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PERIODONTAL DISEASE

Report of an Expert Committee on Dental Health

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WORLD HEALTH ORGANIZATION

PALAIS DES NATIONS

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**PERIODONTAL DISEASE :
REPORT OF AN EXPERT COMMITTEE ON DENTAL HEALTH**

Geneva, 22-27 August 1960

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PERIODONTAL DISEASE

Report of an Expert Committee on Dental Health

The Expert Committee on Dental Health met in Geneva from 22-27 August 1960 to discuss periodontal disease. Opening the session, Dr M. G. Candau, Director-General, stated that in the past periodontal disease had largely been overshadowed by dental caries although the public health problems it raised were just as great, and in some countries even greater. It was essential to ensure that the gains achieved in the prevention and treatment of dental caries were not offset in later life by the ravages of periodontal disease. Dr Candau stressed that a reorientation of dental education and dental health services towards prevention and early treatment and an intensification of dental health education and dental research were required if the problems arising from periodontal disease were to be successfully countered.

Dr A. L. Russell was elected Chairman of the Committee, Dr J. Kostlán, Vice-Chairman, and Dr R. D. Emslie, Rapporteur.

INTRODUCTION

Periodontal disease is one of the most widespread diseases of mankind. No nation and no area of the world is free from it and in most it has a high prevalence, affecting in some degree approximately half the child population and almost the entire adult population.

Research and clinical evidence indicate that the damage caused to the supporting structures of the teeth by periodontal disease in early adult life is irreparable, whilst in middle adult life it destroys a large part of the natural dentition and deprives many people of all their teeth long before old age. The total effects of periodontal disease on the general health of populations is unassessable.

People have come to accept this unfavourable state of affairs as inevitable but research and improved methods of prevention and treatment show that this is not so, and that if the public would accept and follow advice on prevention and seek early dental care, the prevalence of the disease and the severity of its sequelae could be considerably reduced. The generally accepted principle that prevention is better than cure applies to periodontal disease as much as to any other ; this must be fully recognized by governments and the general public because they will be required to

meet the costs of preventive measures. It will therefore be necessary to educate the public as to the benefits of these measures by intensive dental health education.

The relationship between the needs of dental care and the personnel and facilities available varies widely between countries. Even in countries with well-developed dental services, only a small fraction of the periodontal disease problem is being tackled. In others, the attention it is receiving is negligible in relation to the population and the needs. In these circumstances, preventive measures directed towards the child population would pay the best dividends. However, it will be essential to increase the number of dentists, of which there is a world-wide shortage, and to consider augmenting their efforts by providing well-trained auxiliaries. The dental profession also must be prepared to devote a much greater proportion of its time and efforts to the preventive and curative care of periodontal disease than it does at present. This reorientation of outlook is one of the most important needs of the dental profession today.

Finally, as all the problems relating to periodontal disease have world-wide significance, there must be facilities for interchange of personnel and information, particularly as regards research, techniques of health education, the organization of dental care services, and improvement in preventive and curative measures.

With these considerations in mind and with the aim of focusing world-wide attention on periodontal disease, the Director-General, in presenting his programme of activities for 1960, proposed that "an expert committee of seven members should meet to appraise present scientific information in regard to the prevention and control of the disease."¹

1. PRESENT KNOWLEDGE OF THE PERIODONTAL STRUCTURES (PERIODONTIUM)

The Committee decided that, in view of the vast literature on the subject, it would be desirable to review present knowledge of the periodontal structures or periodontium as a necessary prerequisite to a better understanding of the disease processes.

1.1 Definition of the periodontium

All the elements supporting the tooth, represented by cementum, the periodontal membrane, the alveolar bone and the gingiva constitute the periodontium. The periodontium is a functional organ and the different

¹ *Off. Rec. Wld Hlth Org.*, 1960, 89, 39

tissues do not attain their mature form until the tooth has been subject to the forces of mastication.

The term periodontium has been accepted by both the American Academy of Periodontology and the British Society of Periodontology and has gained wide currency in English-speaking countries. Synonymous with periodontium is parodontium which is accepted by ARPA * Internationale. The term parodontium is used in most European countries as well as in South America.

For the understanding of periodontal disease on an international level this difference in nomenclature is not serious, although it would be desirable to adopt a unified terminology.

1.2 Anatomy of the periodontium

The tooth is suspended in its socket by the periodontal membrane which is embedded in the cementum of the root and in the bone of the socket. The alveolar processes of the jaw-bones are covered by cortical bone which fuses buccally and lingually with the cortical plate of the jaws.

The gingiva extends from the gingival margin to the alveolar and palatal mucosa. It has also been subdivided into the free gingiva which surrounds the gingival crevice, and the attached gingiva. Towards the oral cavity it is covered with stratified squamous epithelium. The superficial layer is usually keratinized, which gives the gingiva its pale pink appearance, in contrast to the red of the oral mucosa. The normal gingiva is firm and stippled and is attached to the tooth and bone by the supra-alveolar periodontal fibres and the marginal part of the alveolar periosteum. The buccal and lingual surfaces of the gingiva end with a knife-edged gingival margin. The interdental papilla also forms a knife-edge between the teeth.

Between the gingiva and the tooth is an invagination of the epithelium measuring 1 mm or more. This invagination is closely fitted around the tooth forming the gingival crevice.

1.3 Histology of the periodontium

The cementum consists of collagenous fibres and mineral salts. The periodontal fibres are attached to the cementum. Little is known about the vital processes in the cementum and whether or not they are of any significance in the development of periodontal disease.

