AMALGAM RESTORATIONS
AND
MERCURY TOXICITY

Peter Oliver Sheridan

BDS (Sydney)

A treatise submitted in partial requirement
for the degree of

MASTER OF DENTAL SURGERY

Department of Preventive Dentistry
Faculty of Dentistry
University of Sydney
1991
In the 'Chicago Medical Journal' for July 1875, is an article by Dr. Payne, in which he gravely speaks of the 'poisoning of thousands of people all over the world from corrosive sublimate generated in the mouth from amalgam plugs in the teeth.' And he says that neither Asiatic cholera, nor smallpox, nor any malarious disease is doing half the mischief in the world that is done by this poisoning.


"The idea of mercurial poisoning of the system by amalgam plugs; properly prepared and inserted, is a fallacy. The failures so usual in amalgam fillings are due, not so much to any inherent fault of the material itself, as to lack of knowledge of its properties, improper proportions of the ingredients used, and improper manipulations."

SUMMARY

The safety of amalgam restorations has been challenged, claims having been made that health risks are associated with the constituent mercury. There are assertions that mercury released from amalgam produces mercury poisoning, and is thus responsible for diverse symptoms of impaired health as well as disease states such as Multiple Sclerosis. This study examines the various forms of mercury and their effects and attempts particularly to delineate the significance of dental amalgam as a factor in hypersensitivity reactions and in the human body burden of mercury. Dental personnel are evaluated as a potentially high-risk group for mercury exposure. Dental amalgam and alternative restorative materials are considered, the removal of amalgam being evaluated as a therapeutic modality. The "anti-amalgam" perspective is scrutinised and the validity of the claims assessed. A review of the scientific literature, and the statements of national and international dental and scientific organisations reflect the general support for the safety of dental amalgam. There is no evidence that health risks are associated with the use of dental amalgam other than rare local allergic reactions and oral lichenoid lesions. There are no general health benefits which justify the removal of amalgams. Notwithstanding the usefulness and safety of dental amalgam certain recommendations and conclusions are made in respect of future approaches to the utilisation of this material and for mercury in general. Further objective scientific research is necessary to determine the effects on human health of chronic exposure to low levels of mercury. There is the need for accurate general population threshold levels to be established for mercury vapour with special consideration for the vulnerable members of the community. The health professions have a significant role to play in providing informed opinion and advice for their patients and the public, in countering the more eccentric claims of the anti-amalgamists and assuaging the anxiety and confusion which accompanies this subject.
# TABLE OF CONTENTS

Summary i
Table of Contents ii
List of Tables vii

1. INTRODUCTION 1
2. MERCURY 5
   2.1 CONVERSION FACTORS
   2.2 USES OF MERCURY
   2.3 MERCURY POLLUTION
   2.4 NATURAL SOURCES OF MERCURY

3. TYPES OF MERCURY 9
   3.1 CLASSIFICATION OF MERCURY STATES
   3.2 TOXICITY SCALE
   3.3 ELEMENTAL (METALLIC) MERCURY
      3.3.1 Liquid Elemental Mercury
      3.3.2 Elemental Mercury Vapour
   3.4 INORGANIC MERCURY COMPOUNDS
   3.5 ORGANIC MERCURY COMPOUNDS
      3.5.1 Aryl Mercurials
      3.5.2 Alkyl Mercurials

4. KINETICS AND METABOLISM 16
   4.1 BIOTRANSFORMATION
      4.1.1 Oxidation of Elemental (Metallic) Mercury Vapour to Divalent Ionic Mercury
      4.1.2 Reduction of Divalent Mercury to Elemental (Metallic) Mercury
      4.1.3 Methylation of Inorganic Mercury
      4.1.4 Conversion of Methylmercury to Divalent Inorganic Mercury
   4.2 HALF-LIFE OF MERCURY
   4.3 ABSORPTION
      4.3.1 Inhalation
      4.3.2 Ingestion
      4.3.3 Skin
      4.3.4 Axonal Transport
   4.4 ELIMINATION
   4.5 NEUROTOXICITY OF MERCURY
5. CLINICAL EFFECTS
   5.1 INORGANIC MERCURY
      5.1.1 Acute effects.
      5.1.2 Chronic Effects
   5.2 ORGANIC MERCURY
      5.2.1 Progression of Methylmercury Poisoning
   5.3 NEUROPSYCHOLOGICAL EFFECTS
   5.4 TREATMENT FOR MERCURY POISONING
   5.5 SELENIUM

6. INTAKE AND THE HUMAN BURDEN OF MERCURY
   6.1 MERCURY LEVELS IN FOODS
      6.1.1 Non-Fish Foods
      6.1.2 Fish Foods
   6.2 INTAKE
      6.2.1 Oral
      6.2.2 Inhalation
   6.3 TOTAL BODY BURDEN
   6.4 MAXIMAL ACCEPTED MERCURY INTAKE

7. NORMAL VALUES AND TOXIC DOSES
   7.1 INDICATOR MEDIA
      7.1.1 Blood
      7.1.2 Urine
      7.1.3 Hair
   7.2 TOXIC LEVELS OF MERCURY VAPOUR
   7.3 TOXIC LEVELS OF METHYLMERCURY
   7.4 REPRODUCTION, FOETUS AND EARLY CHILDHOOD

8. EXPOSURE LIMITS FOR MERCURY
   8.1 OCCUPATIONAL LIMITS
      8.1.1 U.S.A.
      8.1.2 United Kingdom
      8.1.3 World Health Organisation
      8.1.4 Australia
      8.1.5 Finland, Sweden and the Soviet Union
      8.1.6 Other Occupational Recommendations
      8.1.7 Secondary Effects of Occupational Exposure
   8.2 GENERAL POPULATION LIMITS
   8.3 MERCURY ABSORPTION
9. DENTAL AMALGAM
  9.1 HISTORY
  9.2 UTILISATION
  9.3 COMPOSITION
    9.3.1 Conventional Alloys
    9.3.2 Copper Enriched Alloys
  9.4 MANIPULATION
  9.5 SETTING REACTIONS AND STRUCTURE
    9.5.1 Conventional Alloys
    9.5.2 Copper Enriched Alloys
  9.6 TARNISH AND CORROSION
    9.6.1 Tarnish
    9.6.2 Corrosion
  9.7 MERCURY HYGIENE

10. ALLERGY TO AMALGAM AND MERCURY
  10.1 TESTING FOR ALLERGIC REACTIONS
  10.2 CLINICAL EFFECTS
  10.3 TREATMENT
  10.4 IMMUNE RESPONSES
  10.5 ORAL LICHENOID LESIONS
  10.6 RESPONSES TO AMALGAM AND DENTAL MATERIALS

11. DENTAL AMALGAM AND MERCURY LOSS
  11.1 INSERTION AND REMOVAL OF AMALGAM
  11.2 MERCURY VAPOUR AND RESTORATIONS IN SITU
    11.2.1 Mercury vapour from amalgam
    11.2.2 Blood and urine mercury from amalgam restorations
    11.2.3 Animal Studies
  11.3 CORROSION AND MERCURY RELEASE
  11.4 LOSS OF MERCURY FROM AMALGAMS AND DAILY DOSE
  11.5 MERCURY VAPOUR FROM CREMATION
  11.6 RELATIONSHIP OF ORGANIC MERCURY TO AMALGAM RESTORATIONS
  11.7 SIGNIFICANCE OF MERCURY FROM AMALGAM
12. MERCURY LEVELS FOR DENTISTS AND DENTAL PERSONNEL

12.1 MERCURY VAPOUR LEVELS
12.2 URINE MERCURY LEVELS
12.3 BLOOD MERCURY LEVELS
12.4 HAIR MERCURY LEVELS
12.5 BRAIN MERCURY LEVELS
12.6 PREGNANCY AND REPRODUCTION
12.7 SYMPTOMS OF MERCURY POISONING
12.8 HYPERSENSITIVITY REACTIONS
12.9 LONGEVITY
12.10 DENTAL PERSONNEL AS A HIGH RISK GROUP FOR MERCURY EXPOSURE

13. EVIDENCE FOR CLAIMS OF MERCURY TOXICITY FROM AMALGAM RESTORATIONS

13.1 STUDIES EXAMINING EVIDENCE OF ILL HEALTH RELATED TO AMALGAM
13.2 ANTI-AMALGAM LITERATURE
13.3 THE MEDIA
13.4 CHRONIC FATIGUE SYNDROME AND CANDIDIASIS
13.5 METHODS OF ANALYSIS
   13.5.1 Symptom Questionnaire
   13.5.2 Electrical Reading of Restorations and Sequential Removal of Amalgam
   13.5.3 Skin Patch Testing
   13.5.4 Urine Mercury Analysis
   13.5.5 Blood Tests
   13.5.6 Hair Analysis
   13.5.7 Oral Mercury Vapour Tests
   13.5.8 Food and Chemical Allergy Tests
13.6 ELECTROGALVANIC EFFECT AND ORAL GALVANISM

14. HOW SAFE IS AMALGAM?

14.1 REVIEWS OF THE LITERATURE
14.2 NATIONAL AND INTERNATIONAL RECOMMENDATIONS
   14.2.1 The Lek Inquiry (Sweden)
14.3. THE VIEWS OF DENTISTS
15. REMOVAL OF AMALGAM  
15.1 REMOVAL OF AMALGAM RESTORATIONS  
15.2 SUPPLEMENTAL THERAPIES AND DETOXIFICATION  
   15.2.1 Lifestyle changes  
   15.2.2 Replacement Protocols  
   15.2.3 Detoxification with Drugs  

16. AMALGAM AND ALTERNATIVE DENTAL MATERIALS  
16.1 AMALGAM  
16.2 TOOTH-COLOURED MATERIALS  
   16.2.1 Synthetic Resins  
   16.2.2 Porcelain and ceramic materials  
16.3 NON-PRECIOUS ALLOYS  
16.4 GOLD  

17. MERCURY AS A FACTOR IN MULTIPLE SCLEROSIS  
17.1 SYMPTOMS OF MULTIPLE SCLEROSIS  
17.2 MERCURY, AMALGAM RESTORATIONS AND MULTIPLE SCLEROSIS  

18. DISCUSSION  
19. RECOMMENDATIONS AND CONCLUSIONS  
20. REFERENCES
Table 1. Progression of Methylmercury poisoning
   Source: Environmental Mercury Contamination (1972)

Table 2. Daily Dose of Mercury 41
Table 3. Blood Levels of Mercury 46
Table 4. Urine Levels of Mercury 47
Table 5. Hair Levels of Mercury 49
Table 6. Occupational and general population exposure limits for mercury vapour 61
Table 7. Uptake of mercury and contribution to body burden
   Source: WHO (1988) 96
Table 8. Daily dose of mercury vapour from dental amalgam 105
Table 9. Urine levels for dental personnel 117
Table 10. Blood levels of mercury for dental personnel 119
Table 11. Daily intake and retention for mercury in the general population
   Source: WHO (1990,1991) 177
Table 12. Daily uptake of mercury
   Source: Clarkson, Friberg, Hursh & Nylander (1988) 179
1. INTRODUCTION

Claims have been made that health risks are associated with the use of dental amalgam. The alleged toxic effects of mercury, which is a requisite ingredient in dental amalgam restorations, have been proposed as the basis for symptoms of ill-health (e.g. depression, fatigue, joint pains and headaches), a major factor in the aetiology of numerous conditions (e.g. infertility, birth defects, leukaemia, cancer, cardiovascular disorders) and the cause of chronic ailments (such as Multiple Sclerosis and other diseases of the immune system, asthma, arthritis, Chronic Fatigue Syndrome, candidiasis as well as many other neurologic and emotional disorders (Pinto & Huggins 1976; Huggins 1982; Hanson 1983; Huggins & Huggins c1983; Lohyn 1983; Pleva 1983; Stock & Jaensch 1983; Hanson c1984; Kupsinel 1984; Lohyn 1984; Ziff 1984; Hanson c1985; Lohyn 1986; Graham 1987; Ziff & Ziff 1987; Ziff & Ziff 1988a; Ziff & Ziff 1988b; Geddes 1989; Black 1990; Godfrey 1990; Heinke 1990).

There is concern and confusion in the general as well as the scientific and dental communities surrounding the recurring claims that mercury poisoning occurs through its use in dental amalgam and the recommendations that the removal of amalgam will alleviate symptoms of diverse disease states. There is a pressing need for the question of mercury in amalgam to be addressed within the broader panorama of mercury in all its forms and its biological impact on humanity.

The purpose of this inquiry is to critically and objectively review the role and effects of mercury in humans and, more specifically, to delineate the significance
of dental amalgam as a source of mercury in the body burden of humans. This study further examines whether mercury emanating from amalgam restorations causes mercury poisoning, is responsible for impaired health and is implicated in the aetiology and/or exacerbations of disease states such as Multiple Sclerosis. The value of removal of amalgam restorations as a therapeutic modality will be evaluated.

In examining the general features of mercury and mercury toxicity attention is given to the types of mercury in the environment, human kinetics and metabolism, and the clinical effects of mercury exposure. The sources of intake of mercury are scrutinized as contributors to the body burden of mercury. Normal and toxic doses are surveyed with particular reference to occupational and population threshold values.

In respect of dental amalgam the major dental considerations relate to the amount and significance of mercury liberated during the preparation, insertion and removal of amalgam restorations and additionally through corrosion and as released mercury vapour during the functional life of the restoration. It is only in the last decade that it has been scientifically established and accepted that mercury vapour is actually released from amalgam restorations during function. Thus, the research and referenced material quoted in this study has, in the main, been taken from recent publications.

The rare occurrence of hypersensitivity to mercury from amalgam is contemplated within the general domain of allergic reactions to dental materials. One specific group with routine high exposure to mercury are dentists and their staff. The health status of this group is an important indicator of toxicity from
mercury vapour. Mercury hygiene is an important determinant of mercury levels in surgeries and the protocols which will minimise exposure to dental staff (as well as patients) are discussed.

Through the media and other sources the general public is continually exposed to commentary and statements which not only question the inherent safety of amalgam restorations in dental treatment, but predict that from their placement arise various symptoms and diseases. These persistent assertions of mercury toxicity from amalgam cannot simply be dismissed but must be properly and seriously addressed and this should include assessment of anti-amalgam literature and the role of the media. Given that the proponents of mercury poisoning from amalgam are often scientifically trained and use science to justify both assessment and treatment, the methods of analysis used to determine mercury toxicity from amalgam are appraised.


Removal of amalgam should be considered not only in respect of the questionable therapeutic value thus achieved, but also in relation to the
advantages and disadvantages of the alternative materials being considered. It has also been contended that oral galvanism occurs with the use of dissimilar metals in dentistry and has deleterious health effects. This phenomenon is included for review as it is another element in the assertions made that health risks accrue from mercury in amalgam.

Multiple Sclerosis is the disease most often quoted as being a consequence of mercury toxicity from amalgam and with the associated claim that removal of amalgam will effect a cure. All over the world many thousands of people with Multiple Sclerosis contemplate removal of their amalgam restorations as a potential remedy. It is vital that a proper and reasoned assessment of these claims is available to professionals and professional groups (neurologists, family physicians, dentists, health care workers, Multiple Sclerosis Societies) such that people with Multiple Sclerosis can expect objective advice, guidance and a balanced view.

In a more environmentally enlightened global community there is increasing awareness of pollutants, both natural and iatrogenic. The public is legitimately concerned as to the safety of dental amalgam and the dental profession must ensure that traditional dental treatment using amalgam restorations is furnished in the best interests of the dental patient.

This study attempts to place in perspective the biologic effects of mercury from dental amalgam in the light of current research and knowledge.
2. MERCURY

2.1 CONVERSION FACTORS

\[
\begin{align*}
1 \text{ ppm} &= 1 \text{ mg/kg} = 1 \mu g/g \\
1 \text{ ppb} &= 1 \mu g/kg = 1 \text{ ng/g} \\
1 \mu \text{mol Hg} &= 200.6 \mu g \text{ Hg} \\
1 \text{ nmol/L} &= 2 \mu g/L = 0.02 \mu g/100 \text{ mL} \\
1 \text{ ng/mL} &= 1 \mu g/L \\
1 \mu \text{mol creatinine} &= 113.1 \mu g \text{ creatinine}
\end{align*}
\]

2.2 USES OF MERCURY

In addition to its use in dentistry, mercury is used industrially in more than 60 industries:

- in the production of chlorine, pulp and paper, insecticides, fungicides;
- the manufacture of neon lights, paper, paint, jewellery, cosmetics, laboratory and medical instruments;
- in electroplating and photographic industries;
- in the extraction of gold.

Consumption of mercury in dentistry is some 3% of its total use in industrialized countries.
In previous centuries mercurial therapy was a recognized form of treatment for virtually all diseases including syphilis, gout, dropsy, cancer and colic. Mercurial compounds were used for phlegm, constipation, malignant scabies, worms and fevers (Wedeen 1989).

In the home environment mercury is added into many household products such as latex paints, adhesives, joint compounds, acoustical plates and cleaning solutions. Many of the products containing mercury do not identify its presence and care and adequate ventilation must be ensured when using potentially toxic household chemicals (MMWR 1990).

2.3 MERCURY POLLUTION

While a reduction has occurred in the utilisation of mercurial formulations in medicine, environmental pollution via evaporation of combustion of fossil fuels and dispersal through industry and agriculture has caused increases in the concentrations of mercury in air, soil and water. Industrial pollution via the production of steel, cement and phosphate, and the smelting of metals may account for the release of substantial amounts of mercury into the environment. Serious toxic pollution has caused poisoning of humans in Minamata, Japan in the 1950's as a result of eating contaminated fish, in New Mexico from contaminated pork and in Iraq in 1972 from contaminated cereal grains. The workers in the felting process of hatmaking, suffered from "Hatter's or Danbury shakes" as a result of mercury pollution of their working environment and were immortalised as the "mad hatter" in Lewis Carrol's "Alice in Wonderland".
The effect of pollution and subsequent contamination of marine life is exemplified by the situation in Sydney, Australia where in the 1970's 4 tonnes of mercury per year as industrial waste were discharged into the sea from ocean outfalls. More recent figures indicate that this amount is steadily increasing. The expected annual allowance of mercury in the 90's permitted through a new outfall being constructed at the Sydney beach suburb of Malabar is some 35 tonnes. Some fish, (e.g. Blackfish) accumulate mercury very easily, particularly in muscle tissue and liver. In a 1973 study Blackfish caught in Sydney's ocean waters had more than five times the National Health and Medical Research Council's maximum level of 0.5 mg/kg mercury and a private examination of Blackfish in 1989 produced levels more than twelve times the maximum recommended level. In a Water Board study in 1989 Red Morwong were consistently over the maximum level and this confirmed a bioaccumulation study in 1987 where Red Morwong and Blue Groper had high levels of mercury in muscle and liver tissue (Beder 1989).

Elemental mercury discharged into the atmosphere may return as acid rain. Pollution of drinking water and, as well, contamination of vegetable and animal food chains may occur. In lakes where this happens, consumption of fish may imply an increased exposure to methylmercury (Svensson, Bjornham, Schutz, Letterval, Nilsson & Skerfving 1987).
2.4 NATURAL SOURCES OF MERCURY

The principal sources of mercury are degassing of the earth’s crust, emissions from volcanoes and evaporation from natural bodies of water.

There is also an uncontrollable source of mercury released through the natural processes of leaching and volatilization. Mercury present in sea water occurs from the leaching of mercury from ores of mercury exposed in the oceans and from run off from the land. These sources must also be considered, in addition to the effects of industrial pollution, when evaluating concentrations of mercury in air, soil and water.

2.5. HUMAN EXPOSURE

The public is exposed to mercury in many forms:

- contaminants in foods (particularly fish) and food additives;
- environmental pollution (heavy industry, fossil fuels);
- medications (diuretics, antibacterial agents, laxatives, skin antiseptics);
- cosmetics (bleaching ointments);
- electric components;
- paints;
- thermometers;
- scientific instruments;
- inorganic disinfectants;
- amalgam restorations.

The main, everyday sources of exposure are through the diet and, to a lesser degree, from dental amalgam. Smoking may also contribute to mercury exposure.
3. TYPES OF MERCURY

Mercury (a.k.a. Quicksilver) is a distinctive metal in that it is liquid at room temperature. It can exist in a wide variety of physical and chemical states and from a toxicological standpoint can be classified into the elemental metal and its vapour, inorganic compounds (salts in the mercurous or mercuric state) and organic compounds (those associated with carbon in their molecular structure). The chemical symbol for mercury, Hg, is derived from its Latin name, hydrargyrum. The alternative name Quicksilver was given by Aristotle in 360 B.C. (Report International Committee 1969; WHO 1980).

3.1 CLASSIFICATION OF MERCURY STATES

**Elemental (Metallic) \([\text{Hg}^0]\)**

i. Elemental mercury

ii. Elemental mercury vapour

**Inorganic Compounds**

i. Mercurous mercury \([\text{Hg}_2^{++}]\) or \([\text{Hg}^+]\)

ii. Mercuric mercury \([\text{Hg}^{++}]\)

**Organic Compounds**

i. Short chain alkyl mercurials (e.g. methylmercury)

ii. Aryl mercurials (e.g. phenylmercury)
There is confusion in the use of the term "Inorganic Mercury" which can imply two particular and quite different categorizations of mercury forms:

1. Inorganic Mercury as a generic term which includes all forms of mercury which are not "Organic Mercury". In this context is included elemental (metallic) mercury (liquid and vapour) as well as inorganic mercurial compounds.

2. The specific subcategory of Inorganic Mercury which includes only the inorganic mercurial compounds and does not include elemental (metallic) mercury (liquid and vapour).

The statement in Sikorski & Paszkowski (1986) that 'the human placenta seems to play an important protective role in preventing inorganic mercury intoxication', makes no distinction as to what particular mercury forms are included in the term "inorganic mercury".

Aschner & Aschner (1990) state 'Inorganic mercury exists in the metallic form (Hg⁰), in the mercurous form (Hg⁺), and in the mercuric (Hg³⁺).' The text of the article, while making occasional broad references to inorganic mercury, (e.g. 'exposure to vapor leads to much higher concentrations of inorganic mercury in the CNS...'), actually is clear in distinguishing the forms being referred to (e.g. 'ingestion of inorganic salts and inhalation of metallic vapor.')
3.2 TOXICITY SCALE

The toxicity of mercury depends on both its concentration and its chemical form. Mercury and its compounds can be categorised in order of increasing toxicity into:

I. Liquid elemental mercury;
II. Inorganic mercury salts and aryl organic mercury compounds;
III. Elemental mercury vapour and short chain alkyl organic mercury compounds such as methylmercury.

From the standpoint of risk to health, metallic mercury vapour and the short chain alkyl mercurials, especially methylmercury, are the most important physicochemical states of this metal to which man is exposed (Enwonwu 1987).

Mercury has a particular affinity for sulphydryl groups of proteins. Because such groups are found in almost all proteins, mercury may be considered a potent but non-specific cellular poison capable of disrupting many cellular functions, particularly those of enzymes (Australian Environment Council 1982).

Organic forms of mercury are far more toxic to micro-organisms, aquatic organisms and birds than inorganic mercury (WHO 1989).
3.3. ELEMENTAL (METALLIC) MERCURY

3.3.1 LIQUID ELEMENTAL MERCURY
Liquid Elemental (Metallic) Mercury is poorly absorbed via the gastrointestinal tract after ingestion (<0.01%), because the mercury occurs as large globular particles and is thus regarded as posing a minimal health threat (Bauer 1985).

3.3.2 ELEMENTAL MERCURY VAPOUR
Exposure to Elemental Mercury is generally occupational, by inhalation of the toxic mercury vapour. Elemental mercury is volatile at room temperature and its rate of vaporisation is a function of both temperature and surface area. The toxicity of elemental mercury is greatly enhanced when in the gaseous form. Inhaled elemental mercury vapour is distinguished from inorganic mercury compounds by its ability to cross the blood-brain barrier and placenta rapidly. Rapid oxidation of mercury vapour occurs in the red blood cells through the mercurous into the mercuric ion. Completion of this reaction requires from one to several minutes and because of the delay, elemental mercury exists in the blood for a sufficiently long time to reach all tissues and organs. In the elemental form mercury is uncharged, lipid soluble and highly diffusible and therefore readily penetrates cell membranes and crosses the two critical biological barriers with ease. After final oxidation converts the mercury to the ionic form it then becomes charged, is no longer fat soluble and with diminished ability to cross membrane barriers. Thus oxidation in these tissues serves as a trap to hold the mercury and leads to accumulation in the brain and foetal
tissues (WHO 1976). Exposure to elemental mercury causes a preferential accumulation in the central nervous system, especially in the cerebral cortex, cerebellar cortex and certain brain nuclei, at a level some ten times higher than after exposure to inorganic mercury compounds.

The accumulation after exposure to mercury vapour is dependant on dose, frequency, duration of exposure as well as individual metabolic factors. The toxic effects of elemental mercury are produced after it has been oxidized to the mercuric ion which has a strong affinity for the sulphhydryl groups of proteins. The mercuric ion interferes with cellular metabolism and function within cells, as well as altering membrane function and transport, including release and uptake of neurotransmitters in the brain (Langan, Fan & Hoos 1987).

3.4 INORGANIC MERCURY COMPOUNDS

Inorganic mercury compounds are not fat soluble and do not easily cross membrane barriers. They are present in many foods in low concentrations and are assumed to cause no ill effects. Inorganic mercury compounds do not vaporize under normal conditions, but may be suspended in air as either dust or aerosols.

Inorganic mercury compounds, regardless of their initial state are immediately dissociated and converted into the mercuric form after entering the bloodstream and thus produce practically identical patterns of distribution. The kidneys contain the highest concentration followed by liver, spleen, brain and other
organs.

3.5 ORGANIC MERCURY COMPOUNDS

Organic Mercury compounds can be divided into mercurials which are relatively stable and those which rapidly split in the body. Alkyl mercury appears to be especially dangerous because of the extremely high degree of stability of the bond between the carbon and mercury atoms which results in this molecule not being degraded. This permits the molecule to maintain its destructive activity for from weeks to years. Aryl mercurials are much less stable; consequently the injuries which they and inorganic mercury cause are almost invariably reversible (Oehme 1978).

3.5.1 ARYL MERCURIALS

Phenyl mercury and methoxyethyl mercury compounds, used extensively in pesticides and preservatives, break down rapidly in the body and this results in a distribution pattern which after a preliminary period, resembles the distribution of inorganic mercury (Aust C'wealth Dept Health 1978).

3.5.2 ALKYL MERCURIALS

Methylmercury is formed naturally in the aquatic and terrestrial environment from elemental mercury and mercuric mercury by a process of bioaccumulation. Following inadequate and improper disposal of wastes into oceans and rivers, inorganic mercury is converted by microorganisms to the more toxic methylmercury (methylation) whence it enters the food chain.
In Minamata, Japan between 1953 and 1960, a plastics manufacturing operation discharged methylmercury chloride into Minamata Bay and River. Consumption of mercury contaminated fish and shellfish caused the death of 46 people. There are now 784 patients officially designated with Minamata Disease, of whom 103 have died and some 3000 persons who are suspected cases (Hanson c1985). In Minamata the polluting effluent contained both methylmercury and elemental mercury, the latter being subsequently methylated to methylmercury.

In Iraq in 1956 and 1960 over 200 people were poisoned by eating bread made from grain treated with methylmercury fungicide, with at least 20 deaths. Again in Iraq in 1972 a similar incident resulted in the hospitalisation of 6530 victims and 500 deaths (Langan,Fan & Hoos 1987).

Organic mercury forms stable compounds during blood transport which easily diffuse from blood to tissues and from tissues to blood and readily cross placental and blood-brain barriers. The stability of alkyl mercury compounds favours their accumulation in the Central Nervous System and the highest neurotoxicity appears to be a special property of short carbon chain alkyl mercury compounds (Oehme 1978). High levels of methylmercury are also located in the liver and kidneys.
4. KINETICS AND METABOLISM

The biokinetics of the different mercury compounds is complicated with consequences for distribution, retention, excretion and toxic effects. Marked differences in metabolic behaviour exist between inorganic and aryl Hg mercury compounds AND the alkyl Hg derivatives, particularly those of short carbon chain such as dimethylmercury. The latter are better absorbed, better retained, more firmly bound in the tissues and induce higher brain mercury levels (Underwood 1977).

4.1 BIOTRANSFORMATION

Biotransformation occurs by several methods:

i. Oxidation of elemental (metallic) mercury vapour to divalent ionic mercury

ii. Reduction of divalent mercury to elemental (metallic) mercury

iii Methylolation of inorganic mercury

iv. Conversion of methylmercury to divalent inorganic mercury

4.1.1 OXIDATION OF ELEMENTAL (METALLIC) MERCURY VAPOUR TO Divalent IONIC MERCURY.

The oxidation of metallic mercury vapour to divalent ionic mercury [see Section 3.3.2] by catalase enzymes in the red blood cells takes place soon after absorption but is sufficiently delayed to allow some portion of the inhaled nonpolar mercury vapour to enter brain and foetal tissues where it is then contained and accumulates after oxidation.
4.1.2 Reduction of Divalent Mercury to Elemental (Metallic) Mercury.
Reduction of divalent mercury to elemental mercury has been demonstrated in animals and humans and a small portion of absorbed inorganic mercury may be exhaled as mercury vapour (WHO 1991).

4.1.3 Methylation of Inorganic Mercury
Minor methylation of inorganic mercury has been reported in vitro by intestinal or oral bacteria but there is scant evidence for synthesis of organomercurial compounds in human tissues. [See Section 11.6]
Trevors (1986) notes that bacteria capable of methylating Hg^{++} have been isolated from sediment, water, soil and the human gastrointestinal tract. Mercury methylation can be either chromosomal or plasmid-encoded in bacteria and mediated by a series of enzymatic reactions. It is possible that certain bacteria use methylation as a resistance/detoxification mechanism.

4.1.4 Conversion of Methylmercury to Divalent Inorganic Mercury.
One pool of inorganic mercury in the body stems from demethylation of retained methylmercury and is considered a fundamental phase in the process of excretion of mercury after exposure to methylmercury (WHO 1990). Demethylation takes place in several organs and during recent years there is a growing indication that a biotransformation of significance may also take place in the brain (WHO 1988).
A study on the brains of monkeys exposed for several years to methylmercury showed that 10-30% of the total mercury content was in the inorganic form at the end of the exposure period and approximately 90% was inorganic mercury 0.5-2.0 years after exposure terminated (Lind, Fribuerg & Nylander 1988).

In humans after high oral intake of methylmercury for two months inorganic mercury levels as a proportion of total mercury were:

- whole blood 7%
- plasma 22%
- breast milk 39%
- urine 73%
- liver 16-40% (WHO 1990)

4.2 HALF-LIFE OF MERCURY

Retention time of mercury in organs is variable, biologic half-life ranging from days to weeks for most of the absorbed mercury, but extending to years for a fraction of the mercury. There is significant variation in tissue retention, the tissue half-life of mercury in blood being 1.7-3.0 days, for the kidneys 60 days and the for the brain in excess of 20 years. Longest retention is found in the brain, kidney and testis. The kidney is the main target organs for the divalent ionic compounds of mercury and after acute exposure. The brain is the critical target organ for methyl mercury and mercury vapour and is most significant in cases of chronic low level exposure to mercury vapour.
Following inhalation of elemental mercury, the average half-times for clearance are: lungs (1.7 days), head (21 days), chest (43 days), kidney region (64 days), and whole body (58 days) (Hursh, Clarkson, Cherian, Vostal & Mallie 1976).

The biologic half-time for methyl mercury is 70 days, that for salts of inorganic mercury 40 days and mercury vapour 35-90 days. The brain may not follow the same kinetics of elimination as the rest of the body and thus the concentration of mercury in the brain may remain at high levels for many years.

Rice (1989) notes that the half-life of methylmercury in humans is 52-93 days for whole body and 49-164 days for blood. Rice’s study in monkeys of chronic exposure to methylmercury showed that the brain half-life is considerably longer than blood half-life.

The concentrations of inorganic mercury, methylmercury and total mercury were determined from autopsy for 46 Japanese subjects (Matsuo, Suzuki & Akagi 1989). Total mercury averages were several hundreds of ng/g in renal cortex, renal medulla and liver, and were several tens of ng/g in cerebrum, cerebellum, heart and spleen. Inorganic mercury accumulated more in kidney (81-84%) and liver (67%) with heart (25%), spleen (22%), cerebrum (20%) and cerebellum (14%). Methylmercury levels were cerebrum, cerebellum, heart and spleen (all approx. 80%), liver (33%), renal medulla (15%) and renal cortex (11%). Age was a significant factor in increased inorganic mercury concentrations in cerebrum and heart.
After long sustained exposure Kosta, Byrne & Zelenko (1975) reported that the thyroid and pituitary glands contained the highest quantities of mercury, lesser levels in kidney, lungs and brain.

Cavanagh (1988) discusses the long term persistence of mercury in the brain and notes that in the brain mercury is sequestered in lysosomal dense bodies of neurons, this mechanism initiated by binding of the toxic ions to proteins. In contrast to excretion of mercury by hair and skin cells, the mercury in neuronal cells may undergo some degree of enzymal degradation to an irreducible chemically inert state (e.g.lipofuscin) which is shown to accumulate with age. Additionally the nerve cells can discharge dense bodies which are taken up by astrocytes, thence into the endothelial cells and ultimately into the blood stream. This slow process may account for the long half-life of mercury in the brain as well as the maintenance of high mercury levels with no clinical sequelae.

4.3 ABSORPTION

4.3.1. INHALATION

Approximately 80-85% of inhaled mercury vapour is retained. This occurs almost entirely in the alveoli of the lungs irrespective of whether inhalation of ambient air containing mercury vapour is nasal or oral. It has been stated that some 10% of an inhaled dose of mercury vapour appears in the blood. 'Very little is known of the pharmacokinetics that convert an inhaled dose of elemental mercury vapour to the critical concentration in the brain, and there are no reliable indicator media for mercury levels in the brain.' (Clarkson 1983).
There is meagre information on the pulmonary retention of inorganic mercury compounds. Particulate matter deposited in the upper respiratory tract would be cleared more quickly than that in the lower respiratory tract. A significant absorption would probably take place directly from the lungs and to some extent from the gastrointestinal tract after mucociliary clearance of non-absorbed mercury.

4.3.2 INGESTION

Inorganic mercury compounds (as seen in solid dental amalgam) are well excreted in the faeces and urine, only about 7-10% of an ingested dose being absorbed from the gastro-intestinal tract, and 0.27% appearing in the blood. Absorption may be higher in young children. Although liquid metallic mercury is minimally absorbed from the gastrointestinal tract, there are some indications that accidental ingestion of metallic mercury (e.g. accidental breakage of thermometers) can increase blood levels. Almost all of alkyl mercury compounds are efficiently absorbed during passage through the digestive tract to the extent of approximately 95%. Organic mercury, after diffusion is very stable..

4.3.3 SKIN

Hursh, Clarkson, Miles & Goldsmith (1989) estimated that the rate of percutaneous uptake of metallic mercury vapour through the skin is approximately 2.2% of that by inhalation.

*Two phenomena operate to reduce the effective toxicity of skin absorption:*

a. approximately one half of the mercury taken up is shed by the desquamating epidermal cells during several weeks, and
b. the remainder is slowly released to the general body system rather than being rapidly transported into the blood, as is the case for inhaled mercury vapour.

There are many documented cases of mercury poisoning due to cutaneous application of inorganic mercury compounds: (De Bont, Lauwerys, Govaerts, Moulin 1986)

- calomel for syphilis;
- ointments containing yellow mercury oxide;
- skin lightening creams containing ammoniated mercury;
  [A case of nephrotic syndrome caused by mercury-containing skin lightening cream is reported by Oliveira, Foster, Savill, Syme, Taylor (1987)]
- mercurochrome for skin lesions.

4.3.4 **Axonal Transport**

Axonal transport of mercury has been shown in animal experiments, Arvidson (1987) reporting retrograde transport in the hypoglossal nerve in rats injected in the tongue with small volumes of inorganic mercury. It is not known whether this takes place in mammals, and whether a similar transport can take place via nerves from the teeth.

It has been postulated that a corrosion process liberates mercury ions from dental amalgam which may reach the central nervous system from the dental pulp via a neural route. The mercury content of trigeminal nerves varies in different studies and the significance of retrograde axonal transport so far seems obscure (Taskinen 1989).
4.4 ELIMINATION

Most forms of mercury are eliminated through urinary and faecal excretion, with lesser amounts through sweat and saliva. A small fraction of absorbed inorganic mercury and inhaled mercury vapour is lost by exhalation and deposition in hair (pilial excretion).

Urinary excretion reflects mainly inorganic and elemental exposure and is more common when mercury exposure is high. After exposure to mercury vapour a small proportion of the urinary mercury may be in the form of elemental mercury. The kidneys have a great capacity to eliminate mercury, allowing for substantial absorption and excretion without untoward effects.

After organic mercury (methylmercury) exposure approximately 90% is eliminated in the faeces, by the two separate processes of biliary excretion and exfoliation of intestinal epithelial cells. Some 40-50% is excreted in the inorganic form.

The amount of mercury excreted through saliva would seem to be insignificant compared with urinary excretion. Symptoms of severe mercury poisoning include an unpleasant metallic taste and increased flow of saliva (Jenkins 1978).

It has been stated that in man with exposures to concentrations of mercury in air below 100 μg/m³ elimination is complete (Bauer 1985). If complete
elimination is equated with lack of toxic effects then this figure would have to be challenged as it is now accepted that onset of symptoms of mercury poisoning can occur below this level of occupational exposure. Also not taken into account is the mercury which is retained for extended periods in such organs as the brain.

