to make informed decisions about your dental treatment. There is no attempt either direct or implied to offer in these pages any medical diagnosis or prognosis. For a variety of medical and legal reasons, there is no suggestion either direct or implied, that any treatment provided in our surgery will give you a health improvement. The information provided here is supported by published scientific literature

All treatment plans will be discussed with you and written fee quotations will be given. Note that this quotation is a close estimate of the fees. If changes must be made during the progress of your treatment, they will be discussed with you as they occur.

We welcome your questions and your interest in our services.

Imagine

By Robert Gammal

Imagine a world where most of the inhabitants (people) are implanted with the third most toxic metal known to science. A metal which is specifically toxic to nerve tissue and developing brains. This metal will cross the placenta and breast milk and poison the developing foetus. (Another group of people come and inject the newborn baby with more of the same metal at levels which may be 60 times higher than the FDA recommend for this body weight.)

When the child is anywhere from 4 – 6 years of age he/she will also get some of these metal implants and possibly a goodly dose of formaldehyde and nickel.

The adult will have these toxic metal implants replaced about every 10 years.

A unique property of this toxic metal is that it turns to vapour at room temperature and rapidly vaporises at body temperature. It is absorbed easily into the body and stored in the fatty tissues including the myelin sheath around the nerves. It crosses the protective blood brain barrier with ease.

In this same world many people will be told that it is a great idea to leave dead, infected and gangrenous tissue in the body. The infection supposedly will not spread and the gasses, which are released from the gangrenous tissue, cannot harm you. These gasses by the way are chemically similar to 'mustard gas' - a known carcinogen and nerve toxin.

Mixtures of metals will then be placed in the adult mouth which promote a greater release of the nerve metal and also will generate electric currents in the order of a 1000 times greater than the circuitry in the brain. Other chemicals will be added to the adult body including zinc oxide which causes calcification in organs, more nickel to further affect the immune system, possibly some gold and titanium for the wealthier and a host of other metals for the rest. Formaldehyde may also be slowly dripped into the body at random times.

These practices will be supported by governments that add rat poison to the drinking water of ALL people. The rat poison is a toxic waste product of the aluminium industry. The rat poison will cause hip fractures and heart attacks and possibly leukaemia. The rat poison will also make both adults and children dumb and apathetic.

When death finally comes and the body is cremated the toxic metals will be released from crematoria chimney at a rate of about 11kg per chimney per year. This pollution will poison the people who are exposed to it and of course the environment.

Each person will pay financially for this abuse at each stage.

No this is not Hitler's torture camps. This is Australia today.

To sidestep the danger of mercury poisoning from dental amalgam, the dental associations and unfortunately many professors call on a different big stick. Amalgam is the strongest and thus supposedly the best filling material for back teeth. Since when has a mechanical argument ever taken priority over much published detrimental health effects? Perhaps thalidomide should still be used to stop morning sickness.

The deans, professors and dental associations promote the mechanical argument even in the face of the following statement by Dr. Harold Loe, the Director of the

National Institute of Dental Research (NIDR), who, in the September 1993 edition of "Dental Products Report" wrote:

"That first filling is a critical step in the life of a tooth. Using amalgam for the first filling requires removing a lot of the tooth substance, not only diseased tooth substance but healthy tooth substance as well. So, in making the undercut you sacrifice a lot, and this results in a weakened tooth. The next thing you know the tooth breaks off, and you need a crown. Then you need to repair the crown...and so it continues to the stage where there is no more to repair and you pull the tooth. With the first filling you should do something that can either restore the tooth or retain more healthy tooth substance. Use new materials – composites - or materials you can bond to the surface without undercuts. You can do this with little removal of the tooth substance so that the core of the tooth is still there."

It is well known that mercury from amalgam is implicated in the development of Alzheimer's disease, Parkinson's disease, autism and other learning disabilities in children. Mercury from amalgam will cause infertility, birth defects and miscarriage. The amount of mercury in the body of a foetus is directly proportional to the number of amalgam fillings in the mother's mouth.

How many have heard the words - "that tooth will need a Root Canal Therapy". Any objection to this proposition is usually answered with "you must keep the tooth at any cost". The costs we are informed of, are the $'s needed to do the root treatment and then the $'s needed to crown the tooth. Dentists are not taught that root therapied teeth can cause cancer and a host of other chronic degenerative diseases. Dentistry uses the words Root Therapy to conceal the fact that you will be keeping dead infected and gangrenous tissue buried in your jaw. The dental professors blindly state that the concept of Focal Infection died in the 1940's soon after the demise of one of the world's greatest researchers Dr Weston Price. In the 1920's Price demonstrated the spread of infection from dead teeth to the rest of the body. The development of arthritic changes and many other health effects, including cancer, were clearly shown. Bacteria and their toxins can migrate with ease from a dead tooth and spread throughout the body. One of the foundations in medical thinking is, in fact, that focal infection is a major concern to your health. Dentistry blatantly asserts that this is not possible and thus denigrates the science, which is even published in their own journals. In 1997 the Journal of Periodontolgy devoted a whole issue to the relationship between gum disease and cardiac disorders, diabetes and a number of other chronic degenerative diseases. Literally thousands of references exist demonstrating focal infection from dead teeth.

Another aspect of the root therapy saga is the continued use of formaldehyde releasing root filling cements. The formaldehyde will spread to all parts of the body. Apart from being carcinogenic, a host of diseases are reported which relate to formaldehyde exposure.

Is there a Pedodontist (specialist for children's teeth) who can tell us the minimum amount of Formaldehyde, which does NOT cause mutagenic change in a four year old? Silly question? Not at all. These people and many GP dentists regularly place formaldehyde into baby teeth. The procedure is called a 'Pulpotomy'. As if this level of physical abuse is not enough, these pulpotomised teeth are then regularly covered with a stainless steel crown – stainless steel releases high levels of nickel, with devastating effects on the immune system.

The teaching of Neural Therapy shows us the connections of the teeth to the rest of the body. Dead teeth and electrical interference from metals may affect the bodies regulatory mechanisms and thereby cause disease states in other parts of the body, often associated with the acupuncture meridian that the offending tooth is on. This

German research changes the way we must think about disease. I have lost count of the number of women who have informed me that their breast lumps disappeared within a week of extracting these teeth.

Of course the criticism is that this is purely anecdotal evidence, and not controlled by double blind studies. Sadly, the word 'anecdote' is used to denigrate 'clinical observation'. Many clinical observations are published in reputable journals and still the professors ignore them.

The professors and dental association even have the audacity to disagree with legally binding court decisions. On 7th Jan, 2003 the Superior Court in San Francisco approved the wording of the following warnings to be displayed in Californian dental surgeries in compliance with proposition 65:

"Dental Amalgam, used in many dental fillings, causes exposure to mercury, a chemical known to the State of California to cause birth defects and other reproductive harm

Root canal treatments and restorations including fillings, crowns and bridges, use chemicals known to the state of California to cause cancer."

Don't despair just yet. Australia has plans to fluoridate ALL drinking water and no one will ever have decay again – or so we are told! Didn't the Australian government read the news reports?

"Friday 09 May 2003

Basel to stop fluoridation of drinking water

The city of Basle in Switzerland is preparing to end the fluoridation of municipal drinking water in the autumn. The local parliament has voted to stop the practice, due to high costs and lack of evidence that fluoride cuts tooth decay."

Some of the well-known effects of fluoride include an increase in bone fragility and thus hip fractures, increased rates of heart disease, and a statistical association between fluoridated areas and leukaemia. A massive epidemiological study of thousands of children in China clearly demonstrated that those in a fluoridated area had substantially lower IQ's than children from non-fluoridated areas. Fluoride makes you dumb. Mercury makes you dumb. Formaldehyde is carcinogenic. Leaves a few questions!

So what's the difference between this insanity

and BioCompatible Dentistry?

In the words of the late Edward Arana, D.D.S.;

"Biological Dentistry can be categorized as dentistry with a consciousness. A consciousness of how the treatments of the teeth and jaws will affect the health of the individual and how it will affect the immune system. Will it be congruent and health enhancing or will the treatments be health stressors to the individual. In the past only lip service was paid to the biocompatibility of materials used in dentistry. The material's compatibility was judged on a general basis and not on an individual basis that is required for biocompatibility."

"The most tragic example of misstated biocompatibility is organized dentistry's position of advocating a known poison - MERCURY- in amalgam fillings just because it has been used for 150 years! In doing so, dentistry has been misled and the truth obfuscated concerning the fact that mercury does indeed cause ill effects when

placed as an implant in the body even to the point of denying that a filling in a prepared tooth cavity is not an implant. Mercury and other heavy metals from dental fillings contribute to all chronic disease states as do multiple chemical sensitizing exposures. From environmentally ill patients there is clinical evidence that the heavy metals from dental fillings and multiple chemical exposures act synergistically to intoxicate and stress the patient, thus causing disease."

The fabrication of mercury dental amalgam in 1812 changed the face of dentistry worldwide. By the 1850's the American and Swedish Dental Societies had insisted that their members sign a pledge to never place this toxic poison in a persons mouth. The dentists at the time refused as they finally had a material, other than molten lead, with which to fill teeth. These societies collapsed. The dentists formed their own new associations, which to this day still regulate the practice of dentistry in almost all countries.

Dental associations, including the Australian Dental Association, are trade organisations. They are NOT scientific organisations. Do they have vested interests? The American Dental Association still owns two patents (since 1978), on the most popular formulation of dental amalgam. Manufacturers pay incredible sums of money to have their products endorsed by the dental associations. These 'endorsements' are merely financial arrangements – they are not a mark of safety or efficacy. In a legal case in 1992 the American Dental Association offered the following statements in their defence against being sued for mercury poisoning form amalgam; "…..The ADA did not manufacture, distribute or install the amalgam fillings. …The ADA has no legal duty of care to protect the public from allegedly harmful dental materials…"

These trade organisations also act as the voice of dentistry. In Australia it is always the ADA who is asked for comments regarding dental practices. Lets forget the ADA - it is time for the Deans of Dentistry to be questioned.

DO NOT allow mercury amalgam to be placed in your mouth

DO NOT have root therapy - it is much healthier to remove dead, infected, gangrenous tissue

DO NOT allow fluoride – rat poison- to be placed in your children's or your mouth.

Information and references are available at www.bcd.com.au. Here you will also find links to many international sites with fully referenced information.

Visit www.iaomt.com to see the short video of mercury vapour coming off a 25 year old amalgam filling - "Smoking Tooth = Poison Gas"

Heavy Metal Detox Protocols 2006

The following protocols are a *tightly summarised compilation* of what we believe to be the most comprehensive and useful at the present time. As there is still a great deal to learn about removing mercury and other heavy metals from the body, we are constantly reviewing and updating this protocol. No two people are the same so please regard these protocols as a guide only. Understand that we are doing our best to present you with the latest information. We cannot take responsibility for side effects or problems associated with these protocols and use of this outline is on the basis of self-responsibility. We advise that you do work with knowledgeable doctors, naturopaths and dieticians to achieve optimal health.

There are many variations on detox procedures – all have benefits.

Recommend Web Sites:

Dietrich Klinghardt, M.D., Ph.D. http://www.neuraltherapy.com Much of what you will read below comes from his work.

Overview of Protocols

Pre Amalgam Removal

thorough dental examination including OPGs and other relevant x-rays

appointment scheduling to avoid the 7th 14th and 21st days for repeat appointments

Bio Compatibility Testing of replacement Dental Materials

Begin Oral Supplements for detox, kidney & liver function improvement

Consult a knowledgeable health professional before and after amalgam removal

An Organic diet will be most helpful in mercury detox

During Amalgam Removal

Maintain and adjust oral detox supplements
Increase Chlorella before and after each appointment
Intravenous Vit C
Report any adverse effects
Use of a Chlorophyll mouthwash is helpful

After Amalgam Removal

Continue Oral Detox

Add Cilantro (Coriander tincture) to detox programme
DMPS Chelation under medical supervision where necessary
Follow up tests for mercury levels can be organised

SERIOUS WARNINGS:

EDTA chelation

EDTA should be avoided as a chelating agent as it has been shown that the EDTA/Mercury complex is potentially more neurotoxic than mercury by itself.

Homoeopathic Amalgam / Mercury

Homoeopathic Amalgam/Mercury will potentially mobilise far too much mercury too quickly. This can make you very ill if other detox procedures

are not followed or if you kidneys and liver are not able to handle such a mass onslaught. We strongly recommend avoiding this form of detox.

Zinc

Zinc potentiates the lethal effects of mercury

N-acetyl cysteine, saunas, vegetarian diets, fasting and excessive sports

all of these have been shown to potentially drag mercury into the brain cells rather than away from the intracellular environment.

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Summary of Recommended Supplements

Vitamin C
Dosage 3-6 grams/day
Must be well separated from BOTH Selenium and Chlorella by at least 2 hours

Vitamin B
We recommend MNB-287
8-10ml twice per day for first month
then 10ml once per day in the mornings

Selenium
Sodium Selenite
200 – 400 mcg / day
Must be separated from Vit C by at least 2 hours

Demertox
A good source of Glutathione
One Cap/day
Stored in fridge

Mineral Complex
LifeStream Colloidal Minerals
5mls 2X per day

Chlorella: SUN brand
start with 1 gram (=4 tabl) 3-4 times/day. This is the standard maintenance dosage for grown ups for the 6-24 months of active detox.

During the more active phase of the detox (every 2-4 weeks for 1 week), whenever cilantro is given, the dose can be increased to 3 grams 3-4 times per day (1 week on, 2-4 weeks back down to the maintenance dosage).

Take 30 minutes before the main meals and at bedtime. This way chlorella is exactly in that portion of the small intestine where the bile squirts into the gut at the beginning of the meal, carrying with it toxic metals and other toxic waste. These are bound by the chlorella cell wall and carried out via the digestive tract.

When amalgam fillings are removed, the higher dose should be given for 2 days before and 2-5 days after the procedure (the more fillings are removed, the longer the higher dose should be given).
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No cilantro should be given around the time of dental work. During this time we do not want to mobilise deeply stored metals in addition to the expected new exposure.

If you take Vitamin C during your detox program, take it as far away from Chlorella as possible (best after meals).

Chlorella Growth Factor
dosage: 1 cap. CGF for each 20 tabl. chlorella

Garlic Blackmore's Freeze Dried
Dosage: 1-3 capsules freeze dried garlic after each meal. Start with 1 capsule after the main meal per day, slowly increase to the higher dosage. Initially the patient may experience die-off reactions (from killing pathogenic fungal or bacterial organisms). Use 5-10 drops bear-garlic on food at least 3 times per day.

Fish oil:
Due to the high levels of mercury in fish we recommend to only use fish oils that are guaranteed free of mercury. Some are available at the surgery

Balanced Electrolyte Solution
Endura powder
2 gram twice a day

Cilantro (Chinese parsley)
No cilantro should be taken until
ALL amalgam is removed –
fillings, under crowns, in bone!

Cilantro causes the gallbladder to dump bile - containing the excreted neurotoxins - into the small intestine. The bile-release occurs naturally as we are eating and is much enhanced by cilantro. If no chlorella is taken, most neurotoxins are reabsorbed on the way down the small intestine by the abundant nerve endings of the enteric nervous system.

2 drops 2 times /day in the beginning, taken just before a meal or 30 minutes after taking chlorella
Gradually increase dose to 10 drops 3 times/day for full benefit.
During the initial phase of the detox cilantro should be given 1 week on, 2 –3 weeks off.

Health Canada's Position Statement on Dental Amalgam

Health Canada advises dentists to take the following measures:

1. Non-mercury filling materials should be considered for restoring the primary teeth of children where the mechanical properties of the material are suitable.

2. Whenever possible, amalgam fillings should not be placed in or removed from the teeth of pregnant women.

3. Amalgam should not be placed in patients with impaired kidney function.

4. In placing and removing amalgam fillings, dentists should use techniques and equipment to minimize the exposure of the patient and the dentist to mercury vapour, and to prevent amalgam waste from being flushed into municipal sewage systems.

5. Dentists should advise individuals who may have allergic hypersensitivity to mercury to avoid the use of amalgam. In patients who have developed hypersensitivity to amalgam, existing amalgam restorations should be replaced with another material where this is recommended by a physician.

6. New amalgam fillings should not be placed in contact with existing metal devices in the mouth such as braces.

7. Dentists should provide their patients with sufficient information to make an informed choice regarding the material used to fill their teeth, including information on the risks and benefits of the material and suitable alternatives.

8. Dentists should acknowledge the patient's right to decline treatment with any dental material.

Mechanics Is Not An Excuse To Disregard Systemic Effects

By Robert Gammal

In arguing the case of whether amalgam is safe or not, the discussion has often been turned to compare the physical properties of various filling materials. The Australian Dental Association still claim that dental mercury amalgam is a far superior filling material and that composite fillings are not only inferior but that they only last a very short time. The types of scare tactics used by such organizations are intended to distract our attention from the fact that amalgam is in fact the greatest source of mercury to the general population and that the stuff simply is not safe. I do not believe that an argument about physical properties of any material can carry more weight than the responsibility of placing health care as our number one priority. It is like suggesting that because Thalidomide stops morning sickness for some women we should still be using it. Mechanics is not an excuse to disregard systemic effects.

To make matters worse the claim that amalgam is a far superior material to composite as a filling material simply does NOT reflect the scientific research, which is even published in the dental journals. Most of this short paper carries annotated references from some of this literature. As you read them it will become clear to you that in fact it is the composite materials which are superior to dental amalgam as a tooth restorative material from a purely mechanical perspective. They carry the added advantage that they also look like teeth and are generally almost non-toxic. Ironically, this view is also held by Dr Peter Magnus who in 1992 was president of the Australian Dental Association (NSW branch). In an interview on radio 2UE, 16th February 1992, the reporter asked him if he used amalgam in his practice.

> His response was: "I personally don't"
>
> Reporter: "Why is that?"
>
> Dr Magnus: "Because I believe today we have materials which are probably better and not so environmentally unfriendly."
>
> Others, in official positions, have expressed the view that amalgam in fact a terrible filling material. These views are not new.

Quintessence International is one of the most respected international dental journals. In 1995 the editor-in-chief of Quintessence (Volume 26, Number 3,1995), Dr Richard Simonsen wrote:

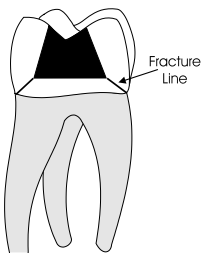
> "Amalgam should not be used as a restorative material in paedatric dentistry. Why? Because better materials are available.
>
> Amalgam should not be used as a first time restorative material. Why? Because better alternatives are available.
>
> **Move Over Amalgam At Last."**

Dr. Harold Loe, the Director of the National Institute of Dental Research (NIDR), stated in the September, 1993 edition of "Dental Products Report":

> "That first filling is a critical step in the life of a tooth. Using amalgam for the first filling requires removing a lot of the tooth substance, not only diseased tooth substance but healthy tooth substance as well. So, in making the undercut you sacrifice a lot, and this results in a weakened tooth. The next thing you know the tooth breaks off, and you need a crown. Then you need to repair the crown...and so it continues to the stage where there is no more to repair and you pull the tooth. With the first filling you should do something that can either restore the tooth or retain more healthy tooth substance. Use new materials-composites or materials you can bond to the surface without undercuts. You can do this with little removal of the tooth substance so that the core of the tooth is still there."
> With these statements, made by such respectable authorities, it is amazing that the dental associations continue to take an opposing position.

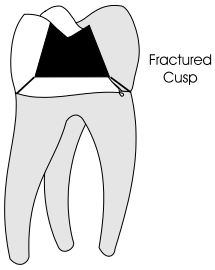
Comparison of Filling Techniques and Their Consequences

Amalgam fillings do not stick to the tooth. To retain the filling in the tooth, the cavity must be prepared with 'undercuts'. These undercuts not only lock in the amalgam filling but also cut off the nutrient supply to the dentine

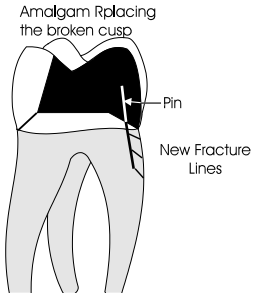


above the cut. Therefore the tooth structure above and to the side of the filling becomes brittle.

All metals in the mouth will undergo some corrosion. Amalgam also corrodes at a reasonably fast rate. When amalgam corrodes it also expands and it does so in all directions. The force created by this expansion will often create minute fractures in the tooth that is already more brittle due to the shape of the cavity preparation. At this stage the patient returns to the dentist to report that all they were eating was some soft bread and the tooth broke!



To repair such a problem, the dentist will usually drill a small hole into the dentine and insert a self-tapping screw - called a pin. The pin is reinforcement for the amalgam filling which will go back in. Even if this pin is made of titanium it will undergo corrosion when in contact with amalgam. Again the corrosion will cause an enlargement of the pin (sometimes up to five times its diameter) which will then crack the tooth further - but this time lower down the root surface.



This tooth is now a candidate for a crown because the filling, which has to go back into the tooth, is now so large that it cannot sustain the forces of chewing for very long.

Composite fillings do stick to the tooth. They are bonded chemically and mechanically to the tooth. They do not require a cavity, which is undercut and therefore do not require such a large or damaging cavity. In fact a composite filling can be used to rebuild a broken cusp without the use of pins or other mechanical support. I personally have not used a pin for years and have had great success with such restorations.

Studies comparing the fracture resistance of the tooth when filled with amalgam or composite indicate that amalgam will weaken the tooth structure whereas bonded composite fillings will strengthen the tooth. There is absolutely NO reason to continue the use of mercury amalgam!

Secondary Decay Under Fillings

Another bit of misinformation, which is often touted about by the dental associations, is that secondary decay is much greater with composite fillings than amalgam. This is completely false. When amalgam corrodes it not only does so on the chewing or exposed surfaces, but also corrodes on the side, which is in contact with the tooth- the deep part of the cavity. The corrosion products react with the calcium and phosphorous in the tooth, with the formation of hydrochloric acid. This acid then dissolves the tooth structure which is called secondary decay. The newer term for this is Crevice Corrosion. This does not happen with composites.

Toxicity

Mercury is one of the most toxic substances known to man. Amalgam is made of 50% mercury which leaches from the set amalgam all of the time. Recent research is indicating that the breakdown products of composites and glass ionomer cements are between 300 times and 1.6 million times below the Tolerable Daily Intake levels. By comparison the mercury from amalgam is about 4 times greater than the Tolerable Daily Intake levels.

Although different people may show sensitivity to different composites, they are not subjected to the high level of poisoning as with dental mercury amalgam.

As a cautionary note, there has been one study published, which shows that some composites (those based on BIS-GMA) may break down to two materials (Bisphenol-A and Bisphenol-A dimethacrylate) which have been shown to be estrogenic. It is therefore advisable, for patients who have a hormone-related cancer, to avoid such materials if possible. With this warning in mind it is still preferable to replace all amalgam fillings. Mercury from amalgam will reduce the body's level of Selenium. Several studies have shown that cancer rates increase as the body's selenium levels drop.

Referenced Abstracts - Composites

References which demonstrate the functional ability

of plastic composite resin fillings

Replacement of missing cusps: an in vitro study. LC; Smith-BG J-Dent. 1994 Apr; 22(2): 118-20
One of the commonest methods of replacing a missing cusp, a pinned amalgam restoration, was compared with three adhesive restorative techniques, two of them with additional pin retention. All the teeth were subjected to occlusal / lateral forces and loaded to fracture in an

Clinical evaluation of a highly wear resistant composite. Dickinson-GL; Gerbo-LR; Leinfelder-KF Am-J-Dent. 1993 Apr; 6(2): 85-7
The purpose of this clinical study was to determine the long-term potential of a resin composite restorative material.

The colour matching ability of the material never fell below 96%. The percent of restorations exhibiting a surface texture similar to enamel never fell below 90% Alfa. At the end of 3 years, the total average loss of material was only 28 microns. No clinical evidence of bulk fracture was detected .... 79% of the restorations were Class II complex restorations with the replacement of at least one cusp!

Evaluation of occlusal marginal adaptation of Class II resin-composite restorations ASDC-J-Dent-Child. Jul-Oct. 1993
describes the results of an evaluation of the occlusal marginal adaptation of Class II restorations in a clinical trial. The margins of 183 resin composite and 61 amalgam restorations, made by three dentists, were assessed. Resin composite restorations showed more 'excellent' margins than amalgam restorations (64.5 percent and 21.3 percent, respectively).

The variable mainly influencing the marginal adaptation of the composite restorations was the dentist.

Three-year follow-up of five posterior composites: in vivo wear. Willems-G; Lambrechts-P; Braem-M; Vanherle-G J-Dent. 1993 Apr; 21(2): 74-8
The wear of five posterior composites was evaluated in Class II cavities over a 3-year period with an accurate 3D-measuring technique. A clinical evaluation was also performed. The ultrafine compact-filled composites (Willems et al., 1992) showed acceptable wear rates ranging from 110 to 149 microns after 3 years. This is very similar to the wear rate of human enamel on molars, which is about 122 microns after 3 years. It can be concluded that the investigated composites can be considered as amalgam alternatives.

Directed Shrinkage Technique in Class V Composite Restorations: in Vivo Microscopic Evaluation and Clinical Procedure, Ferrari, M., Practical Periodontics and Aesthetic Dentistry, Vol. 5, No. 7, September 1993, pp. 29-36.

The study examined the leakage in vivo of Class V restorations with chemically-cured composite bases. Class V cavities were prepared at the CEJ in six periodontally hopeless teeth in six patients. The cavities were total etched, All-Bond 2 and Bisfil 2B (Bisco) were applied, and Z100 (3M) was used to complete the restorations. After 30 days, the teeth were extracted, dyed, sectioned, and scored for leakage. The results showed no enamel margins leaked, with only one cervical margin showing minimal leakage.

Longevity of dental restorations in selected patients from different practice environments. Mahmood-S; Smales-RJ Aust-Dent-J. 1994 Feb; 39(1): 15-7

The objective of the study was to evaluate the long-term survivals or longevity of dental restorations placed in selected patients from different practice environments in two countries. The case histories of 46 adult patients with 622 restorations placed in three private practices in Pakistan were followed for a minimum of 10 years, and compared with similar assessments of 50 adult patients with 966 restorations placed in a dental hospital in Australia. Amalgam and composite resin restorations showed similar survivals in both countries, but cast gold restorations had much lower survivals in the Pakistan group of patients. In both countries, restoration survivals were significantly better in females, and when patients attended less frequently for treatment. For the Australian group, changes in dental operators also gave significantly better survivals, and there were significant restoration survival differences present between the three practices in Pakistan.

