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DENTAL PLAQUE AND PERIODONTAL DISEASE
IN YOUNG ADULTS

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INTRODUCTION

Recent research has emphasized the importance of dental plaque in the causation of periodontal disease.
Although the response to dental plaque is modified by individual tissue resistance factors, the deposition of dental plaque at the dento-gingival junction is the prodromal phase of periodontal disease.

This finding has led to a reappraisal of certain long-established measures in periodontal therapy. At the present time toothbrushing is the most important means of plaque removal in the prevention and treatment of periodontal disease.
As a basis for the assessment of current methods of oral hygiene instruction and toothbrush design, it would be helpful to ascertain the pattern of plaque retention on the teeth of individuals practising conventional oral hygiene procedures and to determine if this pattern varies with the individual's standard of oral hygiene.

It was therefore decided to study the pattern of plaque retention on the facial and lingual surfaces of the various teeth of young adults, and also to examine the distribution of periodontal disease in the dentition of these subjects. The sample population was chosen from dental students and from dental hospital patients to obtain a wide range of values for plaque and
periodontal disease.

In order to assess the effect of different standards of oral hygiene on the pattern of plaque retention, the distribution of plaque in individuals with a high standard of oral hygiene was compared with that in individuals with a low standard of oral hygiene. Similarly, the distribution of periodontal disease in subjects with a low prevalence of periodontal disease was compared with the distribution in subjects with a high prevalence of periodontal disease.

Before examining the distribution of plaque and periodontal disease, it was considered desirable to establish the relationship between these two factors for the entire population sample and to compare this relationship between individuals in both plaque and periodontal disease groups.

In a search for other aetiological factors, certain conditions affecting plaque retention and periodontal disease were also considered. These factors included calculus, restorations and carious lesions involving the gingiva, malocclusion, gingival recession and toothbrushing frequency.
REVIEW OF THE LITERATURE

1. THE FORMATION AND METABOLISM OF DENTAL PLAQUE

Although it is possible to regard the enamel portion of teeth as non-vital\(^\text{106}\), this tissue is not biologically inert. A dynamic equilibrium of ionic exchange between the tooth and its fluid environment has been clearly demonstrated to occur at the tooth surface\(^\text{130}\). The reactions occurring at this surface are mediated by one or more of several organic films which are interposed between the tooth and its fluid environment. Some of these films are of developmental origin and others are acquired after eruption. In most cases the developmental films that have been described appear to be lost after eruption\(^\text{170}\), and therefore play no part in the formation of dental plaque.

The acquired films on the teeth that are relevant to this study of dental plaque include the acquired pellicle, dental plaque itself, materia alba and calculus\(^\text{50, 136, 157, 164, 170}\).

The acquired pellicle (or cuticle) is deposited on the surfaces of the teeth that are exposed to saliva in the oral cavity as a thin organic film, 0.05 - 0.8 microns thick. It is essentially free of bacteria, and stains a light purplish-red with a disclosing solution such as basic fuchsin. Bacteria soon become deposited on the surface of the acquired pellicle, at the same time
surrounding themselves with a matrix that is distinctly different from the pellicle. This aggregation of bacteria with its matrix is known as dental plaque. It has been observed from 5 to 200 microns thick, and stains a bright red with basic fuchsine. Dental plaque forms first at the gingival margin, particularly in protected areas such as apical to the contact point on the proximal surface of teeth. Mature plaques are usually thickest at the gingival margin and taper in thickness occlusally. They are closely adherent to the tooth surface, but may be removed by toothbrushing, although not by rinsing. Dental plaque is thus a product of bacterial growth with a definite structure.

Materia alba (or debris), on the other hand, is a product of accumulation with no discernible structure. It is composed of food remnants, bacteria, desquamated epithelial cells and blood cells which adhere loosely to the surfaces of the teeth, plaques and gingiva. Materia alba stains similarly to dental plaque but, unlike plaque, it can be removed by forceful rinsing.

Under certain conditions crystals of various calcium phosphates may be deposited within the pellicle and the plaque. This process may result in the complete mineralization of the plaque to form supra-gingival calculus. Plaque also forms
under pathological conditions on the part of the tooth surface that is within the gingival sulcus, where the fluid environment is probably not saliva but the exudate of the sulcus. This subgingival plaque may mineralize to form subgingival calculus. The superficial surface of dental calculus is always covered by a layer of uncalcified plaque.

a. Microscopy.

Björn and Carlsson studied plaque morphogenesis directly on the facial surfaces of anterior teeth by examining, with a stereomicroscope, plaques made visible with a disclosing solution. As in the other studies of early plaque formation, the tooth surfaces were cleaned thoroughly and the subjects requested to refrain from oral hygiene procedures during the study. One day after the prophylaxis a lightly staining, bacteria-free film (the acquired pellicle) was observed to cover a large part of the tooth surface. After two to four days a much stronger staining material appeared, first in the defects in the enamel surface, and eventually in the whole surface coating. At the same time a number of small, discrete, densely staining hemispheres also appeared, mainly at the gingival margin. These hemispheres consisted mainly of coccoid bacteria. At five days these colonies of plaque were covered by haloes of a lighter staining material.
The haloes (interbacterial matrix) became more deeply staining as they grew and merged with one another. Further growth of the plaque colonies continued until they coalesced to form a dense microbial mass.  

McDougall studied developing plaque on the interproximal surfaces of extracted teeth. One day after the prophylaxis he found an essentially acellular, non-bacterial matrix overlying the acquired pellicle, which he called immature plaque. On the second day he observed discrete domes of bacteria growing up from the base of the immature plaque. These bacterial clumps rapidly invaded and replaced this material to form mature plaque. McDougall believed that filamentous bacteria remaining in enamel defects after prophylaxis played an important role in the regenerating plaque, and he distinguished between the rapidly reforming plaques produced by the multiplication of these organisms and the more slowly growing plaques produced by the random deposition of cocci and rods from saliva.  

Muhlemann and Schneider found that early plaques, formed on strips placed over the lingual surfaces of lower incisor teeth, consisted mainly of cocci, which after two to four days became progressively replaced by filamentous bacteria. Turesky and his co-workers observed that mature plaque had a
characteristic structure. The basal and older portion of the plaque consisted of tightly packed, parallel filaments arranged with their long axes at right angles to the tooth surface; while the outer, younger layers were made up of irregularly orientated organisms of various types.

Loe and his co-workers and Theilade and his co-workers, sampling plaque by pressing a thin plastic film against the tooth at the gingival margin, noted three phases in its development. At first there was a marked increase of resident gram positive cocci and rods with the appearance of gram negative cocci. Then, two to four days after prophylaxis, they observed a multiplication of filaments. Finally, a more complex flora, including vibrios and spirochaetes became established. These authors suggest that the shift in the composition of the plaque with increasing age is due to alterations in the local environment which favour the growth of certain types of bacteria.

Ritz adds that the change in composition of plaque flora from a predominantly coccoid to a filamentous character may be due to alteration in the oxidation-reduction potential occurring in the deeper layers of the plaque as it increases in thickness: This would favour the growth of anaerobic organisms causing them to supplant the aerobic or facultative flora.
Although most investigators found that the first stage in the formation of plaque was the deposition of the acquired pellicle, Frank and Brendel, using the electron microscope, observed that on proximal surfaces the initial occurrence was the apposition of a bacterial monolayer on the enamel. However in the electron microscope study of Leach and Saxton, the acquired pellicle was found under all plaques, although it tended to be thinner and more invaded by bacteria on proximal than on other surfaces.

Snyder and his co-workers warn that, although gram staining yields information on the staining reaction and number and distribution of different morphological types of bacteria in plaque, this does not constitute precise identification of their genus or species. For instance, they add, because some of the gram positive filamentous bacteria of the oral cavity exist in the coccal as well as the filamentous state, the classification of the organisms into cocci, rods and filaments may lead to an erroneous representation of the different species.

Mature plaque is thus a dense microbial mass, consisting of the acquired pellicle, bacteria and interbacterial matrix. In the majority of studies it was found that the most numerous organisms were gram positive and gram negative cocci,
followed by rods. Filaments were present in lesser numbers, but they appeared to dominate the other organisms due to their comparatively greater size, while their palisade-like arrangement in the deeper layers gave plaque its characteristic structure. Vibrios and spirochaetes were present only in small numbers. It is apparent that the bacterial population of plaque is very complex and variable, and as yet very few of the organisms have been specifically identified.

b. Biochemistry.

The principal components of saliva responsible for its viscosity are a group of complex macro-molecules, each containing a protein and a polysaccharide fraction, which are known as the glycoproteins or collectively as mucin. The glycoproteins of saliva contain two characteristic carbohydrates - sialic acid and fucose,

Winkler and Dirks state that all the surfaces in the oral cavity are covered with a mono-molecular layer of salivary mucoid, and that the acquired pellicle is formed by repeated deposition of this material onto the tooth surface. Walsh suggests that the adsorption of mucin changes the configuration of its protein molecule by a process of denaturation.

In support of these views, McGaughey and Stowell
found that mucin was strongly adsorbed by hydroxyapatite. On the other hand Hay found that the sialoproteins were not amongst the most selectively adsorbed proteins by hydroxyapatite and enamel, which suggests that mucin may be altered before being deposited as the acquired pellicle. However, Leach discovered that the composition of the pellicle resembled very closely that of the salivary glycoproteins, which indicates that in forming pellicle the glycoproteins at least partially resisted breakdown. Meckel investigated the staining reaction of pellicles formed on teeth and squares of enamel, both in vivo and in saliva in vitro, and he found the reactions in all cases to be almost identical. Bacteria did not seem to be involved in this process as neither the addition of bacterial nutrients nor antibiotics affected the formation of these pellicles.

Thus the acquired pellicle appears to be formed by the adsorption of salivary glycoproteins, which may be partially altered in the process, onto the tooth surface and its deposition is probably not dependent on either the activity of bacteria or the presence of dietary residues.

The formation of dental plaque and its retention onto the tooth surfaces is largely dependent upon the development of the plaque matrix. The protein component of the plaque matrix
is derived to a large extent from the salivary glycoproteins. Ferguson, by electrophoresis, found a close correlation between the proteins of saliva and those of plaque. Baumhammers and Stallard, using radioautography, identified labelled protein in salivary gland cells, saliva and plaque.

However, unlike the acquired pellicle, glycoproteins are only incorporated into the plaque matrix after enzymatic breakdown by oral bacteria. The main evidence for the occurrence of this reaction is constituted by the findings of Leach and Middleton, which showed that the sialic acid and fucose of the salivary glycoproteins are virtually absent in plaque. This was confirmed by Leach and Critchley who demonstrated that when oral bacteria are added to duct saliva, these carbohydrates are released from the glycoproteins and metabolized by the bacteria, causing the saliva to lose its viscosity and form a precipitate. The enzyme responsible for the release of sialic acid is neuraminidase, which may not only be derived from oral bacteria but also from leucocytes and epithelial cells.

Another endogenous substrate utilized by bacteria in the formation of plaque may be the gingival fluid. This is supported by the fluorescent studies of Salkind and his co-workers.
who found that, at least in patients with periodontal disease, the gingival fluid contributed to supra-gingival plaque$^{204}$. Apparently the gingival fluid contains all the necessary components to form the plaque matrix$^{231}$; however its contribution to the initial formation of plaque preceding periodontal disease has not been defined as yet.

Thus it seems that the main protein component of the plaque matrix is derived from the salivary glycoproteins (and possibly from the gingival fluid), which are broken down by bacterial action outside the plaque.

Exogenous sources supply most of the carbohydrate component of the plaque matrix which, unlike the protein fraction, is formed by the synthesis of carbohydrate within the plaque. Shaw and his co-workers, along with many other investigators, found that the addition of sucrose to a low plaque forming diet in experimental animals resulted in a dramatic increase in the accumulation of plaque$^{219}$. Significantly, sucrose is the only carbohydrate that can serve as substrate for the formation of large quantities of extracellular polysaccharides, although other sugars can promote limited synthesis of these compounds$^{65}$. Guggenheim and Schroeder have characterized the main polysaccharide component of the plaque matrix as a branched dextran
with a fibrillar structure 89.

Gibbons and his co-workers have shown that the streptococcus isolated from human plaque which can produce periodontal disease in rats, and Howell and Jordan that the filamentous organism which can produce periodontal disease in hamsters, are both able to synthesize large quantities of extracellular dextran 66, 101. The adhesive qualities of dextran and its marked biological stability appear to make an important contribution to the retention of plaque on the tooth surface in the oral cavity. It seems that the relatively small sucrose molecules are able to diffuse into the plaque where they are synthesized by the plaque bacteria to form the large and relatively immobile dextran molecules.

Many investigators, using rats and hamsters, have found that a soft carbohydrate diet (in most cases containing sucrose) favoured plaque formation and periodontal disease 5, 103, 118, 119, 175, 177, 212, 214, 217. On the other hand Carlsson and Elgerberg observed that the addition of sucrose to the diet of dogs did not increase the plaque-forming potential of the diet 40. Moreover Elgerberg found that neither varying the frequency of meals nor tube-feeding affected the accumulation of plaque in dogs. Thus it was concluded that the presence of food residues in the
mouth was not necessary for the formation of plaque\textsuperscript{54}.

In support of this view, McDougall noted that plaque appeared to form readily in humans whose diet contained little fermentable carbohydrate\textsuperscript{158}. Also Littleton and his co-workers found that plaque still formed in patients who were nourished by stomach tube, although it tended to be less extensive than in patients fed by mouth\textsuperscript{147}. But in another experiment on humans, Carlsson and Egelberg found that dietary supplements of sucrose increased the quantity of plaque formed\textsuperscript{40}.

Carlsson is of the opinion that the pioneer colonization of cocci in developing plaque is dependent on endogenous sources of nutrient, while the subsequent growth of the plaque matrix relies more upon dietary sources\textsuperscript{38}.

Leach suggests that, as plaque tends to form principally during fasting periods particularly at night when the oral cavity is depleted of carbohydrate residues from the diet, plaque consumption of endogenous substrate is most important; on the other hand dietary residues available after meals may successfully compete against this endogenous substrate for utilization by the bacteria to form the plaque matrix\textsuperscript{136}. Leach's view is based upon his observation that the addition of readily fermentable sugars to saliva depressed the production of the
enzyme, neuraminidase, which is involved in the breakdown of salivary substrate\textsuperscript{135}. The relative contribution of endogenous and dietary substrates to plaque formation in man has not been fully elucidated as yet.

c. Metabolism.

The metabolic activity of dental plaque is determined largely by the composition of its fluid environment which is constituted by saliva, dietary residues, gingival fluid, and possibly fluid from the enamel. The best defined of these factors are saliva and dietary residues. These nutrients act not only as substrates for bacterial metabolism but also they largely determine the composition of the plaque flora by establishing conditions which favour the growth of particular species of organisms.

Frostell discovered that, in general, the capacity of plaques was greater for the production of alkali than acid\textsuperscript{62}. Kleinberg and Jenkins found that the pH of fasting plaque was higher than that of saliva, which also indicated that alkali production was taking place. They observed that, after fasting, meals low in carbohydrate had little effect on this high pH level, but meals high in carbohydrate lowered the pH value of plaque. They also found that subjects with higher fasting plaque pH levels tended to have faster resting salivary flow rates. So they
concluded that the pH of plaque at any given time depends upon
the balance between acid produced by the plaque bacteria metabo-
lizing dietary carbohydrate and the base being continually pro-
duced by plaque consumption of endogenous substrate. As
carbohydrate residues are cleared from the oral cavity after meals,
less acid is produced to counteract base formation and the pH of
plaque slowly rises. 125

The relative supply of these two major classes of
substrate not only determines the pH of plaque, but also condi-
tions favouring the growth of alkali and acid producing organisms.
Thus Littleton and his co-workers found that the plaque material
from persons who were tube fed was much less acidogenic than
from persons fed by mouth. 147

Plaque has the ability to concentrate calcium.
This is due to the high calcium binding capacity of the organic
matrix of plaque and is an important factor in the movement of
calcium into and out of enamel and also in the formation of
calculus. 164 Koulourides states that undersaturation of the
plaque fluid with respect to hydroxyapatite results in dissolution
of tooth minerals, while supersaturation tends to form solids
either within the tooth structure (remineralization) or in the
organic matrix of plaque (calculus). 130
However in the mineralization of plaque it is unlikely that precipitation of calcium phosphate salts occurs simply when the pH and calcium and phosphate ion concentrations are sufficiently elevated. It is probable that mineralization is initiated by primary seeding of the organic matrix of plaque onto which calcium salts subsequently crystallize in the structural configuration of hydroxyapatite. Thus the organic matrix acts as a template for mineralization which can occur before the calcium and phosphate ion concentration is high enough to cause spontaneous precipitation.163

The role of the bacteria of plaque in the formation of calculus is not clear. Theilade and Schroeder observed that the first detectable signs of mineralization occurred within the matrix and that calcification of the bacteria did not take place until a later stage.241 The metabolic state of the bacteria in the initial stages of crystal formation is not known. Muhlemann suggests that the organisms may release a calcifiable extract which triggers off the seeding mechanism.178 Alternatively, death of the bacteria may precede mineral deposition, for Rizzo and his co-workers have shown that non-viable organisms can mineralize faster than viable organisms, indicating that the metabolic processes of viable bacteria are not an essential part
of this process\textsuperscript{195}. The fact that the bacteria-free acquired pellicle can calcify\textsuperscript{170} and the phenomenon of calculus formation in germ-free animals\textsuperscript{92} tend to support this view.

