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DETOXIFICATION

Sam Ziff

Dorland's Medical Dictionary defines detoxification as: "1. reduction of the toxic properties of poisons. 2.treatment designed to free an addict from his drug habit. Metabolic detoxification, reduction of the toxic properties of a substance by chemical changes induced in the body, producing a compound which is less poisonous or is more readily eliminated."

There are two other words that should be clarified at the outset, chelate and chemotherapy. Dorland's definition of these words are:

Chelate: "to combine with a metal in complexes in which the metal is part of a ring. By extension, a chemical compound in which a metallic ion is sequestered and firmly bound into a ring within the chelating molecule. Chelates are used in chemotherapeutic treatments for metal poisoning."

Chemotherapy: "the treatment of disease by chemical agents; first applied to use of chemicals that affect the causative organism unfavorably but do not harm the patient."

Nonproprietary agents that might possess the desired characteristics to fit the above three definitions, do not command any great interest within the medical community or allocation of government research funds. As a result, there is a paucity of research data on such substances. Conversely, there is quite a bit of data available on proprietary substances, replete with their potential toxic side effects.

The question of which substances will assist in the detoxification of heavy metals is one that is most frequently asked and discussed by BioProbe subscribers. The same is true in relation to queries from the general public. In an effort to satisfy some of these questions Bio-Probe has done an extensive literature search and review on the subject. We will attempt to fill the information void on non-toxic substances available for use as chelates or detoxicants.

The subject is an extremely complex one and the information we will provide will hopefully clarify some of the complexities, while at the same time providing useable data on the substances that can be used. Time and space limitations restrict the amount of information that can be included in any one issue of Bio-Probe. Consequently, we will be providing the information in installments.

To place the complexities of detoxification in perspective we will concern ourselves with the following major aspects:

1. Elimination or reduction of the source.
2. Mediating the toxic effects. This can encompass the prevention of free radical generation; the scavenging of free radicals; preventing enzymatic interference and maintaining homeostasis of essential nutrients and minerals.
3. Controlling transport, tissue distribution and inducing elimination.
4. Repletion of essential nutrients and minerals.

Although applicable to other metals used in dentistry, at this time we will only address the above aspects in relation to mercury. Elimination of the source is, of course, the ideal situation. This would encompass the replacement of amalgam fillings with composites or gold. It should also take into consideration the elimination or reduction of other available sources of mercury such as dietary, cosmetic, and OTC drug preparations. Efforts should be made to reduce or control the intake of foods or use of products or preparations containing mercury or other substances having the potential for negative interactions. There is also the potential for positive diet modification i.e. the inclusion of sulfur/thiol containing foods in the diet that have the potential of augmenting the molecular binding and elimination of mercury from the body. This philosophy should carry through during the preoperative, operative and postoperative phases of treatment and during the postoperative detoxification phase.

Mediating the toxic effects and other aspects of 2 above requires consideration of the relative time-frame of the dental treatment plan. Elapsed time from patient agreement of the proposed dental plan and the first appointment, in some instances, can extend for weeks. If there is obvious symptomatology reflecting a sensitivity to mercury vapor exposure existant at the time of acceptance of the dental plan then a tailored preoperative nutritional program could potentially serve to ameliorate symptomatology, reduce the potential of free radical pathology through control and scavenging and also assist in rebalancing of essential nutrients. In this context it is important that careful consideration be given to any possible interactions with medication the patient may be on.

Two essentially different philosophies exist in regards to preoperative protocols. One school of thought believes the primary consideration should be the enhancement and strengthening of the patient's overall health/nutritional status. The second school of thought centers around a major emphasis for the maximum reduction of mercury body burden prior to starting any treatment plan.

Protocols related to the philosophy of enhancing the nutritional status of the patient would, in all probability, also increase the elimination of mercury to some degree. The degree of elimination induced is of course a function of quantity, potency and chelating efficacy of any of the nutrients involved.

Applying the maximum body burden reduction philosophy has the potential of also offering some degree of diagnostic capability. Diagnostic chelation protocols can be used with mercury urine assays to assess the increase in "mercury dumping". This could serve as a diagnostic indicator of gross mercury body burden and, perhaps more importantly, urine mercury assays would provide evaluation data on the efficacy of the detox protocol being employed.

Both philosophies or approaches to achieving the desired goals have merit. Hopefully the information we provide in this series will permit the clinician to intelligently tailor his or her approach to best fit the patient's needs.