The periodontal membrane (synonyms: desmodonte, periodontal ligament) consists mainly of collagenous fibre bundles. In non-functioning teeth, the periodontal fibres are normally oriented parallel to the root

* Association pour les Recherches sur les Parodontopathies

surface, and are not organized to form strong bundles. When the teeth begin to function the fibres become arranged more or less perpendicular to the root surface.

Most of the periodontal membrane is located between the alveolar bone and the cementum (the remainder being in the supra-alveolar part). When the tooth is fully erupted the distance between the cemento-enamel junction and the alveolar crest is approximately 1 mm. In this area, the periodontal fibres fan out in the gingiva.

There is an ample supply of blood vessels in the periodontal membrane. Arteries and veins penetrate the alveolar wall and branch out into numerous small vessels. The slightest touch on the tooth will be registered by the proprioceptive nerve system and injury will affect the pain receptors. Close to the tooth surface there is a fine epithelial reticulum or "rests of Malassez" which are remnants of the epithelial sheath of Hertwig.

The gingiva is covered with a squamous epithelium nourished by numerous connective tissue papillae. The connective tissue is dense and strong.

The crevicular epithelium and epithelial attachment together form the epithelial cuff or epithelial invagination which faces the tooth and differs from the surface epithelium in many respects.

The relationship between the epithelial invagination and the tooth is still controversial. One school of thought maintains that there is a real attachment between the two and that the bottom of the gingival crevice is located at the coronal limit of the epithelial attachment. Another school of thought maintains that after eruption of the tooth the epithelial cells only adhere to the enamel surface, and that the bottom of the gingival crevice is found at the deepest point of the epithelial cuff. According to this concept the entire epithelial invagination is kept in contact with the tooth surface by the circular fibres of the gingiva and the blood pressure.

The bone of the tooth socket or alveolus commences approximately 1 mm below the apical portion of the epithelial attachment or cuff. It consists of cancellous bone, except where the socket involves part of the cortical plate. In the bony socket the periodontal membrane replaces the periosteum.

1.4 Biology of the periodontium

1.4.1 *Periodontal membrane*

One function of the periodontal membrane is to transfer the force of mastication from the teeth to the jaws. When the force is directed apically, the periodontal membrane is evenly stressed with all the fibres involved at

the same time. When force is applied horizontally to the teeth, only some of the fibres are stretched.

Normal chewing will influence the orientation of the periodontal fibres as a result of the mechanical stimuli. However, increased function will increase the strength of the periodontal membrane and change the orientation of the fibres only to a limited extent. If the force surpasses this threshold, damage will ensue. The first signs of injury are circulatory disturbances and resorption of the alveolar wall. In this way the tooth may adjust itself, taking up a new position in which conditions are more favourable.

The vascular system of the periodontal membrane has been likened to a hydraulic shock absorber. Chewing imparts a series of mechanical impulses which last only for a fraction of a second, during which time blood is pressed out of the vessels. The small dimensions of the capillaries cause considerable resistance, which also partly counteracts the force on the tooth. The intermittent pressure during mastication therefore acts as a pump and helps to increase the blood flow to meet the requirements of the increased function.

1.4.2 *Epithelial rests of Malassez*

The function of these rests is obscure. However, they may play a role in the maintenance of the periodontal space and in the prevention of root resorption.

1.4.3 *The normal gingival crevice*

The gingival crevice has particular significance because the initial stages of periodontal disease usually commence there. There are different concepts regarding the nature of the vital processes taking place in the gingival crevice.

(a) *The epithelial attachment concept.* According to this concept, under ideal conditions the epithelium should be firmly attached to the tooth from the cemento-enamel junction to the split in the epithelium representing the base of a shallow gingival crevice. It has been assumed that, once detached, the epithelium will only become reattached by proliferation of basal cells at the growing end of a new epithelial invagination. However, this view has been modified slightly during the last few years.

(b) *The epithelial cuff concept.* According to this concept, the apposition of epithelium to the tooth depends upon adhesion. Ideal conditions exist when the epithelial cuff adheres to the tooth from its deepest point up to the gingival margin, without interposition of bacterial plaque, calculus, or foreign bodies. The epithelial cuff has a great potentiality for defence and healing.

1.5 Pathology of the periodontium

1.5.1 *Influence of endogenous factors*

Some of the morphological changes due to systemic factors in periodontal disease are well established. Vitamin C deficiency interferes with the formation and maintenance of collagen, and leads to generalized, non-inflammatory destruction of the collagenous fibres throughout the body. Bone formation is retarded or arrested and bone resorption prevails. Owing to the decreased resistance to infection, the development of any inflammatory process or the deepening of a pre-existing periodontal pocket takes place more rapidly than usual. However, apical migration of the epithelial cuff does not seem to take place unless initiated by some exogenous factor. Experiments on scurvy demonstrate the delicate interplay between exogenous and endogenous factors and how they both, in their way, influence the speed and development of periodontal disease.

Periodontal disease is also influenced by other vitamins and hormones, but their effect on the morphological picture is less spectacular than that of Vitamin C. There is no doubt that diabetes predisposes to periodontal disease, but the histological features of inflammation in diabetes do not seem to be specific.

Vitamin A and some of the sex hormones have an effect upon epithelium, and typical changes due to imbalance may be discovered when biopsies are examined microscopically.

Except for vitamin and protein deficiencies, hormonal disturbances, and some blood dyscrasias, systemic factors do not produce specific morphological changes in the periodontal tissues.

1.5.2 *Influence of exogenous factors*

1.5.2.1 *Bacterial irritation*

The anatomical features of the gingival margin predispose towards periodontal disease. The tooth surface permits retention of plaque and materia alba (oral debris) which initially will be in contact with the gingival margin.