Rizzo and his co-workers conclude that if the minimal amount of calculus formed in germ-free animals is due to the lack of bacterial plaque, and if the organisms themselves do not play an active role, then the function of the bacteria may be merely to supply the calcifiable matrix of plaque\textsuperscript{196}.

In experimental animals, Plumbo and his co-workers found that a carbohydrate diet favoured calculus formation\textsuperscript{187}, while Baer and his co-workers and Gressly noted that a diet containing polysaccharide (cornstarch) produced more calculus than one containing disaccharide (sucrose)\textsuperscript{11, 87}. Baer and White, and Stewart and Burnett found that a high protein diet resulted in a greater formation of calculus than a high carbohydrate diet\textsuperscript{14, 230}. Kakehashi and his co-workers and Stewart and Burnett observed that diets supplemented with bicarbonate favoured calculus formation\textsuperscript{110, 230}. Finally Gressly showed that in hamsters phosphate supplements favoured the mineralization of plaques\textsuperscript{87}, although Kakehashi and his co-workers did not confirm this finding in rats\textsuperscript{110}.

In general these experiments signify that although
sucrose diets produce abundant plaques which may calcify, mineralization is favoured by polysaccharides, proteins and certain salts. These latter substrates presumably produce conditions within the plaque more favourable to the growth of alkali-producing organisms, for Little and her co-workers have shown that the remineralization of calculus in vitro is enhanced by high pH levels\textsuperscript{146}.

While the matrix of plaque forms the organic template for supra-gingival calculus formation, saliva appears to be the main source of its minerals. This is supported by the animal experiments of Kakehashi and his co-workers which demonstrated that dietary residues in the oral cavity had little effect on this formation of calculus\textsuperscript{112}, and Hugoson and Persson who showed that partial desalivation decreased the amount of calculus formed\textsuperscript{102}, and Kakehashi and his co-workers who found that total desalivation prevented the formation of calculus\textsuperscript{111}.

On the other hand it seems likely that the source of both the matrix (subgingival plaque) and the minerals of subgingival calculus is the fluid exudate from the gingival sulcus. This view is supported by the following observations: Waehaug showed that it was unlikely that saliva penetrated into the gingival
sulcus \(^{247}\); Baumhammers and Stallard using radioautography found labelled protein in saliva and supragingival calculus, but not in subgingival calculus\(^{17}\); Salkind and his co-workers with a fluorescent technique demonstrated that the protein component of subgingival plaque could not be derived from saliva \(^{204}\); Brill and Bronnestam demonstrated the outward flow of fluid from the gingival sulcus\(^{32}\); the gingival fluid has been shown to contain all the organic and inorganic components necessary for the formation of plaque and calculus \(^{231}\); and finally Gron and his co-workers and Little and Hazen have demonstrated distinct differences between the mineral composition of supragingival and deep subgingival calculus, which tend to reflect similar differences between the composition of saliva and serum exudate \(^{88, 145}\). Although there is convincing evidence that the source of subgingival plaque and calculus is the gingival fluid, no work has been done to elucidate the mechanisms of formation.

Many writers consider that the fall in pH resulting from the metabolism by acidogenic bacteria in plaque of readily fermentable dietary carbohydrate is the initial phase of dental caries \(^{164}\). Conversely it is possible that a decrease in the carbohydrate content of the diet and a corresponding increase in the protein content (which affects the urea content of saliva)
may result in a rise in the protein content and pH of plaque and a tendency to periodontal disease \textsuperscript{124}.

In experimental animals on certain dietary regimes a close correlation between caries and periodontal disease can be obtained, while other diets promote periodontal disease with almost no caries \textsuperscript{234}. Little and her co-workers have shown that in vitro the remineralization of calculus is pH dependent \textsuperscript{146}. Frostell found that the alkali production of plaques tended to increase in patients with periodontal disease, although he was unable to relate this factor to caries \textsuperscript{62}.

Kleinberg and Jenkins observed that just as areas in the mouth of low pH plaque level tended to coincide with regions of higher caries incidence, areas of high pH plaque levels could be related to the distribution of periodontal disease. They concluded therefore that the two oral conditions are related as they are both mediated by plaque, but that they occur on different portions of the pH scale. They proposed a further concept in which the dental plaque is considered to be "an environmental membrane between the tooth and saliva with the metabolism of the plaque bacteria playing a major if not determining role in the net movement of calcium phosphate salts between the two compartments" \textsuperscript{125}.
One objection to the experiments of Kleinberg and Jenkins is their use of antimony electrodes for pH measurements, as these have largely been supplanted by the more reliable glass electrodes for use in biological systems\textsuperscript{41}. But in general their concepts are consistent with the little data available on the metabolism of plaque, although they tend to oversimplify the metabolic changes occurring within plaque. For example, Frank and Brendel under the electron microscope observed both the apposition and destruction of crystalline mineral deposits within the plaque occurring in close proximity to the destruction of apatite in the enamel. This differential action in adjoining micro-areas indicates the complexity of the phenomena resulting from the metabolic activity of different species of bacteria in close proximity to one another\textsuperscript{61}.

Thus it is evident that dental plaque is a variable and dynamic entity with the interrelated factors of diet and bacterial flora largely determining its metabolic activity.
2. THE ROLE OF DENTAL PLAQUE IN PERIODONTAL DISEASE.

a. Epidemiological Studies.

Reliable information about the prevalence and severity of periodontal disease in different populations has only been possible in the last decade since the development of indices which permit a quantitative assessment of the more advanced stages of periodontal disease as well as the earlier stages of gingival pathology. The Periodontal Index of Russell is most commonly used, and provides estimates which have an acceptable degree of comparability when used by different investigators 201. Although this index is relatively simple and quick to use and reduces subjective elements of examiner opinion to a minimum, it tends to underestimate the severity of the disease mainly because there is no standard for the measurement of pocket depth 191.

One of the difficulties of epidemiological studies in this field is that periodontal disease may represent a complex of diseases with different manifestations and aetiologies 213. But in spite of this problem, recent studies consistently demonstrate the predominating correlation of increasing oral uncleanli-
ness and age with increasing prevalence and severity of periodontal disease, so much so that other significant factors may be masked.\textsuperscript{207}

Russell, in a recent review of the results of epidemiological research, states that there is an overwhelmingly positive correlation between oral uncleanliness and the severity of periodontal disease. From a purely statistical point of view, the association between the indices of mouth cleanliness (plus age) and index values for periodontal disease is so strong as to leave very little variation in disease to be accounted for by any factor other than age or oral hygiene. He adds that this same strong association is observed with different criteria of periodontal disease, with both crude and precise techniques of oral hygiene estimation and with varying statistical treatments.\textsuperscript{203}

For example Russell analysed relationships among the Oral Hygiene Index, Periodontal Index and age for multiple and partial correlations for two studies with unlike populations ethnologically, culturally, nutritionally and socio-economically. He found that less than ten per cent of the variance in the group scores, when taken separately or the two studies pooled, remained to be explained after the combined effect of age and oral uncleanliness had been estimated.\textsuperscript{202}
Oral hygiene status is measured in almost all of these studies by the amount of visible materia alba and calculus on the teeth. Greene found that the Periodontal Index and the Oral Hygiene Index increased roughly parallel to increasing age. But when the two components of the Oral Hygiene Index were considered separately, it was evident that the Debris Index reached a high level early in life and increased only slightly with time. The cumulative rise in the Oral Hygiene Index was caused mostly by a steady rise in the Calculus Index with age. Lilienthal and his co-workers found that up to twenty-five years both debris and calculus scores strongly influenced the Periodontal Index, but in the older age groups only calculus exerted any effect. These observations would seem to indicate that chronic periodontal disease is initiated by uncalcified plaque in youth and largely maintained and increased in severity in later years by calculus.

A relationship more consistent with current concepts of periodontal disease is illustrated by the findings of Lovdal and his co-workers, when they correlated the efficiency of oral hygiene with the incidence and distribution of subgingival calculus, gingivitis and periodontitis.

It is not suggested that the results of these
epidemiological studies constitute proof that plaque is the cause of periodontal disease for, as Scherp says, a close correlation is not necessarily a causal relation. There are, no doubt, interaction effects between plaque and periodontal disease. However an important objection to an aetiological interpretation of these studies is that, in general, materia alba and not plaque has been measured. It is probable that there is a close correlation between plaque and materia alba, but this has not been demonstrated. The best evidence for this is perhaps that the high correlation coefficient between plaque and periodontal disease found by O'Leary and his co-workers is almost identical with the correlation coefficient between materia alba and periodontal disease observed by Greene and Vermillion.

The failure to measure plaque may be one reason for the occasional exception to the otherwise uniformly consistent association of oral hygiene and periodontal disease. For example Barros and Witkop found that in Chile fourteen percent of the population over forty years were rated free of debris and calculus, and yet the Periodontal Index of this group was roughly one, which is within the gingivitis range.

Similarly, although Greene found a positive correlation between oral hygiene and periodontal disease in both India
and Atlanta, he observed that, while Indians had lower debris scores than Atlantans at similar ages, their periodontal scores were higher. Greene suggests that the inflammatory response must have been greater in Indians or that some other modifying factor must have been operating. But it is possible if plaque and not just materia alba had been measured, the oral hygiene status of Indians might have been found to be worse than that of Atlantans. This is likely because the oral hygiene methods of the Indians, as reported in this study (mainly rinsing, also finger rubbing and sticks) were probably more efficient in removing materia alba than plaque, which was more effectively removed by the Atlantans (by toothbrushing), who rarely rinsed.

On the other hand Brandtzaeg points out that although the severity of periodontal disease in these studies is a linear function of bacterial debris, the correlation between these two factors is an average one, and it leaves unexplained much individual variation which is most likely to be due to individual tissue resistance factors rather than to variations in the standard of oral hygiene. However it is apparent from these epidemiological studies that the prevalence and severity of periodontal disease are directly related to the level of oral hygiene, as determined by the amount of soft and calcified deposits visible
on the teeth.

b. Clinical Studies.

While epidemiological studies have established the close association of oral hygiene and periodontal disease, clinical investigations have defined this relationship as being essentially between plaque and periodontal disease and have further demonstrated that it is a relationship of cause and effect.

Loe and his co-workers and Theilade and his co-workers showed that the withdrawal of all oral hygiene measures in a group of subjects with clinically healthy gingiva resulted in the rapid accumulation of plaque and the subsequent development of chronic marginal gingivitis within nine to twenty-one days. Reinstitution of oral hygiene procedures causing the removal of the plaque produced a return to health of the gingiva within about one week. Although the time necessary to develop gingivitis, probably reflecting differences in host response, varied between subjects, the removal of plaque resulted in every case in a return to normal gingival health. Moreover, variations in the time required for individual gingival areas to return to normal within different mouths correlated with variations in the time taken to remove the plaque from these areas 151, 239.
Theilade and his co-workers found that the gingival inflammation usually disappeared one to two days after complete removal of the plaque\textsuperscript{239}. However, although Ash and his co-workers found a positive correlation between plaque and gingivitis both before and after prophylaxis on maintenance patients with fairly good oral hygiene, they showed that after prophylaxis the correlation varied with the period of time elapsed since the prophylaxis. As they observed the correlation between plaque and gingivitis to be much more significant at thirty days than at five to seven days, they suggested that there was a refractory period in the tissue response to plaque\textsuperscript{4}.

In studies on the efficiency of the automatic toothbrush, Hoover and Robinson, and Smith and Ash indicated that in patients with good oral hygiene there is not necessarily a correlation between plaque and gingivitis\textsuperscript{100, 222}. This suggests that, although in the vast majority of cases the quantity of plaque is the principle determinant in periodontal disease, under conditions of minimal plaque formation other factors such as tissue response may become more significant.

Hoover and Lefkowitz, after studying the fluctuations of gingival disease, demonstrated that toothbrushing even for one week reduced gingivitis scores significantly, and they
also found a marked net increase in gingivitis in patients who did not brush their teeth for one week. Saxe and his co-workers, using dogs over a period of eighteen months, found that quadrants that were allowed to accumulate large deposits of plaque and calculus developed marked gingival inflammation, while diagonally opposite quadrants that were cleaned every second day exhibited no significant gingival changes.

Lovdal and his co-workers showed that systematic oral hygiene instruction, interdental cleansing and regular scaling over a period of five years reduced the incidence of gingivitis even in patients with periodontitis. Imagawa and his co-workers observed that the decrease in the signs of inflammation following scaling and toothbrushing for two weeks was more marked in patients with reddish gingiva than in those with bluish gingiva or periodontitis.

Brandtzaeg and Jamison noted that one month after oral hygiene instruction, which was repeated once during this period, there was a significant reduction in the Oral Hygiene Index and the Periodontal Index. On the other hand, it has been found that unsupervised oral hygiene instruction and also increasing the frequency of toothbrushing may produce little change in debris and gingivitis scores. These findings
presumably reflected inefficient cleaning techniques. Koch and Lindhe found a statistically significant difference in gingivitis scores between children on prolonged oral hygiene supervision and a control group\textsuperscript{128}. Bernier and his co-workers observed that while the instructed use of a balsa wood stick and the uninstructed use of the toothbrush were equally effective in reducing oral debris and periodontal disease scores, vigorous rinsing was a poor substitute\textsuperscript{22}.

These studies demonstrate that if plaque is allowed to collect on the teeth in sufficient quantity it is likely that gingivitis will soon develop, and conversely that the physical removal of plaque will result in resolution of the gingival inflammation.

These observations are confirmed by studies on the effect of antibiotics on periodontal disease in man. Mitchell and Holmes showed that the topical application of vancomycin onto the teeth of mentally retarded patients with gross accumulations of plaque caused a marked decrease in plaque scores and also a reduction in gingivitis\textsuperscript{176}. Winer and his co-workers found that the systemic administration of penicillin, used as an adjunct to local therapy, produced a significantly greater improvement in gingival disease than local treatment alone\textsuperscript{257}. 
Chan, and Littleton and White observed that gingivitis tended to be less prevalent in children receiving extended antibiotic treatment than in normal children 42, 148.

c. Studies on Experimental Animals.

The many studies made on conventional experimental animals have confirmed the essential role of bacteria in the production of periodontal disease 51. The studies on rodents have culminated in the identification of specific organisms as the primary aetiological agents. Keyes and Jordan, by oral inoculation of subgingival plaque from a species of hamster prone to periodontal disease into another species which normally does not contract the disease, were able to transmit the disease to these uninfected animals 118. Subsequent analysis of the plaque led to the isolation of a pure culture of an anaerobic, gram-positive filamentous organism which induced periodontal disease when inoculated alone into uninfected animals. This organism was subsequently recovered from these animals and thus Koch's postulates were fulfilled 108. Jordan and Keyes succeeded in producing the disease in hamsters using a similar organism isolated from rats with periodontal disease, but they failed to do so with various filamentous bacteria isolated from human plaque. These investigators also induced the disease in germ-free
rats by infecting them with a filament from hamsters. Finally Gibbons and his co-workers demonstrated that a streptococcus isolated from human plaque can produce periodontal disease in germ-free rats. In all these experiments abundant plaques were formed. Thus it would seem that at least part of the destructive potential of these pathogens results from their ability to form plaque.