In this issue we begin the series by providing information about the nutrients and minerals available and some of their biochemical relationships:

CYSTEINE

Cysteine is one of the sulfur-containing amino acids. Its chemical name is 2-amino-3-mercaptopropionic acid. Cysteine is produced by enzymatic or acid hydrolysis of proteins. Cysteine can be oxidized to Cystine (tine) which is rather insoluble in water. It is this characteristic of Cystine that can cause problems. Sometimes it can be found in the urine and in the bladder in a crystal form where it will frequently form cystine calculus (stones) in the kidneys or bladder. Cystine is the main sulfur-containing compound of the protein molecule and upon reduction produces two molecules of cysteine (because of this it is sometimes called dicysteine). Sulfur containing compounds of biochemical importance include insulin, prolactin, growth hormone and vasopressin. Cysteine along with pantothentic acid is a precursor of coenzyme A. Heavy metals catalyze the oxidation of cysteine to cystine and also react with cysteine to form mercaptides.

Cysteine is very soluble in water and therefore can be easily eliminated via the urine. However, cysteine can become oxidized to cystine which can then present the potential of stone problems. Research has demonstrated that if an adequate supply of vitamin C is available, it will help keep the Cysteine in its reduced and soluble form, thereby preventing the formation of stones. The ratio of vitamin C to Cysteine is three to one. Therefore, when supplementing, it is important that you take at least three times as much vitamin C as you take of cysteine.

Jones and Basinger (1982) listed the coordination preferences for L-Cysteine in accordance with the periodic table:

		Co	Ni	Cu	Zn		As
Ru	Rh	Pd	Ag	Cd			Sb
Os	Ir	Pt	Au	Hg		Pb	Bi

The table indicates the metal ion preferences for a cysteine-type structure (which would also include D-Penicillamine). It is also obvious from the chart that L-cysteine would also have the potential of binding the other metals listed.

The ideal chelating agent would only have specificity for the particular toxic metal that you wanted to eliminate from the body. Unfortunately, this appears to be totally impossible because of the biochemical considerations involved. Each of the elements listed above has some biochemical coordination chemistry involving an affinity for sulfhyryls or sulfur containing ligands. The degree may vary but, based on existing knowledge, each would participate to some degree if a sulfhydryl based antidote was being used.

The significance of this phenomenon in any detox protocol relates to repletion considerations for essential ions. Both copper and zinc levels could be reduced concomitantly with the primary objective of reducing mercury body burden. So, from the practitioners standpoint, a

decision has to be made whether it is essential to offset these losses by dietary increases or supplementation. Simple decision? Far from it. For example, there is substantial evidence in the scientific literature indicating most individuals get more copper from exogenous sources than their body requires (bear in mind copper is also toxic). Conversly, there is also scientific evidence indicating that women should have increased intakes of copper, whether dietary or supplemental, to offset losses related to menses. Based on early literature on the subject, a standard of 3 mg was set for inclusion in all multiple mineral supplements. Subsequent research has shown that for a great majority of individuals daily intakes of this magnitude can cause all kinds of problems, especially mental. Most supplement manufacturers now offer products without iron and copper. It is evident therefore, the decision is not a simple one as it relates to copper.

When determining sources of copper, consideration must also be given to the high copper amalgam alloys used for dental fillings. There is scientific research documenting the release of copper ions from these fillings with accumulations noted in the gingival and periodontal tissues.

Although copper is an essential nutritional element, it can also act as a catalyst in lipid peroxidation, under physiologic conditions where the ion is not properly bound. This could initiate or promote a free radical process that leads to atherosclerotic plaques and Coronary Artery Disease.

Denham Harman, M.D., Ph.D. one of the countries leading research scientists on aging has proposed that the aging process might be accelerated by copper acting as a catalyst for the production of free radicals.

There is also scientific research indicating: 1) Increasing blood levels of copper with age in humans; 2) In mice, brain levels of copper increase with age and 3) Elevated blood copper levels in humans have been found in many disease states: leukemia, Hodgkins disease, rheumatoid arthritis, myocardial infarction, atherosclerosis, arteriosclerosis and psoriasis.

Zinc is not as controversial. There is a general population deficit of daily zinc intake from dietary sources. Zinc and many zinc functions are inhibited by mercury. Failure to replete zinc during any detox protocol would exacerbate the deficit. Zinc will be covered in-depth in the next issue of Bio-Probe.

Tandon et al (1983) stated: "Safety of chelating agents is more important than their efficacy to remove harmful metals from the body. Depending on their affinity towards metals they are likely to cause imbalance of essential metals by driving them out of the body, mobilizing them from one tissue to another or modifying their uptake from the diet."

SOME OF THE LITERATURE DEMONSTRATING Hg AFFINITY FOR CYSTEINE:

Stricks and Kolthoff (1953) studied the reactions between mercuric mercury and cysteine and demonstrated that mercury can form at least three compounds with cysteine in which all or a part of the mercury is bound firmly as a mercaptide

Cantoni et al. in their (1984) study indicated that previous research had shown HgCl2 toxicity to be inversely proportional to the extracellular concentration of metal chelating amino acids such as cysteine.