Evaluation of occlusal marginal adaptation of Class II resin composite inlays. Kreulen-CM; van-Amerongen-WE; Borgmeijer-PJ; Gruythuysen-RJ ASDC-J-Dent-Child. 1994 Jan-Feb; 61(1): 29-34

In this paper, the results of a clinical study of the occlusal marginal adaptation of indirect Class II resin composite inlays are presented. The margins of 180 resin composite and 60 amalgam restorations, made by three dentists, were assessed, shortly following their placement. An indirect, photographic method has been applied to assess marginal adaptations. The restorations were classified into excellent and non-excellent marginal adaptation categories and on this basis influencing factors were determined. Resin composite inlays appeared to have a greater percentage of 'excellent' margins than amalgam restorations (46.1 percent and 6.7 percent, respectively). The dentist was the variable that most influenced the marginal adaptation. Variability in the period elapsing between applying the restoration and conducting the assessments is discussed as a factor that may impair a fair comparison with initial results for direct composites.

Three-year follow-up of five posterior composites: in vivo wear. Willems-G; Lambrechts-P; Braem-M; Vanherle-G J-Dent. 1993 Apr; 21(2): 74-8

The wear of five posterior composites at occlusal contact areas (OCA) and contact free occlusal areas (CFOA) was evaluated in Class II cavities over a 3-year period with an accurate 3D-measuring technique. A clinical

evaluation was also performed. The ultrafine compact-filled composites (Willems et al., 1992) showed acceptable OCA-wear rates ranging from 110 to 149 microns after 3 years. This is very similar to the OCA-wear rate of human enamel on molars, which is about 122 microns after 3 years. The fine compact-filled composite had an unacceptable OCA-wear value of 242 microns after 3 years. The ultrafine midway-filled composite showed an exceptionally high CFOA-wear value of 151 microns after 3 years, which gave the impression of it being gradually washed out of the cavity. Clinically, 70% of the restorations made with the ultrafine midway-filled composite showed excellent colour match after 3 years. For most of the compact-filled composites slightly opaque fillings were noted and 63% of the restorations made with one of these materials were clearly opaque. It can be concluded that the investigated ultrafine compact-filled composites can be considered as amalgam alternatives as far as their wear resistance is concerned.

Posterior adhesive composite resin: a historic review. Fusayama-T J-Prosthet-Dent. 1990 Nov; 64(5): 534-8

This landmark study by one of our great pioneers, graphs resin vs. amalgam failures and shows resin (Clearfill Posterior - a self-cured resin) far superior in the long term. This study is included in his text book published last year and makes fascinating reading. It is now relatively old, but Fusayama's team are (were - he's retired) world leaders in resin technology although his technique is clinically complex and I don't use it. In Japan this technique is taught at undergraduate level!

Mercury toxicity: Genetic susceptibility and synergistic effects

Boyd E. Haley, PhD

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B.E. Haley/Medical Veritas 2 (2005) 535–542 535

Note: the full paper is available from the reception

Abstract

Mercury toxicity and intoxication (poisoning) are realities that every American needs to face. Both the Environmental Protection Agency and National Academy of Science state that between 8 to 10% of American women have mercury levels that would render any child they gave birth to neurological disorders. One of six children in the USA have a neurodevelopmental disorder according to the Centers for Disease Control and Prevention. Yet our dentistry and medicine continue to expose all patients to mercury. This article discusses the obvious sources of mercury exposures that can be easily prevented. It also points out that genetic susceptibility and exposures to other materials that synergistically enhance mercury and ethyl-mercury toxicity need to be evaluated, and that by their existence prevent the actual determination of a "safe level" of mercury exposure for all. The mercury sources we consider are from dentistry and from drugs, mainly vaccines, that, in today's world are not only unnecessary sources, but also sources that are being increasingly recognized as being significantly deleterious to the health of many.

Introduction

Mercury toxicity and intoxication (poisoning) are realities that every American needs to face. This article discusses mercury intoxication and several normally appearing factors that increase the susceptibility to mercury toxicity. The sources considered are dentistry and mercury from drugs, mainly vaccines, that, in today's world are not only unnecessary sources, but also sources that are being increasingly recognized as being significantly deleterious to the health of many who are so exposed.

Mercury from dentistry

Let us begin by discussing mercury exposure from dental amalgams. Figure 1 is a segment from a movie showing the emission of mercury vapors from a 50 year old amalgam; it is still releasing mercury at the temperature of a cup of coffee. The point of this figure is to provide visual evidence that mercury is indeed released by dental amalgams. It has been reported in a World Health Organization review of mercury that 80% of the mercury vapors inhaled is retained by the human body. This is why dental amalgams have been found to be the major contributor to human body mercury burden. The visualization of mercury emitting from amalgams presents irrefutable evidence that spokespersons for the American Dental Association (ADA) are exceptionally deceptive when they state there is no danger of mercury exposure from dental amalgams.

11. Conclusions

In summary, it appears as if autistics represent a subset of the population that are more susceptible to the toxic effects of mercury and thimerosal because they are not efficient excretors of these toxic materials. Further, it appears as if the sex hormones play a major role in susceptibility with the male hormones increasing susceptibility to the neurotoxicity of ethyl-mercury and the female hormones affording a good degree of protection. Common sense tells us that a lead toxic person would be more susceptible to mercury toxicity than a healthy, non-toxic person. Research confirms this and we routinely observe that many heavy metals increase the apparent toxicity of low levels of mercury. It is well known that a milk diet will cause the retention of mercury as does the exposure of mammals to certain antibiotics. This would make infants with ear infections prime candidates for mercury retention toxicity. Certainly, the findings of aberrant biochemistries in the autistic child that appear to correlate with mercury sensitive enzymes increases the possibility of mercury involvement in autism causation.

If certain infants are more susceptible to mercury toxicity due to their inability to excrete mercury then it seems plausible that, since this is a genetic susceptibility, older individuals may suffer from the inability to excrete mercury also. Based on the ability of mercury to mimic many of the biochemical aberrancies found in AD brain and to produce aspects of the pathological diagnostic hallmarks of AD it seems plausible that AD is a disease related to mercury toxicity. The published decrease of mercury in the nail tissue of AD versus normal age-matched individuals seems to support this possibility.

Finally, the synergistic effects of other heavy metals, diet, antibiotics, etc. on mercury toxicity make it impossible to define a "safe level of mercury exposure." Therefore it is imperative that we try to eliminate all exposure to mercury; and removal from dentistry and medicines is most important and critical for human health.

Mercury and Cancer

Proceedings from the First World Congress on Cancer
Professor W Kostler President of Austrian Society of Oncology, Austria.
Sydney 1984

Glutadione Peroxidase and Selenium

The glutadione peroxidase is detoxifying peroxides to water and glutadione. We try to improve the scavenging activity by applying selenium. There is a correlation between the level of glutadione peroxidases in the blood and the plasma selenium. A study was made in the US, on AIDS patients and patients with AIDS related complexes, using a control group. A direct correlation was found between the plasma selenium level and glutadione peroxidase activity in the blood. It is very important to supply this selenium, because glutadione peroxidase **is** one of the most effective scavengers.

Excessive intake of selenium (2,000 to 5,000 micrograms a day) is said to produce poisoning. But it is not true in a way - we know that women m Venezuela take 5,000 micrograms **a** day and don't suffer from breast cancer. The optimal intake is in comparison to the sub-optimal intake- sub-optimal means 50 to 200 micrograms a day and optimal intake is 250 to 300 per day. Optimal means you have some reserve, for example, for radiotherapy or chemotherapy, with agents that activate radicals in your body.

The normal level of selenium should be 80 to 130 micrograms per litre of blood. We check the levels of selenium in different cancer patients at their first consultation in our office - most of them suffer from a big deficiency in selenium. One patient had a level of 49 micrograms-a very low level in a person with advanced bladder cancer.

Effects of Mercury in Dental Amalgam

1 want to emphasise that there is a very strong interaction between some trace elements, and I'll focus on mercury and selenium. A surplus of mercury causes a diminution of selenium. Why is that important?

Most of our cancer patients have a lot of amalgam dental fillings. 1 remember **a** study we made few years ago at the University of Vienna. One group of students with amalgam fillings had to chew a chewing gum for twenty minutes, and the other group had to drink hot lemon juice for twenty minutes. We wanted to know what happens with the mercury level in their blood. By chewing the gum, with dental fillings of amalgam, or by drinking hot and acid juices, there was always a big mercury intake in the blood.

In the same way, the level of selenium was lowered, because a lot of selenium was needed to detoxify the mercury to mercury Selenite, and therefore it was not available any longer. What we didn't expect was that the immune status showed us a small decrease in the immune-competent cells after this test-after 20 minutes of chewing gum or drinking hot juice

Therefore, we usually try to remove the dental fillings made out of amalgam, and use chelation therapy to bring out the mercury, leaving the selenium free for the glutadione peroxidase activity again. Such a patient can have normal mercury levels in the blood, but after the injection DMPS there is a large amount of mercury over the next 24 hours, going out with the urine. The mercury is deposited in the brain and the central nervous system, mostly in the hypophysis, and causes a lot of problems with hormonal regulation.

Mercury and Reproductive Damage: Population Control

The Heidelberg research on amalgam and fertility

All preliminary research results should be treated with caution. Still, there are several aspects of the latest Heidelberg study that make it promising:

The fact that it is primarily based on laboratory tests blinds both researchers and patients to some extent and reduces the risk of selection bias. The results of hormone tests, cell culture studies and intervention studies agree. It agrees with at least one earlier study that showed a link between poor mercury hygiene and reduced fertility among dental assistants. Although that study did not measure mercury levels, the highly exposed groups may not be worse off than the most highly exposed individuals in the general population.

Why the difference from other epidemiological studies?

The results do not agree well with epidemiological studies that have not found correlations between amalgam and various health problems. In particular, two earlier epidemiological studies /3,4/ have failed to find an association between allergy and amalgam. In contrast, the preliminary results from Heidelberg indicate a very strong link.

Some possible explanations may be:

The other studies were done with school children, who have been less exposed and for a much shorter time. The patients at the gynecological clinics may be a particularly

mercury-susceptible group; effects that are relatively rare in the general population may be common among these patients. The Heidelberg results may be flawed for some reason that may not be clear until the research is published.

1/ Rowland AS, Baird DD, Weinberg CR, Shore DL, Shy CM, Wilcox AJ. The effect of occupational exposure to mercury vapour on the fertility of female dental assistants. Occup Environ Med 1994; 51 (1): 28-34.

2/ Barregård L, Sallsten G, Jarvholm B. People with high mercury uptake from their own dental amalgam fillings. Occup Environ Med 1995; 52 (2): 124-128. (Abstract.)

3/ Herrstrøm P, Högsted B.Dental restorative materials and the prevalence of eczema, allergic rhino-conjunctivitis, and asthma in schoolchildren. Scand J Prim Health Care 1994; 12: 3-8.

4/ Olstad ML, Holland RI, Wandel N, Hensteen-Pettersen A. Correlation between amalgam restorations and mercury concentrations in urine. J Dent Res 1987; 66(6): 1179-82.

German Scientists Reveal Mercury Amalgam Affects Human Fertility

In a cell culture experiment, mercury affected hormone production at low concentrations.

A link has been found between mercury and hormone disturbances, immune disturbances, recurrent fungal infections, hair loss and allergies. The differences are large. Allergies and hair loss are 2-3 times as common in the high-mercury group.

These recent results confirm earlier findings from Heidelberg on mercury and fertility. According to the researchers, mercury exposure leads to hormone and immune disturbances that can reduce fertility.

In a cell culture experiment at the Tuebingen University gynecological clinic, mercury affected hormone production at low concentrations. The concentrations were so low that cell vitality was otherwise almost unaffected.

Professor Ingrid Gerhard and her co-workers and the university gynecological clinic in Heidelberg have examined more than 1000 patients for mercury, fertility problems and symptoms. Mercury was measured after giving a so-called chelating agent known as DMPS. This substance mobilizes mercury from tissues, particularly the kidneys, so that it is excreted in the urine where it can be measured.

The high-mercury group had more hormonal disturbances, immune disturbances, recurring fungal infections, hair loss and allergies. A number of different hormonal disturbances were found, sex hormones among them. All these differences were statistically significant and some very marked. Allergies and hair loss were 2-3 times more common in the high-mercury group.

The doctors at the clinic have successfully treated fertility problems with a combination of vitamins/minerals and amalgam removal.

According to professor Gerhard, mercury exposure leads to hormone and immune disturbances that can reduce fertility.

Amalgam Removal Does lower the body burden of mercury

Australian Risk Assessment of Mercury Exposure from Dental Amalgam Published August 2000

Prepared by Chem Affairs Pty Ltd PO Box 890 Lane Cove NSW 1595
Published August 2000

This risk assessment was commissioned by the National Health & Medical Research Council of Australia, as part of a series of recommendations put forward by a working party which was set up in 1998 to assess the literature about the dangers of mercury from dental amalgam. NH&MRC have not yet endorsed this document. Although most of the report claims safety for amalgam on the bases of a supposed "Normal Mercury Level" in the body it is important to know that there has never been a level of mercury exposure which is considered safe. The Normal levels suggested in this report are far above the levels set by both the USEPA and the ATSDR.

In point 8 of the Executive Summary the following is stated:

"Amalgam removal has been shown to be effective in reducing mercury levels to the levels of those in people without amalgam fillings. Chelation treatment has also reduced levels in the short-term....in one case **report, amalgam removal has reduced a very high urine mercury level to a normal level. This change was accompanied** by **a decline in symptoms……."**

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Potential Biological Consequences of Mercury Released from Dental Amalgam. A State of the Art Document.

The Australian and other dental associations in the world have consistently claimed that removing amalgam for the sake of health improvements is unethical as there is no relationship between mercury from dental amalgam and disease. They still hold this position. Much of their claims of late are based on a report called "Potential Biological Consequences of Mercury Released from Dental Amalgam. A State of the Art Document. [MFR-panel (Swedish Medical Research Council)]A State of the Art Conference in Stockholm 9-10 April 1992.

They make the following conclusions which are responded to by one of the leaders of the International Academy of Oral Medicine and Toxicology. Note Prof Vimy's credentials.

- "- Mercury released from dental amalgam does not, according to available data, contribute to systemic disease or systemic toxicological effects.
- No significant effects on the immune system have been demonstrated with the amounts of mercury which may be released from dental amalgam fillings.
- - Allergic reactions to mercury from amalgam have been demonstrated, but are extremely rare.
- In very few individuals local reactions such as lichenoid reactions of the mucosa, may occur adjacent to amalgam restorations as well as adjacent to dental restorations made of other materials.
- There are no data supporting that mercury released from dental amalgam give rise to teratological effects.
- The possible environmental consequences of mercury from handling dental amalgam can be controlled by proper waste management, including the installation of efficient amalgam separators in dental offices.
- Available data do not justify discontinuing the use of silver-containing dental amalgam fillings or recommending their replacement."
- In the panel: Bergman B (chairman), Bostrom H, Larsson K S, Li5e H

In An open letter

to Sekreterare Tore Scherstén Medicinska Forskningsrådet Swedish Medical Research Council Box 6713 S-113 85 Stockholm, Sweden December 15, 1992

Re: Potential Biological Consequences of Mercury Released from Dental Amalgam. A Swedish state of the Art Conference,
April 9, 1992.

Dear Secretary Scherstén:

By now you must have felt the pressure of a number of groups who have criticized your "conference". In fairness to you, it is apparent that trust was misplaced in an organizing committee, which had no intention of convening an objective academic scientific forum. Rather, these individuals had a predetermined agenda, as demonstrated by their public positions on the issue of amalgam safety taken on many occasions prior to this meeting.

Drs. Larsson, Löe and Bergman are all on the record as defenders of the status quo. Dr. Bergman's objectivity is tainted by his wife's involvement in the issue; while Dr. Larsson is on the record as a strong supporter of amalgam. Indeed it was incredible to see this person act as both presenter and "judge", especially since he
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has no scientific experimental track record of his own to demonstrate his expertise in this area. Finally, Dr. Löe, politically, administratively and economically affiliated with the American dental establishment, is apparently more concerned with preventing litigation in the U.S.A. than he is with determining scientific truth. His opening biased remarks made it obvious why he was chosen as moderator. Dr. Boström was red herring - a physician "yes"-man with absolutely no research expertise in this area.

The conference presenters showed a general lack of expertise. Most have poor research records and many had not published research papers on either mercury or dental amalgam. This is easily determined by reviewing the bibliographies to their written presentations. They have few if any research papers of their own to cite! The penultimate example was Dr. Petr Skrabanek, a self anointed "quack catcher". This individual, who has no scientific expertise of amalgam, is one of a growing group of self appointed watch-dog "experts". In North America, we have an organization called the National Council Against Health Fraud which purports to be expert in everything. Dr. Skrabanek's mere presence at the meeting totally discredited the scientific purpose of the conference. Sweden, a country of many noted scientists, was better represented by the quality of the expertise in the audience than by the quality of many conference speakers.

Finally, I understand that my invitation to present a paper at this conference was extended reluctantly by the organizing committee, and only after political pressure for a more balanced meeting. If you review the list of speakers chosen it will be obvious that the intention of the organizers was to "white wash" the conclusions. The conclusions of the conference were drawn by the organizing committee and do not represent a consensus view of all the participants or the audience. Since the results were apparently prordained, as I have just described, they are not credible.

I have enclosed for your information a reprint of a recent medical scientific forum on the same issue (Goering et. al., 1992). As you can see, there is now international scientific concurrence on a number of points related to the amalgam mercury issue and its potential effects on human health; a concurrence which is in marked contrast to the "massaged" conclusions of the Swedish Medical Research Council's biased organizing comnmittee.

Respectfully yours,

signed

Murray J. Vimy BA, DMD, FAGD, FIAOMT Clinical Associate Professor Department of Medicine (also Private Practice of Dental Medicine)

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A vast wealth of published research exists which clearly demonstrates that removing the source of the mercury poisoning – the amalgam fillings – will in fact lower the body burden of mercury. Here is a small taste of this literature;

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Recovery from Amyotrophic Lateral Sclerosis and from Allergy after Removal of Dental Amalgam Fillings. Redhe, O; Pleva, J. Int. J. Risk Safety Medicine. (1994): 4, 229-236.

An evaluation of 100 cases of poisoning and immunological effects in dental amalgam patients, documented in clinical practice.

The patient had suffered for a long period from neurological problems. In 1984, following a complete neurologic evaluation, a diagnosis of amyotrophic lateral sclerosis (ALS) was made at the department of neurology of the University Hospital in Umea, Sweden. It is of unknown etiology and considered to be 100% fatal. No further visit to the clinic was proposed, as the disease is pernicious and there is no known therapy for ALS.

A dentist recognised the symptoms as those familiar in the patient group with health problems attributable to dental amalgam fillings. Patient history revealed the onset or exacerbation of neurologic symptoms following placement of amalgam dental fillings. The patient had 34 tooth surfaces filled with amalgam, most of which were shallow and of moderate extent.

With the consent of the patient, all amalgams were removed and replaced with alternative material. Treatment was completed in March 1984. Removal of the amalgam in the first tooth that had originally given post-operative problems resulted in an exacerbation of symptoms, with a continued recurrence of exacerbation following each subsequent replacement.

Following the replacement of the last DA, the patient's entire condition rapidly improved. Six weeks following the final replacement, the patient was able to go up stairs without experiencing back pain. Pains in the mouth also receded and the sore throat, present during the whole history of the disorder, recovered. Five months after completion of the DA removal, the patient returned to the same University Hospital at Umea for a week-long follow-up investigation, after which the following notation was placed in her record: "The neurologic status is completely without comment. Hence, the patient does not show any motor neuron disease of type ALS. She has been informed that she is in neurological respect fully healthy."

Rubber Dam Reduces Mercury Exposure During Amalgam Removal

References Collated June 99

Following are some abstracted referenes which support the use of a rubber dam when removing amalgam. Every effort should be made to use rubber dam to

protect the mouth and airways. Rubber dam may not eliminate mercury exposure but it goes a long way to reducing it.

Mercury levels in plasma and urine after removal of all amalgam restorations: the effect of using rubber dams. Berglund A, Molin M Department of Dental Materials Science, Umea University, Sweden. **Dent Mater 1997 Sep;13(5):297-304**

OBJECTIVE: The aim of the present study was to determine whether removal of all amalgam restorations might significantly affect mercury levels in plasma and urine and whether the use of rubber dams might reduce patient exposure to mercury during amalgam removal.

METHODS: All amalgam restorations were removed from 18 subjects during a single treatment session in which a rubber dam was used and from 10 subjects when a rubber dam was not used. All amalgam restorations were removed by the same dentist using high-speed cutting, water coolant, and high-volume evacuation. The levels of mercury in plasma and urine were analyzed both before and during the subsequent twelve months after amalgam removal. In order to determine whether removal of all amalgam restorations might cause an exposure large enough to significantly increase the mercury levels in two indicator media for mercury exposure, i.e., plasma and urine, and to determine if the removal might cause a significant decrease in the mercury levels found over time, the one-tailed, paired Students' t-test was used. For each individual, the pre-removal levels were compared with both the levels found in plasma on d 1 and in urine on d 10, and also with the levels found 1 y after removal. Furthermore, in order to examine whether the use of rubber dams had any effect on the mercury levels found after removal, the changes in the mercury levels found were compared between the groups using the Wilcoxon-Mann-Whitney rank sum test.

RESULTS: **After removal of all amalgam restorations, only the non-rubber dam group showed significant increases in the mercury levels found in plasma ($p = 0.012$) and urine ($p = 0.037$).** However, one year later, the mercury levels in plasma and urine had sunk significantly below the pre-removal levels for both groups. When the changes in the mercury levels found were compared between the groups, the non-rubber dam group showed a significantly higher increase of mercury in plasma than the rubber dam group the day after removal ($p = 0.0010$). Compared to the pre-removal mercury levels in plasma and urine, the levels found 1 y after removal of all amalgam restorations were on average 52 +/- 23% (range 4-89%) lower in plasma and 76 +/- 21% (range 20-94%) lower in urine.

SIGNIFICANCE: The study showed that dental amalgam had a statistically significant impact on the mercury levels found in plasma and urine in the patients tested, and that the use of a rubber dam during removal of all amalgam restorations significantly reduced the peak of mercury in plasma following removal. PMID: 9823089, UI: 99040423

Systemic transfer of mercury from amalgam fillings before and after cessation of emission. Halbach S, Kremers L, Willruth H, Mehl A, Welzl G, Wack FX, Hickel R, Greim H Institute of Toxicology, Institute of Biomathematics and Biometry, GSF-

National Research Center for Environment and Health, Neuherberg, Oberschleissheim, D-85758, Germany. Environ Res 1998 May;77(2):115-23

In 29 volunteers with a low amalgam load, the number of amalgam-covered tooth surfaces and the occlusal area of the fillings were determined. Concentrations of total mercury were measured in plasma and erythrocytes as well as in urine together with the excretion rate. Absorbed daily doses were estimated from intraoral Hg emission by two separate methods. The transfer of Hg from the fillings via the oral cavity and blood to urinary excretion was evaluated according to the most representative combination of parameters. This consisted of urinary excretion (1), Hg concentration in plasma (2), absorbed dose (3), and occlusal area (4). Pairwise correlation coefficients were 0.75 for parameters 1 vs 2 and 2 vs 3 and 0.49 for parameters 3 vs 4. Within 9 days after removal of the fillings, a transient increase was observed in plasma Hg levels only. **This was reduced in those volunteers to whom a rubber dam had been applied during removal.** Peak plasma Hg was 0.6 ng/ml on average and decreased with halftimes between 5 and 13 days. A significant decrease in Hg excretion was noted not before 100 days after removal. Being relatively insensitive to dietary mercury, the determination of total mercury in plasma and of its urinary excretion rate appears, under practical aspects, most suitable for the investigation of Hg uptake from amalgam. Copyright 1998 Academic Press. PMID: 9600804, UI: 98263198

Mercury concentration in blood and urine--before and after the placement of dental amalgam fillings. [Article in German] Babisch W, Kovacic S, Krause C, Roulet JF, Thron JL, Hoffmann M Institut fur Wasser-, Boden- und Lufthygiene des Bundesgesundheitsamtes, Berlin. Zentralbl Hyg Umweltmed 1992 Aug;193(2):175-87

70 patients of dentist's surgery were given MOD amalgam fillings (non-gamma-2 amalgam) for molars. They were allocated for comparison to four groups defined by their treatment, i.e. the number of old and new restorations and whether a rubber dam was used. Blood and urine samples were collected at regular intervals before and during a 14-day period after treatment and tested for mercury concentration (Hg). Over the observation period the groups with the highest exposure (1-2 old restorations replaced by new ones) showed a tendency, in contrast to the less exposed groups (1 new filling with or without dam), towards increases (p less than 0.10) in group average Hg values of approx. 0.2 microgram/L (blood) and 0.3 microgram/g creatinine (urine) as acute treatment effects. The highest values recorded before and after the treatment, 3.3 micrograms/L (blood) and 16.5 micrograms/L (urine) are higher than normal but do not indicate any increase in the risk to health especially if they are not persistent. PMID: 1388618, UI: 93000323

Robert Gammal Comment;

Although this paper does not demonstrate the effect of the rubber dam it is just another one of those, which demonstrates a dramatic increase in mercury levels during amalgam placement and removal. There would not be many toxicologists who would agree with the conclusion though. An increase of 13 micrograms/L is a dramatic increase in urine levels. Especially in view of the fact that most of the mercury would not have been excreted. Compare these results with those of a DMPS challenge and the increase becomes even more significant.

Profile of respirable particulate produced during amalgam removal. Nimmo A., Werley M.S., Tansy M.F., and Martin J.S **J Dent Res. 68:Abstract 334, page 223, Mar 1989.**

ABSTRACT: Dentists frequently remove existing amalgam restorations with a high-speed handpiece utilizing water spray along with high-velocity evacuation. The purpose of this study was to evaluate the size and range of fully respirable (u) amalgam particles produced under the conditions listed above.