The plaque is built up mainly of living micro-organisms, the deeper layers of which often degenerate and become calcified to form calculus. As the plaque consists of living organisms it has the ability to grow both in thickness and in an apical direction. The speed of this apical growth depends on the aggressive or irritant properties of the micro-organisms as well as on the defence mechanisms of the host.

When the plaque grows below the gingival margin, the area in contact with the soft tissue increases in width. Although the bacteria of the plaque are usually considered to be non-pathogenic, in this situation they are present in the highest possible concentration (in contrast to the organisms

suspended in the saliva) and are kept constantly in contact with the soft tissue. The irritation from the bacteria and their toxins will invariably lead to degeneration of the crevicular epithelium with inflammation in the adjacent connective tissue. *When subgingival plaque has been formed, the gingival crevice has become a pathological pocket.*

Materia alba (oral debris) is the soft white material accumulating in an unclean mouth on the surface of the teeth and gingival margins. The main components of materia alba are the same micro-organisms that form the plaque with particles of food which may be mixed with them. Desquamated epithelial cells and more or less disintegrated leucocytes are other constant components of materia alba.

Both the plaque and the materia alba may form an important matrix for the deposition of calculus. Calculus is composed mainly of filamentous micro-organisms cemented together by organic and inorganic material from the saliva.

There is still disagreement as to whether or not the micro-organisms penetrate the epithelial lining and invade the connective tissue; however, the bacterial toxins and enzymes obviously do, and the inflammatory reaction is probably caused mainly by these.

In some cases, the gingival inflammation may be of the acute type, but in the majority of cases the inflammation is chronic. This chronic inflammation is characterized by the presence of lymphocytes, plasma cells, and macrophages. The number of polymorphonuclear leucocytes in the tissue is small; most of them appear to migrate under the influence of positive chemotaxis through the pocket epithelium to attack the micro-organisms in the pocket. Between the epithelium and the plaque there is usually a great number of polymorphonuclear leucocytes and it is partly due to their action that the plaque does not grow faster in an apical direction. The balance between the bacteria on one side and the defence mechanisms on the other decides the fate of the tooth. If the apical growth of the plaque is not stopped, it may reach the apex of the tooth well within a normal life span.

The polymorphonuclear leucocytes, mixed with tissue fluid and desquamated epithelial cells (pus), are slowly expressed from the pocket. Pus secretion from a pocket indicates the presence of subgingival plaque or calculus, although neither may be readily visible on clinical examination.

Concomitant with the cellular and vascular reaction, there is destruction of the collagenous fibres, probably due to the effect of enzymes and toxins. The cellular infiltration and the collagen destruction are most advanced adjacent to the plaque. Thus there may be a severe inflammation in the connective tissue close to the tooth whereas the visible part of the gingiva may be normal.

When the plaque has grown so close to the cemento-enamel junction that the periodontal fibres below the epithelial cuff are broken down, there

is nothing to prevent the epithelial cells from growing in an apical direction until new and undamaged periodontal fibres are encountered. This represents a loss of supporting structures—and it is a permanent loss. The speed at which the periodontal fibres are destroyed and the pocket is deepened depends first and foremost on the speed of the apical growth of the plaque. At the same time as the periodontal fibres are broken down and the epithelium grows apically, the alveolar wall is being resorbed as a result of the same bacterial irritants and inflammatory reaction.

Bacterial plaque is undoubtedly the most important single etiological factor in most periodontal diseases. Differences in bacterial components of plaques and in tissue resistance account for the varying clinical characteristics of these diseases in different individuals.

1.5.2.2 *Biochemical aspects of periodontal disease*

Provided that the bacteria overcome the defence mechanisms of the host tissue, they generally produce disease by: (1) interfering with the host tissue metabolism; (2) elaborating substances which are toxic to the host tissue cells or disrupting host tissue elements or both; (3) eliciting reactions in the host tissues which reflect some type of abnormal tissue response, i.e., hypersensitivity. There is evidence that members of the gingival microbial flora (staphylococci, various streptococci, diphtheroid organisms and others) produce a number of enzymes capable of acting on different host tissue components.

The resistance of the host tissue is an important factor in determining the extent to which the aggressive tendencies of the bacteria are permitted to manifest themselves. Natural and acquired defence mechanisms operate in order to prevent the parasite from gaining entrance into and establishing itself within the host. The principal natural resistance mechanisms concerned with the immobilization and eventual destruction of harmful bacteria are represented by the phagocytic cells of the host, together with antibacterial components of the serum, the tissue fluids and secretions of various kinds. Acquired resistance is mainly, but not exclusively, connected with the capacity of host tissue cells to produce specific antibody against bacterial antigens.

Both natural and acquired resistance are modified by a number of factors, among which are the nutritional status and the hormonal balance of the host. The biochemical mechanisms underlying the action of the various hormones have not yet been fully elucidated. It appears, however, that hormonal disturbances influence the host tissue resistance by interfering with various aspects of the tissue cell metabolism.

These observations suggest that enzymes produced by gingival bacteria may participate in the development of periodontitis by altering specific properties of the tissues, as well as by causing a general increase in the

permeability of the connective tissue to other bacterial products such as, for example, bacterial endotoxins, which are directly capable of eliciting an inflammatory response.

1.5.2.3 *Mechanical irritation*

There is no convincing histopathological evidence that, in man, mechanical irritation due to rough surfaces of teeth and overhanging margins of restorations will, alone, cause severe inflammation. However, the rough surface retains bacterial plaque and the inflammation seen adjacent to unpolished restorations is more likely to be caused by plaque than by the rough surface. Rough subgingival restorations must therefore be classified as predisposing factors.