4.5 NEUROTOXICITY OF MERCURY

The Central Nervous System is targeted by elemental mercury vapour and organic methylmercury. A compromise in the integrity of the blood-brain barrier has been proposed as the reason for mercury toxicity in the Central Nervous System. After exposure to mercury vapour there is rapid oxidation in erythrocytes to divalent ionic mercury. Some of the monoatomic gas, (because of its lipid solubility), traverses the blood-brain barrier, after which it is rapidly converted to the non-diffusible divalent form. The brain is supplied by paired Carotid and Vertebral Arteries and at rest receives a flow of 750 mL/min from a cardiac output of 5800 mL/min. There is thus some question as to what proportion of mercury vapour actually reaches the blood-brain barrier in its original form (Akers 1991b).

Methylmercury achieves a greater and more complete degree of passage through the blood-brain barrier by the process of molecular mimicry (Aschner & Aschner 1990).

This might account for the high degree of accumulation of methylmercury in the CNS and the associated neurological disturbances. The authors note that although the fate of methylmercury in the brain is unclear, the rate of demethylation may be greater in conditions of chronic exposure to low doses.
5. CLINICAL EFFECTS

There are subtle differences in the clinical effects of toxic exposure to inorganic and organic mercury. Although both forms of mercury concentrate to a high degree in the central nervous system, the actions are different with the effects of elemental mercury being neuropsychiatric, whereas those of organic mercury are sensorimotor (Klaassen, Amdur & Doull (Eds) 1980).

Mercury poisoning leads to impairments of the cerebellum, the basal ganglia and the cerebral cortex. Organic forms of mercury such as methylmercury are far more toxic to the Central Nervous System than inorganic mercury (Lille, Hazemann, Garnier, Dally 1988).

The developing nervous system may be particularly vulnerable to methylmercury, with neuropathological effects such as poor myelination occurring in children of unaffected mothers in Minamata. However peripheral nerves of adults seem more vulnerable than the Central Nervous System. Animal studies have not shown significant neuropathology except with very high doses of methylmercury and no effect in the case of inorganic mercury administered in the range of 10-20 mg/kg/day for more than one year (Wiggins 1986).
5.1 INORGANIC MERCURY

5.1.1 Acute Effects.

Severe intoxication by inorganic mercury can be provoked by:

i) accidental short-term inhalation of high concentrations of elemental mercury vapour causing:

- bronchial irritation,
- erosive bronchitis and
- diffuse interstitial pneumonitis

ii) ingestion of electrolytic inorganic salts of mercury producing:

- local necrotic changes in the gastro-intestinal tract,
- circulatory collapse or
- acute renal failure.

5.1.2 Chronic Effects

Chronic poisoning by inorganic mercury usually occurs by occupational exposure to elemental mercury vapour alone or in combination with mercuric dust. This toxicity is classic mercurialism and effects mainly the central nervous system producing a wide range of clinical symptoms.

i) EARLY SIGNS (micromercurialism)

Early signs are non-specific and include such symptoms as:

- muscular weakness,
- fatigue,
- anorexia,
- weight loss and
- gastro-intestinal problems.

Bauer (1985) includes within the definition of micromercurialism the symptoms of increased excitability of the central and autonomic nervous system, fine tremor and salivation, but not lesions of the central nervous system.
ii) INCREASED EXPOSURE

With increasing exposure the characteristic mercurial tremor appears as fine trembling of muscles interrupted by coarse shaking movements. Psychological and behavioural changes (erethism) parallel the development of tremor.

Symptoms may include:

- increased excitability,
- loss of memory,
- insomnia,
- severe depression,
- irritability and confusion,
- ataxia,
- speech disorders,
- reflex abnormalities,
- visual disturbances and
- impaired nerve conduction.

Oral symptoms include:

- gingivitis,
- excessive salivation,
- metallic taste and
- loosening of teeth (adapted from Langan, Fan & Hoos 1987).

The classic symptomatic triad of increased excitability, tremor and gingivitis have been regarded as conventional manifestations of mercury poisoning from inhalation of mercury vapour.

Describing mercurial disease among Hatters in New Jersey, USA in 1860, Freeman commented that hundreds showed all the characteristics of Mercurial Salivation and Stomatitis... 'ulceration of the gums, loosening of the teeth, foetor of the breath, abnormal saliva, tremors of the upper extremities, or a shaking palsy.'

The incidence of gingivitis evidenced by inflammation of gingiva with swollen and bleeding margins may not be as significant as previously assumed. In the period of data accumulation, oral hygiene was poor and gingivitis was a universal
condition. A chronic gingivitis may have been exacerbated by the diminished health and natural resistance of the subject exposed to mercury vapour but the aetiological factor in the gingivitis may not have been mercury. Similar comments have been made with regard to the status of gingival disease in the symptomology of scurvy.

Erethism is the collective term for the behaviour and personality changes which result from chronic mercury poisoning. This term derives from the Greek word for shyness. There is timidity under observation coupled with quarrelsome behaviour. As well there is restlessness and though tired, difficulty in sleeping (Uzzell & Oler 1986).

Roels, Abdeladim, Braun, Malchaire, Lauwers (1989) measured hand tremor in a group of 54 workers exposed to moderate levels of mercury vapour reflected by mean blood mercury levels of 2.4 μg/dL (24 μg/L) and urine mercury levels of 63 μg/g creatinine. Psychomotor tests (hand steadiness and eye-hand coordination) revealed preclinical alterations in postural and intentional tremor. The authors suggest that young adults (<21 years) may be more susceptible to the neurotoxic effects of mercury.
5.2 ORGANIC MERCURY

The effects of organic mercury poisoning are mainly neurological and depend on degree of exposure, with a long latent period lasting several months. The alkylmercury compounds (particularly methylmercury) readily pass through such physiological barriers as the blood-brain barrier, blood-testes barrier and the placenta. There is a high quantity accumulation in the brain, with the major pathological effects being on nervous and reproductive systems as well as the developing embryo and foetus. Within the central nervous system, the damage from methylmercury is selectively limited to specific focal areas such as the granule cells of the cerebellum and the neurons in the interstices of the visual cortex. The initial damage is non-selective, inhibiting protein synthesis, but these particular cells may be unable to effect repair (Clarkson 1987).

Common functions affected include sensory, visual and auditory functions, and co-ordination related to the cerebellum (WHO 1990). Symptoms include tremors, digital paraesthesia, progressive incoordination, loss of vision and hearing, and mental deterioration.
The progressive nature of the effects of methylmercury poisoning is shown in Table 1.

<table>
<thead>
<tr>
<th>PROGRESSION OF METHYLMERCURY POISONING</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prognostic Zone</strong></td>
</tr>
<tr>
<td>Reversible:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Possibly Reversible:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Irreversible:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Fatal:</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Table 1. Source: Environmental Mercury Contamination 1972

5.3 NEUROPSYCHOLOGICAL EFFECTS

Modern psychological testing has elicited subtle but significant neuropsychologic effects from chronic exposure to low levels of mercury.

Uzzell & Oler (1986) measured neuropsychological performance of 13 female dental auxiliary workers with elevated head mercury levels. Chronic subtoxic levels of inorganic mercury appear to produce mild changes in short-term
nonverbal recall and heightened distress generally, and particularly in categories of obsessive compulsion, anxiety and psychoticism, without alterations in general intellectual functioning, attention, verbal recall and motor skills.

Tollefson & Cordle (1986) state that: 'there is some evidence to suggest that methylmercury may be the cause of subtle neurological impairments when ingested at even low to moderate levels, particularly the prenatal and early childhood periods'.

Soleo, Urbano, Petrera, Ambrosi (1990) studied the psychological effects of low exposure to inorganic mercury on 28 workers, comprising 8 chronically exposed and 20 occasionally exposed. Mean urinary mercury concentrations in the exposed group were 30-40 μg/L. Of the psychic functions only short term auditory memory was impaired in the chronically exposed workers. No changes of visual motor functions were observed. The personality and mood of the occupationally exposed workers was considerably changed from the control group suggesting depression.

Rosenman, Valciukas, Glickman, Meyers, Cinotti (1986) evaluated 42 workers in a chemical plant producing inorganic mercury compounds. Routine clinical testing using physical examination, blood chemistries and urinalysis were generally normal. In contrast there were complaints of neuropsychological symptoms, elevated n-acetylB-D-glucosaminidase (NAG) levels, decreased motor nerve conduction velocities and the presence of lenticular opacities when organ systems known to be affected by mercury were targeted. The authors suggest that
more sensitive indicators of mercury toxicity need to be included in routine medical screening to help diagnose the aetiology of neuropsychological symptoms and prevent long-term sequelae in workers exposed to mercury.

Siblerud (1989) claims a relationship between mercury in amalgam and mental illness but this study is seriously flawed [See Section 13.2]

5.4 TREATMENT OF MERCURY POISONING

The type of mercury, the degree of exposure and the period involved are major prognostic indicators. In low-level chronic exposure, where symptoms are minimal, removal of the source of mercury can often result in complete resolution. If ataxia, loss of motor control and mental deterioration are exhibited, these symptoms are generally not reversible and the prognosis poor.

The traditional treatment for mercury poisoning has been by diaphoresis and physiotherapy. The benefits of sweating as a therapy has been recognised for centuries. Chelating agents such as dimercaprol (e.g B.A.L.) and d-penicillamine have been recommended as antidotes for mercury poisoning for many years. The reports are somewhat contradictory, but the chelating agents seem to be more effective in organic exposure than for inorganic mercury poisoning (Sunderman 1988).

Campbell, Clarkson & Omar (1986) reported the use of 2,3-dimercaptopropane-1-sulfonate as an effective treatment for two patients with elemental mercury
vapour poisoning. One patient was asymptomatic despite high urine mercury levels, the other presented with toxic symptoms including abnormal electromyograms and haematuria. Use of the drug showed a prompt clearing of the haematuria and reversal of electromyographic abnormalities in the symptomatic patient. Excretion half-life of the mercury changed from 33.1 days before therapy to 11.2 days during therapy.

Six workers were exposed to metallic mercury vapour in a confined space. One patient had acute renal and respiratory failure, another acute bilateral pneumonitis and the remaining four corrosive oropharyngeal mucositis with a 'flu-like' syndrome. After removal from the source of exposure and the institution of support measures (including chelant therapy in the first two patients), all recovered without evidence of residual damage (Aguado, de Quiros, Marin et al 1989).

5.5 SELENIUM

Selenium is a trace element that has been linked to mercury, laboratory experiments indicating that selenium acts as a powerful antagonist to mercury intoxication (Hansen 1988). Low selenium levels have been coupled with modern disease states (Trimmer 1988) and included as supplements in the pre-amalgam replacement phase of mercury detoxification (Ziff 1988a). [See Section 15.2.2]
Hansen (1988) notes that the mercuric ion binds to selenium to form a biologically inert complex leading to decreased body burden of both elements. This reaction seems to take place only when a threshold of mercury exposure is exceeded. Selenium decreases the oxidation rate of elemental mercury, which can cause an increased brain uptake. The author hypothesises that to man selenium is of no benefit in cases of exposure to mercury either as mercuric mercury or as mercury vapour.

The formation of a selenium complex may be responsible for the long half-time of a fraction of the mercury (WHO 1991).

Molin (1990) found that mercury release from dental amalgam does not influence the selenium status in man.
6. INTAKE AND THE HUMAN BURDEN OF MERCURY

6.1 MERCURY LEVELS IN FOODS

6.1.1 Non-Fish Foods
Mercury in food other than fish is generally below 20-30 µg/kg (20-30 ng/g).

6.1.2 Fish Foods
The main dietary source of mercury is seafood, the majority of which (up to 90-95%) is methylmercury. The dietary intake of mercury depends primarily on the concentrations of methylmercury in fish and the amount of fish consumed. Alkylmercury, formed in the bottom sediment of the ocean and in freshwater systems, is enriched to a high degree in the aquatic food chain with the highest levels occurring in the predatory fishes. Freshwater fish and most oceanic species have levels around 150-200 µg/kg (150-200 ng/g). The large carnivorous species (Shark, Tuna, Swordfish) have higher levels ranging from 200-1500 µg/kg and these often exceed the regulatory limit for mercury of 500 µg/kg (0.5 mg/kg) established by several countries. Mean concentrations in molluscs and crustacea seldom exceed 100 µg/kg (UNEP/WHO 1988). In contaminated freshwater areas mercury levels in fish may exceed 500 µg/kg.
The half-life of methylmercury in fish is up to 2 years. The loss occurs in two stages:

a. Methylmercury is distributed throughout the tissues, mainly muscle, over a period of a few weeks and then is discharged from the binding sites very slowly. This is why fish (particularly salt water) are a major source of mercury exposure in humans.

b. During this period the fish are continuously supplied with methylmercury from the water providing a mechanism for the continuous increase of mercury residues. Older fish accumulate considerable amounts of mercury (Tollefson & Cordle 1986).

6.2 INTAKE OF MERCURY

6.2.1. ORAL

Approximately 80% of the daily intake of mercury is in the form of methylmercury, the average daily consumption ranging from 20 μg/day to in excess of 80 μg/day (Mottet, Shaw & Burbacher 1985).

The daily intake of inorganic mercury will probably not exceed 10 μg/day in the absence of occupational exposure from, e.g. mercury inhalation, drinking water and food (Berlin 1986).

A U.K survey of mercury in food estimated the intake of mercury from 1.5 kg food (U.K. daily average) to be 5-10 μg mercury daily.
In Sweden the average daily intake of uncontaminated fish gives rise to a range of methylmercury intake of 1-20 μg/day.

A study in the Netherlands by van Dokkum, de Vos, Muys, Wesstra (1989) analyzing 221 different food items over a period of 2.5 years found the mean daily amount of mercury to be 0.7 μg/day, with a range of 0.43-1.44 μg/day. In the United Kingdom, the average intake of mercury is 0.3 μg/kg per week. This figure rises to 1.5-1.9 μg/kg in coastal fishing communities. Although some 12% of survey populations exceeded the Provisional Tolerable Weekly Intake (PTWI) for methylmercury, blood levels did not give rise to any health concerns.

The MECCA Study (1973) reported that the national average fish consumption in the USA is 14 gm/day which at the methylmercury limit of 0.5 ppm converts to 7 μg methylmercury daily. The average high consumption of approximately 60 g/day would amount to 30 μg/day. A U.S survey showed an average intake of 2.48 μg/day methylmercury, with 98% of the test group being below 17 μg/day and the maximum intake being 31.7 μg/day (Finch 1973).

A Finnish study (Mykkanen, Rasanen, Ahola, Kimmpa 1986) studied 1768 children up to 18 years of age for dietary heavy metal consumption. Consumption of fish was positively associated with intakes of mercury and arsenic. Daily intake of heavy metals increased with age but the authors noted that heavy metal exposure via diet is highest in young children since they consume more food in proportion to body weight than adults.
A study by Buzina, Suboticanec, Vukusic, Sapunar, Antonic, Zorica (1989) assessed the effect of industrial pollution on seafood content and dietary intake of total mercury and methylmercury. The authors surveyed 79 families (314 people) in an industrially polluted area on the Adriatic coast and 63 families (255 people) in an unpolluted control area of the same coast. In 40 species of local fish analyzed, methylmercury, on average, accounted for 52.5% of total mercury in the polluted area as against 66.7% methylmercury in the control area. Although there were variations from 10% to 100% in the proportion of methylmercury to total mercury, the result in the non-polluted area varies quite dramatically from the accepted view that nearly all mercury in fish is methylmercury.

From seafood alone the consumption pattern showed a daily intake of total mercury of 9.2-25.2 μg in the industrially polluted area and 6.3-17.9 μg in the control area. As a proportion of total mercury, methylmercury intake constituted 4.9-12.9 μg (polluted area) and 3.9-14.6 μg (control area). A number of subjects in both polluted and control areas, particularly in the 7-19 years age group exceeded the WHO PTWI levels of 300 μg of total mercury or 200 μg of methylmercury. The study showed a higher intake of methylmercury in the control group and noted that this was due to higher fish consumption (>5 times per week) compared to that in polluted waters. The reduced consumption of fish in polluted waters may attest to increasing awareness of the population of health effects of mercury. They conclude that increased coastal water and marine sediment mercury levels due to local industrial pollution have increased mercury content of seafood, which has in turn affected dietary intake of the local population.
6.2.2 Inhalation

Airborne mercury is predominantly elemental. The average concentration is approximately 20 ng/m³ with variations from 0.5-50 ng/m³.

6.3 Total Body Burden

Cassarett and Doull's Toxicology (1986) estimates that the general population is exposed to daily metallic mercury levels of:

- Air - 1 µg Hg/day
- Water - 2 µg Hg/day
- Food - 20 µg Hg/day .... but up to 75 µg Hg/day depending on fish consumption.

Similar figures have been expressed by Snyder et al (1975) estimating:

- Air - 1 µg Hg/day
- Food and Fluids - 15 µg Hg/day

A daily figure of 10 µg Hg/day intake of inorganic mercury has been estimated by Berlin (1987). The natural mercury content of the daily diet varies from 5-20 µg and may reach up to 100-300 µg in coastal areas (Nauen, Tomasi & Santorini 1982).
The United States Environmental Protection Agency (USEPA) (1984) has estimated that for the adult population not occupationally exposed to mercury the average daily retention rate to be:

Mercury vapour- 153 ng Hg/day (0.15 μg Hg/day).
Organomercurials (mainly methylmercury from fish)- 3666 ng Hg/day (3.7 μg Hg/day)
Inorganic mercury compounds (from dietary products other than fish)- 2000 ng Hg/day (2.0 μg Hg/day)
Mercury in drinking water- 5 ng Hg/day (0.005 μg Hg/day)

The word 'retention' in the USEPA estimates must be clarified as to whether it denotes exposure or uptake, since these figures are approximately 10% of comparable estimates of daily dose.

Cooley and Young (1984) estimated the daily intake of mercury by humans as:

Air- <60 ng (0.06 μg)
Water- <2 ng (0.002 μg)
Food- 5000-10000 ng (5-10 μg).

WHO (1991) gives a daily intake for mercury and mercury compounds:
Elementary mercury vapour- 3.9-21 μg Hg/day
Inorganic mercury compounds- 4.3 μg Hg/day
Methylmercury- 2.41 μg Hg/day

A summary of published figures for the daily dose of mercury and maximum accepted daily levels is given in Table 2. All these figures must be assessed on
the basis of the toxicity of the type of mercury and the percentage taken up by
the body. Inorganic mercury compounds are the least toxic, mercury vapour and
methylmercury being most toxic.

**Body uptake is as follows:**

1. Inorganic mercury compounds- 10%

2. Mercury vapour- 85%

3. Alkyl Organomercurials (methylmercury)- 95%

No clear consensus has been established as to what is a safe level of mercury
consumption for the general population and the proportion retained from
different types of mercury needs to be considered over and above a simple
quantitative assessment of mercury exposure.

<table>
<thead>
<tr>
<th>DAILY DOSE MERCURY AND MAXIMUM ACCEPTED DAILY LEVELS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>USEPA (1984)</td>
</tr>
<tr>
<td>Cassarett &amp; Doulls Toxicology (1986)</td>
</tr>
<tr>
<td>Snyder (1975)</td>
</tr>
<tr>
<td>Berlin (1987)</td>
</tr>
<tr>
<td>Friberg (1986)</td>
</tr>
<tr>
<td>Naumen (1982)</td>
</tr>
<tr>
<td>Buzna (1989)</td>
</tr>
<tr>
<td>Coolay (1984)</td>
</tr>
<tr>
<td>Nottet (1985)</td>
</tr>
<tr>
<td>U.K. survey</td>
</tr>
<tr>
<td>Sweden</td>
</tr>
<tr>
<td>Van Dokkum (1989)</td>
</tr>
<tr>
<td>MECCA (1973)</td>
</tr>
<tr>
<td>Finch (1973)</td>
</tr>
<tr>
<td>WHO (1990, 1991)</td>
</tr>
</tbody>
</table>

**MAXIMUM ACCEPTED DAILY LEVELS**

|          |              |              |              |              |
| USEPA    | 30           |              |              |              |
| JEFCA    | 43c          |              |              |              |
| MECCA (1973) | 30      |              |              |              |
| ELEY & COX (1987) |          |              |              | 200.00       |
| WHO (1991) |            |              |              | 29           |

Table 2

- *Fish source from non-polluted area...contains both methylmercury and inorganic mercury*
- *Non-specific foods...includes fish*
- *No more than 28 µg/day methylmercury*
- *From dental amalgams*
6.4 MAXIMAL ACCEPTED MERCURY INTAKE

The United States Environmental Protection Agency (USEPA) recommends a maximum mercury intake of 30 μg/day from all sources (USEPA 1984).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has established a provisional tolerable weekly intake (PTWI) of 300 μg of total mercury per person (43 μg/day) of which no more than 200 μg (28 μg/day) should be present as methylmercury. These amounts are equal to 5 μg/kg body weight and 3.3 μg/kg body weight respectively (UNEP/WHO 1988).

The PTWI of FAO/WHO can be exceeded if 2 meals of 327 gm of edible fish parts are eaten during the week if one of them is based on big predatory fish (Aust C’wealth Dept of Health, 1978 Section 4).

The MECCA Study in 1973 assumed that man can consume up to 30 μg of methylmercury daily (Finch 1973).

WHO (1991) notes that the weekly consumption of 200 g of fish having 500 μg Hg/kg will cause an intake of 100 μg of predominantly methylmercury. This amount is half the tolerable recommended weekly intake - 200 μg/week; 29 μg/day).
8. EXPOSURE LIMITS FOR MERCURY

8.1 OCCUPATIONAL LIMITS FOR EXPOSURE TO MERCURY

The toxic effects of mercury have been documented since early times and in Idria (1665) the first industrial hygiene law was passed protecting mercury miners by limiting their work day to six hours (Burt 1986).

8.1.1 U.S.A

The American Conference of Governmental Industrial Hygienists (ACGIH) in 1983 recommended the following threshold limit values as time weighted average in workroom air for an 8 hour day:

- 100 $\mu$g/m$^3$ for mercury, aryl and inorganic compounds
- 50 $\mu$g/m$^3$ for mercury vapour (all forms except alkyl)
- 10 $\mu$g/m$^3$ for organo(alkyl)mercury

The short-term exposure limit for organo(alkyl)mercury is:
- 30 $\mu$g/m$^3$ per 15 minutes.

8.1.2 UNITED KINGDOM

In Great Britain the environmental safety limit for mercury vapour is set at 50 $\mu$g/m$^3$ (50 ng/L).
8.1.3. World Health Organization

The WHO Study Group in 1980 concluded that long term exposure (8 hrs/day for 225 days/yr) to 100-200 $\mu g/m^3$ of mercury vapour in air would give rise to tremor and similar exposure to 50 $\mu g/m^3$ would be associated with non-specific symptoms.

Thus they recommended that the occupational exposure limits be:

- 25 $\mu g/m^3$ for metallic mercury vapour (time weighted average) for air and
- 50 $\mu g/m^3$ creatinine for individual levels of urine.

For short term exposure of mercury vapour the WHO Study Group recommended a value of 500 $\mu g/m^3$.

Additionally they noted that due to the potential foetotoxicity of mercury, exposure to women of child-bearing age should be kept as low as possible.

Inorganic mercury, in other forms than elemental mercury, is less toxic and the occupational exposure was recommended by the WHO Study Group to be set at 50 $\mu g/m^3$.

8.1.4 Australia

The National Occupational Health and Safety Commission in May 1990 in its "Exposure Standards for Atmospheric Contaminants in the Occupational Environment"(1990) issued the following limits:

- Mercury (elemental)- 0.05 mg/m$^3$ (50 $\mu g/m^3$ )
- Mercury (alkyl compounds)- 0.01 mg/m$^3$ (10 $\mu g/m^3$ )
- Mercury (aryl & inorganic compounds)- 0.1 mg/m$^3$ (100 $\mu g/m^3$ )

These figures are equivalent to the ACGIH (1983) levels quoted above 8.1.1.
8.1.5 **Sweden, Finland and the Soviet Union**

Finland and Sweden have an occupational limit of 25 $\mu g/m^3$ for mercury vapour, whereas the Soviet Union has had a limit of 10 $\mu g/m^3$ since 1944.

8.1.6 **Other Occupational Recommendations**

Roels et al (1982 & 1987) have proposed a biological occupational limit for urine of 50 $\mu g$ Hg/g creatinine to prevent preclinical alterations of hand-eye coordination and hand steadiness. This figure corresponds to 40 $\mu g/m^3$ time weighted average exposure of mercury vapour in air.

Soleo et al (1990) suggest that the TLV-TWA for mercury vapour should be lowered to 0.025 mg/m$^3$ (25 $\mu g/m^3$) and that the biological urinary exposure indicator for biological monitoring should be 25 $\mu g/L$.

8.1.7 **Secondary Effects of Occupational Exposure**

The significance of the peripheral effects of occupational exposure to mercury vapour can be seen in cases such as that reported by Swinehart (1988) concerning a 20 month old infant, the child of a thermometer plant worker. The house was 500 ft from the smelter and the child played with his father's hat which was worn during work. The child exhibited frank signs of mercurialism and high urine levels of mercury.
8.2 GENERAL POPULATION LIMITS FOR EXPOSURE TO MERCURY VAPOUR

Casarett & Doull’s Toxicology (1986) quotes the recommended standard for permissible exposure limits for inorganic mercury in the air in the workplace as being 0.05 mg/m\(^3\) Hg (50 \(\mu g/m^3\)) and equivalent to an ambient air level of 0.015 mg/m\(^3\) (15 \(\mu g/m^3\)) mercury for the general population (24 hour exposure).

A different figure for the general population can be calculated using occupational exposure of 8hr/day for 225 days/yr as a base, from which continuous long term exposure of the general public at 24 hrs/day for 365 days/yr involves a five fold increase in exposure time. Thus by simple extrapolation, the British occupational figure of 50 \(\mu g/m^3\) [quoted above], reduced by a factor of five, would suggest that 10 \(\mu g/m^3\) of mercury vapour in air be the threshold limit for the general population and thus yield the same degree of risk. According to Reinhardt (1988) continuous exposure to 10 \(\mu g/m^3\) mercury vapour would result in a daily intake of 24 \(\mu g\) mercury.

Alternatively, using the WHO (1980) figure of 25 \(\mu g/m^3\) for occupational exposure and applying the same conditions would result in a threshold limit of 5 \(\mu g/m^3\) for the general population. This may be even too high for the variety of health states found in the general populous and Gerstner & Huff (1977) have proposed an arbitrary value of 1 \(\mu g/m^3\) as the level below which mercury vapour in the ambient air poses no health hazards to the general population. This latter figure seems unnecessarily low considering the fact that the normal
daily exposure to mercury in the air and from other sources may be far in excess of this figure and at these levels appears to produce no obvious deleterious effects in the general population. Nevertheless, there is a serious need for the identification of threshold limits for long-term exposure for the general population, with special consideration for pregnant women, young children and the elderly and ill.

The American Agency for Toxic Substances and Disease Registry's acceptable residential indoor air mercury concentration is equal to or less than 0.5 μg/ m³ (Agency for Toxic Substances 1988).

Craig (1986) in a review of mercury biocompatibility states that exposure limits should be treated as tentative because of insufficient data and that there may be a need for separate limits for individuals at high risk and for short term high levels of exposure.

8.3 MERCURY ABSORPTION

From occupational health studies it has been estimated that a time-weighted average air concentration of 50 μg/ m³ Hg (0.05 mg/ m³) corresponds to a concentration of 35 μg/L Hg (3.5 μm/ 100mL; 175 nmol/L; 35 ng/ mL) in blood and 150 μg/L Hg in urine. 50 μg/ m³ Hg in air should be consistent with an uptake of 200-300 μg/ day (Skare & Engqvist 1990).
Mercury vapour at a concentration of 25 μg/m³ corresponds to 20 ng/mL in blood and 75 μg/L in urine (Eley & Cox 1987). Using Gerstner & Huff’s 1 μg/m³ Hg recommended limit for continuous exposure of the general public the corresponding maximums are 4 ng/mL (4 μg/L) for blood and 15 μg/L for urine. The ratio of mercury in air to urine is generally considered to be approximately 1:2-3 (i.e. 50 μg/m³ Hg in air corresponds to 100-150 μg/L Hg urine) although recently ratios as low as 1:1 have been utilised. This latter ratio is closer to that in the WHO Report on Inorganic Mercury (1991) which considers recent exposure data more reliable than those previously quoted and notes that occupational exposure to 40 μg Hg/m³ of air will produce 15-20 μg Hg/L blood and 50 μg/g creatinine for urine.

Table 6 lists published occupational and general population exposure limits for mercury vapour as well as toxic levels.

<table>
<thead>
<tr>
<th>OCCUPATIONAL AND GENERAL POPULATION EXPOSURE LIMITS FOR MERCURY VAPOUR</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOURCE</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>OCCUPATIONAL LIMITS</td>
</tr>
<tr>
<td>WHO (1976)</td>
</tr>
<tr>
<td>ACGIH (1983)</td>
</tr>
<tr>
<td>USA, UNITED KINGDOM</td>
</tr>
<tr>
<td>CASARETT &amp; DOULL</td>
</tr>
<tr>
<td>USSR,SWEDEN</td>
</tr>
<tr>
<td>WHO (1980)</td>
</tr>
<tr>
<td>WHO (1991)</td>
</tr>
<tr>
<td>GENERAL POPULATION LIMITS</td>
</tr>
<tr>
<td>CASARETT &amp; DOULL</td>
</tr>
<tr>
<td>BASED ON WHO (1980-1976)</td>
</tr>
<tr>
<td>GERSTNER &amp; HUFF (1977)</td>
</tr>
<tr>
<td>AGENCY FOR TOXIC SUB (1988)</td>
</tr>
</tbody>
</table>

'NORMAL' Hg LEVEL (Reference) <5 <0.5 <5 5

TOXIC LEVELS |
| WHO (1976) | 100-200 (tremor) |
| BERLIN (1986) | 50 (non-specific) |

Table 6
9. DENTAL AMALGAM

9.1 HISTORY
There are indications that dental amalgam was first used in the early period of the Tang Dynasty in China (618-907 A.D.) and in Germany by Dr Strockerus about 1528. It was introduced into general dental use in the 1830's, originally made by mixing mercury with the filings of silver coins (Bjorklund 1989) and promoted as "royal mineral succedaneum" being an alternative to gold.

9.2 UTILISATION
In industrial countries dental amalgam represents 3% of the total consumption of mercury. Amalgam has been used for some 130 years as a dental restorative material and accounts for some 80% of single tooth restorations. In England and Wales in 1983 25 million amalgams were placed under the National Health System. In America some 160 million amalgams are placed each year and each american dentist in private practice uses approximately 1 kgm of amalgam per year. Eggleston (1989) reports that dentistry in the USA uses over 100 tons of mercury annually.
9.3 COMPOSITION

An amalgam is an alloy of mercury with another metal or metals. Dental amalgam (or silver amalgam) is an alloy of liquid elemental mercury and an alloy powder.

9.3.1 CONVENTIONAL ALLOYS

Conventional alloy powders usually contain less than 6% Copper and the constituents have changed very little over the years.

Traditionally they contain:

Silver 67-74%  Copper 1-6%  Tin 25-27%  Zinc 0-2%

In conventional alloys the particles may be lathe cut or spherical or a blend of both. Silver contributes to the resistance to tarnish of the amalgam. Tin reacts readily with mercury and makes amalgamation of the alloy easier. Copper replaces silver and increases hardness and strength of the amalgam. Zinc acts as a scavenger for oxygen in the fusion of the alloy.

9.3.2 COPPER ENRICHED ALLOYS

A modern type of dental amalgam (copper enriched, high copper) is currently in favour which contains higher amounts of copper (up to 25-30%).

[Much of the material in the following Sections 9.3; 9.4; 9.5 and 9.6 has been taken directly from the text "NOTES ON DENTAL MATERIALS" by E.C. Combes 1986]
This group includes:

a. **Blended Alloys** (dispersion modified) containing two parts conventional lathe cut particles plus one part spheres of silver-copper eutectic alloy.

   Silver 69%  Copper 13%  Tin 17%  Zinc 1%

b. **Single Composition Alloys** which include:

   i. Ternary Alloys (in spherical or spheroidal form)

      Silver 40-60%  Copper 15-30%  Tin 25-30%

   ii. Quaternary Alloys in spheroidal form

      Silver 59%  Copper 13%  Tin 24%  Indium 4%

The modern high copper amalgams should not be confused with "copper amalgams" which were used in paediatric dentistry until the 1950’s. This type of amalgam contained 60-70% mercury and 30-40% copper and was prepared by open heating in the surgery which produced high levels of occupational mercury vapour exposure.

**9.4 MANIPULATION**

Dental amalgam is prepared by mixing approximately similar weights of silver-tin powder with liquid mercury. The resulting plastic mixture is then incrementally packed with hand instruments into a prepared cavity in the tooth under pressure. Mechanical mixing of proportioned alloy and mercury have superseded hand mixing with mortar and pestle.
9.5 SETTING REACTIONS AND STRUCTURE

The amalgam hardens to form a crystalline solid. The bulk of hardening takes place within minutes but amalgam continues to set and strengthen over the next 12-24 hours. Spherical and copper rich amalgams have high early strengths. There is no free elemental mercury in amalgam restorations after setting, but it can be released as mercury vapour or mercuric ion.

9.5.1 CONVENTIONAL ALLOYS

The reaction between alloy (intermetallic silver-tin compound; $\text{Ag}_3\text{Sn}$; gamma phase) and mercury is complex.

On mixing the gamma phase dissolves in mercury with crystal growth of two phases: gamma-1 Phase...$\text{Ag}_2\text{Hg}_3$ (cubic structure) and gamma-2 phase...$\text{Sn}_{7.8}\text{Hg}$ (hexagonal structure)

The set material is a cored structure with a core of unreacted gamma phase and a matrix of gamma-1 and gamma-2. After setting further diffusion processes may occur. The gamma-2 phase is the weakest and softest and as such prone to degradation.

9.5.2. COPPER ENRICHED ALLOYS

The set structure is free of gamma-2 phase which creates improved corrosion and creep properties, increased strength and durability, and reduced release of mercury vapour.

For blended alloys the reaction occurs between a mixture of $\text{Ag}_3\text{Sn}$ plus $\text{Ag-Cu}$ spheres and mercury. In the first stage the reaction is the same as for
conventional alloys with Ag-Cu taking no part. In the second stage gamma-2 component reacts with Ag-Cu spheres, creating a copper-tin compound (Cu$_6$Sn$_5$) and more gamma-1. The Cu$_6$Sn$_5$ is a halo surrounding the Ag-Cu particles. The final set consists of a core of Ag$_3$Sn and Ag-Cu surrounded by a halo of Cu$_6$Sn$_5$ and a matrix of gamma-1.

In single composition alloys the structure of the material is similar to that for blended alloys however the Cu$_6$Sn$_5$ is present in the gamma-1 matrix rather than as a halo.

9.6 TARNISH AND CORROSION

9.6.1 TARNISH

Tarnishing occurs in the presence of sulphur, creating a sulphide layer on the surface of the restorations.

9.6.2 CORROSION

[See Section 11.3 for further discussion of corrosion and mercury release]

9.6.2.1 Conventional Amalgams

The set amalgam is heterogeneous, which encourages corrosion. Gamma-2 Phase is the most active electrochemically, being anodic to both gamma and gamma-1 Phases. In the mouth as gamma-2 corrodes in the presence of saliva, tin products are liberated (SnO$_2$ and Sn(OH)$_6$Cl). Additionally mercury can be released which reacts with remaining unreacted gamma phase.

9.6.2.2 Copper Enriched Amalgams

There is no Gamma-2 phase present, Cu$_6$Sn$_5$ being most liable to corrosion. Corrosion currents and the volume of corrosion products is less than with
conventional amalgams. Minimal or no mercury is released as part of the corrosion process.

9.7 MERCURY HYGIENE

In previous decades amalgam was prepared with a mortar and pestle, the resultant mixture then being placed in a cloth filter and mercury expressed to produce a dry packable consistency. This technique produces mercury vapour and, as well, spillage is difficult to avoid. Current trituration techniques utilise sealed capsules and mixing machines and exposure to mercury vapour is markedly decreased. Modern dental suction systems and spittoons have accessible traps which collect amalgam particles generated during amalgam removal and insertion. There has not been sufficient consideration of adequate design for waste amalgam collection in dental units and operatories and this may constitute an environmental hazard both in the surgery and at sites remote from dental practices.

All accumulated amalgam residue should be stored in a sealed container under x-ray fixer solution, this being far more effective than water in preventing escape of mercury vapour (Yu 1989). Eggleston (1989) notes that dental amalgam is classified as a hazardous material by OSHA and stresses the need for proper disposal of scrap amalgam. The use of a metal recycler using a heat distillation process caused mercury contamination and resulted in successful litigation against 58 dentists in the USA by the Environmental Protection Agency (ADA News 1988).
A number of authors have indicated necessary stratagems for mercury hygiene and for minimising mercury vapour loss in dental surgeries. These include design of clinics (particularly floor coverings and adequately ventilated surgeries); equipment (particularly modern capsulated mixing and effective waste removal); routines (high speed evacuation and water cooling) and regular testing of facilities and staff. (Bauer 1985; Mann, Eisman & Ernest 1986; Jackson 1986; Nilsson & Nilsson 1986; Heydt 1988) More recently there has been advocacy of face masks to be used by dental staff particularly during amalgam removal.

Pohl, Berglund, Bergmann & Olsson (1988) describe a new instrument, combining the functions of a dental mirror and an evacuator which would remove mercury vapour in the breathing zone of patient and dental staff. This system is thought to be particularly useful where no chairside assistance is available to the dentist during amalgam procedures.

An interesting study from Cooley, Stilley & Lubow (1985) measured mercury vapour from steam sterilization of amalgam contaminated instruments. They found mercury vapour levels of 30 µg/m³ from 2 unwrapped amalgam carriers and 270 µg/m³ from unwrapped amalgam cylinders. When placed in paper sterilization bags the levels dropped to 0 and 30 µg/m³ respectively. Mercury was released from the sterilizer when the instruments were removed if bags were not used.
10. ALLERGY TO AMALGAM AND MERCURY

Allergy to amalgam is a rare occurrence perhaps some 40-60 definite cases having been reported in the scientific literature. The frequency of side effects of amalgam placement has been rated at 1 case per million by Donovan and Handlers (1984) and by Kallus (1985) at 0.05%, whereas Vimy & Lorscheider (1988) suggest that somewhere between 3-16% of the general population will demonstrate some form of hypersensitivity to mercury. Of the components of dental amalgam (mercury, silver, tin, copper, zinc) mercury is the element most often implicated as an allergen, although some reports have indicated that the allergen involved may not be mercury (Djerassi & Berova 1969).

