AMALGAM RESTORATIONS
AND
MERCURY TOXICITY

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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 malevolent 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.
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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; Lohny 1983; Pleva 1983; Stock & Jaensch 1983; Hanson c1984; Kupsinel 1984; Lohny 1984; Ziff 1984; Hanson c1985; Lohny 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

1 ppm = 1 mg/kg = 1 µg/g
1 ppb = 1 µg/kg = 1 ng/g
1 µmol Hg = 200.6 µg Hg
1 nmol/L = 2 µg/L = 0.02 µg/100 mL
1 ng/mL = 1 µg/L
1 µmol creatinine = 113.1 µg creatinine

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) [Hg\(^0\)]**

i. Elemental mercury

ii. Elemental mercury vapour

**Inorganic Compounds**

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

ii. Mercuric mercury [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 (Hg²⁺).’ 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 sulphhydryl 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 sulphydryl 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
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 M ethylation 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, Friiberg & 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 (ereethism) 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, Lauwerys (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 co-ordination) 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.

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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 (Nauern, 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.

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**MAXIMUM ACCEPTED DAILY LEVELS**

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<tr>
<td>USEPA</td>
<td>30</td>
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<tr>
<td>JFCA</td>
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<tr>
<td>MECCA (1973)</td>
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<td></td>
</tr>
<tr>
<td>Eley &amp; Cox (1987)</td>
<td>200.00</td>
<td></td>
</tr>
<tr>
<td>WHO (1991)</td>
<td></td>
<td>29</td>
</tr>
</tbody>
</table>

Table 2

- a Fish source from non-polluted area...contains both methylmercury and inorganic mercury
- b Non-specific foods....includes fish
- c No more than 28 µg/day methylmercury
- d 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 µg/m³ for mercury, aryl and inorganic compounds
- 50 µg/m³ for mercury vapour (all forms except alkyl)
- 10 µg/m³ for organo(alkyl)mercury

The short-term exposure limit for organo(alkyl)mercury is:

- 30 µg/m³ per 15 minutes.

8.1.2 UNITED KINGDOM

In Great Britain the environmental safety limit for mercury vapour is set at 50 µg/m³ (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)- $0.05 \text{ mg/m}^3$ (50 $\mu g/m^3$)
- Mercury (alkyl compounds)- $0.01 \text{ mg/m}^3$ (10 $\mu g/m^3$)
- Mercury (aryl & inorganic compounds)- $0.1 \text{ mg/m}^3$ (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 μg/ m³ for mercury vapour, whereas the Soviet Union has had a limit of 10 μg/ m³ since 1944.

8.1.6 Other Occupational Recommendations

Roels et al (1982 & 1987) have proposed a biological occupational limit for urine of 50 μg Hg/g creatinine to prevent preclinical alterations of hand-eye coordination and hand steadiness. This figure corresponds to 40 μg/ m³ 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/ m³ (25 μg/ m³) and that the biological urinary exposure indicator for biological monitoring should be 25 μg/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>SOURCE</th>
<th>AIR</th>
<th>BLOOD</th>
<th>URINE</th>
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<td>μg/m³</td>
<td>μg/l</td>
<td>ng/mL</td>
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<tr>
<td></td>
<td>μg/m³</td>
<td>μg/l</td>
<td>ng/100ml</td>
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<td>35 175</td>
</tr>
<tr>
<td>ACGIH (1983)</td>
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<td>USA, UNITED KINGDOM</td>
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<tr>
<td>CASARETT &amp; DOULL</td>
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<td></td>
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<tr>
<td>USSR,SWEDEN</td>
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<td>WHO (1980)</td>
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<tr>
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<td>BASED ON WHO(1980-1976)</td>
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<td>GERSTNER &amp; HUFF (1977)</td>
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<td>4 15</td>
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<td>100-200 (tremor)</td>
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<tr>
<td>BERLIN (1986)</td>
<td>50 (non-specific)</td>
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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.
[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]

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%).
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

\[
\text{gamma-2 phase...Sn}_{7.8}\text{Hg} \text{ (hexagonal structure)}
\]

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 (Cu₆Sn₅) and more gamma-1. The Cu₆Sn₅ is a halo surrounding the Ag-Cu particles. The final set consists of a core of Ag₃Sn and Ag-Cu surrounded by a halo of Cu₆Sn₅ and a matrix of gamma-1.

In single composition alloys the structure of the material is similar to that for blended alloys however the Cu₆Sn₅ 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 (SnO₂ and Sn(OH)₆Cl). 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, Cu₆Sn₅ 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 (mercurochrome, 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-Puttersen 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, Moeller-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 aetiological 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 etiologic 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/m$^3$ and from 30-60 μg/m$^3$ for particulate matter. These levels are well below the WHO values for short-term exposure of 500 μg/m$^3$. 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 μm 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 μg/m³ 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 μg/m³ 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 μg/m³ (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 μg/m³ is absorbed during 4 hrs of stimulated conditions and 0.4 μg/m³ 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 μg. The authors consider that the results of Vimy and Lorscheider (15-30 μg/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 μg. 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/m$^3$ 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/m$^3$ 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>
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<th>SOURCE</th>
<th>EXPOSED TO/UPTAKE</th>
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<th>URINE</th>
<th>BRAIN</th>
<th>KIDNEY</th>
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<td>μg Hg/L</td>
<td>μg Hg/kg</td>
<td>μg Hg/kg</td>
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<td>1563</td>
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<td>0.77</td>
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<td>0.7</td>
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<td>224</td>
</tr>
<tr>
<td>Viny &amp; Lorscheider (1985)</td>
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<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 anaesthetia. 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 \( \mu g \) day. These are presented in chronological order in Table 8.