1.5.2.4 *Traumatic occlusion (traumatic articulation)*

Knowledge of the histopathological changes in traumatic occlusion is based mainly on animal experiments. If a tooth is subjected to increased function, the periodontal membrane will first react with hypertrophy; the fibres increase in number and thickness and the bone trabeculae become stronger. However, the stress cannot be increased above a certain level without the initiation of pathological processes.

Circulatory changes may be the first sign of damage. The alveolar wall may be resorbed and the tooth moved into a new position. This will probably not lead to any irreparable damage in the periodontium.

If the stress is increased still further, the periodontal membrane on the pressure side may be so much compressed that it becomes necrotic. The necrosis in the periodontal membrane is usually limited to the intra-alveolar part, and does not extend into the supra-alveolar portion between the alveolar crest and the deepest point of the epithelial cuff. Thus it is improbable that gingivitis can be caused by traumatic occlusion alone.

The necrosis in the periodontal membrane seems to be aseptic if bacteria are not introduced through the circulatory system. Such a contamination is indeed possible and, if it occurs, an abscess may be formed. The abscess may penetrate into the gingival pocket, in which case the way is open for growth of the epithelium apically.

An aseptic necrosis of the periodontal membrane will probably heal without any permanent damage as soon as the occlusal trauma is discontinued. The necrotic tissue is resorbed and replaced with granulation tissue from which new cementum and periodontal membrane may be organized.

A number of experimental investigations on the effect of traumatic occlusion in animals have shown that it is very difficult to produce pocket deepening. Theoretically it is feasible to produce down-growth of epithelium without contamination of the necrotized periodontal membrane, but this will probably take place only in exceptional cases.

To what extent traumatic occlusion can lead to pocket formation in man has not been established on histopathological evidence. It is possible that damage to the intra-alveolar part of the periodontal membrane may speed up the pocket deepening which is caused by subgingival bacterial plaque. The subgingival plaque causes inflammation which involves the supra-alveolar periodontal membrane. If at the same time necrosis of the intra-alveolar periodontal membrane, due to trauma, spreads in a coronal direction this may contribute to the destruction of the fibres below the epithelial cuff.

1.5.2.5 *Chemical irritation from crown and filling materials*

Some filling materials undoubtedly cause chemical or electrical irritation or both, but the inflammation is limited in its extent. The greatest danger with crowns and fillings that are extended below the gingival margin is that they contribute to the retention of plaque owing to their roughness.

1.6 **Clinical pathology of the periodontium**

Histopathological changes in the different periodontal tissues may or may not be detectable clinically. In the diagnosis of periodontal disease it is extremely important to be able to correlate the clinical findings with the histopathological state. To some extent this can be done and some of the main features are outlined below.

1. *Colour*. If the gingiva is red or magenta, this is a sign of inflammation. However, the colour of the gingiva is also dependent on the degree of keratinization and the thickness of the epithelium, the amount of oedema, and the presence or absence of pigments. In some cases keratinization may be absent; if so, the capillaries will shine through even in the absence of inflammation. In other cases pigmentation may camouflage an underlying inflammation. A severe inflammation in the part of the gingiva bordering the crevice or pocket is often not evident from the oral surface.

Evaluation of the colour of the gingiva is a valuable diagnostic guide, but cannot be used alone to determine the presence or absence of gingivitis.

2. *Contour*. Thickening of the gingival margin and gingival clefts may be signs of periodontal disease, but it may also have been caused by too vigorous brushing of the teeth, or may merely represent anatomical variations.

3. *Bleeding*. Spontaneous bleeding and bleeding on pressure are obvious signs of inflammation. Profuse bleeding during probing and instrumentation in pockets is also a sign of inflammation; it is due to crevicular ulceration and increased vascularity of the inflamed connective tissue.

4. *Pus secretion.* If pus can be squeezed out of the pocket, this is a definite sign of severe inflammation.

5. *Pocket deepening.* Pockets of more than 3 mm are probably in most cases pathological. There may be exceptions, however, such as pockets around erupting teeth and pockets where successful treatment has been carried out.

6. *Increased tooth mobility.* Increased mobility in the absence of pathology can sometimes be observed in teeth that are subjected to heavy function. However, in most cases increased mobility is a sign of pathology. Evaluation of mobility is part of the clinical examination, but other signs of disease must be present before increased mobility can be taken as a sign of periodontitis.

7. *Tooth migration.* Tooth migration is caused by pressure from granulation tissue within the walls of pockets. If a pocket is of irregular depth, this pressure, although slight, will be uneven around the tooth and may cause it to migrate with the development of a diastema.

2. DEFINITION AND CLASSIFICATION OF PERIODONTAL DISEASE

2.1 Definition

In the broadest sense, periodontal disease may be defined as being all those pathological processes that involve the periodontium (see page 4). They can be limited to one of the constituents of the periodontium (e.g., in gingivitis) or include several or all of the tissues. Most of the periodontal diseases are limited to the periodontium, but in some cases they may be manifestations of general diseases or diseases of other organs.

2.2 General classification

Considerations of etiology, although important, can play only a secondary and accessory part in a classification by contributing certain details for minor sub-divisions. Study of causes is also an unsuitable basis for classification, as many etiological factors, some possibly unknown, may be involved in any particular case.

Clinical assessment lacks sufficient precision to serve as a foundation for classification, especially in view of the fact that different pathological processes may present very similar clinical appearances.