Mercury compounds are formed by the binding of mercury to the biological binders, albumin or cysteine. (Margel and Hirsh 1982).

Cellular nonprotein thiols (NPSH) consist of glutathione (GSH) and other low molecular weight species such as cysteine, cysteamine, and coenzyme A. (Biaglow et al., 1983)

These results suggest that NPSH play an important role in Hg uptake and subsequent development of Hg toxicity. (Johnson D.R., 1982)

Chao and Frenkel (1983) utilized a model system for experiments to measure the ability of various mercury compounds to compete with a nonmercurous sulfhydryl reagent in binding to cysteine. The results showed that even compounds such as phenylmercury and the inorganic mercurials, which are unable to stimulate RNA synthesis, are able to bind to a sulfhydryl group.

NOTE: THERE ARE LITERALLY HUNDREDS OF PAPERS DEMONSTRATING THAT CYSTEINE PARTICIPATES IN AN EXTREMELY COMPLEX SERIES OF METABOLIC REACTIONS AND IS INCORPORATED INTO MOST PROTEINS AND GLUTATHIONE. AT SOME FUTURE DATE WE WILL DISCUSS SOME OF THESE AS POTENTIAL BIOCHEMICAL PATHWAYS FOR MERCURY. HOWEVER, THAT IS BEYOND THE SCOPE OR INTENT OF THIS SERIES OF ARTICLES.

GLUTATHIONE

Glutathione is a sulfur containing tripeptide composed of glutamic acid, cysteine, and glycine. Glutathione is present in almost all cells in rather high concentrations. Glutathione serves as a storage and transport form of cysteine and also as a respiratory carrier of oxygen. It also functions as an intracellular reductant and has an important role in catalysis and metabolism and protects cells against free radicals and the destructive effects of hydrogen peroxide. In red blood cells the iron of hemoglobin is normally in the ferrous [Fe(II)] form, but is readily oxidized to the ferric form by hydrogen peroxide to yield methemoglobin, which is inactive in carrying oxygen.

Although, as with copper, we are not talking about the ability of cysteine or glutathione as a chelating agent to remove iron from the body, it is of similar importance in relation to multiple mineral or antioxidant supplements that contain iron. It also extremely important when viewed in context with mercury's binding affinity for glutathione, which ultimately has the effect of reducing the amount available to function in its protective role against hydrogen peroxide. This in turn could then have metabolic consequences on the amount of ferrous iron oxidized to the ferric form.

To understand the significance of this more thoroughly lets digress for a moment to talk about iron. Advanced Medical Nutrition Inc (AMNI) in their newsletters of Nov 1983 and Mar 1984 gave an excellent analysis of the problem of iron excess: "Once iron is absorbed, there is no simple mechanism for its excretion. The body guards its iron stores very carefully and reuses any that is broken down in the body over and over again. Only the small amounts lost in the urine, sweat, sloughing of skin and by menstruation need to be replaced.....Free iron promotes the formation of the highly toxic

hydroxyl radical from the superoxide anion radical and hydrogen peroxide, thereby stimulating lipid peroxidation.....Further, free iron may directly promote inflammation.. Synovial fluid from rheumatoid patients contains non-protein bound iron salts that promote lipid peroxidation. Use of antioxidants and iron chelating agents may have important anti-inflammatory properties that are clinically applicable in the treatment of rheumatoid disease and other disorders. A substantial amount of evidence thus exists that various tissue disorders are the result of iron becoming decomparmentalized. Decompartmentalization occurs due to cellular damage caused by the action of certain metal ions or hormones, enzyme induced hydrolysis, the action of drugs, toxic chemicals, or viruses, as well as by numerous processes related to ill health or aging. When excessive decompartmentalization occurs and the body's protective mechanisms are overloaded or weakened by ill health, damaging oxidative free radical reactions may take place."

The subject of iron bioavailability would not be complete without bringing out the fact that almost all detoxification protocols include vitamin C which dramatically enhances the absorption of iron from the gut.

In the liver, glutathione acts a reservoir of cysteine which is utilized whenever necessary for protein synthesis. The cysteine component of glutathione is protected from conversion to the insoluble form cystine. Because of this quality, there is some research indicating glutathione to be preferable to cysteine when supplementing in a detoxification protocol. The rationale being to eliminate the potential problems related to cysteine oxidation. However, as long as vitamin C is taken concurrently with cysteine to prevent its oxidation to cystine, a more favorable protocol would be the combination of both cysteine (which has great specificity for the binding of mercury) and glutathione, thereby providing supplementation to augment many metabolic functions. Glutathione can also replace cysteine derived from methionine (another sulfur amino acid) thus exerting a methionine sparing action, which would also tend to modify any need to provide supplemental methionine.