The patient model consisted of a manikin head and dentoform (Columbia Dentoform) connected to an Andersen Cascade Impactor particle sizer. The dentist model consisted of a particle sizer placed in the dentist's breathing zone. Nine MOD amalgam restorations were placed in maxillary premolar ivorine teeth with Tytin (Kerr) amalgam. The restorations were removed using a high-speed handpiece with water spray and high-velocity evacuation. The particle sizes were used to evaluate patient and dentist particulate inhalation for each restoration.

The patient model collected 4.00 + 2.60 mg of particulates ranging from <0.10 to 10 u, and having a mean particle size of 1.44 + 0.60 u. The dentist model collected 4.40 + 4.20 mg over a similar range with a mean particle size of 1.88 + 1.83 u.

These results suggest that both patient and dentist are subjected to similar particulate exposures during amalgam removal. Particle mass distribution was approximately equal across the range.

Particulate inhalation during the removal of amalgam restorations. Nimmo A, Werley MS, Martin JS, Tansy MF Department of Prosthodontics, Temple University, School of Dentistry, Philadelphia, Pa. J Prosthet Dent 1990 Feb;63(2):228-33

An aerosol that contains amalgam particles is created when a high-speed hand-piece is used to remove an existing amalgam restoration. Those particles smaller than 10 microns are considered to be fully respirable. This means that a significant percentage of the particles have the potential to travel to the terminal alveoli, where they may become lodged. Long-term exposure to fully respirable particles may compromise a person's respiratory function. Amalgam restorations were placed in the typodont teeth of a mannequin designed to simulate the head and the respiratory tract of a patient. The amalgam restorations were removed under three experimental conditions: dry cut (control), wet cut (water spray) with high-velocity evacuation, and wet cut with high-velocity evacuation and a rubber dam. Particulate exposure was evaluated in the simulated respiratory tracts of the patient and the dentist that were equipped with ambient particle sizing samplers. Use of water spray and high-velocity evacuation significantly reduced patient exposure to particles. The use of a rubber dam, together with water spray and high-velocity evacuation, was responsible for a further significant reduction of exposure to particles when compared with water spray and high-velocity evacuation alone. The dentist, however, was exposed to moderate levels of fully respirable particles for all conditions tested. It is therefore recommended that all dental personnel wear face masks while removing existing amalgam restorations.

Filtration efficiency of dental face masks during amalgam removal. Nimmo A., Werley M.S., Tansy M.F. and Martin J.S. J Dent Res. 68:Abstract 333, page 223, Mar 1989.

ABSTRACT: Dentists are exposed to moderate amounts of fully respirable particulates during amalgam removal (Nimmo et al. J Dent Res 67:335, 1988). The purpose of this study was to evaluate the in vitro filtration efficiency of two types of dental face masks during amalgam removal.

MOD amalgam restorations were placed in 40 ivorine premolar teeth using Tytin (Kerr) amalgam. Restorations were removed from two teeth during a 14 min period using a high-speed handpiece without water spray. The aerosol produced was analyzed by two Anderson Cascade Impactor samplers; one served as the control (C), while the other was equipped with a dental face mask. The two face masks tested were a tie-on (TO) mask (3M Tie-on Surgical Mask, model 1818), and a molded cup type (CT) mask (3M Aseptex Face Mask, Model 1942). These procedures were repeated for a total of 10 samples of two amalgams removed for each mask type. Paired t-test analyses were done.

The TO mask reduced the amount of particulates collected from 4.10 + 3.62 mg (C) to 0.98 + 1.58 mg. The CT mask reduced the amount of particulates collected from 5.36 +7.05 mg (C) to 0.45 + 0.77 mg. The average size of particles collected was 3.76 + - 3.10 u (C) compared to 0.68 + 0.91 u (TO) and 3.71 + 3.91 u (C) compared to 0.47 + 0.82 u (CT). The TO mask demonstrated a significant reduction ($p<0.05$) in the amount and size of fully respirable particulates produced during amalgam removal.

Amalgam Mercury In Mothers' Milk Risk To Infants!

A newly published study has firmly established the presence of mercury from dental amalgam in the milk of nursing females! [Vimy, MJ; et al, 1997] Since this is a matter of the utmost importance, BioProbe has reviewed the existing literature on the subject, with the pertinent studies abstracted below in the Science section.

Several studies have already established the transfer of dental amalgam mercury into the tissues of unborn babies, in both animals and humans. [Vimy, MJ; 1990; Drasch, G; et al, 1994] The study on humans by Drasch and associates concluded: "Future discussion on the pros and cons of dental amalgam should not be limited to adults or children with their own amalgam fillings, but also include fetal exposure. The unrestricted application of amalgam for dental restorations in women before and during the child bearing age should be reconsidered." The publication of these studies has already resulted in the issuing of government advisories against the use of mercury amalgam dental fillings in pregnant females (Germany, Sweden and Canada). In Germany, public opinion is encouraging a ban on the use of dental amalgam in all fertile women. The current research on amalgam mercury in breast milk adds further evidence to the wisdom of such an action.

The ability of metal ions to concentrate in mothers' milk has been scientifically established for years, as has the ability for methyl mercury to transfer to breast milk and cause neurologic damage to infants. [AminZaki, L; et al, 1981] The investigation of the possible transfer of mercury specifically from amalgam dental fillings to mothers' milk began in 1990. A study by Vimy and associates implanted amalgam fillings, seeded with radioactively labeled mercury, into pregnant ewes. [Vimy, MJ; et al, 1990] Since radioactively labeled mercury does not occur naturally, it was possible to detect mercury in tissues that was specifically derived from the amalgam dental fillings. The amalgam mercury was found to quickly accumulate in tissues of mothers and fetuses. In the lactating ewes, the levels of labeled mercury in milk were as much as six times higher than the levels of labeled mercury in their blood.

The current study [Vimy, MJ; et al, 1997] evaluated mercury related to amalgam dental fillings transferring to breast milk in both animals and humans. In the animal study, lactating ewes with amalgam fillings nursed foster lambs from ewes without amalgam fillings. The amalgam fillings contained a portion of radioactively labeled mercury, which was found in the tissues of the foster lambs. This confirmed the transfer of mercury from the amalgam fillings of the mothers, into the breast milk, then into the tissues of the foster lambs. The human study examined mercury levels in breast milk of 33 lactating women. The mercury levels correlated with the number of amalgam fillings or mercury vapor concentration levels in mouth air. The infant exposure levels were compared to the United States Public Health Service Minimal Risk Level (MRL) standard for adults, and caution was urged. The combination of prenatal mercury exposure and lactating exposure to maternal amalgam mercury was addressed. Other important factors addressed were mercury exposures related to the differences in body mass between infants and adults and the particular sensitivity of infants to heavy metal toxic effects. This latter concern has also been pointed out by other authors. [Schümann, 1990]

By 1995, the comparison of activity of different forms of mercury had been investigated. [Schümann, K, 1990; Yoshida, M; et al, 1994; Oskarsson, A; et al, 1995] It has been found that any form of mercury can transfer to breast milk and, from there, into the tissues of infants, although the fat soluble forms of mercury (methyl mercury and mercury vapor) will concentrate more in brain tissue of infants. The Schümann study pointed out that milk increases the bioavailability of Hg++ as the ionic mercury is bound to a greater extent in the red blood cells of the suckling infants. In an evaluation of lactating human females, the study by Oskarsson and associates found that dental amalgam mercury transferred to mothers' milk, but that methyl mercury from consumption of fish correlated to mercury levels in blood but not to levels in milk. In the portion of the study on rats and mice, the mercury was found to cause pathologic effects in the offspring, including alteration of the thymocytes, increased lymphocyte activities, and effects on noradrenaline and nerve growth factor in the developing brains. These effects occurred in the animals exposed to methyl mercury.

It has been well established scientifically that mercury vapor, being lipid soluble, functions very similar to methyl mercury pathologically. There have been other studies confirming the harmful effect of mercury vapor on unborn babies and developing infants. [Danielsson, BR; et al, 1993; Warfinge, K; et al, 1994; Fredriksson, A; et al, 1996] It should be emphasized that the studies cited herein clearly show that mercury damage to unborn babies and infants is not readily observable early on. The neurologic damage is developmental in nature, primarily effecting learning, behavior and neurologic function. These effects can dramatically alter the functioning of the individual throughout life. Early exposure to inorganic or organic mercury can even result in mental retardation. [Schümann, K, 1990]

In a subsequent study, Oskarsson and colleagues confirmed the accumulation of dental amalgam mercury in mothers' milk. [Oskarsson, A; et al, 1996] This study found that amalgam mercury dental fillings were the main source of mercury in the milk of lactating humans, related the exposure to the World Health Organization standard for daily intake for adults, and concluded it to be significant enough to be a risk to infants.

At this point, the scientific evidence clearly establishes that mercury transfers from amalgam dental fillings to the tissues of unborn babies and to mothers' milk, from the milk to body tissues of infants, and, according to existing standards, presents a health risk to the infants. The combination of prenatal exposure and neonatal exposure from nursing presents an undeniable concern. Oskarsson and associates

[1996] stated: "We concluded that efforts should be made to decrease mercury burden in fertile women." Since amalgam dental fillings have now been identified as a significant, if not the major, contributor of mercury in mothers' milk, the formal regulatory limitation of amalgam fillings in fertile women is clearly indicated! BioProbe now calls upon all responsible public officials to immediately initiate action to protect unborn babies and infants from the scientifically proven health risk of mercury exposure from the amalgam fillings of their mothers. The use of mercury amalgam fillings in all fertile women should be banned forthwith!

More References

Mercury From Maternal "Silver" Tooth Fillings in Sheep and Human Breast Milk: A Source of Neonatal Exposure. Vimy, MJ; Hooper, DE; King, WW; Lorscheider, FL. Biological Trace Element Res., 56:14352, 1997.

ABSTRACT: Neonatal uptake of Hg from milk was examined in a pregnant sheep model, where radioactive mercury (Hg203)/silver tooth fillings (amalgam) were newly placed. A crossover experimental design was used in which lactating ewes nursed foster lambs. In a parallel study, the relationship between dental history and breast milk concentration of Hg was also examined.

Results from the animal studies showed that, during pregnancy, a primary fetal site of amalgam Hg concentration is the liver, and, after delivery, the neonatal lamb kidney receives additional amalgam Hg from mother's milk. In lactating women with aged amalgam fillings, increased Hg excretion in breast milk and urine correlated with the number of fillings or Hg vapor concentration levels in mouth air.

It was concluded that Hg originating from maternal amalgam tooth fillings transfers across the placenta to the fetus, across the mammary gland into milk ingested by the newborn, and ultimately into neonatal body tissues. Comparisons are made to the U.S. minimal risk level recently established for adult Hg exposure. These findings suggest that placement and removal of "silver" tooth fillings in pregnant and lactating humans will subject the fetus and neonate to unnecessary risk of Hg exposure.

Total and Inorganic Mercury in Breast Milk in Relation to Fish Consumption and Amalgam in Lactating Women. Oskarsson, A; Schültz, A; Skerfving, S; Hallén, IP; Ohlin, B; Lagerkvist, BJ. Arch Environ Health, 51(3):23451, 1996.

ABSTRACT: Total mercury concentrations (mean ±standard deviation) in breast milk, blood, and hair samples collected 6 wk after delivery from 30 women who lived in the north of Sweden were 0.6 ± 0.4 ng/g (3.0 ± 2.0 nmol/kg), 2.3 ±1.0 ng/g (11.5 ±5.0 nmol/kg), and 0.28 ± 0.16 microg/g (1.40 ± 0.80 micromol/kg) respectively. In milk, an average of 51% of total mercury was in the form of inorganic mercury, whereas in blood an average of only 26% was present in the inorganic form.
Total and inorganic mercury levels in blood ($r = .55$, $p = .003$; and $r = .46$, $p = .016$; respectively) and milk ($r = .55$, $p = .01$; and $r = .45$, $p = .018$; respectively) were correlated with the number of amalgam fillings. The concentrations of total mercury and organic mercury (calculated by subtraction of inorganic mercury from total Mercury) in blood ($r = .59$, $p = .0006$, and $r = .56$, $p = .001$; respectively) and total mercury in hair ($r = .52$, $p = .006$) were correlated with the estimated recent exposure to methyl mercury via intake of fish.

There was no significant correlation between the milk levels of mercury in any chemical form and the estimated methyl mercury intake. A significant correlation was found between levels of total mercury in blood and in milk (r = .66, p = .0001), with milk levels being an average of 27% of the blood levels. There was an association between inorganic mercury in blood and milk (r = .96, p .0001); the average level of inorganic mercury in milk was 55% of the level of inorganic mercury in blood. No significant correlations were found between the levels of any form of mercury in milk and the levels of organic mercury in blood.
The results indicated that there was an efficient transfer of inorganic mercury from blood to milk and that, in this population, mercury from amalgam fillings was the main source of mercury in milk. Exposure of the infant to mercury from breast milk was calculated to range up to 0.3 microg/kg x d, of which approximately onehalf was inorganic mercury. This exposure, however, corresponds to approximately onehalf the tolerable daily intake for adults recommended by the World Health Organization. We concluded that efforts should be made to decrease mercury burden in fertile women.

Exposure to Toxic Elements Via Breast Milk. Oskarsson, A; Palminger Hallaen, I; Sundberg, J. Analyst, 120(3):76570, 1995.

ABSTRACT: Breast milk is the ideal nutrient for the newborn, but unfortunately also a route of excretion for some toxic substances. Very little attention has been paid to breast milk as a source of exposure to toxic elements. The dosedependent excretion in breast milk and the uptake in the neonate of inorganic mercury, methyl mercury and lead were studied in an experimental model for rats and mice.
The transfer of mercury from plasma to milk was found to be higher in dams exposed to inorganic mercury than to methyl mercury. In contrast, the uptake of mercury from milk was higher in the sucklings of dams exposed to methyl mercury than to inorganic mercury. Pre and postnatal exposure to methyl mercury resulted in increased numbers and altered proportions of the thymocyte subpopulation and increased lymphocyte activities in the offspring of mice and also effects on the levels of noradrenaline and nerve growth factor in the developing brain of rats.
Mercury in blood and breast milk in lactating women in Sweden was studied in relation to the exposure to mercury from fish and amalgam. Low levels were found; the mean levels were 0.6 ng/g1 in milk and 2.3 ng/g1 in blood. There was a statistically significant correlation between mercury levels in blood and milk, showing that milk levels were approximately 30% of the levels in blood. Inorganic mercury exposure from amalgam was reflected in blood and milk mercury levels. Recent exposure to methyl mercury from consumption of fish was reflected in mercury levels in the blood but not in milk. (Abstract truncated at 250 words.)

Maternal Fetal Distribution of Mercury (203Hg) Released From Dental Amalgam Fillings. Vimy, MJ; Takahashi, Y; Lorscheider, FL.
Amer J Physiol., 258(RICP 27):R93945, 1990.

ABSTRACT: In humans, the continuous release of Hg vapor from dental amalgam tooth restorations is markedly increased for prolonged periods after chewing. The present study establishes a timecourse distribution for amalgam Hg in body tissues of adult and fetal sheep.
Under general anesthesia, five pregnant ewes had twelve occlusal amalgam fillings containing radioactive 203Hg placed in teeth at 112 days gestation. Blood, amniotic fluid, feces, and urine specimens were collected at 1 to 3 day intervals for 16 days. From days 16-140 after amalgam placement (16-41 days for fetal lambs), tissue

specimens were analyzed for radioactivity, and total Hg concentrations were calculated.
Results demonstrate that Hg from dental amalgam will appear in maternal and fetal blood and amniotic fluid within 2 days after placement of amalgam tooth restorations. Excretion of some of this Hg will also commence within 2 days. All tissues examined displayed Hg accumulation. Highest concentrations of Hg from amalgam in the adult occurred in kidney and liver, whereas in the fetus the highest amalgam Hg concentrations appeared in liver and pituitary gland. The placenta progressively concentrated Hg as gestation advanced to term, and milk concentration of amalgam Hg postpartum provides a potential source of Hg exposure to newborn.

It is concluded that accumulation of amalgam Hg progresses in maternal and fetal tissues to a steady state with advancing gestation and is maintained. Dental amalgam usage as a tooth restorative material in pregnant women and children should be reconsidered.

Mercury Burden of Human Fetal and Infant Tissues. Drasch, G; Schupp, I; Hofl, H; Reinke, R; Roider, G. Pediatrics, 153(8):60710, 1994.

ABSTRACT: The total mercury concentrations in the liver (HgL), the kidney cortex (HgK) and the cerebral cortex (HgC) of 108 children aged 1 day to 5 years, and the HgK and HgL of 46 fetuses were determined. As far as possible, the mothers were interviewed and their dental status was recorded. The results were compared to mercury concentrations in the tissues of adults from the same geographical area. The HgK (n= 38) and HgL (n= 40) of fetuses and HgK (n= 35) of older infants (11-50 weeks of life) correlated significantly with the number of dental amalgam fillings of the mother. The toxicological relevance of the unexpected high HgK of older infants from mothers with higher numbers of dental amalgam fillings is discussed.

CONCLUSION: Future discussion on the pros and cons of dental amalgam should not be limited to adults or children with their own amalgam fillings, but also include fetal exposure. The unrestricted application of amalgam for dental restorations in women before and during the child bearing age should be reconsidered.

Milk Transfer and Tissue Uptake of Mercury in Suckling Offspring After Exposure of Lactating Maternal Guinea Pigs to Inorganic or Methyl mercury. Yoshida, M; Watanabe, C; Satoh, H; Kishimoto, Y.
Arch Toxicol, 68(3):1748, 1994.

ABSTRACT: Maternal guinea pigs were injected with mercuric chloride ($HgCl_2$; 1 mg Hg/Kg body weight) or methyl mercury (MeHg; 1 mg Hg/kg) 12 h after parturition, and exposure of the offspring to mercury (Hg) via breast milk were studied on days 3, 5 and 10 postpartum. Milk Hg concentrations were lower than maternal plasma Hg concentrations regardless of the form of Hg given to the dams.
Milk Hg was higher in $HgCl_2$ treated dams than in MeHg treated dams. In MeHg treated dams, MeHg was separately determined. While the ratio of MeHg to THg decreased in the dams' plasma, it did not in the milk. There was a strong correlation between milk and plasma THg concentrations in $HgCl_2$ treated dams. In the milk of MeHg treated dams, the plasma MeHg concentrations correlated better than did the plasma THg concentrations.

In the offspring, regardless of the chemical forms of Hg given to the dams, the highest Hg concentrations were found in the kidney, followed by the liver and the brain. Brain Hg concentrations were, however, significantly higher in the offspring of MeHg treated dams than in those of HgCl2 treated dams. In addition, Hg levels in the major organs of the offspring of HgCl2 treated peaked on day 5 postpartum, while those of MeHg treated dams did not show a significant decrease up to day 10 postpartum.
These facts indicate that the two chemical forms of Hg were transferred to the offspring via the breast milk and were distributed differently, depending on the chemical form, to the offspring's tissues.

Methyl mercury Poisoning in the Iraqi Suckling Infant: A Longitudinal Study Over Five Years. AminZaki, L; Majeed, MA; Greenwood, MR; Elhassani, SB; Clarkson, TW; Doherty, RA. J Appl Toxicol, 1(4):2104, 1981.

ABSTRACT: In a five year longitudinal study of mothers and infants exposed to methyl mercury during the Iraq epidemic of 1972, the frequencies of signs and symptoms exhibited by the mothers were typical of methyl mercury poisoning. When blood concentrations of mercury are corrected to 1 March 1972, mothers with the most severe signs and symptoms had an average blood mercury concentration significantly higher (p less than 0.01) than either the milder or asymptomatic groups.
Analytical data indicate that the predominant route of exposure for the infant was through breast milk in which approximately 60% of total mercury was determined, by cold vapor atomic absorption, to be organic mercury. Abnormal neurological signs in these infants became more obvious with time: hyperreflexia was observed in 8 of 22 infants at first examination, and in 17 of 22 at second examination. Delayed motor development became evident at the second and third examinations. The frequency of pathological reflexes and delayed motor developmental milestones was so high as to be considered significant even the absence of a controlled study. There was no increase in mortality as compared to a control group.

The Toxicological Estimation of the Heavy Metal Content (Cd, Hg, Pb) in Food for Infants and Small Children. Schümann, K. Z Ernahrungswiss, 29(1):5473, 1990.

ABSTRACT: There are differences between young and adult organisms regarding toxokinetic aspects and clinical manifestations of heavy metal intoxications. Chronically, toxic Cd intake causes a microcytotic hypochromic anemia in young rats at lower exposure levels and after shorter exposure periods than in adult animals. Cd absorption is increased by coadministration of milk and in conjunction with iron deficiency. After long exposure periods toxic Cd concentrations accumulate in the kidney cortex; this process starts very early in life. In 3 year old children Cd concentrations in the kidney can reach up to onethird of those found in adults.
Hg++ and methyl Hg can cause Hg encephalopathia, and frequently cause mental retardation in adults. Correspondingly, Hg++ accumulation in the brains of suckling rats is approx. 10 times higher than in grown animals. Milk increases the bioavailability of Hg++. In suckling rats Hg is bound to a greater extent to ligands in the erythrocytes. Methyl Hg concentrations in breast milk reach 5% of those in maternal plasma and that is a severe hazard for breastfed children of exposed mothers.

Toxic Pb concentrations can lead to Pb encephalopathia. A high percentage of surviving children have seizures and show signs of mental retardation. Anemia and reduced intelligence scores were recently observed in children after exposure to very low levels of Pb. Pb absorption is increased in children and after coadministration of Milk.
The average heavy metal uptake from such diets [infant formulas or breast milk] exceeds the provisional tolerable weekly intake levels set by the WHO for adults, calculated on the basis of an average food intake and a down scaled body weight. These considerations do not even provide for differences in absorption and distribution or for the increased sensitivity of children to heavy metal exposure. [Abstract truncated.]

Behavioral Effects of Prenatal Metallic Mercury Inhalation Exposure in Rats. Danielsson, BR; Fredriksson, A; Dahlgren, L; G'ardlund, AT; Olsson, L; Dencker, L; Archer, T. Neurotoxicol Teratol., 15(6):3916, 1993.

ABSTRACT: The effects of administration by inhalation of metallic mercury vapour (Hg0) to pregnant rats, approximately corresponding to doses of 0.2 mg Hg0/kg/day (high dose) or 0.07 mg Hg0/kg/day (low dose), on the developmental and behavioral repertoire of the offspring were studied. Exposure occurred during days 11-14 plus 17-20 of gestation. The dose levels were selected so as not to induce maternal toxicity.
Maturation variables such as surface righting, negative geotaxis, pinna unfolding, and tooth eruption revealed no difference between Hg0treated offspring and controls. Tests of spontaneous motor activity showed that the Hg0treated offspring were hypoactive at 3 months of age, but hyperactive at 14 months. In spatial learning tasks the prenatally exposed offspring showed retarded acquisition in the radial arm maze but no differences in circular swim maze. A simple test of learning, habituation to a novel environment (activity chambers), indicated a reduced ability to adapt.
These data suggest that prenatal exposure to Hg0 vapour results in similar behavior changes in the offspring as reported for methyl mercury.

The Effect on Pregnancy Outcome and Fetal Brain Development of Prenatal Exposure to Mercury Vapour. Warfinge, K; Berlin, M; Logdberg, B. Neurotoxicology, 15(4), 1994.

ABSTRACT: Fourteen pregnant female squirrel monkeys were exposed to mercury vapour (Hg0) 5 days/week from 57 weeks of gestation until delivery in an exposure chamber. Hg0 exposure varied from 1 mg/m3 for 22 hr/d (1 monkey), 7 hr/d or 4 hr/d to 0.5 mg/m3 for 7 hr/d or 4 hr/d. Hg concentration in maternal blood ranged 0.05-0.09 mcg/g.
There was a dose related increase in abortion rate and perinatal mortality in the exposed monkeys compared to unexposed controls. The morphology of perinatally sacrificed or succumbed offspring brains showed signs of migration disturbances such as increased cell density in the cerebral subcortical white matter, abnormal cell collections near the cerebral lateral ventricles. Autometallographically, Hg was preferentially localized in the heterotopic cells and in the verticular aspects of the pseudostratified neuroepithelium. Hg concentration in the brain of exposed offspring ranged 0.01-0.70 mcg/g.
Autometallography of the maternal brains revealed that the pyramidal neurons of neocortical layer V contained more visualized Hg than the other neurons. In the offspring brains, Hg was visualized throughout the whole neocortex and no laminar

distribution pattern was found. In the fiber systems, the offspring brains contained more Hg than the adult brains. In the cerebellum, the Purkinje cells, the Bergmann glial cells, the astrocytes of the medullary layer and the deep cerebellar nuclei were the main targets, for Hg accumulation in both maternal and offspring brains.

Prenatal Coexposure to Metallic Mercury Vapour and Methyl mercury Produce Interactive Behavioral Changes in Adult Rats. Fredriksson, A; Dencker, L; Archer, T; Danielsson, BR. Neurotoxicol Teratol., 18(2):12934, 1996.

ABSTRACT: Pregnant rats were 1) administered methyl mercury (MeHg) by gavage, 2 mg/kg/day during days 69 of gestation, 2) exposed by inhalation to metallic mercury (Hg0) vapor (1.8 mg/m3 air for 1.5 h per day) during gestation days 1419, 3) exposed to both MeHg by gavage and Hg0 vapor by inhalation (MeHg + Hg0), or 4) were given combined vehicle administration for each of the two treatments (control). The inhalation regimen corresponded to an approximate dose of 0.1 mg Hg0/kg/day.
Clinical observations and developmental markers up to weaning showed no differences between any of the groups. Testing of behavioral function was performed between 4 and 5 months of age and included spontaneous motor activity, spatial learning in a circular bath, and instrumental maze learning for food reward.
Offspring of dams exposed to Hg0 showed hyperactivity in the motor activity test chambers over all three parameters: locomotion, rearing and total activity; this effect was potentiated in the animals of the MeHg + Hg0 group. In the swim maze test, the MeHg + Hg0 and Hg0 groups evidenced longer latencies to reach a submerged platform, which they had learned to mount the day before, compared to either the control or MeHg group. In the modified, enclosed radial arm maze, both the MeHg + Hg0 and Hg0 groups showed more ambulations and rearings in the activity test prior to the learning test. During the learning trial, the same groups (i.e., MeHg + Hg0 and Hg0) showed longer latencies and made more errors in acquiring all eight pellets.
Generally, the results indicate that prenatal exposure to Hg0 causes alterations to both spontaneous and learned behaviours, suggesting some deficit in adaptive functions. Coexposure to MeHg, which by itself did not alter these functions at the dose given in this study, served to significantly aggravate the change.