The most valid basis for a classification of periodontal diseases is therefore one based on general pathology. In this connexion, three

fundamental types of pathological process, differing distinctly in characteristics, origin and course, are well recognized :

(a) *Inflammatory processes* (gingivitis, periodontitis)

An inflammatory process develops whenever an irritant affects the integrity of the tissues by physical or chemico-infectious action. It is a type of reaction and repair involving primarily the non-differentiated connective tissue and its circulatory system. The differentiated connective tissues (for example, bone, cartilage, dentine, cementum, etc.) the parenchymatous tissues, and the neighbouring epithelia may also undergo secondary changes.

(b) *Degenerative processes* (periodontosis ?)

The term degenerative is applied to regressive conditions related to a general or local metabolic deviation. They are characterized by structural changes resulting in the disappearance from the histological picture of certain elements, or the appearance of substances foreign to the normal composition of the cells or tissues.

(c) *Neoplastic processes*

The periodontium may be the site of primary neoplasms deriving from its various constituents (epithelium, connective tissue, vessels). It may also rarely be the site of metastases from neoplasms elsewhere.

(Note. Studies of the pathology of the periodontium have shown that all the three types of general pathological process described above may be present ; in many cases, however, these processes are not found in a pure form but in association with one another.)

2.3 Classification of inflammatory periodontal diseases

Among the periodontal diseases the inflammatory types are not only by far the most common, but are also more readily preventable and treatable. The Committee therefore decided to concentrate its efforts on these and to omit consideration of the rarer periodontal diseases. The following classification was proposed.

1. *Gingivitis*

(a) *Acute*

(i) Acute ulcerative gingivitis (Vincent's disease)

(ii) Acute non-specific gingivitis (associated with local trauma, or other diseases, such as acute herpetic stomatitis, streptococcal throat, blood dyscrasias, etc.)

(b) Chronic

- | | | |
|--|---|---|
| <ul style="list-style-type: none"> (i) Chronic gingivitis (ii) Chronic hyperplastic gingivitis | } | (may be modified by systemic disease, or conditions such as pregnancy, or the use of drugs such as diphenylhydantoin) |
|--|---|---|

2. *Periodontitis*

(a) *Acute* (such as periodontal abscess, or developing from acute ulcerative gingivitis)

(b) Chronic

(i) Periodontitis simplex : marginal horizontal bone resorption and true pocket formation—pockets usually of regular depth (the result of marginal irritation and possibly aggravated at some stage by occlusal trauma)

(ii) Periodontitis complex : irregular bone resorption and pockets of varying depth, often in younger individuals (occasionally, associated with recognizable systemic disease, but may have commenced as periodontosis, sometimes considered to be primarily a degenerative lesion with inconspicuous local factors ; occlusal trauma is often an aggravating local factor)

3. ETIOLOGY OF PERIODONTAL DISEASE

3.1 The role of local factors (direct and predisposing)

As explained in the section on pathology of the periodontium, bacterial plaque is the most important single etiological factor in most periodontal diseases. A number of contributory and predisposing local etiological factors may increase the irritating qualities of the plaque, favour its deposition, or interfere with its removal by natural or artificial oral hygiene. In addition, some other irritants which are not of bacterial origin may affect the periodontal tissues directly.

The following factors may modify the plaque or its effects :

3.1.1 *Calculus (tartar)*

The deposition of calcium salts within the bacterial plaque produces calculus which is firmly attached to the tooth either just above the gingival margin (supragingival calculus) or within the gingival crevice or pocket (subgingival calculus). Excessive supragingival calculus causes gingival recession and the eventual loss of the tooth, but from study of ancient skulls it would seem that a small amount of supragingival calculus can

be tolerated by the periodontal tissues without necessarily leading to progressive periodontal disease. Subgingival calculus is found only where gingival inflammation has been present for some time and should therefore be regarded as an aggravating factor. Carefully prepared decalcified sections show that the calculus is always separated from the soft tissue by a layer of leucocytes, desquamated epithelial cells, and bacteria. It is therefore probable that the products of the bacteria living on its surface and favoured by its presence are mainly responsible for the irritating action of calculus. The surface of the calculus is often rough, however, and direct trauma to the soft tissues may occur during mastication as a result of the gingiva being pressed against this rough surface. Individual tooth movement may also result in trauma to the interdental gingiva from calculus in this region.

3.1.2 *Consistency of food*

Raw and fibrous foods help to prevent the accumulation of bacterial plaque and calculus by their scouring effect during mastication. Conversely, soft and pappy foods have little cleansing action on the teeth, and are often swallowed without any mastication at all.

A long-term effect of the mastication of raw, fibrous or "sandy" foods is increased wear (attrition) on the teeth. This compensates for the continuous eruption of the teeth and prevents any lengthening of the clinical crowns. Where attrition is very marked, the clinical crowns may even become shorter with age, as is found in primitive communities. With soft diets, however, the increased length of the clinical crown from lack of wear may adversely affect periodontal health in two ways :

(a) With increase in age, the gingival margins of the opposing jaws become progressively further apart in centric occlusion. Thus the stagnation areas for bacterial plaque and calculus become further removed from the occlusal surfaces and incisal edges of the teeth, and less subject to the self-cleansing action of mastication.

(b) The mechanical advantage of lateral tilting forces on the crowns of the teeth becomes greater with age. Cusps also remain long, interfering with lateral excursions of the mandible. Both these factors increase the likelihood that occlusal trauma may eventually become an aggravating factor of any periodontal disease.

3.1.3 *Overcrowding of teeth*

Teeth that are instanding or outstanding from the dental arch inevitably interfere with natural or artificial cleansing, and increase the stagnation of debris against the teeth. In addition, the anatomy of the interdental gingiva is changed when the contacts between the teeth are close to the neck of the teeth or involve part of the roots. The space above the crest of the

exaggerated interdental "col" of gingival tissue represents a stagnation zone which is quite uncleanable. This is sometimes the main cause of localized deep pocketing in overcrowded mouths.