The oral mucosa may respond to both primary irritation and allergic sensitisations from dental materials. The buffering and irrigating effect of saliva makes oral mucosa more resistant than skin. Primary irritation is non-specific, caused by many substances, trauma and heat. Allergic reactions are indicative of bioincompatibility due to specific immunologic sensitisation (Cooley & Young 1984).

The majority of documented reactions follow the pattern of delayed hypersensitivity reactions and clinically may be described as allergic contact dermatitis or stomatitis due to skin or mucosal contact with externally encountered allergens. Virtually all cases report a previous sensitisation from either an occupational or medicinal exposure. In the case of mercury allergy it
is difficult to ascertain when initial sensitization takes place but there are perhaps other sensitizers more common than dental amalgam whose status as a sensitizer to mercury is unresolved. These include topical disinfectants (mercuriochrome, merbromin); preservatives in vaccines and contact lens solutions (thimerosal/merthiolate); yellow eye ointment (mercury precipitate); contraceptive jellies (phenylmercuric compounds); red pigments of tattoos (cinnabar/mercury sulphide); and metallic mercury from broken thermometers (Hensen-Pettersen 1989). Merthiolate (thimerosal) is an organic mercury compound which has been implicated as a frequent contact allergen. In 1987/1988 thimerosal was the second most common contact allergen after nickel (Wekkeli, Hippmann, Rosenkranz, Jarisch & Gotz 1990).

A number of distinctions need to be made between:

a. Toxic exposures to mercury in the dental environment relate to mercury hygiene and reflect high levels of mercury vapour in the ambient air. This is seen in dental personnel and is not an allergic response to mercury.

b. traditional allergy/hypersensitivity which may be related to amalgam and mercury, and produces a contact dermatitis or stomatitis.

c. the terms "mercury sensitivity", "mercury hypersensitivity" which are used in an expanded sense to include, as well as local allergic responses, systemic effects (changes in blood pressure, pulse rate, body temperature and white cell count) and general symptoms (headaches, irritability, depression, fatigue). It is claimed by those who condemn amalgam that certain people have a low tolerance for mercury which results in signs and symptoms of mercury poisoning at very low burdens of mercury.
Kaaber 1990 attempts to define risk groups among dental patients for hypersensitivity problems to the use of silver amalgam and acrylic denture base. For silver amalgam the main risk group is patients with contact lesions in the oral mucosa adjacent to the restoration, as this group exhibits a high frequency of skin sensitivity to mercury and other base materials in dental amalgam.

10.1 TESTING FOR ALLERGIC REACTIONS

Testing for suspected allergic reactions to amalgam and mercury should involve referral to specialist allergists or dermatologists. Patch testing is the traditional test for bona fide type IV (cell-mediated, delayed) hypersensitivity. This diagnostic procedure has no relevance for "mercury sensitivity" or "mercury hypersensitivity".

Mercury poisoning or non-allergic hypersensitivity cannot be determined by the same methods as those which detect true allergic responses to mercury (e.g. patch testing). Therefore skin testing is unsuitable for testing to determine whether a wide variety of general symptoms such as depression and gastrointestinal disorders and diseases such as Multiple Sclerosis can be caused by amalgam restorations (Fisher 1985).

Patch testing is complicated by problems with irritational reactions to the vehicle and adhesive, false positives and subjective interpretations (Mackert 1985).

[See Section 13.5.3.]
There is no basis for the use of galvanic current measurements as a diagnostic tool for allergy or toxicity (Renson 1989).

10.2 CLINICAL EFFECTS

There are a variety of clinical manifestations of allergy/hypersensitivity reactions to dental amalgams with the bulk of cases showing remote skin reactions (facial dermatitis or eczema of trunk and extremities) or local skin reactions (redness on proximal mucosa and gingiva, oedema). Oral symptoms include burning sensation, pain, numbness, loss of taste and are usually described as gingivostomatitis.

10.3 TREATMENT

Routine treatment for allergic episodes has been removal of amalgam restorations, but successful resolution has occurred with prophylactic antihistamine therapy without amalgam removal as most of these allergic reactions to amalgam appear to be self-limiting (approximately 2 weeks) (Fung & Molvar 1987).
10.4 IMMUNE RESPONSES

Allergic and autoimmune reactions to chemicals can be distinguished as follows: in allergy the adverse immune response is restricted to the offending exogenous agent present in the tissue. In chemically-induced autoimmunity, by contrast, the adverse immune response is not restricted to the chemical compound inducing it, but involves responses to self-antigens as well. In both allergy and autoimmunity the immune system is stimulated to specific responses that are harmful to the body. As well there are very strong effects of genetic factors predisposing to both allergic and autoimmune reactions to chemicals (Gleichmann, Kimber & Purchase 1989).

There may be in humans (as occurs in some strains of animals) a genetic predisposition to the immune response to mercury. Where a dose/response measurement cannot be utilised for immunologically sensitive individuals, there may be no threshold levels for mercury (e.g. in blood or urine) below which (in individual cases) mercury related symptoms will not occur.

Gleichmann et al (1989) in a discussion of immunotoxicology note that a variety of drugs and environmental chemicals have the potential to impair components of the immune system. There is a growing list of drugs and chemicals which are capable of eliciting autoantibodies and pathological autoimmune reactions. Mercury has been shown to produce an autoimmune syndrome in rodents, a prominent feature of which is glomerulonephritis. This disease has also been documented in humans in cases of mercury poisoning or exposure to mercury
containing drugs and cosmetics. In all these human cases the subjects were exposed to high concentrations of mercury over a short period of time. Autoimmunity in rats caused by mercuric chloride are T cell dependant and there is an excessive activation of T helper cells, which suggests that mercury autoimmunity is similar to Systemic Lupus Erythematosus-like autoimmunity.

A study by Eggleston (1984) analyzed the effect of dental amalgam and nickel alloys on human T lymphocytes. In two patients the removal of amalgam restorations increased the T lymphocytes from 47%-73% and 60%-71%. In one patient the removal of a nickel based alloy increased the T lymphocytes from 56%-77%. The author notes that the T cell balance of helper T lymphocytes and suppressor T lymphocytes is essential for immune homeostasis and variations may predispose to autoimmune diseases such as Systemic Lupus Erythematosis (SLE), Multiple Sclerosis (MS) etc. It cannot be concluded from this study that incidences of allergic responses to amalgam and nickel are the result of T cell variations and the extension to autoimmune diseases being mediated by T cell variations caused by amalgam and nickel is speculative. The variation in T lymphocytes occurring in this study may be explained by other phenomena (e.g. diurnal changes) and may not have any clinical significance as is evidenced by the fact that none of the three patients had any symptoms of allergic or immune response. Since no controls were used the results in such a small group may be coincidental. Additionally intermittent changes in T cell populations occur without heralding specific health problems and do not indicate that cell mediated immunity has been affected (Robinson 1986).
10.5 ORAL LICHENOID LESIONS

Oral lichen planus is a common chronic disease of unknown aetiology with a prevalence of approximately 2% of the adult population. Lichenoid lesions are most often located at the buccal mucosa and on the lateral border of the tongue (Bowleska, Holmstrup, Möller-Madsen, Kenrad & Danscher 1990b). It is considered a cell-mediated type of immune response with the majority of cells being T lymphocytes and an increased number of Langerhans cells. Oral lichen planus and lichenoid lesions have been suggested as caused or aggravated by contact with amalgam restorations and may represent a contact hypersensitivity to mercury (Finne, Goransson & Winckler 1982; Jolly, Moule & Freeman 1986; James, Ferguson, Forsyth, Tulloch & Lamey 1987; Stenman & Bergman 1989).

Oral Lichen Planus has also been associated with diabetes, hypertension, immunologic disorders as well as drugs such as methyldopa, beta blockers and antiphlogistics (Lind, Hurlen, Lyberg & Aas 1986). Other aetiopathological factors which have been suggested for oral lichen planus include bacterial, fungal and viral infections, local trauma, mental stress, galvanic phenomena, betel chewing and immunological defects (Finne et al 1982). Frykholm, Frithiof, Fernstrom, Moberger, Blohm & Bjorn (1969) demonstrated a case in which allergy to copper from dental alloys was suggested as a possible cause of lichen planus.

There is the possibility is that substances liberated from dental restorative materials can serve as haptens, which on adsorption and binding to epithelial elements can form complete antigens capable of eliciting delayed hypersensitivity
reactions (Hensen-Pettersen 1989). However, surface roughness of amalgam may prove a significant factor as an irritant. Mercury hypersensitivity has been reported to occur in 16-62% of patients with oral lichen planus, but there are conflicting opinions in the literature (see below). It is of interest that composites (tooth coloured synthetic resins) have also been linked to the same oral lichenoid condition which may relate to the liberation of formaldehyde (Lind 1988).

Bolewska & Reibel (1989) evaluated the presence of T lymphocytes, Langerhans Cells and HLA-DR expression on keratinocytes in oral lesions in contact with amalgam restorations and in those patients with no dental restorations at all. The findings did not differ between the groups and the authors conclude that the pattern observed is a common reaction of the oral mucosa to known (amalgam restorations) and unknown factors.

Eversole & Ringer (1984) found that 21% of 24 patients with oral lichen planus exhibited positive skin responses in patch tests to dental restorative materials and selected metallic salts. This compared with an 8% response by the control group. The authors conclude that oral lichen planus patients show a higher correlation with delayed hypersensitivity to dental materials than a control population: however, a cause and effect relationship cannot be substantiated.

James et al (1987) patch tested 29 consecutive patients with lichen planus for the range of metals contained in dental amalgam. Allergic reactions to mercury were found in 10 of the 29 (34%) and all of these had old corroding amalgams,
presumably releasing mercury ions. Six patients had the amalgams replaced with alternative materials which resulted in resolution of the ulcerated areas. In the follow-up period one patient had recurrence of the oral lesions and another persistent discomfort despite resolution.

Lind, Hurlen, Lyberg & Aas (1986) studied 52 patients with oral lichen planus related to amalgam restorations. In 18 patients replacement of amalgam caused complete remission in 16 patients within 1-12 months.

A study by Bolewska, Hansen, Holmstrup et al (1990a) of 49 patients with lesions of the oral mucosa in contact with corroding dental amalgams found that those patients with lesions restricted to the close proximity of the amalgam showed a greater proportion of positive reactions to mercury. Replacement of amalgam or prevention of contact with the lesion produced greater regression of the lesions in this group. The authors concluded that contact allergy to mercury was a possible aetiologic factor for the mucosal changes in this group. The group of patients with lesions extending beyond the area of contact with amalgam were considered unrelated to mercury and the authors suggest that causes such as lichen planus should be considered.

A different result was found by Hietanen, Pihlman, Forstrom, Linder & Reunala (1987) who studied 12 patients with oral lichen planus suspected of dental metal allergy. Only 1 patient of the 12 reacted positively to mercury compounds on a patch test but this was not confirmed in further testing nor did X-ray microanalysis show any contaminating metals in the lesion. As well no mercury
allergy was found in a further reference group of 17 patients suspected of dental restorative material allergy but with no oral lichenoid lesions. Bowleska et al (1990b) in a further study of 43 patients claim to show: 'for the first time 1.) that mercury is taken up by the lesioned oral mucosal membrane, 2.) that under certain, at present unknown, conditions mercury can also penetrate the intact oral mucosa without causing clinical or histopathologic changes.'

10.6 RESPONSES TO AMALGAM AND DENTAL MATERIALS

Stenman & Bergman (1989) studied 151 patients who were subjected to epicutaneous testing for possible side-effects of dental materials. Thirty nine women and seven men had positive skin reactions, the majority being related to nickel, although a number of positive reactions were noted to organic materials. Twelve patients with positive skin reactions to mercury salts all had amalgam restorations and oral mucosal changes were present in five of these. The authors note that while some of the complaints could be explained by hypersensitivity to dental materials, they also strongly emphasise that other explanations have to be considered.

A report of a case of allergy to an amalgam filling which manifested as a burning mouth was reported by James, Ferguson & Forsyth (1985). The symptoms of burning mouth and metallic taste occurred two days after the insertion of an amalgam and persisted for some 5 months being refractory to treatment. Medical history and blood tests were normal, patch testing showed an allergy to ammoniated mercury and elemental mercury. There was no
evidence of previous sensitisation to mercury from occupational or medical exposure. The particular amalgam was removed and replaced with a composite material. Symptoms resolved within 2 weeks and remained symptom free for 18 months subsequently.

The cause of the previous case is disputed by Albert (1986) who queries the clinical manifestations and particularly the fact that the patient had other amalgams which appeared not to cause an allergic response.

A case of occupational allergic contact dermatitis from metallic mercury is reported by Goh & Ng (1988) in which a 45 year old female dental nurse developed chronic vesicular eczema of the thumb and forefinger. The technique of applying soft newly mixed amalgam into dental cavities involved the use of a cotton roll for levelling of amalgam restorations and the subsequent re-rolling of this mercury contaminated cotton roll between the affected fingers. The eczema cleared with the use of topical steroids and a change of technique. This incident, while being caused by a non-standard technique does, however, highlight the potential for allergic responses to mercury in dental staff as well as the patients who receive the amalgam restorations.
11. DENTAL AMALGAM AND MERCURY LOSS

Amalgam cannot be described as an inert substance. There is a dynamic state in the mouth whereby with time and function there is change in the structure of the amalgam and ions are released into saliva, oral air and dental tissues. These changes have been recorded in a multitude of studies, but it is the differing interpretations and significance attributed to the released material (particularly mercury) which evokes so much comment and controversy, and which still requires further investigation, quantification and qualification.

Metallic ions (corrosion products) from restorations, crowns, and denture clasps have been shown to flow into enamel and dentine (Soremark, Wing, Olsson & Goldin 1968). In enamel and dentine surrounding amalgam restorations there were marked increases in silver and mercury and moderate increases of zinc.

The release from amalgam of metals which have toxic properties has an antibacterial effect. A study by Orstavik (1985) on the inhibitive effects of a number of amalgams on streptococcus mutans (which is implicated in cariogenic plaque) showed antibacterial effects of mercury, silver and copper.

The cytotoxicity of dental amalgam has been investigated, mainly in vitro and the results seem to indicate that zinc and copper are more cytotoxic than mercury. The modern non-gamma 2 amalgams with high copper content release greater numbers of cytotoxic copper ions.
There is evidence that mercury is released from amalgam restorations during insertion, setting, polishing and removal. [see Section 11.1] Additionally a number of researchers have recorded mercury vapour in the expired air of patients with amalgam restorations with increases following mastication and tooth brushing (Svare, Peterson, Reinhardt, Boyer, Frank, Gay & Cox 1981; Patterson, Weissberg & Dennison 1985; Vimy & Lorscheider 1985a, 1985b; Berglund, Pohl, Olsson & Bergman 1988).

[See Section 11.2]

In an in vitro study by Marek (1990) the method of mercury release from dental amalgam restorations has been described as conforming to a dissolution/evaporation model. In vitro experiments have demonstrated that corrosion of amalgam releases mercury vapour but this has not been confirmed in vivo.

The status of saliva in the release of mercury from amalgam has yet to be fully clarified. Saliva serves many functions in the oral cavity and its buffering action has been postulated as a stabilizing factor in the potential chemical activities that might and do occur in the mouth. In teeth covered with saliva, part or all of the mercury released from amalgam may immediately pass from the vapour form into an ionized form, and this being the least toxic form of mercury is, if ingested, poorly absorbed from the gastrointestinal tract.
Olsson, Berglund, Pohl, Bergman (1989) describe a model of the manner in which mercury vapour is transported from amalgam restorations. Released mercury atoms pass through the saliva and are distributed partly to the gas phase, from which they are respired to the lungs and environment, and partly to the saliva and gastrointestinal tract by swallowing.

11.1 INSERTION AND REMOVAL OF AMALGAM

Mercury vapour is to some degree released during insertion, condensation and carving of amalgam and subsequently during setting. During removal of amalgam both mercury vapour and mercury dust containing fine amalgam particles are released (Reinhardt, Boyer, Svare et al 1983; Reinhardt, Chan & Schulein 1983).

Raised mercury levels fall rapidly on completion of both insertion and removal procedures. With the use of appropriate dental procedures, levels of mercury vapour were below 15 μg/m³ and from 30-60 μg/m³ for particulate matter. These levels are well below the WHO values for short-term exposure of 500 μg/m³. Mercury levels in urine are increased after insertion of amalgams, but fall to original levels after some seven days (Frykholm reported in Snapp, Boyer, Peterson & Svare 1989).

In Snapp et al (1989) the method of amalgam removal was not controlled but removing an average of 14 surfaces of amalgam at one sitting produced in the blood stream an additional exposure of 1.46 ng Hg/mL (1.46 μg/L) that was rapidly cleared from the blood with a half-time of 2.9 days.
A study by Olstad, Holland & Pettersen (1990) on nine children who had a single session of amalgam placement found no effect on the urine mercury concentration. They determined that: 'Conclusively, one single session of amalgam treatment did not per se represent a mercury exposure of sufficient quantity to be detectable in a longitudinal, individual study.'

Ott, Vogler, Kroncke et al (1989) studied the effects of placement of non-gamma 2 amalgam restorations in 45 subjects all of whom had amalgam fillings and were not occupationally exposed to mercury. Blood and urine values were below normal upper limits prior to placement of the restorations and showed no increase in the 24 hours after placement.

Richard & Warren (1985) found that removal of old amalgam could create mercury vapour concentrations in the breathing zone of dental operators up to the threshold limit for mercury (0.05 mg/m³; 50 μg/m³).

Haikel, Gasser, Salek & Voegel (1990) measured the levels of mercury vapour in the oral cavity during removal, setting and polishing of amalgam. All procedures released mercury, the mean levels being between 85 and 326 μg/m³. The authors found a significant correlation between the mercury vapour concentrations and the size of the amalgams in each of the procedures. Water coolant during amalgam polishing made no difference to the level of mercury vapour released.
Molin, Bergman, Marklund, Schutz & Skerfving (1990) measured blood and urinary mercury levels of 10 healthy persons whose amalgam restorations were replaced with gold inlays. Immediately after removal plasma mercury rose 3-4 fold, whereas urinary and erythrocyte mercury rose approximately 50%. Twelve months after removal of amalgams, plasma and urinary mercury levels were significantly reduced to 50% and 25%, respectively, in the experimental group. Although amalgam fillings contributed to the plasma and urinary mercury levels, a large number of supplementary biochemical analyses did not show influence on organ functions or any differences between the groups before and after amalgam removal.

There is perhaps the possibility that certain additives to amalgam may reduce the amount of mercury vapour released during insertion, setting, function and removal. In a study by Powell, Johnson & Bales (1989) adding 8-14% indium to the alloy powder significantly reduced the amount of mercury vapour released from dental amalgam, particularly during the setting phase. Others have indicated that gallium amalgams may offer an alternative to mercury amalgam. These have similar powder constituents with the inclusion of palladium and indium, but gallium replaces mercury. More development and research is necessary to assess suitability and ensure biocompatibility.

A case study reported by Taskinen (1989) outlines a possible case of mercury toxicity with multiple symptoms paralleling those of micromercurialism as a consequence of multiple grinding and removal of dental restorations.
Thus, dental procedures involving insertion and removal of amalgam, being of short duration and infrequent occurrence, are not of any major clinical significance, provided appropriate modern methods are observed. This would require the use of water cooled drilling, high volume aspiration (both currently routinely used in modern dental surgeries) and perhaps rubber dam. This can dramatically reduce, if not virtually eliminate, mercury thus liberated.

This was demonstrated in a study by Nimmo, Werley, Martin & Tansy (1990) who noted that an aerosol containing amalgam particles is created when a high speed handpiece is used to remove amalgam restorations. Particles smaller than 10 μ are considered fully respirable and may become lodged in the terminal alveoli and may compromise respiratory function if the exposure is long-term. Water spray with high velocity suction significantly reduced exposure to particulate matter compared to dry cut amalgam and this was again notably reduced with the added use of rubber dam to the water spray and high velocity suction. The authors, however, noted that: 'the dentist 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'.
11.2 MERCURY VAPOUR AND RESTORATIONS IN SITU

11.2.1 MERCURY VAPOUR FROM AMALGAM

Mercury vapour can be measured in the expired air of patients with and without amalgam restorations. A number of studies have shown that persons with amalgam restorations have higher levels of mercury vapour than those without, and levels in the former rise with chewing and brushing, while negligible changes occur in the levels of those with no amalgams. The studies vary greatly in their methodology, results and interpretations and the significance of the data is yet to be unequivocally established.

Svare et al (1981) reported that for an amalgamless group of 8 patients the mean mercury level in expired air was 0.26 $\mu$g/m$^3$ before chewing and 0.13 $\mu$g/m$^3$ after 10 minutes vigorous chewing. The mean level of the amalgam group of 40 patients was 0.88 $\mu$g/m$^3$ before chewing and 13.74 $\mu$g/m$^3$ after chewing. Svare concluded that the level of mercury vapour was higher with those patients having amalgam restorations; increased an average of 15.6 fold after chewing in the amalgam group while remaining unchanged in those without amalgams and appeared to increase in relation to the number of amalgam restorations in the mouth.

Vimy and Lorscheider (1985a) noted mean mercury levels of 0.54 $\mu$g/m$^3$ in the unstimulated amalgamless group (11 patients) and 0.72 $\mu$g/m$^3$ after chewing. The amalgam group (24 patients) had a mean prechewing level of 4.91 $\mu$g/m$^3$ and mean post-chewing level of 29.1 $\mu$g/m$^3$. A further article by the same
authors (1985b) measured mercury concentrations in intra-oral air after chewing. The calculated average daily dose of mercury was approximately 20 μg, subjects with more than 12 occlusal amalgam surfaces receiving 29 μg and those with less than 4 occlusal amalgam surfaces receiving 8 μg. Vimy and Lorscheider (1986) estimated that given continuous exposure to elemental mercury vapour from dental amalgam at 30 μg/day, the CNS could accumulate a substantial amount of mercury over extended time.

Patterson et al (1985) studied the effects of toothbrushing on 172 people with amalgam restorations finding mean mercury vapour levels of 8.2 μg/m³ after brushing compared with 3.1 μg/m³ before brushing. The mean mercury level of 5 subjects without amalgam restorations was 0.06 μg/m³. The enhanced values obtained by brushing the teeth decreased slowly over the following hour to approximately one third of the peak value.

Berglund et al (1988) studied the method of collection and analysis of mercury vapour and concluded that, on the basis of experimental and theoretical considerations, the amount of mercury released from the oral cavity was time-dependent. On seven subjects with nine or more amalgam restorations the rate of mercury release was 0.03-0.34 ng/sec and <0.01 ng/sec on three subjects with no amalgams. Berglund recalculated the results of Vimy and Lorscheider giving levels of 0.06 ng/sec (originally 4.91 μg/m³ for the unstimulated group) and 0.36 ng/sec (originally 29.1 μg/m³ after stimulation).
Mackert (1987) re-examined the daily dose estimations from amalgam performed by Vimy and Lorscheider (1985a) and stated: 'Calculation of the mercury vaporizing rates responsible for the mercury vapour concentrations previously reported enabled the daily dose of mercury to be estimated for subjects with various amalgam restorations. The corrected estimates for daily dose of mercury from amalgam restorations are a factor of sixteen lower than those previously reported.'

In a similar vein Olsson and Bergman (1987) and Wallis, Kaiser & Menke (1986,1988) argue that the results of Vimy and Lorscheider (1985a,b) in measuring the level of intra-oral mercury vapour are erroneous and 16 times too high. The WHO Report on Inorganic Mercury (1991) questions the accuracy of the gold amalgamation technique used by Vimy and Lorscheider (1985), and Svare et al (1981) to evaluate the release of elemental mercury vapour in the oral cavity.

Hume (Dean of the Faculty of Dentistry, Sydney University)(1989) used the results of Berglund et al (1988) to calculate the relative exposure of patients with amalgams compared to industrial standards and the dental work environment. Utilising the maximum stimulated levels (0.3 ng/sec) of mercury vapour released into the oral air of those with multiple amalgam restorations, and assuming a worst case scenario where the patient is a mouth-breather twenty four hours per day and additionally that all mercury vapour is inspired, the rate of mercury inflow is 9 ng/ min.
As comparisons Hume uses:

a. the British environmental safety limit for mercury vapour is 50 \( \mu g/m^3 \) giving a rate of inflow of 300 ng/min and

b. the levels of mercury in the ambient air in a dental hospital environment as measured by Wilson and Wilson (1985) which rate at a level of 30 ng/min.

Thus Hume concludes that: 'a total mouth breather with a mouth heavily restored with dental amalgam would suffer a mercury inflow 3% of that deemed to be acceptable as an occupational safety standard and 30% of that encountered by individuals in a dental working environment. Since in the great majority of individuals the proportion of nasal to oral inflow during breathing is large, the mercury inflow from amalgam restorations would be correspondingly smaller than that calculated above. However it must be acknowledged that from the data referred to some mercury, albeit at levels well below those accepted as safe, is likely to enter the body from dental amalgam restorations by the respiratory route when mouth breathing occurs'.

It should be noted that the WHO Industrial limit (1980) is 25 \( \mu g/m^3 \) of mercury vapour and therefore the occupational threshold value calculated by Hume of 300 ng/min should be halved to 150 ng/min. Thus the individual would inspire approx 6% of the industrial limit. Further if one accepts that 5 \( \mu g/m^3 \) (equivalent to 30 ng/min) is a suitable upper limit for continuous exposure for the general public, then the 9 ng/min calculated by Hume is well below this level. However, a realistic assessment of the proportion of mercury vapour
actually inspired would be at least 50% or less than the worst case scenario utilised for the calculations. If the higher stimulated level occurs after mastication and toothbrushing and lasts a maximum of 2-3 hours before returning to prestimulated levels, then only approx 12 hours/day could be rated at the higher amount. Additionally total mouth breathing is unusual and the average person breathing nasally would inspire only a small proportion of the contaminated oral air.

Langworth, Kolbeck and Akesson (1988) measured tracheal levels of mercury vapour and found that the tracheal levels were considerably lower than intra-oral levels. They note that: *the low mercury levels in the trachea are due to dilution of the small volume of intra-oral air (40-50 ml) containing mercury vapour with more than ten times greater volume of inhaled air with a very low content of mercury. There may also exist some degree of mercury binding and inactivation in the mucous membranes of the airways*. On a daily basis they estimate 2 $\mu$g/m$^3$ is absorbed during 4 hrs of stimulated conditions and 0.4 $\mu$g/m$^3$ from 20 hours unstimulated. Using a 50% factor for nose/oral breathing and an alveolar mercury absorption factor of 80% they calculate the daily dose of mercury from dental amalgams to be approximately 3 $\mu$g. The authors consider that the results of Vimy and Lorscheider (15-30 $\mu$g/day) are based on erroneous assumptions.

Berglund (1990) in a continuation of his work on intra-oral mercury vapour studied 15 subjects over a 24 hour period and estimated the daily dose of mercury from dental amalgam to be 1.7 $\mu$g. The daily release of mercury from
amalgam was corrected for retention of inspired mercury vapour (80%), for inspiration/expiration ratio (50%) and for nose/mouth air flow proportions.

At rest (deemed to be 8 hours) there is 0.4% oral respiration; during conversation (a further 8 hours) there is 58% oral respiration; and during sleep (8 hours) there is 17% oral respiration.

*Many of the results contradicted earlier studies:*

i. The daily dose of inhaled mercury vapour was not significantly related to either the occlusal, or the total number, or the area of the amalgam surfaces.

ii. With the exception of breakfast, ordinary meals caused no significant increase in release of mercury vapour from amalgams.

iii. Hot drinks caused no increase in mercury release.

It was confirmed that toothbrushing did cause a significant increase in mercury vapour release. Berglund calculates that an occupational threshold limit value (TLV) of 50 μg/m³ of mercury in air reflects an inhaled daily dose 300-500 μg/day. The daily dose from amalgam (1.7 μg) represents approximately 1% of this total.

Ultimately there is still confusion and controversy as to the concentration of mercury in intra-oral air and the inspired dose of mercury vapour.
11.2.2 Blood and Urine Mercury from Amalgam Restorations

There is continuing controversy over the actuality and the degree to which mercury from amalgam is reflected in blood and urine. People with amalgam restorations have been reported to have a blood mercury of 0.6-1.9 ng/mL, which is well below maximum levels and in many cases differs little from controls. Nevertheless a number of studies have found a relationship between the total number of amalgams (or total number of amalgam surfaces, or total surface area of amalgams) and urine (and to a lesser degree plasma) mercury levels (Svare et al 1981; Nilsson & Nilsson 1986; Olstad, Holland, Wandel & Pettersen 1987; Langworth, Kolbeck & Akesson 1988; Molin et al 1990). There is however, no scientific evidence that the increased mercury levels in blood and urine from amalgam restorations is at a toxic level nor that it produces any deleterious effects on organ or health states.

Abraham, Svare and Frank (1984) measured mercury levels in blood and mouth air before and after chewing in 47 persons with and 14 persons without dental amalgams. They concluded that blood mercury concentrations were higher in subjects with amalgams (0.7 ng/mL; 0.7 µg/L) than without (0.3 ng/mL; 0.3 µg/L) and attributed this to the presence of dental amalgams, hypothesizing that mercury volatilized from the amalgam surface is inhaled and reaches the blood via pulmonary absorption. Other studies have failed to show a correlation between blood mercury concentrations and the number of amalgam restorations (Kroncke, Ott, Petschelt, Schaller, Szecsi & Valentin 1980; Ott & Kroncke 1981).
Svare (1984) reports a study wherein on one female patient the blood levels after amalgam removal were 10 times lower than the average pre-removal level at 214 days.

Forsten (1989) contradicts previous reports of increased blood mercury levels after mastication. After chewing paraffin for 30-60 minutes blood was analyzed from 35 patients, each with at least 10 large restorations. Total blood mercury content of all patients was <35 nmol/L (7 μg/L; 7 ng/mL) [which the author compares to a figure of 175 nmol/L (35 μg/L; 35 ng/mL) in blood - equates to an occupational limit of 50 μg/m³ of mercury in air]. Additionally, the inorganic component of blood mercury, wherein the effects of elemental mercury would be represented, was 5-10 nmol/L (1-2 μg/L; 1-2 ng/mL) for 32 of the patients and 15-20 nmol/L (3-4 μg/L; 3-4 ng/mL) for 3 patients.

It should be noted that using the lower alternative of 5 μg/m³ of mercury in air as a threshold for the general population equates to 17.5 nmol/L (3.5 μg/L; 3.5 ng/mL) blood level and thus for total blood mercury, half of the patients in this study exceeded this level. Nevertheless, in 32 out of 35 patients the inorganic component, which would include a reflection of absorbed mercury vapour, was below this threshold level.

A study by Snapp et al (1989) determined the exposure to mercury from dental amalgams by comparison of blood levels of mercury before and after removal of all amalgams from ten subjects. The mean baseline blood level of mercury was 2.18 ng/mL Hg (2.18 μg/L) prior to removal of amalgams, which had a
closer linear relationship to the number of occlusal surfaces than total surfaces of amalgam restorations [average 14 surfaces, 7 of which were occlusal]. The mean decrease in blood mercury after amalgam removal was 1.13 ng/mL (1.13 µg/L), nine of the ten subjects exhibiting a statistically significant decrease in blood mercury. The half-time for the elimination of mercury from the blood after amalgam removal was 30.2 days, with an additional exposure of 1.46 ng/mL (1.46 µg/L) actually due to the physical removal of amalgam, which was rapidly cleared from the blood with a half-time of 2.9 days. The daily intake of mercury from the subjects was estimated to be at least 1.3 µg.

Fung, Molvar, Strom, Schneider and Carlson (1990) placed up to 8 new amalgams in 24 patients and analyzed blood and urine at intervals from 2 days to 12 months. Neither mercury nor organic mercury could be detected at equal to or greater than 20 ng/mL in the blood or urine. The authors state: 'Levels of mercury or methyl mercury equal to or less than 20 ng/mL are considered safe in blood or urine. Thus, in this study, patients were not subjected to unsafe levels of mercury from old restorations or after the placement of new ones.'

The detection limit of 20 ng/mL in this study seems nowhere near as sensitive as the majority of other studies quoted. The claim of safety relies on the relationship of the figures with occupational safety thresholds which may not be appropriate guidelines for general population conditions.

Olstad et al (1987) studied 73 schoolchildren and found a positive correlation between urine mercury (mean value 0.58 nmol/mmol creatinine) and extent of
amalgam restorations. They concluded that these levels were below any value of toxicological significance, the Institute of Occupational Health in Norway considering 10 nmol Hg/mmol creatinine as the threshold limit for urine mercury concentration in the Norwegian population without occupational exposure to mercury. Additionally they could find no correlation between the levels of mercury in urine and days of absence from school due to illness, resolving that the actual dosage of mercury caused by the amalgam is too small to exert any adverse effects on the patients.

Langworth, Elinder and Akesson (1988) confirm the significance of the relationship between urinary excretion of mercury and the number of amalgam surfaces.

Eggleston and Nylander (1987) show a positive correlation between the number of occlusal surfaces of amalgam and mercury levels in the brain. The authors acknowledge that total mercury values were used because of the: "bi-directional conversion between inorganic and organic mercury in humans'. This factor allows for confounding due to the presence of organic mercury and, as well, inorganic mercury demethylated from original organic exposure. The authors note that the amount of mercury in human brain tissue may not be clinically significant, but they suggest that dental amalgam exposure should be considered in monitoring sources of mercury accumulation in the human brain tissue.
Nylander, Friberg and Lind (1987) studied 34 cadavers and noted an association between the number of tooth surfaces containing amalgam and concentration of mercury in the occipital lobe of the brain and the renal cortex. In 6 cases analysis of total mercury revealed inorganic mercury assuming a mean proportion of 77%. The authors conclude that the cause of the association between amalgam load and accumulation of mercury in tissues is the release of mercury vapour from amalgam fillings, but do acknowledge the possibility that demethylation of organic mercury could also be contributory.

The WHO Report on Inorganic Mercury (1991) notes that from autopsies on subjects not occupationally exposed, a moderate number (25) of amalgam surfaces may increase the brain mercury concentration by 10 μg/kg.

In the Internal Technical Report of an International Programme on Chemical Safety 1988 (WHO 1988) the estimated average uptake of mercury and steady state contribution to blood, urine, brain and kidney from amalgam fillings in four different studies are presented from Clarkson, Friberg, Hursh and Nylander (1988). [Table 7]

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>EXPOSED TO/UPTAKE</th>
<th>BLOOD</th>
<th>URINE</th>
<th>BRAIN</th>
<th>KIDNEY</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>μg Hg/day</td>
<td>μg Hg/L</td>
<td>μg Hg/L</td>
<td>μg Hg/kg</td>
<td>μg Hg/kg</td>
</tr>
<tr>
<td>Svare et al (1981)</td>
<td>17.5</td>
<td>1.75</td>
<td>5.3</td>
<td>29</td>
<td>1563</td>
</tr>
<tr>
<td>Abraham et al (1984)</td>
<td>8.0</td>
<td>0.77</td>
<td>2.4</td>
<td>12</td>
<td>714</td>
</tr>
<tr>
<td>Patterson et al (1985)</td>
<td>2.5</td>
<td>0.25</td>
<td>0.7</td>
<td>4</td>
<td>224</td>
</tr>
<tr>
<td>Vimy &amp; Lorscheider (1985)</td>
<td>2.9</td>
<td>0.29</td>
<td>0.9</td>
<td>4.3</td>
<td>259</td>
</tr>
</tbody>
</table>

Table 7 Source: WHO (1988)
The study by Snapp (quoted above) shows a blood level of 1.13 µg Hg/L due to amalgam restorations and calculates the daily intake by division of the steady-state blood concentration by the coefficient 0.866, resulting in a daily intake value of 1.3 µg/day. Snapp quotes the work of Mackert (1987) who calculated a daily dose of 1.24 µg/day from the data of Vimy and Lorscheider.

This calculation from blood to uptake level differs from the method of Clarkson et al (1988) where blood level is calculated as 10% of the uptake figure. Snapp states: 'Inspired mercury vapour, on other hand, is highly absorbed, with 80% retained [Nielsen-Kudsk, 1965], and 10% appears in the total blood volume [Cherian et al, 1978].' On this basis the blood level of 1.3 µg/L would represent 10% of 11.3 µg Hg taken up which is 80% of 14.13 µg Hg inspired.

WHO (1988) states that: 'As judged from urinary concentrations of mercury, average exposure levels from dental amalgam fillings among the general population are well below average exposure levels associated with effects among occupationally exposed individuals. The individual spread is considerable, however.'

11.2.3 Animal Studies
Animal studies are important in assessing potential toxic materials and dosage in man. Nevertheless, the data from experimental animal research should not, in isolation, be given undue significance for man. The study may not duplicate human exposure parameters and nor may the animal behave like or physiologically parallel the human counterpart.
Using whole-body image scan and tissue analysis Hahn, Kloiber, Vimy, Takahashi and Lorscheider (1989) investigated the uptake routes and distribution of mercury from amalgam fillings placed in sheep. The mercury isotope appeared in various organs and tissues within 29 days. There were three uptake sites: lung, gastrointestinal and jaw tissue, and once absorbed high concentrations of mercury localized in kidneys and liver. A further study by the same group of authors (Vimy, Takahashi & Lorscheider 1990) establishes a time-course distribution for amalgam mercury in body tissues of adult and fetal sheep. Mercury from dental amalgam appears in maternal and foetal blood and amniotic fluid within 2 days after placement, while within the same period excretion has commenced. In the adult sheep the highest concentrations of mercury occurred in kidney and liver, whereas for the foetus the highest amalgam mercury concentrations appeared in the liver and pituitary gland. The authors conclude that: 'accumulation of amalgam mercury 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 considered.'