Perhaps a more important reason to supplement glutathione in addition to cysteine is its biological role in the antioxidant recycling process. Both vitamin C and E require the presence of an adequate supply of reduced glutathione for regeneration from their oxidized and inactivated state after participating in the free radical scavenger process. In addition to insuring the clinical effectiveness of vitamin C and E, glutathione plays a double role in the effectiveness of selenium. Selenium is an essential component of several enzymes, particularly glutathione peroxidase which is an antioxidant enzyme that protects unsaturated phospholipids and cholesterol in cell membranes from free radical attack. Research has demonstrated that oxidized cholesterol can lead to abnormal calcium buildup within cells and connective tissue.

GSH is the abbreviation for reduced glutathione which is also a tripeptide and is present in red blood cells. It is functionally associated with glucose-6-phosphate dehydrogenase and reduced nicotinamide-adenine dinucleotide phosphate (NADP) in the maintenance of red cell integrity.

SOME OF THE LITERATURE RELATING MERCURY TO GLUTATHIONE:

Rabenstein and Isab (1982) utilizing intact human erythrocytes found that HgCl2 when added to the suspension would cross the membrane and reach an equilibrium distribution among the molecules of the erythrocyte within 4 minutes. In the intracellular region Hg(II) reacts with GSH and hemoglobin to form the ternary mixed-ligand complex GSH-Hg(II)-hemoglobin. The lifetime of the GSH in the GSH-Hg(II)-hemoglobin complex is shown to be less than 30 seconds, which provides direct evidence for the first time that Hg(II) complexes in biological systems are quite labile.

Ballatori and Clarkson (1984) investigated the interrelation between biliary transport of glutathione (GSH) and inorganic mercury. They found that mercury secretion into bile was closely related to the rate of GSH secretion and that sex differences and individual variability in the biliary secretion of inorganic mercury was correlated with differing abilities to secrete GSH into bile.

In a paper by Weed et al. (1962), the erythrocytes were found to bind Hg(II) in amounts up to 7-times the glutathione (GSH) concentration of the cells before binding by GSH was detected.

MacGregor and Clarkson (1974) utilizing intact human erythrocytes found that Zinc(II), methylmercury(II) and trimethyllead(IV) were complexed by GSH and hemoglobin.

VITAMIN C

Some of the physiological functions of vitamin C are: absorption of iron; maintenance of adrenal cortex; major role as an antioxidant; wound healing; formation of cartilage, dentine, bone and teeth; metabolism of tryptophan, phenylalanine, and tyrosine; synthesis of polysacchardies and collagen; maintenance of capillaries and growth.

Some of the target tissues of vitamin C are the adrenal cortex, pituitary, ovary, connective tissue, bone, liver, teeth and gums.

Other functions or actions of vitamin C are: detoxification through maintenance of the peroxidase system; stimulation of phagocytosis; acts as an antimitotic agent; involved in cellular respiration through reductions and oxidation and organ respiration by maintaining oxygen turnover; chelating agent for heavy metals; prevents formation of nitrosamines from nitrates; and serves as an antistress and anti-infection factor.

NOTE: VITAMIN C WILL BE COVERED IN-DEPTH IN THE NEXT ISSUE.

VITAMIN E

Vitamin E is a fat-soluble vitamin. Vitamin A, D, and K also fall within this category or grouping. Fat-soluble or water-soluble relates to the original discovery findings on vitamins. The first vitamin categorized in this manner was called fat-soluble vitamin A because it was found that it was soluble in fat and fat solvents (alcohol and ether). Subsequent discoveries found that certain vitamins were only soluble in water.

Fat-soluble vitamins differ from the water-soluble group in other ways: they can be stored in the body whereas water-soluble cannot; they are excreted chiefly by the fecal pathway versus the urinary pathway for the water-soluble vitamins; and perhaps the most important difference lies in the fact that they are absorbed along with dietary fats and conditions of extremely low fat intake or impaired uptake of fats will also interfere with their absorption. Antibiotics and certain other drugs as well as certain disease states such as malabsorption syndromes decrease the absorption of fat-soluble vitamins from the intestinal tract.

The chemical name for Vitamin E is tocopherol and is derived from Greek tokos (childbirth) and pherin (to bear) and the ending of ol is the chemical suffix to denote an alcohol. The name tocopherol was bestowed on this vitamin in 1938 and relates directly to the original discovery work by Evans and Bishop in 1922 who found that rats given a purified diet containing all the then-known nutrients, could not reproduce. When fresh green leaves or dried alfalfa was added, the rats could again reproduce. The unknown factor was called substance X and in 1924, Sure named it vitamin E or the antisterility vitamin.