<table>
<thead>
<tr>
<th>DAILY DOSE OF MERCURY VAPOUR FROM DENTAL AMALGAM (&gt;10 surfaces amalgam)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radics et al (1970)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sware et al (1981)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Abraham et al (1984)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Brune and Evje (1985)&lt;sup&gt;d&lt;/sup&gt;</td>
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<tr>
<td>Patterson (1985)</td>
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<tr>
<td>Patterson (1985)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Vimy &amp; Lorscheider (1985b)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Vimy &amp; Lorscheider (1985b)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Mackert (1987)</td>
</tr>
<tr>
<td>Langworth et al (1988)</td>
</tr>
<tr>
<td>Snapp et al (1989)</td>
</tr>
<tr>
<td>Marshall (1989)&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>Derand (1989)</td>
</tr>
<tr>
<td>Berglund et al (1990)</td>
</tr>
<tr>
<td>WHO (1991)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Table 8

<sup>a</sup> calculated in Clarkson et al (1988)

<sup>b</sup> based on Clarkson et al (1988) calculations of Sware, Abraham, Patterson, Vimy & Lorscheider.

<sup>c</sup> questionable conclusion

<sup>d</sup> mainly inorganic mercury compounds in amalgam particles

<sup>e</sup> Radics, Schwander and Gasser (1970)

The work of Berglund, Langworth, Snapp, and Mackert varies markedly from the earlier estimates of the studies by Sware, 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 & Lorscheider 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 & Lorscheider (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 μg/m³. Some floor level readings approached the British occupational exposure limit of 50 μg/m³ and on one floor where amalgam was being used continuously (with poor ventilation) the ambient mercury vapour level was 5-10 μg/m³. High readings (up to 160 μg/m³) were obtained within some dental spittoons presumably due to 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 μg/m³, with certain clinics having levels up to 150-170 μg/m³.

Nilsson and Nilsson (1986 a,b) reported mercury levels of 4 μg/m³ 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/m³ (50 μg/m³).
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 (Dentists)</td>
<td>4.9-36.0 (Assistants)</td>
</tr>
<tr>
<td>KELMAN (1978)</td>
<td>38.0 (Assistants)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>22.0 (Dentists)</td>
<td></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>1-21</td>
</tr>
<tr>
<td></td>
<td>7.0 (Assistants)</td>
<td>1-70</td>
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<td>NALOVAY (1985)</td>
<td>14.2</td>
<td>0-556</td>
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<td>JOKSTAD (1990)</td>
<td>78.0 (1986)</td>
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<td>VERSCHOOR (1988)</td>
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<tr>
<td>NORMAL (Reference)</td>
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</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.
BLOOD LEVELS OF MERCURY FOR DENTAL PERSONNEL (µg/L)

<table>
<thead>
<tr>
<th>SOURCE</th>
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<th>RANGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>BATTISTONE (1976)</td>
<td>9.8</td>
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</tr>
<tr>
<td>BRADY (1980)</td>
<td>8.5</td>
<td></td>
</tr>
<tr>
<td>CARREL (1981)</td>
<td>11.3 (Dentists)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>8.2 (Students)</td>
<td></td>
</tr>
<tr>
<td>MOLLER (1988)</td>
<td>4.0</td>
<td>1.2-19.2</td>
</tr>
<tr>
<td>NORMAL (Reference)</td>
<td>&lt;5.0</td>
<td></td>
</tr>
</tbody>
</table>

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 Jugoslavia where post-mortem samples showed high accumulation and retention of mercury in the thyroid and pituitary glands.

Langworth, Rojdamark 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, Juszkiewicz, 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 \( \mu \text{g} / \text{m}^3 \). Urine concentrations were also low (1.4-2.9 nmol Hg/mmole creatinine), being similar to that of the general Swedish population (5 \( \mu \text{g} / \text{L} \); 2.8 nmol Hg/mmole 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 μg/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 $\mu$g/m$^3$. 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 weighed 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
14. Difficulty recalling word  15. Depression  16. Loss of interest in work or former activities or hobbies
17. Crying spells  18. Tendency for fixed ideas, recycling or repeating of ideas

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
1. Fatigue (physical or mental)
2. Loss of former energy ("getting old")
3. Weakness
4. Edema (swelling)
5. Pallor
6. Inappropriate chilliness or excessive warmth
7. Excessive perspiration without fever
8. Unexplained fevers

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
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 dragging, 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 "mercuropohobes" 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.