Amalgam Manufacturer States

Their Product Is Dangerous

Dispersalloy Amalgam

A new age is dawning! Below is an abstract from the internet home page of Caulk Company, the manufacturer of on of the most popular dental amalgams on the market. We now have the ludicrous situation where the Australian Dental Association claims that there is no evidence of harm from amalgams and the manufacturer states that there are clear contraindications to its use. They go on to list most of the symptoms caused by mercury poisoning from amalgam!

The information below was taken form their web site at http://www.caulk.com/MSDSDFU/DispersDFU.html. The information was removed very soon after it was placed when many people discovered it.

Stated by Caulk Co.:

Contraindication

The use of amalgam is contraindicated;

- In proximal or occlusal contact to dissimilar metal restorations.
- In patients with severe renal deficiency.
- In patients with known allergies to amalgam.
- For retrograde or endodontic filling.
- As a filling material for cast crown.
- In children 6 and under.
- In expectant mothers.

Precautions

The number of amalgam restorations for one patient should be kept to a minimum.

Inhalation of mercury vapor by dental staff may be avoided by proper handling of the amalgam, the use of masks, along with adequate ventilation.

Avoid contact with skin and wear safety glasses and gloves.

Store amalgam scrap in well sealed containers. Regulations for disposal must be observed.

Health affects and first aid

Inhalation: Chronic: Inhalation of mercury vapor over a long period may cause mercurialism which is characterized by fine tremors and erethism. Tremors may affect the hands first, but may also become evident in the face, arms, and legs. Erethism may be manifested by abnormal shyness, blushing, self-consciousness, depression or despondency resentment of criticism, irritability or excitability, headache, fatigue, and insomnia. In severe cases, hallucinations, loss of memory, and mental deterioration may occur. Concentrations as low and 0.03 mg/m3 have induced psychiatric symptoms in humans. Renal involvement may be indicated by proteinuria, albuminuria, enzymuria, and anuria. Other effects may include salivation, gingivitis, stomatitis, loosening of the teeth, blue lines on the gums, diarrhea, chronic pneumonitis and mild anemia. Repeated exposure to mercury and its compounds may result in sensitization. Intrauterine exposure may result in tremors and involuntary movements in the infants. Mercury is excreted in breast milk. Paternal reproductive effects and effects on fertility have been reported in male rats following repeated inhalation exposures.
First Aid: Remove from exposure area to fresh air immediately. If breathing has stopped, give artificial respiration. Maintain airway and blood pressure and administer oxygen if available. Keep affected person warm and at rest. Treat symptomatically and supportively. Administration of oxygen should be performed by qualified personnel. Get medical attention immediately.

Mercury's Effects on the Heart

Marked Elevation of Myocardial Trace Elements in Idiopathic Dilated Cardiomyopathy Compared With Secondary Dysfunction

Frustaci, A., Magnavita, N., Chimenti, C., Caldarulo, M., Sabbioni, E., Pietra, R., Cellini, C., Possati, G.F. and Maseri, A. Journal of the American College of Cardiology Vol. 33, No. 6, 1999, pp. 1578-1583

Objectives: We sought to investigate the possible pathogenic role of myocardial trace elements (TE) in patients with various forms of cardiac failure.

Background: Both myocardial TE accumulation and deficiency have been associated with the development of heart failure indistinguishable from an idiopathic dilated cardiomyopathy.

Methods: Myocardial and muscular content of 32 TE has been assessed in biopsy samples of 13 patients (pts) with clinical, hemodynamic and histologic diagnosis of idiopathic dilated cardiomyopathy (IDCM), all without past or current exposure to TE. One muscular and one left ventricular (LV) endomyocardial specimen from each patient, drawn with metal contamination-free technique, were analyzed by neutron activation analysis and compared with 1) similar surgical samples from patients with valvular (12 pts) and ischemic (13 pts) heart disease comparable for age and degree of LV dysfunction; 2) papillary and skeletal muscle surgical biopsies from 10 pts with mitral stenosis and normal LV function, and 3) LV endomyocardial biopsies from four normal subjects.

Results: A large increase (>10,000 times for mercury and antimony) of TE concentration has been observed in myocardial but not in muscular samples in all pts with IDCM. Patients with secondary cardiac dysfunction had mild increase (<5 times) of myocardial TE and normal muscular TE. In particular, in pts with IDCM mean mercury concentration was 22,000 times (178,400 ng/g vs. 8 ng/g), antimony 12,000 times (19,260 ng/g vs. 1.5 ng/g), gold 11 times (26 ng/g vs. 2.3 ng/g), chromium 13 times (2,300 ng/g vs. 177 ng/g) and cobalt 4 times (86.5 ng/g vs. 20 ng/g) higher than in control subjects.

Conclusions: A large, significant increase of myocardial TE is present in IDCM but not in secondary cardiac dysfunction. The increased concentration of TE in pts with IDCM may adversely affect mitochondrial activity and myocardial metabolism and worsen cellular function.

Effects of mercury on the isolated heart muscle are prevented by DTT and cysteine.

Vassallo DV, Moreira CM, Oliveira EM, Bertollo DM, Veloso TC

Toxicol Appl Pharmacol 1999 Apr 15;156(2):113-8

The protective effects of dithiothreitol (DTT, 50 &mgr;M) and cysteine (CYS, 100 &mgr;M) against toxic effects of HgCl2 (1, 2.5, 5, and 10 &mgr;M) were studied in isolated, isometrically contracting rat papillary muscles. Force reduction promoted by Hg2+ was prevented by both DTT and CYS. Also, after both treatments, no significant changes in dF/dt were observed. A progressive reduction in the time to peak tension was observed when increased concentrations of HgCl2 were used after CYS and DTT treatment. This was an indication that the enhancement of calcium release from the sarcoplasmic reticulum produced by mercury was not affected by DTT and CYS. Tetanic contractions were also studied. After treatment with DTT or CYS tetanic tension did not change. No significant reduction of tetanic tension was

observed during treatment with 1 &mgr;M Hg2+ but its reduction was observed after 5 &mgr;M Hg2+. Myosin ATPase activity was also affect by Hg2+, being completely blocked by 1 &mgr;M Hg2+ and reduced by 50% with 0.15 &mgr;M Hg2+. Full activity was restored by using 500 nM DTT. These findings suggest that several but not all toxic effects of Hg2+ on the mechanical activity of the heart muscle are prevented by protectors of SH groups such as DTT and CYS. The enhancement of the Ca2+ release from the sarcoplasmic reticulum by Hg2+ during activation was not affected by prior treatment with DTT and CYS, suggesting that interactions with SH groups may not be important for the activation of the Ca2+ channel of the sarcoplasmic reticulum

The chamber exposure of laboratory rats to metal oxides originating from metal producing industry. Kovacikova Z, Chorvatovicova D

Physiol Res 1997;46(1):41-5

Laboratory rats were exposed to the inhalation of dust from an agglomeration unit which is the greatest contributor to dust pollution in the vicinity of a mercury producing plant. The exposure lasted for 6 months (4 hours daily, 5 days per week), the concentration of aerosol in the chamber was 10 mg x m(-3). After finishing the exposure, the animals were examined and compared with the controls which were held under standard laboratory conditions. The number of alveolar macrophages was highly elevated (P< 0.001) in the exposed animals, Mg2+ ATPase activity in the heart muscle was decreased. The alanine aminotransferase activity in the serum was not changed, the aspartate aminotransferase was slightly enhanced. No differences in the frequency of abnormal sperm and in the frequency of polychromatic erythrocytes in bone marrow were detected.

Mercury effects on the contractile activity of isolated heart muscle. Oliveira EM, Vassallo DV, Sarkis JJ, Mill JG

Toxicol Appl Pharmacol 1994 Sep;128(1):86-91

The toxic effects of HgCl2 (1, 2.5, 5, and 10 microM) were studied in isolated, isometrically contracting rat papillary muscles and frog ventricular strips. In rat papillary muscles 1 microM Hg2+ produced a small increase in the force of contraction. Higher concentrations of HgCl2 produced a dose-dependent decrease in contractile force. The rate of force development was affected differently, increasing at 1 and 2.5 microM Hg2+ and decreasing to control levels at 5 and 10 microM Hg2+. This was the result of a progressive reduction in the time to peak tension observed when HgCl2 concentrations increased. This effect probably reflects the binding of Hg2+ to SH groups inducing Ca2+ release from the sarcoplasmic reticulum. The relative potentiation of postrest contractions was used as an index of sarcoplasmic reticulum activity. It was measured after pauses of increasing duration and was reduced at concentrations of 1 microM Hg2+ when compared to that of the control. A further decrement in the relative potentiation was observed with higher Hg2+ concentrations, indicating that the activity of the sarcoplasmic reticulum was depressed by mercury in a dose-dependent manner. Tetanic contractions were also studied in the rat myocardium. The tetanic tension did not change during treatment with 1 microM Hg2+ but decreased with 5 microM Hg2+, suggesting a toxic effect on the contractile proteins only at high Hg2+ concentrations. Frog ventricular strips were studied using the same HgCl2 concentrations and no effects on either force or relative potentiation were observed. These findings suggest that Hg2+ promotes dose-dependent toxic effects on heart

muscle via actions on the sarcolemma, the sarcoplasmic reticulum, and contractile proteins.

Mercury compounds: lipophilicity and toxic effects on isolated myocardial tissue. Halbach S

Arch Toxicol 1990;64(4):315-9

Lipophilicity is suggested to modulate the diffusion and the cytotoxic effects of mercury compounds. To investigate this, the positive inotropic effect of four Hg compounds (HgCl2, CH3HgCl, chlormerodrin, bromomercurihydroxypropane) was studied in catecholamine-depleted isolated heart muscle preparations. The rate of development of the positive effect was inversely correlated to the concentration in the case of HgCl2 and chlormerodrin, i.e. the product of concentration (c) and time to half-maximal effect (t50) remained constant. This was in accordance with the assumption of a permeation-controlled rate of action, as was shown earlier for p-chloromercuriphenyl-sulfonic acid. In addition, the c X t50 values of the individual mercurials decreased hyperbolically with the increase in lipophilicity as measured by the octanol/water partition. The results support the view that the toxicity of mercurials increases with their lipid solubility. In conjunction with the previously reported negative inotropic effect of Hg compounds, a model is proposed allocating thiol groups responsible for the negative inotropic action to lipid compartments within the cell membrane, while SH groups conveying the increase in contraction force are thought to be situated at the internal surface of the sarcolemma.

The relationship between mercury from dental amalgam and the cardiovascular system. Siblerud RL Sci Total Environ 1990 Dec 1;99(1-2):23-35

The findings presented here suggest that mercury poisoning from dental amalgam may play a role in the etiology of cardiovascular disorders. Comparisons between subjects with and without amalgam showed amalgam-bearing subjects had significantly higher blood pressure, lower heart rate, lower hemoglobin, and lower hematocrit. Hemoglobin, hematocrit, and red blood cells were significantly lower when correlated to increased levels of urine mercury. The amalgam subjects had a greater incidence of chest pains, tachycardia, anemia, fatigue, tiring easily, and being tired in the morning. The data suggest that inorganic mercury poisoning from dental amalgam does affect the cardiovascular system.

Hemodynamic and electrophysiological effects of mercury in intact anesthetized rabbits and in isolated perfused hearts. Rhee HM, Choi BH

Exp Mol Pathol 1989 Jun;50(3):281-90

Using intact anesthetized rabbits and isolated perfused hearts, the hemodynamic and electrophysiological effects of mercury (Hg) were examined in order to assess the role of cardiovascular dysfunction in Hg intoxication. The most consistent and prominent cardiovascular effect was a significant reduction in blood pressure. This cardiodepressive action was probably brought about by the primary action of Hg on the heart rather than by altered sympathetic activity, as evidenced by normal renal nerve activity at times when the hemodynamic actions of Hg were clearly manifest. Although the principal target organ for the toxic actions of inorganic Hg is the kidney, chronic exposure to both inorganic and organic Hg frequently results in signs and symptoms of CNS dysfunction. The profound hemodynamic effects of Hg

that we have observed emphasize the potential importance of Hg cardiotoxicity and indicate the need to differentiate between the primary and the secondary effects of Hg intoxication on CNS tissues for evaluation of the toxic effects of Hg compounds.

Cardiovascular homeostasis in rats chronically exposed to mercuric chloride. Carmignani M, Boscolo P Arch Toxicol Suppl 1984;7:383-8

Two groups of male Sprague-Dawley rats received from weaning 50 micrograms/ml of mercury as mercuric chloride (HgCl2) in drinking water for 320 and 350 days. Hg exposure increased cardiac inotropism, without chronotropic changes, in both groups, and induced arterial hypertension in the rats exposed for 350 days. In the exposed rats, cardiovascular responses to the stimulation of peripheral alpha and beta adrenoceptors were decreased and increased, respectively, possibly through a reduced intracellular availability of calcium ions for contractile mechanisms. Hg exposure did not affect either vagal or sympathetic activity or cardiovascular reactivity to several physiological agonists. On the other hand, Hg exposure induced baroreflex hyposensitivity and produced a drastic alteration of the levels of copper and zinc in brain and kidney.

Mechanisms in cardiovascular regulation following chronic exposure of male rats to inorganic mercury. Carmignani M, Finelli VN, Boscolo P

Toxicol Appl Pharmacol 1983 Jul;69(3):442-50

In this study we verified the possibility that chronic exposure to inorganic mercury may induce hemodynamic changes in the rat by affecting some neurogenic and/or humoral mechanisms regulating cardiovascular function. For this reason, aortic blood pressure, maximum rate of rise of the left ventricular pressure, heart rate, and electrocardiogram were monitored under pentothal anesthesia in rats which received 50 micrograms/ml of mercury (as HgCL2) in drinking water for 320 days and in control rats. No pressor or electrocardiographic changes were found in mercury-treated animals, which showed increase of cardiac inotropism and decrease of the pressor and inotropic responses to bilateral carotid occlusion. Cardiovascular responses to bilateral vagotomy and iv hexamethonium under vagotomy were unchanged in the mercury-exposed rats. In these animals both pressor and inotropic responses to iv norepinephrine and to higher doses of epinephrine were reduced, while the vascular beta-adrenergic response to 0.125 micrograms/kg of iv epinephrine was potentiated. Cardiovascular responses to acetylcholine, angiotensin I, angiotensin II, bradykinin, histamine, and serotonin did not differ in the two groups of rats. These results indicated that chronic mercury exposure affects cardiovascular function by interfering with the baroreflex mechanisms and/or the reactivity to catecholamines. Higher amounts of mercury were found in kidney, but the metal was significantly accumulated also in urine, blood, and brain. Mercury exposure greatly increased the levels of copper and zinc, but not that of iron, in brain and kidney. The increased accumulation of copper and zinc in tissues may be related in part to the mercury-induced synthesis of metallothionein, a protein able to bind these essential metals. It may be suggested that zinc and copper interact with mercury in inducing cardiovascular changes.

Mercury Affects the Blood

Dr Walter J Clifford MS, RM(AAM), BLD, FIAOMT

Proceedings of the First World Congress on Cancer Sydney 1994

One of the areas that is always of extreme interest is that of **mercury.** There is good reason to have concern. Cells challenged with mercury show the marked and defined changes that even 1 part per 10 million in Locke's solution can have on red blood cells. The changes in the thickness of the membranes, changes in the metabolism rate, shifts in color, aniso and poikilocytosis, the dramatic drop in somatids in the black spaces, is of extreme interest. W find that mercury is one of the most directly toxic of all the materials.

Microbiology of Challenged Blood

When **mercury** has been added to the cells and they have incubated for about 30 minutes, it is not unusual to see both accelerated and unusual changes taking place in the microbiology. We see rods, and we see circular bodies that have developed appendages and are rapidly developing towards pleomorphic mycelial forms of bacteria. These changes are exacerbated by mercury, and can be forced to occur **in** even the most healthy of individuals.

In red blood cells, we see the formation of intracellular bacterial forms. Large circular bodies inside the red cells correspond to the yeast forms described by Naessens. When we watch the progress of these bodies, we see that they will seemingly connect and form a mycelial mat. This is a function that is exacerbated by mercury. It can also be induced by beryllium and tin. It is often found in cancer patients, and we have also seen it in certain arthritic patients.

Normally, it takes several hours for this to happen. But when mercury has been added to the specimen, we can accelerate the development of the microbiology so that these phenomena can be seen in perhaps 20 to 30 minutes. We see the structure enclosing the smaller bodies, and additional development m the yeast form. This is not unusual **m** many of the red blood cells that have not become crenated, or in which the cell membrane has been damaged.]be distension continues to form.

In mercury-challenged cells from an apparently healthy donor, we see the interior microbial content developing **in** less than one hour in virtually every red cell that is visible. Some lymphocytes show a very peculiar lack of texture, and the differentiation of the nucleus diminishes substantially.]here is splitting of the refractile bodies that Naessens characterises as the viral aggregation body. It is apparent that both structure and function have been severely altered in these lymphocytes.

One facet often observed in bloods that have been exposed to mercury, tin, beryllium, aluminium, cadmium and perhaps several others is the accelerated formation of the Naessens thallus stage. This stage is the end of the Naessens macro cycle. The long fibrous casings give rise to literally millions of new 'ds. We notice many red cells with intracellular parasitism by bacterial forms, but the thalluses can become huge and differentiated, and some have lost most of their content. The thallus, which is long and fibrous, will often have a bulbous head at one end. There is often substantial activity and the somatids scatter as breakage occurs.

Remembering that metals can exacerbate this, normally the development of the thallus is seen only in the late stages of cancer, AIDS, and other extremely severe and debilitating diseases.

Even in patients where the cells are not severely affected by mercury and other heavy metals, the microbiology still can be. Is the thought not suggested that those patients who may not have extreme mercury symptoms, but still have debilitated immune systems and still have the inability to fight off disease, may have the effects of mercury because of the microbiology that has been induced.

We expose cells to mercury for less than one hour, and notice the extent to which microbial growth has formed. In cells that have been exposed to nickel at dilute levels for approximately 30 minutes, we can see the effects of bacterial growth inside each of these red cells. Nearly all the cells show the progress of microbiology. We see not only the development of bacterial forms in the cells, but also the so-called yeast forms. We see lymphocytes with bright, refractile bodies, described by Naessens as viral aggregations, and the extensive mat formation-the simplast, in the terminology of Enderlein.

We took blood from another "healthy" donor. No peculiar or unusual development was seen in the microbiology, after observing the untreated blood for 90 minutes. The same blood was mixed with aluminium at very dilute solutions. We noticed bacterial rod structures appended to red blood cells, and some early thecits forming. There was degradation of some of the membranes. It is not unusual in aluminium exposures for this happen within 30 to 45 minutes.

Blood from another healthy donor showed no propensity to develop unusual microbiology within the first 40 minutes in normal organisation. We challenged the blood with cadmium at a very dilute level. We noted the formation of early rods inside some of the red cells, the unusual formation of thecits, and some degradation of the platelets.

In these cadmium exposures, it is not unusual for us to see the formation of the long, slender motile rods. These are frequently seen in cancer patients and in AIDS patients. 'These were generated within 30 to 40 minutes after exposure to cadmium, and again these cells came from a donor where no unusual microbiology was seen within the first 90 minutes under normal observation techniques.

We challenged blood cells with dilute beryllium. Some of the membranes began to deteriorate, with some lipid stripping along a couple of the red cells, and the development of rod-like bacteria in a number of cells. Some of these changes take longer with beryllium, perhaps as long as 45 minutes to an hour. However, after watching the same blood, unchallenged, for up to 2 hours, we did not see any of these phenomena - only when beryllium exposure is induced.

When healthy blood is mixed with a dilute aluminium solution, one observation we frequently see is that many of the PMNs prematurely age and begin to undergo their death phase. The color shifts dramatically and there is formation of thecits. We also see enhancement of the centriole centrosome network in the PMNs.

A peculiar kind of microbial cellular stack is often seen when we mix blood of normal patients with dilute iron salt solution. Iron seems to have a peculiarity in forming these little chains of cells. These cells are especially fragile and very, very heat sensitive when we try to fix and stain them. There is some indication of mycelial formation as well.

One aspect frequently seen when dilute mercury solutions are used is that many of the lymphocytes will suddenly develop an expanded envelope of cytoplasm, with a number of developing microbial forms inside the cytoplasm of the lymphocyte. These protuberances are, again, typical of what we see in malignancy patients. These can be induced within 30 to 40 minutes after mercury exposure.

When we have lymphocytes that have been so expanded and enlarged after mercury exposure, it is also not unusual that we will see PMNs begin to move in and attach themselves, or begin a phagocytic action against many of these infected limbs. This is peculiar to mercury, and we believe we can induce this over longer periods of time with nickel and beryllium. It raises the question that we may be looking at some of the initiation and onset of auto-immune disease problems when this type of action takes place.

Finally, there is a form referred to as a medusae or medusal head. It is a microbial form which has mycelial and fungal-like qualities. It is frequently seen in AIDS patients, and occasionally may be found in cancer patients. We have induced the medusal form by mercury exposure for about one hour in the blood of a patient who has no diagnosed disease and appears completely healthy.

Mercury may reach into a number of disease processes that are far removed from anything that is yet recognised, even by the Academy and other scientific bodies. We recognise that the microbiology must be dealt with, and 1 would also add that these are forms that are not classically recognised by hospital-based microbiologists. If the medusal form can be metal-induced, then can metals be a contributive factor in degenerative disease?

Dr. Waiter Jess Clifford

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Scientists Connect Alzheimer's Disease To Mercury

International Academy of Oral Medicine and Toxicology
www.iaomt.com
SPECIAL NOTE: The University of Calgary has placed a copy of the video on their site at: http://movies.commons.ucalgary.ca/mercury/

Research conducted at the University of Calgary Faculty of Medicine has demonstrated that trace amounts of mercury can cause the type of damage to nerves that is characteristic of the damage found in Alzheimer’s Disease. The level of mercury exposure is consistent with those levels found in humans with

mercury/silver amalgam dental fillings. The exposure to mercury caused the formation of "neurofibrillar tangles," which are one of the two diagnostic markers for Alzheimer's Disease. The scientists found that other metals, including aluminum, did not cause the damage. Previous research has shown that mercury can cause the formation of the other Alzheimer's Disease diagnostic marker, "amyloid plaques."
The research, published in a peer-reviewed medical journal, is accompanied by a video visual presentation of the effect. Utilizing digital time-lapse photography, this video shows rapid damage to the nerve cells after introduction of minute amounts of mercury. Funding for this video was provided by the International Academy of Oral Medicine and Toxicology (IAOMT).
"Retrograde Degeneration of Neurite Membrane Structural Integrity of Nerve Growth Cones Following In Vitro Exposure To mercury."
Leong, CW; Syed, NI; Lorscheider, FL. NeuroReport, 12(4):733-737, 2001.

BIOPROBE COMMENT: This study should remove all doubt regarding the role that dental mercury from amalgam fillings plays in the development of Alzheimer's Disease (AD). Although the American Dental Association would like to have you believe otherwise, science has clearly demonstrated that there is a positive correlation between brain mercury levels and the number and surfaces of "mercury/silver" amalgam dental fillings. The mercury levels that caused the devastating damage to nerve cells in the above referenced study were 100 to 1000 times below those found in the brains of people with "mercury/silver" amalgam dental fillings.
In 1997, researchers at the University of Calgary Medical School and the College of Pharmacy at the University of Kentucky clearly demonstrated that exposing rats to the same levels of mercury vapor that can be released from "mercury/silver" amalgam dental fillings caused the mercury to interact with brain tubulin and disassemble microtubles that maintain neurite structure. The identical neurochemical lesion of similar or greater magnitude is evident in Alzheimer brain homogenates from approximately 80% of patients, when compared to human age-matched neurological controls. (Neurotoxicology 1997;18(2):315-324)
In 2000, researchers at the Neurobiology Laboratory, Psychiatric University Hospital in Basel, Switzerland using neuroblastoma cells exposed to mercury demonstrated an increase in production of amyloid protein that makes up the amyloid plaques as well as significantly increasing the phophorylation of Tau protein. (J Neurochem 2000 Jan;74(1):231- 236)

Studies demonstrating a correlation between amalgam dental fillings and brain mercury levels:

> Lakartidningen 1986 Feb 12;83(7):519-522 Swedish Dental Journal 1987;11(5):179-187, Sci Total Environ 1987 Oct;66:263-268, J Prosthet Dent 1987 Dec:58(6):704-707, FASEB J 1989 Dec;3(14):2651-2646, Sci Total Environ 1990 Dec 1;99(1-2):1-22, Sci Total Environ 1993 Sep 30;138(1-3):101-115, J Trace Elem Med Biol 1995 Jul;9(2):82-87, Zentralbl Hyg U:mweltmed 1996 Feb;198(3):275-291 FASEB J 1998 Aug;12(11):971-980, Biometals 1999 Sep;12(3):227-231

Following is the editorial which accompanied the publication of this study;
Mercury induced growth cone collapse: Owen Hamill, Physiology and Biophysics, UTMB, Galveston, TX, USA

Cases where exposure to heavy metals in the domestic and work environment have contributed to human disease extend back to antiquity with the use of lead in water

pipes and wine storage vessels. It has been proposed that pandemic lead poisoning, resulting in mental incompetence and declining birth rate, especially amongst the ruling class, contributed to the fall of Rome [1] (see [2] for another view). More recent lead poisoning in the general population has arisen from lead-based paints and lead- additives in petrol. A well-documented case of occupational poisoning arose in workers of the 19th century felt hat industry due to the use of mercury as a stiffener of rabbit fur. Increased irritability, mood swings, tremulousness, ataxia and impairment in intellectual capacity characterize Mad Hatter's disease [3]. Currently there is ongoing public health debate on whether low level chronic exposure to mercury due to dental repair work results in subclinical behavioral changes associated with CNS damage (see [4] for review). For example, in the USA the most common material used in dental fillings is a mercury/silver mixture (amalgam) in which an estimated 70 000 kg is used in 100 million fillings/year. Furthermore, evidence indicates that mercury vapor is continuously released from tooth fillings where it is breathed in by the lungs and converted into mercuric ions. Although there is no debate on the toxic effects of high concentrations of mercury (i.e. associated with urinary concentrations .50 ìg/l), a challenge exists to demonstrate more subtle, preclinical effects associated with chronic low level mercury exposure in the general population with fillings. At least consistent with this notion is the study published in this issue [5] showing that exposure to mercury concentrations of ,0.1 ìM results in rapid (i.e. within 10 min) retraction of growth cones in snail neurons and is correlated with disruption of microtubules. Interestingly, the authors point out that similar disruption of microtubules is associated with Alzheimer's disease. These recent findings give added impetus for the development and implementation of alternative materials for fillings and may provide parents with added ammunition in teaching their children to floss.