The key role of bacteria in the causation of periodontal disease in conventional experimental animals has been confirmed by the use of antibiotics. In hamsters and rats it has been found that antibiotics used topically or added to the diet cause a marked reduction in the accumulation of plaque and periodontal disease.

It would be expected that, as antibiotics inhibit the formation of plaque, they should have a similar effect on calculus formation. Although Fitzgerald and his co-workers and König and Muhlemann found that antibiotics had little effect on calculus formation in rats, the studies of Gressly on hamsters, Richardson on cats and Theilade and Fitzgerald on rats demonstrated that certain antibiotics at sufficiently high levels in animals on certain dietary regimes do inhibit the formation of calculus.
The role of the composition of the diet in the production of periodontal disease in experimental animals has already been discussed. It has been found that the most efficient way to induce the abundant formation of plaque in rodents is by the use of a carbohydrate diet containing sucrose. The effect of sucrose is essentially on the plaque flora.

When attempting to evaluate the deleterious effects of nutritionally adequate diets of varying consistencies on the periodontal tissues of experimental animals, one must distinguish between the plaque-promoting effect of a soft diet and the traumatic effect of a coarse diet. In general, it has been found that a soft diet tends to cause a generalized type of periodontal disease which is associated with the accumulation of plaque\textsuperscript{5, 35, 45, 53, 118, 121, 131, 175, 200, 212}. On the other hand a coarse diet produces a more severe but localized type of lesion resulting from the traumatic impaction of hard food particles into the periodontium\textsuperscript{5, 12, 45, 174, 175, 186, 226}. However even when mechanical trauma is involved in the aetiology of periodontal disease, the experiments of Stahl, and Rovin and his co-workers demonstrate that the bacterial component is still a most significant factor.

Stahl showed that the healing of a standardized
gingival wound in rats was accelerated by antibiotics, and Rovin and his co-workers found that the gingival irritation produced by cervical ligatures was minimal in germ-free rats, while in conventional animals there was a marked inflammatory response.

The experiments on the traumatic effects of coarse diets would seem to have little relation to the pathogenesis of periodontal disease in civilised man due to the soft nature of his diet. This underlines the difficulty of extrapolating data from experimental animals to man. Moreover impaction of hair due to preening is said to be a common aetiological factor of periodontal disease in many of these animals. Leung states that one must accept with reservation any statements concerning similarities in the pathogenesis of periodontal disease between animals and man until there is convincing evidence that the morphological, biochemical and physiological properties of the periodontal tissues are similar. One striking species difference is that the gingival sulcus in the rat and the hamster, unlike that of man, is lined by keratinized epithelium.

The occurrence of a type of periodontal disease in germ-free animals has been represented as a challenge to the bacterial concept of this disease. It is significant that in general
the lesions were characterized not only by little food or hair impaction but also by the absence of gingival inflammation\textsuperscript{6, 8, 9, 10}. Shaw noted that in the rice rat there appeared to be two periodontal syndromes - one resembling periodontitis in man, characterized by the accumulation of plaque, gingival inflammation and bone loss - the other like periodontosis in man with little plaque and gingival inflammation, but progressive bone loss. Shaw says that the periodontitis-like syndrome undoubtedly has a bacterial component and suggests that some experimental animals are also subject to an atrophic alveolar resorptive process - the periodontosis-like syndrome\textsuperscript{213}. This view is supported by the observations of Gupta and his co-workers who found that the use of antibiotics in rats caused a major reduction in soft tissue lesions but a much smaller decrease in alveolar bone loss\textsuperscript{90}, and those of Shaw and Krumins who showed that to a certain degree alveolar resorption progressed independently of gingival disease\textsuperscript{218}.

Baer and his co-workers have undoubtedly demonstrated a type of periodontal disease in germ-free animals, however irrespective of its aetiology it still remains that because of the lack of inflammation in their animals this type of periodontal disease differs from that commonly found in man\textsuperscript{198}. 
In general, these studies on experimental animals support the primary role of bacteria and plaque in the pathogenesis of periodontal disease.

d. The Role of Dental Plaque in the Pathogenesis of Periodontal Disease.

Dental plaque concentrates large numbers of bacteria in close relation to the gingival margin and protects them from removal during functional activity of the oral structures and possibly from the normal defense mechanisms of the oral cavity. This high concentration of bacteria maintained in close proximity to the gingival sulcus, which is the site of the initial lesion of periodontal disease, represents the prodromal phase of this disease. Although there is convincing evidence that bacteria are the primary aetiological agents of periodontal disease, their mode of action has not yet been elucidated.

Nor has it been possible to identify the specific bacteria that are involved in the production of periodontal disease in man. However Loe and his co-workers, and Theilade and his co-workers, in their studies of experimental gingivitis in man, observed certain significant changes in the bacterial composition of plaque which appeared to be associated with the development of gingivitis. These qualitative changes in the
plaque flora were a marked proliferation of gram negative cocci and filamentous bacteria.\textsuperscript{151, 239} Also MacDonald and his co-workers have presented evidence for the key role of Bacteroides Melaninogenicus as a pathogen.\textsuperscript{156} However Kelstrup was unable to correlate the incidence of this organism with either oral hygiene status or the degree of gingival inflammation.\textsuperscript{116}

Neither Gibbons and his co-workers, counting the predominant cultivable bacteria, nor Socransky and his co-workers, making direct counts and counting all viable and specific organisms, were able to find any significant qualitative differences in the flora of the gingival debris between normal and periodontally involved mouths.\textsuperscript{63, 224} This suggests that periodontal disease results from a quantitative increase in the bacterial flora rather than any specific change in its composition. This view is supported by the observations of Sharawy and his co-workers that the mass of bacterial debris was greatly increased in patients with periodontal disease,\textsuperscript{211} and the experiments of Courant and his co-workers which showed that the infectivity of debris from patients with periodontal disease differed little from that of patients with healthy gingiva.\textsuperscript{47} However more sensitive methods of detecting changes in the composition and virulence of the plaque flora may eventually
implicate certain specific bacteria as causative agents of periodontal disease in man.

There is no doubt of the pathological potential of the bacterial flora of dental plaque. This has been demonstrated by the subcutaneous injection of plaque into guinea pigs with the resultant development of abscesses and transmissible infections. However in chronic periodontal disease it is unlikely that the organisms actually invade the tissues. It is more probable that the pathological activity of the bacteria is due to the elaboration of toxic substances. Schultz-Haudt suggests that the initial phase of periodontal disease may be interference by bacterial enzymes with the adhesion of cells forming the dynamic epithelial attachment; this could permit the diffusion into the tissues of other enzymes elaborated by bacteria, such as collagenase and hyaluronidase, which may participate in the breakdown of gingival connective tissue. Proteolytic enzymes from the plaque flora may act directly on substrates in the gingival tissues or they may stimulate inflammation by releasing bound histamine and kinins within the tissues.

The failure as yet to relate the pathological activities of the bacteria of plaque directly to the initiation of
periodontal disease has led to a consideration of the disease in terms of host response. For even in the absence of clinically detectable inflammation the gingival sulcus harbours bacteria and the outcome of this host-parasite relationship must be partly determined by factors of tissue reactivity.

Taichman and his co-workers suggest that the leucocyte response to bacteria may result in a release of enzymes which could cause tissue damage \(^{238}\). Bennick and Hunt have demonstrated collagenolytic activity in inflamed gingiva \(^{21}\). Schneider and his co-workers have revealed the existence of an antigen-antibody reaction taking place between the bacteria of the gingival sulcus and globulins from the gingival tissues \(^{208}\). Mergenhagen has identified the endotoxins of gram-negative bacteria acting as the antigens in this immunological reaction \(^{171}\). Cowley suggests that as long as the antigens are produced by the sulcular flora then this is essentially a protective reaction; however it is possible that tissue components in the form of breakdown products of inflammation may act as antigens in an auto-immune reaction which would be harmful to the tissues of the host \(^{48}\). Thus it is possible that the plaque flora may cause periodontal disease by releasing toxic substances themselves or by stimulating the release of harmful agents from the host tissues.
3. THE PHYSIOLOGY OF THE DEPOSITION AND RETENTION OF DENTAL PLAQUE.

a. Masticatory Function and Dietary Consistency.

Kawamura states that one of the physiological purposes of chewing is the maintenance of oral hygiene\textsuperscript{113}, and indeed it is generally accepted that vigorous function promotes the health of the periodontal tissues. It has been shown that the pattern of masticatory movement varies with the character of the food eaten\textsuperscript{114}. Although it seems that every individual has his own characteristic type of masticatory pattern\textsuperscript{1}, each chewing stroke is essentially adaptive to the nature of the food being chewed\textsuperscript{70, 105}. It is apparent that in general the more resistant foods induce more vigorous chewing strokes\textsuperscript{180}, longer chewing times\textsuperscript{132}, and a greater number of chewing strokes\textsuperscript{70}. Thus vigorous and prolonged mastication is best achieved when the masticatory apparatus is challenged by a hard diet.

Many writers have attributed the relative freedom from periodontal disease enjoyed by wild animals and primitive man to the vigorous function imposed on their oral structures by their coarse diet. However the lack of consistency in the findings of different investigators makes it difficult to draw any
firm conclusions. On the other hand the protective effect of vigorous function against periodontal disease has been defined by animal experimentation as operating mainly through its inhibiting effect on the formation of dental plaque. Reference has already been made to the numerous studies on experimental animals indicating that diets of a soft consistency promote the accumulation of plaque and the development of periodontal disease. These studies have also demonstrated (apart from causing localized lesions due to food impaction) the protective action of diets of coarse consistency on the periodontal tissues due to their inhibition of plaque formation.

In humans, Hydak and his co-workers found that a diet confined to milk and honey for thirty days produced an increase in gingivitis\textsuperscript{103}. King observed that the chewing of sugar cane had a beneficial effect on the gingiva\textsuperscript{121}. Slack and Martin found that the eating of apples after meals for two years improved the gingival condition of children when compared to a control group\textsuperscript{221}. O'Leary and his co-workers showed that a liquid diet of the consistency of baby food maintained for thirty five days resulted in an increase in plaque formation and gingivitis\textsuperscript{184}. Although the design of most of these human experiments precludes positive inference, they tend to confirm
the results of animal experimentation on the effect of varying dietary consistencies on the periodontium.

Egelberg found that tube-fed dogs had even more gingival disease than those fed on a soft diet. On the other hand Littleton and his co-workers noted that the plaque formed in patients who were nourished by stomach tube was less extensive than that in patients fed by mouth. But Hine says that in humans it is a common clinical observation that a quadrant not used for mastication is predisposed to plaque deposition and periodontal disease. Emslie adds that in the skulls of primitive man, gross accumulations of calculus and periodontal disease are found only in non-functioning teeth that have lost their antagonists. This finding was confirmed in rats by Baer and White, who showed that non-functioning teeth had higher calculus scores than those with antagonists. Finally Leach states that plaque tends to form mainly during fasting periods, particularly at night.

Thus the experiments and observations on humans and laboratory animals support the view that lack of function or the minimal function associated with a soft diet predisposes to the accumulation of plaque, and conversely that the vigorous function induced by a hard diet inhibits the formation of plaque.
and the development of periodontal disease.

Klatsky in 1948, and many other writers since, have interpreted the beneficial action of a coarse diet as operating mainly through its mechanical cleansing action. However, clinical experiments testing the efficacy of the detergent action of various foods do not support this contention. Wilcox and Everett painted the teeth with a preparation that would easily rub off with a little friction, and observed the amount of material removed by the mastication of certain types of foods. They found that although fibrous foods had a slightly greater detergent action than soft foods, generally none of them cleaned any more than the occlusal surfaces and the occlusal one third of the facial and lingual surfaces of the teeth. Arnim stained the plaque on the teeth of subjects who had suspended their oral hygiene procedures for one week. Then the subjects chewed a variety of foods vigorously over a period of three hours with the intention of removing as much plaque as possible. Planimeter measurements of the restained plaque remaining on the teeth indicated that only the chewing surfaces were well cleaned. Facial and lingual surfaces were poorly cleaned, and there was no perceptible change in the plaque on the proximal surfaces. The poor plaque-removing property of the so-called detergent
foods was again confirmed by Emslie. If the beneficial effect of a coarse diet does not result from its cleansing action in removing plaque that has already formed on the teeth, then it must act in other ways—presumably by inhibiting the formation of plaque and/or by increasing the resistance of the gingival tissues to the deleterious effects of plaque. At this point it is relevant to reconsider the role of the composition of the diet in the formation of plaque. Of course, it may be objected that the composition of the diet could have a significant systemic effect on the health of the periodontal tissues. However it is unlikely that nutritional factors are important in the etiology of common chronic periodontal disease in man, and even in frank deficiency, states it seems that the initiation of periodontal disease usually requires the local irritation of plaque.

Reference has already been made to the numerous studies on humans and experimental animals indicating that refined carbohydrate diets, particularly those containing sucrose, promote the formation of abundant plaques. It seems probable that dietary residues in the oral cavity aid the formation of plaque by supplying substrate for bacterial metabolism. Apart from their high content of refined carbohydrate, many of the
constituents of the modern diet have another characteristic - that of adhesiveness. In this respect it is interesting to note that most of the epidemiological surveys implicating oral uncleanliness as the major aetiological factor in periodontal disease have measured not plaque but materia alba, which usually consists partly of food debris. Thus it seems probable that an important aspect of the effect of the modern diet on plaque formation, quite apart from its lack of coarse consistency and its composition, is its retentive quality in the oral cavity.

Bibby and his co-workers have reported that foodstuffs vary considerably in their clearance times from the oral cavity, and that this factor is significantly affected by their texture. Several investigators have shown that many of the soft foods in the modern diet are retained on the teeth due to their adhesive qualities for a much longer time than other foods of a more fibrous or coarser texture. Moreover Shaw found that replacement of the relatively sticky and adhesive casien by an amino acid mixture in the diet of rats resulted in decreased plaque formation and gingival disease.

Although the so-called detergent foods have been shown to remove little plaque once it has formed, it is possible that they inhibit the initial formation of plaque by removing
dietary residues from the oral cavity. Turesky and Bibby found that foods, which are harder or require more chewing, eliminate more retained food debris from the oral cavity than softer foods.

Thus it appears that the decreased function on a soft diet, which predisposes to periodontal disease, operates partly through the retentive qualities of the diet by supplying substrate for the formation of plaque. On the other hand the protective effect of the vigorous function induced by a hard diet may be partly due to the removal of dietary residues from the oral cavity, thus inhibiting the formation of plaque.

b. Saliva.

The limiting effect of a hard diet on the formation of plaque is largely mediated through its action upon the secretion of saliva. For the physical character of the diet, by affecting the vigour and duration of mastication, in turn affects the rate of flow of saliva and thus the rate of clearance of debris from the oral cavity.

Schneyer and his co-workers found that little saliva was secreted during sleep. Clark and his co-workers and Cobb and his co-workers have demonstrated that although the basal salivary flow (during waking hours) has a beneficial
effect on the removal of carbohydrate debris from the oral cavity, the stimulated, profuse flow of saliva is much more efficient. Knighton has shown that fibrous foods tend to flush away more food debris than soft foods.

The salivary flushing mechanism is also an important regulator of the oral bacterial population. The functional movements of the oral soft tissues produce currents whereby food debris and bacteria are washed to the rear of the oral cavity where they are eliminated by swallowing. Lear and his co-workers found that the frequency of swallowing, and therefore the rate of salivary flow, was greatest during eating and least during sleep with other activities occupying an intermediate position. The bacterial population of the oral cavity appears to be inversely related to the swallowing frequency; it is lowest after meals and highest after a night's sleep.

There are no doubt antibacterial properties contained in the secretions of the salivary glands, although it seems that the best defined of these factors to date - lysozyme - may be ineffective against the bacteria that form plaque. Also by virtue of its physico-chemical properties saliva may play an important part in reducing the adhesiveness of foods and in emulsifying and dispersing foodstuffs. The dependence of
the health of the periodontal tissues upon the normal salivary secretion has been demonstrated by the pathological changes following the desalivation of experimental animals 91, 102, 192, 199.

Thus, on one hand, functional stimulation of a profuse flow of saliva flushes bacteria and food debris from the oral cavity which inhibits the formation of plaque, while on the other hand, under conditions of stasis, saliva acts as one of the substrates which bacteria metabolize to form dental plaque.

c. Gingival Fluid.