Dodes in a letter to the editor in the FASEB Journal (1990) argues that the findings of Hahn et al (1989) and Vimy et al (1990) are false. He points out that (contrary to the view of Hahn et al 1989) there is clear experimental evidence of the safety of silver amalgam, pointing to the Hahn’s dismissal of the fact that American dentists have no higher an incidence of morbidity or mortality despite a body burden of mercury 3-15 times that of the general
population. Dodes notes that dentists should be the first to exhibit symptoms of mercury release and that the anti-amalgam lobby has never been able to tie any disease to the release of mercury from silver fillings. He claims the research of Hahn is faulty due to the method of mastication, rumination and digestion of the sheep used for the experiment being a poor model for human dental extrapolation.

A further study by Hahn, Kloiber, Leininger, Vimy and Lorscheider (1990) demonstrates the bodily distribution of amalgam mercury in a monkey whose dentition, diet, feeding regimen and chewing pattern parallel those of humans. When amalgam fillings incorporating radioactive isotopes are placed in monkey teeth, the isotope appears in high concentration in various organs and tissues within 4 weeks. Highest levels of mercury were located in the kidney, gastrointestinal tract and jaw. The authors resolve that: 'the dental profession’s advocacy of silver amalgam as a stable tooth restorative material is not supported by these findings.'

A study by Danscher, Horsted-Bindslev and Runby (1990) placed occlusal amalgams on teeth and amalgam implants in maxillary bone of 3 monkeys. After one year they found deposition of mercury in spinal ganglia, anterior pituitary, adrenal medulla, liver, kidneys, lungs and intestinal lymph glands. The authors conclude that the results strongly support the view that: 'dental fillings in primates cause absorption of mercury released from amalgam fillings through lungs and intestinal tract and that depending on exposure mercury is distributed to
most organs and will eventually be found in the central nervous system.'

Nilner, Akerman and Klinge (1985) studied both humans and beagles to assess the mercury burden of nerve tissue. In the beagles amalgam restorations were placed to assess their influence on the mercury content of the nervous tissue. The mercury content in man and dogs differed widely from one nerve to another, with no apparent relation to the number, type or location of tooth restorations.

Vimy, Boyd, Hooper and Lorscheider (1990) claim decreased kidney function due to glomerular filtration impairment in six sheep, each of which had placed 12 occlusal amalgam fillings.

Truono (President, American Dental Association) (1991) criticizes this research in that it was not a peer-reviewed article and the sample size 'absurdly small'. Additionally the reported values appear contradictory (if kidney function were reduced by 50% then blood urea should have been elevated not decreased). Also (continues Truono) the choice of sheep for the Calgary study as a model is flawed. The sheep being a ruminant animal, chewing for two thirds of the day on a coarse diet, and having all amalgams placed in one visit 'would not provide reliable information on the effects of amalgam in humans'.

Summers, Vimy & Lorscheider (1990) (this being another study by the Calgary Group) placed, in each of two monkeys, 16 occlusal amalgams under general anaesthesia. The authors claim that: 'ingested mercury is sufficiently bio-available
to select for a substantial increase in the proportion of mercury resistant bacteria in both the oral cavity and intestine. It is then argued that: 'mercury resistant bacteria can convert Hg(II) or methyl-Hg(I) to volatile, lipid soluble Hg(O), the increased incidence of such bacteria in flora being able to influence the pharmacodynamics and toxicity of ingested mercury from dental amalgam.'

This study suffers from the same problem as Vimy et al (1990) in that a sample size of 2 has limited scientific value. The bulk of studies which involve the University of Calgary and specifically the research group led by Vimy & Lorscheider have consistently argued that it is the mercury vapour released from amalgam which, due to its high percentage uptake through inhalation, is potentially toxic. The opening line of the abstract for this paper states: 'Mercury vapor is continuously released from silver amalgam fillings in humans.' This paper however, tenders an argument for the effects of ingested mercury (which would be in the less toxic, inorganic compound form). The environmental propensity of mercury resistant bacteria in soil and water may not be applicable in man (exactly as is the case of methylation of inorganic mercury which occurs in the environment but not in humans). The claim that bacteria can convert inorganic mercury and methylmercury to mercury vapour in the human oral cavity and intestine appears rash and no other studies have made similar observations.
11.3 CORROSION AND MERCURY RELEASE

All dental alloys and particularly amalgam are inherently susceptible to corrosion due to their heterogeneous structure. Corrosion behaviour of structure such as amalgam depends upon both the corrosion properties of the individual phases and the electrochemical interaction between them and the oral environment (Palaghias 1985).

The majority of the studies on corrosion of amalgam and release of ions such as mercury and copper have been carried out in vitro with results having a questionable relationship to actual in vivo conditions. Palaghias (1985) suggests that saliva has protective and inhibitive properties to hinder metal dissolution. Buffer systems and some organic compounds offer sufficient protection against the corrosion of dental alloys for the majority of patients. An in vitro study by Moberg (1985) concluded that corrosion products, especially copper and mercury, released from dental alloys can probably reach local concentrations in the oral mucous membrane great enough to influence the excitable tissue. In other parts of the body the concentrations of active corrosion products are probably too low to exert such actions. This might explain the rare hypersensitive or local allergic reactions as distinct from proposed non-allergic systemic responses and disease states for which there is little clinical or documented evidence.
Traditionally corrosion of amalgam referred to particles of amalgam and included mercuric ions and compounds which were released into the oral environment and ingested with saliva. In this form and by this process, mercury as a byproduct of corrosion would have minimal significance. There is a further question as to whether the release of mercury vapour during the functional life of amalgam may be regarded as a consequence of or part of the corrosion process.

It has been advocated that copper rich amalgams may be superior to conventional amalgams in that they are less porous and exhibit reduced corrosion which is confined to the surface and may account for the reported superior marginal integrity (Marshall, Jackson & Marshall 1980). A recent study by Patsurakos and Moberg (1990) tested conventional and high copper amalgams for marginal microhardness. The microstructure of the amalgam was tested during corrosion specifically for tin, copper, zinc, silver and mercury dissolution. It was found that the high copper amalgam retained the greatest microhardness after corrosion and this was attributed to the differential degradation of the eta phase in the case of the high copper amalgams as against the breakdown of the gamma-2 phase for conventional amalgam.

Eley (1985) notes that when the copper amalgams corrode the reaction affects the Cu₅Sn₆ phase and does not release mercury. Gettleman (1986) quotes Holland and Asgar (1974) and Domagala, Van Thyne and Lenke (1968) in concluding that the preponderance of evidence indicates that it is the tin and
copper in amalgam that corrodes releasing metallic mercury back into the restoration and not into the tooth.

Brune (1985) found the release rate of mercury from conventional, dispersed phase or spherical high copper content amalgam as decreasing approximately exponentially with time. Similarly, in an in vitro experiment, corrosion current decreased exponentially with time. After simulated brushing corrosion current increased, indicating the removal of loosely bound corrosion products. Brune (1986) also notes that the mercury release from amalgam surface seems to be strongly influenced by the interaction of mechanical forces e.g. chewing and seems to be released according to a cyclic pattern.

An in vitro study by Derand (1989) on different types of amalgams contradicts the reports that high copper amalgams release less mercury vapour. "Sybralloy" (30% Cu) and "ANA 2000" (25% Cu) had significantly higher mercury vapour release rates than other amalgams.

Eley and Cox (1987) calculate a maximum and daily exposure limit of 200 μg of inorganic mercuric compounds. They thus discount the danger of adverse effects from ingestion of saliva containing corrosion products of amalgam restorations.
11.4 LOSS OF MERCURY FROM AMALGAM AND DAILY DOSE

There is as yet no scientifically accepted daily dosage of mercury vapour attributable to dental amalgams, which from different in vivo and in vitro studies has been estimated at ranging from negligible to 150 µg/day. These are presented in chronological order in Table 8.

| DAILY DOSE OF MERCURY VAPOUR FROM DENTAL AMALGAM (>10 surfaces amalgam) |
|-----------------------------|------------------|
| Radics et al (1970)         | 150.00 µg        |
| Sware et al (1981)          | 17.50 µg         |
| Abraham et al (1984)        | 8.00 µg          |
| Brune and Evje (1985)       | 20.00 µg         |
| Patterson (1985)            | 27.00 µg         |
| Patterson (1985)            | 2.50 µg          |
| Vimy & Lorscheider (1985b)  | 30.00 µg         |
| Vimy & Lorscheider (1985b)  | 2.90 µg          |
| Mackert (1987)              | 1.83 µg (recalc of Vimy & Lorsch) |
| Langworth et al (1988)      | 3.00 µg          |
| Snapp et al (1989)          | >1.30 µg         |
| Marshall (1989)             | 22.00 µg         |
| Derand (1989)               | 0.60 µg (in vitro study) |
| Berglund et al (1990)       | 1.70 µg          |
| WHO (1991)                  | 3.8-21 µg        |

Table 8

a calculated in Clarkson et al (1988)
[See 14.2.2 for discussion]
c questionable conclusion
d mainly inorganic mercury compounds in amalgam particles and as well as in vitro experiment
e Radics, Schwander and Gasser (1970)

The work of Berglund, Langworth, Snapp, and Mackert varies markedly from the earlier estimates of the studies by Svare, Abraham, Patterson and Vimy & Lorscheider. There are varying appraisals of Vimy & Lorscheider (1985a,b), markedly differing from that of the authors themselves, and the figures
attributed to Svare, Abraham, Patterson and Vimy & Lorschieder marked with superscript b are calculations made by Clarkson et al (1988) which involve various assumptions regarding sampling methods, accuracy, breathing patterns and time considerations. There now exist two distinct schools of thought on the quantity of mercury released and taken up from amalgam restorations and the potential significance in respect of body burden. There seems a definite trend in the more recent studies to identify smaller amounts of mercury being released and taken up per day from amalgam (1.3-3.0 μg).

Vimy & Lorschieder (1990) have, at least in part, accepted that their original 1985 calculations were incorrectly based and overestimated the amount of mercury vapour released from amalgam: 'Corrections for the sampling factors of flow rate and sampling dilution, and the respiratory factor of mercury accumulation in the closed mouth between oral inhalations, reduce our original daily dose estimates by approximately 50%.'

Marshall, Marshall and Letzel (1989) found that a loss of mercury occurred in dental amalgams after clinical use. Using five brands of copper rich amalgams there was variation between 0.7%-2.8% loss of mercury over a clinical life of between 3.6 and 8.9 years. In the case of greatest loss of mercury with an average clinical life of 6.6 years this equated to a loss of 2.2 μg per restoration per day. The authors note that corrosion product formation during clinical use could account for an apparent mercury loss up to 2.5%, suggesting that the apparent loss might not be real. As well they suggest that a portion of the
variability may be a consequence of batch differences and operator technique. The authors conclude that: 'amalgam restorations appear to contribute only a minor amount to total daily mercury dose and that amalgam restorations removed after prolonged clinical use contained nearly all the original mercury present after their placement.'

Taken at face value the results of this research is disturbing and the conclusions facile. If 2.2 \( \mu g \) of mercury per day per restoration are lost then a person with 10 amalgam restorations would be exposed to 22 \( \mu g \) of mercury per day which, if absorbed, could be a substantial addition to the body burden of mercury.

In the study by Marshall there is need to know in what form the mercury is lost - whether contained in inorganic particles, (in which case although swallowed there is minimum absorption [7-10%]) or as mercury vapour (in which case there is greater absorption [85%] but only a fraction of which would be actually inhaled). The authors note the complicating factor of corrosion product formation affecting the calculations of mercury loss and it seems necessary that this research be duplicated and extended in scope to clarify the form of mercury loss and as well the validity and significance of the level of mercury loss.

On a broader level the research of Marshall does invalidate the unsubstantiated claims in the anti-amalgam literature that up to 50% of mercury is lost from amalgam restorations.

Brune and Evje (1985) equated the body's mercury load derived from amalgam dental restorations to that obtained from intake of food (i.e. approximately 20 \( \mu g \) Hg/day from each source). This calculation was based on an in vitro study
of amalgam in natural saliva simulating cyclic loading as well as static conditions. The majority of mercury released was present in amalgam particles. These inorganic compounds of mercury would be ingested and only 7-10% absorbed. The release of mercury vapour is discussed but perhaps because the vehicle in the study is saliva, this factor was not accurately measured or quantified. There is an unresolved question as to the effects of smoking on mercury levels and the confounding potential that this might have on studies relating to mercury release from amalgam. Tobacco leaves contain mercury and thus smoking may contribute to inhalation exposure (Suzuki, Shishido & Urushiyama 1976).

Svare (1984) cites Bhatnagar (1980) who reported higher blood mercury concentrations in smokers. Svare, however, found lower post-chewing mercury breath levels in smokers than in non-smokers although numbers were small (4).

**11.5 MERCURY VAPOUR FROM CREMATION**

Cremation of cadavers vaporizes all mercury from amalgam restorations and releases regular and perhaps significant quantities of metallic mercury vapour into the atmosphere, usually within residential areas. Mills (1990) calculates that one crematorium in Leicester, U.K., carrying out an average of some 3800 cremations per annum may release 11 kg of mercury into the atmosphere through an unfiltered single crematorium chimney. Mills calculates the mercury content of an amalgam (0.6 g) on the basis of unmixed ampoules of mercury and alloy powder. Compared with Marshall et al (1989) who quote a typical
restoration as containing 0.19 g mercury after setting, Mills figures are an overestimation by a factor of three. Mills other assumption that each person cremated would have an average of 5 amalgams in their mouths is in need of validation. The author wisely advocates proper ground and air sampling programmes to be initiated to assess any possible health hazard and suggests (if necessary) activated charcoal filters be installed in the crematorium chimneys. Rivola, Krejci, Imfeld and Lutz (1990) evaluated the dental status of 130 deceased Zurich persons. The mean mass of mercury per dentate deceased was calculated to be 2.49 +/- 0.37 g (a total not dissimilar to that of Mills (1990). The authors further calculate that in Switzerland, with 55.5% of funerals being cremations, mercury contamination by cremation comprised only 0.61-1.53% of total mercury contamination produced by all waste incineration methods and the contribution of dental amalgam is thus minimal.

11.6 RELATIONSHIP OF ORGANIC MERCURY TO AMALGAM RESTORATIONS

Generally organic mercury is considered unrelated to dentistry but there exists a tenuous relationship whereby it has been postulated that chronic exposure to elemental mercury via silver-mercury restorations may be followed by biotransfer to the more toxic methyl mercury (Gay, Cox & Reinhardt 1979). The biological conversion of ingested inorganic mercury to organic mercury (specifically methylmercury) has been demonstrated in fish, bacteria in sediment, and, under laboratory conditions, in strains of bacteria from animals and humans. In vitro experiments producing methylation of mercury by oral streptococci (Heintze, Edwardsson, Derand & Birkhed 1983) have not been recreated in vivo and no
methylmercury from dental sources has been detected in the oral cavity. A slight increase in methylmercury levels has been reported in the blood and urine of dentists, but this may in fact be due to experimental errors or confounding by exposure to methylmercury (e.g. fish in diet).

The methylation process is extremely slow and occurs under strict environmental and chemical conditions. Additionally, in respect of the experiments cited above, it has been noted that digestion of bacteria would be necessary to free the intracellular methylmercury resulting in quantities (0.029 mg methylmercury) that would only be a fraction of the minimum safe level. To place this hypothesis in proper perspective it has been reported that after five years of consumption of contaminated fish containing 0.8 mg methylmercury per day no symptoms of mercury poisoning were evident.

Chang, Siew and Gruninger (1987) evaluated the possible existence of an in vivo biotransformation of elemental mercury from dental amalgam into more toxic organic mercurials. Using 205 practising dentists they showed that high levels of inorganic mercury in the blood was not correlated with high organomercurial levels, which were insignificant. The authors concluded that: 'significant enzymatic conversion of inorganic to organic mercury compounds does not occur in vivo'.

The theories postulating a significant human methylation of inorganic to organic mercury (which are capitalised on by the anti-amalgamists to indicate a more severe consequence for the mercury released from amalgam) are conjectural and
entirely unsupported by any clinical data. As a corollary, it should be noted that inorganic levels in the blood which have been attributed to mercury release from amalgam may in fact be part of the demethylation process from what was original methylmercury exposure.

11.7 SIGNIFICANCE OF MERCURY FROM AMALGAM

It is important to note that evidence of toxicity from long term exposure to high levels of mercury vapour (50-100 μg/ m³) is not so easily translatable to effects from long term exposure to very low levels (1-10 μg/ m³). High doses of mercury produce obvious and characteristic symptoms, but the vague non-specific features which might indicate low levels of mercury poisoning are also features of a multitude of other maladies. Although there is little doubt that amalgam restorations produce demonstrable levels of mercury vapour and stimulation increases these levels, there is much variation in the published data and, more importantly, the significance of that data is debatable.

There is however a difference between occupational exposure, where the ambient air is suffused with mercury vapour at a given concentration (e.g. in a chloralkali plant), and exposure of general population to unpolluted atmospheric air where there may be situational variations during the day in the air that is breathed (e.g. toothbrushing releasing mercury from amalgam restorations). Additionally, if mercury vapour from amalgam restorations is being assessed as contributory to the body burden of mercury, the individual levels of intra-oral air reflecting release of mercury vapour from amalgam restorations will be
diluted by the surrounding air which contains lesser levels of mercury vapour.

Furthermore the intra-oral air is neither regularly nor fully inspired given the variations between nasal and oral inspiration and between expiration and inspiration. Thus there is perhaps doubt as to the validity of comparing air mercury levels in the occupational environment with mercury which is only in intra-oral air and caution should be exercised in further extrapolating results to imply a correspondence to other systemic parameters such as blood and urine levels.

An additional factor (albeit minor) which should be considered is the quantity of mercury vapour in exhaled air which occurs as a minor byproduct of the inorganic breakdown of mercury in the body and which accounts for some 7% of total excretion.
12. MERCURY LEVELS FOR DENTISTS AND DENTAL PERSONNEL

12.1 MERCURY VAPOUR LEVELS

Mercury vapour levels were measured at a British Dental Hospital (Wilson & Wilson 1985) and the vast majority of readings were below 5 $\mu$g/m$^3$. Some floor level readings approached the British occupational exposure limit of 50 $\mu$g/m$^3$ and on one floor where amalgam was being use continuously (with poor ventilation) the ambient mercury vapour level was 5-10 $\mu$g/m$^3$. High readings (up to 160 $\mu$g/m$^3$) were obtained within some dental spittoons presumably due the drying of waste amalgam in unemptied traps.

WHO (1991) reports a variety of studies measuring mercury vapour levels in dental clinics. Average levels were 20-30 $\mu$g/m$^3$, with certain clinics having levels up to 150-170 $\mu$g/m$^3$.

Nilsson and Nilsson (1986 a,b) reported mercury levels of 4 $\mu$g/m$^3$ in the air of private dental clinics.

A survey by the Council on Dental Materials and Devices in the United States in 1974 indicated that a significant number of dental offices had a mercury vapour level equal to or in excess of the current threshold limit value for airborne mercury vapour of 0.05 mg Hg/m$^3$ (50 $\mu$g/m$^3$).
12.2 URINE MERCURY LEVELS

The American Council on Dental Materials and Devices (1974) found that a significant number of urine samples contained more than the normal levels of mercury and a correlation existed between general air exposure and urinary mercury excretion by both dentists and assistants.

Urinary mercury levels in 4272 U.S. dentists were studied for the period 1975-1983 (Naleway, Sakaguchi, Mitchell et al 1985). The mean level was 14.2 μg/L with a range from 0 to 556 μg/L [compared to a "normal" level for the unexposed population of 0.5-3.0 μg/L]. The type and character of dental practice, together with the method of amalgam/mercury handling, can influence the amount of absorption of mercury into the body. High levels were noted in those dentists in general practice, working more than forty hours per week, placing larger numbers of amalgams per week and in surgeries with heating/cooling systems producing minimal air turnover. The authors concluded that less than 1.3% of the dentists surveyed possessed urinary levels above 100 μg/L, a level at which documented physiologic effects first appear. This latter figure should be compared to the recommended WHO occupational exposure limit for mercury in urine of 75 μg/L which if utilised would see 2.3% of the sample over the threshold level.

Surveys were conducted at the Norwegian Dental Association Congresses in 1986 and 1987 (Jokstad 1990) to assess mercury exposure. Morning urine samples and questionnaires were collected from 672 participants in 1986 and 273 participants
in 1987. Mean values of urinary mercury excretion were 39 nmol/L (78 μg/L) in 1986 and 43 nmol/L (86 μg/L) in 1987. The data indicate that the following factors affect the level of urinary mercury: gender (lower for female than male), restorative status, the number of placed restorations per week as well the number of polished and replaced amalgam restorations per week. Surgery environmental factors such as wooden floors and scrap amalgam separators caused elevated mercury values.

A study of 18 dental personnel with higher than normal urinary mercury levels showed a relationship between plasma mercury levels and the total number of amalgam surfaces (Molin, Marklund, Bergman & Nilsson 1989). A large number of supplementary analyses were carried out which did not indicate any influence of the mercury on organ functions. 'Although the persons in the present study were occupationally exposed to mercury, none of the biologic variables analyzed seem to be affected.'

Skare and Engqvist (1990) examined 6 dentists and 4 dental nurses prior to and after vacations to assess overall halftime for clearance of urinary mercury after cessation of mercury vapour exposure. The halftime for urinary mercury was a mean of 41 days with a range from 21-91 days.

Herber, de Gee and Wibowo (1988) studied 162 dentists and assistants for mercury levels in hair and urine. Both number of fillings placed and hours in the surgery related to urine mercury: a 10-fold increase in number of fillings placed gives a 4-fold higher mercury urine level; a 5-fold longer duration in practice
gives a 70% higher urine mercury level.

Nilsson and Nilsson (1986 a,b) studied 505 dental personnel and found mercury urine values (6-7 μg/L) which were higher for dental personnel than for the control group, but were very low and in general below the upper limit for normal non-exposed subjects. No subjects reported an allergy against mercury. The authors studied the prevalence of symptoms which may be caused by long-term exposure to mercury vapour: fatigue, anxiety, insomnia, loss of appetite, tremor and short-term memory. The greatest number of symptoms were found among women dentists, which group also had the greatest number of subjects with three or more symptoms. However the prevalence of symptoms was not related to exposure parameters which thus clouds the relevance of these results.

Mercury exposure and renal function parameters were examined by Verschoor, Herber and Zielhuis (1988) in 68 dentists and 64 dental assistants. Levels of mercury in urine were low with only three individuals exceeding 20 μg/L Hg. There was increased urinary protein excretion and increased activity of urinary enzymes. These functional changes were not related to mercury urine level, age sex, smoking or drinking habits. The authors conclude that the proteinuria is an indicator of increased risk factor and may be due to one or more potential nephrotoxic agents used in dental practice.
A study by Akif, Seckin, Aygun and Ataman (1986) used cold vapour atomic spectrometry and gas-liquid chromatography for determination and speciation of mercury in a group of staff and student dentists. Of interest is the fact that of 15 urine samples selected for mercury speciation almost 50% contained organic mercury mainly in the form of methylmercury. The percentage of organic mercury in the total mercury ranged between 19% and 87.5%. It is generally regarded that urine mercury reflects inorganic exposure, whereas this study indicates that diet and environmental conditions predisposing to organic mercury exposure may contribute to urine mercury levels.

A summary of the results of studies measuring urine mercury levels in dental personnel is shown in Table 9.

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>MEAN</th>
<th>RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>MASSACHUSETTS (1970/71)</td>
<td>4.9-23.0</td>
<td>4.9-36.0</td>
</tr>
<tr>
<td>KELMAN (1978)</td>
<td>38.0 (Assistants)</td>
<td>22.0 (Dentists)</td>
</tr>
<tr>
<td>NIXON (1981)</td>
<td>26.0</td>
<td>2-149</td>
</tr>
<tr>
<td>NILSSON &amp; NILSSON (1986)</td>
<td>6.0 (Dentists)</td>
<td>7.0 (Assistants)</td>
</tr>
<tr>
<td>NALEVAY (1985)</td>
<td>14.2</td>
<td>0-556</td>
</tr>
<tr>
<td>JOKSTAD (1990)</td>
<td>78.0 (1986)</td>
<td>43.0 (1987)</td>
</tr>
<tr>
<td>VERSCHOOR (1988)</td>
<td>&lt;20.0</td>
<td></td>
</tr>
</tbody>
</table>

Table 9
12.3 BLOOD MERCUry Levels

Dentists who have been in active practice for more than twenty years have higher serum mercury levels (1.13 +/- 0.46 μg/100mL; 11.3 +/- 4.6 μg/L) than do dental students (0.82 +/- 0.23 μg/100mL; 8.2 +/- 2.3 μg/L) or a control group of healthy individuals whose occupation is not related to dentistry (0.72 +/- 0.22 μg/100mL; 7.2 +/- 2.2 μg/L) (Carrel, Mackowiak, Chialastri & Binns 1981). The author concludes that while the dental practitioners had significantly higher levels of mercury in their sera, no participant had a serum mercury level higher than 5 μg/100mL (50 μg/L), a level far below that which usually precipitates clinical symptoms of mercury toxicity. This last statement may require further assessment as a number of authors have considered 50 μg/L blood mercury as being sufficient to cause symptoms of mercury toxicity. [See Table 3, Section 7.1.1.]

In a study of 130 Danish dentists and 40 blood donor controls Moller-Madsen, Hansen and Kragstrup (1988) found a median blood concentration of mercury of 4.0 μg/L (range 1.2-19.2 μg/L) for dentists and 2.0 μg/L (range 1.1-4.6 μg/L) for controls. Practice characteristics showed no relationship to blood mercury, but 49 dentists having one or more fish meals per week had a median blood mercury value 47% higher than those dentists consuming fish infrequently.

A summary of the results of studies measuring blood mercury levels in dental personnel is shown in Table 10.
12.4 HAIR MERCURY LEVELS

High levels of mercury were found in dentist’s hair with 14% of a group of 150 dentists having mercury levels higher than 10 ppm (Pritchard, McMullin & Sikondari 1982). Some dentists tolerated levels up to 64 ppm without symptoms or complaint, although one dentist with a level of 40 ppm demonstrated ataxia and cerebellar speech problems. The high levels were almost entirely related to occupational exposure to mercury vapour in the dental surgery and the majority normalised once proper mercury hygiene measures were instituted. Given the probability of external adsorption by direct contamination of hair by mercury vapour as well as systemic deposition of mercury in the forming elements of hair, the results are not applicable to the general population except for the high levels tolerated without symptoms. Another study (Francis et al 1982) analyzed hair samples from dental and non-dental personnel and found no significant difference between the two groups.
Similar findings were reported by Nishino, Morita and Shimomura (1986) who studied the mercury levels in hair of 139 male and 9 female dentists to ascertain whether accumulation occurs as a function of years in practice and quantity of amalgam fillings placed per month. The control group had mercury hair levels of 3.11 ppm (males) and 2.28 (females), the dentists had 3.41 ppm (males) and 2.28 ppm (females). Although the authors resolve that hair mercury levels in dentists were not different from the general population and were not correlated with years in practice and number of amalgams placed per month, the fact that 7 of 8 dentists with high mercury hair levels had placed more than 50 amalgams per month seems to contradict this last conclusion.

Scarlett, Gutenmann and Lisk (1985) found a significant difference in the mercury hair concentration of a group of dentists studied in 1972 (16.77 +/- 4.00 ppm) and again in 1985 (5.27 +/- 1.85 ppm). They judged this reduction due to the modern use of capsulated amalgam as against the former use of manual mixing.
12.5 BRAIN MERCURY LEVELS

Nylander (1986) found high levels of mercury (135-349 ng/g wet weight) in the pituitary glands of 3 dentists compared with minimum levels in the occipital cortex. Additionally he reported a level in the pituitary of 97 ng/g Hg in one control subject who had several amalgam restorations removed in the last year of life and 50 ng/g Hg in another control subject who had multiple amalgam restorations. He suggested that these levels were due to inhalation of mercury vapour and postulated a route of absorption by the nasal mucosa, with a difference in degree of penetration from arteries allowing direct transport to cranial cavity and pituitary gland. In a further autopsy study Nylander, Friberg, Eggleston and Bjorkman (1989) found high levels of mercury in the pituitary and thyroid glands of dentists as against controls. These findings are given further weight in the report by Kosta, Byrne and Zelenko (1975) of workers in mercury mines in Yugoslavia where post-mortem samples showed high accumulation and retention of mercury in the thyroid and pituitary glands.

Langworth, Rojdmak and Akesson (1990) tested a group of dental personnel exposed to mercury vapour from dental amalgam and found no evidence of a negative influence on pituitary function at the existing level of low grade chronic mercury exposure.
12.6 PREGNANCY AND REPRODUCTION

In a survey of 21,000 dentists and 21,000 dental assistants the relationship between mercury exposure and pregnancy outcome was investigated. Exposure to mercury was categorised as "high" if greater than 40 amalgams per week were effected. For both direct exposure for dental assistants and indirect exposure for wives of dentists there was no increase in the incidence of spontaneous abortions and congenital deformities in the period 1968-1978 irrespective of the level of mercury exposure (Brodsky et al 1985).

Magos (1988) raises a number of doubts as to the validity of the results in Sikorski, Juszkieiwicz, Paszkowski and Szprengier-Juszkiewicz (1987) which reported that in the dental profession exposure to mercury vapour increased the frequency of reproductive failure and that there is a positive correlation between mercury in scalp hair and reproductive failure or menstrual disorders. Magos notes, however, historical evidence supporting the view that toxic exposure to mercury vapour at a relatively early gestation stage has an abortifacient effect and emphasises that the question is one of dose/response relation.
12.7 SYMPTOMS OF MERCURY POISONING

A study by Nilsson, Gerhardsson and Nordberg (1990) examined 505 dental personnel for exposure to mercury, urine mercury levels and symptoms of mercury poisoning. Mercury vapour levels in the dental surgeries was lower than in previous studies, ranging from 1.5-3.6 $\mu g$/m$^3$. Urine concentrations were also low (1.4-2.9 nmol Hg/mmol creatinine), being similar to that of the general Swedish population (5 $\mu g$/L; 2.8 nmol Hg/mmol creatinine). The authors note that there is an equivalence between the amount of mercury intake from occupational exposure and that from amalgam restorations. Four symptoms were studied; loss of appetite, tremor, insomnia and anxiety. The prevalence was low (<11%). In this study which included mercury intakes up to twice the contribution from amalgam fillings, no increase in the prevalence of symptoms could be detected.

Similar results were recorded by Wirz, Ivanovic and Schmidli (1990) who tested dentists and assistants, and control groups with and without amalgam restorations. The two control groups showed no significant difference in blood and urine mercury levels, implying that these slight traces of mercury can be attributed to food and the environment. Although the blood and urine mercury levels of the dental office personnel were twice that of the control groups there was no threat of mercury poisoning for any of the tested groups. The authors conclude that the continued use of amalgam fillings in teeth can be recommended without reservation and at no risk to the patient.
Ayer, Getter, Machen and Haller (1976) studied blood levels of mercury and hand steadiness in 501 male dentists as found no differences between normal scores and those which were above 15 \( \mu g/L \).

Shapiro, Sumner, Spitz, Cornblath, Uzzell, Ship and Bloch (1982), using x-ray fluorescence measurement of head and wrist mercury content, found 7 dentists out of 298 with sub-clinical polyneuropathies.

### 12.8 Hypersensitivity Reactions

Miller, Perry and Agner (1987) tested a group of dental students for hypersensitivity to mercuric chloride. They found no increase in the development of allergic reactions as the students progressed through their course. The finding that a standard 31.6% of the students had mercury hypersensitivity is far in excess of the results of White and Brandt (1976) which was a mean of 5.6%. The latter study, however, showed an increase in positive patch testing to mercury in students from 2% in freshmen to 10.8% in seniors.

### 12.9 Longevity

Data on age at death of dentists compared to that of the same age group in the general male population (1961-1966) showed no difference in longevity (71.2 years) (Bureau of Economic Research and Statistics 1968).
Dentists have been traditionally noted as more likely to commit suicide than other members of the population. A study by Arnetz, Horte, Hedberg and Malker (1987) showed that though male dentists were twice as likely to commit suicide than other academics, the rate was similar to that of the general population. The authors postulate the possible role of occupational exposure to mercury vapour as a contributory risk factor. This seems highly unlikely given that female dentists had the same suicidal tendencies as other academics and, as well, that the other high risk group for suicide were physicians. This latter group while being within the health care arena, are not exposed to mercury and thus it is more likely that psychosocial and personality factors are the cause of the suicide rate of dentists and physicians.

12.10 DENTAL PERSONNEL AS A HIGH RISK GROUP FOR MERCURY EXPOSURE

The bulk of evidence shows that a significant number of dentists and dental assistants are routinely occupationally exposed to elemental mercury vapour. As many as 10% of dental offices have been shown to have mercury vapour concentrations in excess of 100 μg/m³. General dental practitioners as a group have blood, urine and tissue levels of mercury higher than those of the general population, yet symptoms of toxicity are rare. Dental personnel with their raised exposure to mercury (and with the additional contribution of their own amalgams) would surely appear as a distinct epidemiological group if the symptoms and diseases actually existed as a result of the toxicity attributed to mercury and amalgams. The raised levels and minimum consequent health
problems highlight the lack of risk to the general population. The reason for high levels in surgeries is due in major part to poor mercury hygiene practices which are preventable.

De Freitas (1981) reviewed international and Australian surveys in respect of airborne mercury in dental surgeries and biological mercury levels of dental staff. Although Australian conditions did not justify concern the author recommends regular testing of dental facilities with portable detectors and personnel with monitoring badges.

In addition to mercury vapour inhalation there is the risk to dental personnel of inhalation of small amalgam particles (<10 μg) during removal of amalgams which may become lodged in the alveoli. This potential problem is preventable by the routine use of masks by dentist and staff during amalgam removal.

More research is needed in an extended examination of offspring of dental personnel, to eliminate the possibility of minor neurological changes which may not be evident at or immediately after birth.
13. THE EVIDENCE FOR CLAIMS OF MERCURY

TOXICITY FROM AMALGAM RESTORATIONS

The use of dental amalgam is under a broad-fronted attack. There is growing pressure from a very vocal group of anti-amalgamists, whose number in the health care industry outside of the dental profession is increasing (Bales 1991). There are many people who, having been made aware of the claimed link between amalgam restorations, mercury poisoning and ill health, become convinced that their complaints are caused by their own amalgam restorations. The symptoms reported to be associated with amalgam include virtually all deviations from normal (see Pleva and Ziff below) and the diseases states implicated include cancer, birth defects, migraine, epilepsy, arthritis, blindness, candidiasis, AIDS, Crohn's Disease, Parkinson's Disease, Alzheimer's Disease, Multiple Sclerosis and Chronic Fatigue Syndrome. This study specifically addresses the conditions of Multiple Sclerosis, Chronic Fatigue Syndrome and candidiasis as regards their alleged relationship with mercury and amalgam. Other conditions and disease states are not dealt with in any detail, although the evidence and discussion which specifically relates to those diseases investigated in this study can readily be applied in general to any or all of the aforementioned ailments. While a vast catalogue of diseases are listed or mentioned in lay publications as emanating from mercury in amalgam there are
virtually no studies in the scientific literature examining these associations, the
general view in the medical and scientific community being that there is no
justification to examine such capricious speculations.

Strict scientific studies on subjects with alleged mercury toxicity from amalgam
have not found any evidence which would allow acceptance of a traditional cause
and effect correlation between amalgam and specific symptoms or disease states.
However, there seems a perpetual inclination on the part of those who claim
amalgam is a danger to health to see even a lack of evidence as support for
their stance. The majority of reports which describe deleterious effects and
symptoms from amalgam restorations and improvement after amalgam removal
are usually anecdotal, uncontrolled, are often biased and at best inconclusive.
The absence of incontrovertible proof that mercury from amalgam does not
cause disease is interpreted to imply that it might. Conscientious scientific
methodology does not reject such an hypothesis entirely, (even though the bulk
of evidence fails to show any relationship), where there are unresolved questions.
Unfortunately the remote possibility that some individuals may be affected by
their amalgams is often exaggerated and extrapolated to the extent that the
thesis that amalgam is a health hazard to the general public is considered
proved.

Thus, in a strict scientific and philosophic sense, the question of whether the
mercury from amalgam is a health hazard has not been entirely nor satisfactorily
resolved. Amalgam restorations have become ensconced in the armamentarium
of the dental profession and the lack of obvious health deficits from amalgam and the mercury it contains has created a long period of complacency. While there is no evidence to support any of the claims that mercury from amalgam causes ill health it is important that more research is carried out (both clinically and epidemiologically) to scientifically reinforce the traditional sentiment that assumes amalgam is a safe therapeutic agent.

Weiner, Nylander and Berglund (1990) claim that: 'from a toxicological point of view, amalgam is an unsuitable material for dental restorations'. This same phrase occurs in the Lek Report (1987) and is slightly misleading in that it makes an assumption that amalgam produces toxic effects. Amalgam may rarely produce allergic responses (contact stomatitis, oral lichenoid lesions) and which may be due to the mercury contained therein. However amalgam itself is not "toxic" which would imply unproven systemic sequelae, and thus amalgam cannot be regarded from a 'toxicological point of view'. In fact it is the mercury (in the generic sense) that, because of its toxic properties, could be regarded as an inappropriate material for use in dentistry. By extension one can suggest (with some justification) that amalgam, because it contains mercury, is, in principle at least, unsuitable for use as a dental restoration. Once this fact is accepted, the core of this dialectic becomes the question of whether the levels of mercury released from amalgam, when compared to the other sources of mercury which impact on the human subject, assume any toxic significance. Weiner et al note that high levels of mercury released from amalgam could place individuals at risk and they are concerned that genetically susceptible individuals may develop
auto-immunity from mercury with more severe effects. The fact that a very small number of individuals show a hypersensitivity to one or more of the constituents of amalgam, does not justify an extrapolation to imply that there are vast numbers of the community whose health is at risk because of amalgam restorations. A reasonable analogy is that there are a small number of the population who are allergic to penicillin. This group must avoid this particular drug, but there is no pressure to deprive the rest of the community from its benefits. In the same way, those rare allergic responses to mercury and amalgam must be weighted against the bulk of the community who show no ill effects from the use of amalgam.