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Scientific Facts about Mercury And Dental Amalgam

Robert Gammal BDS.
June 1998

What really is that 'Silver' filling?

Dental amalgam is an alloy of silver, tin, zinc and copper, which is combined with an equal amount of elemental mercury. The 'Silver Fillings' in your mouth are about 50% mercury.

As there is a continuous release of mercury from dental amalgam, dentists must dispose of scrap amalgam as toxic waste following strict guidelines. It is illegal to put it in the garbage, the sewer or the drain. In fact it seems that the only legal place to keep this material is in the mouth of a living person! (It is estimated that 11kg mercury per year is released from each crematorium chimney!) [1,2,3]

How Much Mercury is Too Much?

Mercury escapes from amalgam in the forms of mercury vapour, elemental mercury and mercury ions. The rate of release of mercury is increased by an increase in temperature, friction and electrical currents. Elevated levels remain for about 90 minutes after such stimulation.[4]

The dental authorities claim that only a minute amount of mercury is released from amalgam fillings and although this is true, it is worth remembering that mercury is a cumulative poison. It stays in your body and the levels are topped up continuously. This type of poisoning is called micromercurialism. The earliest symptoms are usually sub-clinical and neurological, namely fatigue, headaches, forgetfulness, reduced short term memory, poor concentration, shyness and timidity, confusion, rapid mood swings, unprovoked anger, depression and suicidal tendencies. [5,6,7]

A variety of scientific studies [8,9,10,11,12,13,14] indicates that 20mcg/m3 to 150mcg/m3 of mercury vapour may be found in the mouth of a person with amalgam fillings. 1 mcg of mercury vapour is 5 times greater than the level quoted by the United States Environmental Protection Agency (USEPA) as safe.[15] It is 50 times greater than the level regarded as an acute exposure by the Agency for Toxic Substances and Disease Registry (ATSDR) in the USA.[16] The ATSDR lists mercury as one of the top 20 most hazardous substances known to man!

There is NO know safe level of mercury vapour.

World Health Organization stated in 1991 that for mercury vapour, there is no known "no-observable-effect level (NOEL)".[17] In other words all levels of mercury vapour are harmful. The WHO also demonstrated that dental amalgam is the single greatest source of mercury to the general population - up to 10 times more than from all other sources combined.

Latex paints, which contained mercury, had to be taken off the market after releasing only 2-3mcg/m^3.

The Richardson Report, a study completed for Health Canada in 1995, found that the tolerable daily intake of mercury was exceeded in different age groups with the following number of amalgam fillings: adults - 4, teenagers – 3, children and toddlers – 1.[18]

Retention of mercury in the body is estimated to be 1mcg/filling/day.[11,12] Up to 80% of inhaled mercury vapour is absorbed through the lungs.[19,13] A percentage of mercury vapour adheres to the lining of the nose and mouth and is transported directly into the brain.[6]

Mercury from amalgam easily crosses the blood brain barrier and can damage any part of the central nervous system. [6], Some mercury is also transported along nerve fibres (retrograde axonal transport) back to the brain. [20,21,22,23,24] Mercury from amalgam has been found all the way down the spinal cord. [6] The levels of mercury in the brain are directly proportional to the number of fillings in the mouth.[8,10,12] Minute amounts of mercury in the brain will cause the same type of damage as is found in the brains of patients with Alzheimer's Disease. [25] Low levels of mercury in the brain will severely disturb cellular function and reduce the growth of nerve fibres. [6]

Dentists regularly implant amalgam fillings directly into the bone in the form of retrograde root fillings (a filling placed at the end of the root). Mercury can pass readily from such an implant into the brain. Would any other branch of medicine condone such an absurd practice? One amalgam manufacturer, Caulk, states that amalgam is contraindicated for use as a retrograde filling, yet the Australian dental authorities teach and condone this practice![26]

Mercury from amalgam may be found in all cells of the body (highest concentrations are usually in the kidney, liver and brain).
There will also be a very high concentration of mercury in the jaw bones and the soft tissue lining the mouth.
Blood and urine sampling are poor ways of estimating body burdens of mercury as most of the mercury is retained in the cells of the body (known as Retention Toxicity).
DMPS is a chelating agent, which will remove some mercury from cells and bind it in such a way that it can be excreted. Changes in urine mercury levels can then be measured.[27 ,28]

Mercury from amalgam does not cause a specific disease-

it causes mercury poisoning which, is characterized by a wide range of symptoms. Many organs and functions of the body may be affected.

The following are some basic facts from the published research:

- Mercury from amalgam fillings has been shown to cause a 50% reduction in kidney filtration after just two months in the mouth (animal studies) [29] Kidney damage from mercury has been reported often in the literature.[30 ,31 ,32 ,33 ,34]
- The most common symptoms of long-term low-level mercury poisoning are headaches and psycho-emotional disturbances. Muscle twitches and body shakes are later symptoms and thus more severe.
- Research from 1993 onwards has shown that mercury from amalgam fillings will cause an increase in the number of antibiotic resistant bacteria in the gut and mouth. [35, 36, 37] The number of antibiotic resistant bacteria fall rapidly after the amalgams are removed.
- Mercury from amalgams can cause a weakening in the wall of the small blood vessels (micro-angiopathies) – this results in a reduction of blood supply to the tissues resulting in reduced function and/or cell death.[6]
- Heart function may be affected by mercury and electrical currents from amalgam.[38 , 39]
- Some reports [40 ,14] suggest that elevated cholesterol levels are related to mercury in the body. It has been noted that cholesterol levels drop after removal of amalgam fillings.
- Although the dental associations claim that less than 1% of the population show true allergy to amalgam, the latest research[41] indicates that the real figure is closer to 13%. Assuming that only half the population in Australia has amalgam fillings, this would mean that over 1,700,000 people might be sick due to an allergic reaction to these fillings. Since the medical profession as a whole do not acknowledge the dangers of amalgam, it is most likely that the majority of these people are misdiagnosed and therefore mistreated.
- True allergy is only one type of immune reaction.[42]
- Mercury will always have a detrimental effect on the immune system. This creates an environment in the body for other diseases to develop. [43 ,44 , 45 ,46 ,47 ,48 ,49 ,50]
- Mercury binds to proteins, and thus makes them look like foreign material to the cells of the immune system. [50,51] . Overt auto-immune diseases may then ensue.
- There are literally hundreds of peer reviewed scientific papers discussing the damaging effects that mercury has on the immune system.[50]
- Mercury from amalgam may cause an increase in allergies, skin rashes and itching. [52 ,53]

- Mercury will bind strongly to selenium, a trace element needed for a wide variety of enzyme functions. Latest research indicates a direct relationship between reduced blood selenium levels and an increase in the rate of some types of cancer.[54 ,55 ,56 ,57 ,58 ,59]
- Many studies indicate that selenium supplementation will help to protect from the damaging effects of mercury.[60 ,61 ,62 ,63]
- Mercury binds to haemoglobin in the blood and reduces its capacity to transport oxygen[40] This may be one of the causes of chronic fatigue.
- Mercury at levels as low as 1 part / ten million will destroy the walls of red blood cells.[64 ,65 ,43]
- In May 1998 the British Government recommended that dentists
- not place or remove amalgam in pregnant women.
- Mercury from amalgam fillings will cross the placenta and concentrate in the foetus [66 ,67 ,68 ,69 ,70 ,71 ,72 ,73 ,74 ,75 ,76]
- Mercury from amalgam can also be transported via the breast milk and concentrate in the body of the feeding infant.
- Breast milk increases the bioavailability of mercury to the infant.[77 ,78]
- Prenatal exposure to mercury may cause developmental defects and may cause permanent neurological damage in the unborn child.[69,70]
- Tissue levels of mercury in the foetus, new-born and infant are directly proportional to the number of amalgam fillings in the mother's mouth.[79]
- Mercury is mutagenic - it can cause single strand breaks in DNA..[80 ,81 ,82 ,83 ,84]
- Female dental personnel exposed to mercury, exhibit twice the rate of miscarriage, infertility and still births as compared to the rest of the population. [58,59,12,49]

If you are pregnant, never allow amalgam fillings to be placed in your mouth.

Do not go into a dental surgery where amalgam is used, as the mercury vapour levels in the air may be harmful to the foetus.[16]

- Electric currents, generated by the interaction of different metals in the mouth, can be measured in micro-amps. The central nervous system operates in the range of nano-amps. This is about 1,000 times less than the currents generated in the mouth. This is in the same order of magnitude as that induced in a person standing under high-tension power cables.[85 ,86 ,87]
- Electrical currents, formed by placing gold into a mouth with amalgam fillings, will create an increase in electrical currents in the fillings, resulting in an increase in mercury released from all of the fillings.
- Placing a gold crown over an amalgam filling may cause a four-fold increase in the amount of mercury being driven through the tooth.[62, 44] Gold crowns, on top of amalgam, create a permanent galvanic cell. Amalgam is still the most commonly used material to build a core for a crown. This practice is contra-indicated by the manufacturers Caulk and Ivoclar.
- Dental fillings are an implant of materials into living tissues. Neither the United States Food and Drug Administration nor the Australian Therapeutic Goods Administration have approved mixed dental amalgam as an implant material.
- Although the dental authorities make claims about amalgam safety, they have not presented one scientific paper which indicates that this material is toxicologically safe.
- In dental surgeries where amalgam is used, the mercury vapour levels may be so high as to be hazardous to health. Dental associations have said that if mercury from amalgam is so dangerous for the patient, then why is it that the

dentists, who are exposed to far greater levels of mercury, are not sick? This claim is not substantiated by the scientific literature.

In fact, dental personnel show a range of medical effects different from the rest of the population.

- Twice the rate of glioblastomas than the rest of the population. [90]
- Reduced IQ levels have been demonstrated [91 ,92 ,93]
- Psycho-motor and psycho-emotional studies of dentists, demonstrate a severe drop in scores compared to the rest of the population. [94]
- Twice the rate of suicide of any professional group.
- 20% of Canadian dentists are on permanent disability for psychological reasons [9]

Detoxification and Amalgam Removal

Clinical experience has demonstrated that people affected by mercury from dental amalgams will often enhance the benefits of amalgam removal if removal is combined with a detoxification routine prior to, during and after the amalgam removal.

Removal of amalgam fillings has been shown to substantially lower the body burden of mercury.[88 ,89] Protocols do exist for the safer removal of dental amalgam from your mouth. Failure to follow these guidelines may result in exposure to an unacceptable level of mercury. Removing old amalgam fillings must be performed with extreme care.

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Written for ASOMAT by Dr Robert Gammal BDS

The Australasian Society of Oral Medicine and Toxicology was formed by a group of dedicated dentists and doctors with the aim of educating the public and the profession about the concepts of bio-compatible dentistry.
ASOMAT is a non-profit organization and can be contacted at
PO Box A860 Sydney South NSW
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Scientific Facts on the Biological Effects of Fluorides

"Fluoridation is the greatest case of scientific fraud of this century"

1.Fluoride exposure disrupts the synthesis of collagen and leads to the breakdown of collagen in bone, tendon, muscle, skin, cartilage, lungs, kidney and trachea.

A.K. Susheela and Mohan Jha, "Effects of Fluoride on Cortical and Cancellous Bone Composition", IRCS Medical Sciences: Library Compendium, Vol 9, No.11, pp.1021-1022

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2. Fluoride stimulates granule formation and oxygen consumption in white blood cells, but inhibits these processes when the white blood cell is challenged by a foreign agent in the blood.

Robert A. Clark, "Neutrophil Iodintion Reaction Induced by Fluoride: Implications for

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3. Fluoride depletes the energy reserves and the ability of white blood cells to properly destroy foreign agents by the process of phagocytosis. As little as 0.2 ppm fluoride stimulates superoxide production in resting white blood cells, virtually abolishing phagocytosis. Even micro-molar amounts of fluoride, below 1ppm, may seriously depress the ability of white blood cells to destroy pathogenic agents.

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S.K. Jain and A.K. Susheela, "Effect of Sodium Fluoride on Antibody Formation in Rabbits", Environmental Research, Vol.44, pp.117-125 (1987).

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Viktor Gorlitzer Von Mundy, "Influence of Fluorine and Iodine on the Metabolism, Particularly on the Thyroid Gland," Muenchener Medicische Wochenschrift, Vol 105, pp182-186 (1963);

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T.Takamorim "The Heart Changes in Growing Albino Rats Fed on Varied Contents oif Fluorine," The Toxicology of Fluorine, Symposium, Bern, Switzerland, Oct 1962, pp.125-129; Vilber A.O. Bello and Hillel J. Gitelman, "High Fluoride Exposure in

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J.K. Mauer, et al., "Two-year cacinogenicity study of sodium fluoride in rats", Journal of the National Cancer Institute, Vol 82, pp1118-1126 (1990);

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P.D. Cohn, " A Brief Report on the Association of Drinking Water Fluoridation and Incidence of Osteosarcoma in Young Males", New Jersey Department of Health, Trenton, New Jersey, Nov 1992; M.C. Mahoney et al.,"Bone Cancer Incidence Rates in New York", American Journal of Public Health, Vol 81, pp.81, 475 (1991);

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Nicholas Leone, et al., "Medical Aspects of Excessive Fluoride in a Water Supply", Public Health Reports, Vol 69, pp.925-936 (1954);

J. David Erikson, "Mortality of Selected Cities with Fluoridated and Non-Fluoridated Water Supplies", New England Journal of Medicine, Vol. 298, pp.1112-1116 (1978);

"The Village Where People are Old

Before their Time", Stern Magazine, Vol 30, pp.107-108,111-112 (1978);

10. Fluoride ingestion from mouthrinses and dentifrices in children is extremely hazardous to biological development, life span and general health.

Yngve Ericsson and Britta Forsman, "Fluoride retained from mouthrinses and dentifrices in preschool children", Caries Research, Vol.3, pp.290-299 (1969);

W.L. Augenstein, et al., "Fluoride ingestion in children: a review of 87 cases", Pediatrics, Vol 88, pp.907-912, (1991);

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George Waldbott, "Mass Intoxication from Over-Fluoridation in Drinking Water", Clinical Toxicology, Vol 18, No.5, pp.531-541 (1981)

11. Fluorides diminish the intelligence capability of the human brain.

X.S.Li et al, Fluoride, Vol 26, No.4, pp.189-192, 1995, "Effect of Fluoride Exposure on Intelligence In Children". Presented to the 20th Conference of the International Society for Fluoride Research, Beijing, China, September 5-9, 1994.

12. Fluoride studies in rats can be indicative of a potential for motor disruption, intelligence deficits and learningdisabilities in humans. Humans are exposed to plasma levels of fluoride as high as those in rat studies. Fluoride involves interruption of normal brain development. Fluoride affects the hipppcampus in the brain, which integrates inputs from the environment, memory, and motivational stimuli, to produce behavioral decisions and modify memory. Experience with other developmental neurotoxicants prompts expectations that changes in behavioral functions will be comparable across species, especially humans and rats.

Neurotoxicology and Teratology, Vol 17, No,2, p.176, "Neurotoxicity of Sodium Fluoride in Rats", Muellenix, Denbesten, Schunior, Kernan, 1995.

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Neurotoxicology and Teratology, Vol 17, No,2, p.176, "Neurotoxicity of Sodium Fluoride in Rats", Muellenix, Denbesten, Schunior, Kernan, 1995.

14. "Fluorides are general protoplasma poisons, with the capacity to modify the metabolism of cells by inhibting certain enzymes. Sources of fluoride intoxication include drinking water containing 1ppm or more of fluorine."

Journal of the American Medical Association, September 18, 1943.

15. "Drinking water containing as little as 1.2 ppm fluoride will cause developmental disturbances. We cannot run the risk of producing such serious systemic disturbances. The potentialities for harm outweigh those for good."

Journal of the American Dental Association, Editorial, October 1, 1944.

Other Facts

- The contents of a family-size tube of fluoridated toothpaste is enough to kill a 25-pound child.
- In 1991, the Akron (Ohio) Regional Poison Center reported that "death has been reported following ingestion of 16mg/kg of fluoride. Only 1/10 of an ounce of fluoride could kill a 100 pound adult. According to the Center, "fluoride toothpaste contains up to 1mg/gram of fluoride." Even Proctor and Gamble, the makers of Crest, acknowledge that a family-sized tube "theoretically contains enough fluoride to kill a small child."

- Fluorides have been used to modify behavior and mood of human beings.
- It is a little known fact that fluoride compounds were added to the drinking water of prisoners to keep them docile and inhibit questioning of authority, both in Nazi prison camps in World War II and in the Soviet gulags in Siberia.
- Fluorides are medically categorized as protoplasmic poisons, which is why they are used to kill rodents.
- 1943 The Journal of the American Medical Association on September 18, 1943, states, "fluorides are general protoplasmic poisons, changing the permeability of the cell membrane by inhibiting certain enzymes. The exact mechanismof such actions are obscure.".
- Fluoride consumption by human beings increases the general cancer death rate.
- 1975 Dr. John Yiamouyiannis publishes a preliminary survey which shows that people in fluoridated areas have a higher cancer death rate than those in non-fluoridated areas. The National Cancer Institute attempts to refute the studies. Later in 1975, Yiamouyiannis joins with Dr. Dean Burk, chief chemist of the National Cancer Institute (1939-1974) in performing other studies which are then included in the Congressional Record by Congressman Delaney, who was the original author of the Delaney Amendment, which prohibited the addition of cancer-causing substances to food used for human consumption.
- Both reports confirmed the existence of a link between fluoridation and cancer. (Note: Obviously Dr. Burk felt free to agree with scientific truth only after his tenure at NCI ended, since his job depended on towing the party line).

Fluorides have little or no effect on decay prevention in humans:

In 1990 Dr. John Colquhoun is forced into early requirement in New Zealand after he conducts a study on 60,000 school children and finds no difference in tooth decay between fluoridated and unfluoridated areas. He additionally finds that a substantial number of children in fluoridated areas suffered from dental fluorosis. He makes the study public. There is no scientific data that shows that fluoride mouth rinses and tablets are safe for human use.

1989 A study by Hildebolt, et al. on 6,000 school children contradicts any alleged benefit from the use of sodium fluorides.

In 1990 a study by Dr.John Yiamouyiannis on 39,000 school children contradicts any alleged benefits from the use of sodium fluorides.

In 1992 Michael Perrone, a legislative assistant in New Jersey, contacts the FDA requesting all information regarding the safety and effectiveness of fluoride tablets and drops. After 6 months of stalling, the FDA admitted they had no data to show that fluoride tablets or drops were either safe or effective. They informed Perrone that they will "probably have to pull the tablets and drops off the market."

The fact that fluoride toothpastes and school-based mouth rinses are packaged in aluminum accentuates the effect on the body.

In 1976, Dr. D.W.Allman and co-workers from Indiana University School of Medicine feed animals 1 part-per-million (ppm) fluoride and found that in the presence of aluminum in a concentration as small as 20 parts per billion, (like in a toothpaste tube, using aluminum pans to boil water, or drinking beverages in

aluminum cans, fluoride is able to cause an even larger increase in cyclic AMP levels.

Cyclic AMP inhibits the migration rate of white blood cells, as well as the ability of the white blood cell to destroy pathogenic organisms. Ref: Journal of Dental Research, Vol 55, Sup B, p523, 1976, "Effect of Inorganic Fluoride Salts on Urine and Tissue Cyclic AMP Concentration in Vivo". (Note: It is no small accident that toothpaste tubes containing fluoride are often made of aluminum)

"Fluoridation is the greatest case of scientific fraud of this century"

> Robert Carlton, Ph.D.,former U.S. EPA scientist on "Marketplace" Canadian Broadcast Company Nov 24, 1992

"Regarding fluorodation, the EPA should act immediately to protect the public, not just on the cancer data, but on the evidence of bone fractures, arthritis, mutagenicity and other effects"

> William Marcus, Ph.D., senior EPA toxicologist, Covert Action, Fall 1992, p.66

Fluoride Poisoning

From The Townsend Newsletter For Doctors

May 2000

The following column is Chapter Fourteen of the recently published book from Hampton Roads Publishing Co., Inc. authored by Dr. Morton Walker, *Elements of Danger: Protect Yourself Against the Hazards of Modern Dentistry.* Notice that many of the references cited are taken from issues of the *Townsend Letter for Doctors & Patients.*

Fluoride - an industrial waste product commonly sold as poison for killing rats and insects - is not good for the human body either. Yet, most dentists spouting the American Dental Association's propaganda insist that there must be fluoridation of our drinking water. These dentists, as proponents of adding fluoride to public water supplies or administering dental fluoride treatments to patients or making fluoride an ingredient of toothpaste, are perpetrating an evil against all of us. Most especially they're harming our children.

FDA Warning:

Fluoride Toothpaste Poisons Little Children

The Food and Drug Administration has sent down a ruling. It requires that on all fluoride toothpastes manufactured after April 1997, a printed admonition must appear on the fluoridated toothpaste package reading: *"Warning: Keep out of the reach of children under six years of age. If you accidentally swallow more than used*

for brushing, seek professional assistance or contact a Poison Control center immediately."

Following that FDA ruling, the manufacturers of fluoride toothpastes either disregarded it altogether, ignored the ruling's voluntary guidelines or interpreted them overly broadly; consequently, the FDA has put teeth in its current rules of enforcement. Inasmuch as pharmacies, groceries, convenience stores, health food stores, supermarkets, and other stores have finally sold out old and overstocked inventories of fluoridated toothpastes, you can now read the warning on various, newly distributed toothpaste tubes, boxes, bottles, and cans. For example, Procter & Gamble has included the message inside packages of Crest Multicare(TM) advising customers to stop using the brand if they experience any form of body irritation. The Colgate-Palmolive Corporation prints the FDA warning on its newest toothpaste, Colgate Total'. Church & Dwight, the manufacturer of Arm & Hammer toothpaste, provides a new toll free telephone number for any reports of fluoride poisoning. Unilever PLC, the maker of Mentadent(TM) has put fluoride toxicity information on its Web site.

Some toothpaste brands, like Oral B bubble-gum flavor and the Tom's of Maine "Silly Strawberry" formula are so delicious that little children, even as they brush, eat their fluoridated toothpaste like candy. There is danger here, because the kids can die or at the very least become very sick from ingesting the fluoride poison. Certainly they may come down with dental fluorosis.

Dental Fluorosis is a condition characterized by mottled tooth enamel, which is opaque and may be stained. Its incidence increases when the level of fluoride in the water supply is above two parts per million. When the drinking water's level of fluoride reaches over eight parts per million, systemic fluorosis may occur, with calcification of ligaments. In general, fluorosis is a bony overgrowth accompanied by neurologic complications and arthritis brought on by long-term fluoride intake, such as occurs in industrial workers.

If asked, manufacturers do admit that no child could get through a six-ounce tube of fluoridated toothpaste without vomiting. Fluoride causes vomiting because large doses of it combined with gastric juices tend to irritate the child's - and even an adult's - stomach and intestines. Of the 4,453 cases of unintended "fluoride exposure" reported to poison-control centers in 1997, 99% turned out not to be life-threatening but definitely had the potential to bring on death. In the remaining 1%, severe illness, near-death, or actual death did occur.'

Harmful Effects Caused by Fluoride Ingestion

In 1974, a three-year-old Brooklyn boy had stannous fluoride gel swabbed over his teeth by a pedodontist (specialist in children's dentistry) as the means of preventing tooth decay. Five hours later this child died from fluoride poisoning because of one fatal mistake. After rinsing his mouth the small boy did not spit out the rinse water but swallowed it instead.'

In New York City five years before, another boy, age four, went into violent convulsions and died directly after receiving topical fluoride applications to his teeth. Personnel of the dental clinic claimed this child had sustained a heart attack, even though there was no history of cardiac disease for him or any members of his family. In fact, the ingestion of even small amounts of fluoride is known among cardiologists to be a possible cause of cardiac arrest. It's one of water fluoridation's "side effects. 113

On May 23, 1998, in Hooper Bay, Alaska, 41 year-old Dominic Smith drank fluoridated well water along with thirty other residents of this Bering Sea coastal village. All of them got sick, but Mr. Smith died. It seems that a broken pump had [1] injected a little more than the usual fluoride quantity into Hooper Bay's water supply. As a result, Dr. Peter Kakamura, director of the A] askan State Division of Public Health, reports: "The man's death occurred by reason of fluoridation." He was poisoned. There was near-death of Dominic Smith's sister too, plus flu-like symptoms struck 29 others who drank from one of the two public wells. Fluoride is routinely added to Alaska's public water supplies, including those in Anchorage and many Eskimo villages "to reduce tooth decay. -4

From Auckland, New Zealand, nutritionist Toni Jeffreys, PhD, asks, "I wonder if the epidemic of osteoporosis and escalating heart disease in women is not due to the current conventional medical advice to take calcium and fluoride tablets? Most of us know that calcium is antagonistic to magnesium, and that it is magnesium that protects our hearts. But also, magnesium is the mineral that provides elasticity in bones. Without magnesium we can build lots of bone but it's a poor grade bone which shatters and fractures at any strain.'

"Unfortunately fluoride is also antagonistic to magnesium and will cancel it out," continues Dr. Jeffreys. "It is therefore quite murderous to give women calcium and fluoride tablets, when we are already overburdened with fluoride in the environment in numerous ways and are deficient in magnesilim."[6]

As shown by a scientific study conducted by four prestigious research institutions, Harvard Medical School, Eastman Dental Center, Iowa State University, and Forsyth Research Institute, fluoride has an adverse effect on the brain and central nervous system (CNS). It causes "motor dysfunction, IQ deficits, and/or learning disabilities in humans," say the institutions' cooperating researchers .7

At approximately one part per million (1 ppm), fluoride has been added to most public water supplies throughout the United States for over four decades at the urging of dentists, but these four research groups report that the CNS's functional output is vulnerable to fluoride. This scholarly laboratory study indicates that fluoride ingestion's "neurotoxic risks deserve further evaluation.""