Teeth that are instanding or outstanding from the dental arch may be associated with localized gingival recession. When the teeth erupt, the gingival margins on the outstanding side will be receded in relation to the gingivae surrounding adjacent teeth. These receded areas are less likely to be cleaned either by natural or by artificial methods, with the result that bacterial plaque and supragingival calculus are able to build up and cause further recession. A high labial frenum may also interfere with cleansing in this region and movements of the lip cause retraction of the receded gingival margin from contact with the tooth, permitting accumulation of plaque within the pocket.

3.1.4 *Lip-seal*

Mouth-breathing, or lack of lip-seal, may be an important aggravating factor in all types of periodontal disease. Edentulous areas do not appear to be affected by mouth-breathing, which suggests that drying in itself is not an irritant. When teeth are present, however, the products of bacterial action at the gingival margin or within the crevice are not subject to the usual diluting and cleansing effects of saliva in patients who are mouth-breathers or who habitually keep their lips apart. In addition, when there is a lack of lip-seal debris tends to dry on the teeth and is more difficult to remove by the usual measures of oral hygiene.

3.1.5 *Design of dental restorations and prosthetic or orthodontic appliances*

Dental restorations that do not reproduce the normal anatomy of the tooth may lead to localized periodontal disease. Absence of tight contact points close to the occlusal surfaces of the teeth results in food impaction, and insufficient space between the necks of teeth produces an uncleanable interdental embrasure with an improperly shaped interdental gingiva. Overhanging and rough edges favour stagnation of bacterial plaque and consequent chronic gingivitis.

Removable partial prosthetic or orthodontic appliances favour bacterial stagnation around the gingival margins that are covered, and interfere with the diluting and neutralizing action of the saliva on the bacterial products. These effects are aggravated when the appliances are worn at night and infrequently removed for cleaning. When the periphery of such an appliance is less than 5 mm (approximately) from the gingival margin, or the gingival margin is covered by the appliance which has been "relieved" in this region, gingival hyperplasia with false pocketing tends to develop.

Stripping of gingival tissue from the teeth may be caused by "sinking" of partial prosthetic appliances without adequate occlusal rests. Occasionally, overstress of clasped abutment teeth may aggravate existing periodontal disease.

3.1.6 *Food impaction*

In a well-formed dental arch the shape of the teeth and the position of contact points between adjacent teeth ensure that during mastication food is deflected away from the gingival crevice and interdental embrasures. With loss of teeth, dental caries, or some types of malocclusion, these mechanisms may be disrupted, and localized impaction of food debris within the buccal or lingual gingival crevice, or interdentally, may occur. Continuation of this process will result in localized pocketing due to pressure from the impacted debris. Subsequent bacterial action within this debris will inevitably occur if it is not removed by the patient with, say, an interdental stick, dental floss or an elastic band. Although impaction of food causes only localized pocketing in some mouths, it is probably an important factor in the loss of alveolar bone which sometimes occurs in small experimental animals (e.g., impaction in hairs interdentally in rats).

3.1.7 *Direct trauma*

Damage to the gingival tissue from occasional hard particles in the food is probably not an important initiating factor in chronic periodontal disease. When the gingiva is enlarged and inflamed as a result of other local irritants, direct trauma from food during mastication can be an aggravating factor.

Damage to the gingiva from teeth of the opposing jaw may occur with certain malocclusions of Angle's Class II type. The regions affected are usually in the anterior part of the mouth, the lower labial and upper palatal gingivae being traumatized by the tips of the opposing incisor teeth.

Gingival trauma may also be produced by incorrect tooth-brushing techniques, the use of cleaning sticks, or various other habits. The use of chewing sticks may be an important factor in some countries where they are traditionally used instead of toothbrushes. Other habits, such as betel chewing, may also be of importance, although little definite evidence is available. Some recent surveys have, however, shown a significant relation between smoking and periodontal disease. Direct trauma may be an initiating factor in acute ulcerative gingivitis (Vincent's disease), e.g., gum flap over lower third molar being traumatized by upper tooth.

3.1.8 *Occlusal trauma*

Oversstress on the teeth from the teeth of the opposing jaw does not appear to be an initiating factor in periodontal disease. However, when

the supporting structures have been partially destroyed by progressive periodontal disease, a stage must inevitably occur when occlusal trauma becomes an important aggravating factor (secondary occlusal trauma). In this connexion, non-functional tooth-grinding (bruxism) may sometimes be more important than functional chewing.

3.2 The role of systemic factors

Little is known regarding the role of systemic factors in most of the human periodontal diseases, and an assessment of the relative importance of local and systemic factors can be no more than speculation. It is clear, however, that as more local etiological factors are discovered the necessity of invoking some unidentified systemic factor or factors decreases.

It is generally believed that systemic factors play a part in the etiology of periodontal diseases by in some way reducing local tissue resistance. Nevertheless, there are relatively few cases in which it is possible to point with any certainty to the systemic factor involved.

The following systemic factors appear to be of importance in some periodontal diseases.

3.2.1 *Racial or hereditary factors*

It has been generally accepted that certain races are more prone than others to periodontal disease. An "inherited inferiority of the dental organ" has been suggested to account for this. The results of some recent surveys have thrown doubt on the long-held conviction that racial factors are of great importance in susceptibility to periodontal diseases, but they have certainly not excluded such a possibility.