The exact physiological function of vitamin E in man is still not comletely understood, although recently some of its functions have been identified. It appears that one of the primary biological functions of vitamin E relates to its role as an antioxidant. Vitamin E's antioxidant ability is enhanced by selenium. This synergistic effect preserves membranes from destruction by oxidation products and especially retards hemolysis of red blood cells.

The mystery of vitamin E continues unabated. There are many hypotheses related to functions other than antioxidant. The real research problem relates to the fact that many of these functions can be demonstrated in animals but reproducibility in humans has been elusive and much more research is required. For example, impairment of membrane function, red cell defects and increased platelet aggregation can be demonstrated in vitamin E-deficient animals. However, there is no solid evidence that alterations of the same nature occur in humans. The same has been demonstrated in relation to the immune system. In rat studies, vitamin E deficient rats had a twofold higher index of lipid peroxidation of polymorphonuclear (PMN) leukocytes than controls as well as decreased chemotaxis and increased oxygen consumption. Other animal studies have clearly demonstrated that iron metabolism tends to be abnormal in vitamin E deficiency states. In humans there has been some evidence that oral supplementation with vitamin E will tend to correct a deficiency in the red cell of glucose-6-phosphate dehyrogenase, in some patients. However, not all patients with this condition respond to vitamin E supplementation.

Another synergistic relationship is evident with vitamin E and vitamin A. Vitamin E protects vitamin A during digestion and absorption and also when both are present in tissues.

The literature shows that vitamin E can reduce the toxic effects of mercury. In laboratory tests , vitamin E was able to reduce the chromosomal breakage caused by mercury.

Fukino et al (1984) demonstrated lipid peroxidation and a decrease in vitamin E content in the rat kidney 12 hours after mercury administration.

Vitamin E has been shown to have sulfhydryl-protective activity. (Seelig M.S., 1982)

Vitamin E was able to inhibit phagocyte-induced mutagenesis. (Weitzman and Stossel, 1982).

Vitamin E was able to protect mouse spleen cells from lipid peroxidation and thereby enhance their primary antibody response and their lipopolysaccharide-stimulated proliferation response. (Hoffeld, 1981).

IN THE NEXT ISSUE OF THE DETOXIFICATION SERIES WE WILL COVER:SELENIUM, ZINC, VITAMIN C, AND VITAMIN B6. IN THE THIRD INSTALLMENT WE WILL COVER: PANTOTHENIC ACID, VITAMIN B1, MAGNESIUM AND VITAMIN A.

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Although letters to the Editor are normally handled under the FORUM section of the Newsletter the content of the following letter received from Alfred V. Zamm, M.D., F.A.C.A., F.A.C.P. is such that it should be considered as an "original article". Dr. Zamm brings in to perspective a complex issue that is of major significance to every practicing dentist and by implication, every physician. Please read it carefully.

On page 520 of the April 1983 issue of the JADA, the American Dental Association declared its position of the mercury issue: "The Association wishes to emphasize that, except in individuals **sensitive** (emphasis mine) to mercury, there is no reason why a patient should seek to have amalgam restorations (silver fillings) removed". I believe this statement is not so innocent and simple as it might appear.

Note: the choice of the word **"sensitive"** is critical to understanding how obfuscating this statement is and how confusion has arisen among dentists in discussing (and understanding) the mercury issue.

Although numerous definitions of the following terms have been used, the following definitions of these terms would be generally accepted by most professionals:

"Allergic": This term is most often used to denote either immediate or delayed hypersensitivity, i.e., Type I and Type IV respectively, Gell and Coombs classification. An example of immediate hypersensitivity (Type I) is hay fever. An example of delayed hypersensitivity (Type IV) is poison ivy dermatitis.

"Hypersensitive": This term is broader than "allergic" and is generally used to refer to Types I, II, III, and IV reactions (Gell and Coombs classification) and may also relate to other reactions whose mechanisms are not yet fully understood.

"Sensitive": This is the broadest term of all and can be used to denote a variety of reactions: allergic, immunological, biochemical, and biologic. This is a general term and does not exclusively refer to any particular process.

One can readily see that the choice of the word **"sensitive"** is obfuscating and misleading, since it might lead those not conversant with the "in-group jargon" of allergists and immunologists to the **erroneous** conclusion that sensitivity = hypersensitivity = allergy. This non-statement by the ADA does **not** say that **immunological reactivity** or **allergic intolerance** are the **only** criteria for mercury removal. It specifically says "sensitivity" - **a much broader and more inclusive general term.**

Taken at its face value, the ADA's position that only those individuals **sensitive** to mercury should have their mercury fillings removed is **in agreement with and is indeed, concentric with that of most dentists who advocate mercury removal.** The reasoning goes as follows:

Mercury is a known biological poison. **Everyone** is **"sensitive"** to a biological poison to a greater or lesser degree. Hence, **everyone** is a candidate for having their mercury fillings removed.