Taking in fluoride definitely has deleterious consequences for the brain. Pathological conditions of the brain have been studied by Russians, Chinese, the United States Public Health Service (USPHS), and others since 1978. For instance, in their 1978 book *Fluoridation, the Great Dilemma,* three medical authors describe the findings of practicing Soviet physicians. The Russian physicians observed that 79% of patients with occupational fluorosis show a series of chalky-white, irregularly distributed patches on the surface of the enamel which become infiltrated by yellow or brown staining or other discolorations on teeth from fluoride ingestion. With these patches, say the Russians, the patients "demonstrate dysfunction of subcortical axial nonspecific structures of the brain."9

Moreover, the 1991 review, *Fluoride Benefits and Risks,* published by the USPHS, states that there is "relative impermability of the blood-brain barrier to fluoride." This mineral does penetrate the brain's first line of defense against toxins and potentially may be responsible for various brain syndromes such as senile dementia, schizophrenia, and Alzheimer's disease.10

Recent studies from China on the relationship between drinking fluoridated water by residents in endemic Chinese dental fluorosis areas and the population's intelligence quotient, contain significant references and discussions. They indicate that diminishing IQ for people living in dental fluorosis areas has been known since

1989. Chinese studies indicate that the influence of a high fluoride environment on the intelligence of children may occur early in development such as during the stages of embryonic life or infancy when differentiation and growth are more rapid. Ultramicroscopi study of embryonic brain tissue obtained from termination of pregnancy operations in endemic fluorosis regions showed "differentiation of brain nerve cells were poor, and brain development was delayed. -11,12

The incidence of thigh bone fractures at the femoral neck in those people 65 years of age and older was compared in three communities in the State of Utah. Among the Utah towns, one of them had its water artificially fluoridated to one part per million. The other two did not. Measured over a seven year period, the relative risk of hip fracture for women drinking fluoridated water increased by 1.27, and for men the risk rose to 1.41. As a conclusion to their study, the four medical researchers state, "We found a significant increase in the risk of hip fracture in both men and women exposed to artificial fluoridation at one ppm, suggesting that low levels of fluoride increase the risk of hip fracture in the elderly"13

Commenting on this finding, Seattle, Washington medical nutritional therapist Alan R. Gaby, MD, made an observation similar to that of Dr. Toni Jeffreys (above). Dr. Gaby said: "Hip fracture is the second most common cause of admission to nursing homes, accounting for approximately 60,000 admissions each year. Fluoride apparently causes new bone formation of inferior quality, especially in the femoral head, where there is more cortical bone. Some studies suggest that fluoride is also a carcinogen."14

Fluoride as a Carcinogen

In 1977, epidemiological studies on fluoridation carried out by Dean Burk, PhD, former head of the Cytochemistry Section of the National Cancer Institute, in conjunction with John Yiamouyiannis, PhD, President of the Safe Water Foundation of Delaware, Ohio, were the subject of full-scale United States Congressional Hearings. The Burk/Yiamouyiannis studies showed that fluoridation is linked to about 10,000 cancer deaths annually in this nation. The US Public Health Service, co-promoter of fluoridation with the American Dental Association (ADA), opposed the Burk/Yiamouyiannis investigations. The USPHS tried to refute findings of the two scientists with its own report. But Drs. Yiamouyiannis and Burk evaluated the USPHS findings. Then they explained to the Congress how "conflicting findings of the USPHS are due to the fact that the Service had made mathematical errors by leaving out 80-90% of the recorded data. When these errors and omissions are corrected, its method of simultaneously adjusting for age, race, and sex confirm that 10,000 excess cancer deaths per year are linked to water fluoridation in the United States."

To their amazement, officials of the USPHS discovered that their own findings really did coincide with findings from Drs. Burk and Yiamouyiannis. All of the findings from both studies, going back a quarter century, clearly point to fluoride as a cancer culprit. When added to drinking water fluoride creates a carcinogen. More than this, the following are results released in 1990 by the National Toxicology Program (NTP) under the auspices of the USPHS:15

- Precancerous. changes occur in human oral squamous cells as a result of elevating the levels of fluoride in drinking water.
- There is an increase in the incidence of tumors and cancers in oral squamous cells as a result of increasing levels of fluoride in the drinking water.

Osteosarcoma, a rare form of bone cancer, occurs only in animals with fluoride in their drinking water.

There is an increase in the incidence of thyroid follicular cell tumors as a result of increasing levels of fluoride in the drinking water.

Hepatocholangiocarcinoma a rare form of liver cancer, occurs in animals with fluoride in their drinking water.

The doses of fluoride that are linked to cancer in this NTP study are only one tenth to one fiftieth of the amount used to produce can by benzene. Thus, fluoride is up to fifty times more carcinogenic than benzene.

The cancer-causing potential of fluoride is not limited to one type of cancer.

Similar to cancer-coverups by cigarette manufacturers in the tobacco industry, Proctor & Gamble, manufacturer of the many Crest' fluoridated toothpaste brands, which are endorsed (for a fee) by the American Dental Association, performed carcinogenicity studies with sodium fluoride four years before the above-reported 1990 NTP/USPHS report was released. Dose-dependent increases in cancer were observed in every parameter tested, including squamous cell metaplasias, but the USPHS held back this information and only released it when forced to do so by Dr. John Yiamouyiannis under the Freedom of Information Act."

Added to all that, the Department of Health and Human Services (HHS) gathered a massive amount of evidence for its own 1991 report, "Review of Fluoride Benefits and Risks." This additional report supports the link between drinking and bathing in fluoridated water and the creation of human cancers. Here is what was learned by the HHS:17

- Based on rates of 279 cancer cases expected in nonfluoridated areas, 290 people contracted bone and joint cancers when they lived in areas whose water is fluoridated.
- Although 30.33 cases were expected, there was an excess of 49 people suffering from Ewing's sarcoma in fluoridated counties.
- The observed-to-expected rate of soft tissue cancer for both sexes in Seattle, Washington increased with the duration of fluoridation of the city's water supply.
- For kidney cancer, the risk ratios for both sexes in Seattle with its fluoridated drinking water rose by 10%, a trend that the HHS considered statistically significant.

In December 1992, the New Jersey Department of Environmental Protection and Energy and the New Jersey Department of Health released their joint study of November 8, 1992. New Jersey State findings were that bone cancer rates among ten- to nineteen-year-old males living in all New Jersey municipalities having fluoridated drinking water is 6.9 times higher than in other areas of the state. There is no doubt that New Jersey residents drinking from fluoridated public water supplies suffer from a much higher incidence of bone cancer.',, Because it continues to fluoridate its public drinking water, the State of New Jersey is a candidate for a class action suit by its residents and by visitors to the state.

Additional Hazardous Results from Fluoride Ingestion In the United States Pharmacopoeia, the poisonous hazards of fluoride ingestion are listed. They include the less lethal side effects of nausea, vomiting, stomach cramps, tremors, faintness, weakness, unusual psychological excitement, skin rash, sores in the mouth and on the lips, pain and aching of bones, and white, brown, or near-black discolorations of teeth identified as dental fluorosis.

Dental or occupational fluorosis is actually a visible sign that fluoride content of the body has caused the enamelforming cells, the ameloblasts, to produce damaged collagen. Collagen makes up 30% of the body's protein. It provides the structural framework for skin, ligaments, tendons, muscles, cartilage, bones, and teeth.

In his well-documented and detailed book, *Fluoride, The Aging Factor,* Dr. John Yiamouyiannis explains that fluoride ingestion causes "increased production of imperfect collagen or collagen-like protein," not just in the teeth but throughout the body. The body's structural components that should not become mineralized such as ligaments, cartilage, and tendons turn into hardened tissues. The skin which produces the disease scleroderma, as well as arteries, not only harden but also calcify.

Fluoride ingestion also affects the structure and strength of bone by causing fused vertebrae, calcified joints, arthritis, and an increase in fractures. It decreases the bone's healing ability. Several studies that evaluate fluoride as a treatment for osteoporosis found that this mineral increases, rather than prevents, skeletal fragility. Only one ppm fluoride in drinking water, disrupts collagen metabolism. Yet, the US Environmental Protection Agency allows 4 ppm fluoride in our nation's water supply."

Not only does drinking fluoridated water disrupt collagen, it affects other proteins as well, causing widespread dysfunction of enzymes and the immune system, and even chromosomal damage. Fluoride ingestion breaks up existing protein bonds and forms an extremely strong bond to the one particular protein bond known as "H2," disrupting the normal shape and function of other necessary proteins. When the proteins that form enzymes are disrupted in this way, the enzymes themselves become inactivated. Enzymes are the catalysts that cause the biochemical changes in the body.

Not only are these enzymatic proteins inactivated, they are rendered unrecognizable to the body's immune system, setting up an autoimmune allergic reaction. Because of this peculiarity, fluoride causes a vast variety of ill effects. "The United States National Academy of Sciences (USNAS)20 and the World Health Organization (WHO) 21 as well as other institutions, have published lists of enzymes that are inhibited at fluoride levels of one ppm or less," writes Dr. Yiamouyiannis. Among them, acetylcholinesterase, glutamine synthetase, ATPase, and the DNA Repair Enzyme System are just a few of the known enzymes inhibited by fluoride ingestion at one ppm.

One part per million fluoride in drinking water or other ingested solvents (such as diet cola drinks which contain fluoride) cuts the activity of the DNA repair enzyme by 50%, resulting in increased genetic damage. In his 1993 book, Dr. Yiamouyiannis lists nineteen studies since 1973, that show evidence of fluoride-induced genetic damage in mammals, one of which was done by Proctor and Gamble (already cited). Scientific studies prove that fluoride levels found in the autopsied brains of persons drinking fluoridated water average 1.5 ppm fluoride; in their autopsied hearts 1.8 ppm was found; and in their thyroid glands there was 4.0 ppm. Fluoride is used, according to the *Merck Index,* to suppress thyroid activity. Clearly, a fluoridated substance taken into the body affects more than teeth .22

With All this Damage, Why Are We Fluoridating Ourselves?

Despite so much documentation of fluoride's ill-effects on the body and the very real danger all of us face from its ingestion, we continue to fluoridate ourselves by means of adding it to drinking water, toothpastes, oral rinses, baked goods, prescribed dental treatments, food supplements, soft drinks, beer, wine, fruit juices

made from concentrates, and in additional ways. In one analysis, Coke Classic Tm, bottled in Chicago, is shown to contain 2.56 ppm fluoride. And, as was alluded to, the Chicago-made Diet Coke@ contains 2.96 ppm. Produce grown with fluorine-containing fertilizers offer up from six to 12 times more fluoride than those fruits an vegetables not "fed" with these fertilizers. Not only does fluoride pollute our food and water, but certain manufacturing plants such as aluminum, phosphate, steel, clay, glass, enamel, and many other factory types release high levels of fluoride into the air, soil, rivers, and lakes.23

You have every right to ask, why are we continuing to fluoridate ourselves into sickness and death? We do it as a result of media bombardment: emphatic promotions by the United States Public Health Service, propaganda from the American Dental Association, advertising by fluoride toothpaste manufacturers, and other more subtle capitalistic reasons having nothing to do with dental health. So, allow me to answer your question as to why we are fluoridating ourselves. First, however, you should know the political background of fluoridation 'that's forced on our populace in the United States.

American fluoridation proponents are ever-continuing to assert that fluoride is a mineral essential to the body and responsible for preventing tooth decay. But a 1971 review of numerous studies concerning the nutritional value of fluoride, performed by the US National Academy of Sciences, found no evidence to support the claim that fluoride is an essential mineral. Further, both the US Center for Disease Control and Prevention in Atlanta, Georgia and the British Ministry of Health admit that no laboratory or epidemiological study supports the claim that adding fluoride to the drinking water prevents tooth decays

The reason Americans are fluoridating their water supplies is strictly related to big business greed and the buying off of bureaucrats. Yes, it has everything to do with making money for certain vested interests and nothing to do with our dental health.

Money-Eyed Interests Fluoridate Us

As the aluminum and phosphate fertilizer industries grew in the 1920s and 1930s, manufacturers were faced with how to get rid of their poisonous byproduct, the fluoride waste. They could sell only so much rodenticide and insecticide and desperately needed another means of getting rid of fluoride without incurring public censor. The manufacturers hit on an idea of using it for fluoridating reservoirs, lakes, ponds, rivers, streams, aquifiers and other sources of drinking water. They lobbied the US Public Health Service to introduce this fluoridation measure into the public trough. H. Trendley Dean, MD, then a bureaucrat with the USPHS, performed surveys of areas in the 1930s that relied for drinking water on fluoridepolluted streams and rivers. In a 1937 report that Dr. Dean published, he found that the higher the content of fluoride in the water, the greater the incidence of mottled teeth (dental fluorosis). He wrote an excellent paper which clearly made the case against fluoridation.`

Then Dr. Gerald J. Cox, an official of the Mellon Institute pronounced that, while too much fluoride can cause mottling of teeth, low levels of one ppm actually was nutritionally beneficial and prevented tooth decay. Dr. Cox's boss, the Mellon family, owned the Aluminum Company of America (ALCOA), a major fluoride polluter. The Mellons were seeking a way to unload their massive amount of fluoride byproduct and maybe even make money from its sale.

Persuaded by a high-paying position provided by the Mellons as the first director of the National Institute of Dental Research, in 1938 Dr. H. Trendley Dean changed his findings from seven years before. He reversed himself by publishing skewed data to

support Dr. Cox's cavity-prevention Pronouncement Then, six years later, Oscar Ewing, an ALCOA attorney, was appointed the United States' Federal Security Administrator in 1944 and took charge of the USPHS. Mr. Ewing appointed Edward L. Bernays as the "father of public relations" for popularizing ALCOA's water fluoridation campaign. Thus, Bernays turned rat poison into "a beneficial provider of gleaming smiles." He did it with clinical reports praising the use of fluoride for cavity prevention published in various medical and dental journals.

Mr. Bernays knew that by offering appropriate "under-the-counter" compensation, he could get officials of the American Medical Association, the American Dental association, Proctor and Gamble, independent scientists and laboratories, physicians, dentists, and certain government bureaucrats to proclaim fluoride's safety and advantages for teeth. This public relations gambit Mr. Bernays achieved long before any clinical studies were completed. By accomplishing such positivesounding publication and declarations, the government officials, physicians, dentists, business executives, scientists, laboratory directors, and others would be too embarrassed and frightened of lawsuits to renege on their well-paid endorsements - regardless of the results of scientific studies.

Those whose scientific research refuted fluoride and showed its toxicity were ignored, labeled crackpots, fired from jobs, denied grants, and stripped of dental and medical licenses. In his very fine book, Dr. Yiamouyiannis contends that the fluoridation campaign still continues under the leadership of New York City psychiatrist, Stephen Barrett, MD, who is cofounder of the National Council Against Health Fraud (NCAHF), which recently changed its name to the National Council for Reliable Health Information (NCRHI).26 (To learn how Dr. Barrett and his fellow members of the NCAHF or NCRHI have situated themselves among the candidates for a class action suit, see the end of this article.)

Does Fluoride Prevent Dental Cavities?

Fluoride, a natural trace mineral in the diet, is found in drinking water naturally in widely varying concentrations from trace amounts to a dozen parts per million. Much like selenium, manganese and other trace minerals, there is an ideal level of intake. When fluoride's intake is too low, dental caries could possibly occur. When too high, dental fluorosis will occur.

Cardiologist/dentologist Thomas Levy, MD, of Colorado Springs, Colorado, who had worked with biocompatible dentist Dr. Hal Huggins, tells us about the mottling of teeth by the pathological onset of fluorosis. "Known in the United States

since at least 1916, dental fluorosis is sometimes referred to as "Colorado Brown Stain" and "Texas Teeth" as these two states have a high endemic fluoride level in much of their drinking water," states Dr. Levy. "In its advanced stages, affected teeth demonstrate pitting and brittleness. Often chipping, and a yellow, brown, or black appearance [shows up] in different areas [of the teeth]. Earlier stages show a chalky, mottled, inconsistent appearance .1127

The elegant study in 1937 by Dr. H. Trendley Dean of the USPHS that I described earlier clearly points out that the incidence of dental fluorosis is directly related to fluoride concentration in drinking water, reaching virtually 100% when the level exceeds 4.5 ppm. Levels of only 2.2 ppm show a roughly 70% incidence of this affliction. 28 Dr. Dean's initial dental fluorosis finding, well after his refutation of it for money, was later supported substantially by another 1984 study published in the *Journal of the American Dental Association.29*

Worse, USPHS medical scientists uncovered in 1954 that Bartlett, Texas, with 8 ppm fluoride in its water supply, presented a mortality rate among its residents three times greater than that of a neighboring town with a fluoride drinking water level of merely 0.4 ppm.30 Added to this Bartlett township discovery was another study published in a 1978 issue of the *New England Journal of Medicine* that stated death occurs more frequently among the populace of cities and towns drinking fluoridated water as compared to those folks drinking unfluoridated water.31

Fluoride Weakens Bones and Teeth

Fluoride is not essential for sound teeth and it does not prevent cavities. However, as a result of a 1940s study commissioned by the USPHS that determined one ppm of fluoride in water reduced tooth decay by 60%, many countries acted on this erroneous finding. Today, two-thirds of the population of Australia, half the population of the United States, Canada, Ireland and New Zealand, 30% of the population of Brazil, and 10% of the population of Great Britain, mostly in the west Midlands and Northern regions of England, drink water from municipal supplies that have been artificially fluoridated. Other, more enlightened European nations such as Sweden, Holland and Germany have reversed their policy and discontinued the practice."

While fluoride ingestion tends to stimulate bone density, the fluoride-stimulated bone is structurally unsound. Restoration of bone mass by use of this mineral not only fails to reduce the risk of fractures in women who suffer with postmenopausal osteoporosis, but it actually increases the risk of such fractures .33 And more disturbing is accumulating evidence that the fluoridation of public water supplies eventually increases the risk of fractures in the whole community.34

Scientific studies are currently reporting higher prevalence of dental fluorosis following the fluoridation of drinking water than had been predicted .35 Melvyn R. Werbach, MD, of Tarzana, California, an internationally acclaimed medical journalist, questions the propriety of the decision to allow fluoridation to remain in United States' drinking water. Dr. Werbach writes, "The fluoride controversy is simply another example of the peculiar bias mainstream medicine shows towards apparently powerful new treatments whose dangers are largely unknown, while it continues to show bias against many gentler therapies with safety records far exceeding those of standard treatments."36

A University of Arizona study, published in the July 27, 1992 issue of *Chemical & Engineering News* by Cornelius Steelink, PhD Professor Emeritus in the university's Department of Chemistry, reported that the more fluoride a child drank in its water supply, the more cavities appeared in the child's teeth .37

The City of Tucson, Arizona provided Dr. Steelink with a unique opportunity to test the many fluoridation hypotheses handed out wholesale by proponents of fluoride additives to drinking water. The professor writes: "Historically, this city has had discrete geographic areas of groundwater with high fluoride contents of 0.8 ppm and areas of low fluoride contents with 0.3 ppm." When Dr. Steelink's evaluation committee plotted the incidence of tooth decay versus fluoride content in a child's neighborhood drinking water a positive correlation was revealed. As stated above, Professor Steelink's committee reported with this exact summarizing quote: "In other words, the more fluoride a child drank, the more cavities appeared in the teeth. "38

David C. Kennedy, DDS, of San Diego, California, the author of How to *Save Your Teeth: Toxic-Free Preventive Dentistry,* outright declares that fluorides do not reduce tooth decay. 39

In a published letter, Dr. Kennedy writes to the Safe Water Coalition of Washington State: "In Canada, the areas which report the lowest incidence of decay are the unfluoridated areas. Tooth decay is declining world-wide with no statistical difference between fluoridated and unfluoridated areas. Some authors [proponents of fluoridation] attempt to attribute the decline of cavities in unfluoridated areas to a decrease in the consumption of refined sugars. I believe statistics show we are consuming more, not less refined sugar. The latest data from the National Institute of Dental Research (NIDR) found no difference in the incidence of tooth decay in children ages five through 17 years raised in nonfluoridated, partially fluoridated, and fluoridated communities. NIDR studies show no relationship between fluoridation and tooth decay rates."40

Fluoride Reacts with Aluminum to Cause Alzheimer's Disease

Newly declassified documents, obtained under the US Freedom of Information legislation, today provides shocking medical facts known but concealed by the US Government since the 1940's. During that same period when Dr. H. Trendley Dean changed his laboratory test findings and ALCOA attorney Oscar Ewing won his political appointment, the United States Public Health Service knew that fluoride produces adverse human central nervous system effects. It's true! For over 50 years, the USPHS has hidden this horrible information about fluoride as a polluter and deteriorater of the human brain.

Ellie Rudolph, Director of the Pennsylvania Chapter of the Health Alliance International, advises that pathological changes in the brain tissue of animals given fluoride and aluminum-fluoride combined are the same changes found in the brains of people with Alzheimer's disease and other forms of dementia. Director Rudolph states: "Low levels of fluoride have serious health implications for people and the effect is enhanced in the presence of other neurotoxins like aluminum .1141 (To discuss this hidden aspect of fluoridation pathology, you may contact Ellie Rudolph in person by consulting Appendix A of my book, *Elements of Danger: Protect Yourself Against the Hazards of Modern Dentistry.)*

The peer-reviewed medicaljournal, Brain Researchh, reveals that aluminum-induced neural degeneration in rats is greatly increased when the animals are fed low doses of fluoride. *The* presence of fluoride enhances the bio-availability of aluminum causing more aluminum to cross the blood-brain barrier and become deposited in the brain. The brain researchers write: "The aluminum level in the brains of the fluoride-treated group of animals was double that of the controls ."42

Even worse, the study's authors say, "While the small amount of aluminum-fluoride in the drinking water of rats required for neurotoxic effects is surprising, perhaps more significant are the neurotoxic results of sodium fluoride (Nor) at the dose given of 2.1 ppm Nor. This 2.1 parts per million NaF equals 1.0 mg fluoride ion per lure of water *which is the same level found in 1.0 ppm "optimally" fluoridated drinking water.*

Note: The formula for converting NaF to fluoride ion is ppm x 45% so that 2.1 ppm x 45% =.95 ppm (ppm = mgs/litre).

The present fluoride/aluminum study on the brains of laboratory animals confirms the work of two separate groups of scientists in China, each group publishing their investigations in 1995 and reported on earlier in this chapter. Both Chinese studies showed that drinking water containing fluoride adversely affects the intelligence quotients of children.","

Colgate-Palmolive Compensates Brits for Staining Teeth Brown

Selling its fluoridated Colgate toothpaste in the United Kingdom, America's Colgate-Palmolive Corporation paid out the equivalent of US$2,000 to the parents of a ten-year-old British child for fluoride damage to his teeth. It was "goodwill" compensation after an independent specialist diagnosed the boy as having developed dental fluorosis from swallowing small amounts of fluoride toothpaste over a period of time. Headlines appeared featuring the November 24, 1996 payout in every major British newspaper, including The *London Times*

Dental fluorosis, a permanent brown discoloration and mottling of the teeth caused by exposure to fluorides in the drinking water and products such as fluoride toothpaste, is a sign of systemic toxicity. Even though the Colgate@ case was settled out of court, British lawyers said that it set an international precedent, opening the door for future toxic tort litigation related to fluoride-containing products.

Solicitor (attorney) Julian Middleton of the Nottingham, England law firm Freeth, Cartwright, Hunt & Dickins said that he represents over 200 families with children suffering from dental fluorosis. This Colgate@ payment will help families in their battle for legal aid to file a class action suit. The Nottingham law firm has made several attempts in the past few years at obtaining legal aid for the families and have been turned down by the UK courts. Unlike the United States' legal system in which law firms can handle class action suits on a contingency basis, in the UK government funding is required for such suits. Also in the USA, it requires only one person to file a class action suit on behalf of everyone who has suffered injury from a product or action.

In any country, dental fluorosis is highly litigable for a number of clearcut reasons:

1. Dental fluorosis is the most visible of the adverse effects of fluoride ingestion. 2. Dental fluorosis is the most easily diagnosed. 3. Dental fluorosis occurs from only one cause.

In the United States, an estimated 30% of all children in nonfluoridated water areas suffer with some small degree of dental fluorosis from all sources. In contrast, about 62% of the children in areas with fluoridated water show obvious signs of dental fluorosis. Therefore, the United Kingdom's attorneys are amazed that numbers of law suits haven't already been filed by American law firms. Traditionally, in product liability cases, the Americans are ahead of us," said Paul Balen, solicitor for Freeth, Cartwright, Hunt & Dickens , 'but for once we appear to be ahead of the Americans."'15

The key to dental fluorosis litigation involves: *Failure to adequately warn the public of adverse effects caused from exposure to fluorides.* Certainly the ADA and the USPHS have failed miserably in this regard. They declare dental fluorisis as merely a cosmetic effect, but it's not. Such a declaration is illustrative of the big lie - a coverup! Expert witnesses such as toxicologists, physiologists, internists, pediatricians, holistic dentists, anatomists, and other health professionals would prove the lie in such a statement. Then, malpractice and product liability lawsuits could be leveled at the following groups:

pediatricians, conventionally-practicing dentists, and other health professionals who prescribe fluoride supplements,

fluoride toothpaste manufacturers,

communities and townships fluoridating drinking water,

organizations and media outlets such as newspapers endorsing the use of fluoride supplements or water

fluoridation,

manufacturers of fluoridation equipment,

manufacturers of foods and beverages containing fluorides,

associations and individuals involved in the promotion of fluorides as a palliative,

media outlets advertising fluoridated products,

advertising agencies handling the accounts of fluoridaters.

It appears that attorneys have a potential bonanza waiting. in the wings, once they pick up on the massive amount of liability that prescribing health professionals, cities, states, and commercial organizations have created for themselves. Such liability is unquestionable because the damage to patients with dental fluorosis is permanent. It's classified into three categories:

1.Mild fluorosis means that there is barely discernable chalky blotching on the teeth.

2.Moderate fluorosis indicates that there is discernable chalky blotching to rust-colored stains on the teeth.

3.Severe fluorosis is shown by brown-stained, pitted, and friable teeth.