It has for many years been suggested that part of the beneficial action of a hard diet on the periodontal tissues and also of certain oral hygiene procedures is due on increase in the local gingival circulation caused by the stimulation of foodstuffs and oral hygiene devices passing over the gingiva 74, 185. This view appears to have been confirmed by the recent research on the gingival fluid.

Hard diets, vigorous mastication, gingival massage and toothbrushing cause an increase in the flow of fluid from the gingival sulcus, which follows changes in the blood flow and permeability of the gingival blood vessels. It has been suggested that this flow of gingival fluid has a protective action on the gingival tissues by mechanically washing away bacteria.
and their products from the gingival sulcus and by preventing the formation of plaque. It has also been shown that there is an increased flow of gingival fluid on a soft diet following plaque formation and gingival inflammation.\textsuperscript{25, 32, 33, 34, 55} It has been proposed that the protective action of the gingival fluid may be partly mediated through its leucocyte and immunoglobulin content.\textsuperscript{28}

On the other hand, when Loe and Holm-Pederson were unable to detect a flow of fluid from normal gingival sulci, they took the view that it was a pathological entity resulting from inflammatory changes in the periodontal tissues.\textsuperscript{150} Although others have detected small but definite amounts of fluid flowing from normal sulci,\textsuperscript{255} most writers still consider it to be essentially an inflammatory exudate, and thus throw doubt upon its protective role in preventing the formation of plaque or in counteracting the harmful effects of plaque.

Collins and Gavin found that, far from inhibiting the viability of oral bacteria, the gingival fluid supported the growth of many organisms.\textsuperscript{46} It seems that the gingival exudate is one of the substrates contributing to the metabolism of supragingival plaque\textsuperscript{204}, and there is little doubt that it forms the principle substrate for the formation of subgingival plaque.\textsuperscript{231}
Thus although it is probable that the gingival exudate following inflammation of the gingiva supports the growth of plaque, the plaque-inhibiting or protective effect of the gingival fluid under normal conditions still remains to be demonstrated.

It is difficult to reconcile these two conflicting concepts of the gingival fluid but Salkind and his co-workers, and Weinstein and Mandel suggest the possibility that it may consist of two separate entities - a modified tissue fluid flowing from healthy sulci which forms part of the defense mechanism of the normal gingiva - and an inflammatory exudate produced by pathological changes in the gingival tissues and contributing to the metabolism of plaque^204, 254.

O'Rourke in 1947, and many other writers since, have suggested that the protective function of a coarse diet and artificial gingival massage is partly due to a keratinizing effect on the gingival epithelium^185. But as only the oral surface of the gingiva is so affected^37, and as the non-keratinized, sulcular surface is the site of the initial lesion in periodontal disease, it seems unlikely that the process of keratinization plays a significant part in the prevention of periodontal disease in man.

After reviewing the physiological aspects of
masticatory function and dietary consistency, saliva and gingival fluid that are related to the formation of dental plaque, it is apparent that although these factors are significant in the inhibition and formation of plaque and even in its subsequent growth, once plaque has formed it can only be removed by artificial methods of oral hygiene.

d. Tooth Form.

The characteristic tendency of dental plaque to form on certain selected areas of the teeth is recognized in the concept which divides the surfaces of the teeth into self-cleansing and non-self-cleansing areas. This phenomenon must be related to physiological activities within the oral cavity, which protect some regions of the tooth surface more than others against the retention of plaque.

During mastication food is forced from the occlusal surfaces of the teeth over the facial and lingual surfaces. At the same time the facial mucosa exerts pressure against the teeth to prevent food from falling into the vestibule, and the tongue presses foodstuff against the hard palate and lingual surface of the upper dental arch. The movement of the soft tissues over the surfaces of the teeth also aids the mechanical
cleansing of these surfaces by forcing saliva against them to wash the teeth.\textsuperscript{76}.

The tongue is constantly in action during mastication and deglutition\textsuperscript{115}. After swallowing, sweeping movements with the tongue and lips are often made to remove food debris from the teeth\textsuperscript{133}. Other functional activities of the mouth such as speaking also cause frictional contact between the soft tissues and the teeth.

Wilcox and Everett studied the surfaces of the teeth that were most subject to frictional forces during mastication. They found that in general the surfaces most affected were the occlusal and incisal surfaces of the teeth, the occlusal one third of the facial and lingual surfaces of the maxillary posteriors and the occlusal two thirds of the facial surfaces of the mandibular posteriors. The facial surfaces of the maxillary and mandibular anteriors and the lingual surfaces of the mandibular posteriors received little friction. The lingual surfaces of the anteriors were affected somewhat more. There was little frictional contact of interproximal surfaces and of the gingiva except in the lingual region of the maxilla\textsuperscript{256}.

Thus it appears that the areas in which plaque first forms - the gingival portion of the facial and lingual surfaces
and the interproximal region - are also the areas of the teeth least subject to frictional forces during mastication.

The individual and collective contours of the teeth are largely responsible for this differential action of frictional contact. It is commonly accepted that the function of the facial and lingual curvatures of the crowns of the teeth is to deflect food away from the gingival margin and interdental papilla. However Herlands and his co-workers, while accepting the necessity for the prevention of food impaction interproximally, doubt the existence of a potential impaction mechanism on the facial and lingual surfaces under normal conditions. Van Reenen is of the opinion that the protection afforded to the marginal gingiva of primitive man by this convex tooth form may have outlived its usefulness in modern man due to the change in his diet. Moreover Shear suggests that man's incompetent dentition (predisposing to the accumulation of plaque) is unlikely to be improved by evolution to a more efficient form as man is no longer dependent upon his teeth for survival.

Irrespective of the validity of these speculations, it appears that plaque first forms on the areas of the teeth most inaccessible to the natural frictional mechanisms of the oral
cavity. This view is supported by the finding of Baer who reported that calculus first formed in experimental rats on the mesial surface of the maxillary first molar which is the only concave surface in the dentition of these animals. Also Turesky and his co-workers indicated the importance of the local environment in the formation of plaque and calculus, when they found that the placing of a celluloid strip over the lingual surface of the lower anterior teeth of children induced calculus formation.

These last two findings may indicate that morphological factors influence not only the formation of plaque but also its subsequent metabolism. Nevin and Walsh found that variation of interproximal shapes altered the rate of diffusion of substrates into and out of interproximal spaces. Winkler and Dirks have demonstrated that the effective plaque thickness (the distance from the enamel to the plaque-saliva interface) is greater in interproximal plaques than in plaques on the facial and lingual surfaces. Stralfors has shown that the thickness of the plaque has an important effect on the dilution of the end products of plaque metabolism by the oral fluids. Makinen found that the activity of proteolytic enzymes was higher in plaques from protected areas of the
dentition than from areas being continually washed by saliva 162.

Perhaps the most important aspect of plaque depth is its influence on the character of the plaque flora by producing conditions within the deeper layers of the plaque which cause a change in the composition of the bacterial flora there. For this reason it is possible that plaque must reach a critical thickness before exerting a pathological effect on the gingival tissues.

On the other hand ageing of the plaque may be the more important factor determining the composition of the flora. Although the significance of plaque thickness and ageing in terms of pathological potential awaits further definition, there is little doubt that morphological factors due to their effect on the local environment have an important influence on the growth and metabolism of plaque. Leach and Saxton observed that proximal plaques had a dendritic structure and that they were continuous with the subsurface enamel components with often little evidence of the acquired pellicle. Labial plaques had a thicker pellicle and a thinner bacterial portion, while lingual plaques had a greater tendency to calcification than either their proximal or labial counterparts 138.

Of course the physiology of plaque deposition
and retention on the teeth of man is modified greatly by the relative efficiency of his oral hygiene procedures on the different surfaces of the teeth.

It is a common observation that in most dentitions only the facial surfaces of the teeth are subject to any efficient degree of artificial cleansing, and that proximal surfaces are the most neglected. Gingivitis and pathological pockets are most common interproximally, less common lingually and least frequent on the facial surfaces.\textsuperscript{98, 141, 151, 152, 153} This same distribution holds true for the accumulation of plaque and calculus.\textsuperscript{251}

It is interesting to note that, as a result of his studies on the efficiency of toothbrushing, Goldman came to the conclusion that teeth with healthy gingiva were easier to keep free of plaque than teeth with a previous history of periodontal disease.\textsuperscript{75} This is no doubt largely due to the more complex morphology of the cervical and embrasure areas of the teeth when they are exposed by periodontal disease.

These findings indicate that plaque forms most commonly on those areas of the teeth that are most inaccessible to physiological frictional activities and artificial oral hygiene procedures. However, any attempt to explain the tendency to
localization of plaque as being solely due to the morphological features of the teeth may exclude other significant factors. For instance the cervical contour cannot be the major determinant, as plaque appears to deposit just as readily at the gingival margin of partially erupted teeth as at the gingival margin of teeth that have erupted beyond their maximum cervical contour.

This suggests that an important consideration may be simply the proximity of the area to the gingival sulcus. If there is a protective mechanism operating within the gingival sulcus due to the gingival fluid, and also a cleansing mechanism in the oral cavity mediated by saliva, then it is conceivable that, as plaque forms first at the junction of these two environments, the interaction between the two fluids may play a significant part in the formation of plaque.

As the retention of plaque in man is influenced most strongly by the efficacy of his oral hygiene procedures, the relative accessibility of the various surfaces of the teeth to these measures will largely determine the pattern of plaque retention in his dentition.
4. **SOME LOCAL FACTORS PREDISPOSING TO PLAQUE RETENTION.**

a. Malocclusion.

The preceding observations on the influence of morphological factors on the deposition and retention of plaque imply that the physiological protective and artificial cleansing mechanisms are most likely to be optimal when the teeth are normally located in relation to other oral structures and correctly aligned with one another, and conversely that mal-alignment of the teeth may predispose to periodontal disease by making some surfaces of the teeth less accessible to these cleansing mechanisms. If it is true that malocclusion predisposes to periodontal disease, it seems probable that the pathological potential of malocclusion operates mainly through its effect on plaque retention.

Hirschfeld and Prichard have stressed the effect of irregularities of tooth alignment in creating areas of stagnation both between the teeth themselves and between the teeth and the gingiva, resulting in the latter case from variations from ideal alveolar and gingival form. The animal experiments of Klingsberg and his co-workers indicate that if inadequate lip-seal is an aetiological factor in periodontal disease, it is
more likely to be due to interference with cleansing mechanisms than to dehydration of the soft tissues. Also hypofunction resulting from abnormal occlusal relationships may encourage the accumulation of plaque.

However malocclusion may predispose to periodontal disease other than through its effect on plaque retention. Although in the normal dentition of modern man food impaction mechanisms may not be operative on facial and lingual surfaces, there seems little doubt that under certain conditions, such as deep anterior overbite and isolated facial or lingual version, masticatory action can result in food impaction or direct trauma to the gingiva. Furthermore it is generally accepted that alteration of the protective proximal form or contact relation of teeth is a common cause of interproximal food impaction. Similarly, malocclusion can result in plunger cusps forcing food into the periodontium of opposing teeth.

Although it is possible that disharmonies of the occlusion may predispose to traumatic occlusion, most occlusal disharmonies are not traumatic and, even then, traumatic occlusion does not initiate periodontal disease. However it is probable that in the presence of an already existing periodontitis, traumatic occlusion acts as a serious aggravating factor.
increasing the severity of the lesions.\(^{71}\)

Stern has drawn attention to the need for the accumulation of data to substantiate these concepts.\(^{223}\) Several writers, in epidemiological studies mainly on children and young adults, have reported finding significant correlations between malocclusion and gingivitis.\(^{169, 173, 197}\)

More specifically, Hellgren, McCombie and Stothard, and Trott and his co-workers have related crowding to gingivitis. McCombie and Stothard also found a significant relationship between anterior overbite and overjet and gingivitis, but this finding was not confirmed by Hellgren. Trott and his co-workers were unable to relate lip posture to gingivitis.\(^{94, 155, 242}\)

From studies of individuals in similar age groups, Emslie, and Sanjana and his co-workers concluded that gingivitis could be ascribed to food stagnation, calculus and lack of lip seal, but not to functional loads from the occlusion.\(^{57, 205}\)

There is far less agreement in the reports of different investigators on the effects of malocclusion on periodontal disease, which includes the more advanced stages of the disease as well as gingivitis. This is to be expected due to the greater number of modifying factors operating in periodontitis.
compared to gingivitis.

Ditto and Hall observed a higher prevalence of crowding in patients with periodontal disease compared with a group of students in periodontal health.\textsuperscript{52}

Beagrie and James, Geiger, and Gould and Picton were unable to relate malalignment to periodontal disease. These studies were conducted on a small number of individuals. Gould and Picton did find a significant correlation between increased overbite and overjet, but this finding was not confirmed in the study of Geiger. On the other hand Geiger noted that the severity of periodontal disease was significantly greater about teeth in cross-bite relation and teeth migrated following extractions. Gould and Picton observed no correlation between periodontal disease and cross-bite, although they found a positive correlation between periodontal disease and teeth with poor proximal contacts\textsuperscript{19, 64, 79}.

Yuodelis and Mann observed periodontal disease to be more severe around teeth with non-working facets of wear\textsuperscript{260}.

Using the Occlusal Feature Index and the Russell Periodontal Index in 963 young adults, Poulton and Aaronson correlated periodontal disease with anterior overbite, posterior cuspal interdigation, lower anterior crowding and horizontal
overjet, in decreasing order of significance\textsuperscript{189}. Bilimoria using the same criteria for malocclusion and periodontal disease and also the Oral Hygiene Index in 800 young adults, found a strong correlation between these two conditions and concluded that oral hygiene was less significant than occlusal factors in periodontal disease\textsuperscript{24}.

On the other hand Greene states that, employing these same three indices in 2000 individuals, he was unable to establish any relationship between the Occlusal Feature Index (or any of its parts) and the Periodontal Index when the oral hygiene status was taken into account\textsuperscript{84}.

While the conclusions of Bilimoria do not support the hypothesis that the pathogenic potential of a malocclusion operates mainly through its effect on the retention of plaque, the findings of Greene are not inconsistent with this view.

The studies on the effect of malocclusion on simple gingivitis appear to support the belief that malocclusion, and particularly crowding predispose to gingivitis. However the studies on malocclusion and periodontal disease indicate that much more data is required to define the effects of the various features of malocclusion on plaque retention, food impaction and other occlusal factors in the causation of periodontal disease,
b. Restorations and Carious Lesions.

Restorations involving the gingival portion of teeth have frequently been implicated as aetiological agents of periodontal disease. This results partly from the surface qualities of the restorations as rough, unpolished surfaces facilitate the accumulation of plaque. Of the materials commonly used only gold alloys and glazed porcelain appear to possess the ideal qualities of a smooth, non-porous surface, which is chemically and electrically inert and can be polished before insertion of the restoration; amalgam, plastic materials and silicate cements are questionable, the surface of the last two being particularly unstable in the oral cavity.

But the marginal fit of restorations, particularly of those extending subgingivally, is probably the most common factor predisposing to periodontal disease. The mechanical insult to the gingiva of an overhanging or under-extended gingival margin is of little significance compared to the retention of plaque that such defects encourage.

Morphological features of restorations also predispose to periodontal disease. Inadequate coronal contours or exaggerated contours particularly in the embrasure area may lead to gingival overgrowth and create areas for the retention of
plaque. Faulty proximal contacts and failure to reproduce marginal ridges are common causes of interproximal food impaction. Also restorations not in harmony with the occlusion may lead to traumatic occlusion 73.

Carious teeth may have a threefold action in initiating periodontal disease - food impaction interproximally, mechanical irritation from the jagged edge of the cavity, and plaque retention resulting from hypofunction or inadequate cleaning due to local pain 142.

The main evidence for the role of faulty restorations and carious lesions in the causation of periodontal disease is based on clinical experience. This is apparently so overwhelming that Loe, and O’Leary, have incorporated these factors along with plaque and calculus in their indices used to measure the quantity of local irritant exposed to the gingiva 149, 182.

Animal experimentation has confirmed the effect of faulty restorations on the periodontal tissues, although some of the minor histological changes found in association with acceptable restorations may not be of clinical significance 166, 252, 261.

c. Gingival Recession.

Recession of the gingival margin beyond the
cemento-enamel junction is uncommon before 20 years of age, and the isolated recession observed in subjects around this age on facial surfaces with normal bone levels interproximally is generally not considered to be periodontal disease nor to be a result of this disease\textsuperscript{251}.