In order to fully understand the concept of sensitivity as a biological phenomenon, we must understand the concept of LD_{50} (Lethal Dose, 50%). If the LD_{50} dose of mercury, i.e., specific predetermined milligrams of mercury, is given to a group of rats, **50% of them will die of a LD_{50} dose.** This is a measure of toxicity. Is it not reasonable to ask: What about the 49th percentile of rats? Can we assume they were free of adverse effects simply because they did not die? Did they feel ill? I will conjecture they felt a fraction less ill than did their compatriots who died. I would further conjecture that the 48th percentile felt a little less sick than the 49th percentile, but yet sick, and so forth down to the first percentile, who probably felt the least sick of the group - **but still sick.** Was not the first percentile rat sensitive to mercury? Death did not ensue, yet sickness would ensue. How about the 0.1 percentile? or the 0.01 percentile? **Should we view patients on the basis of a binary function: death or not death? or on an analog function of the degree of wellness (or the degree of illness)?**

As you can see, it all boils down to the frequency distribution curve and the amount of mercury being presented to an individual patient and where on the frequency distribution curve this patient statistically lies in terms of his resistance to mercury poisoning.

In my experience, those people who exhibit sensitivities to chemicals (vapors of petrochemical origin, etc.) or allergies to ingestants and inhalants will generally display beneficial clinical improvements from mercury removal, depending on how sick they are from these intolerances. **The more symptoms, allergic and otherwise, the patient displays, i.e., the sicker they are, the more likely they are to have a discernable benefit from the removal of their mercury fillings.** This should not be construed to say that they are the only ones

that should remove their mercury fillings. No one should be exposed to poison, and even an ostensibly totally healthy person would be better off without poison in his mouth - if he wants to remain healthy.

I believe that, in stating their position in this document, the ADA carefully chose the term **"sensitive"** so that when, at some time in the near future, the problem of mercury toxicity from dental amalgam fillings will be more widely accepted, someone at the ADA can say "but we said it all along; we were in agreement; we said individuals who are **sensitive** to mercury should remove their mercury fillings; there it is in writing."

What is the future for the individual dental practitioner who is not now attuned to the subtleties of this carefully contrived sophism? Where will he stand in terms of his patients who will come to him saying, "the ADA said that **sensitive** patients should have the mercury fillings removed; I am a mercury-sensitive person (at least to some extent); why did you put poisonous fillings in my mouth **when in 1983 it was clearly stated by the ADA that you should not have done this?"** What will be the position of the dental practitioner? **He will be left "holding the bag"**! It is only during this present golden period in which the issues are still being clarified that the practicing dentist has an opportunity to cease providing mercury fillings for his patients and make some other choice. Once the issue becomes standardized and mercury is declared for what it has always been: a poison, **at that point the individual practitioner will suddenly find that he was left holding the bag** - since it is up to the individual practitioner (not the ADA or the government) to make these decisions for the patient.

In summary: "sensitivity to mercury" does not equal hypersensitivity does not equal allergy. **Sensitivity is a term with broader significance and I believe was carefully chosen by the ADA for that reason.**

A more recent statement by the ADA that removal of mercury is suggested if **hypersensitivity** (allergy) is a factor **did not negate the previous broad statement that "sensitivity" is a criterion**, as hypersensitivity was not stated as the **only** exclusive factor to be considered.

In a number of places in Bio-Probe the eroneous term of "hypersensitivity" crept into the text. Hypersensitive responsiveness was not the issue. The word "sensitivity" should be substituted for hypersensitivity in the following places:

Bio-Probe Vol II #3 May 1985, page 2
Bio_Probe Vol II #6 Sep 1985, page 2,3,4
Bio-Probe Vol II #7 Nov 1985, page 12

Signed Alfred V. Zamm, M.D.

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COUNTERATTACK

The recent license suspension of a mercury-free dentist in Utah (see ADA News Nov 4, 1985) clearly demonstrates the desperation and total frustration of the pro-amalgam forces. Unable to scientifically defend the biocompatibility of mercury amalgam and its continued use as a dental filling material, pro-amalgam forces are resorting in desperation to their last remaining weapon "political power and economic blackmail" to crush and silence the voices of opposition. This "McCarthyism mentality" has precipitated a rash of unjustifiable attacks on mercury-free dentists across the continent.

This irrational behavior and apparent inability of the pro-amalgam forces to accomodate to the constantly increasing pressure from the professional and scientific communities, the media, and the public has inadvertently presented the mercury-free advocates with a golden opportunity. I say inadvertant because I am sure that the scenario as conceived, did not consider that their individual attacks might engender a collective response. Which is exactly what I am advocating. The future of dentistry, health of the public, and preservation of the principle of "freedom of choice" dictates that now is the time to collectively STAND UP AND FIGHT.