3.2.2 *Nutritional deficiencies*

That gingivitis may be associated with scurvy is well recognized, but surveys of gingivitis in areas where nutritional deficiencies are evident in the population have not shown any consistent association between the deficiencies and gingivitis. However, these findings do not prove that total nutrition and periodontal disease are unrelated; before any progress can be made towards answering this question, thorough dietary and nutritional surveys are needed, both in groups with a very low prevalence of periodontal disease and in groups with a very high prevalence.

3.2.3 *Systemic diseases*

Severe diseases such as diabetes mellitus or blood dyscrasias lower tissue resistance, and acute febrile disease may adversely affect conditions

in the mouth. In parts of the world where severe chronic diseases are common, they may play an important etiological role in periodontal disease in some of the population.

3.2.4 *Hormonal disturbances*

Certain types of gingivitis appear to be associated with endocrine imbalance. During pregnancy any pre-existing gingivitis tends to become more severe and to change in character. The rather rare chronic desquamative gingivitis occurs mainly in women at the time of the menopause, or during puberty and early adolescence. The most common form of gingivitis in children and young adults is the hyperplastic type, and it has been postulated that the temporary endocrine imbalance associated with puberty is responsible for this particular type of inflammatory reaction. A variation in the severity of gingivitis has also been noted in relation to the menstrual periods.

With the exception of the cases occurring during pregnancy, however, it has seldom been possible to demonstrate other signs of endocrine imbalance, and treatment with hormone preparations has so far proved unsatisfactory.

3.2.5 *Psychosomatic factors*

Very little is known about the effect of psychosomatic factors on periodontal disease. There is some evidence that they play a definite part in the etiology of acute ulcerative gingivitis (Vincent's disease). They may also be important in conditions such as bruxism which can aggravate periodontal disease.

3.2.6 *Drugs and heavy metals*

Regular use of the anticonvulsant drug diphenylhydantoin is often associated with gross gingival enlargement in persons with standing teeth. In edentulous mouths, no change occurs in the mucosa covering the alveolar process, and it therefore appears probable that the effect of the drug is merely to modify a pre-existing gingivitis. Control of periodontal disease in patients taking this drug is nevertheless a difficult problem.

Ingestion of salts of some metals (bismuth, mercury, or lead) causes dark deposits in the gingiva around teeth with bacterial plaque or calculus. Again, this would appear to be a modifying influence on the inflammatory reaction, salts of the metal being deposited around capillaries in the connective tissue; presumably absorbed products of bacteria react with the salts in the tissue fluids to form insoluble products (sulfides?) which are precipitated. There is no evidence that drugs or metals generally are important in the etiology of other periodontal diseases.

4. INDICES FOR RECORDING PERIODONTAL DISEASE

Population indices for the periodontal diseases are numbers that define the relative status of a population on a graduated scale, with definite upper and lower limits, for comparison with other populations classified by the same criteria and methods.

Population indices have little value in that type of study which aims at a total inventory of all defects in a specific group. One general family of indices is based on deviation of tissues from normal, and this is sometimes defined quite stringently. Others are limited to signs of disease considered to be of clinical significance. None of these indices represents a precision measurement (in the sense that one may measure the speed of light, for example); all rely to a greater or lesser extent upon the judgement of the examiner. Some may be determined very swiftly for large numbers of people; others require so much time as to limit the numbers that can be studied. Some require the use of bulky accessory aids, such as X-ray equipment; others are designed for a minimum of instruments. Some (like the DMF * indices in dental caries) are based on total tissue damage over the lifetime of the individual and are therefore irreversible; others represent estimates of present and active disease and can indicate response to treatment or beneficial circumstance. The choice of an index in any specific situation must be based upon the nature of the information required, the numbers of persons to be considered, and the type and amount of equipment that the circumstances and the location of the study will permit.

4.1 Reversible indices

In most populations, much the same conclusions will be drawn whether the periodontal disease index used is a reversible or a cumulative one, but a change for the better (following successful treatment; for example) can be demonstrated much more swiftly by measuring the morbidity than by means of an irreversible index. Whenever a relatively prompt recognition of improvement in a group is required the reversible index will be the index of choice. Two indices of this type have been widely used. These are the P-M-A index, and the Periodontal Index developed by the United States Public Health Service.

4.1.1 *The P-M-A ** index of gingivitis*

The P-M-A index of gingivitis is based upon the concept that the extent of gingivitis serves to indicate the severity of the affection.

* Decayed, missing, filled

** Papillary, marginal, attached

The criteria direct that a gingival unit be scored as a positive if it departs in any way from normal. This assessment is carried out for each papillary and marginal area of gingiva and for the attached gingiva associated with each tooth included in the examination plan. No mandatory examination method or procedure for determining the index has been prescribed or followed. The examination may include the entire mouth, or merely an anterior segment; it may be visual or supplemented by colour photographs. Some investigators have determined only the numbers of inflamed gingival papillae; others have counted the numbers of inflamed papillae, the numbers of inflamed marginal areas and, instead of the numbers of inflamed attached gingivae, the numbers of teeth showing gingival recession involving the cemento-enamel junction, these items being reported separately as the mean percentage involved per man. Still others have estimated the relative severity as well as the extent of gingivitis. Comparability between examiners may be difficult to achieve.

The index is very sensitive to small changes when the disease is mild and, given an experienced examiner, is probably the method of choice in a clinical trial of the effect of such agents as a mouthwash or toothbrushing on gingivitis.

P-M-A data are sometimes reported as prevalence (i.e., as the percentage of persons showing one or more inflamed gingival units) or as the average number of P-M-A units per person, a constant that takes into account the relative extent of inflammation as well as its prevalence. As long as the data are presented as percentages of the whole group of persons, or as mean scores per person, P-M-A findings may be analysed by modern statistical methods.