Epidemiological studies have shown that recession increases with age\textsuperscript{187, 227}. The most common aetiological factors associated with recession are malalignment and toothbrush trauma; other factors may include calculus, inflammation, occlusal trauma, frenum attachment, cervical restorations and partial dentures\textsuperscript{78, 243}.

Gingival recession may predispose to periodontal disease particularly in later life by creating food traps interproximally or inconsistent gingival margins which are missed in oral hygiene procedures. The plaque retentive qualities of the exposed cemental surface may be enhanced by its comparatively rough surface\textsuperscript{232}, and also possibly by the unphysiological crown contour created. However when recession is due to toothbrush trauma it seems likely that, at least in the earlier stages, the exposed cementum surface will be well cleaned.
MATERIALS AND METHODS

This study was conducted on 232 individuals of whom half were dental students and half dental patients. The students comprised 116 undergraduate students in the faculty of dentistry, University of Sydney. Of these, 54 were in the third year of their academic course, 49 in fourth year and 13 in fifth year. The patient group consisted of 116 individuals admitted to the United Dental Hospital, Sydney, for dental treatment.

The student and patient groups were carefully matched with each other in age and sex. All persons were between the ages of 18 and 22 years at the time of examination. The mean age of the student group was 20.8 years and that of the patient group 20.3 years. Each group included 10 females.

The students were examined in the Department of Periodontics, University of Sydney, and the patient group in the Examination Department of the United Dental Hospital. All individuals were examined between the months of November, 1967 and March, 1968 by the same examiner under conditions of good natural light with the aid of mouth mirrors, calibrated periodontal probes (Vetter pattern), explorers, gauze swabs, disclosing solution (2% Mercurochrome), cotton wool applicators and specially prepared charts.
METHOD OF CHARTING AND SCORING CRITERIA.

DMFT / S.

✓ sound tooth present, free of caries and restorations.

M no tooth present because extracted due to caries.

M no tooth present because extracted for reasons other than caries.

UE no tooth present because it has not erupted.

O no tooth present and space closed.

PE tooth only partially erupted so that either facial or lingual surface exposed is too small for plaque scores - less than 3 mm., or not erupted into occlusal plane.

X crown destroyed to such an extent that plaque scores are not possible on facial or lingual surfaces.

F sound tooth with restoration. Number of surfaces involved in restoration recorded.

D tooth with caries. Number of surfaces involved by caries and restorations, if any, recorded.

Only clinically obvious, and readily detectable carious lesions were recorded.

DFS Recorded separately for facial and lingual surfaces.

D tooth surface has carious lesion involving gingival one third of surface.
$D_1$ surface has carious lesion not involving gingival third of the surface.

$F$ surface has restoration involving gingival third of the surface.

$F_1$ surface has restoration not involving gingival third of the surface.

Prosthesis.

$D$ missing tooth replaced by removable partial denture.

$X$ tooth or its gingiva in contact with any portion of the partial denture apart from clasps.

$C$ tooth with clasp.

$B$ missing tooth replaced by fixed bridge.

$A$ abutments utilized to support fixed bridge.

$O$ teeth in contact with any part of an orthodontic appliance.

There were so few individuals in this prosthesis group that they were subsequently discarded from this study.

Malposition.

$\checkmark$ tooth malposed or malaligned in any readily detectable manner in relation to the dental arch of which it forms part.

$C$ tooth in crossbite.
Recession.

Recorded separately for facial and lingual surfaces.

√ tooth surface with clinically obvious recession of the gingival margin beyond the cemento-enamel function.

RPI the criteria of Russell's Periodontal Index were used to evaluate the health of the periodontium.

O negative; there is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissue.

I mild gingivitis; there is an overt area of inflammation in the free gingiva which does not circumscribe the tooth.

2 gingivitis; inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment.

6 gingivitis with pocket formation; the epithelial attachment has been broken and there is a pocket (not merely a deepened gingival crevice due to swelling in the free gingiva). There is no interference with normal masticatory function, the tooth is firm in its socket and has not drifted.
advanced destruction with loss of masticatory function. The tooth may be loose; may have drifted; may sound dull on percussion with a metallic instrument; may be depressible in its socket.

In cases of doubtful assessment, the lesser score was assigned.

Calculus,

was scored separately on facial and lingual surfaces in the manner of the Calculus Index of Greene and Vermillion.  

0  no calculus present.
1  supragingival calculus covering not more than one third of the exposed tooth surface.
2  supragingival calculus covering more than one third but not more than two thirds of the exposed tooth surface, or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth or both.
3  supragingival calculus covering more than two thirds of the exposed tooth surface, or a continuous heavy band of subgingival calculus around the cervical portion of the tooth or both.
Debris.

After painting the teeth with disclosing solution and instructing the patient to rinse his mouth with water, the amount of debris and dental plaque was scored separately on facial and lingual surfaces.

0 no plaque or stain present.

X no plaque but extrinsic brown stain present.

I plaque not extending completely around the cervical portion of the tooth surface.

2 plaque extending completely around the cervical portion of the tooth surface but covering not more than one third of the surface.

3 plaque extending completely around the tooth surface and covering more than one third of the surface.

Record was made of the date and time of the examination, the name, group category and sex of the individual. The place of birth of the subject and that of his parents was also noted.

Frequency of toothbrushing.

Subjects were asked the minimum number of times a day that they brushed their teeth and their replies were recorded
in the following manner:

1. toothbrushing less than once a day;
2. toothbrushing at least once a day but not always twice a day;
3. toothbrushing at least twice a day or more frequently.

Arch Relation.

Molars.

The relationship of the molar teeth was assessed according to the Angle classification.

1. neutrocclusion of the lower first molar in relation to the upper first molar.
2. distocclusion of the lower first molar in relation to the upper first molar.
3. mesiocclusion of the lower first molar in relation to the upper first molar.

Overbite.

1. normal relation of the upper and lower anterior teeth in a vertical plane when the posterior teeth are in centric occlusion.
2. over half of the facial surface of the lower anterior teeth covered by the upper teeth.
3. openbite - any space between the incisal edges of the
upper and lower anterior teeth when the posterior teeth are in centric occlusion.

Overjet.

1 normal relation of the upper and lower anterior teeth in a horizontal plane when the posterior teeth are in centric occlusion.

2 greater than 4 mm. between the facial surfaces of the upper and lower anteriors in a horizontal plane when the posterior teeth are in centric occlusion.

3 edge to edge or crossbite relationship of more than two anterior teeth in both the upper and lower arch.

Statistical Method.

In the statistical treatment of this data a maximum number of 28 teeth were considered for each individual. Third molars were included only if one or more teeth in the same quadrant were missing and if the third molar itself was fully erupted into the occlusal plane.

Russell's Periodontal Index was calculated for each individual by dividing the sum of the periodontal scores for the individual teeth by the number of teeth assessed. A Malalignment Index was calculated in a similar manner. As recession, calculus
and plaque were scored separately on facial and lingual surfaces, the indices for these conditions were determined by dividing the total score for all tooth-surfaces by the number of surfaces measured.

A strong clinical impression was gained during the examination of these subjects that malalignment was more common in the anterior teeth, and that restorations and carious lesions involving the gingiva were more prevalent in the posterior teeth. Therefore the Periodontal and Plaque Indices were calculated for each individual separately for his malaligned anterior teeth and for all his anterior teeth, and also for his posterior teeth with gingival restorations or caries and for all his posterior teeth.

Mean Plaque Index scores were calculated separately on the facial and lingual surfaces of the anterior, premolar and molar teeth in the upper and lower arches. Mean Periodontal Index scores were also calculated for each of these groups of teeth.

The population sample in this study was chosen from two sources - dental hospital patients and undergraduate dental students. Although the dental hospital patients included a small number of university students from faculties other than dentistry, they were mainly individuals from a low socio-economic and educational status in the particular community under study.
Many of these individuals were seeking treatment on an emergency basis and appeared to have little interest in their oral health. The dental students on the other hand appeared to be better motivated in this respect.

The main purpose of this study was to examine the patterns of plaque retention and periodontal disease in subjects with a high standard of oral hygiene and a low prevalence of periodontal disease, and to compare these with the patterns in subjects with a lower standard of oral hygiene and a higher prevalence of periodontal disease.

The dental students and the dental hospital patients were considered to broadly represent these two categories. In order to make the conclusions of this study more meaningful, all the subjects were classified into categories on the basis of their Plaque and Periodontal Indices. Values were chosen which divided the entire sample into groups of approximately equal numbers.

These categories comprised a low Plaque Index group (Plaque Index < 1.4), a high Plaque Index group (Plaque Index \( \geq 1.4 \)), a low Periodontal Index group (Periodontal Index < 1.0), and a high Periodontal Index group (Periodontal Index \( \geq 1.0 \)).
The various indices were then calculated separately for the dental students, the hospital patients and for each of the two Plaque and Periodontal Index categories. This data was subjected to correlative analysis to ascertain the relationships between these indices for the entire sample and for each of the above-mentioned groups.
RESULTS

1. Relationships between the Periodontal Index and the Plaque and Calculus Indices.

   Table 1. a presents the mean Periodontal, Plaque and Calculus Indices for all individuals in this study, and separately for the dental students, the hospital patients and for the two Plaque and Periodontal Index groups.

   The mean Periodontal Index for all individuals in this study was 1.10. The mean Periodontal, Plaque and Calculus Indices for the dental students were all significantly lower than those for the hospital patients (P < .001). These relationships held true for the two Plaque and Periodontal Index groups.

   Table 1. b shows the correlations observed between the Periodontal and Plaque Indices and between the Periodontal and Calculus Indices for the different groups.

   For all individuals in this study, there was a moderate correlation between the Periodontal and Plaque Indices (r = .55) and between the Periodontal and Calculus Indices (r = .55). The correlation between the Periodontal and Calculus Indices was weaker in the low Periodontal Index group (r = .20) than in the high Periodontal Index group (r = .44).
Table 1. a

Mean Periodontal, Calculus and Plaque Indices.

<table>
<thead>
<tr>
<th>population group</th>
<th>mean Perio. Index</th>
<th>mean Plaque Index</th>
<th>mean Calculus Index</th>
<th>number in group</th>
</tr>
</thead>
<tbody>
<tr>
<td>all individuals</td>
<td>1.10 (0.68)</td>
<td>1.37 (0.49)</td>
<td>0.17 (0.30)</td>
<td>232</td>
</tr>
<tr>
<td>dental students</td>
<td>0.81 (0.52)</td>
<td>1.10 (0.38)</td>
<td>0.06 (0.13)</td>
<td>116</td>
</tr>
<tr>
<td>hospital patients</td>
<td>1.39 (0.69)</td>
<td>1.64 (0.44)</td>
<td>0.27 (0.33)</td>
<td>116</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>0.73 (0.50)</td>
<td>0.95 (0.27)</td>
<td>0.07 (0.18)</td>
<td>107</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>1.33 (0.69)</td>
<td>1.73 (0.32)</td>
<td>0.24 (0.36)</td>
<td>125</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>0.58 (0.24)</td>
<td>1.12 (0.39)</td>
<td>0.05 (0.10)</td>
<td>119</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>1.65 (0.55)</td>
<td>1.63 (0.44)</td>
<td>0.29 (0.38)</td>
<td>113</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Table 1. b

Correlations between the Periodontal Index and the Plaque and Calculus Indices.

<table>
<thead>
<tr>
<th>population group</th>
<th>Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Perio. Index</td>
</tr>
<tr>
<td>all individuals</td>
<td>.55</td>
</tr>
<tr>
<td>dental students</td>
<td>.37</td>
</tr>
<tr>
<td>hospital patients</td>
<td>.44</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>.36</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>.38</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>.32</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>.25</td>
</tr>
</tbody>
</table>
Table 1. c presents the correlations observed between the Periodontal and Plaque Indices for anterior and for posterior teeth separately.

For all individuals in this study, there was a moderate correlation between these two Indices in the anterior teeth ($r = .58$); this correlation was slightly weaker in the posterior teeth ($r = .43$). The difference between the anterior and posterior teeth was most marked in the high Periodontal Index group, where no correlation was found between the Periodontal and Plaque Indices for posterior teeth ($r = .11$).

2. Relationships between the Periodontal and the Plaque Indices for posterior teeth with gingival restorations or carious lesions.

Table 2. a presents the mean number of posterior teeth with gingival restorations or carious lesions and the mean total number of posterior teeth for the different groups.

The dental students had a significantly greater mean number of posterior teeth with gingival restorations or carious lesions compared to the hospital patients, as did the low Plaque Index group compared to the high Plaque Index group ($P < .001$). The dental students also had significantly
Table 1.c
Correlations between the Periodontal and Plaque Indices for anterior and posterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>Correlations between Periodontal Index and Plaque Index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>anterior teeth</td>
</tr>
<tr>
<td>all individuals</td>
<td>.58</td>
</tr>
<tr>
<td>dental students</td>
<td>.36</td>
</tr>
<tr>
<td>hospital patients</td>
<td>.49</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>.28</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>.44</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>.40</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>.43</td>
</tr>
</tbody>
</table>
Table 2.a

Mean number of posterior teeth with gingival restorations or carious lesions and mean total number of posterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>mean number of posterior teeth with gingival restorations*</th>
<th>mean total number of posterior teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>all individuals</td>
<td>6.12 (4.46)**</td>
<td>14.50 (2.44)</td>
</tr>
<tr>
<td>dental students</td>
<td>3.19 (4.42)</td>
<td>15.43 (1.20)</td>
</tr>
<tr>
<td>hospital patients</td>
<td>4.04 (3.41)</td>
<td>13.58 (2.97)</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>7.36 (4.47)</td>
<td>14.73 (2.13)</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>5.05 (4.12)</td>
<td>14.31 (2.68)</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>6.60 (4.51)</td>
<td>14.79 (2.14)</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>5.60 (4.36)</td>
<td>14.20 (2.71)</td>
</tr>
</tbody>
</table>

* includes carious lesions involving the gingiva.

** standard deviation in brackets.
more posterior teeth than the hospital patients ($P < .001$).

In Table 2, b the mean Periodontal Index and the mean Plaque Index for posterior teeth with gingival restorations or carious lesions are compared with these Indices for all posterior teeth.

For all individuals, the mean Periodontal Index of posterior teeth with gingival restorations or carious lesions was higher than that of all posterior teeth (probably significant, $P < .05$). However the mean Plaque Index of posterior teeth with gingival restorations or carious lesions was lower than that of all posterior teeth ($P < .001$).

The mean Periodontal Index of posterior teeth with gingival restorations or carious lesions was also higher than that of all posterior teeth for the dental students (probably significant, $P < .05$) and for the high Periodontal Index group ($P < .001$).

Table 2, c presents three series of correlations for posterior teeth with gingival restorations or carious lesions.

For all individuals and for each of the groups considered, there was a moderate correlation between the Periodontal Index and the Plaque Index of posterior teeth with gingival restorations or carious lesions.

For all individuals, no correlation was found
Table 2.b

Mean Periodontal and Plaque Indices for posterior teeth with gingival restorations or carious lesions and for all posterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>mean Perio. Index</th>
<th>mean Plaque Index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>post. teeth</td>
<td>gingival restorations</td>
</tr>
<tr>
<td>all individuals</td>
<td>1.69 (1.36)**</td>
<td>1.48 (0.91)</td>
</tr>
<tr>
<td>dental students</td>
<td>1.48 (1.14)</td>
<td>1.21 (0.78)</td>
</tr>
<tr>
<td>hospital patients</td>
<td>1.89 (1.53)</td>
<td>1.75 (0.95)</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>1.36 (1.12)</td>
<td>1.16 (0.63)</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>1.97 (1.48)</td>
<td>1.75 (0.99)</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>0.98 (0.62)</td>
<td>0.89 (0.36)</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>2.42 (1.53)</td>
<td>2.09 (0.90)</td>
</tr>
</tbody>
</table>

* includes carious lesions involving the gingiva.
** standard deviation in brackets.
Table 2. c

Correlations for posterior teeth with gingival restorations or carious lesions.

<table>
<thead>
<tr>
<th>Population group</th>
<th>Correlations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Perio. Index of post. with gingival restorations*</td>
</tr>
<tr>
<td></td>
<td>Plaque Index of post. with gingival restorations*</td>
</tr>
<tr>
<td>all individuals</td>
<td>.52</td>
</tr>
<tr>
<td>dental students</td>
<td>.41</td>
</tr>
<tr>
<td>hospital patients</td>
<td>.55</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>.41</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>.51</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>.49</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>.44</td>
</tr>
</tbody>
</table>

* includes carious lesions involving the gingiva.
between the number of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth. However for the dental students a weak correlation was found between these two factors \( r = .30 \).