The essence of the anti-amalgam argument centres on a number of key features:

1. That the mercury released from amalgam is at a high level and constitutes a serious health hazard to the population;

2. Allergy to amalgam is prevalent in the community and large numbers of the population have developed toxic systemic reactions to mercury;

3. The clinical signs of mercury poisoning include a vast number of symptoms and diseases;

4. Oral galvanism is a consequence of contact between amalgam and other metal dental materials and contributes to mercury poisoning and symptoms;

5. Inorganic mercury from amalgam can be methylated by the body to become the more toxic methylmercury;

6. Removal of amalgam will alleviate symptoms and diseases;

7. Certain tests can elicit "mercury sensitivity" and "mercury toxicity".
There are quantum jumps of attribution whereby:

a. the symptoms and diseases recorded (particularly neurological and/or immunological of unclear aetiology) are presumed to be initiated or exacerbated by mercury toxicity;

b. the mercury in amalgam restorations (and particularly released mercury vapour) is assumed to be the aetiological factor in mercury poisoning.

Much of the material quoted in support of mercury toxicity from dental amalgam is selectively culled from scientific and peripheral literature. Case studies reporting cures are always anecdotal in nature and the reports are generally not supported by data from objective medical examinations. There are usually no control groups, and the results are not evaluated for placebo effect and observer bias.

The anti-amalgamists using the broadest of brushes, have created a polymorphic amalgam syndrome. In Pleva (1983) the alleged signs and symptoms of mercury amalgam toxicity include:

a. PSYCHOLOGICAL DISTURBANCES such as irritability, nervousness, shyness, depression, anxiety, fits of anger, lack of attention, drowsiness and insomnia.

b. ORAL CAVITY DISORDERS including bleeding gums, foul breath, ulceration and excessive salivation

c. GASTROINTESTINAL EFFECTS such as abdominal cramps, colitis and diarrhoea.

d. SYSTEMIC EFFECTS:
   i. Cardiovascular- irregular heartbeat and pulse, altered blood pressure, chest pain
   ii. Neurologic- headache, dizziness, ringing in ears, fine tremor
   iii. Respiratory- cough, emphysema, shallow or irregular respiration
   iv. Immunological- allergies, asthma, sinusitis
   v. Endocrine- cold clammy skin, excessive perspiration
      Also muscle weakness, fatigue, anaemia, loss of appetite, joint pains

   e. SEVERE CASES include hallucinations and manic-depression.
Ziff (1984, 1988a,b) includes a list of common complaints which may be attributed to the effects of mercury from amalgam restorations:

**NERVE OR MUSCLE PROBLEMS**

**MOOD CHANGES**

**ORGANS AND SYSTEMS PROBLEMS**
1. SKIN- rashes, excessive perspiration 2. EYES- burning, itching, excessive tearing, feeling of heaviness and pressure within eyes 3. EARS- dizziness (Meniere’s syndrome), decreased hearing, buzzing in ears (tinnitus), “plugged” ears (swollen eustachian tubes) 4. NOSE- nasal obstruction, sinus congestion, sneezing (rubbing nose upward is a sign of allergy). 5. THROAT- hoarseness, “itching” throat (leading to sore throat), excessive mucus 6. LUNGS- wheezing 7. CARDIOVASCULAR- palpitations, flushing 8. GASTROINTESTINAL- nausea, loss of appetite, voracious appetite or sudden weight gain (5 pound in 2 days), chronic obesity, excessive thirst 9. GENITOURINARY- urgent urination, frequent urination, bedwetting, vaginal itching, excessively painful menstruation 10. MUSCULO-SKELETAL- muscle soreness, joint pains, uncertain gait

**GENERAL PHYSICAL PROBLEMS**

This plethora of non-specific subjective manifestations is in its profuse scope its greatest weakness. No one person would exhibit all these symptoms concurrently and any one symptom in isolation cannot be regarded as legitimate evidence of mercury poisoning. In fact, many, if not most of the symptoms mentioned above could be as easily attributed to the effects of food additives, for example. The use of opposite symptoms (e.g. loss of appetite and voracious appetite) caused
Evidence for Claims of Mercury 133  Toxicity from Amalgam Restorations

by the same initiator defies logic. Katz (1991) makes the comment that the list of diseases and conditions 'is as long as the imagination is broad.'

Huggins claims that: '67% of the American population have already developed systemic (toxic) reactions to mercury as would be expected in a group exposed to amalgam' (Kaiser 1988).

Hanson and Pleva (1991), two stalwart anti-amalgamists, in a review of the dental amalgam issue stress the potential impact of amalgam mercury on human health, but fail to show any specific evidence of cause and effect between mercury in amalgam and disease states. In concluding, the authors limply resolve that discussion of the dental amalgam issue has suffered from the lack of an interdisciplinary approach and that amalgam mercury should be considered among other possible factors in neurological and immunological diseases of unclear aetiology.

The major public proponents of amalgam mercury toxicity, Huggins and Ziff, must be questioned as to the legitimacy of their claims as well as the financial basis of their marketing activities. Dr Huggins, an American dentist, writes: 'Have you ever felt draggy, listless and even fatigued when you wake up?: Have you ever felt depressed, irritable and jumpy, lashed out at people for no reason?:.....for many of my patients, the culprit is mercury toxicity.'

In the modern world, tension, stress and the pressures of daily existence commonly cause brief or even prolonged bouts of emotional and physical disturbance. The 'mercuriophobes' use non-specific and common symptomology
(as in the above description) in order to broaden the base for their prospective clients, and then resort to quasi-scientific practices to influence and convince the lay public of the scientific foundation for their recommendations.

Another approach involves the concept of "Optimal Health". Bellman (1987) suggests that the question is not whether mercury in amalgam fillings is causing ill health, rather to what degree mercury in amalgam is affecting an individual's ability to achieve optimal health. This semantical reflection on the difference between absence of disease and peak health rather begs the question of mercury toxicity from amalgam as a cause of disease and symptoms. The author does however note that the acceptable normal values are based on a population with a high diet of saturated fat, sugar and refined foods, who have minimal exercise and high stress levels and who are exposed to air and water tainted with chemicals and pollutants. These variables only serve to raise further doubts as to the certainty of any relationship posed between amalgam and health problems.
13.1 STUDIES EXAMINING EVIDENCE OF ILL HEALTH RELATED TO AMALGAM

An indication of the infrequency of toxic and allergenic reactions to dental treatment is seen in the study by Kallus (1985), who with the participation of 137 dentists monitored 13,325 patients in order to assess the incidence of adverse reactions from dental materials. In 15,820 visits 24 comments by patients were recorded and 22 observations made by dentists concerning assumed side effects. No reactions were proved (4 probable and 3 possible) and none of these caused by materials intended for permanent use. At worst (assuming that the 7 cases were adverse reactions to dental materials) this is a frequency of 0.05%.

An epidemiological study by Ahlqwist, Bengtsson, Furunes, Hollender and Lapidus (1988) of 1024 women found no positive correlations between number of amalgam fillings and number of symptoms, or between number of amalgam fillings and prevalence of specified single symptoms or complaints.

A Danish study by Meurman, Porko and Murtomaa (1990) investigated 20 patients who allegedly had amalgam-related symptoms. While the results showed that the group suffered more medical illnesses and chronic craniofacial pain than controls, there was no difference in any other clinical, allergic responses, salivary chemical or microbiological findings between this group and the control group. In the five patients who gave blood samples, both inorganic and organic mercury levels were below threshold values.

Michel, Norback and Edling (1989) studied whether fatigue was related to the
number of tooth surfaces of amalgam or to other factors in a group of 108 hospital workers. There was a significant positive relationship between symptoms of fatigue and psychosocial factors and the frequency of sick-leave in respiratory diseases. A positive relation was found between the number of surfaces of amalgam and age as well as smoking habits. The authors conclude that factors such as respiratory infections and psychosocial factors could explain the symptoms of fatigue other than the release of mercury from dental amalgam.

Hietanen et al (1987) found no evidence of hypersensitivity to amalgam or mercury in a group of 29 patients with suspected dental restorative metal allergy.

Lothigius, Smedberg, Angmar-Mansson and Nilner (1989) studied 80 patients for complaints related to dental restorative materials or for supposed adverse effects from mercury released from dental amalgams. A correlation was found between symptoms and lowered pH values in stimulated saliva. It is postulated that, for example, smoking and medications may reduce salivary pH and predispose to oral symptoms, although this study failed to show any demonstrable cause for the reported symptoms.

Lavsted and Sundberg (1989) found no significant increase in the percentage of individuals with symptoms in groups with increased numbers of amalgam fillings after controlling for confounding factors such as gender, social group and smoking habits.
13.2 ANTI-AMALGAM LITERATURE

This literature can generally be distinguished from scientific research and reviews because it tends to support a dogmatic and rigid view of the deleterious effects of amalgam restorations on health as an overriding premise and propounds this view in an almost obsessive, even hysterical, manner. There is a general condemnation of the dental profession and an almost religious deference to obscure tests and authorities. It is unfortunate that the anti-amalgamists suffer to an even greater degree from the blinkered view which they (with some justification) attribute to the dental and medical scientific establishments.

There are a number of organizations which support the concept of mercury toxicity from amalgam and actively produce and distribute printed, audio and video material.

The two major groups are:

- The Toxic Element Research Foundation directed by Dr Hal Huggins, a Colorado Springs Dentist and

- Bio-Probe Inc which circulates the material of Sam and Dr Michael Ziff (the latter a dentist).

Other groups in the USA include:
- American Academy of Biological Dentistry
- National Centre for Homeopathy
- International Centre for Preventive Medicine
- International College of Applied Nutrition
- American Academy of Environmental Medicine
- American Holistic Medical Association
- International Academy of Oral Medicine and Toxicology
- Foundation for Toxic Free Dentistry

In England:
- The British Dental Society for Clinical Nutrition
- The Allergy & Environmental Medicine Department of the Nightingale Hospital
In Australia:
-The Australian Society of Biological Dentistry

These groups promote the concepts of mercury toxicity and direct people to dentists in "mercury free dental practices" who will replace amalgams on the basis of mercury poisoning. Additionally they promote nutritional, supplemental and drug detoxification treatments.

Much of the published material can be found in handouts, leaflets, booklets and texts (Hanson 1983; Lohyn 1983; Huggins 1983; Ziff & Ziff 1988a,b; Black 1990) made available to dentists promoting amalgam removal and distributed through fringe or alternative health oriented societies, bookshops and book publishers. Quite often in general books on alternative health there are chapters or sections repeating the claims of mercury poisoning from amalgam (Horne 1985; Graham 1987).

The titles of these books, monographs and chapters are generally all strident and emotive, denouncing the amalgam restoration with grandiose claims of mercury poisoning causing a multiplicity of afflictions. For example:

-"SILVER DENTAL FILLINGS, THE TOXIC TIME BOMB" (Ziff 1984);
-"ARE YOUR DENTAL FILLINGS HURTING YOU? THE HAZARDS OF HAVING MERCURY IN YOUR MOUTH" (Fasciana c1986);
-"MERCURY: A FACTOR IN MENTAL DISEASE? CAN MERCURY-SILVER FILLINGS CAUSE PSYCHIATRIC SYMPTOMS?" (Huggins 1983);
-"MERCURY AMALGAM TOXICITY - A MAJOR COMMON DENOMINATOR OF DEGENERATIVE DISEASE" (Kupsinel 1984);
"INFERTILITY AND BIRTH DEFECTS. IS MERCURY FROM SILVER DENTAL FILLINGS AN UNSUSPECTED CAUSE?" (Ziff & Ziff 1987).

It must be said that some of these books are produced in a proper scientific style, well documented and with copious references (e.g. "Infertility and Birth Defects" 1987). However, it is the prejudiced and selective utilisation of data to formulate unfounded and speculative conclusions which ultimately consigns these manuals to become mere footnotes in the scientific literature.

In 'THE HEALTH REVOLUTION' by Horne (1985) there is a section on "Mercury Poisoning from Teeth Fillings". As is common in these types of books there is a presumption that mercury poisoning from amalgams is known to produce various chronic disease conditions. On this basis and quoting the names of various clinicians and authors, numerous cases are referred to in a general sense for whom health has been restored with the replacement of amalgams. A Dr Scwharzkopf is reputed to have eliminated 'cancer, erratic heart beats, pancreas weakness, erratic menstruation, headaches, thyrotoxicosis, endocarditis, hyperthyroidism, neuralgia, muscular pains and rheumatism' by amalgam replacement therapy. In a more specific vein the author himself cites a case of Mrs Gun Thoresson of Burea, Sweden, who had been gradually going blind, but after removal of amalgam regained 90% of her vision within 6 months. In this style of publication these cases and those who report them are accepted without query - they support the basic thesis and are, as such, given an unwarranted and unproven status.
A comical, but extremely illuminating, example of nonsense in the guise of science is seen in the book "HOW TO BE YOUR OWN NUTRITIONIST". (Berger 1989) The author asks 'Do you have silver fillings in your mouth? It has been shown that these can leach lead and mercury into the body over a long period. A few experts believe that a quarter of the people with serious lead poisoning get it from dental fillings.'

This epistle exposes the vacuous nature of the oft used phrase 'it has been shown' and the meaningless value of reference to 'experts' without substance. In reality lead is never a constituent of amalgam and thus it cannot be shown that lead leaches into the mouth from dental fillings. Even less compelling is the reference to the experts who contend that 25% of serious cases of lead poisoning come from dental fillings which do not contain lead.

The scientific style articles are published in journals which are either in an esoteric professional category: e.g. Journal of Orthomolecular Psychiatry (Huggins 1982; Pleva 1983; Hanson 1983) and Journal of the International Academy of Preventive Medicine (Pinto & Huggins 1976) or lay publications which promote anti-establishment or alternative approaches to health; e.g. "AUSTRALASIAN HEALTH AND HEALING...A HOLISTIC APPROACH TO ALTERNATIVES IN LIVING." (1990)

As an example of this genre an article by Siblerud (1989) in the American Journal of Psychotherapy claims that mercury poisoning from dental amalgams
may play a role in the aetiology of mental illness. The author notes initially that the study was designed to prove a preconceived thesis: 'The strong evidence linking dental amalgam with mercury poisoning and associated psychological disorders led us to design a study that evaluated the mental health of subjects with and without dental amalgams.' It is difficult then not to be cynical about the results and findings thus obtained. Two separate studies were carried out, the first with 100 college students (50 without amalgams and 50 with amalgams) who completed two mental health questionnaires and had hair and urine samples assessed for mercury. In keeping with the expected results the amalgam subjects reported being significantly less happy, having less peace of mind, rated their reading comprehension as lower, had more emotional distress symptoms with significantly more episodes of sudden anger, depression and irritability as well as suicidal tendencies and anxiety. The amalgam group had higher mercury levels in urine and hair. The urine mercury concentration of the amalgam group was 3.7 ppb as against 1.23 ppb for the non-amalgam group. The standard deviation in the amalgam group was 3.78 ppb implying a significant variation such that a proportion of this group had levels which would have approximated those of the non-amalgam group. There are narrow theoretical considerations in this study highlighted by the fact that, in assessing the results of both clinical tests (hair and urine), sources of mercury intake other than from amalgams have been scrupulously ignored. There are also questions as to whether the group with amalgams were blinded to the object of the questionnaire - it is hard to envisage a question which would allow the individuals in the amalgam group to rate their own reading comprehension that would have any objective reality
when compared with the amalgam free group.

The second study reflects a questionnaire response from 86 out of 300 patients who had undergone amalgam removal at a specific dental practice. The results of this survey are almost entirely invalid...people who have their amalgams replaced constitute a specific group with preconceived ideas about their health status, the role of amalgam as a source of mercury poisoning and assorted health symptoms, and the benefits of amalgam removal. In this study there is no control group and the placebo effect is unknown. Additionally, the respondents constitute a further select group within those who have had amalgams replaced (86/300) which should be factored into any assessment of their subjective opinions.

Thus the result that 80% felt better since removal, 86% were glad they had undergone the procedure and 88% would undergo the procedure again is to be expected given the source of the information. In order to further clarify the 14% response of those who were 'feeling worse' after amalgam removal, the following is offered: 'Of the 11 subjects who said they felt worse, 9 said they also felt better after removal. Some said they felt worse immediately after removal, then felt better. Only 3 felt worse after amalgam removal than before. On a scale of 0%-100%, the 11 subjects that felt worse said they felt 21% worse, but said they felt 47% better on the 'feeling better' question.'

The studies overall suffer from a prejudgmental attitude on the part of the author and the second study is additionally weakened by the choice of subjects. The author improperly attempts to relate the two studies assuming that the positive response in the second study to the removal of amalgam supports the
findings in the first that mental symptoms are a result of mercury toxicity from amalgam. The author further postulates that the studies support that mercury via amalgam is responsible for stress, fatigue, memory loss, premenstrual syndrome and poorer lifestyle. This article will be quoted by the anti-amalgam lobby as support for their contentions, but the results are not satisfactorily proved by impartial, objective and scientific methodology.

Siblerud in other related articles makes further postulations regarding the effects of amalgam on health. He contends that mercury from amalgam causes diseases of the mouth (Siblerud 1990a) and that mercury poisoning from dental amalgam may play a role in the aetiology of cardiovascular disorders (Siblerud 1990b). In this latter report the author claims that amalgam bearing subjects had significantly higher blood pressure, lower heart rate, lower haemoglobin and lower haematocrit. The amalgam subjects had a greater incidence of chest pains, tachycardia, anaemia, fatigue, tiring easily and being tired in the morning.

There is no equivalent research in the literature to support these findings. It is essential that the results of these studies by Siblerud be duplicated by independent researchers with proper controls and methodology.

In many instances the amalgam/mercury toxicity question is but one of a multiplicity of contentions regarding disease and ill-health emanating from current dental therapy. For example in the Feb-April 1990 Issue of the aforementioned Australasian Health and Healing which is a special issue on "Hidden Dental Health Hazards" there are, in addition to the mercury related material, articles and commentary on:
- 'The carcinogenic effects of devitalised teeth.....root canal- cancer link.....why is a root-filled tooth a major health hazard?'
- 'Wisdom teeth - heart link.....heart disease linked to tooth decay'
- 'Neural therapy.....interference fields cause chronic inflammation and osteitis in the jawbone'

Recommendations to alleviate these problems include removal of bone around current and previous extraction sites, particularly those involving amalgam restorations, root canal therapies and wisdom teeth. Additionally, these publications contain numerous testimonials from individuals attesting to the success of one or more of the recommended alternative dental treatments.

E.g. "A cure for my aching heart" and "The miracle that saved my eye" (Australasian Health and Healing 1990 p24-25).

13.3 THE MEDIA

The role of the media is a potent factor in the dissemination and propagation of the anti-amalgam point of view. While espousing a philosophy of public knowledge and professional accountability there is a tendency for the media to sensationalise and simplify complicated issues. This may be, in the first place, because the journalists do not really understand the issues and secondly, because the object is to sell papers and satisfy the public’s taste for shock and horror. This is highlighted in the continued oversimplified regurgitation of the thesis that there is a proven relationship between amalgam restorations and mercury poisoning and thence extended to an association with other diseases.
The New York Times (Dec 1990) is guilty of the grossest distortion and simplification when it states in an article "Debate flares over the safety of dental fillings": 'It has long been known that exposure to large doses of mercury can cause symptoms ranging from arthritis to depression, as well as a variety of neurological problems and autoimmune disorders, like multiple sclerosis.' The use of 'It has long been known' is simply a fallacious argument (Fearnside & Holther 1959) in that it is an appeal to authority and tradition which tends to legitimise the statement which follows and forestalls disagreement, but in no way indicates the basis or the authenticity of the authority alluded to. The remaining statement is a classic example of faulty causal generalization. Arthritis is erroneously described as a symptom of mercury exposure when it is a distinct disease state with no direct relationship to mercury and definitely not caused by mercury. Depression is a common symptom which is manifested in a multitude of disease states and is hardly specific for mercury poisoning.

Excessive mercury exposure can cause neurological damage which results in symptoms and problems of sensory and motor dysfunction. Neurological damage is also evident in other disease states including Multiple Sclerosis, Alzheimer's disease, stroke etc. Though overt neurological symptoms may be similar (e.g. tremor) there is no justification for a causal relationship between mercury and Multiple Sclerosis to be implied. There is use here of faulty analogy and post hoc (assuming the cause) reasoning. Multiple Sclerosis has been described as an auto-immune disease, although this not entirely a definite description as the cause of Multiple Sclerosis is as yet unknown. There is a widely held opinion
that mercury poisoning produces damage to the immune system. To make the statement that mercury causes autoimmune diseases such as MS is again an illogical, unproved and fallacious associative argument.

Titles such as "Heavy metal in your mouth - Hidden dangers at the dentist" (Simply Living 1990); "A Mouthful of Trouble" (Geddes 1983); "The mercury starts to rise over filling debate..But can it really cause poisoning in the mouth?" (Daily Mail 1990); "Drilling for Danger?" (Newsweek October 1990) and "Mercury in Teeth. No silver linings" (Economist February 1991), appear to prejudge the issue.

The articles in the lay press generally accept unquestioningly the anecdotal claims of cures subsequent to the removal of amalgam restorations. For example: 'The strong denials from conventional dentists and the NHMRC (National Health and Medical Research Council) also do not account for the large numbers of testimonials from people who were sick, had their amalgams removed and now feel much better...Further testimonials range from cured face aches, improved digestion, relief from chest and ear pain, to one person claiming a radical improvement in her advanced and severe muscular sclerosis.' (Simply Living 1990);

'Instances abound of patients making amazing recoveries once their mercury amalgams are replaced with non-toxic fillings, according to Lohyn.'(Geddes 1989);
'I noticed an immediate improvement in my health after the worst amalgam filling was replaced- I had a big increase in energy' (Daily Mail 1990).

It is difficult for the public (and particularly those in ill-health and looking for therapy), reading scientific material within a journalistic context, to apply objective criteria to assess;

a. hypotheses and opinions which are dogmatically avowed as if universally proven and accepted and

b. unsubstantiated assertions (both generalised and specific) of improvement in health states following amalgam removal;

In America in December 1990 the CBS-TV’s "60 MINUTES" news programme posed the rhetorical question: "Is there poison in your mouth?" The answer was sought through a series of interviews, chiefly with opponents of amalgam usage. The programme gave the impression that amalgam restorations contribute toxic levels of mercury to the body. Using anecdotal accounts the programme also suggested that removal of these fillings can cure a variety of medical diseases, including arthritis and Multiple Sclerosis.

The bias as well as factual errors in the 60 Minutes programme has been detailed in a "Special Report" sent with the January 1991 issue of the Journal of the American Dental Association (ADA 1991) and the link with Multiple Sclerosis refuted by the National Multiple Sclerosis Society (Reingold 1990).
A symposium was carried out by the British Dental Society for Clinical Nutrition in 1986 titled "Hazards in Dentistry: The Mercury Debate." and which resulted in a number of stories appearing in the media about the 'dreadful harm' that amalgam fillings were capable of causing. The editor of Dental Update commented: 'The handling of this story in the media is a stark example of the worst type of so-called "investigative" reporting. Sensationalism sells newspapers and frightening the masses appears to be considered fair game. Like many such stories, it disappears into nothingness when examined in an objective and scientific fashion.' (Dental Update 1986). The fact that the sponsors of this meeting were ICI, the makers of the tooth coloured composite material "Occlusin" seems to undermine the objectivity of the proceedings.

13.4 CHRONIC FATIGUE SYNDROME AND CANDIDIASIS

Chronic Fatigue Syndrome (previously known as Myalgic Encephalitis [ME] and also Post Viral Fatigue Syndrome) is a controversial disease entity which has been linked with mercury toxicity from amalgam restorations. Chronic Fatigue Syndrome is characterised by chronic variable and fluctuating symptoms particularly muscle fatigue and may be caused by viral infection (Ramsay 1988).

In "TOXIC CHEMICAL-FREE LIVING" (Whitmore 1990) the author recounts the history of 'an acquaintance' whose long term ME and head pain had improved radically since having her amalgam fillings systematically removed.
Candidiasis (Thrush) is a proliferation of the fungus candida albicans which can occur when normal bacterial flora are diminished by the use of antibiotics. Additionally it is suggested that with an altered immune status and/or chemical and food allergies, candida can proliferate. In "CANDIDA: A TWENTIETH CENTURY DISEASE" (Lorenzani 1986) in a section on "Causes of Candidiasis", the author implicates mercury toxicity (specifically from dental restorations) by suggesting that if the activities of the major organ systems are continuously compromised by toxic metals slowly and steadily entering the blood stream, foreign organisms, such as Candida, may find it easy to proliferate. Crook in "THE YEAST CONNECTION"(1989) in the section "Could your dental fillings be hazardous to your health?" discusses in much detail the effective adjunctive value of dental amalgam removal as anti-candida albicans therapy. In conclusion the author states: 'Should you have your dental fillings removed? I don’t know. Yet, if you suffer from chronic health disorders which haven’t improved in spite of all your efforts and those of your physicians, I feel a silver/mercury-illness relationship should be considered.' Crook quotes a monograph by Zamm (1986) titled "Anticandida Albicans Therapy: Is There Ever An End To It? Dental Mercury Removal: An Effective Adjunct"

The aetiology of both Chronic Fatigue Syndrome and candidiasis are often related to the effects of food and chemical allergies causing immunosuppression. In the case of Chronic Fatigue Syndrome a general multi-allergic base is implied whereas with candidiasis, yeast intolerance is quoted as a major factor (Griffiths
1990). In both these diseases mercury (in a general environmental sense and specifically from amalgam restorations) is often mentioned as an underlying element in the allergic/immunosuppressive/toxic background.

Occasionally a direct interrelation between Chronic Fatigue Syndrome, candidiasis and mercury from amalgam restorations is claimed and this is evidenced in an article in Interaction, the Journal of Myalgic Encephalitis Action (Viewpoint 1989) where the "Case Against Dental Amalgam" is put forward by Mr Jack Levenson, a dentist and President of the British Dental Society for Clinical Nutrition. The article relates that Dr Victor Penzer of Boston University stated: 'It appears that mercury leaking from amalgam fillings, when ingested and converted to methylmercury, can disturb the intestinal flora of saprophytes, and can encourage the growth of fungus'. This is followed by two typically vague statements: 'Reports from physicians indicate that patients who did not get well on anti-fungal therapy alone recovered after the source of mercury intoxication was also eliminated.' and 'Reports from Europe indicate that intestinal dysbiosis is often related to amalgam fillings', which are classic examples of unsubstantiated assertions. The article continues: 'Mr Levenson explained the process in simple terms. When you chew food with teeth that have amalgam fillings, the mercury is released into your saliva. When this toxic saliva is swallowed it kills off the 'good' flora in the gut, allowing the 'bad' such as candida, to proliferate.'

Mr Levenson's description may seem plausible to the person with Chronic Fatigue Syndrome who is considering candidiasis as a contributing factor in the disease, but several questions can be raised as to the scientific legitimacy of the
facts and sequences offered. Of the small amounts of mercury released from amalgam during chewing, most concern focuses on mercury vapour, some of which will be inhaled. The inorganic mercury compounds in saliva are composed of some proportion of the vapour which dissolves in saliva and perhaps actual particles of amalgam (corrosion products) which contain inorganic mercury compounds. The inorganic compounds of mercury that would exist in saliva and be ingested are minimal in quantity and also the least toxic form of mercury. Even assuming some mercury from amalgam was dissolved in saliva, there is no evidence that, after ingestion and during passage through the gut, there is any alteration in intestinal flora sufficient to depress normal bacteria and promote fungal growth. Dr Penzer's assumption that inorganic mercury is converted to the organic methylmercury is a very tenuous and speculative hypothesis, the conversion only based on an in vitro experiment and never having been established in humans. [see Sections 4.1.3 and 11.6]

Two definite facts can be accepted:

a. mercury is released from amalgam restorations during chewing and

b. changes in intestinal flora can cause candidiasis....

The subsequent inferences drawn are incorrect as well as hypothetical. The remote possibility that these assertions may be true should not be allowed to undermine the broad and major scientific body of knowledge which rejects the extended assumptions regardless of the conviction with which they are held.
Although strongly implied in the article there is actually NO evidence for the following contentions:

i. the amount of mercury released and then absorbed is significant to the body burden of mercury;

ii. mercury from amalgams can produce any degree of mercury poisoning or toxicity;

iii. all mercury released from amalgam is dissolved in saliva and swallowed;

iv. mercury in saliva ingested can affect intestinal flora and specifically reduce certain flora allowing candida to proliferate;

v. inorganic mercury or mercury vapour is converted to the extremely toxic organic methylmercury in the gut;

vi. mercury has any role in the aetiology of either Chronic Fatigue Syndrome or candidiasis;

vii. removal of amalgam restorations has any affect on candidiasis and by extension Chronic Fatigue Syndrome.

13.5 METHODS OF ANALYSIS

A number of screening measures are often employed to ostensibly assess mercury levels and provide evidence of toxicity. Some of the tests (e.g. blood, urine, hair) are potentially valid indicators if carried out by appropriate trained personnel and utilising sophisticated technical analysis. In general the testing carried out for mercury sensitivity and mercury poisoning from amalgam restorations is extremely questionable and quite often unacceptable, inappropriate and invalid. The mercury sensitivity allegedly revealed by these
tests is used as justification for the recommendation for amalgam removal.

1. Symptom Questionnaire
2. Electrical Reading of Restorations
3. Skin Patch Testing
4. Urine Mercury Analysis
5. Blood Tests
6. Hair Analysis
7. Oral Mercury Vapour Tests
8. Food and Chemical Allergy Tests

[adapted from Dodes 1988]

13.5.1 Symptom Questionnaires

Hundreds of questions elicit information on all health aspects, including areas such as cardiac, pulmonary, nervous, skin, digestive, blood, urine, emotional etc. The questions are general even when assessing specific problems and the list is so inclusive that any healthy person would find it hard not to confirm the presence of some of the symptoms or conditions.

13.5.2 Electrical Reading of Restorations and Sequential Removal of Amalgams.

A quasi-scientific metering device assesses the electrical status of the amalgam restorations and the "negative" restorations are accorded pre-eminence in the sequence of removal. Ostensibly this device is recording electric potentials which is presumed to be a measure of corrosion rate and thus the release of mercury, but in the first place the device does not accurately measure this parameter and in the second there is no scientific evidence that corrosion of amalgams is a significant factor in the body burden of mercury. The sequence of removal of amalgams is claimed to be important in the release of mercury from the tissues. Electrical potential testing and sequential removal of amalgam restorations is
even criticised by one of the major patrons of mercury toxicity from amalgam restorations. Ziff and Ziff (1988b) state: 'There is no scientific data to support the use of sequential removal. More importantly, there is absolutely no scientific data to support the statements being made by the proponents of sequential removal that 'If your dentist doesn't use sequential removal it will cause the mercury to remain locked in the tissues'. Moreover, it has been well established scientifically that precise measurements of these electrical potentials is not possible.'

[See also Section 13.6 Electrogalvanic Effect and Oral Galvanism]

13.5.3 Skin Patch Testing

Skin patch testing can be used to determine allergic and hypersensitivity responses to materials such as mercury but they are not able to test for mercury poisoning (toxicity), micromercurialism or non-allergic hypersensitivity. Skin tests are not valid to assess for relationships between general symptoms of ill health and amalgam restorations.

Fisher (1985) notes that: 'Patch tests with mercury chloride are notoriously unreliable because even dilute solutions, such as 0.01% aqueous solutions, may show 'irritant' patch test reactions in many normal controls. The North American Contact Dermatitis Research Group has determined that the proper test for allergic mercury hypersensitivity is 5% ammoniated mercury. Even a positive patch test with 5% ammoniated mercury is meaningless as far as mercury "poisoning" is concerned. Hence the removal of a mercury amalgam dental filling because of a positive patch test is irrational in suspected cases of mercury "poisoning".'
The "Merc-Kit", a mercury patch test kit which is marketed for dentists to test for mercury hypersensitivity and toxicity, suffers from a number of flaws which virtually invalidate its use:

- use of 0.02% mercuric chloride...an inappropriate and irritant allergen;
- Unfounded extrapolation of interpretive factors to include systemic as well as cutaneous responses. Minor changes in blood pressure, pulse and body temperature, as well as indigestion, fatigue, depression, redness of eyes etc are considered as significant sequelae of the patch test;
- the adhesive bandage may also act as an irritant

The large percentage of positive responses (20-25%) reported with the use of the "Merc Kit" has been one of bases on which diagnosis of mercury poisoning is made and the removal of amalgams promoted and justified (Mackert 1985).

Results of skin patch testing are inconsistent, with many false positive and negative reactions. Patch testing must be performed by professionals (dermatologists and immunologists), and interpretation is complex and requires expertise. Additionally the reaction of skin and oral mucosa may differ.

[Also see Section 10. Allergy to amalgam and mercury]

13.5.4 Urine Mercury Analysis and
13.5.5 Blood Tests

These tests are only accurate when performed by medical personnel and assessed in proper pathology laboratories. Urine and blood tests are significant for group analysis on a linear long-term basis, but highly variable for individuals. Serial
monitoring of blood and urine may allow for assessment of exposure by individuals, with urinary levels reflecting steady-state exposure and blood levels indicating recent exposure. The inorganic component of the blood concentration of mercury (which in part would reflect mercury vapour from amalgam) should be differentiated from the organic component (which would generally reflect methylmercury exposure through fish consumption).

Home urine testing with ultra-violet light is advocate by some clinical ecologists as a test for food and chemical allergies. This involves taking a urine sample some three hours after eating a particular substance. *If the urine is pink or red this is said to indicate porphyria (mercury toxicity) which is also linked to an allergic reaction* (Griffiths 1990). There seems to be no medical or biochemical basis for these assumptions.

13.5.6 Hair Analysis

The legitimate use of hair analysis by qualified researchers should not be confused with the misuse of hair analysis to assess nutritional status or diagnose toxicity to various trace elements (Langan, Fan & Hoos p873 1987). Hair samples from two healthy teenagers were sent to 13 commercial laboratories performing multimineral hair analysis. Laboratories had contradictory normal values for the minerals and *most reports contained computerised interpretations that were voluminous, bizarre and potentially frightening to patients. Literature from most of the laboratories suggested their reports were useful in managing a wide variety of diseases and supposed nutrient imbalances. However commercial use of*
hair analysis in this manner is unscientific, economically wasteful and probably illegal" (Barrett 1985).

Sherertz (1985) questions the usefulness of hair analysis on the variability in environmental effects (hair care, occupational exposure and geographic location), differing growth rates (health, drug effect, age, gender) and lack of standardisation in analysis techniques. She concludes: 'Little has changed in this regard since the editorial in 1974 in JAMA, in which Lazar, reflecting the opinion of the American Medical Association's Committee on Cutaneous health and Cosmetics, stated "present scientific knowledge does not support the use of metal levels in hair for broad sophisticated, subtle diagnostic purposes...and certainly, hair analysis is not desirable for routine use."

Hair is recognised as an indicator medium for exposure to methylmercury, but is not suitable to indicate exposure to inorganic mercury and mercury vapour.

13.5.7 Oral Mercury Vapour Tests

A probe inserted in the mouth after mastication or stimulation measures mercury vapour levels. The mercury vapour levels thus measured do not reflect a continuous pattern of exposure and are highly variable. Carried out in dental surgeries, where ambient mercury vapour levels may be above normal, the results may not specifically reflect mercury vapour emanating from the patient's amalgams.

The "Jerome Mercury Vapor Analyzer" (Jerome Instruments, Jerome, AZ, USA) is an instrument commonly used to estimate mercury vapour levels released from
amalgams and additionally estimate potential body burden of mercury. It is based on the absorption of mercury by a thin gold film. The accuracy of the Jerome Analyzer is, at best, +/- 15%, with repeats agreeing to +/- 5 μg/m3 (Akers 1991).

WHO (1991) notes a standard deviation of 3-10% for the Jerome Analyzer but notes that: 'at higher mercury concentrations, the films become saturated with mercury and precision decreases. There is no data on the accuracy of the method when used in actual field studies by Svare et al (1981) or Vimy & Lorscheider (1985a,b).'

Ziff & Ziff (1988b) in the booklet "THE HAZARDS OF SILVER/MERCURY DENTAL FILLINGS", in the discussion on mercury vapor analyzers (and specifically the much touted Jerome Mercury Analyzer) admit: 'Again unfortunately, the degree of validity of these equations is still controversial, as is the very use of these analyzers for dental mercury exposures.'

Another type of direct reading mercury vapour detector is the "Bacharach Mercury Sniffer" (Bacharach Instrument Co., Pittsburgh, PA, USA) which is based on the use of ultraviolet light. Correct readings can be distorted by organic acids, dust, cigarette smoke and water vapour. The machine should not be used for sampling in the mouth (Cooley & Young 1984).

To properly detect vapour from the amalgam the tooth must be dry and this does not reflect the normal status in the mouth where the teeth are coated with saliva. Perhaps this fact may account, at least in part, for the disparity between
some of the high readings of intra-oral mercury release and the subsequent poor correlation with indicator media levels and clinical effects.