Being a permanent condition, there are only costly remedies to disguise the discolorations. They include dental bleaching, micro-abrasion, cosmetic bonding, and capping. Most cases of severe dental fluorosis results in loss of teeth or the formation of cavities in fluorosed areas of the teeth. In cases where such teeth have become friable, repairing such cavities is difficult if not impossible.

While some fluoridation suits have already taken place, as for instance *Feldman v. Lederle Laboratories,* American lawyers have only begun to sue for the enforcement of fluoridation on the public. Now the fluoride proponents will finally have their day in court. In my opinion, that's a good thing!

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Correspondence

The following are some letters to the Australian Dental Association and the Australian Society of Endodontology, requesting information regarding the scientific verification of statements made in their literature. The replies are also published.

As you will see neither of these associations have any scientific references to support their statements. If they do exist then they are certainly unwilling to share them with me. To date, after numerous requests from myself and other colleagues, the Australian Dental Association, have not presented one scientific paper to support their position.

Take the time to write and ask the same questions!

From Robert Gammal May 21 1997

To; Dr Robert Butler, Chief Executive Officer, AUSTRALIAN DENTAL ASSOCIATION INC.

P.O. Box 520 St. Leonards. NSW 2065.

Dear Dr Butler,

Recent publications of the Australian Dental Association seem to be producing confusion regarding the position the Australian Dental Association holds about certain amalgam issues. I have read in one journal that some mercury comes out of amalgam and in other publications of the Australian Dental Association that no mercury is released. I would appreciate an answer to some questions so that I might know the exact position that the Australian Dental Association holds in these matters.

Does the Australian Dental Association accept that mercury is released from set dental amalgam

If the answer is yes, how much mercury is released in the opinion of the Australian Dental Association. Please give a specific quantity for release of mercury vapour in micrograms per cubic meter. Please cite the reference, which supports this view.

Does the Australian Dental Association consider dental amalgam to be a potential source of mercury.

Does the Australian Dental Association consider 'dental amalgam' to be an amalgam of metals or a true alloy of metals.

Please let me know the position the Australian Dental Association takes in regard to the ethical considerations of amalgam replacement.

What is the position of the Australian Dental Association regarding the estimated percentage of people who show an allergy to mercury. Please cite the references which support this position.

Does the Australian Dental Association accept that other immune reactions to mercury and (metals in the mouth) are possible.

Does the Australian Dental Association accept that mercury from amalgam crosses the placenta and is absorbed by the fetus, and also crosses via the breast milk to be absorbed by the feeding infant.

What is the current Australian Dental Association position regarding a safe level of mercury vapour for people who are occupationally exposed and for the rest of the population. Please cite the references on this position

With regard to Root Canal Therapy issues would you please verify that the position the Australian Dental Association has, is supported by the position and statements of the Australian Society of Endodontology.

I look forward in anticipation to your reply.

Yours truly,

Robert Gammal

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Reply from AUSTRALIAN DENTAL ASSOCIATION INC.
P.O. Box 520, St. Leonards NSW 2065.

June 11, 1997

Dear Dr. Gammal,

The confusion concerning the position of the Australian Dental Association on amalgam issues is only in your mind. Read the world-wide research and make your own decision.

Concerning root canal therapy, I would refer you to the Australian Society of Endodontology.

As a member of the ADA, you are entitled to all of the services and publications of the Association. It is my personal decision that our busy Executive Director not spend an inordinate amount of time answering questions which you can research yourself. The enclosed documentation may be of assistance to you.

This is the end of the correspondence, Dr. Gammal.
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Yours sincerely,

Herb Hammer

Federal President

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From Robert Gammal July 14, 1997

To the Australian Dental Association

Dear Dr Hammer,

Thank you for your letter and the vast quantity of information about the opinions of various committees, who have reviewed some of the literature, and then made conclusions regarding the safety of dental amalgam. Unfortunately this material has nothing to do with the questions asked in my previous letter. These questions relate exclusively to the opinion held by, or the position taken by, the Australian Dental Association, in regard to some specific issues regarding the use of dental amalgam.

I understand from your letter that neither you nor the Australian Dental Association may wish to continue this correspondence with me. I must make comment to this matter at the outset.

Dr Hammer, as president of the Australian Dental Association you must appreciate that many members would look to you for leadership. The issue of leadership becomes more difficult when controversial issues must be discussed. As you can see from a perusal of your records, I have been a fully paid member of the Australian Dental Association for the past twenty years. As such, I believe that I and any other member of the Australian Dental Association, have the right to know the position of the association concerning major issues of dentistry and public health. The Australian Dental Association is after all, supposed to provide leadership for the dental profession. A refusal to supply this information must be seen at worst as a breach of contract between the Australian Dental Association and myself, and at best an abuse of my rights as a member of the Australian Dental Association.

I am well aware that the official position of the Australian Dental Association regarding the safety of amalgam, is that the material is perfectly safe. I also understand that you support this position on the advice of various committees such as the NH&MRC, the FDA, and the Swedish Medical Council. I am also aware that you understand the position that I take regarding the safety of dental amalgam. No matter what the beliefs of a particular member of the association, I hereby formally request, as a member of the Australian Dental Association, that the Australian Dental Association provide me with information concerning the following questions. If the Australian Dental Association does not have an opinion or a position in regard to these issues, would you be kind enough to let me know.

Does the Australian Dental Association believe that mercury is released from set dental amalgam in the oral cavity?

Does the Australian Dental Association believe that mercury is released from set dental amalgam which is regarded as ‘waste’ amalgam?

If the answer to question 1 is ‘yes’, please specify the amount of mercury which the Australian Dental Association believes comes from amalgam in the mouth.
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If the answers to questions 1 & 2 are 'no', please specify the references used to support this position.

What level of mercury vapour does the Australian Dental Association believe is a safe level for the following situations. (Please supply an answer in micrograms per cubic meter.)

Industrial exposure

Dental Surgeries

Oral Mercury Vapor in children

Oral Mercury Vapor in adults

Does the Australian Dental Association support the conclusions of the World Health Organization Criteria 118 1991?

Does the Australian Dental Association support the continued use of dental amalgam as a restorative material in school dental clinics?

Does the Australian Dental Association believe that there is a minimum age below which dental amalgam should not be used?

Does the Australian Dental Association consider 'dental amalgam' to be an amalgam of metals or a true alloy of metals?

Please let me know the position the Australian Dental Association takes in regard to the ethical considerations of amalgam replacement?

What is the position of the Australian Dental Association regarding the estimated percentage of people who show an allergy to mercury. Please cite the references which support this position?

Does the Australian Dental Association accept that other immune reactions to mercury and (metals in the mouth) are possible?

Yours truly,

Robert Gammal

The above letter has still not been answered

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From Robert Gammal

To Australian Society of Endodontology (Inc.),

Dr Ralph Reid

12th Floor

TNG Building

141 Queen St

Brisbane

4000

Ph 07-3229-4209
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Dear Dr Reid,

I am writing with a request for information which I hope you, as president of the Australian Society of Endodontology (Inc.), will be able to supply. I am a practicing general dentist in Sydney and have a great interest in the area of Endodontics. My queries are in relation to the patient education pamphlet;

"Relax- there is no need to lose your tooth…ENDODONTICS (Root Canal Therapy) can save it for you".

1) In paragraph 2 it is written "Once the tooth is fully formed the main source of nutrition for the tooth comes from the tissues surrounding the root."

Could you please supply the references for this statement? Would you also be kind enough to explain to me exactly how the tooth is nourished from its surrounding tissues. Is this via the blood supply, the lymph or by osmosis?

2) In the third paragraph it is written;

"Therefore, a tooth can function normally without its pulp and can be kept indefinitely. After endodontic treatment the tooth is pulpless, but it is NOT a dead tooth."

Again I would appreciate references to support this statement. By suggesting that the tooth is not dead, one can only assume that it is alive. For this to be so it must have some vascular supply. If I am not mistaken the very procedure of Root Canal Therapy is to remove the blood supply.

The statement (7th Paragraph) "During endodontic treatment, the infected or damaged pulp is removed from the inside (i.e. root canal) of your tooth."

Is it necessary to remove all infected dead pulp tissue from the tooth? If not please supply references which describe the fate and effect of remaining infected tissue.

If so please supply the references which demonstrate that all necrotic and infected tissue can be removed from the tooth.

The 8th Paragraph states: "The root canals are then cleaned, sterilised and shaped to a form that can be completely sealed." Firstly I again request references to support this statement. Next would you be kind enough to explain to me;

the procedure and medication recommended by the society which does sterilise a tooth.

how is sterility of the tooth determined? Is it necessary to take a swab of the tooth for culturing. If so should this be aerobic or anaerobic.

if anaerobic testing is required could you please inform me of the correct procedures.

please supply references which demonstrate the complete sealing of a root canal.

Paragraph 11 talks of the sedative dressings and temporary fillings which are used to settle the tooth "and destroy any remaining bacteria" . References supporting

this statement would be appreciated. Would you also list for me the medicaments which are currently recommended to achive this outcome.

I appreciate that you may not be the author of this pamphlet and that this is indeed quite a large request. I believe though, that if I am to pass this pamphlet on to my patients, I would like to be in a position to be able to verify each of these staements by published, peer reviewed scientific papers.

If you are unable to furnish the answers to this request I would appreciate it if you could point me to the author of this papaer. I thank you in advance for your response.

Yours sincerely

Robert Gammal

Reply from Australian Society of Endodontology (Inc.),

Dear Dr Gammal

Thank you for your original letter of the 12th April. The request was handed on to our committee which handles educational matters. I have just returned from three weeks away, hence the delay in replying.

The committee made the following recommendations which are passed on for your

information:

- the pamphlet was written by a committee of specialist endodontists as a public service to dentist's patients.
- the pamphlet was then circulated to all specialist endodontists in Australia for their
- comment, additions, etc before final printing.
- the material was based on the committee members' general knowledge of endodontics and not on specific references.
- the statements are universally accepted by endodontists worldwide and by the dental profession in general.
- there are no controversial issues raised in the pamphlets (this was intentionally avoided by the committee).
- NO specific references were used to **write the pamphlets.** ANY text book on endodontics could be used to justify the statements made in the pamphlets.

I hope this information is of some help in showing where the pamphlets have come from.

Yours sincerely,

Ralph J Reid President, ASK Inc.

Australia's leading specialist endodontic society

could not find ONE scientific reference
to support the advice they give to patients.

NOT ONE!

This begs the question;
if the brochure was put together from the 'general knowledge'
of endodontists, then what is that general knowledge based on
if not on any published science?

NICO And Cavitations

This paper is from Dr W Shankland

http://www.drshankland.com/osteocavitation_lesions.html

History and Overview

Cavitations or NICO lesions are hollow places in jaw bones. These hollow areas may never cause pain or a problem. However, cavitations can produce trigeminal pain, headaches, and facial pain. Cavitations are common in all bones that have bone marrow. Many cavitations linger for years without producing facial pain. Most people know what we mean when we say cavity, but the word cavitation is confusing. Both of these words come from the same root word meaning hole. A cavity is a hole in the tooth, whereas a cavitation is a hole in bone. Unlike most tooth cavities, bone cavitations can't be detected by simply looking at the bone, and even using x-rays, many cavitations are missed. The termed cavitation was coined in 1930 by an orthopedic researcher to describe a disease process in which a lack of blood flow into the area produced a hole in the jawbone and other bones in the body. Dr. G.V. Black, the father of modern dentistry, described this cavitation process as early as 1915 where he described a progressive disease process in the jawbone, which killed bone cells and produced a large cavitation area or areas within the jawbones. He was intrigued by the unique ability of this disease to produce extensive jawbone destruction without causing redness in the gingiva (gums), jaw swelling, or an elevation in the patient's body temperature. Essentially, this disease process, which produces osteonecrosis (dead bone) is actually a progressive impairment which produces small blockages (infarctions) of the tiny blood vessels in the jawbones, thus resulting in osteonecrosis, or areas of

dead bone. These dead, cavitational areas, which produce pain, are now called NICO (Neuralgia Inducing Osteonecrosis) lesions (Figure 1). In his book on oral pathology, Dr. Black suggested surgical removal of these dead bone areas.

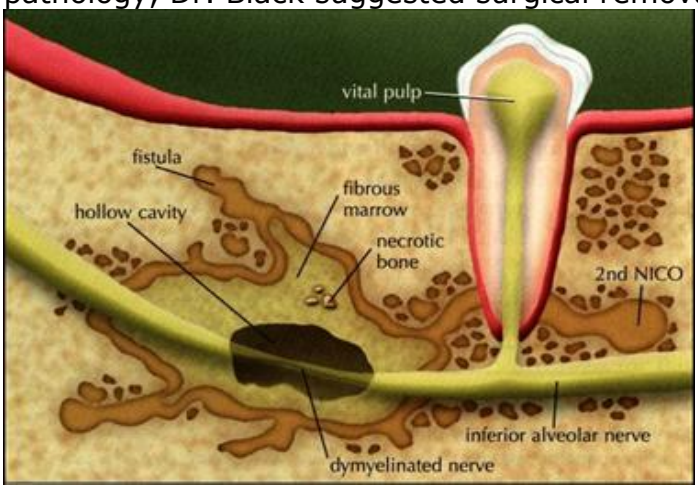


Figure 1: Diagram of cavitation lesions in the mandible.

Symptoms of Cavitations

Cavitational lesions may produce no symptoms at all, especially if we find no redness over the area or signs of drainage. However, these lesions may also produce intense, trigeminal neuralgia-like symptoms, which cause suffering to such an extent that it's a wonder patients can stand the pain and suffering.

There are established, characteristic referred pain patterns (Figure 1), which we find very consistent in most symptomatic cavitation cases. Patients with pain usually have an underlying, constant dull aching. Along with this gnawing, deep pain, often there's a sharp, shooting pain, which, understandably, convinces doctors that the diagnosis is trigeminal neuralgia.

A very common symptom we find is a sour, persistent drainage from the cavitation directly into the mouth. This foul taste makes many patients and doctors alike consider a diagnosis of sinusitis. Unfortunately, all the sinus surgery in the world will not correct the problem if the sour fluid is draining from areas of dead bone, namely, a jawbone cavitation.

Some of the more common symptoms of cavitations are:

Deep bone pain and pressure, which may be constant but vary in intensity
A sour, bitter taste, which often causes gagging and bad breath
Sharp, shooting pain from the jaws, which eludes doctor's diagnostic attempts
Chronic maxillary sinusitis, congestion and pain
A history of large dental fillings followed by pain, root canal therapy, and ultimately, removal of the tooth
Multiple root canals
Endodontic surgery (apicoectomy)
Difficult tooth extraction, including wisdom teeth, several years earlier
Post-operative complications, especially the development of a dry socket
Failed attempts to treat trigeminal neuralgia

To confuse matters more, many patients report systemic symptoms like arm or leg pain and generalized fatigue. We've seen these systemic symptoms improve, or completely resolve, once the cavitation (or cavitations) is removed. The same has been seen in some chronic fatigue cases.

The most common scenario we see usually starts with a simple dental restoration. The family dentist replaces an old restoration (filling) and the tooth becomes

sensitive, especially to cold temperatures. The doctor may replace the filling again or several more times, but the sensitivity never decreases. Then, in most cases, the tooth is treated with root canal therapy. But guess what? The pain continues. Another doctor is consulted, only to have the tooth re-treated with root canal therapy, but the pain persists . . . generally worse than in the beginning. Finally, out of sheer desperation (of both patient and doctor), the tooth is extracted, only to have the pain continue and intensify.

In this scenario, the finest dentistry was performed, but something went wrong. It wasn't neglect by the dentist but damage to the tiny vessels in the jaw around and beneath the injured tooth. Due to the constant inflammation and swelling, an infarction occurs in one or more of the tiny vessels, producing ischemia and, ultimately, bone death and cavitation formation (Figure 2).

Remember, cavitations may be completely painless. This is not unique to the jawbones. In other bones, such as the femur, often there is no pain even when the bone destruction is extreme.
Top

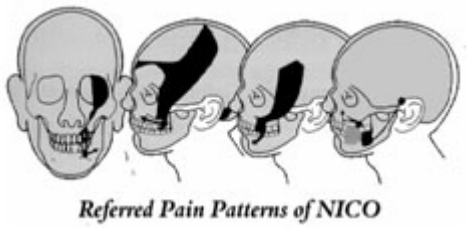
Location of Cavitations

Table 1: Common locations of NICO lesions.

Figure 2: Referred pain patterns of NICO lesions.

Alveolar location	Maxilla	Mandible	Total
Central incisor area	2.5 %	0.2 %	2.7 %
Lateral incisor area	3.6	0.2	3.8
Cuspid area	5.0	2.0	7.0
First bicuspid area	5.2	1.1	6.3
Second bicuspid area	4.8	3.4	8.2
First molar area	6.8	12.6	19.4
Second molar area	2.6	5.1	7.7
Third molar area *	20.0	24.9	44.9
Total:	51.5 %	48.5 %	100.0 %

* includes tuberosity and retromolar areas



Referred Pain Patterns of NICO

In the last several years, the term cavitation has been used to describe various bone lesions which appear both as empty holes in the jawbones and holes filled with dead bone and bone marrow. In Table 1, common locations of NICO lesions are listed. Note that the most common locations overall are areas of wisdom teeth (third molars).
Often, these NICO lesions take years to develop, usually producing few if any symptoms . . . for a while. Then, generally for unknown reasons, pain in the jaws, face, head and neck may develop. There are characteristic referred pain patterns, which generally confuse patients and doctors alike (Figure 2).

Current Research

The results of recent research of Dr. Boyd Haley (former Chairman, Department of Chemistry, University of Kentucky) show that **ALL** cavitation tissue samples he's tested contain toxins, which significantly inhibit one or more of the five basic body

enzyme systems necessary in the production of energy. These toxins, which are most likely metabolic waste products of anaerobic bacteria (bacteria which don't live in oxygen), may produce significant systemic effects, as well as play an important role in localized disease processes, which negatively affect the blood supply in the jawbone. There are indications that when these toxins combine with certain chemicals or heavy metals (for example, mercury), much more potent toxins may form.

Systemic Problems Associated With NICO Lesions

Researchers early in the 20th century and now recently have been concerned with systemic diseases caused by a primary problem (a focus of infection). The focal theory of infection fell out of favor with medical and dental doctors after the advent of antibiotics, but may researchers today believe that in spite of antibiotics, the focal theory of infection is alive and well. Ask and veterinarian doctor, and he or she will immediately agree that the focal theory of infection is a great concern of theirs.

Many researchers today believe that NICO lesions are the focus of various infections which may spread throughout the body. In the last few years, some of the most surprising medical news has been the discovery that bacteria from the mouth appear to be very influential in causing various heart, liver and kidney problems. If you have a joint implant or mitral valve prolapse, your dentist must prescribe an antibiotic before any dental treatment. Why? Because bacteria from the mouth can spread through the blood to cause serious problems elsewhere in the body. Could the toxins from NICO lesions do the same?

Initiating, Predisposing, and Risk Factors for NICO

There are many initiating, predisposing, and risk factors associated with cavitational lesions. It's likely that a combination of these factors present in a someone may influence the occurrence, type, size, progression and growth patterns of a cavitational bone lesion.

Initiating Factors: Probably the major initiating factors are dental trauma, which produce physical, bacterial, and toxic components, as described below.

Table 2: Dental traumas (**initiating factors**) associated with cavitational bone lesion development.

Physical Trauma
Bacterial Trauma
Toxic Trauma
Tooth Extractions
Periodontal Disease
Dental Materials
Dental Injections
Cysts
Root Canal Toxins
Periodontal Surgery
Abscesses
Anesthetic by-Products
Root Canal Procedures
Root Canal Bacteria
Anesthetic Vasoconstrictors
Grinding and Clenching
Non-vital (dead) Teeth

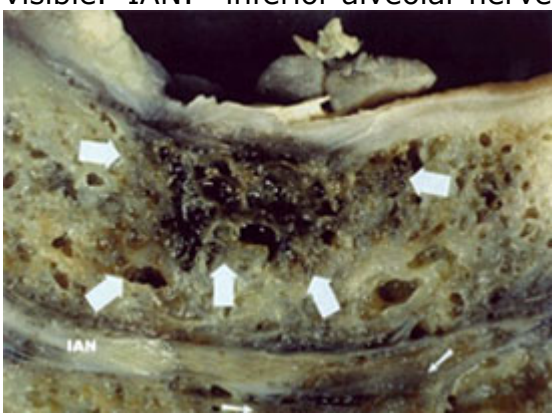
Chemical Toxins
Electrical Trauma from Dissimilar Metallic Restorations
Improper Removal of Periodontal Ligament after Tooth Extraction?
Bacterial Toxins
Heat from High Speed Drilling
Infected Wisdom Teeth
Other Toxins

Predisposing Factors: There are many predisposing factors and no doubt, many more will be discovered. Most of the known predisposing factors include: blood clotting disorders such as thromophilia, hypofibrinolysis, or others; age -- evidence suggests that as many as 11% of older persons may have major or complete blockage of arteries feeding the jaws or of the smaller arterioles within the jaws themselves; radiation or chemotherapy for cancer; rheumatoid arthritis; lymphoma or bone dysplasia; changes in atmospheric pressures in occupations; osteoporosis; systemic lupus erythematosis; sickle cell anemia; homcystinemia; Gaucher's disease; hyperlipidemia; hemodialysis; gout; antiphospholipid antibody syndrome; physical inactivity (bedridden); and deficiencies of thyroid or growth hormones.

Risk Factors: There are many risk factors which greatly increase the probability of the development of cavitational lesions, especially in the occlusion or blockage of tiny blood vessels within the jawbones. The most common risk factors are: heavy smoking; high and long-term cortisone usage; pregnancy; estrogen use; alcoholism; and pancreatitis. Undoubtedly, there are many other risk factors.
Wisdom Teeth Sites: Research findings indicate that 45% to 94% of all cavitational lesions are found at wisdom teeth extraction sites. These areas are anatomically predisposed to develop these bony lesions because they contain numerous tiny blood vessels which are apparently, easily damaged from trauma (oral surgery in these areas) and osteonecrosis can easily develop. Also, many local anesthetic injections are given in the wisdom tooth areas and many of the local anesthetic solutions contain vasoconstrictors (especially epinephrine) which is used to intentionally close or shut-down the blood supply to the bone, teeth and gingiva to prolong the effects of the anesthetic and reduce bleeding. The actions of closing down the blood supply to these wisdom tooth areas may be a major cause for NICO development.

The Appearance of NICO Lesions

Figure 3: Gross appearance of NICO lesions. Note that at least 4 lesions are visible. IAN: inferior alveolar nerve.



Cavitational lesions are difficult to discover. On most x-rays, unless the doctor is specifically trained, these bony lesions are usually missed.

Gross examination of NICO lesions are shown in Figure 2. Note the large nerve, the inferior alveolar, as it travels through and between NICO lesions.

Dental students and residents spend a lot of time learning to properly read x-rays of all types. A very useful x-ray view in dentistry is the panoramic radiograph. Unfortunately, all of us were trained to read certain irregularities as normal! We now know that these irregularities on panoramic x-rays are quite often cavitational lesions.
(Photos Deleted for this handout – check out the website)

Diagnosis of Cavitations

The diagnosis of cavitation lesions is complicated by the fact that x-ray examination of the jawbones often appears normal . . . to the untrained eye. Considerable diagnostic experience is required to detect disorders that mimic cavitations, including variations of normal anatomy.

Why is this so? Osteonecrosis is a disease of the marrow spaces of bone and 40% to 50% of such bone must be destroyed before changes can be seen on x-rays. So, if your dentist or oral surgeon takes an x-ray and pronounces the film normal in spite of your symptoms, don't necessarily believe it. X-rays may be interpreted as normal unless (1) there's a significant amount of bony destruction or (2) the doctor is experienced in reading x-rays specifically for cavitations.

Although MRI (magnetic resonance imaging) is the imaging technique of choice for long bones, flat bones of the face are not imaged well with regular MRI scans. CT scans are also ineffective in locating most cavitations in the jawbones.

However, we've discovered that using the technique of MRI STIR imaging (Figure 15) is very effective and accurate in locating areas of bone marrow edema (swelling) and ischemia (areas of reduced oxygen). Both of these conditions can and do lead to the formation of cavitations.

(Photos Deleted for this handout – check out the website)

Bone scans using a radioactive isotope are somewhat helpful in locating cavitations but very difficult to interpret. Also, radiologists, not expecting these lesions in jawbones, often note the lesions in their radiology reports but interpret the results as normal.

The best, most effective method to locate cavitations is the Cavitat bone scanning device (Figure 17). This computer-based sonar imaging system was designed to aid the medical community with a detailed profile of the interior of bones. The Cavitat computer generates digitized two and three dimensional images of the interior of the jawbones from sound waves passed through the bones.

Because liquid is a near perfect conductor of sound waves, when these waves enter into voids or porosities in bone (areas that have compromised bone flow; i.e., cavitations), the sound waves slow down considerably, which produces images of the interior of the bony area being scanned. We've found the Cavitat results to be very accurate, especially when compared with patients' panoramic x-rays. Our

diagnostic results have improved dramatically. Most importantly, our surgical successes have soared since we began using this revolutionary device.

Therefore, since both MRI STIR imaging and ultrasound imaging (Cavitat) are so effective and accurate (Figure 18), since November of 2003 we're been using both imaging techniques with most patients. Using both of these diagnostic tests have helped improve our diagnostic abilities and better yet, have improved our overall success rate in treating cavitations of the jaws.

For patients experiencing pain, diagnosis is further improved through anesthetic confirmation or anesthetic blocking. By giving a local anesthetic injection (similar to having your dentist numb the jaw before he or she performs a dental procedure), pain in the jaws can be selectively turned-off, meaning the sense or feeling of pain can be chemically and temporarily eliminated. If the pain goes away after the injection, then we can be reasonably certain that there's a problem in the anesthetized area, generating pain.

Recommended Treatment of Cavitational Lesions of the Jaws

The only treatment available at this time to remove cavitational lesions is surgical removal. Some have attempted to inject homeopathic remedies or ozone into these areas of dead bone, but unfortunately, there's no blood circulation within cavitational lesions, so any medications, drugs, or remedies can't get into and permeate these lesions, let alone allow toxins and metabolic products to be removed. Homeopathic remedies certainly have their place in NICO treatment, especially in healing after surgical removal of the lesions themselves.