For all individuals, there was a moderate correlation between the Periodontal Index of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth \( r = .69 \). This correlation was stronger in the dental student, low Plaque Index, and low Periodontal Index groups.

3. Relationships between the Malalignment Index and the Periodontal and Plaque Indices for all teeth.

Table 3 shows that for all teeth no correlations were found between the Malalignment and the Periodontal Indices or between the Malalignment and the Plaque Indices for all individuals or in any of the groups considered.

4. Relationships between the Periodontal and the Plaque Indices for malaligned anterior teeth.

Table 4 presents the mean number of malaligned anterior teeth and the mean total number of anterior
Table 3

Correlations between the Malalignment Index and the Periodontal and Plaque Indices for all teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>Malalignment Index</th>
<th>Perio. Index</th>
<th>Malalignment Index</th>
<th>Perio. Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>all individuals</td>
<td>.08</td>
<td>.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dental students</td>
<td>-.05</td>
<td>.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hospital patients</td>
<td>.13</td>
<td>.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>-.06</td>
<td>-.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>.12</td>
<td>.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>-.01</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>.09</td>
<td>.15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 4. a

Mean number of malaligned anterior teeth and mean total number of anterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>mean number of malaligned anterior teeth</th>
<th>mean total number of anterior teeth</th>
</tr>
</thead>
<tbody>
<tr>
<td>all individuals</td>
<td>3.93 (2.63) *</td>
<td>11.69 (1.13)</td>
</tr>
<tr>
<td>dental students</td>
<td>4.22 (2.66)</td>
<td>12.00 (0.42)</td>
</tr>
<tr>
<td>hospital patients</td>
<td>3.63 (2.57)</td>
<td>11.39 (1.49)</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>4.00 (2.51)</td>
<td>11.80 (1.00)</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>3.37 (2.73)</td>
<td>11.60 (1.23)</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>3.97 (2.63)</td>
<td>11.86 (0.92)</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>3.88 (2.63)</td>
<td>11.52 (1.30)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
teeth for the different groups.

There were no significant differences between the mean number of malaligned anterior teeth for the different groups. The dental students had a significantly greater mean total number of anterior teeth compared to the hospital patients \( (P < .001) \).

In Table 4.b the mean Periodontal Index of malaligned anterior teeth is compared with that of all anterior teeth and the mean Plaque Index of malaligned anterior teeth is compared with that of all anterior teeth.

No significant differences were noted between either the mean Periodontal or Plaque Indices for malaligned anterior teeth and the mean Periodontal or Plaque Indices for all anterior teeth.

Table 4.c presents four series of correlations for malaligned anterior teeth.

For all individuals, no correlation was found between the number of malaligned anterior teeth and the Periodontal Index of all anterior teeth. However there was a weak correlation between these two factors for the dental students \( (r = .27) \), low Plaque Index group \( (r = .22) \), and for the low Periodontal Index group \( (r = .34) \).
Table 4.b

Mean Periodontal and Plaque Indices for malaligned anterior teeth and for all anterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>mean Perio. Index</th>
<th>mean Plaque Index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>malaligned anteriors</td>
<td>all anteriors</td>
</tr>
<tr>
<td>all individuals</td>
<td>0.69 (0.79)*</td>
<td>0.66 (0.64)</td>
</tr>
<tr>
<td>dental students</td>
<td>0.37 (0.54)</td>
<td>0.34 (0.45)</td>
</tr>
<tr>
<td>hospital patients</td>
<td>1.01 (0.88)</td>
<td>0.98 (0.65)</td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>0.39 (0.53)</td>
<td>0.33 (0.42)</td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>0.94 (0.89)</td>
<td>0.94 (0.67)</td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>0.29 (0.38)</td>
<td>0.25 (0.26)</td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>1.10 (0.91)</td>
<td>1.09 (0.64)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Table 4.c

Correlations for malaligned anterior teeth.

<table>
<thead>
<tr>
<th>population group</th>
<th>correlations</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. mal-aligned anteriors</td>
<td>no. mal-aligned anteriors</td>
<td>Perio.Index malaligned anteriors</td>
<td>Plaque Index malaligned anteriors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Perio.Index of all anteriors</td>
<td>Plaque Index of all anteriors</td>
<td>Perio.Index of all anteriors</td>
<td>Plaque Index of all anteriors</td>
<td></td>
</tr>
<tr>
<td>all individuals</td>
<td>.08</td>
<td>-.04</td>
<td>.82</td>
<td>.59</td>
<td></td>
</tr>
<tr>
<td>dental students</td>
<td>.27</td>
<td>.09</td>
<td>.89</td>
<td>.59</td>
<td></td>
</tr>
<tr>
<td>hospital patients</td>
<td>.08</td>
<td>-.05</td>
<td>.74</td>
<td>.50</td>
<td></td>
</tr>
<tr>
<td>low Plaque Index group</td>
<td>.22</td>
<td>.05</td>
<td>.83</td>
<td>.70</td>
<td></td>
</tr>
<tr>
<td>high Plaque Index group</td>
<td>.04</td>
<td>-.08</td>
<td>.78</td>
<td>.35</td>
<td></td>
</tr>
<tr>
<td>low Perio. Index group</td>
<td>.34</td>
<td>-.03</td>
<td>.84</td>
<td>.59</td>
<td></td>
</tr>
<tr>
<td>high Perio. Index group</td>
<td>.03</td>
<td>-.04</td>
<td>.73</td>
<td>.49</td>
<td></td>
</tr>
</tbody>
</table>
No correlations were observed between the number of malaligned anterior teeth and the Plaque Index of all anterior teeth.

There was a strong correlation between the Periodontal Index of malaligned anterior teeth and the Periodontal Index of all anterior teeth in each of the groups considered.

There was a moderate correlation between the Plaque Index of malaligned anterior teeth and the Plaque Index of all anterior teeth for all individuals and for each of the groups with the exception of the two Plaque Index groups. The correlation between these two factors was much stronger in the low Plaque Index group \( r = .70 \) than in the high Plaque Index group \( r = .35 \).  

5. Distribution of teeth with gingival recession.

The distribution of gingival recession on the different teeth for individuals in the low and the high Plaque Index groups separately is shown in Table 5.

Individuals in the low Plaque Index group had over twice the number of teeth with recession compared to individuals in the high Plaque Index group.

The tooth most commonly affected by recession
Table 5

Distribution of teeth with gingival recession for individuals in the low and the high Plaque Index groups.

<table>
<thead>
<tr>
<th>tooth</th>
<th>no. of teeth with recession</th>
<th>total no. of teeth with recession</th>
<th>percentage distribution of teeth with recession</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>low Plaque Index group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U2</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U3</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U4</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U5</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U6</td>
<td>25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>U7</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L1</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L2</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L3</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L4</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L6</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>L7</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>high Plaque Index group</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>169</td>
<td>70</td>
<td>239</td>
</tr>
<tr>
<td>total number of teeth</td>
<td>2808</td>
<td>3239</td>
<td>100%</td>
</tr>
</tbody>
</table>
was the upper first premolar. This tooth was followed by the upper first molar and then the upper second premolar.

6. Comparison of plaque scores on tooth surfaces with recession against plaque scores on all similar tooth surfaces.

In Table 6 the mean plaque scores on tooth surfaces with gingival recession are compared with the mean plaque scores on all similar tooth surfaces.

Of the 239 surfaces with recession, all but 17 were facial surfaces. Of the 17 lingual surfaces with recession, 11 were in the lower anterior teeth in the low Plaque Index group. Therefore in Table 6 the plaque scores of tooth surfaces with recession are compared with facial surface plaque scores in the same groups of teeth in individuals of the same Plaque Index group, except for the lower anteriors in the low Plaque Index group. In this case the plaque score of tooth surfaces with recession is compared with the mean of the facial and lingual plaque scores of similar teeth.

The mean plaque scores of tooth surfaces with recession did not differ significantly from those of all similar tooth surfaces.
Table 6

Mean plaque scores on tooth surfaces with recession and on all similar tooth surfaces.

<table>
<thead>
<tr>
<th>teeth</th>
<th>mean plaque score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>low Plaque Index group</td>
</tr>
<tr>
<td></td>
<td>tooth surfaces with recession</td>
</tr>
<tr>
<td>upper anteriors</td>
<td>0.56</td>
</tr>
<tr>
<td>upper premolars</td>
<td>0.95</td>
</tr>
<tr>
<td>upper molars</td>
<td>0.90</td>
</tr>
<tr>
<td>lower anteriors</td>
<td>1.00</td>
</tr>
<tr>
<td>lower premolars</td>
<td>0.85</td>
</tr>
<tr>
<td>lower molars</td>
<td>0.70</td>
</tr>
</tbody>
</table>

* these are facial scores only.
** this score is mean of facial and lingual scores.
7. Relationship of the frequency of toothbrushing to the Recession, Periodontal, Plaque and Calculus Indices.

Table 7 presents the mean Recession, Periodontal, Plaque and Calculus Indices for individuals according to their reported frequency of toothbrushing.

The mean Recession Index for individuals brushing at least once daily was higher than for those brushing less than once daily (probably significant, \( P < .05 \)). There was no significant difference between the mean Recession Index for individuals brushing at least once daily and for those brushing at least twice daily (\( P > .05 \)). The mean Recession Index for individuals brushing more than twice daily was significantly higher than for those brushing less than once daily (\( P < .001 \)).

Both the mean Periodontal Index and the mean Plaque Index were significantly lower for individuals brushing at least once daily compared to those brushing less than once daily (\( P < .01 \)). The mean Periodontal and Plaque Indices were also significantly lower in individuals brushing at least twice daily compared to those brushing at least once daily (\( P < .001 \)).

There was no significant difference between the mean Calculus Index for individuals brushing at least once daily compared to those brushing less than once daily (\( P > .05 \)).
Table 7

Mean Recession, Periodontal, Plaque and Calculus Indices according to toothbrushing frequency.

<table>
<thead>
<tr>
<th>brushing frequency per day</th>
<th>$\leq 1$</th>
<th>$\geq 1$</th>
<th>$\geq 2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>number of individuals</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>57</td>
<td>89</td>
<td>86</td>
</tr>
<tr>
<td>Recession Index</td>
<td>0.29 (0.95)*</td>
<td>0.88 (2.24)</td>
<td>1.66 (3.39)</td>
</tr>
<tr>
<td>Perio. Index</td>
<td>1.46 (0.54)</td>
<td>1.19 (0.45)</td>
<td>0.83 (0.51)</td>
</tr>
<tr>
<td>Plaque Index</td>
<td>1.77 (0.39)</td>
<td>1.37 (0.40)</td>
<td>1.07 (0.35)</td>
</tr>
<tr>
<td>Calculus Index</td>
<td>0.29 (0.32)</td>
<td>0.19 (0.35)</td>
<td>0.06 (0.14)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Thus individuals who reported that they brushed their teeth more frequently had more gingival recession but less periodontal disease, plaque and calculus. The most significant differences were found for periodontal disease and plaque.
8. Distribution of plaque.

a. Distribution of plaque for all individuals.

Table 8. a and Figure 1 present the distribution of mean plaque scores on the different groups of teeth for all individuals in this study.

No significant differences were found between the mean plaque scores of the anteriors and premolars in the upper or the lower arches. The upper and lower premolars had significantly lesser scores than the upper and lower molars respectively ($P < .01$).

The mean plaque scores of each group of lower teeth were more than those of the corresponding upper teeth (probably significant, $P < .05$).

b. Distribution of plaque on facial and lingual surfaces for all individuals.

Table 8. b and Figure 2 show the distribution of mean plaque scores separately on the facial and lingual surfaces of the different teeth for all individuals in this study.

On facial surfaces, there were no significant differences between the mean plaque scores of the anteriors and the premolars in the upper or lower arches. The mean plaque scores on the facial surfaces of the upper and lower premolars
Table 8. a.

Mean plaque scores on the different groups of teeth for all individuals.

<table>
<thead>
<tr>
<th>teeth</th>
<th>mean plaque score</th>
</tr>
</thead>
<tbody>
<tr>
<td>upper anteriors</td>
<td>2.05 (1.22)*</td>
</tr>
<tr>
<td>premolars</td>
<td>2.26 (1.33)</td>
</tr>
<tr>
<td>molars</td>
<td>3.05 (1.43)</td>
</tr>
<tr>
<td>lower anteriors</td>
<td>2.77 (1.52)</td>
</tr>
<tr>
<td>premolars</td>
<td>3.02 (1.30)</td>
</tr>
<tr>
<td>molars</td>
<td>3.37 (1.34)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Fig. 1

MEAN PLAQUE SCORES – ALL INDIVIDUALS

antennors

upper

premolars

molars

antennors

lower

premolars

molars

mean plaque score
Table 8.b

Mean plaque scores on facial and lingual surfaces of the different groups of teeth for all individuals.

<table>
<thead>
<tr>
<th>teeth</th>
<th>surface</th>
<th>mean plaque score</th>
</tr>
</thead>
<tbody>
<tr>
<td>upper anteriors</td>
<td>facial</td>
<td>1.23 (0.72)*</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>0.82 (0.52)</td>
</tr>
<tr>
<td>premolars</td>
<td>facial</td>
<td>1.27 (0.81)</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>0.99 (0.53)</td>
</tr>
<tr>
<td>molars</td>
<td>facial</td>
<td>1.83 (0.85)</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>1.22 (0.58)</td>
</tr>
<tr>
<td>lower anteriors</td>
<td>facial</td>
<td>1.31 (0.77)</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>1.46 (0.75)</td>
</tr>
<tr>
<td>premolars</td>
<td>facial</td>
<td>1.22 (0.68)</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>1.80 (0.62)</td>
</tr>
<tr>
<td>molars</td>
<td>facial</td>
<td>1.43 (0.68)</td>
</tr>
<tr>
<td></td>
<td>lingual</td>
<td>1.94 (0.65)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Fig. 2
MEAN PLAQUE SCORES ON FACIAL AND LINGUAL SURFACES — ALL INDIVIDUALS
were significantly less than those on the facial surfaces of the upper and lower molars respectively ($P < .01$). The mean facial score of the upper molars was significantly more than the mean facial score of the lower molars and of the facial scores on the other teeth ($P < .001$).

On lingual surfaces, the mean plaque score of the anteriors was less than that of the premolars and the mean plaque score of the premolars was less than that of the molars in both the upper and the lower arches (probably significant, $P < .05$).

The mean plaque scores on the lingual surfaces of the lower teeth were significantly more than those on the lingual surfaces of the corresponding upper teeth ($P < .001$).

In the upper arch, mean facial scores were significantly more than mean lingual scores on the anteriors, premolars and molars ($P < .001$). In the lower arch, mean facial scores were less than mean lingual scores for the anteriors (probably significant, $P < .05$), premolars and molars ($P < .001$).

The highest mean plaque scores were found on the lingual surfaces of the lower premolars and molars and the facial surfaces of the upper molars; the lingual surfaces of the
upper anteriors had the lowest scores followed by the lingual surfaces of the upper premolars ($P < .01$).

c. Distribution of plaque on facial and lingual surfaces for individuals in the low and high Plaque Index groups separately.

In Table 8, c and Figure 3 the distribution of mean plaque scores on the facial and lingual surfaces of the different teeth for individuals in the low Plaque Index group is compared with the distribution for individuals in the high Plaque Index group.

In the low Plaque Index group, the distribution of plaque between the anteriors, premolars and molars in each arch was essentially the same as that for all individuals.

On facial surfaces in the low Plaque Index group, there were no significant differences between the mean plaque scores on the anteriors and the premolars in the upper or lower arches. The mean plaque scores on the facial surfaces of the upper and lower premolars were significantly less than those on the facial surfaces of the upper and lower molars respectively ($P < .01$). The mean facial score of the upper molars was significantly more than the mean facial score of the lower molars and of the facial scores of the other teeth ($P < .01$).
Table 8.c

Mean plaque scores on facial and lingual surfaces of the different groups of teeth for individuals in the low and the high Plaque Index groups.