13.5.8 Food and Chemical Allergy Tests

Food and chemical allergy tests are often used to determine a hypersensitivity to mercury and other heavy metals.

i. Vega Test - 'works on the principles of Bio-Energetic Medicine. It looks at the body from an electrical (bio-energetic) sense. It combines aspects of both acupuncture and homeopathy.' (Graham 1987). Glass phials containing foods and chemical are placed in a machine and an electric current applied to the finger or toe of the subject. The sound from the machine occurs when there is a drop in measured resistance and indicates the substance as sensitive or toxic. The relationship of skin impedance to food and chemical sensitivity is not scientifically validated nor is there any indication that the measurements produced have any meaningful connotation.

ii. Applied Kinesiology - 'also works on the principles of Bio-Energetic Regulatory Medicine. It tests the reaction of muscles as a way of detecting food allergies and nutrient imbalances.' (Graham 1987). The subject holds a glass phial containing a particular substance and tries to raise his arm. The tester, by putting pressure on the muscles of the raised arm, is able to identify (by relative strength and weakness of the arm) those foods and substances to which the patient is allergic.
This test is also conducted by placing the substance to be tested under the tongue after chewing (Griffiths 1990).

The Vega and Applied Kinesiology tests are the epitome of quasi-science and would be comical were it not for the fact that they are presented as authentic and proper and the public cannot distinguish legitimate medicine from fraudulent quackery. This is highlighted in the letter referred to in Section 17.2 relating to removal of amalgams for Multiple Sclerosis.

13.6 ELECTROGALVANIC EFFECT AND ORAL GALVANISM

A related claim to mercury toxicity from amalgam restorations is that a combination of dental metals (e.g. gold and silver fillings) in combination with saliva can create an electrogalvanic effect which adversely affects the cells of the body, causing neurological dysfunction. Oral galvanism was previously thought to be induced by a short circuit resulting from contact between metal restorations, but more recently has been attributed to a flow of ions or to the "battery effect" of metal restorations.

It is common for a minor galvanic reaction to occur in the mouth initially when a mixture of metals, or a new metal, is utilised. This applies to gold, amalgam, non-precious alloys (used as an economical alternative to gold) and the chrome cobalt alloys used in partial dentures. These minor "electric shocks" occur while the new material has an active surface and until a passive (oxide or sulphide) layer forms. It is caused when contact occurs with a dissimilar metal in the
mouth, but is of short-term duration and inconvenience. Beryllium, which is used in non-precious alloys, has been alleged to be released into the mouth from dental restorations and implicated in disease states, but there are no substantive clinical reports in the literature verifying this thesis. On the other hand there are extremely large numbers of people with a mixture of dental metals in their mouths and there have been no reports of general and consistent adverse effects.

An example of the claims of oral galvanism related to ill-health is seen in "TOXIC CHEMICAL-FREE LIVING" Whitmore (1990) where the author relates that an acquaintance whose 'bizarre symptoms of head pain' had improved radically after the systematic removal of her amalgam restorations was also of the opinion that: 'the metal fillings affected the electrical charges in her body and contributed to dizziness and pain'.

Muller, Van Loon and Davidson (1990) studied 28 patients without oral complaints (galvanism, leukoplakia, oral lichen planus, toxic or allergic reactions to restorations) and measured the electrical potentials of 183 amalgams and 11 precious metal restorations. In most subjects potential differences of more than 50 mV were present between restorations. The authors thus conclude that this phenomenon is common in healthy populations.

Hampf, Ekholm, Salo et al (1987) conclude that: 'oral galvanism is primarily a psychosomatic disorder'. In this study there was no statistical difference in electrical currents, potential or energy capacity in the dental metallic restorations.
between patients and controls. The frequency of allergies and candida infection was similar to the normal population, and the mercury exposure was excluded. Of the 38 patients with signs and symptoms of oral galvanism, 68% exhibited signs of mental disturbance. Of the 20 patients referred for psychiatric investigation, 5 refused consultation, 2 were mentally healthy and the remaining 13 had varying problems ranging from neurosis to severe psychiatric disturbance.

Molin, Marklund, Bergman, Bergman and Stenman (1987) investigated 12 patients with subjective symptoms, ascribed by the patients themselves to mercury released from dental restorations. There were higher intra-oral currents in the patient group related to a control group, although no differences could be seen in the amount of selenium, glutathione peroxidase and mercury in the blood of the two groups. There was a positive correlation between total number of amalgam surfaces and plasma mercury levels for all participants.

A study by Ortendahl, Holland and Rockert (1989) analyzed levels of mercury and copper in blood, blood plasma, urine and saliva of navy divers working under water with electrical cutting equipment in order to assess any changes in the presence and level of oral galvanism and the potential for occupational health risk. They concluded that the risk of increased intra-oral electrical activity during welding or cutting activities was small.

The addition of a small amount of palladium to a high copper spherical amalgam showed less electrochemical activity (Mahler, Engle & Adley 1990).
14. HOW SAFE IS AMALGAM?

14.1 REVIEWS OF THE LITERATURE

A number of authors have, in recent years, reviewed the literature and the evidence for mercury toxicity from amalgam restorations.

Reinhardt (1988) in a risk assessment analysis of mercury exposure from dental amalgams concludes that: 'the margin of safety for mercury toxicity in humans is approximately 8-30 fold.' The author's figures are calculated using as a basis a daily dosage range of 8-29 μg of mercury which are taken from the works of Brune and Evje (1985) and Vimy & Lorscheider (1985). Current opinion is that these figures are an overestimate and that the daily dosage from amalgams is more in the region of 1-3 μg. Thus the margin of safety is actually 80-240 fold. Additionally, the use of 50 μg/m$^3$ as an occupational TLV in the calculations may need to be reduced by a factor of 2 [See Section 8]. This then would alter the safety margin to 40-240. The author notes that in deriving estimates of acceptable daily intakes of noncarcinogens a tenfold safety factor is an arbitrary margin established to protect sensitive members of the human population. Thus from this analysis the mercury from amalgam does not constitute a risk for mercury toxicity.

Langan, Fan and Hoos (1987) conclude that: 'There is no evidence in the scientific literature that the minute amounts of mercury vapour that may be released from amalgam restorations cause mercury poisoning. Allergic reactions to mercury and
other constituents of amalgam have been documented, but are exceedingly rare. Finally, dental amalgam, which has been used extensively for more than 100 years, has an exemplary record of safety and benefit to the dental patient.

The conclusions of Ely and Cox (1988) were that: 'At the present time, it must be concluded that there is insufficient evidence to justify claims that mercury from amalgam restorations has an adverse effect on the health of the vast majority of patients, excepting allergic reactions in a few individuals sensitive to amalgam constituents.'

Ironside (1988) comments on the fact that: 'the studies conducted by well credentialed researchers and published in well credentialed journals with unbiased editorial review panels present overall a picture where the role of mercury from dental amalgam as a potent neuro-toxin to the patients carrying such restorations is not near as harmful as some would want us to believe.'

Dodes (1988) makes the judgement that: 'In critically evaluating some of the theories, diagnostic tests, and treatments promoted by the anti-amalgam fringe, I have found a lack of any valid scientifically sound data to support the contention that amalgam is dangerous, much less that it causes serious disease.'

Enwonwu (1987) states that: 'The potentially serious health risks of mercury notwithstanding, there are as yet no unequivocally convincing published data linking any specific major human health problem to mercury vapour derived mainly from dental amalgam restorations which are prepared and inserted according to
recommended standard procedures.'

Gettleman (1986) notes that: 'although the increases in mercury vapour in the oral cavity have been shown to be statistically significant, disease or pathology has not been demonstrated as a result of mercury toxicity.'

Akers (1989) asserts that: 'There is no statistically reliable scientific study which links amalgam restorations with specific disease states. This excludes the well documented but rare cases of allergy to mercury. The current 'conflict' is not due to a problem with amalgam, but rather to the perception of a problem by extreme minority 'scientific' opinion and the media. The mercurophobes have, on occasions, been exposed as fraudulent, scientifically inaccurate and philosophically paradoxical. This is a social, not scientific, "debate".'

Smart (1985) concludes that: 'the dangers to the patient would seem to be negligible'.

Balevi (1988) notes that: 'It appears that any risk of mercury in amalgam to the dental health team and the general public is acceptably low. Until such time as low levels of mercury are definitively linked to ill-health, we must assume that the use of mercury amalgam is safe. However we should be diligent in protecting our patients and ourselves from mercury in our working environment, especially in light of the percentage of offices where mercury levels exceed the recommended levels.'
Clinical Research Associates reported on "Silver amalgam and its alternatives - 1991" (Clinical Research Associates 1991) and concludes: 'Empirical evidence and minimal scientific research support silver amalgam use. Increasing research and highly vocal antagonists discourage its use. Replacement for mercury in dental restorative materials should be sought. Currently most viable alternatives to silver amalgam appear to be cast gold and composite resins. Unfortunately, a low cost, easily placed, well researched replacement for silver amalgam does not exist in 1991. Worldwide dentistry needs less emotion and politics and more sound research on this subject.'

Eggleston (1989) considers that the belief that amalgam has over 100 years of exemplary record of safety and benefit to the dental patient does not reflect the concern generated by recent additions to the literature on dental amalgam. This review highlights the author's concerns regarding the risk of toxic accumulation of inorganic mercury and hypersensitivity in dental patients and dental personnel, and the hazardous waste disposal of mercury. This review does not support the general acceptance of amalgam and its safety and the author concludes: 'By necessity, the use of amalgam will continue until a true substitute is available. When dental amalgam is recommended, dental patients and personnel have the right to informed consent regarding the hazards of dental amalgam as well as the benefits.'
14.2 NATIONAL, INTERNATIONAL RECOMMENDATIONS, DENTAL PUBLICATIONS

Support for the safety of amalgam has been underwritten by the vast majority of national and international dental bodies:

- **Federation Dentaire Internationale (1988)** In their "Recommendations on dental mercury Hygiene" (Federation Dentaire Internationale 1988) the FDI state: 'Mercury is a potential health hazard in dental practice. Available evidence suggests that the presence of amalgam restorations does not constitute a significant hazard to the patient, except in rare cases of hypersensitivity.'

- **Federation Dentaire International (1989)** In the FDI's "Safety of dental Amalgam" (Federation Dentaire Internationale 1989) they conclude: 'There is no documented scientific evidence to show adverse affects from mercury in dental amalgam restorations, except in rare cases of mercury hypersensitivity. Therefore, except for individuals sensitive to mercury, there is no reason why a person would seek at this time to have amalgam restorations replaced. Indeed, the effect of such a procedure and further restorative operations could be detrimental to a patient's oral health and cannot be justified. There is no alternative to dental amalgam which has been proved to give durable restorations at a comparable cost, and the routine use of composite as an amalgam substitute cannot be endorsed.'
- National Health and Medical Research Council of Australia. (Statements in 1984, 1986, 1989) The 1986 "Recommendations in Dental Mercury Hygiene" state that: 'Mercury is a potential health hazard in dental practice. Available evidence does not suggest that the presence of amalgam restorations constitutes a significant hazard to the patient, except in the rare cases of hypersensitivity."


**In summary they found that:**

- no data had been published in scientific journals which supported claims that dental restorations are a source of mercury poisoning;
- a small proportion of the population may have an allergy to mercury;
- replacement of amalgam restorations is only justified when the restoration has failed.

The authors also recommended that: 'patients should be tested for allergy or hypersensitivity to alternative dental materials used to replace silver amalgam restorations before these materials are inserted.' This recommendation needs to be clarified further as it should really only apply to those who are having amalgams replaced for reasons of true hypersensitivity and for whom it would be sensible to make prior assessment of the allergic potential of the replacement material.

As a broad and general application, allergy testing is impractical and unnecessary.
This review was also the core of the feature article in the April 1991 issue of the Asia Pacific Dental News (Asia Pacific Dental News 1991).

- The American Dental Association has recently (American Dental Association April 1990) published an overview of the subject in "When your patients ask about mercury in amalgam" which aims at educating the dental profession such that they can accurately advise patients about mercury in amalgam. The American Dental Association maintains that amalgam restorations are safe and that except in individuals sensitive to mercury there is no reason why a patient should have his amalgam restorations removed and replaced by composite fillings. The ADA warns that any dentist who advocates the removal of amalgam fillings for the alleged purpose of removing toxic substances or for health reasons is acting unethically and violating principles of ethics and codes of professional conduct.

The American Dental Association in January 1991 in response to a "60 Minutes" television programme provided a supplement to the Journal of the American Dental Association. In this is outlined "What '60 Minutes' didn't tell you" concluding with: 'The American Dental Association continues to believe that dental amalgam is a safe and effective restorative material.' (American Dental Association 1991).

- The Canadian Dental Association There are no definitive studies or case reports published in refereed scientific journals supporting the statements that dental amalgams are the cause of mercury toxicity. There is no data to suggest that the removal of amalgam restorations should be performed in an attempt to treat

- The American National Institute of Dental Research. 'Based on the available research, the NIDR concludes that dental amalgams pose no known health risk to individuals who are not sensitive to the materials. At this time, there is no reason for recommending either the discontinuation of dental amalgam from patients who have no demonstrated hypersensitivity to mercury or other components of amalgam.' (National Institute of Dental Research 1991).

- Stamm (Dean of the School of Dentistry & Chief of the Dental Research Centre, University of North Carolina, USA) (American Dental Association 1991) states: 'The contribution of mercury from amalgam to the overall mercury level in the body has never been demonstrated to be sufficient to cause or contribute to toxicity reactions.' One might however question this author's toxicological hierarchy of mercury wherein he claims that elemental mercury is the least toxic of the states of mercury and the implication that dental amalgam fillings containing elemental mercury are thus safe. There seems here an unfortunate choice of terminology and simplified conclusions which do not adequately express the particular toxicity of mercury vapour released from amalgam and the degree of uptake which inhalation provokes.

A similar problem in terminology occurs in an article by Katz (Associate Dean of McGill University, Faculty of Dentistry, Montreal, Canada) titled "Unwarranted and Unprofessional: the Superfluous Removal of Clinically Acceptable Amalgams" (Katz 1991) which is an impassioned and generally lucid
condemnation of the anti-amalgam point of view. The statement: *The inorganic forms of mercury, particularly the inorganic form used in dentistry, carries the least potential for harm, even if it leaches out from set fillings*' seems to imply (incorrectly) that all mercury released from amalgam is in the form of inorganic compounds, ignoring the existence of the high percentage uptake and the potential toxicity of mercury vapour.

- Heydt (1988), Editor of the Journal of the Dental Association of South Africa states: *There is no alternative to dental amalgam which has been proved to give durable restorations at a comparable cost, and the routine use of composite as an amalgam substitute cannot be endorsed.‘


The nine members of this scientific advisory committee was unanimous in its recommendation that dental fillings containing mercury are safe for most people. They also recommended that more research be done to allay the fears of the public that the fillings can cause health problems.

Additionally, independent organizations such as the Consumer’s Union of USA in their Consumer Reports of March 1986 have supported the continued use of amalgam.

Within the dental literature the views in the main refute the claims of the anti-amalgam lobby and offer a supporting view for amalgam (Smart 1985; Chase

Graver (1986), while stating that: 'for the majority of patients the benefits of dental amalgam far outweigh the risks' advises dental practitioners to: 'use patients' medical histories to evaluate the possibility of systemic problems; inform patients of the toxic potential of amalgam so that they may be given the opportunity to choose another restorative material; and replace amalgam restorations with other posterior restorative materials only in patients who have a medically justified need for such replacement'. Occasionally an article in a dental journal may present more strident claims as to the dangers of amalgam (Penzer 1986; Bellman 1987; Faguy 1987; Eggleston 1989), but usually these papers and comments are presented in more peripheral literature.

14.2.1 The LEK Inquiry (Sweden)

The LEK Inquiry (referred to in some publications as "Socialstyrelsen Redovisar"), was commissioned by the Department of Health and Social Welfare in Stockholm, Sweden in 1985 and the report submitted in 1987 (Lek Inquiry 1987). The group responsible for the inquiry included lay representatives as well as scientific experts. They looked into the possible risks at low dose exposure to mercury and particularly examined the effects of mercury in dentistry. In their conclusions they make a number of statements relating to questions posed in their original brief, but it should be noted that these are recommendations not
laws, and that they represent a cautious and studied concern with unknowns. These proposals have been widely misrepresented as being legitimate support for the anti-amalgam lobby. While the authors acknowledge that alternative materials should be fostered, they present no proof of mercury poisoning from amalgam and advise caution in pregnancy only because of mercury's ability to pass across the placenta, not because of any evidence of foetotoxicity from amalgam. They promote research into amalgam as well as all other dental materials. As regards whether mercury from amalgam could cause mercury poisoning and mercury allergy they point to the possibility that allergic responses such as contact eczemas and oral lichen planus may be associated with amalgam. In respect of mercury poisoning, because of the non-specific nature of the symptoms, the low level of the exposure from amalgam and the few cases occurring, the authors question whether such cases do exist. They mention the possibility of birth defects, but note that: 'this hazard is not verified experimentally or epidemiologically'.

The question of whether there exist any documented case histories proving firstly that mercury from amalgam caused symptoms of mercury poisoning and allergy and secondly verifying the alleviation of symptoms by removal of amalgam fillings, was answered thus: 'There are no epidemiological surveys available which permit conclusions regarding the effects of amalgam.' While the authors accept that many people believe their symptoms are caused by amalgam and some studies have described improvement after amalgam replacement, they note that: 'The inquiry finds that these studies are not controlled in such a way that any conclusions can be drawn'.
In setting future guidelines the authors make the following recommendations:

- 'Amalgam is from a toxicological point of view an unsuitable material for tooth fillings. The group of experts because of this would like to suggest to the Department of Health and Social Welfare the following action regarding the use of amalgam;

- The Department is proposed to stimulate the development of new materials for tooth filling which are technically and biologically/toxicologically satisfactory. When such materials are available, the group of experts suggest that the use of amalgam should be discontinued;

- Because of the potentially damaging effects on foetii, it is proposed that the Department should advise against extensive amalgam works during pregnancy. If the treatment of teeth has to take place during the pregnancy, filling materials of the cement type should primarily be used.'

The authors do note that all materials for tooth fillings should go through a biological/toxicological examination, citing gold alloys, titanium and ceramics as satisfactory at the current level of knowledge. Dental cements and 'above all' composite materials should be the subject of further examination.

One of the major recommendations of the Lek Inquiry was to caution against extensive amalgam work during pregnancy. There is little evidence that inorganic mercury is a danger to the foetus, whereas methylmercury is known to cross the placenta and cause adverse affects to the developing foetus. The term "inorganic" is often used to indicate "non-organic" and can include elemental mercury vapour as well as inorganic compounds. However if inhaled mercury vapour follows the same kinetics in crossing the placenta as it does in the blood brain barrier, then
before oxidation in the blood to the mercuric ion there would be a passage of some elemental mercury vapour into the foetal tissues where final oxidation would occur. Thus, though the placenta may be a barrier to inorganic mercury compounds this says nothing of the passage of "inorganic" elemental mercury vapour to the foetus.

The World Health Organization in 1980 recommended that the exposure of women of child-bearing age to mercury vapour should be minimised due to the fact that elemental mercury readily passes the placental barrier.

A recent study by Klemann, Weinhold and Strubelt (1990) gauged the effects of amalgam fillings in the mother on mercury concentrations in amniotic fluid and breast milk. In 95 women there were no correlations between the surface area of dental restorations and the concentrations of inorganic mercury in amniotic fluid and, as well, no correlation between amalgam fillings and total mercury in maternal blood and breast milk. The authors conclude that: ‘maternal amalgam fillings are of no importance for the mercury load of the foetus and the neonate.’

Sections of these two reports appear to have been prepared in a less than impartial manner and present an incomplete, unbalanced and overstated view of the extent and danger of mercury exposure from amalgam restorations.

In WHO 1991 Section 1.9 "Effects on Humans" of Section 1 "Summary and Conclusions", the following appears:

'Recently, there has been an intense debate on the safety of dental amalgams and claims have been made that mercury from amalgam may cause severe health hazards. Reports describing different types of symptoms and signs and the results of the few epidemiological studies produced are inconclusive.' This statement implies that no studies have shown that "severe health hazards" are not caused by mercury from dental amalgam. Similarly Section 9.7 (Dental Amalgam and General Health) is incomplete and unconvincing in its attempt to clarify the question of whether amalgam has any effect on general health. There are no reports in the scientific literature where severe health hazards can in any way be attributed to the mercury from amalgam. The studies by Kallus (1985), Ahlquist et al (1988), Meurman et al (1990), Michel et al (1989), Hietanen et al (1987), Lothigius et al (1989), Lavsted and Sundberg (1989) [reviewed in this text Section 13.1] and Hampf et al (1987) [reviewed this text Section 13.6], all failed to show any causal relationship between amalgam restorations and the symptoms
reported. Additionally none of the symptoms studied could in any way be regarded as "severe". It is only in the peripheral literature and health publications that the spectre of "severe" sequelae (such as Multiple Sclerosis, cancer, birth defects) from amalgam is proposed. The use of the word "inconclusive" to describe the reports and results is not only misleading but adds further emphasis to the possible validity of the claims of "severe health hazards".

The approach of WHO 1991 should be contrasted with that of the National Institute of Dental Research (1991) which states: *There is no scientifically sound evidence linking mercury in dental amalgam to multiple sclerosis, arthritis, mental disorders, or other diseases. Despite a number of case reports and anecdotes implicating amalgam in systemic diseases, there have been no controlled studies demonstrating adverse effects of amalgam restorations on human health.*

Reproduced below in Table 11 is Table 2 of the WHO Report 1991, (originally published as Table 4 in WHO Environmental Health Criteria 101 - Methylmercury 1990) which gives estimated daily intake and retention (in parentheses) figures for mercury in the general population not occupationally exposed to mercury.

<table>
<thead>
<tr>
<th>EXPOSURE</th>
<th>ELEMENTAL MERCURY VAPOUR</th>
<th>INORGANIC MERCURY COMPOUNDS</th>
<th>METHYLMERCURY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>µg Hg</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Air</td>
<td>0.030 (0.024)</td>
<td>0.002 (0.001)</td>
<td>0.008 (0.0064)</td>
</tr>
<tr>
<td>Food</td>
<td>0.0002 (0.001)</td>
<td>0.008 (0.0064)</td>
<td></td>
</tr>
<tr>
<td>Fish</td>
<td>0.600 (0.042)</td>
<td>2.4 (2.3)</td>
<td></td>
</tr>
<tr>
<td>Non-fish</td>
<td>3.6 (0.25)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Drinking Water</td>
<td>0.050 (0.0035)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Dental Amalgams</td>
<td>3.8-21 (3-17)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>3.9-21 (3.1-17)</td>
<td>4.3 (0.3)</td>
<td>2.41 (2.31)</td>
</tr>
</tbody>
</table>

Table 11  Source: WHO (1990, 1991)
It is interesting that a range is given for mercury vapour from amalgam whereas for methylmercury an average low figure is quoted. Therefore this table gives an unbalanced view of the contribution of the different forms and sources of mercury to the daily dosage of mercury. In each of the categories in Table 2 (elemental mercury vapour, inorganic mercury compounds and methylmercury) the figures quoted can be criticised or at least questioned not only in terms of the amounts cited but the sources used (and ignored) and the selective use, or non-use, of ranges.

14.2.2.1 Elemental Mercury Vapour.

Section 5.1.1.1. in the WHO Report 1991, which discusses human studies on exposure from dental amalgam, ignores the trend in recent studies to lower levels of mercury being recorded as released from amalgam and more importantly the chorus of independent studies which have criticised earlier reports and particularly the measurements of Vimy and Lorscheider as being up to 16 times too high and grossly inaccurate. Even within the WHO 1991 document in 2.4.2. there are questions raised as to the accuracy of the sampling methods of Svare and Vimy & Lorscheider.

It should be noted that this document was originally produced as a draft Internal Technical Report in December, 1988 (WHO 1988) and in that version it was stated: (p10) *In some studies the authors have undoubtedly overestimated the uptake of mercury, as in the papers by Vimy and Lorscheider (1985 a,b) and Vimy et al (1986).* In the current version (WHO 1991) this has been deleted and replaced with a meaningless dilution: *some studies may have overestimated and others underestimated the daily dose of mercury, while others may have*
underestimated or overestimated the mercury uptake.' In fact the text seems to reinforce the results of the earlier studies and glosses over or ignores those by Mackert (1987), Berglund (1988, 1990), Olsson and Bergman (1987), Wallis (1986, 1988), Langworth (1988), Snapp (1989) and Derand (1989) all of whom either found the daily dose of mercury from amalgam to be 3 μg or less or criticised the overestimated calculations and conclusions of Vimy & Lorscheider.

[See Table 7 Section 11.4 this text]

The estimates of 3.8-21 μg Hg intake per day (3-17 μg Hg retention per day) for mercury vapour from dental amalgams are originally quoted in WHO 1990 Section 5.1.1. and in Table 4, (WHO 1990 p38) with no references or indication as to the basis of the figures.

It can be deduced that the source of these figures is as follows:

a. The author of WHO 1990 is Dr T. Clarkson (WHO 1990 p9);


<table>
<thead>
<tr>
<th>Source</th>
<th>Total Daily Uptake μg Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Svare et al 1981</td>
<td>17.5</td>
</tr>
<tr>
<td>Abraham et al 1984</td>
<td>8.0</td>
</tr>
<tr>
<td>Patterson et al 1985</td>
<td>2.5</td>
</tr>
<tr>
<td>Vimy &amp; Lorscheider 1985b</td>
<td>2.9</td>
</tr>
</tbody>
</table>

Table 12 Source: Clarkson, Friberg, Hursh & Nylander (1988)

The range of figures 2.9-17.5 μg Hg/day is almost identical to the range 3-17 μg Hg/day quoted in WHO 1990, 1991 as retention rates. The significant feature
of this data is that it does not incorporate material subsequent to 1985 (referred to above) and is thus grossly out of date. Additionally the figure of 2.9 μg Hg/day quoted for Vimy & Lorscheider (1985b) is only 10% of that calculated by the original authors as the daily dose for subjects with twelve or more occlusal amalgam surfaces (29 μg Hg/day). In Clarkson et al (1988) the recalculation of the results of Vimy & Lorscheider parallels the current view of overestimation. However the work of Vimy & Lorscheider is given a high profile in the text of WHO 1991.

It is not mentioned that Svare and Abraham are research collaborators, which could account for the duplicated high release rates in their two studies and which are the basis for the calculation of daily dosage by Clarkson et al (1988).

c. Further supporting evidence for this publication being used as resource material for WHO 1990,1991 is that Figures 3,4,5,6 and Table 5 are directly reproduced from Clarkson et al 1988 in WHO 1991

d. The author of the first draft of WHO 1991 is Dr L. Friberg one of the co-authors with Clarkson in Clarkson et al 1988

e. Table 2. in Clarkson et al 1988 p256 was included in the draft copy WHO 1988 but was not in the final publication WHO 1991

f. Dr M.J. Vimy was a member of the WHO Task Group involved in the production of WHO 1991.

The animal experiments cited in 5.1.1.2 of the WHO Report 1991, reflect almost exclusively the work of Hahn (1989) and Vimy (1990) and Summers (1990) who are research collaborators. The experiments conducted by this group at the University of Calgary have been widely criticised but the text of WHO 1991
fails to acknowledge this fact.

14.2.2.2 Inorganic Mercury Compounds:
The level of mercury intake from non-fish foods is given as 3.6 \( \mu g \) Hg/day. This level of inorganic mercury compounds is below the normal considered range which is 5-20 \( \mu g \)/day. [See Table 1 in Section 6.3 this text].

Section 5.2.1. of WHO 1990 states: *The estimated dietary intake of inorganic mercury of 4.3 \( \mu g \)/day is the least reliable of the estimates in Table 4. Data are not available on the species of mercury in most foodstuffs. In addition, the figures for dietary intake of total mercury come from only two countries, Belgium and the USA.* In fact the two Belgian studies had total mercury intakes from foodstuffs of 13 \( \mu g \)/day and 6.5 \( \mu g \)/day. It is the low USA figure of 3.5 \( \mu g \)/day which is given maximum weighting in the calculations. To achieve a mean figure the two Belgian figures are averaged and then the resultant amount averaged again with the USA figure. This produces a 1.1 \( \mu g \)/day less amount than if the three studies were averaged in a single calculation, which is a 14\% difference. In WHO 1990 a Polish study by Szprezinger-Juszkiewicz (1988) giving a figure of 15.8 \( \mu g \)/day is quoted but not incorporated in the calculations used to formulate the final figure of 3.6 \( \mu g \) Hg. Given the admitted unreliability of the figure quoted, the absence of a range of intake figures in this category is curious.

14.2.2.3 Methylmercury
The use of a figure of 2.4 \( \mu g \)/day methylmercury is representative of a low fish consumption and this is conceded in the text of WHO 1990 Section 5.2.1. As well mention is made of communities where fish consumption produces intakes
Studies have shown that the daily dose of organic mercury (presumably the bulk of which is methylmercury) is in the range of 2.4-80 μg/day depending entirely on fish consumption. [See Table 1 in Section 6.3. this text]. Again the absence of a range to properly indicate the intake of methylmercury skews the significance of the results.

Thus the three categories show inconsistencies in the way figures are presented, giving a false impression that the bulk of mercury intake and retention is a reflection of mercury vapour from amalgam restorations.

It is disturbing that these two documents (WHO 1990, 1991) have actually added to the confusion surrounding the release of mercury from amalgam, its significance as regards body burden and the question of toxic effects on health. The data regarding mercury release from amalgam is dated, the comparison of dental sources of mercury to other dietary sources is misleading and the indecisive conclusions regarding health effects from amalgam reflect poorly on the scientific credibility of such an august body as the World Health Organization.
14.3 The Views of Dentists

In a survey of some 10,000 dentists, Clinical Research Associates Newsletter (1990) reports dentists attitudes about mercury use in dentistry:

- Highly concerned about mercury use....6%
- Concerned about mercury use........33%
- Not concerned about mercury use.....16%
- Not concerned: feel anti-amalgam movement is overdone..............45%

Additionally 94% of respondents use amalgam as their major class 2 restorative material. The number of respondents that do not use amalgam at all had increased from 3% in the 1985 survey to 6% in 1990.
15. REMOVAL OF AMALGAM

15.1 REMOVAL OF AMALGAM RESTORATIONS

The removal of amalgam restorations has been recommended as a method of eliminating the source of mercury poisoning which is propounded as the cause of a variety of diseases. The anti-amalgamists are preoccupied with the effects of mercury from one source...amalgam restorations. Both in the studies and hypotheses which implicate mercury from amalgam they make little or no provision for mercury from other sources. The contribution of methylmercury from fish and inorganic mercury from topical medicines, cosmetics etc is generally ignored, both as a source of body burden mercury and as a cause of hypersensitivity to mercury. The removal of amalgam may eliminate one source of low level chronic exposure, but may not pivotally alter the total body burden of mercury.

In assessing the merits of the procedure of amalgam removal to eliminate mercury poisoning there are a number of considerations:

1. There is no evidence of difference in the incidence of diseases nor in longevity between people who have amalgam restorations and those with no amalgam restorations.

2. Edentulous people are in general not healthier than people with amalgam-filled teeth and specifically do not show less evidence of neurological disease
nor improvement thereof.

3. Except for individual anecdotal evidence (which is usually short-term) there are no studies which indicate that people who have amalgam fillings removed experience any lasting improvement in health.

One report from Drouet, Le Sellin, Bonneau and Sabbah (1990) describes a single patient whom the authors observed had improvement from asthma after removal of dental amalgam. The authors suggest that mercury acts in this case as a respiratory tract allergen rather than the normal mucosal or systemic type hypersensitivity. This isolated report and hypothesis would need to be duplicated to validate the claims made. Additionally there would be the need for control groups to monitor placebo effects which might well account for the results.

4. Removal of amalgams would not eliminate mercury in brain tissue.

5. Removal of amalgam produces short-term high levels of mercury vapour.

Bulk removal of amalgam restorations may possibly create a significant mercury uptake by the body.

Removal of amalgam (or any other restorative material) involves damage to tooth structure and should only be effected if the original restoration is defective or if there is recurrent decay. A study by Qvist, Qvist and Mjor (1990) in Denmark assessed the reasons for placement of 5000 amalgams. 39% of restorations were placed because of primary caries and 61% were replacements of failed restorations. The failed restorations were due to secondary caries (38%), marginal discrepancies and fracture. The age of the restorations replaced ranged from 0 to 46 years and 50% of the failed restorations in permanent teeth were more than 8 years old.
Short-term elevation of mercury vapour levels occur during removal of old amalgams. The resultant replacement restorations (whether amalgam or alternate materials) are always larger and consequently remaining tooth is thinner, weaker and more fragile. The pulpal tissues are traumatised by restorative procedures with increased potential for pulpal injury and possible pulpal death.

Anusavice (Chairman, Department of Biomaterials, University of Florida) and Major (Director, Scandinavian Institute of Dental Materials) are quoted in the American Dental Association’s January, 1991 supplement to the JADA (American Dental Association 1991). They state: ‘The removal of serviceable amalgam restorations for the alleged purpose of removing toxic substances from the body is not currently considered acceptable dental practice. Presently the most biocompatible, well-established materials are direct filling gold, gold alloys and ceramics. Compared with amalgams, restorations made from these materials are much more costly and may require the removal of more tooth structure. As a general group, the plastic fillings called composites have failed thus far to demonstrate comparable strength, durability, and resistance to bacterial leakage along the tooth-restoration interface. They also show less resistance to degradation after long-term exposure to saliva and they are associated with significant shrinkage during hardening and higher levels of plaque accumulation compared with amalgam restorations.’
In respect of allergy to amalgam, van Noort and Johns in a letter to the Editor of the British Dental Journal (1985) make the point that indiscriminate replacement of dental amalgams with composites could lead to more problems than those encountered by the very small group of patients with a low tolerance to mercury.

The Chairman of the Dental health and Science Committee of the British Dental Association (Dowell 1985) notes that: 'It is misleading and cruel to suggest to worried patients that their neurological symptoms may be cured by extensive, and probably expensive, dental treatment in the absence of a responsible diagnosis. I would advise any dentist considering the replacement of amalgam fillings under these circumstances not to undertake work without obtaining the advice of a consultant neurologist.'

15.2 SUPPLEMENTAL THERAPIES AND DETOXIFICATION

There is no evidence as to benefits of food supplements, vitamins, minerals and chelating agents advocated as part of the amalgam detoxification process.

15.2.1 LIFESTYLE CHANGES

Lifestyle changes in diet and habits advocated are usually quite reasonable and commensurate with many of the recommendations of Heart Foundations etc.

These include:
- reduction of refined carbohydrates, sugars and saturated fats
- increase in high fibre food consumption
- reduction in stress
- limit alcohol and smoking
15.2.2 Replacement Protocols

Some anti-amalgamists (Ziff & Ziff 1988a) propose amalgam replacement protocols which by the addition of specific nutrients to the diet supposedly assist in a mercury detoxification process. These are initiated two weeks before amalgam replacement and continued subsequently for an additional one to two months.

*Pre Amalgam replacement;* Glutathione, Cysteine, Vitamin C, Zinc, Selenium, Vitamin B1, B complex, Magnesium, Acidophilus capsules.

*Post Amalgam replacement;* Increased glutathione, cysteine and Vitamin C. Additionally Pantothenic Acid, Vitamin E, Amino Acid complex.

There is no evidence to support the claims that any additional mercury is eliminated from the body by the use of these supplements.

The effects of Vitamin C supplements (500 mg and 1000 mg) were tested by Calabrese, Stoddard, Leonard and Dinardi (1987) on 52 adult males. Results indicate that Vitamin C did not significantly effect levels of cadmium, lead or mercury in either hair or blood samples.

15.2.3 Detoxification with Drugs

Dimercaprol and D-Penicillamine have been used as chelating agents for mercury poisoning. Intravenous EDTA is sometimes used in the USA for mercury detoxification although EDTA is not known to have a great binding affinity with mercury.
Currently DMPS (Dimaval), a water soluble derivative of Dimercaprol, is being recommended for "mercury poisoning" from amalgam. DMPS is initially given intravenously and then followed up with oral administration over a period of some months. DMPS has not been approved for use in the USA (Ziff & Ziff 1988a; Daunderer 1989).
16. AMALGAM AND ALTERNATIVE DENTAL MATERIALS

In the interests of informed consent it is important that patients are advised of the benefits, problems, risks, costs and alternatives for any proposed treatment and technique in dentistry. If amalgam restorations are being suggested, the patient should be advised that they contain mercury, with perhaps a brief overview and comment on the mercury debate and the current view of the profession on the longevity and safety of amalgam restorations.

Dental biomaterials which are used to replace damaged or lost oral tissue are in principle intended to be inert. With the exception of unalloyed gold and titanium most dental biomaterials can be expected to release constituents into the complex and highly variable environment of the oral cavity (Bergman, 1990).

16.1 AMALGAM

Amalgam has been used successfully as a restorative material for over 150 years. It was quickly taken up by dentists after its introduction by the Crawcours, who advertised the material in London in 1831 and in New York in 1833. Amalgam restorations may be serviceable from 5-15 years and are generally used in posterior teeth for strength and where aesthetics is as a secondary consideration. Dental amalgam is still the material of choice for the
repair of most medium-sized areas of decay in premolar and molar teeth. In 1983 nearly 25 million amalgams were placed under the National Health Service in England and Wales (Dental Estimates Board 1985) and it is estimated that in excess of 160 million amalgams are inserted in the USA annually (Moen & Poetsch 1970). In both countries amalgam is used for 75-80% of all posterior restorations.

Amalgam does not support surrounding tooth structure and requires significant tooth removal for proper design and placement. Amalgam is clinically well tolerated and has an established durability, generally regarded as between 5 and 15 years. A recent study showed that medium to large size amalgam restorations have a survival rate of 72% after fifteen years (Smales 1991). Dental amalgam is also successfully used as a retrograde root filling material in which case it is not exposed to the oral cavity and serves as an implant material.

The legitimate concerns surrounding the release of mercury vapour from amalgam restorations suggests that if a viable alternative restorative material to amalgam were available it should be utilised. The speculation that mercury release from amalgam restorations may be effected internally through the tooth to the pulpal and periodontal tissues has yet to be substantiated.

Potential release of mercury during insertion, removal and polishing of amalgam restorations can be reduced markedly by use of water spray and high-volume aspiration. The modern ternary amalgam alloys, which have reduced silver and tin and higher copper content, have improved properties and may reduce mercury loss during function.
16.2 ALTERNATIVE TOOTH-COLOURED MATERIALS

The tooth coloured alternatives comprise plastics, glass ionomer cements, composite materials, ceramics and porcelains. As an alternative to amalgam they offer improved aesthetics, but with significant complications in respect of technique and longevity which should be explained to patients.