There is NO REASON OR RATIONALE, ethically, morally, legally or scientifically to succomb to this "power" attack, real or implied! There is an irrefutable scientific rationale for acting in the best interests of our patients:

Documented scientific research has firmly established the release of mercury from amalgam fillings throughout the lifetime of the fillings, as well as confirming the contribution of that mercury to the body burden of patients. Moreover, the pathological damage from and the signs and symptoms of mercury exposure are well documented. Although OSHA, NIOSH, the EPA and others have SUGGESTED Threshold Limit Values for mercury exposure, investigation of the documents of these agencies reveals that the opinions of these agencies not only differ, but are based only on the prevention of a limited number of clinically observable Signs and Symptoms.

These agencies, as well as the world experts on mercury, publicly acknowledge that the minimum toxic dose of mercury for humans is NOT known! Moreover, NEVER in 160 years of use have controlled scientific studies demonstrating the harmlessness of dental mercury amalgam fillings in patients been provided.

The continued use, by mercury amalgam advocates, of the argument that 160 years of use in patients is the best evidence of its harmlessness is totally without merit. It is a specious argument, because in 160 years, with the exception of a few "heretics", dentists and physicians NEVER LOOKED FOR a relationship between mercury amalgam fillings and clinical symptomatology. More importantly, and perhaps a greater indictment of the spurious nature of the argument is that in 160 years of use the institutions with the assigned and/or moral obligation to do so, have not provided dentists or physicians with any guidance, evidence, or correlative symptomatology upon which to base even the most rudimentary evaluation. If you want proof of those refutations, merely ask any clinician two questions: 1) Is he aware

that mercury vapor is released from "silver dental fillings" in sufficient quantities to produce overt symptomatology in some individuals? and 2) Does he have any idea of what the signs and symptoms are for chronic mercury vapor exposure?

The scientific and clinical evidence against use of dental mercury amalgam continues to grow. In scientific terms I would have to say that there is a possitive correlation between this phenomenon and the concomitant rapid deterioration of the "validity" of the empiracle ANECDOTAL defense theory exploited for so many years by the advocates of dental mercury amalgam.

WHAT CAN WE DO LEGALLY?

The same Nov 4, 1985 issue of the ADA News contained another article that I consider much more important than their front page exploitation of a license revocation (without grounds). The article related the disposition of a case by the Supreme Court of Sweden and was headlined "Swedish court backs mercury amalgam use, rejects bid for appeal". According to the article, "The suit posed two questions of law to the court: could mercury from amalgam be supported as the cause of the stated health problems and did the government have the right to forbid dentists to use mercury amalgam. The court held that since it had not been proved that amalgam caused the stated damage and illness, the government had no reason to forbid the use of amalgam. But even had the opposite been proved, the court held, IT WAS THE INDIVIDUAL DENTIST - NOT THE GOVERNMENT - WHO IS RESPONSIBLE FOR THE MATERIALS USED." (emphasis mine).

In the United States, the American Dental Association has, in writing, admitted it is merely a trade organization and has no legal authority to dictate the use of filling materials. Moreover, no U.S. Governmental Agency has approved of or forbidden the use of any dental materials. This leaves the liability for the selection and use of any dental material directly on the shoulders of the individual dentist. A position in accordance with the precedent set by the Swedish Supreme Court.

This presents an interesting paradox: The vast and ever increasing volume of scientifc and clinical evidence provides legal security for the actions of the mercury-free dentist. Conversely, the same scientific evidence insures that the dentist who continues placing amalgams today, is legally at risk. Even the ADA has publicly printed that "dentists should become familiar with the signs and symptoms of metal exposure". In this context, you must also consider the insidious nature of evidence presented by Dr. Zamm in the preceding article.

So far, the attack on mercury-free dentists has been predicated on two issues: "Practicing medicine without a license" and "the use of certain proprietary instruments and materials". Prevention is the best and most effective protection against such allegations:

1. Never claim that amalgam removal will "cure" systemic ailments in any individual. Amalgam replacement is the removal of a poison from the patients mouth and the elimination of a chronic cause of vagrant electrical currents. Inform the patient of the documented facts and the pathologic potential of mercury.

Prosecution of dentists for "practicing medicine without a license" is as specious as the arguments used to defend continued use of amalgam. Dentists have a legal responsibility to practice "dental medicine" which includes the evaluation and diagnosis of oral symptomatology and pathology, whether local or systemic. This aspect also provides a basis for amalgam replacement, i.e., the pathological effect of mercury and dental amalgam on periodontal structures is well documented. It is also a diagnosis suffering from "oversight"

2. Use Informed Consent Forms routinely for all operative procedures.

3. Use instruments and materials that have been registered in "research protocols" for that purpose and not as diagnostic tools.