<table>
<thead>
<tr>
<th>teeth</th>
<th>surface</th>
<th>mean plaque score</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>low Plaque Index group</td>
<td>high Plaque Index group</td>
<td></td>
</tr>
<tr>
<td>upper anteriors</td>
<td>facial</td>
<td>0.74 (0.46)*</td>
<td>1.64 (0.64)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.49 (0.45)</td>
<td>1.09 (0.41)</td>
<td></td>
</tr>
<tr>
<td>premolars</td>
<td>facial</td>
<td>0.76 (0.47)</td>
<td>1.71 (0.77)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.75 (0.54)</td>
<td>1.20 (0.41)</td>
<td></td>
</tr>
<tr>
<td>molars</td>
<td>facial</td>
<td>1.27 (0.67)</td>
<td>2.30 (0.68)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.96 (0.52)</td>
<td>1.45 (0.54)</td>
<td></td>
</tr>
<tr>
<td>lower anteriors</td>
<td>facial</td>
<td>0.82 (0.51)</td>
<td>1.73 (0.69)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.99 (0.59)</td>
<td>1.86 (0.63)</td>
<td></td>
</tr>
<tr>
<td>premolars</td>
<td>facial</td>
<td>0.79 (0.45)</td>
<td>1.58 (0.64)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.48 (0.63)</td>
<td>2.07 (0.45)</td>
<td></td>
</tr>
<tr>
<td>molars</td>
<td>facial</td>
<td>1.06 (0.47)</td>
<td>1.75 (0.68)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.73 (0.58)</td>
<td>2.13 (0.65)</td>
<td></td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Fig. 3

MEAN PLAQUE SCORES
FACIAL AND LINGUAL SURFACES
LOW AND HIGH PLAQUE INDEX GROUPS
On lingual surfaces in the low Plaque Index group, mean plaque scores of the anteriors were significantly less than those of the premolars, and the mean plaque scores of the premolars less than those of the molars in both upper and lower arches ($P < .01$). Also the mean plaque scores on the lingual surfaces of the lower teeth were significantly more than those on the lingual surfaces of the corresponding upper teeth ($P < .001$).

In the high Plaque Index group the distribution of plaque between the anteriors, premolars and molars differed from that for all individuals and for the low Plaque Index group.

On facial surfaces in the high Plaque Index group, the only significant difference between the mean plaque scores of the various upper and lower teeth was that the upper molar score was above that of the other teeth ($P < .01$).

On lingual surfaces in the high Plaque Index group, the only significant differences in mean plaque scores were that the upper molar scores were above that of the other upper teeth and the lower anterior score was below that of the other lower teeth ($P < .01$).

Thus the rise in mean plaque scores from the anteriors to the posteriors was more marked in the low Plaque Index group than in the high Plaque Index group.
Comparison of the mean plaque scores on the facial and lingual surfaces of the same groups of teeth also revealed differences between the low and the high Plaque Index groups.

In the low Plaque Index group, upper facial scores were significantly higher than upper lingual only on the anterior and molar teeth (P < .001); in the lower arch facial scores were less than lingual on the anteriors (probably significant, P < .05); premolars and molars (P < .001).

In the high Plaque Index group, upper facial scores were significantly higher than upper lingual scores on the anteriors, premolars and molars (P < .001); in the lower arch facial scores were less than lingual only on the premolars and molars (P < .001).

In general in the upper arch, the differences between facial and lingual scores were more marked in the high Plaque Index group. While in the lower arch, the differences between facial and lingual scores were more marked in the low Plaque Index group. In both arches, the differences in plaque scores between the low and the high Plaque Index groups were more marked on facial than on lingual surfaces.

a. Distribution of periodontal disease for all individuals.

Table 9. a and Figure 4 present the distribution of mean periodontal scores for the different groups of teeth for all individuals in this study.

In both the upper and lower arches, the mean periodontal score of the anteriors was significantly less than that of the premolars and the mean periodontal score of the premolars was less than that of the molars ($P < .001$).

In comparing the mean periodontal scores of the upper teeth with those of the corresponding lower teeth, the only difference was that the upper premolar scores were higher than the lower premolar scores (probably significant, $P < .05$).

The upper and lower molars had the highest mean periodontal scores, followed by the upper and then the lower premolars; the upper and lower anteriors had the lowest scores ($P < .01$).

b. Distribution of periodontal disease for individuals in the low and the high Periodontal Index groups separately.

In Table 9. b and Figure 5 the distribution of
Table 9. a

Mean periodontal scores for the different groups of teeth for all individuals.

<table>
<thead>
<tr>
<th>teeth</th>
<th>mean periodontal score</th>
</tr>
</thead>
<tbody>
<tr>
<td>upper anteriors</td>
<td>0.60 (0.71)*</td>
</tr>
<tr>
<td>premolars</td>
<td>1.32 (1.16)</td>
</tr>
<tr>
<td>molars</td>
<td>1.75 (1.36)</td>
</tr>
<tr>
<td>lower anteriors</td>
<td>0.67 (0.73)</td>
</tr>
<tr>
<td>premolars</td>
<td>1.10 (0.82)</td>
</tr>
<tr>
<td>molars</td>
<td>1.64 (1.25)</td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Fig. 4
MEAN PERIODONTAL SCORES
ALL INDIVIDUALS

Mean periodontal score
Table 9.b

Mean periodontal scores for the different groups of teeth for individuals in the low and the high Periodontal Index groups.

<table>
<thead>
<tr>
<th>teeth</th>
<th>mean periodontal score</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>low Perio. Index group</td>
<td>high Perio. Index group</td>
<td></td>
</tr>
<tr>
<td>upper anteriors</td>
<td>0.24 (0.32)*</td>
<td>0.99  (0.80)</td>
<td></td>
</tr>
<tr>
<td>premolars</td>
<td>0.69 (0.50)</td>
<td>1.97  (1.28)</td>
<td></td>
</tr>
<tr>
<td>molars</td>
<td>1.03 (0.75)</td>
<td>2.51  (1.44)</td>
<td></td>
</tr>
<tr>
<td>lower anteriors</td>
<td>0.23 (0.34)</td>
<td>1.14  (0.75)</td>
<td></td>
</tr>
<tr>
<td>premolars</td>
<td>0.72 (0.59)</td>
<td>1.49  (0.85)</td>
<td></td>
</tr>
<tr>
<td>molars</td>
<td>1.10 (0.61)</td>
<td>2.20  (1.48)</td>
<td></td>
</tr>
</tbody>
</table>

* standard deviation in brackets.
Fig. 5
MEAN PERIODONTAL SCORES
INDIVIDUALS IN LOW AND HIGH PERIODONTAL INDEX GROUPS

low P.I.group

high P.I.group

antelors

UPPER

premolars

molars

LOWER

antelors

premolars

molars

mean periodontal score
mean periodontal scores for individuals in the low Periodontal Index group is compared with that for individuals in the high Periodontal Index group.

In both low and high Periodontal Index groups and in both upper and lower arches, the mean periodontal scores of the anteriors were significantly less than those of the premolars and the mean periodontal scores of the premolars were less than those of the molars ($P < .01$).

In the low Periodontal Index group, there were no significant differences between the mean periodontal scores of the upper teeth and those of the corresponding lower teeth. In the high Periodontal Index group, the mean periodontal score of the upper premolars was significantly above that of the lower premolars ($P < .001$).

10. Prevalence of gingivitis on selected teeth according to their plaque scores.

Table 10 and Figure 6 present the percentage of selected teeth that were affected by gingivitis according to the plaque scores on their facial and lingual surfaces.

Only teeth were selected which did not have any condition which could be considered to predispose to gingivitis other than by the coronal plaque retained on them. The teeth
Table 10

Percentage of selected teeth with gingivitis according to their plaque scores.

<table>
<thead>
<tr>
<th>Plaque scores facial/lingual or lingual/facial</th>
<th>Russell periodontal score</th>
<th>Percentage of teeth affected by gingivitis *</th>
</tr>
</thead>
<tbody>
<tr>
<td>0/0</td>
<td>83  4  0</td>
<td>0</td>
</tr>
<tr>
<td>0/1</td>
<td>158 24 1</td>
<td>.14</td>
</tr>
<tr>
<td>1/1</td>
<td>161 105 6</td>
<td>42</td>
</tr>
<tr>
<td>0/2</td>
<td>18 24 0</td>
<td>57</td>
</tr>
<tr>
<td>1/2</td>
<td>116 197 17</td>
<td>67</td>
</tr>
<tr>
<td>2/2</td>
<td>37 102 19</td>
<td>79</td>
</tr>
<tr>
<td>0/3</td>
<td>0 4 0</td>
<td>100</td>
</tr>
<tr>
<td>1/3</td>
<td>23 49 16</td>
<td>78</td>
</tr>
<tr>
<td>2/3</td>
<td>2 57 18</td>
<td>96</td>
</tr>
<tr>
<td>3/3</td>
<td>0 7 17</td>
<td>100</td>
</tr>
</tbody>
</table>

* teeth with Russell periodontal score of 2 are counted twice.
Fig. 6

PERCENTAGE OF SELECTED TEETH WITH GINGIVITIS ACCORDING TO PLAQUE SCORES
excluded comprised teeth with calculus, restorations or carious lesions involving the gingiva, malaligned teeth, teeth in cross-bite or abnormal overbite or overjet relation, teeth adjacent to diastemas and any teeth adjacent to teeth with any of these conditions. Teeth with periodontitis were also excluded.

It will be seen from Figure 6 that the percentage of teeth affected by gingivitis rose in a linear fashion as the plaque score on the facial and/or lingual surface increased by one unit of the Plaque Index.
DISCUSSION

1. Relationships between the Periodontal Index and the Plaque and Calculus Indices.

The mean Periodontal Index of 1.10 found for all individuals in this study is high compared to that found in similar samples of age and sex in Norway by Brandtzaeg and Jamison (0.80), and in the United States of America by Russell (0.62) \(^{203}\). This may be due to examiner variation. However it is noteworthy that Lilienthal and his co-workers in a similar population sample in Australia observed the same Periodontal Index (1.10)\(^{144}\).

The entire sample was divided into two Periodontal and Plaque Index groups in order to assess the differences in the distribution of periodontal disease and plaque at different levels of prevalence of these two conditions. In examining the relationships between periodontal disease, plaque, calculus, and other factors predisposing to periodontal disease, it was also felt desirable to compare these relationships between the two Periodontal and Plaque Index groups.

As was anticipated, the mean Periodontal, Plaque and Calculus Indices of the dental students were all significantly below those of the hospital patients. These relation-
ships held true for the two Plaque and Periodontal Index groups.

The correlations between the Periodontal and the Plaque Indices ($r = .55$) and between the Periodontal and the Calculus Indices ($r = .55$) for all individuals in this study were similar to those found by Brandtzaeg and Jamison ($r = .58$, $r = .48$ respectively) in a similar sample. These findings indicate that both plaque and calculus were equally associated with periodontal disease. This is consistent with the results of Lilienthal and his co-workers, who found that up to 20 - 24 years of age both debris and calculus strongly influenced the Periodontal Index.

The weaker correlation between periodontal disease and calculus in the low Periodontal Index group ($r = .20$) compared to that in the high Periodontal Index group ($r = .44$) no doubt reflected the little calculus found in the former group.

The correlation between the Periodontal and the Plaque Indices for all individuals in this study was somewhat weaker in the posterior teeth ($r = .43$) than in the anterior teeth ($r = .58$). This finding indicates that, compared to the anterior teeth, periodontal disease in posterior teeth tended to be more frequently associated with factors other than plaque.

Only in the high Periodontal Index group was no
correlation found between the Periodontal and Plaque Indices for posterior teeth ($r = .11$). Russell's Periodontal Index is weighted in favour of pocket formation. This suggests that the factors other than plaque, which were associated with periodontal disease in posterior teeth, tended to be associated with periodontitis.

2. Restorations and carious lesions involving the gingiva.

For all individuals in this study, the mean Periodontal Index of posterior teeth with gingival restorations or carious lesions was higher than that of all posterior teeth (probably significant, $P < .05$). This relationship only held true for the dental students and the high Periodontal Index group.

The most significant difference between the Periodontal Index of posterior teeth with gingival restorations or carious lesions and that of all posterior teeth was in the high Periodontal Index group ($P < .001$). This may indicate that gingival restorations and carious lesions in posterior teeth tended to be associated with periodontitis. However even in this group there was only a small difference between the Periodontal Index of posterior teeth with gingival restorations or carious lesions (2.42) and that of all posterior teeth (2.09): Only in the student group was any correlation
observed between the number of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth \( r = .30 \). This finding no doubt reflects the greater mean number of posterior teeth with gingival restorations or carious lesions in the student group compared to the other groups.

For all individuals, there was a moderate correlation between the Periodontal Index of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth \( r = .69 \). This correlation was stronger in the student, low Plaque Index and low Periodontal Index groups.

Thus the relationship between gingival restorations and carious lesions and periodontal disease in posterior teeth was most clearly observed in the groups where the values for plaque and calculus were lowest.

Wright found that 57 percent of his sample had restorations with overhanging margins and 43 percent had carious lesions involving the gingiva. Of these restorations and carious lesions, 85 percent were associated with gingival inflammation. These factors were also more frequently associated with gingival inflammation than debris; calculus
was the factor most frequently associated with periodontitis. The sample investigated by Wright included older subjects than the present study, which would implicate calculus to a greater degree; also he did not measure plaque. Nevertheless his study does underline the significance of poor restorations and carious lesions involving the gingiva in the aetiology of periodontal disease.

In the present study no attempt was made to evaluate the quality of the restorations. The relationship between restorations and carious lesions involving the gingiva and periodontal disease in posterior teeth was most clearly observed when other factors, plaque and calculus, were minimal.

3. Malalignment.

No correlations were observed between the Malalignment and the Periodontal Indices for all teeth or between the Malalignment and the Plaque Indices for all teeth.

No significant differences were found between either the mean Periodontal or the mean Plaque Index for mal-aligned anterior teeth compared to the mean Periodontal or Plaque Index for all anterior teeth.

One difficulty in comparing the periodontal or
plaque scores of malaligned teeth with those of correctly aligned teeth in the same arch is that, if an unphysiological situation exists around a malaligned tooth, it is also created around an adjacent correctly aligned tooth.

Another problem is the multifactoral nature of periodontal disease. Neither Beagrie and James, nor Geiger, nor Gould and Picton were able to relate malalignment to periodontal disease \(^{19, 64, 79}\). However Poulton and Aaronson in a young adult sample found a weak correlation between lower anterior crowding and periodontal disease \((r = .24)\) \(^{189}\); the relatively low mean Periodontal Index of their sample \((0.48)\) may have been responsible for this positive finding. For Hellgren, McCombie and Stothard, and Trott and his co-workers were able to relate crowding to gingivitis \(^{94, 155, 242}\).

In the present study it was only possible to find any correlation between the number of malaligned anterior teeth and the Periodontal Index of all anterior teeth in the student group \((r = .27)\), the low Plaque Index group \((r = .22)\), and the low Periodontal Index group \((r = .34)\). Thus a relationship between malalignment and periodontal disease in anterior teeth was best observed when periodontal disease was low.

The influence of malalignment on the retention of plaque (or debris) does not appear to have been investigated
previously. The weak correlation between the Plaque Index of malaligned anterior teeth and the Plaque Index of all anterior teeth in the high Plaque Index group \( r = .35 \) contrasted with the stronger correlation between these two factors in the low Plaque Index group \( r = .70 \). This finding is interpreted as indicating that in subjects with better oral hygiene, malaligned anterior teeth tended to have similar plaque scores as the remaining anterior teeth. While in subjects with poorer oral hygiene, malaligned anterior teeth tended to have plaque scores either above or below those of the remaining anterior teeth.

Malalignment, of course, may make some surfaces of the teeth more accessible to oral hygiene and others less so. These findings suggest that morphological factors may become less important in determining the pattern of plaque retention as oral hygiene improves. This is probably because efficient toothbrushing is able to overcome most tooth contours predisposing to the retention of plaque.


Individuals in the low Plaque Index group had over twice the number of teeth with gingival recession than those in the high Plaque Index group.

Increase in the reported frequency of toothbrushing
was accompanied by an increase in the prevalence of gingival recession.

Gorman and Kitchin also found that individuals with good oral hygiene had more gingival recession than those with poor oral hygiene \(^78, 122\).

In the present study, the most commonly affected surfaces were the facial surfaces of the upper quadrant from first premolar to first molar. This finding agrees with previous studies of similar age groups by Gorman and O'Leary \(^78, 182\).