The surgery basically consists of making an incision, exposing the bony defects, and scraping them clean (termed debridement) to remove all unhealthy bone and other pathological problems like abscesses and cysts. It's not sufficient to simply punch a hole in the bone and rinse the area out, like some doctors recommend. In fact, treating these expanding bony lesions in such a conservative fashion often makes the lesion and subsequent pain much worse.

After removing the dead bone and other pathological products, the goal in healing is bone regeneration. But first, if possible, we remove all predisposing and risk factors.

What Can A Patient Do?

If you think you might have a NICO lesion, what can you do? First, find a doctor who understands this disease process; one who is trained in effectively diagnosing and treating these bony problems. Unfortunately, there are precious few such doctors in the world and very few in North America at this time.

If you're experiencing pain, don't allow anyone to operate without first proving where your pain originates. This is done most effectively by closely evaluating x-rays and using diagnostic anesthetic injections to actually turn-off the suspected NICO areas to see if the pain is turned-off. There are characteristic referred pain patterns of NICO lesions and there are also characteristic responses to local anesthetic testing. Find a doctor who knows about these characteristic patterns and realize that most doctors who treat orofacial and TMJ pain know nothing about NICO lesions.

Be certain that the doctor obtains Cavitat scans, MRI STIR imaging, or both in the process of diagnosing your problem. Both of these imaging tests give us a view of the size and extent of cavitations and can also indicate if surgery is truly needed or not.

Root Canal Therapy

Prepared by Robert Gammal BDS February 1997

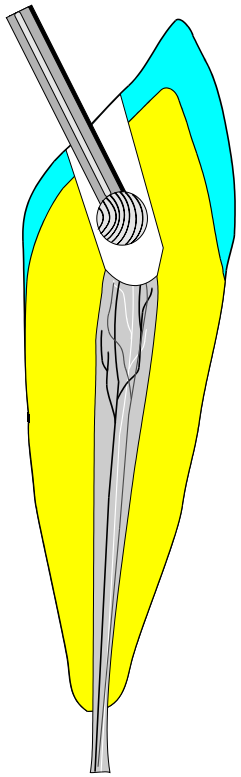
A brief paper summarizing some of the difficulties associated with the treatment of dead teeth

What is Root Canal Therapy

The aim of Root Canal Therapy is to 'save' a tooth which has become infected or dead, in an attempt to make it functional and pain free.

After scraping out the inside of the tooth the dentist will attempt to disinfect the tooth and the canals to eliminate any source of infection. The canal is then filled with a combination of cement and Gutta Percha in an attempt to completely occlude these canals. This is supposedly to prevent any microorganisms from entering the tooth either through the crown or the root.

If you consider pain control, mechanical function and aesthetics to be the limit of good dental treatment, then you will have "SAVED" the tooth.



If systemic effects are included in your concept of dentistry, than you must understand that all that has happened, is that you have kept dead, infected tissue, buried in the bone, within a couple of inches from your brain.

For some obscure reason we are all conditioned to think that teeth are not a part of the body, but that they are inert calcified material, and that they are sort of dead anyway. Dentistry is the only one of all the medical & para-medical professions that thinks it is a good idea to keep dead, gangrenous tissue in the body. The way to do this is to perform a Root Canal Therapy .

One eminent Endodontist says:

"It is wrong to speak of (Root Canal Therapy) as a dead tooth; it is more correct to describe such a tooth as nonvital or , better, pulpless. Even though the central blood supply to the tooth has been lost, the tooth itself still retains it's connection to the body via the periodontal membrane and the cementum."[1]

This is like saying that even though the blood supply to your leg may be completely cut off , it would be wrong to suggest that the leg is dead, because it is still connected to your body by your hip joint! The Oxford dictionary defines 'non-vital' as "Fatal To Life". It defines 'Dead' as "No longer Alive".

The Ritual Of False Beliefs.

There are many presumptions about Root Canal Therapy which are based in myth rather than science. The philosophy underlying the teaching of dentistry limits it's practice to mechanics, pain control and aesthetics. The systemic effects of dental treatment are rarely considered.

Dr. Weston Price was the leading dental researcher at the turn of the century. He was the head of the American Dental Association and wrote numerous papers on subjects as diverse as the role of nutrition on dental health to the effects of dead teeth and root canal therapy on systemic health. Dr. Price researched the effects of Root Canal Therapy for over twenty years. He was able to correlate different disease states with the types of pathology seen around dead teeth. He demonstrated thousands of times, the creation of diseases from non-vital teeth. He demonstrated how every belief about Root Canal Therapy, held by the dental community at the time, was based on a complete lack of scientific research. They were myths which developed and were then believed. These beliefs have now become set in concrete as truths by the current dental communities.

If you think that the research is out of date, you should realise that the techniques, most of the materials, and some of the instruments that were used then are identical to those used today. The medicaments used to 'sterilize' teeth then, are still being used today - Camphor, Phenol, Formaldehyde, Menthol.

Recently published research, completely supports that done by Dr Price. Specially that of Dr. Patrick Störtebeker, Assoc. Professor of Neural Surgery at Karolinska University in Sweden[2,3,4,5] and the work of Dr. Eugene Ratner[6],[7] in the United States.

Some of the myths that are still perpetuated include:

1 You can see infection on an x-ray.!

FALSE! Only if the angle is correct you may see some bone loss on an x-ray. It is impossible to demonstrate infection with an x-ray as dental radiographs only 'see' hard tissue. They do not see soft tissue or infections. Due to the shadow cast by the root it may also be impossible to see the bone loss.

2 You can gauge the extent of infection by the amount of bone loss on an x-ray.

FALSE! It is assumed in dentistry that the extent of bone loss is a direct indication of the amount of infection present. This is a false assumption because the bone loss may take time to develop. The extent of the bone loss about the end of the root is also a function of the body's immune system being able to isolate the infection process. It has little to do with the degree of infection.[8]
Sometimes there is no bone loss, but instead, a condensation of bone about the end of a dead tooth. We are taught in dentistry that this indicates a lack of infection. The reality is that teeth showing a 'Condensing Osteitis' are demonstrating that the body's immune system is <u>incapable</u> of quarantining the infection locally.[19] These are often the teeth which cause the greatest systemic effects. This is put neatly by Dr Josef Issels 1995 (translated direct from German):
"If the local resistance is already so weakened that the inflammatory focus no longer can become encapsulated, the inflammatory toxins will infiltrate without hindrance into the pulpa and the whole organism.
If an inflammatory process can no longer be localised and encapsulated, it proves, as emphasised by Pischinger and Kellner that the organism has become largely non reactive. On an X-ray, these teeth normally show no translucence. This is characterised as X-ray negative .

In our cancer patients, such non-encapsulated focus, and therefore X-ray negative teeth, do frequently exist. This indicates the enormity of low resistance of these patients."[9]

3 You can determine the length of a tooth by x-ray.

FALSE!
Dentistry teaches that a root canal must be filled to within 1mm of the root apex. The apex of a root canal is only rarely determinable by X-ray. Thus most root canals are worked too short, or so long that the root filling will protrude through the end of the tooth and into the bone. This is born out by research published in the dental literature:

"Thirty two canals in four mongrel dogs were treated endodontically. The mandibular third and fourth premolars were selected for study because their apices were widely spaced and could be studied individually without danger of confusion"

"Examination of the histologic sections revealed that in some cases root canal instrumentation had been terminated slightly short of the anatomic apex. Moreover some canals which appeared reontgenographically to be filled slightly short of the apex actually were associated with extrusion of some particles of sealer into the periodontal ligament space"

Five canals were accidentally overfilled. Of the 32 tested, 4 were overfilled. Therefore 5 out of 28 canals which were radiographically under-filled were in fact overfilled. **This is a failure rate of 17% in terms of basic endodontic procedure.**

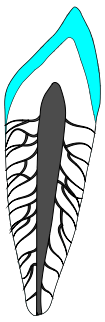
"In the canals which were overfilled, the extruded materials were always associated with advanced destruction of the surrounding tissue and liquification necrosis"[10]

It is not possible with an x-ray to see

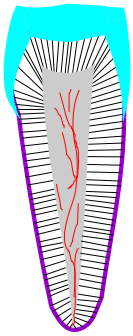
- the end of the root canal,
- the angle of the root canal,
- the number of canals or
- the various branches of each canal

4 It is possible to actually treat all of the hollow areas of the tooth. This is assumed to be limited to the actual root canals.

False!
It is assumed that the only part of the tooth which contains soft tissue is the actual root canal. Even in the latest Australian Dental Association handout on root therapy they state "All root canals in the effected tooth must be treated". [11] Unfortunately the root canals are the smallest area of the tooth which contains nerves, blood vessels and connective tissue.



The root canals are really like the tap root of a tree - one main root with hundreds of branches coming off it and opening to the edge of the root all the way along its length. It is impossible to treat these accessory canals.



As well, the dentine is not a solid structure. It is made of tubules which extend from the surface of the root canal to the enamel of the crown and to the cementum on the root surface. Each tubule is estimated to be able to contain 8 bacteria across its diameter. In a front tooth which has only one root there is over three kilometers of tubing. This equates to billions of microorganisms in just one tooth.

In comparison to the volume contained in the accessory canals and the dentine tubules, that of the root canal is actually quite small. It is not possible to remove dead infected soft tissue from whole of the tooth. When only the root canals are treated there remains a massive amount of gangrenous tissue which is infected by anaerobic microorganisms.

Dr Issels puts it this way; (note that this is a translation from German and directly quoted) [9]

"Altmann, Doepke and Pritz, as well as Fischer, Hess and other researchers have become involved with the fine structure of the tooth. They have found that the hard substance of the tooth in no way resembles an avital structure but maintains an active metabolic process with pulpa and dental periosteum. The pulper cavity and the external surface of the root are connected with each other via very fine canals. They are again connected via the mesenchymal fissures and capillars of the central periosteum with the canal system of the jaw bone and its pulper spaces and therefore with the general organism. This knowledge has refuted the concept, which had existed for decades, that the tooth, after removal and sealing off the pulper cavity, would be an isolated, avital structure no longer maintaining further exchange transactions. Even the most perfect preservation will only reach the most vertical intermediary trunk of the root canal system. In no way will it reach the lateral branches or the numerous dental canalculi, which likewise takes its exit from the root canal. Even after the most precise preparation of the root canal, there will always remain protein in the adjoining areas. This protein is usually infected and denaturated by filling materials, whereby toxic decomposition products will be formed. It was demonstrated by MEYER (Goettingen), that the dental canaliculi exhibits an exuberant bacterial flora. The decomposition toxins produced by these microbes can, with a dental root filling, no longer empty into the oral cavity. They can only be derived via the cross connection and the unsealed branches of the root canal finally reaching the pulper spaces of the jaw and thereby the flowing systems of the organism. Because of the devitalising and preservation procedures, the tooth has become a "toxin factory" by which the organism will be continually damaged."

It is claimed by most dental authorities that the bodies immune system will take care of what is left over. This is an assumption based in fantasy. If the blood supply of the tooth has been removed (which is what happens when the root canal is 'cleaned out') the cells of the immune system cannot get there.

Often during or before root therapy is started the dentist will administer antibiotics. This may lead to a rapid reduction in pain. Unfortunately both the dentist and the patient assume that the infection has been eradicated. The reason that the pain disappears is only because there is a reduction in pressure from around the end of the root. The antibiotics do not effect the organisms which reside within the tooth which are the original and continuing source of microorganisms and their toxins.

As there is no blood supply to the tooth it is impossible to get the antibiotics in there either.[12]

" In the case of an acutely infected tooth there is no natural process of drainage and there is no mechanism by which the antibiotics which have been administered can reach the bacteria inside the tooth" [1]

5 It is possible to sterilize the canal by using medicaments placed inside the canal.

FALSE!
It is impossible to sterilize the canals. The medicaments and antibiotics used do not penetrate the dentine tubules. Dr. Price was even able to culture bacteria from teeth through which he had poured fuming formaldehyde. Even the recent dental literature reflects this:

"It is now known that complete sterilization of an infected root canal is very difficult to achieve and complete removal of all pulp tissue remnants frequently is not possible." [13]

6 Bacteria that penetrate the canals and tubules are usually the 'aerobic' type found in the mouth. When the canal is sealed and the oxygen supply cut of, these bacteria die.

FALSE!
The bacteria, yeasts and other organisms which enter the tooth do not die when the oxygen supply is reduced (as happens inside the root canal system). They undergo what is called a pleomorphic change[14],[15] and become 'anaerobic' bacteria. They literally change form and become bacteria that do not need oxygen to live. It is now known that dead teeth are usually heavily infected with gram negative anaerobic bacteria. [16] Sundqvist, in 1976 isolated 88 species of bacteria out of 32 root canals with periapical disease.[17] "Only 5 of those bacteria could grow in air. Strict anaerobic bacteria must have played a decisive pathological role although a limited number of facultative species have been show to induce periapical lesions……."

long standing populations of infected root canals do contain a mixture of strict anaerobes. Low grade but chronic periapical inflammation is the result that may last for years."

Other organisms such as yeasts, funguses and 'cell-wall-deficient forms' (Lida Mattman) also inhabit this tissue.[18] The dead teeth thus become a focus of infection which can cause numerous disease states throughout the body. Anaerobic bacteria produce incredibly potent neurologic and hemolytic toxins. A true "Toxin Factory".

7 If it does not hurt it must be OK!

FALSE!
Weston Price's comments are most succinct;

"Local comfort..... may constitute both what is probably one of the greatest paradoxes and one of the costliest diagnostic mistakes through injury to health, that exists in dental and medical practice ..... the absence of this local reaction

and the consequent destruction by the infection products, permits them to pass through the body to irritate and break down that patient's most susceptible tissue".

Lack of pain around the tooth is usually taken to mean a successful root therapy. Unfortunately it does not rule out the possibility of systemic effects.

8 Systemic effects need not be thought of in relation to dental disease.

FALSE!
All researchers from Weston Price[19], Billings, Rosenow, Stortebecker, Ratner and many others, have demonstrated the spread of systemic disease from infected teeth and gums. It is only the dental profession, who are not trained in medicine, that refuse to accept this basic concept. The research of Steinman [20] in the 70's conclusively demonstrates the relationship of metabolic dysfunction and dental disease.

Patrick Stortebecker and others have demonstrated the transport of all materials, microorganisms and their toxins directly from the tooth back to the brain via the blood and by transport along the nerve fibres.[2,3,4,5] Many other research articles have shown that whatever you put in a tooth can be transported to the rest of the body.[21],[22],[23],[24]

As Schondorf states "A root canal treatment which does not plant a focus, does not exist"

Focal Infection Theory

The concept of focal infection has been around for well over 150 years. Since the time of Pasteur the medical and dental authorities have claimed that the concept of focal infection firstly cannot exist and secondly does not hold relevance to dead teeth which have been root therapied. Lately the dental associations are stating that to promote this theory is to set dentistry back by 150 years. Many researches over the years have successfully demonstrated that dead, root therapied teeth can in fact release organisms and their toxins into the body. These can then initiate disease states in other parts of the body. Stortebecker has even demonstrated that these organisms and their toxins can be transported directly back to the brain via the blood and also by transport along the nerve fibers. Other researchers have demonstrated that parts of the brain can be directly infected from dead teeth[25,26,27,28]

There is also research which demonstrates the presence of Tumor Necrotising Factor at the end in the apical area of infected roots.[29,30,31,32] Tumor Necrotising Factor is capable of causing; Chronic wasting syndrome, Anorexia & Weight Loss, Bone resorption (by it's osteoclast activating potential), Inflammatory disease states.

"A *Focus of infection* has been defined as a circumscribed area infected with microorganisms which may or may not give rise to clinical manifestations.

A *Focal Infection* has been defined as sepsis arising from a focus of infection that initiates a secondary infection in a nearby or distant tissue or organs."

" the concept of focal infection in relation to systemic disease is firmly established. The origin of many toxic or metastatic diseases may be traced to primary local or focal areas of infection. (Reimann and Havens)

Two mechanisms can produce focal infection:

1- an actual metastasis of organisms from a focus,
2-the spread of toxins or toxic products from a remote focus to other tissues by the blood stream. [33]

Also in the Journal of the American Dental Association we read:

"If the bacteria pass the barrier (of the abscess wall) a number of things may happen;

(Appleton)

the bacteria may be discharged from the focus onto a free surface whence, conveyed by mechanical means, they determine an extension of the disease by re-inoculation.

the bacteria escaping from the focus may be conveyed to distant parts of the body by way of the lymphatics or blood. Once the bacteria leave the focus they may be arrested by the nearest lymph nodes. A lymphadonitis gong on to abscess formation may develop. If the bacteria pass this barrier three things may happen (a) they may multiply in the blood setting up an acute or chronic septicemia. (b) they may be carried live to a suitable nidus where they infect the surrounding tissue. (c) they may produce a slow but progressive atrophy with replacement fibrosis in various organs of the body.

Products of bacterial metabolism or of the interaction of bacteria and the cells …….may reach remote parts of the body.

the bacteria at the focus may undergo autolysis or dissolution. Some of the products of this dissolution, diffusing into the blood or lymph , may sensitize in an allergic sense various tissues of the body. A later diffusion of these products on reaching the sensitized tissue may call forth an allergic reaction"

There is a suggestion in dentistry that if the infection is 'quarantined' it will not pose a danger to the rest of the body. The quarantining is regarded to be in the form of a Dental Granuloma (an encapsulated abscess). Unfortunately this position is not supported by the dental literature;

"the capsule contains a meshwork of capillaries among its fibers and is penetrated abundantly by larger vessels; thus direct communication is established in the inner part, or seat of inflammation and the circulation……"

In 1931 Freeman reported " there is no question that bacteria or their toxins are not limited by the fibrous capsule." [33]
To ignore the reality of focal infection is to allow dentistry to operate in the dark ages.

Also from the dental literature, we find an approach which defines loosely the type of people who will be effected by focal infections"

"A patient becomes susceptible to infection if any of these mechanisms *(immune function and reticuloendothelial system)* decrease in function, or if an organ is damaged to the extent that microorganisms can localize and produce an infection."

"Patients with rheumatic heart disease, congenital heart disease, heart valvular prosthesis, or patients with an inadequate defense mechanism are susceptible to severe consequences if they are subjected to a bacteremia. Inadequate defense mechanisms to resist bacteremias may result in cases of; debilitation or dehydration, exposure to radiation, diabetes, cancer, blood dyscrasias, malnutrition, vitamin deficiency, leukemia, multiple myeloma, diseases of the liver

or kidney, and in patients undergoing prolonged therapy with antibiotic, corticosteroids, immunosuppresives, and antimetabolites."[34]

This is just about everyone who undergoes any stress in their lives. Increase in the amount and variety of types of stress produces a severe drop in immune function.

Neural Focal Interference

Till now we have spoken of the effects of Root Canal Therapy in relation to microorganisms. There is another way of looking at the problem of dead teeth. It is related to a concept of medicine which came out of Germany in the 1950's. It was developed by two German doctors called the Heuneke brothers. What they found was that areas of dead tissue, scar tissue, foreign bodies, cystic tissue and infected tissue could interfere with the body's regulatory systems. They called these areas "Foci of Neural Interference."

The German Medical Association for Focal Research and Control, defines focus as: "an abnormally localised alteration in the organism, with the capacity to induce distant actions out of its immediate proximity." Any local circumscribed pathogenous organic alteration such as a chronic inflammation, a degenerative alteration, or a scar (independent of its size and location), can be active as a focus or as an "interference field".

The "focus" is defined by Pichinger and Kellner as a "chronic devious localised alteration in the connective tissue, which can cause the most diversive reactions out of its immediate environment and consequently is located in a permanent active relationship with the localised and general immune system."

Any chronic inflammation, any scar, any degenerative or other alteration can obviously satisfy this condition. The focus is embedded in the mesenchymal base tissue and in that way has direct contact with the capillary system of the blood and lymphatic vessels and the neuro-vegetative nerve fiber. This produces the connection to the whole organism. Through any of these conduction systems, it will be able to cause distant actions in other organs. The focal nerve impulse will be first projected into the vegetative centers, where it can cause a vegetative dysregulation which likewise can become retroactive to the whole organism again. On the other hand, focal toxins and bacteria will be infiltrated by the vessel systems where they are able to spread their infectious, toxic and allergenic properties everywhere."[9]

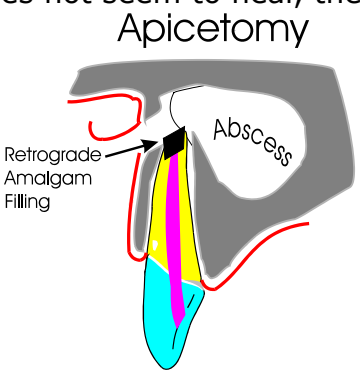
A neural interference field will create an imbalance in the bodies regulatory mechanisms, which include the tissue fluid around all of the cells of the body. Dead and infected teeth fulfill all the criteria to become Primary Foci of Neural Interference. The imbalance in the regulatory system will then either create or potentiate disease states in other parts of the body, which are remote from the original focus. These disease states will often coincide with areas of the body that are on the same acupuncture meridians as the primary focus. This has been verified by the work of Voll who was a German physician and electro-acupuncturist. For example we often see disease states in the areas of reproductive system, kidney and knees in relation to non-vital front teeth.

The mouth and teeth are a primary source of focal infection and neural interference fields. No other parts of the body have dead tissue routinely left in place. The only thing which seems to separate individual reactions is the state of that person's immune system and genetic factors. Consequently other factors which may reduce

immune function will allow a greater reaction to the non-vital teeth. (e.g. Mercury from dental amalgam fillings will have a direct and deleterious effect on the immune system)

Apicectomy & Retrograde Root Fillings

Sometimes, when an infection at the end of a root does not seem to heal, the dental surgeon will perform a surgical technique to clean the abscessed area. This is called an Apicectomy. This surgery is based on the belief that infected material escapes only through the end of the root (myth). Therefore as part of this procedure, a filling is often placed at the end of the root. This is called a Retrograde Root Filling. The material of choice which is most commonly advocated by the dental profession is often AMALGAM.

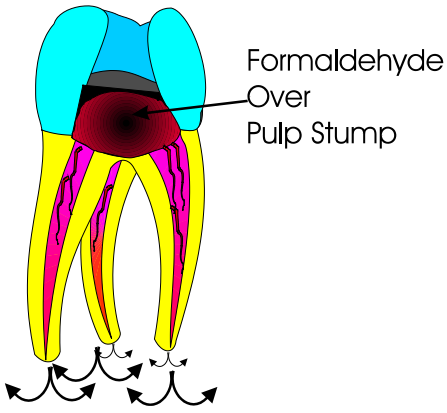


There is not one area of medicine that would condone the implantation of amalgam or mercury into bone. This is in fact what is being done daily in dentistry. It is equivalent to an implant of mercury directly into the brain! This is not an exaggeration. Many researchers have demonstrated that mercury vapor released from dental amalgam will migrate through the palate and the nasal linings to pass directly into the brain. [35] If the mercury is already inside the bone it will migrate freely to the brain.

If you have had this treatment it is vital to remove all bits of amalgam from the bone.

Pulpotomy

Due to the anatomy of the end of the root of a baby (deciduous) tooth it is not possible to do a root therapy. If a baby tooth is infected or dead the treatment which is still taught at Sydney University is called a Pulpotomy. This involves the removal of only the crown section of the pulp while leaving the remainder of the infected pulp in the root of the tooth. This pulp stump is then covered with a material which 'mummifies' the remaining tissue. The mummifying material is in fact a mixture of Formaldehyde and Cresol. The belief is that this material remains in the tooth. There is NO scientific foundation for this belief! In fact there is published research which demonstrates that Formaldehyde placed in teeth of cats and rats will migrate easily to every tissue in the body[36]. Formaldehyde is carcinogenic (cancer producing) in minute amounts.



Pulpotomies not only mummify the pulp but may start to mummify the child as well.

Symptoms

The types of disease states which relate to dead teeth are so numerous that it is impossible in an article of this size to discuss them all. They range from head and neck pain all the way through to rheumatism and cancer.

The most common symptom is in the form of head and neck pain. This may range from mild headaches to migraine to Trigeminal Neuralgia.

Sinusitis is very often associated with non-vital and Root Canal Therapied teeth especially if they are in the upper arch.

Price found that most patients with non-vital teeth had some thyroid dysfunction.

A number of researchers and physicians are finding a relationship between cancer and non vital teeth.

Reduced immune function is common.

Eye and Ear problems are common with root therapied teeth.

Rheumatic and Arthritic changes are almost the norm amongst people with dead teeth in their mouths.

Many heart problems and nervous disorders are associated with dead teeth.

Multiple Sclerosis has also been linked to the toxins and organisms from dead teeth.[37],[38]

The location of the tooth, the types of organisms inside it and the nature of the persons genetic make up will determine the areas of disease found clinically. The one thing that is certain is that if you are sick you should look very carefully at all non-vital teeth, whether root therapied or not.

Treatment

Dentistry is the only medical/paramedical profession that consider it O.K. to leave dead infected tissue in the body. (Not only is it OK but it is condoned and paid for by the health funds.) No medical practitioner would consider leaving gangrenous tissue in the body.

Unfortunately there are no good alternatives for this situation. The only treatment for dead tissue in the body is to remove it. Therefore the treatment of choice is to extract a dead tooth rather than root fill it. It is also important to remove any infected tissue from around the tooth. This usually requires a very easy surgical approach to access the end of the socket. Although this does not sound attractive, the results usually are, and the actual surgery is usually very easy.

As dentists we are taught to extract teeth with forceps and that any infected tissue left in the bony socket will be dealt with by the cells of the immune system. This does sometimes happen. Often, though, the bone will heal around the infected tissue which remains indefinitely as an infected hole in the bone. These areas are usually colonized by gram negative bacteria.[39] They are called areas of Osteitis or NICO Lesions (Neuralgia Inducing Cavitational Osteonecrosis) NICO lesions[40],[41] can act as Foci of Infection and also Neural Foci just as the Root Therapied teeth can. This is the main reason that a surgical approach is used for most extraction.

The next obvious question is 'How do you fill the space?' The solution depends on the location of the space and the condition of the adjacent teeth and or lack of teeth in the area. It will usually involve the creation of some sort of bridge or partial denture. Each person must be assessed individually.

I do not believe that Titanium implants are a suitable solution. The electric currents generated by these devices may also act as a neural interference field.

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