In most of these techniques the material is bonded to the enamel and dentine of the tooth using an acid-etch system for adhesion. The results are very technique sensitive and the ultimate long-term integrity and quality of the bond is not entirely guaranteed by current methods and technology.

Modern tooth coloured materials (synthetic resins) which are bonded to tooth structure (particularly enamel) effect (at least initially) a measure of support to surrounding tooth. One of the great advantages of bonding technique is that only carious material need be removed and in the case of new cavities, where no previous restoration exists, a small composite restoration could be preferable to a larger amalgam.

Nevertheless the decision to replace an amalgam with a synthetic alternative should only be made at the time of necessary replacement of the existing restoration and cannot be supported on the basis of mercury toxicity.
16.2.1 Synthetic Resins
Modern synthetic resins (tooth-coloured restorations) have been successfully used in the anterior region of the mouth for the past 10-15 years. These restorations used in posterior teeth, which bear great masticatory load, are technically more complicated and more difficult to construct than are amalgams. The cost is greater and the life of the restoration is often much shorter than amalgam. Posterior composites are very technique sensitive and this coupled with polymerisation shrinkage and incomplete curing means there is more likelihood of salivary contamination, post-operative sensitivity, leakage around margins and, as well, recurrent decay is more rapid beneath these restorations. Composite resin may have its own degree of toxicity with a recent report demonstrating the presence of formaldehyde as a byproduct of the polymerisation process (Lind 1988).

The Federation Dentaire Internationale in their Technical Report 33 on the Safety of amalgam (1989) note that: 'at the present time resin-based materials cannot be used unrestrictedly in stress bearing posterior cavities. Such materials are unproved, with deficiencies particularly in abrasion resistance, stiffness and radiopacity, and should only be used in selected cases.'

16.2.2 Porcelain and Ceramic Materials
Porcelain or ceramic materials which are fabricated outside the mouth have significant advantages over the resins in respect of strength, internal integrity and aesthetics. They represent an biocompatible alternative to gold and
porcelain fused to gold crowns and perhaps indicate the future replacement alternative to amalgam. Improved laboratory techniques have created ceramic restorations which fit accurately and are less likely to fracture. In addition to being brittle ceramics can also be highly abrasive to opposing teeth. The covalent bonding of ceramics does not permit the surface mobility which characterises the precious metals.

Another weakness of these materials lies in the bonding arrangement of the restoration to tooth structure, but as marginal accuracy approaches 50-100 μ and bond strength improves, ceramic restorations will be both aesthetic and serviceable.

16.3 NON-PRECIOUS ALLOYS

Nickel is used commonly in metal dental prostheses, both in stainless steel alloy (10%-14%) and alloyed with chromium as chrome cobalt alloy (2.5%) (Burrows 1986). It is now estimated that some 20% of women and 3% of men exhibit allergic responses to Nickel. This is apparently due to ear piercing which is the initial sensitizing exposure. The addition of beryllium to Nickel/Chromium alloys produces an increased rate of corrosion. Noble elements (e.g. gold) are considered far safer than the non-precious alloys.

Intimately related to amalgam is the common use of stainless steel pins containing nickel for additional retention in larger restorations. There is little information regarding possible degradation, corrosion or galvanic reaction of the nickel containing stainless steel pins within tooth substance and amalgam restorations. "Stainless steel is strongest, but today many practitioners are avoiding
the use of nickel that is present in stainless steel. Some countries will not allow the use of nickel in humans. Titanium alloy or pure titanium pins are now available from most pin manufacturers.' (CRA 1991).

16.4 GOLD

Cast gold has long been considered the ideal dental material, being inert and biocompatible. Precious metals are ideal for the restoration of natural teeth because of the lubricating effect of the metal atoms during clenching and grinding. Gold has been the most enduring of dental materials but high costs restrict its generalised use and the current fixation by public and dental profession alike with aesthetics has limited its application. The porcelain fused to gold crowns have extended the scope of gold by virtue of the aesthetic benefits of the porcelain coverage.

The procedure whereby large amalgams are covered by cast gold or gold/porcelain restorations may offset the fears of release of mercury vapour from amalgam during function. Some 30% of build-ups for crowns is achieved with amalgam (CRA 1991). This however would also increase the possibility of electrolytic reaction between the contiguous surfaces of gold and amalgam, although no evidence exists that this is significant, continuous, or harmful. The cementation process, whereby a luting cement (e.g. glass ionomer or zinc phosphate) aids in retention of the crown to the tooth and forms a layer between gold and amalgam, may reduce the potential for this process. There appear to be no studies to determine if mercury vapour is released from
amalgam restorations which are covered by other materials such as gold.

However Combe (1986) states that: 'if amalgam comes into contact with a gold restoration, an electrolytic cell may be set up leading to corrosion of the amalgam and incorporation of mercury on the gold restoration.' This process may require saliva and aerobic conditions, neither of which may be present where gold totally covers amalgam.
17. MERCURY AS A FACTOR IN MULTIPLE SCLEROSIS

Mercury poisoning, from whatever source and regardless of whether organic or inorganic, has not been conclusively proven to be associated with, or the cause of, Multiple Sclerosis (MS). Nevertheless Multiple Sclerosis is constantly cited as being a known consequence of, or linked to, mercury poisoning from dental amalgams.

Multiple Sclerosis may briefly and for the lay public be described as: 'An unpredictable neurological disorder with its onset in early adult life, mostly affecting walking, and often with associated urinary problems' (Scheinberg 1987) or more scientifically as: 'a slowly progressive Central Nervous System disease characterised by disseminated patches of demyelination, resulting in multiple and varied neurological signs and symptoms, usually distinguished by exacerbations and remissions.'

Multiple Sclerosis is thought to affect some 70,000 people in the U.K. and perhaps 250,000 in the USA. The disease generally manifests between the ages of twenty and forty and affects more women than men. Multiple Sclerosis is the most common chronic neurological condition affecting young adults, peak of onset being at 22 years of age. Half the known cases begin before the age of 30, three-quarters before 40. Multiple Sclerosis occurs more commonly in the white
races, in cooler climates (further away from the equator) and in areas with high standards of sanitation (National Multiple Sclerosis Society of USA 1978; Multiple Sclerosis Society of Great Britain & Nth Ireland c1985; US Dept Health and Human Services 1985; Waksman, Reingold & Reynolds 1987; National Multiple Sclerosis Society of Australia 1990).

These elements have led some researchers to believe that an environmental factor is responsible for Multiple Sclerosis, but it is entirely possible that migration patterns within certain genetically predisposed groups of people could account for the geographic distribution of the disease.

The disease process is variously referred to as inflammatory, auto-immune, metabolic, vascular, viral and genetic ... all of which attest to the unknown aetiology and permit a variety of hypotheses as to initiating and exacerbating factors, as well as a host of treatment modalities (Russell 1976; Greer 1982; Swank & Dugan 1987; Graham 1987). The enigma that is Multiple Sclerosis permits it to be routinely appended to lists of "caused by" and "could be cured by". In this case those who believe that mercury from amalgam is the font of ill-health include Multiple Sclerosis as caused by mercury from amalgam, and claim that Multiple Sclerosis can be cured by the removal of amalgam. The extraordinary diversity of therapeutic modalities offered for Multiple Sclerosis are comprehensively and objectively assessed in "THERAPEUTIC CLAIMS IN MULTIPLE SCLEROSIS" (Sibley 1988).
17.1 SYMPTOMS OF MULTIPLE SCLEROSIS

Initial Symptoms

The incidence of initial symptoms are approximately as follows: (Matthews et al 1985)

i. Weakness in one or more limbs 40%
ii. Optic neuritis 22%
iii. Paraesthesia 21%
iv. Diplopia 12%
v. Vertigo 5%
vi. Disturbance of micturition 5%

Advanced Symptoms

Certain symptoms and signs are commonly present in advanced cases:

i. Weakness
   This is most commonly seen in the lower limbs. Onset varies but initially presents as fatigue or weakness on exertion, gradually increasing until present constantly.

ii. Spasticity

iii. Ataxia

iv. Sensory loss

v. Loss of bladder control

vi. Pyramidal signs ...(depression or absence of superficial abdominal reflexes, increased tendon reflexes and an extensor plantar reflex.)

Lech滕enberג (1988) notes that the systems often affected include; vision, coordination, speech, strength, sensation and bladder control and lists the common symptoms in Multiple Sclerosis as:

- Blurred or double vision
- Loss of vision in one eye
- Slurred or slow speech
- Easy fatiguability
- Psychologic changes
- Weakness or paralysis of limbs
- Poor coordination
- Shaking of limb
- Staggering gait
- Poor balance
- Dragging feet
- Numbness or pins-and-needles
- Poor bladder or bowel control
17.2 MERCURY, AMALGAM RESTORATIONS AND MULTIPLE SCLEROSIS

The two groups generally responsible for maintaining a causative link between mercury (via amalgam restorations) and neurological disease are clinical ecologists and a very small sub-group of "holistic" dentists and doctors.

Dr Hal Huggins, head of the Toxic Element Research Foundation in Colorado USA makes statements such as: 'It may well be that MS starts in the dentist's office.' (Martin 1987). Martin further quotes Huggins: 'I used to have MS or something near - but I gave it up!' (It is the mercury in his nervous system, however, which makes him unable to draw a straight line today).’ Huggins also claims that an unspecified 'European research group have allocated US $1 million to investigate these techniques in their improvement in Multiple Sclerosis and Leukaemia.' (Australian Society of Biological Dentistry 1988).

Dr M. Daundere (1989), (a German Internist) is quoted in the anti-amalgam "Bio-Probe Newsletter" claiming that Multiple Sclerosis is an 'immunodeficiency caused by amalgam' and that 'many cases of colitis (ulcerative) and some with Multiple Sclerosis improved considerably after removal of the amalgam fillings and detoxification with DMPS.'
The rationale of those who implicate dental amalgams in Multiple Sclerosis is that:

a. mercury is a neurological poison which serves as a causative or aggravating factor in neurological diseases including Multiple Sclerosis. 

Additionally they contend that:

b. mercury leakage from amalgam restorations is of sufficient dosage and toxicity to inflict damage to the body tissues and organs as well as to the immune system, causing a broad range of health problems of which Multiple Sclerosis is one.

c. removal of amalgam restorations will cure Multiple Sclerosis

Over the last 10-15 years the claims of links between Multiple Sclerosis and amalgam restorations have continued to surface periodically, attracting media and public attention. The more recent data establishing release of mercury vapour from amalgam restorations has provided further material for the proponents of this theory and stimulated additional controversy. There are many groups involved in promoting this theory and it is difficult, given the multiplicity of sources, to simply dismiss the claims as outrageous, unsubstantiated and unscientific.

The righteous, dogmatic conviction, which complements these assertions, places limits of credulity on any claim to scientific objectivity and clouds the validity of the data and case histories utilised as substantiation. Much of the research referred to in the copious anti-amalgam literature is published in obscure journals whose impartiality is questionable and the material rarely relates specifically to Multiple Sclerosis. The term Multiple Sclerosis is almost ritually
appended to lists of diseases or symptoms thought to be initiated by mercury poisoning from dental amalgam. For example a book by Stortebecker is titled "DENTAL CARIES AS A CAUSE OF NERVOUS DISORDERS - EPILEPSY - SCHIZOPHRENIA - MULTIPLE SCLEROSIS - BRAIN CANCER." The continued reckless and unwarranted inclusion of Multiple Sclerosis creates a false and totally unsubstantiated impression of a genuine affiliation with mercury poisoning and amalgam and has become enshrined as a cruel piece of anti-amalgam folklore. The case histories and personal testimonials of dramatic improvement of Multiple Sclerosis after amalgam removal, while emotionally convincing, are uncontrolled and rarely followed for any reasonable period.

Nevertheless, there is legitimate research to demonstrate that mercury and mercury vapour is released from amalgam restorations in the mouth. The anti-amalgam lobby makes a number of strained assumptions leading from this premise, one being that Multiple Sclerosis is a consequence of mercury release from dental amalgams and that their removal will cure the disease. There can be no argument with the call for the ultimate exclusion of mercury from dental techniques based on its inherent bio-incompatibility, nor the call for further research to demonstrate threshold levels and toxic effects of low level chronic exposure to mercury. Notwithstanding these valid considerations, there is no evidence that the dose of mercury which is released from amalgam is toxic or that it causes symptoms of mercury poisoning. There is even less justification for a causal relationship to be attributed to mercury (either by itself or as a product of amalgam) and Multiple Sclerosis.
Again one must criticise those health professionals, who espouse an anti-amalgam viewpoint, for their manipulation of scientific data to convince the gullible public of their claims. Whether well-meaning or simply exploitive, these health professionals ignore their responsibility to ensure that desperate and unhealthy people are fully informed of all the facts before embarking on techniques which, particularly in the case of Multiple Sclerosis, will be expensive, stressful, time-consuming, unnecessary and achieving of no long-term health benefit.

The strands of the amalgam/MS rationale are clearly seen in the following extracts where the author, a person with Multiple Sclerosis, having been recommended amalgam replacement requests communications on the subject. Although the responses concluded no improvement from amalgam removal, there is still maintained a presumptive acceptance of the general mercury toxicity hypothesis as a factor in Multiple Sclerosis.

The following is from a letter published in the Jan/Feb 1990 issue of "Arms Link" [produced by Action and Research for Multiple Sclerosis, London] (p30) (Bowring 1990a) from a person with Multiple Sclerosis: *In common with many of your readers I consulted a Clinical Ecologist after MS was diagnosed and was put on a strict diet. I was also advised to have my dental amalgam fillings changed to a non-mercury filling. I would be very interested to hear from anyone who has had their fillings changed, or who knows of anyone, and what they think about this course of action. It would also be of interest to know whether they had a Vega test first, and if they then had their teeth re-filled in the "correct" order.*
In a later issue of "Arms Link" (May/June 1990 p22) (Bowring 1990b) from the same writer: *I heard from many people on this subject, not one of whom reported any notable improvement in their condition after having their fillings changed, and I detected an understandable note of disappointment in several on this account. However most who had consulted clinical ecologists had been found to be sensitive to mercury and one or two proffered the view that mercury poisoning was actually blocking other forms of treatment.*

These letters exemplify the confusion that quasi-science can create in a person with Multiple Sclerosis who is both literate and sensible.

The following claims are included in the letters:

- diet and removal of amalgams will alter the course of Multiple Sclerosis;
- the Vega Test (see Section 13.5.8) and the "correct order" of amalgam removal are necessary adjuncts to the amalgam removal procedure;
- some people with Multiple Sclerosis are sensitivity to mercury;
- mercury poisoning blocks other forms of treatment for Multiple Sclerosis;

None of these claims are true, but offered in a "professional" environment and shrouded in, what appears to be, scientific terminology and analysis, they are appealing and reasonable.

Multiple Sclerosis is a disease that is often ignored by the medical profession - there is no cause and only symptomatic therapy. Many (if not the majority) of people with Multiple Sclerosis seek assistance outside the established medical framework. Thus it is not unusual nor unlikely that they will be exposed to peripheral and alternative medicine offering attention and therapies which the medical establishment is unwilling or unable to offer. Fear and frustration tend
to dilute commonsense and even outlandish theories and therapies are tacitly accepted. The clinical ecologists, holistic dentists etc whether sincere or exploitive, capitalize on the abandoned status of the person with Multiple Sclerosis. There is need for the medical profession (both family doctors and neurologists) to support and advise their patients with Multiple Sclerosis on an ongoing basis (particularly in relation to claims of cures and treatment), and guide them to appropriate organizations (e.g. Multiple Sclerosis Societies) where they can seek further information and assistance.

That one individual's Multiple Sclerosis remits after amalgam removal should not be interpreted as proof of a relationship between amalgam and Multiple Sclerosis, nor that others will have similar results. Ms Louise Herbeck claims a 'remarkable recovery' from her Multiple Sclerosis after amalgam removal such that she has founded a group "D.A.M.S." (Dental Amalgam Mercury Syndrome Victims Support Group). The title of her testimonial "Amalgam poisoning and MS - a case history - Or a walk through hell" (Herbeck 1990) conveys her anguish and frustration with the disease and the text her obvious conviction as to the benefits of amalgam removal. In the majority of testimonials, (whether alluding to Multiple Sclerosis or other disease states) there is no supporting medical evidence, either of the state of health prior to amalgam removal or of the reality of altered symptoms subsequent to removal.
There is very little in the scientific literature specifically relating mercury from amalgam to Multiple Sclerosis:

Heavy metal toxicity is often cited as a cause of disease and, in the case of Multiple Sclerosis, lead and mercury are often implicated. Ingalls (1986) discusses clusters of outbreaks of Multiple Sclerosis, in particular 30-40 cases in Key West, Florida, USA in 1983-1985. He concludes that this outbreak was due to environmental pollution of mercury from a local dump pile. Copper deficiency has been implicated in the aetiology of Multiple Sclerosis on the basis of similar demyelinating diseases in sheep (Warren 1989).

An earlier article by Ingalls (1983) associates slow, retrograde seepage of ionic mercury from amalgam restorations over many years with the advent of Multiple Sclerosis in middle age. Ingalls considers that lead may operate interchangeably with mercury. Using rather oblique reasoning he states: 'Possibly cases of unilateral MS derive from mercury-amalgam fillings in ipsilateral teeth, whereas the generalised disease may result from ingestion or inhalation of volatile mercury or exhaust fumes of lead additives to gasoline.'

Craelius (1972) claims that the aetiology of dental caries and Multiple Sclerosis are similar and proposes that dental caries may be just a precursor stage in many cases of Multiple Sclerosis due to mercury toxicity secondary to mercury seepage from amalgam fillings.

This claim can only be regarded as nonsensical. Dental caries is a specific bacterial disease of teeth and one of the most common diseases of modern man.
To equate the aetiologies of dental caries and Multiple Sclerosis is simply bizarre.

Currier (1972) interviewed a series of 87 patients with Multiple Sclerosis for historical factors over a 10 year period and hypothesised that the amount of dental work carried out prior to onset might be unusual and bear further investigation. This idea is based on the fact that 83 of the 87 had diverse forms of dental treatment prior to the onset of Multiple Sclerosis and all of the 83 having had local anaesthetic administered. These statistics are not compared to a control group of 84 patients whose dental experience is not documented. The fact that the remaining 4 Multiple Sclerosis patients had no dental treatment tends to further dilute the basis for this hypothesis. The author sees support for this thesis in the fact that in China and India, where Multiple Sclerosis is virtually unknown, both dental care and dental caries are infrequent, teeth being lost from periodontal disease.

This type of associative hypothesis is purely conjectural and not supported by any factual data. The low incidence of Multiple Sclerosis in China and India may be a reflection of lack of medical facilities such that the disease is not recognised nor documented. On the other hand dietary factors present in the western world and absent in these countries could as easily be substituted for the claim that dentistry is the source of Multiple Sclerosis.

Sibley, Bamford and Clark (1990) studied 170 Multiple Sclerosis patients and 130 healthy controls over an 8 year period to determine possible environmental factors which might be important in the disease. Stress had only a marginal
affect and physical trauma was not a risk factor. Dental treatment was included in the category of physical trauma and there was no evidence of any relationship between dentistry and the exacerbations of Multiple Sclerosis. The authors noted that the only significant risk factor was clinical viral infection and they conclude that Multiple Sclerosis is probably a post-infectious phenomenon.

The research by Eggleston (1984) [See Section 10.4] into alterations in T lymphocyte populations related to the presence and removal of amalgam restorations has been cited by some as evidence of a relationship between amalgam and autoimmune disease. This preliminary study, which has not been duplicated nor confirmed, showed an increase in T cell numbers with the removal of amalgam in 2 patients. Neither of these people exhibited any evidence of allergic response, nor sensitivity to amalgam or mercury, much less any indication of immune suppression. Multiple Sclerosis has been described as an autoimmune disease and it has been established that there is a reduction in the numbers of circulating T lymphocytes during the exacerbation phase of Multiple Sclerosis. Although mercury is known to be able to cause autoimmune responses in rodents, this study does not provide any logical link between amalgam and Multiple Sclerosis, other than a superficial similarity of response mechanisms.

In making an assessment of a possible connection between Multiple Sclerosis and mercury poisoning, there is confusion because, both Multiple Sclerosis and chronic toxic exposure to mercury particularly target central nervous system structures and are manifested as neurological effects. Thus many of the
symptoms of mercury poisoning are similar to those of Multiple Sclerosis including early toxicity signs of weakness and fatigue, and later signs of advanced toxicity such as tremor, memory loss, ataxia and speech disorders. The correspondence of symptoms does not, however, indicate a common aetiology. Thus mercury is the cause of mercury poisoning, but mercury is not the cause of Multiple Sclerosis, the aetiology of which is as yet unknown.

*Factors to be considered in excluding mercury and amalgam restorations as a cause of Multiple Sclerosis:*

1. Although first identified as a distinctive disease in 1868 by Charcot, Multiple Sclerosis predates the invention of dental silver amalgam in c1818 and its general usage in dentistry which developed in the mid 19th century.

2. There is no evidence of an increased incidence of Multiple Sclerosis in those occupationally exposed to elemental mercury vapour and in whom high levels of mercury have been reported. In the dental sphere, dentists and staff have higher mercury levels than both the general unexposed population and those people with extensive amalgams, but not a higher incidence of Multiple Sclerosis.

3. Multiple Sclerosis is not limited to people with amalgam restorations:
   a. Multiple Sclerosis occurs in children and adolescents who have had no amalgam restorations and therefore mercury from dentistry cannot be the initiating factor for Multiple Sclerosis in these cases.
   b. Multiple Sclerosis develops in edentulous people who have no teeth and therefore no amalgam restorations. However, it must be accepted that
the teeth extracted may have been restored with amalgam and that prior exposure to mercury may have occurred. Nevertheless, the fact that edentulous people are not healthier than those with amalgam-filled teeth questions the claims of those who recommend removal of amalgam restorations as a treatment for Multiple Sclerosis.

4. Multiple Sclerosis is a highly unpredictable disease, marked by exacerbations and remissions which vary in degree and frequency. Thus to conclude that the cause of onset or an exacerbation of Multiple Sclerosis is the existence of or the placement of amalgam restorations and that the basis for a remission is the removal of same is highly tenuous. Additionally, the placebo effect is as high as 70% in Multiple Sclerosis patients (National Multiple Sclerosis Society 1983) and thus the assessment of improvement or deterioration requires rigorously controlled studies. None of the cases of Multiple Sclerosis supposedly caused by mercury from amalgam or the anecdotal cures resulting from amalgam removal have been supported by evidence of changes in the characteristic findings in Multiple Sclerosis seen in Magnetic Resonance Imaging (MRI), spinal fluid and evoked potentials.

5. Stress has been considered as a factor in the onset and recurrence of symptoms of Multiple Sclerosis. The possibility (even remote) that increased stress could affect immune response and be a contributory catalyst suggests that unnecessary surgery and anaesthesia should be avoided. The removal and replacement of multiple amalgam restorations is a protracted procedure involving extended periods in the dental chair and the injection of considerable
amounts of local anaesthesia. A large percentage of the population is apprehensive of dental treatment and for the person with Multiple Sclerosis there would be enhanced levels of anxiety and stress associated with these procedures.

6. The consumption of methylmercury in fish increases the body burden of mercury far more than the release of mercury vapour from amalgam restorations. Methymercury is also more toxic than mercury vapour. The daily consumption of fish in many parts of the world produces high levels of mercury in these populations, but no epidemiological evidence of increased incidence of Multiple Sclerosis.

7. There is no pathological or histological evidence that the neurological demyelinating process which is a characteristic of Multiple Sclerosis is in any way similarly produced by mercury poisoning.

Thompson (1986) in his review of dentistry and the Multiple Sclerosis patient concludes that: 'there was no bonafide scientific evidence that either mercury-silver amalgam restorations or correction of occlusal dysfunction has any bearing on the course of Multiple Sclerosis'.

In reviewing the replacement of mercury amalgam fillings as a treatment for Multiple Sclerosis the Therapeutic Claims Committee of the International Federation of Multiple Sclerosis Societies (IFMSS)(1988) concludes that: 'there appears to be no generally accepted scientific basis for use of this therapy. It has
never been tested in a proper controlled trial. It is relatively free of serious adverse side-effects during long-term use. It is very expensive.'

Dr Reingold, Vice President for Research and Medical Programs of the American National Multiple Sclerosis Society notes in a memorandum (November 1990): 'The theory that MS is in some way caused by amalgam fillings, and that their replacement can lead to a remission of the disease, has been present for a number of years. The reality is that there has never been a direct association made between amalgam and MS. Reports of remissions resulting from amalgam removal appear to be anecdotal and cannot be separated from placebo effect or spontaneous changes in the disease. There are many people with MS who do not have such fillings, and others with MS who have had amalgam fillings replaced with no demonstrated benefit. Additionally, MS as a disease existed well before amalgam fillings became commonly used in dental care.

The Medical Advisory Board of the National Multiple Sclerosis Society has concluded that there is no sound epidemiological evidence which relates mercury amalgam fillings to MS, and no sound clinical evidence, gained through controlled clinical trials, which suggests that replacement of dental amalgams leads to any improvement in MS.'

In an editorial in Dental Update (1986) the Editor states: 'The statements, made in the media, that dental amalgam fillings are harmful are both highly misleading and lacking in good scientific evidence to support them. The linking of mercury toxicity with symptoms mimicking Multiple Sclerosis is highly speculative and it is most improbable that the mercury levels from dental amalgam could lead to
neurological symptoms.'

The National Institute of Dental Research (NIDR)(1991) states: 'There is no scientifically sound evidence linking mercury in dental amalgam to multiple sclerosis, arthritis, mental disorders, or other diseases.'

People with Multiple Sclerosis and their loved ones are particularly susceptible to claims of curative treatment. The relationship purported to exist between Multiple Sclerosis and mercury poisoning hinges on a simplistic comparison of similar neurological symptoms with dissimilar aetiologies. For the desperate and afflicted this quasi-scientific thesis is reassuringly reasonable and many people with Multiple Sclerosis have little ability to objectively assess the legitimacy of these claims. It should therefore be clearly communicated to the Multiple Sclerosis community that there is no evidence that Multiple Sclerosis is associated with mercury poisoning from amalgam restorations. Additionally no benefit occurs to the person with Multiple Sclerosis by the removal and replacement of amalgam restorations.
18. DISCUSSION

In assessing the question of mercury toxicity from amalgam there is an interesting analogy within the dental arena of concerns regarding the toxicity of fluoride which might imply a more general social and political dimension to these controversies. This salient point has been emphasised by Akers (1989) and Chenoweth (1985) aptly notes that: "most of the present difficulties in the field of toxicology are not technical but political, psychological and sociological".

In this civilized and technical world there are many health measures imposed by governments, local authorities and health professionals which may appear to impinge on the rights of the individual. The chlorination and fluoridation of water supplies is a classic example, as may be the dental profession’s general and optionless usage of amalgam. Notwithstanding society’s medical excellence, diseases persist and the modern style of existence creates new symptoms and conditions which are yet to be resolved. There are thus fears within the community, almost archetypal in nature, of bureaucratic health impositions which might in fact be contaminating part or all of the populus.

These misgivings are the domain of certain groups within the community:

- the sincere (but unfortunately often obsessive) crusaders who make elaborate claims as to health dangers posed by certain substances. There are very few people who can claim a complete grasp of the specialised fields of knowledge involved in toxins and disease. Nevertheless there are, on the one
hand, experts in a single field making pronouncements in other disciplines and, on the other, lay generalists with a broad understanding of fundamentals making assertions in complex specialist areas. In many respects the arguments presented are as faulty in detail as they are mistaken in principle.

- the healers whose particular therapy offers cures for the multiplicity of symptoms and diseases not satisfactorily catered for by the medical establishment. It is often hard to ascertain whether these are charlatans, their fabrications purely for fame and profit, or genuine believers, they themselves as deluded as their patients as to the basis of their belief and the effects of their therapies. Outside of the holistic dentists who promote mercury toxicity from amalgam and amalgam removal there are a range of para- and pseudo-health professionals who are involved in the concept of disease by mercury poisoning; clinical ecologists, nutritionists, herbalists, homeopaths, chiropractors, naturopaths.

- the media, which in general shows contempt both for the public and for the truth. There is thus a vehicle for the recycling of the revelations of those mentioned above which is exaggerated and simplified for easy and gratuitous consumption;

- some members of the community whose perception and attitude finds satisfaction and credibility in these types of claims. This is particularly so of those who are critical of imposed authority, establishment values, and particularly cynical of the ethics of the healing professions. For some, perhaps,
there is a willingness to personally accept far greater danger than is acceptable to be imposed upon them from another source. While challenging orthodoxy is healthy and valuable, the unquestioned embracing of alternative claims and cures seems more a social posture than a reasoned judgement. In addition to its human effects, mercury is also an environmental pollutant. Thus environmentally oriented individuals and groups become concerned with the ecological impact of mercury which indirectly adds a measure of credence to the anti-amalgam claims;

- **the sick**, whose search for therapy must encompasses all possible avenues. Unfortunately, irrationalism born of hope never dies - which accounts for the continued existence of those who offer remedies. This also explains the anecdotes recounting miraculous cures which are always paraded as supporting evidence of claims and therapies, but which are usually the result of short-term placebo effects, natural remissions, and psychosomatic considerations.

In discussing the above groups and their contribution to the mercury from amalgam debate it is relatively simplistic to explain the phenomenon as a combination of naivete, gullibility and confused logic, with the lack of scientific expertise and less than objective reasoning producing false conclusions. Perhaps it is just that, as sentient beings, there is an unwillingness to accept that certain configurations of our lives are as yet unknown. There is a need to create patterns out of randomness, and then impart meaning to the pattern.
For the quacks and charlatans it is probably fair to say that when the cited evidence is deficient and yet the conclusions have grave and significant implications, there are perhaps motivations which are other than a concern for the truth.

However, as in the fluoride controversy, there are a few groups of respected researchers within the established scientific community who continually report data which gravely impugns the safety of amalgam. Some of these results have been questioned and even discredited by a larger and diverse number of similarly well respected researchers. Where a scientific impasse exists there is the obvious need for objective and independent research to duplicate and either validate or refute these findings. However, there is also a question as to why these few groups (the prime example being the "University of Calgary Group" comprising researchers Vimy, Lorscheider, Hahn, Summers), having established an anti-amalgam stance, seem to be able to produce data from varied animal and human experiments which repeatedly support this position. Numerous articles printed in diverse publications, with members of teams rotating as primary authors, gives a bulk of literature, apparently of independent source, producing similar results. Katz (1991) criticises the results of the studies at the University of Calgary as not being peer-reviewed and according to the FDA, very flawed.

Perhaps there is in these cases the cardinal fallacy that, once having predetermined the conclusions, both the method of research and the manner of argument become selective and manipulative in order to satisfy and justify the fixed equation. This comment may of course have a wider application and just
as legitimately apply to some members of the scientific and dental establishment (for whom change is an anathema and who seek to preserve the inertia and hierarchy of the status quo) as to the radicals and dissenters (whose desire for change can be an anarchical response to those who have become self-satisfied and sedentary, and so consuming as to become logically inconsistent).

Beyond the emotional and sensational, there is little to substantiate the basic claims made by the hard-core anti-amalgamists such as Huggins and his Toxic Element Research Foundation and the Ziffs and their Bio-Probe Incorporated:

1. *Mercury escapes from amalgam in significant amounts;*

   There is mercury released from amalgam, but the levels are low and there is simply no evidence that it is either significant in quantity or in effect.

2. *WHO Threshold Limit Values are exceeded by the mercury which escapes from amalgam;*

   There are no proper general population thresholds for chronic exposure to mercury vapour, but WHO occupational levels are not exceeded.

3. *Mercury from amalgam is in sufficient quantities to impair health;*

   There is no evidence of ill health being a consequence of mercury release from amalgam. The rare cases of contact allergy to amalgam and oral lichenoid conditions are not confirmation of any widespread community health damage.

   There is no proof that the human immune system is affected by the mercury released from amalgam.
4. Elemental mercury is one of the deadliest poisons known to man;

The elemental mercury vapour released from amalgam is one of the most toxic forms, but less toxic than methylmercury consumed in fish. In negating the claims of the anti-amalgamists, the inherent dangers of mercury in all its forms cannot be ignored.

5. The human body can methylate elemental mercury;

This rather important plank in the mercury toxicity platform is total conjecture and has not been validated. The human body does not methylate inorganic mercury. This assertion had allowed an inclusion of all methylmercury in the body (with its attendant high neurotoxicity and reproductive sequelae) to be inferred as a consequence of inhalation of mercury vapour from amalgam. In this way all the repercussions of methylmercury poisoning (birth defects etc) could be laid at the door of amalgam. The reality is that all methylmercury in the body is from fish consumption.

6. Inorganic Mercury in the body (in the absence of occupational exposure) is mainly due to mercury from amalgam;

A body of recent research, which shows that demethylation of methylmercury contributes to a major proportion of the body’s inorganic mercury levels, confounds the claim that the bulk of inorganic mercury in the body must come from amalgam mercury release. This means that the inorganic mercury in the body could be derived from both inorganic and organic
7. Mercury from amalgam is harmful during pregnancy, causes stillbirths and malformations and retardation;

Only methylmercury has been shown to have these effects. Methylmercury is not a consequence of mercury release from amalgam.

8. Mercury from amalgam accumulates in brain, thyroid and pituitary glands;

Mercury does target the central nervous system, but the source of the inorganic accumulation is not necessarily and solely amalgam. In spite of this there seem no clinical effects as a consequence of mercury accumulation in the brain which may emanate from amalgam.

9. Oral galvanic impact from dissimilar metals in the mouth (including amalgam) is a serious concern;

There is no evidence that, if oral galvanism exists, it produces any damage to health.

10. Removal of amalgam improves health;

Only anecdotal reports make these assertions and no studies have shown any improvement in health states, diseases, or symptoms by the removal of amalgam restorations.
Research in the next few years will clearly identify whether:

a. the dental and scientific community have complacently supported an enshrined dogma of amalgam safety such that opinion and research has continually confirmed what was believed to be true or

b. those who claim mercury poisoning from amalgam restorations have created a grand fiction from modest facts. The presumption of guilt has allowed misrepresentations and misinterpretations to colour the debate such that untenable assumptions and conclusions have maligned a long standing and historically satisfactory dental therapy.

The bulk of evidence currently favours the latter interpretation.

Unlike fluoride (which in large doses is toxic, yet in small doses is safe and has a profoundly beneficial influence on tooth structure), mercury has no redeeming biological features for the human body and should be avoided. The therapeutic margin (the difference between a therapeutic and toxic dose) for fluoride is wide whereas for mercury there is little therapeutic value (it being a weak antimicrobial agent) and threshold levels (particularly for mercury vapour) are not satisfactorily established.

It is important that amalgam restorations be seen as a therapeutic modality in the same light as medical applications which contain mercury (e.g. skin lightening creams, topical antiseptics etc). There has been a tendency for the dental profession to view amalgam as a static prosthetic implant in solid tooth rather than as a form of medication. However, amalgam is not inert and the site is human tissue, and thus consideration of the use of amalgam in humans should
be approached in the same way as medical applications containing mercury. The health considerations by which medications containing mercury are being selectively excised from medical practice should also be applied to dental applications containing mercury. There appears to be a paradox in comparing the therapeutic value of the amalgam restoration in the dental field with the lack of therapeutic value (and potential toxicity) of the mercury which it contains.

Avoidance of mercury altogether is not possible.....natural as well as polluted sources in soil, air and water ensure that a background level exists. Additionally it is impossible to avoid some level of intake from foods. The inorganic mercury in non-fish foods is poorly absorbed and is thus less consequential than fish foods which constitute the major source of retained dietary mercury, almost all of which is the highly toxic methylmercury.

It must be acknowledged that from current data available some mercury vapour is regularly entering the body from dental amalgam restorations by the respiratory route when mouth breathing occurs. The important considerations which stem from this fact are whether the levels of mercury taken into the body from this source are significant, add measurably to the body burden of mercury, are toxic to the extent of causing mercury poisoning and the cause of disease states.
Compared to mercury intake from other sources the quantities are small, but must be weighted due to the repetitive nature of the exposure and the toxicity of mercury vapour. Nevertheless, many people and populations regularly consume fish containing methylmercury in far higher concentrations than that of mercury vapour released from amalgam restorations and, in the case of the methylmercury, more is retained and it is far more toxic than mercury vapour. The possibility of further increased community methylmercury exposure exists because there is a worldwide trend to more regular fish consumption because of health benefits accruing from the Omega-3 essential fatty acids which are at high levels in fish. Many National Heart Foundations and official health organizations recommend increased fish meals per week as part of a balanced diet, with the aim of a reduction in saturated fats and an increase in marine unsaturated oils.

Symptoms of mercury toxicity may reflect organic or inorganic exposure or a combination of both. Both methylmercury and inorganic mercury vapour have a specificity for the Central Nervous System. There are subtle differences in symptoms [See Section 5.1 & 5.2] but, because of ultimate demethylation of much of the methylmercury in the body to inorganic mercury, the final clinical effects may represent a combination of the effects of inorganic and organic forms of mercury. The various symptoms reported as being a consequence of mercury released from amalgam do not fit any recognised pattern or progression of mercury toxicity. Many of the non-specific symptoms may reflect a vast panorama of alternative disease processes, psychological manifestations and, as
well, mirror toxicities from other elements and heavy metals. Clarkson (1987) states: 'Inorganic compounds of aluminium, lead, lithium, manganese, mercury and thallium are well known for their neurological and behavioural effects in humans. The alkyl derivatives of certain metals - lead, mercury and tin - are specially neurotoxic. The most severe damage (to the nervous system) is produced by organo-metallic forms such as methylmercury.'