O'Leary observed that the mean plaque scores in the segments of his subjects' arches that were associated with recession were lower than the mean plaque scores of the same segments in subjects without recession \(^182\). However, it would appear that this finding merely reflected the different oral hygiene levels in these subjects.

In the present study, no differences were found between the mean plaque scores of tooth surfaces with recession and those of all similar tooth surfaces. Generally, the findings in the present study implicate toothbrush action as a major cause of gingival recession in this age group.

5. Frequency of toothbrushing.

Brandtzaeg and Jamison, and Sumnicht found that
the frequency of toothbrushing, as reported by subjects, was not necessarily related to the prevalence of periodontal disease. However, Stanmeyer, and Greene and Vermillion observed a close relation between these two factors. Stanmeyer found a significant decrease in gingivitis scores from subjects brushing less than once daily to those brushing at least once daily, and also between the latter subjects and those brushing at least twice daily; the decrease in gingivitis scores in subjects brushing still more frequently was very small. Greene and Vermillion found that the decrease in the Periodontal Index from subjects brushing less than once daily to those brushing at least once daily was more significant than the decrease from the latter subjects to those brushing at least twice daily.

In the present study it was found that each increase in the reported frequency of toothbrushing was accompanied by a significant decrease in the Periodontal Index and in the Plaque Index.

Greene and Vermillion also found significant decreases in the Debris Index with each increase in the brushing frequency. The Calculus Index was decreased in the more
frequent brushing groups in the present study, but less significantly than the Periodontal and Plaque Indices. This is in accordance with the findings of Brandtzaeg and Jamison\textsuperscript{30}.

Thus it appears that daily toothbrushing significantly reduces periodontal disease and plaque, and that twice daily toothbrushing reduces these conditions even further. However this conclusion is based upon unverified reports of toothbrushing frequency from mean data of epidemiological studies. The different findings of Brantzaeg and Jamison\textsuperscript{30} and Sumnicht\textsuperscript{235} indicate that these observations are not valid for all individuals. It is likely that it is the efficiency rather than the frequency of toothbrushing that is most significant in removing plaque and reducing periodontal disease.
6. Distribution of plaque.

For all individuals in this study, on facial surfaces there were no significant differences in mean plaque scores between the anterior and premolar teeth in the upper or the lower arch. Mean plaque scores on the facial surfaces of the molars were higher than those of the premolars in both upper and lower arches.

On lingual surfaces, mean plaque scores rose from the anteriors to the premolars and again to the molars in both upper and lower arches.

In the upper arch on each group of teeth, facial scores were higher than lingual. In the lower arch, lingual scores were higher than facial.

When facial and lingual scores were combined, the lower teeth had higher mean plaque scores than the corresponding upper teeth.

Lilienthal and his co-workers, in individuals 20 - 24 years of age, found that debris scores were higher on anterior than on posterior teeth and that debris scores on lower teeth were higher than on upper teeth. These investigators and also Lovdal and his co-workers observed that in general facial surfaces retained less debris than lingual surfaces\(^{144, 152}\).
In the present study, the highest mean plaque scores were found on the lingual surfaces of the lower molars and premolars and the facial surfaces of the upper molars. The lowest scores were found on the lingual surfaces of the upper anteriors, followed by the lingual surfaces of the upper premolars.

Lightner and his co-workers, in subjects 17–21 years of age, found that the largest accumulations of plaque occurred in descending order on the facial surfaces of the upper molars, the facial and lingual surfaces of the lower incisors and the lingual surfaces of the lower molars 143.

Thus the distribution of plaque for all individuals in the present study was similar to that found by other investigators with one exception. The lower anteriors in the present study had relatively lower mean plaque scores when compared with the findings of Lightner and his co-workers. This difference will be discussed later.

After estimating the distribution of plaque on the facial and lingual surfaces of the teeth for all individuals, the distribution of plaque was examined separately for individuals falling into two arbitrary categories - the low and the high Plaque Index groups. This was done in order to assess differences in the pattern of plaque retention at different levels of oral hygiene.
Loe and his co-workers, measuring the thickness of plaque rather than its coronal extension, observed the distribution of plaque at the end of a period (10 - 21 days) during which their subjects were requested not to clean their teeth\textsuperscript{151}. Bay and his co-workers, testing the efficiency of various types of toothbrushes, measured the amount of plaque remaining on the various surfaces of the teeth of dental students after they had brushed their teeth until they felt absolutely clean. The average brushing time was seven minutes\textsuperscript{18}.

Thus the findings of Loe and his co-workers and Bay and his co-workers may be taken as representing respectively the natural pattern of plaque retention and this pattern as affected by painstaking toothbrushing.

Loe and his co-workers did not observe any significant differences between the mean plaque scores of the anterior and the posterior teeth. However Bay and his co-workers found that plaque scores increased progressively from the anteriors through the premolars to the molars.

In the present study, the rise in plaque scores from the anteriors to the posteriors was more marked in the low Plaque Index group than in the high Plaque Index group.

Loe and his co-workers observed that facial
surfaces had higher plaque scores than lingual surfaces; generally, upper facial surfaces had the highest scores followed by lower facial and then lower lingual surfaces. However Bay and his co-workers found that, except for the upper premolars and lower anteriors, lingual scores were higher than facial. These findings indicate the greater efficiency of toothbrushing on facial surfaces.

In the present study in both the low and the high Plaque Index groups, facial scores were generally higher than lingual in the upper arch; while in the lower arch lingual scores were higher than facial. However in both arches, the differences in plaque scores between the low and the high Plaque Index groups were more marked on facial than on lingual surfaces.

Thus the differences in the distribution of plaque between the low and the high Plaque Index groups showed a distinct tendency to reflect the differences between the findings of Bay and his co-workers, and Loe and his co-workers respectively. Therefore it may be inferred that the pattern of plaque retention in the high Plaque Index group reflected more closely the natural distribution of plaque, and the pattern in the low Plaque Index group reflected to a greater extent the effect of toothbrushing on this distribution.
In the present study it was also found that increases in the reported frequency of toothbrushing were accompanied by significant decreases in the Plaque Index.

Individuals no doubt vary in their disposition to form and retain dental plaque. However it appears that the retention of plaque and more particularly the pattern of plaque retention is most strongly influenced by the relative efficiency of oral hygiene procedures on the various surfaces of the teeth.

The results of the present study and of the other studies quoted confirm certain common clinical observations. Apparently the natural distribution of plaque does not favour the anterior or the posterior teeth, but the differential action of toothbrushing as it is normally practised results in a progressive increase in plaque retention from the anterior to the posterior teeth.

It also appears that physiological activity within the oral cavity results in more plaque being retained on the facial than the lingual surfaces of the teeth, particularly in the upper arch. However the greater efficiency of toothbrushing on the facial surfaces alters this pattern, so that the lower lingual surfaces tend to retain most plaque while the upper lingual surfaces still remain most free of plaque.
One comment is pertinent concerning the Simplified Oral Hygiene Index of Greene and Vermillion. There were marked differences between the mean plaque scores in the low and high Plaque Index groups for the facial surfaces of the upper molars and upper and lower anteriors. However, the relatively small difference between the mean plaque scores of the two Plaque Index groups for the lingual surfaces of the lower molars indicates that these surfaces did not provide a good index of oral hygiene.

The findings of the present study suggest that in oral hygiene instruction more attention should be given to the posterior teeth and also to the lingual surfaces in the lower arch, particularly in subjects with relatively good oral hygiene. Also, some attention should be given to improving the design of toothbrushes in order to facilitate cleaning of the lingual surfaces of these teeth.
7. Distribution of periodontal disease.

Lovdal and his co-workers assessed the reduction in gingivitis scores on the facial and lingual surfaces of the teeth following a five year programme of oral hygiene instruction. Before commencement of the programme, gingivitis scores in subjects with poor oral hygiene were approximately equal on facial and lingual surfaces; in subjects with good oral hygiene, facial scores were less than lingual. At the end of the programme facial scores were less than lingual for all subjects, and the improvement in facial compared to lingual scores was greatest in subjects who had poor oral hygiene at the commencement of the programme and least in subjects with good oral hygiene then.

Suomi and Barbano, studying the distribution of gingivitis on facial and lingual surfaces in individuals 20 - 24 years of age, found that in the upper arch facial scores exceeded lingual scores; in the lower arch, except for the lower anteriors, lingual scores were higher than facial.

In the present study no record was made of the distribution of periodontal disease on the facial and lingual surfaces. However the findings of the two studies quoted above confirm the results of the present study concerning the distri-
bution of plaque on the facial and lingual surfaces.

In the present study the distribution of periodontal disease showed a significant increase in mean scores from the anteriors to the premolars and again to the molars in both upper and lower arches. This relationship was observed for all individuals in this study and held true for both the low and the high Periodontal Index groups.

The only difference between the mean periodontal scores of the upper and corresponding lower teeth was that the upper premolars had higher scores than the lower premolars. This relationship was observed for all individuals in the study, but only held true for the high Periodontal Index group.

Suomi and Barbano observed a posterior increase in gingivitis scores on lingual surfaces, but on facial surfaces the highest scores were found on the upper molars followed by the lower anteriors 236.

Bossert and Marks, in individuals 16 - 24 years of age, found that, except for the lower anteriors which were the most commonly affected teeth, there was a posterior increase in the percentage of teeth affected with periodontitis. The percentage of upper and lower teeth affected by periodontitis was the same, although more upper than lower teeth had been extracted
for this reason. Lightner and his co-workers also observed that the lower anteriors had the highest mean score for periodontal disease. It will be recalled that the mean plaque score of the lower anteriors found by these investigators was also relatively higher than that in the present study.

In this respect the results of the present study differ from those of certain previous investigators. The relatively low periodontal and plaque scores for lower anteriors in this study were probably due to the large number of subjects in the sample with good oral hygiene. For this has been shown to reduce plaque scores more effectively on the anterior than the posterior teeth.

8. Comparison between the distribution of plaque and periodontal disease.

Generally for all individuals in this study, the distribution of plaque was similar to that of periodontal disease. The only important difference was that the periodontal scores of the premolars, particularly in the upper arch, were relatively higher than the plaque scores of these teeth.

There were no significant differences between the mean plaque scores of the anteriors and premolars, whereas
the mean periodontal scores of the premolars were significantly higher than those of the anteriors in both upper and lower arches.

Also for all individuals, the mean plaque scores of the upper teeth were significantly less than those of the corresponding lower teeth. Whereas there were no significant differences between the mean periodontal scores of the upper and lower teeth, except that the upper premolar scores were higher than those for the lower premolars. This relationship between the premolars only held true in the high Periodontal Index group.

From the findings on the effect of gingival restorations and carious lesions on the Periodontal Index of posterior teeth, it is suggested that this factor was most likely to have accounted for the differences between the distribution of plaque and periodontal disease.

9. Prevalence of gingivitis on selected teeth according to their plaque scores.

As the causal relationship between plaque and periodontal disease is now well established, a useful direction for epidemiological research in this field may be to ascertain if there exists a general level at which plaque exerts its pathological potential. No doubt this would vary between individuals,
but a common threshold of plaque activity (perhaps varying with age) might exist for most individuals. Rephrasing Mandel 165, it is possible that a sixty per cent reduction in plaque may translate into zero per cent pathogenicity for periodontal disease.

The analysis of the percentage of selected teeth affected by gingivitis according to their different plaque scores is an essay in this direction. It was found that the percentage of teeth affected by gingivitis rose in a linear fashion as the plaque score increased on the facial and/or lingual surface.

This finding indicates that measurement of the coronal extension of plaque was a valid method of assessing the pathological activity of plaque. However it was not possible to detect a critical level of plaque activity in terms of gingivitis.

The criteria used for the estimation of plaque, as long as it is plaque that is measured, would appear to be of less significance than those used for periodontal disease. For in measuring the coronal extension of plaque, one is at the same time measuring other factors such as the thickness and age of the plaque. However the measurement of the earliest clinical signs of gingivitis in a consistently reproducible manner is a more serious problem. It is possible that a more sensitive index such as the Gingival Index of Loe149 might yield more significant results than Russell's Periodontal Index.
SUMMARY AND CONCLUSIONS

The mean Periodontal Index for all individuals in the young adult sample chosen for this study was 1.10.

Of the two sources for this sample, the dental students had significantly lower mean Periodontal, Plaque and Calculus Indices than the hospital patients.

All the individuals in this study were divided into two Plaque and two Periodontal Index groups.

The mean Periodontal, Plaque and Calculus Indices were all significantly less in the low Plaque and Periodontal Index groups than in the high Plaque and Periodontal Index groups.

For all individuals, there was a moderate correlation between the Periodontal and Plaque Indices and between the Periodontal and Calculus Indices.

The correlations between the Periodontal and Plaque Indices were weaker in posterior teeth than anterior teeth, particularly for individuals in the high Periodontal Index group. This was interpreted as indicating that factors other than plaque, which were associated with periodontal disease in posterior teeth, tended to be associated with periodontitis.

For all individuals in this study, the mean
Periodontal Index of posterior teeth with gingival restorations or carious lesions was higher than that of all posterior teeth. This relationship only held true for the dental students and the high Periodontal Index group.

Only in the student group was even a weak correlation found between the number of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth.

The correlation between the Periodontal Index of posterior teeth with gingival restorations or carious lesions and the Periodontal Index of all posterior teeth was stronger in the student, low Plaque and low Periodontal Index groups.

Thus the relationship between restorations and carious lesions involving the gingiva and periodontal disease in posterior teeth was most clearly observed when other factors, plaque and calculus, were minimal.

No correlation was found between the Malalignment and the Periodontal Indices for all teeth or between the Malalignment and the Plaque Indices for all teeth.

No differences were found between either the mean Periodontal or the mean Plaque Index for malaligned anterior teeth compared to the mean Periodontal or Plaque Index for all
anterior teeth.

A correlation between the number of malaligned anterior teeth and the Periodontal Index of all anterior teeth was found only in the student, low Plaque and low Periodontal Index groups. This correlation was weak and was best observed in the group where periodontal disease was low.

The weak correlation in the high Plaque Index group between the Plaque Index of malaligned anterior teeth and the Plaque Index of all anterior teeth contrasted with the stronger correlation between these two Indices in the low Plaque Index group. This was interpreted as indicating that malalignment had more effect, not necessarily to increase scores, on the distribution of plaque in subjects with poor oral hygiene than in subjects with good oral hygiene.

The prevalence of gingival recession increased with the reported frequency of toothbrushing.

The surfaces most commonly affected by recession were the facial surfaces of the upper quadrant from first premolar to first molar.

No differences were observed between the mean plaque scores of tooth surfaces affected by recession and the mean plaque scores of all similar tooth surfaces.
Increases in the reported frequency of tooth-brushing were accompanied by significant decreases in the mean Periodontal, Plaque and Calculus Indices.

The distribution of plaque was analysed in some detail, firstly for all individuals in this study and then for individuals in the low and the high Plaque Index groups separately. This was done in order to compare the distribution of plaque at different levels of oral hygiene.

On facial surfaces, there were no significant differences in plaque scores between the anteriors and the premolars in the upper or the lower arch. But facial plaque scores in both arches increased from the premolars to the molars. On lingual surfaces, plaque scores increased from the anteriors to the premolars and again to the molars.

This increase in plaque scores on the posterior teeth was more marked in the low than in the high Plaque Index group. It was thus interpreted as resulting from toothbrushing as practised by the subjects in this study.

In the upper arch, facial plaque scores were greater than lingual plaque scores. In the lower arch, lingual scores were greater than facial scores. In both arches, the differences in plaque scores between the low and the high Plaque Index groups were more marked on facial than on lingual surfaces.
This was interpreted as indicating that toothbrushing was more efficient on facial than lingual surfaces.

The distribution of periodontal disease was similar to that for plaque.

Periodontal scores in both arches increased from the anteriors to the premolars and again to the molars.

The only important difference between the distribution of these two conditions was that the premolars, particularly in the upper arch, had relatively higher scores for periodontal disease.

The percentage of selected teeth affected by gingivitis rose in a linear fashion as their plaque scores increased.


70. Gillings, Barrie. - Personal communication, 1967.


74. Glickman, Irving. - Ibid. (p. 866-867).


114. Kawamura, Yojiro. - Ibid. (p. 81).


133. Lanke, Lisa S. - Ibid. (p. 127).