4. If advised that you are under investigation or you are being challenged because of practice protocols or applications, regardless of the source, engage the servcices of a competant and informed attorney. Just as important, don't take the attitude that you are out there alone "slowly twisting in the breeze" We are all in this fight together and if we don't know you are being attacked, there is nothing we can do to help.

In selecting an attorney, you should be aware that two different philosophies exist within the legal profession regarding these types of cases. Simply put, defensive or offensive. Under the defensive approach, counsel will attempt to prepare a suitable defense to be presented at the actual hearing. Under the offensive approach, counsel will want to take aggressive action against your antagonists as soon as possible after the first indication of potential jeopardy. In all cases this will be prior to any hearing and could include preemptive suit under "anti-trust", "civil rights" or any other legal framework considered applicable and appropriate.

If you presently have an attorney but he doesn't subscribe to the course of action you want to take, then get another attorney who does.

Regardless of your choice of attorneys, there are some basic procedures you should follow:

1. Do not accept telephone notifications of some action in progress. Demand written notification. Do not make any statements pro or con over the telephone even if you think the caller is your friend.

2. Do not assume that the implications of the action are not serious or that it is merely an informal discussion to obtain information. View it as the action it is - "A direct attack on your professional credibility".

3. If you want help in obtaining scientific documentation, let us know what your needs are. Bio-Probe now has on file approximately 1400 scientific articles and books dealing with the potential toxicity of mercury and its affect on the various systems of the body.

4. If you want to discuss your legal position with someone who has been vigorously investigating the mercury issue for the last three years, we suggest you contact Robert E. Reeves. Mr. Reeves is based in Lexington, Kentucky and can be reached at (606) 259-1827. Be prepared to be billed a nominal fee for your telephone consultation. Like you, Bob is in business to make a living. Up till now he has not charged for these consultations. However, it recently dawned on him that he was spending upwards of two hours a day on the phone providing free legal consultations to people around the country and that this was

having an impact on his case load. Another aspect of this situation is that Bob cannot represent you unless you live in Kentucky but he can work with the attorney of your choice in your area on a consulting basis. He may also be aware of attorneys in your area who are familiar with the mercury issue and these types of cases.

5. Regardless of whether you attack before a hearing or attack during or after the hearing, the byword is attack. Lets all make it abundantly clear to everyone concerned that the days of "whipping in public" for those, who for the health of their patients elect to practice alternative dentistry, are over. Remember, officially, there are two acceptible modes of practicing dentistry and the choice is up to the individual dentist to make.

6. Once you have selected legal counsel, do not make any appearances before investigative personnel or boards without counsel being present.

7. Seriously consider using the provisions of the "freedom of information act" to obtain information concerning your case. Also consider suing for damages to recover expenses and lost time from your practice to defend yourself.

8. Lastly, if you need financial assistance to defend yourself, I can't promise we can help, but we can sure solicit help in your behalf. If we all pull together during these crucial times there is no question in my mind about a victorious outcome.

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EVENTS

I.A.O.M.T. BOARD MEETING.

The next Board Meeting of the International Academy of Oral Medicine and Toxicology will be held at Caesers Palace Hotel, Las Vegas, Nevada, March 1-2, 1986. The Board meetings are open to all of the membership who wish to attend and an invitation is also extended to anyone seriously considering joining the Academy.

INTERNATIONAL ASSOCIATION OF E.A.V. SEMINAR.

The Hawaiian Chapter of the International Association of E.A.V. is presenting Dr. Joachim Thomsen, D.D.S. in a five day seminar to be held at the Four Queens Hotel, Las Vegas, Nevada, March 15-19, 1986. Anyone desiring more information please write to the International Association of E.A.V. Hawaii Chapter, Suite 721, 1441 Kapiolani Blvd., Honolulu, HI 96814.

AMERICAN ACADEMY OF BIOLOGICAL DENTISTRY SEMINAR.

A 4 day program title "Mouth Acupuncture and Quantifying Amalgam Generated Energies and Its Biological Effects" will be given at the Carmel Mission Inn, Carmel, California, April 25-28, 1986. Dr's. Kramer and Gleditsch of West Germany will be the presentors. For more information contact the American Academy of Biological Dentistry, P.O. Box 856, Carmel Valley, CA 93924.

GREATER LONG ISLAND DENTAL MEETING DEBATE.

A classic debate on the mercury amalgam controversy is in the making. The anti-amalgam side will be taken by Murray J. Vimy, D.M.D. and Michael F. Ziff, D.D.S. Opposing on the pro-amalgam side will be Robert S.Baratz, D.D.S., Nelson W. Rupp, D.D.S. and Mark S. Wolf, DDS. April 2, 1986 at Islip, New York.