Huggins claims that 67% of the population (presumably a figure representing all those with amalgam restorations) show symptoms of systemic mercury toxicity. Yet, in the descriptions of "mercury toxicity" and the cases cited, there is little mention of classic mercurial tremor, a symptom which would be prevalent if such a vast proportion of the population exhibited the toxic effects of mercury exposure.

One major area of concern in regard to alleged symptoms from mercury poisoning from amalgam relates to mild neurological effects. Although it is accepted that a proportion of mercury, from whatever source, targets the brain and is retained for many years, little is known as to how neurological symptoms are produced and what level of tissue or cellular concentration can be accepted without neurological deficit. The efficiency of the body’s cellular defence mechanisms is characterised by the fact that even though the pituitary and thyroid glands have been shown to retain remarkably high concentrations of mercury there is no evidence that their function has been compromised.
The relationship between mercury release from amalgams and uptake by the body is obscured by uncertainties about analytical quality control, sampling methodology, breathing patterns and dilution with inhaled air. There is controversy as to the actual amount of mercury vapour released from amalgam restorations on a daily basis, and the relationship of this to physical indicators such as blood, and urine, which are traditionally more indicative of the degree and extent of exposure to mercury vapour. The effects of mercury poisoning are reasonably explicit where large doses are involved, but the vague and non-specific symptoms which are equated with lower levels of mercury exposure are neither distinct nor exclusively characteristic of mercury poisoning.

If amalgam restorations contribute to mercury poisoning, it can only be in terms of the uptake of mercury vapour thus released. The effects of inorganic mercury ingested in saliva are negligible in comparison with that from mercury vapour, only a fraction being absorbed and being less toxic.

Perhaps the most telling evidence for the relatively innocuous nature of the mercury vapour from amalgam is found in studies done on dental personnel who can be exposed to significant amounts of mercury vapour in the surgery (to which must be added the mercury from their own amalgam restorations). Despite having urine mercury levels 3-5 times higher than the average population, blood levels 2-3 times the normal and mercury levels in surgery air up to the maximum accepted threshold occupational level of 50 \( \mu g/ m^3 \), dentists and their assistants exhibit no elevated levels of morbidity and mortality.
As an example of provisional tolerable weekly intake, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) [See Section 6.4] established a Provisional Tolerable Weekly Intake (PTWI) which allows 43 \( \mu \text{g} \) of total mercury consumption per day of which no more than 28 \( \mu \text{g} \) should be methylmercury. Given the toxicity of methylmercury, the probable 1-3 \( \mu \text{g} / \text{day} \) of mercury vapour released from amalgam restorations presents little threat. This contribution of amalgam restorations to the body burden of mercury is probably equivalent to approximately 10-20 \( \mu \text{g} \) from general foods but minimal in relation to the consumption of methylmercury via fish which is often in the range of 20-30 \( \mu \text{g} / \text{day} \). The comparison with methylmercury is particularly significant because of the similar uptake factors (mercury vapour 80\% and methylmercury 95\%), whereas only 7\% of inorganic mercury compounds (found in non-fish foods) is absorbed into the body. There is grave doubt that the mercury released from amalgam could actually cause symptoms of micromercurialism or mercury toxicity. There is simply no scientific evidence which identifies any symptom of ill health, or particular disease state which can be causally related to the effects of mercury released from amalgam.

Berlin (1986), in discussing the consumption of 10 \( \mu \text{g} / \text{day} \) of inorganic mercury in non occupationally exposed populations, states: 'Some contribution may result from the release of mercury vapour from dental fillings if these are present. This figure is low in relation to the possible intake of methylmercury.'
It is an important global health consideration that human mercury consumption be qualified and quantified. Mercury poisoning should be a legitimate concern of all health authorities... examples of pollution and occupational excesses indicate clearly that threshold levels do exist and toxic levels of mercury can be reached, producing overt disease symptoms. The inherent dangers both to the environment and to people that can accrue from the industrial use of mercury may result in actions by governments limiting or completely restricting its use. As an example there is currently a proposal in Denmark to ban the use of amalgam from 1999 as part of an environmental prohibition on Mercury.

Research indicating dosage which produces preclinical changes in neuropsychology and neurophysiology confirms the urgency for threshold levels to be reassessed. Caution should be applied in the case of treatment of pregnant women with amalgam, but this should not be seen as proof of mercury poisoning, but as a sensible health measure undertaken as a precaution until better data is available and threshold levels are clearly enunciated. The concerns with the effects of mercury on the foetus may well relate only to organic mercury.

There are complexities in evaluating the sources and forms of mercury which contribute to the body burden of mercury. There is transformation of organic mercury into inorganic mercury within the body and the reverse process in the environment. Not all mercury in fish is methylmercury, the contribution of industrial pollution adding inorganic mercury to the calculations. Thus inorganic mercury levels in brain, blood and urine may arise from fish sources as well as
from demethylation. It is simplistic and erroneous to apportion the bulk of body inorganic mercury to the release of mercury vapour from amalgam. All sources of mercury must be identified and incorporated into the assessment of the body burden of mercury and only at that time will we know the real contribution of dental amalgam. At this stage it appears mercury from dental amalgam is a minor contributor to the levels of mercury which many people are currently routinely exposed to, and which fortunately appears to cause little evidence of mercury poisoning or ill-health.

There is only an unproven and speculative relationship between mercury exposure due to amalgam restorations and the generalised and non-specific syndromes which claim mercury from amalgams and subsequent mercury poisoning as the cause. In the absence of objective data showing a direct and sequential relationship between amalgam restorations, mercury toxicity and disease states, there currently exists no need to restrict the use of amalgam as a routine dental restorative material. There appears no justification for gross replacement of amalgam restorations. Claims of improvement after replacement of amalgam fillings are usually anecdotal and with no control for potential placebo effects and observer bias.

Nevertheless there is validity in the premise that mercury (as with many chemicals) is inherently dangerous to the human system and exposure should be minimised. Even though the levels of mercury vapour taken up after release
from amalgams are low, it is of concern that there are no precise and accepted
safe levels of mercury consumption for the general population and particularly
for pregnant females, young children, the aged and the infirm. Therefore,
without detracting from the usefulness of amalgam in dentistry, suitable
alternative materials and treatment modalities should also be considered when
amalgam restorations fail and require replacement. Again, in the case where a
new restoration is required, informed consent is obligatory where amalgam is the
restoration of choice.

The future for amalgam is pleasantly bleak in a number of respects. In the first
place it will in time be superseded by materials which are more biocompatible
and have adequate function and longevity, as well as improved aesthetics. In the
second place in countries where fluoridation of water supplies has been
instituted the affected population show minimal caries and require far less
restorative treatment. Thirdly, a better educated and health conscious population
will maintain a higher standard of oral health. Fluoridation of water supplies
plus appropriate regular preventive care (both professionally in the dental
surgery and personally at home) should create a community for whom dental
caries and the ensuing restorative process is a rarity. The routine need for
restorations only exists if the prevention of caries fails.
The great value in the current controversy is that it alerts the dental profession to a possible problem with mercury as a constituent of amalgam restorations. It is the responsibility of the dental profession to ensure that any treatment provided to the public is beneficial and safe. Rigorous legitimate scientific research coupled with objective clinical assessment will eventually resolve the questions surrounding mercury release from amalgam. We need to know exactly how much mercury is released from amalgam restorations in humans, the uptake dosage, the effect this has on the body burden and the clinical significance as an element in mercury toxicity and disease states. At this stage we only know that mercury is inherently toxic and that small doses of mercury are released from amalgam. All else is supposition and speculative association. Nonetheless, the long and successful use of amalgam as a restorative material does not permit rank dismissal of valid inquiry, nor should it encourage professional complacency. In refuting the gross assertions of the anti-amalgamists there is the lesson that "rigid" and "inert" are not satisfactory characteristics to describe amalgam and nor should these qualities portray the professional approach to the health service provided to the dental patient.
1. Although there is measurable mercury released from amalgam restorations, no evidence of mercury poisoning has been established as a consequence of this. Additionally, no causal link can be substantiated between the mercury released from amalgam restorations and diverse symptoms of ill health and disease states. There is no substance to the claim that Multiple Sclerosis is related to mercury release from amalgam restorations.

2. Dental personnel constitute a select group whose mercury vapour exposure is significantly higher than the general population and yet who show no particular evidence of mercury poisoning or unusual levels of ill-health.

3. There is need for further research to establish not only the exact amount of mercury released from amalgam restorations into the oral environment, but to what degree this is taken up and incorporated into the body and its ultimate distribution and fate. There is need for the development of accurate measuring and analytical tools to measure low levels of mercury and for speciation of the different forms of mercury.
4. The release of mercury from amalgam must be considered in the broader panorama of mercury as it impacts on humanity. The contribution of methylmercury from fish sources constitutes a far greater factor (both in terms of quantity and toxicity) in the human burden of mercury than does mercury from amalgam.

5. There is a requirement, given the varied sources of human mercury intake, to clearly distinguish at what level of mercury exposure (particularly in respect of mercury vapour) the earliest evidence of toxicity occurs. Testing for subtle neurobehavioural effects should particularly target those occupationally exposed with special attention to the occupationally exposed dental fraternity and their offspring. There needs to be verification of the results of animal studies which have reported widespread dissemination of mercury throughout the body from amalgam implants and the possibility of auto-immune reactions to mercury. There is need for further research to assess the prevalence of true hypersensitivity and allergic responses to the constituents of dental amalgam.

6. Authentic allergy or hypersensitivity to the constituents of amalgam is a rare occurrence and only affects a very small group within the population. Contact allergic reactions are not evidence of mercury poisoning (micromercurialism, mercury sensitivity) which involve systemic parameters. There is consistent data indicating that oral lichenoid lesions may be causally related to the components or the roughness of amalgam restorations. It should be noted that there is a far greater incidence of hypersensitivity to nickel in dentistry than there is to mercury.
7. There is no therapeutic value in the removal of amalgams except in the rare cases of true allergy or hypersensitivity to the constituents of amalgam. No disease states or symptoms of ill-health are improved by the removal of amalgams. In general, amalgam restorations should only be removed when they fail.

8. There is no justification for the exclusion of amalgam from the repertoire of dental materials offered to patients. The benefits and disadvantages of both amalgam and alternative materials should be discussed as part of the treatment plan where restorative dentistry is intended. Informed consent is important in respect of planned amalgam restorations as with all forms of dental therapy.

9. Accurate threshold levels for mercury exposure need to be established for the general population and for the vulnerable members of the community - children, the foetus, the aged and the sick. While there is no evidence at all that mercury vapour from amalgam restorations causes any adverse effects on the developing foetus, a more cautious and restricted approach should be taken by the dental profession to the removal or insertion of amalgam restorations in pregnant women and in young children until such time as proper exposure standards for all forms of mercury for these sub-groups have been determined.

10. Preventive dental health measures should be reinforced in order to minimise the need for all dental restorative procedures.
11. The health professions should aim to exclude mercury from all therapeutic treatments. In the light of current knowledge the mercury released from dental amalgam does not constitute a threat to human health. Nevertheless, mercury has no biologic benefit and efforts should be made to provide alternative and more biocompatible dental materials to ultimately offer a genuine replacement for amalgam in dentistry.

12. The dental, medical and scientific communities should be more aware of the vigour and diversity of anti-amalgam sentiment and the accompanying substantial bulk of aggressive literature promoting the danger in dental amalgam. This perspective is regularly highlighted and even legitimised by sections of the media who readily sacrifice scholarship for sensation. These claims of symptoms and diseases being caused by mercury poisoning from amalgam restorations impact, not only upon those who are ill, but upon a very large percentage of the general public who have amalgam restorations. Within this larger group there are normal concerns regarding health and, because most people have periods of less than optimum well-being, the safety of amalgam may be questioned. The dental and medical professions, particularly, must address the fear and confusion that surrounds this subject, and ensure that their patients, and indeed the whole community, receive a balanced and educated overview of the matter.

13. Continued rigorous, objective scientific research should be fostered to clarify the role of mercury in human health. Within these broad parameters, there will ultimately be precise attribution of the contribution of that mercury which is contained in dental amalgam.
20. REFERENCES

The effect of dental amalgam restorations on blood mercury levels.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (1988).
Preliminary health assessment.

Acute mercury vapour intoxication: Report of six cases.

Number of amalgam tooth fillings in relation to subjectively experienced symptoms.

The amalgam debate- A personal viewpoint.
Aust Dent Assoc (Qld Branch) Newsletter. (Sep):5-6.

Letter to the editor.

Letter to the Editor.
Aust Dent Assoc (Qld Branch) Newsletter (in press).

AKIF M, SECKIN, AYGUN S, ATAMAN OY (1986).
Determination and speciation of mercury in a dental work-place by cold vapour atomic absorption spectrometry and gas-liquid chromatography.

ALBERT D (1986).
Mercury allergy as a cause of burning mouth. Letter.

ALCSER KH, BRIX KA, FINE LJ, KALLENBACH LR, WOLFE RA (1989).
Occupational mercury exposure and male reproductive health.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (1983).
TLVs threshold limit values for chemical substances and physical agents in the work environment with intended changes for 1983-84.
Cincinnati.
When your patients ask about mercury in amalgam.

Special Report. Supplement to JADA.
J Am Dent Assoc 122(1).

Suicide among Swedish dentists.

Retrograde axonal transport of mercury.
Exp Neurol 98:198-203.
Cited in: BOWLESKA J, HOLMSTRUP P, MOLLER-MADSEN B, KENRAD B,
DANSCHER G (1990b).
Amalgam associated mercury accumulations in normal oral mucosa, oral mucosal lesions of lichen planus and contact lesions associated with amalgam.

Mercury toxicity: Mechanisms of blood brain barrier transport.

Dental Amalgams.

AUSTRALIAN COMMONWEALTH DEPARTMENT OF HEALTH (c1978).
Health aspects of mercury in fish.
p16.

AUSTRALIAN DENTAL ASSOCIATION: MERCURY SUB-COMMITTEE OF THE
THERAPEUTICS, INSTRUMENTS, MATERIALS AND EQUIPMENT COMMITTEE (Dec
1989).
Dental Amalgam - a safe and proven dental filling material.
Sydney:Australian Dental Association.

AUSTRALIAN SOCIETY OF BIOLOGICAL DENTISTRY (Nov 1988).
Hal Huggins Course Application.

Hand steadiness and mercury blood levels among practising dentists: Preliminary findings.
J Amer Cent Assoc 106:519.
Potential health hazard of use of mercury in dentistry: Critical review of the literature.

Silver amalgam under attack. Editorial.
Mercury and dentistry - The controversy continues.

Commercial hair analysis.

BATTISTONE GC, HEFFERREN JJ, MILLER RA, CUTRIGHT DE (1976).
Mercury: Its relation to the dentist's health and dental practice characteristics.
Environmental Health Criteria 118 - Inorganic mercury.
IPCS [International Programme on Chemical Safety]; Geneva.

Action of mercury in dental exposures to mercury.

Toxic fish and sewer surfing.
Australia: Allen & Unwin.

Drilling for Danger? A debate over the safety of 'silver' fillings.

Safety of mercury. Letter to the editor.

BERGER SM (1989).
How to be your own nutritionist.

Determination of the rate of release of intra-oral mercury vapour from amalgam.

Estimation by a 24 hour study of the daily dose of intra-oral mercury vapor inhaled after release from dental amalgam.

Side-effects of amalgam and its alternatives: local, systemic and environmental.

BERLIN M (1986).
Mercury.
In: FRIBERG L, NORDBERG G, VOUK V. (1986)
Mercury.
In: Toxicology of metals - Vol II
Toxicology and biological monitoring of metals in humans.
USA: Lewis Publishers. p150.

BHATNAGAR RS (1980).
Molecular basis of environmental toxicity.
Dental amalgam related mercury vapor exposure.
Calif Dent Assoc J 12:54-60.

The history of dental amalgam.
Tidskr Nor Laegeforen 109(34-36):3582-5.

Dental amalgam - does it threaten our health?

T lymphocytes, Langerhans cells and HLA-DR expression on keratinocyted in oral lesions
associated with amalgam restorations.

Oral mucosal lesions related to silver amalgam restorations.

BOWLESKA J, HOLMSTRUP P, MOLLER-MADSSEN B, KENRAD B, DANSCHER G (1990b)
Amalgam associated mercury accumulations in normal oral mucosa, oral mucosal lesions of lichen
planus and contact lesions associated with amalgam.

BOWRING L (Jan/Feb 1990a).
Letter to Postal Link.
England: Arms Link. Published by ARMS Central. p31

BOWRING L (May/June 1990b).
Letter to Postal Link.
England: Arms Link. Published by ARMS Central. p22-23

BRADY JA, GEMITTI-NUNN D, POLAN AK, MITCHELL D, WEIL R, VIANNA NJ. (1980)
The relationship of dental practice characteristics to blood mercury levels.
Environmental Health Criteria 118 - Inorganic mercury.
IPCS [International Programme on Chemical Safety]: Geneva.


Examination of blood levels of mercurials in practicing dentists using cold-vapor atomic absorption spectrometry.

CHASE RH (1986).
Is the amalgam filling safe?
Chronicle 49(1):6-7,11.

Perspectives in toxicology.
Potential health hazard of use of mercury in dentistry: Critical review of the literature.

Radioactive mercury distribution in biological fluids and excretion in human subjects after inhalation of mercury vapor.
Arch Environ Health 33:109-114.
The contribution of dental amalgam to mercury in blood.

The effects of methylmercury on the developing brain.

CLARKSON TW (1983).
Mercury.
Potential health hazard of use of mercury in dentistry: Critical review of the literature.

CLARKSON TW (1987).
Metal toxicity in the central nervous system.
Environmental Health Perspective 75:59-64.

The prediction of intake of mercury from amalgams.
Biological monitoring of metals.

CLARKSON TW, NORDBERG GF, SAGER PR (1987).
Biological monitoring of metals with special reference to the early stages of the life cycle.
Sangyo Ikka Daigaku Zasshi 9(Supplement):50-58.

1. Amalgam use. 3. Attitudes about mercury in dentistry.

Tooth build-up - Status report.

COMBE EC (1986).
Amalgam.
In: COMBE EC (1986).
Notes on dental materials. 5th edn.

CONSERVATIVE GYNECOLOGY CLINIC, INSTITUTE OF OBSTETRICS AND
GYNECOLOGY, ACADEMY OF MEDICINE, LUBLIN, POLAND (1986).
Mercury in neonatal scalp hair.
Sci Total Environ 57:105-110.

Mercury vapor produced during sterilization of amalgam contaminated instruments
J Pros Dent 53:304-308.
Biocompatibility of mercury derivatives.
Dent Mat 2(3):91-96.

Detection and diagnosis of bioincompatibility of mercury.
Calif Dent Assoc J 12:36-43.

COUNCIL ON DENTAL MATERIALS AND DEVICES. REPORTS OF COUNCILS AND
BUREAUS (1974).
Mercury surveys in dental offices.

CRAELIUS W (1972).
Comparative epidemiology of multiple sclerosis and dental caries.

CRAIG RG (1986).
Biocompatibility of mercury derivatives.
Dent Mat 2(3):91-96.

CROOK WG (1989).
The yeast connection. 3rd edn.

CURIER RD (1971).
Multiple Sclerosis and dental work.

DAILY MAIL. MEDICAL NOTEBOOK (Sep 10 1990).
The mercury starts to rise over filling debate.
Traces of mercury in organs from primates with amalgam fillings. 

Mobilization test for environmental poisonings 

Yellow mercuric oxide ointment and mercury intoxication. 

Mercury in the dental work place: An assessment of health hazards and safeguards. 

DENTAL ESTIMATES BOARD (1985). 
p52.

DENTAL UPDATE (1986). 
The mercury hazard in dentistry. Editorial. 
Dental Update 13(1):5-6.


The possibility of allergic reaction from silver amalgam restorations. 

Amalgam toxicity: A review of the literature. 

Dental silver tooth fillings. Letter to the editor. 

A rational policy on the use of dental amalgam. 
Side-effects of amalgam and its alternatives: local, systemic and environmental. 

Hazards in dentistry: The great debate. Letter to the editor. 

Mercury - is it a respiratory tract allergen? 
Effect of dental amalgam and nickel alloys on T-lymphocytes: Preliminary report.

EGGLESTON DW (1989).
Dental amalgam: A review of the literature.
Compend Contin Educ Dent 10(9):500-505.

EGGLESTON DW, NYLANDER M (1987).
Correlation of dental amalgam with mercury in brain tissue.

ELEY BM (1985).
Hazards in dentistry: The great debate.

ELEY BM, COX SW (1987).
Mercury from dental fillings in patients.

ELEY BM, COX SW (1988).
"Mercury poisoning" from dental amalgam - an evaluation of the evidence.
J Dent 16(2):90-95.

Potential health hazard of use of mercury in dentistry: critical review of the literature.

The role of dental restorative metals in the pathogenesis of oral lichen planus.

The mercury scare. Letter to the editor

FASCIANA G (c1986).
Are your dental fillings hurting you? The hazards of having mercury in your mouth.
New Canaan CT: Keats Publishing.

FEDERATION DENTAIRE INTERNATIONALE (1988).
Recommendations on dental mercury hygiene. Revision of FDI Technical Report No. 7

FEDERATION DENTAIRE INTERNATIONALE (1989).
Safety of dental amalgam.
FEARNSIDE WW, HOLThER WB (1959).  
Fallacy. The counterfeit of argument.  

FINCH R (1973).  
Effects of regulatory guidelines on the intake of mercury from fish - The Mecca project  
Fishery Bulletin 71(3):615.  
Health aspects of mercury in fish.

Oral lichen planus and contact allergy to mercury.  

Response to MACKERT JR.  

The misuse of the patch test to determine "hypersensitivity" to mercury amalgam dental fillings.  
Cutis 35:110-117 1985  
Hypersensitivity reactions in a dental group of patients.  

Mercury in scalp hair of healthy Singapore residents.  

FORSTEN L (1989).  
Blood mercury content after chewing.  

FRANCIS P.C ET AL (1982).  
Mercury content of human hair: A survey of dental personnel.  
J Toxicol & Env Health 10:667-672.

FREEMAN JA (1869).  
Mercurial disease among hatters.  
Were the hatters of New Jersey "mad"?  

Allergy to copper derived from dental alloys as a possible cause of oral lesions of lichen planus.  

Health implications of mercury in dental amalgam.  
In vivo mercury and methyl mercury levels in patients with amalgam restorations.

Chewing releases mercury from fillings.

A Mouthful of trouble.
Australian Wellbeing 34:67-68.

Debate flares over the safety of dental fillings.
New York Times (Dec 5).

Clinical toxicology of mercury.
J Toxicol & Env Health 2:491-526.

GETTLEMAN L (1986).
What can we do about the mercurophobes? Editorial opinion.

GLEICHMANN E, KIMBER I, PURCHASE IFH (1989).
Immunotoxicology: suppressive and stimulatory effects of drugs and environmental chemicals on
the immune system...a discussion.

GODFREY ME (1990).
A review of the amalgam toxicity controversy.

Occupational allergic contact dermatitis from metallic mercury contact.

Absorption and excretion of mercury in man. VII. Significance of mercury in blood.
Arch Environ Health 9:735-741.

Multiple Sclerosis a self help guide to its management.

GRAVER HT (1986).
Mercury toxicity and dental amalgam: an update.

GREER R (1982).
Diets to help Multiple Sclerosis. 2nd edn.
England:Thorsons Publishing Group.


Hidden dental health hazards.  

Methylation of mercury from dental amalgam and mercuric chloride by oral streptococci in vitro.  

Replacement of restorations based on material allergies.  
Quality evaluation of dental restorations.  
Chicago: Quintessence Publishing Co.

HERBECK L (1990).  
Amalgam poisoning and MS - a case history - or a walk through hell. Testimonial V.  

Exposure of dentists and assistants to mercury: mercury levels in urine and hair related to conditions of practice.  

Recommendations: Dental mercury hygiene.  

No evidence of hypersensitivity to dental restorative materials in oral lichen planus.  

The health revolution. 4th edn.  
Sydney: Southwood Press. p337-338.

HOWIE RA, SMITH HJ (1967).  
Trace elements in human and animal nutrition. 4th edn.  

HUGGINS HA (1982).  
Mercury: A factor in Mental Disease?  

HUGGINS HA (1983).  
Mercury - a factor in mental disease? Can mercury-silver fillings cause psychiatric symptoms?  

HUGGINS H, HUGGINS S (c1983).  
It's all in your head...diseases caused by silver-mercury fillings.  
Available from Health and Healing Bookshop Kingscliff, NSW, Australia.
Mercury vapour from amalgam fillings into oral air.  
Aust Dent Assoc (NSW Branch) Newsletter. (Sep):12

Clearance of mercury (Hg-197, Hg-203) vapor inhaled by human subjects.  
Arch Environ Health 31:302-309.  
Risk assessment of mercury exposure from dental amalgams.  

Percutaneous absorption of mercury vapor by man.  
Arch Environ Health 44(2):120-7.

Mercury concentration change in human hair after the ingestion of canned tuna fish.  

INGALLS TH (1983).  
Epidemiology, etiology and prevention of Multiple Sclerosis. Hypothesis and fact.  

INGALLS TH (1986).  
Endemic clustering of Multiple Sclerosis in time and place, 1934-1984...Confirmation of a hypothesis.  

Mercury toxicity.  
Report for Australian Dental Association (South Australian Branch).

JACKSON L (1986).  
Mercury: Clinical considerations in dentistry for children.  

Absorption and excretion of mercury in man. VI Significance of mercury in urine.  
Arch Environ Health 9:454-463.

Mercury allergy as a cause of burning mouth. Letter.  

Oral lichenoid reactions related to mercury sensitivity.  

JENKINS GN (1978).  
The physiology and biochemistry of the mouth. 4th edn.  
Mercury excretion and occupational exposure of dental personnel.

Amalgam-related chronic ulceration of oral mucosa.

Allergy to dental materials with special reference to the use of amalgam and polymethylmethacrylate.

KAISSER CR (Jan 1988).
Who will protect the children.
USA: Brochure.


Unwarranted and unprofessional: The superfluous removal of clinically acceptable amalgams.

KELMAN GR. (1978)
Notes and miscellanea: Urinary mercury excretion in dental personnel.
Environmental Health Criteria 118 - Inorganic mercury.
IPCS [International Programme on Chemical Safety]: Geneva.

Physical and mental development of stage 1: Preliminary tests at age 4. Solna Sweden, National Swedish Environmental Protection Board Report 3080.
The effects of methylmercury on the developing brain.

KLAASSEN CD, AMDUR MO, DOULL J Eds (1980).
Casarett and Doull's Toxicology.
Action of mercury in dental exposures to mercury.

KLAASSEN CD, AMDUR MO, DOULL J Eds (1986).
Casarett and Doull's Toxicology.

Effects of amalgam fillings on the mercury concentrations in amniotic fluid and breast milk.
Dtsch Zahnarzt (Germany) 45(3):142-145.
KOSTA L, BYRNE AR, ZELENKO V (1975).
Correlation between selenium and mercury in man following exposure to inorganic mercury.

Über die guerecksilber-konzentrationen in blut und urin von personin mit und ohne
amalgemfallungen.
Dtsch Zahnarzt 35:803-808.
Dental amalgam related mercury vapor exposure.
Calif Dent Assoc J 12:54-60.

Mercury amalgam toxicity- A major common denominator of degenerative disease.

LANGAN DC, FAN PL, HOOS AA (1987).
The use of mercury in dentistry: a critical review of the recent literature.

Mercury exposure from dental fillings I. Mercury concentrations in blood and urine.

Mercury exposure from dental fillings. II. Release and absorption.

Normal pituitary response to thyrotrophin releasing hormone in dental personnel exposed to
mercury.

LAUWERYS RR (1983).
Biological monitoring of exposure to inorganic and organometallic substances.
In: Industrial Chemical Exposure Guidelines for Biological Monitoring.
Davis C.A. Biomedical Publications. p9-50.
The contribution of dental amalgam to mercury in blood.

Prenatal and early postnatal intoxication by inorganic mercury resulting from the maternal use of
mercury containing soap.
Hum Toxicol 6(3):253-256.

Medical diagnosis and disease symptoms related to amalgam fillings.
Tandlakart Idningen (Swedish) 3:81-88.
Environmental Health Criteria 118 - Inorganic mercury.
IPCS [International Programme on Chemical Safety]:Geneva.
Multiple Sclerosis fact book.
Philadelphia:F.A.Davis Co.

LEK INQUIRY REPORT (CHAIRMAN PROFESSOR PO LUNDBERG) (May 1987),
Conclusion and answers to the questions put by the Department of Health and Social Welfare.
Sweden.

Effects of lead and mercury intoxications on evoked potentials.

Preliminary studies on methylmercury biotransformation and clearance in the brain of primates II.
Demethylation of mercury in brain.
Mercury concentrations in organs of contemporary Japanese.

LIND PO, HURLEN B, LYBERG T, AAS E (1986).
Amalgam related oral lichenoid reaction.

LIND PO (1988).
Oral lichenoid reactions related to composite restoration.

LOHYN R (Oct 1983).
Are your silver fillings making you sick?

A possible source of unrecognized mercury poisoning. Is the problem more widespread than we realize? Allergic responses to mercury based amalgam dental fillings.

LOHYN R (Mar 1989).
Mercury toxicity from dental amalgams.

Relationship between oral symptoms, smoking habits and general health in patients with complaints related to dental restorative materials.
Swed Dent J. 13:245-254

LORENZANI SS (1986).
Candida: a twentieth century disease.

MACKERT JR (1985).
Hypersensitivity to mercury from dental amalgams.
Factors affecting the estimation of dental amalgam mercury exposure from measurements of mercury vapour levels in intraoral and expired air.

MAGOS L (1988).
Thoughts on life with untested and adequately tested chemicals. Editorial.

Effect of Pd on the clinical performance of amalgam.

Mercury: health hazards in dentistry. Literature review and recommendations.

The release of mercury from dental amalgam: the mechanism and in vitro testing.

Fetal methylmercury poisoning: Relationship between concentration in single strands of maternal hair and child effects.
Arch Neurol 44:1017-1022.
The effects of methylmercury on the developing brain.

Copper rich and conventional amalgam restorations after clinical use.
J Am Dent Assoc 100:43-47.

Mercury content of amalgam restorations.

MARTIN S (Sep 1987).
Huggins exposes the great mercury cover up.

Mercury concentrations in organs of contemporary Japanese.
Arch Env Health 44(5):298-303.

McAlpine's Multiple Sclerosis.
Edinburgh: Churchill Livingstone.

Patients complaining about amalgam-related symptoms suffer more often from illnesses and chronic craniofacial pain than their controls.
An epidemiologic study of the relation between symptoms of fatigue, dental amalgam and other factors.  

Prevalence of mercury hypersensitivity in dental students.  

Mercury and crematorium chimneys. Scientific Correspondence.  
Nature 346(6285):615.

MJOR IA (1987).  
The safe and effective use of dental amalgam.  

MMWR (AUTHOR NOT GIVEN) (1990).  
Elemental mercury poisoning in a household - Ohio 1989.  

MOBERG L (1985).  
Corrosion products from dental alloys and effects of mercuric and cupric ions on a neuroeffector system.  

More preventive care, less tooth repair.  

Mercury release from dental amalgam in man.  

Mercury, selenium, and glutathione peroxidase before and after amalgam removal in man.  

Plasma-selenium, glutathione peroxidase in erythrocytes and mercury in plasma in patients allegedly subject to oral galvanism.  

Mercury, selenium and glutathione peroxidase in dental Personnel.  

Mercury concentrations in blood from Danish dentists.  

Health risks from increases in methylmercury exposure.  
Environ Health Perspectives 69:133-140.

MULTIPLE SCLEROSIS SOCIETY OF GREAT BRITAIN AND NORTHERN IRELAND (c1985). So you have MS?


NATIONAL HEALTH AND MEDICAL RESEARCH COUNCIL, C'WEALTH OF AUSTRALIA (Jun 1989). Dental amalgam and mercury in dentistry.


NATIONAL MULTIPLE SCLEROSIS SOCIETY OF USA (1978). What everyone should know about Multiple Sclerosis.


Heavy metal in your mouth. Hidden dangers at the dentist.

Effect of admixed indium on mercury vapour release from dental amalgam.

The prevalence of high levels of mercury in dentists' hair.

Placement and longevity of amalgam restorations in Denmark.

Die kristallinen komponenten der silberamalgam-untersuchungen mit der electronischen roentgenmikrosonde.
Zahnarzt Welt 79:1031-1036.

RAMSAY AM (1988).
Myalgic encephalomyelitis and postviral fatigue states. 2nd edn.
London:Gower Medical Publishing.

REINGOLD S (Nov 1985).
Increased media attention to the relationship between dental amalgam (fillings) and multiple sclerosis.

Exhaled mercury following removal and insertion of amalgam restorations.

Mercury vaporization during amalgam removal.

Risk assessment of mercury exposure from dental amalgams.

RENSON CE (1989).
Amalgam in modern dentistry.
Dental Update 16(10):415-416.

Maximum allowable concentrations of mercury compounds.
Arch Environ Health 19:891-905.

RICE DC (1989).
Brain and tissue levels of mercury after chronic methylmercury exposure in the monkey.
Mercury vapour released during the removal of old amalgam restorations.  

Cremation and the environmental mercury burden.  
Schweiz Monatsschr Zahnmed 100(11):1299-1303.

ROBINSON MF (PATHOLOGIST) (1986).  
Australia:Private communication

Detection of hand tremor in workers exposed to mercury vapour: A comparative study of three methods.  
Environ Res 49(2):152-165.

Relationships between the concentrations of mercury in air and in blood or urine in workers exposed to mercury vapour.  

Comparison of renal function and psychomotor performance in workers exposed to elemental mercury.  
Int Arch Occup Environ Health 50:77-93.  
Detection of hand tremor in workers exposed to mercury vapour: A comparative study of three methods.  
Environ Res 49(2):152-165.

Sensitive indicators of inorganic mercury toxicity.  

Multiple Sclerosis. Control of the disease.  
Oxford:Pergamon Press.

SANDSTEAD HH (1986).  
A brief history of the influence of trace elements on brain function.  

SCARLETT JM, GUTENMANN WH, LISK DJ (1988).  
A study of mercury in the hair of dentists and dental-related professionals in 1985 and subcohort comparison of 1972 and 1985 mercury hair levels.  

Multiple Sclerosis. A guide for patients and their families. 2nd edn.  


SMART ER (1985).
The hazards of mercury in dentistry.
Reviews on Environmental Health 5(1):59-86.

The contribution of dental amalgam to mercury in blood.

New York. ICRP Publication 23.
Toxicology and biological monitoring of metals in humans.
USA: Lewis Publishers. p151.

Effects of low exposure to inorganic mercury on psychological performance.

Penetration of metallic ions from restorations into teeth.

Mercury concentration in cord blood.
Arch Dis Child 63(2):202-203.

Hypersensitivity reactions in a dental group of patients.

Nothing new under the sun. Experiences with mercury poisoning.
Related by Dr Alfred Stock and Dr E Jaensch in 1926.

STORTEBECKER P. (c1985)
Dental caries as a cause of nervous disorders - Epilepsy - Schizophrenia - Multiple Sclerosis - Brain cancer.
Infertility and birth defects. Is mercury from silver dental fillings an unsuspected cause?
Florida: Bio-Probe Inc.

Increase mercury resistance in monkey gingival and intestinal bacterial flora after placement of
Physiologist 33:4.

SUNDERMAN FW (1988).
Perils of mercury.


Glomerular filtration impairment by mercury released from dental 'silver' fillings in sheep.
Abstract 94.
Physiologist 33:4.

Intra-oral air mercury released from dental amalgam.

Serial measurements of intra-oral air mercury: estimation of daily dose from dental amalgam.

Letter to the Editor.
Am Ind Hyg Assoc J 42(2):A92-A94.

Dental amalgam mercury daily dose estimated from intra-oral vapor measurements:
A predictor of mercury accumulation in human tissues.
J Trace Elements in Experimental Med 3:111-123.

Estimation of body burden from dental amalgam: Computer simulation of a metabolic compartment model.

Maternal-fetal distribution of mercury (203Hg) released from dental amalgam fillings.

Research on Multiple Sclerosis. 3rd edn.
New York:Demos Publications.

Letter to the Editor.
Am Ind Hyg Assoc J 47:A782-784.

Letter to the Editor.
Am Ind Hyg Assoc J 42(2):A92-A94.

WARREN HV (1989).
Geology, trace elements and health.

Were the hatters of New Jersey "mad"?
Does mercury from amalgam restorations constitute a health hazard?

Mercury as a contact allergen. Short Communication.
Contact Dermatitis 22(5):295-296.

WHITE RR, BRANDT RL (1976).
Development of mercury hypersensitivity among dental students.
Prevalence of mercury hypersensitivity in dental students.

Toxic chemical-free living and recovering from ME/CFS.
NSW Australia:Sally Milner Publishing. p68.

WHO (WORLD HEALTH ORGANIZATION) (1976).
Environmental Health Criteria 1 Mercury.
Geneva.
Cited in: AUST C'WEALTH DEPT HEALTH (1978)
Health aspects of mercury in fish.

WHO (WORLD HEALTH ORGANIZATION STUDY GROUP) (1980).
Recommended health-based limits in occupational exposure to heavy metals.

WHO (WORLD HEALTH ORGANIZATION) [UNITED NATIONS ENVIRONMENT PROGRAMME/INTERNATIONAL LABOUR ORGANIZATION/WORLD HEALTH ORGANIZATION] (Dec 1988).
IPCS [International Programme on Chemical Safety];Geneva.

Environmental Health Criteria 86 Mercury - Environmental Aspects
IPCS [International Programme on Chemical Safety];Geneva.

Environmental Health Criteria 101 - Methylmercury.
IPCS [International Programme on Chemical Safety];Geneva.

Environmental Health Criteria 118 - Inorganic mercury.
IPCS [International Programme on Chemical Safety];Geneva.

WIGGINS RC (1986).
Myelination: a critical stage in development.
Neurotoxicology 7(2):103-120.

Mercury vapour levels in a dental hospital environment.