4.1.2 *The Periodontal Index*

The P-M-A index was developed solely for the assessment of gingivitis and was not, in any way, intended to apply to diseases affecting the deeper periodontal tissues. The Periodontal Index (PI) was designed as a more comprehensive index to cover both gingivitis and periodontitis. Progressive stages involve inflammation of the gingiva, pocket formation with consequent loss of alveolar bone, actual loss of function of the tooth, and eventually loss of the tooth itself. In essence, the examiner is asked to make a swift appraisal of the supportive tissues surrounding a tooth; to decide whether marginal periodontitis is present; and, if it is, to score the stage of the process according to the criteria described in Table 1 (see page 23). The average score for the teeth in the mouth of an individual is taken as the score for that person. Most persons with a clinical diagnosis of gingivitis score from 0.1 to 1.0, those with frankly-established destructive disease from 1.5 to 5.0, and those with disease in terminal stages from about 4.0 to 8.0, the maximum score.

TABLE 1. CRITERIA FOR THE PERIODONTAL INDEX

Score	Criteria
0	NEGATIVE — There is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissues.
1	MILD GINGIVITIS — There is an overt area of inflammation in the free gingivae, but this area 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 gingivae). There is no interference with normal masticatory function; the tooth is firm in its socket, and has not drifted.
8	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.

RULE : When in doubt, assign the lower score

Comparability of population findings between trained examiners can be quite as good with PI as with counts of DMF teeth. This degree of comparability depends upon mandatory procedures, and a series of criteria designed to leave as little as possible to the judgement of the examiner. *Reporting is limited to advanced and quite obvious lesions—those “clearly evident at first glance in good light”—upon which most examiners should agree. The examiner is instructed, whenever he is in doubt, to record the lower of the two scores that might be assigned.*

This degree of comparability has been achieved at the cost of an underestimation of the total number of lesions, a procedure that by some standards is drastic. It can be shown that, in chronic progressive conditions as highly prevalent as the periodontal diseases, the effect of a constant underestimation on sample size or accuracy of conclusion is virtually nil.

The index may be applied very swiftly and a minimum of equipment is required. It is sensitive to differences between moderate and advanced stages of disease. It has been used with success to make comparisons between two populations, within segments of a population, and to estimate change in status of a population over short or long spans of time.

Findings may be reported in several ways, each amenable to statistical analysis. The group score (the average of the scores of the individuals comprising the group) reflects both prevalence and relative severity of disease. Prevalence may be reported on several levels, as the proportion of persons with any sign of disease, or with one or more periodontal pockets, or with one or more teeth beyond function. It is sometimes useful to contrast the proportions of persons free from disease in two populations, and proceed to consider the relative severity of disease in persons in whom

disease was present. Studies have been reported in which periodontal disease was about equally prevalent in two populations, but the individual cases were much more severe in one population than the other ; conversely, periodontal disease may be much more prevalent in one population than another although the individual cases are of about equal severity in both.

4.2 Irreversible indices

If the reversible index can demonstrate a beneficial effect with a minimum of delay, it is likewise susceptible to the influence of fortuitous and transient variables which may seriously complicate the interpretation of data. The irreversible index, given time, provides data that are usually unequivocal. One irreversible end-result of periodontal disease is loss of alveolar bone. Most population indices of bone loss have been based upon radiographic examination.

4.2.1 *X-ray indices of bone loss*

There are limitations to this method of estimation but these limitations apply more to the diagnosis of the individual patient than to an estimate of community status. The degree of underestimation is relatively slight, and the reading error can be kept random. In the hands of meticulous workers, the method is capable of giving a reasonably correct average resorption figure for each age or group.

There has been no uniformity in reporting. Resorption has been rated on a scale from 0 through 10, with and without the aid of a measuring device. The resulting figure can be read directly as the percentage of bone lost in any single instance, or the average percentage loss for a group. Non-linear scales of bone loss have also been employed, with the mean or median score for the teeth present in the mouth representing the score for the individual. Before data returned by such methods can be compared with data reported in percentages, adjustments must be made which, in some cases, result in quite crude approximations.

Resorption of alveolar bone to a depth of more than 2 mm or 3 mm below the cemento-enamel junction has also been used to divide groups into two categories considered to be with, or without, periodontal disease.

Some loss of alveolar bone with age has been demonstrated in rats and humans, though no precise relation has been established. If bone loss due to physiological aging can be presumed to be relatively uniform between populations, it can have little effect on comparisons based on a scale of measurement, as the error would modify with equal force the findings for all as long as comparisons were made between groups of persons of equivalent age. For this reason, relative measurement on a scale would seem to be preferable for comparison between two populations.

Because the X-ray film is a relatively permanent record, independent of the patient himself, it can be read "blind", that is, without knowledge of any fact about the patient that might sway the examiner's judgement. Films taken years apart in time may be compared side by side and assessed by identical criteria, avoiding the "learning" effect in which examination procedures and criteria become more skilled and stringent, or more lenient, with the passage of time and the accumulation of experience. This type of error is very difficult to detect and virtually impossible to allow for by statistical means. Where an irreversible population index is appropriate, X-ray examination is probably the method of choice. Its principal drawbacks are the greater time and expense required for the assessment of each patient, and the difficulty of employing X-rays in the field, particularly in those regions of the world where transport facilities are poor and electricity is not in general use.

4.2.2 *Gingival recession*

A second irreversible lifetime measure of periodontal destruction which has proved useful in some situations is an index of gingival recession. The number of teeth in which gingival recession has exposed the cemento-enamel junction may be counted and computed as a percentage of the total teeth present in the mouth; the whole-group score is then computed as the mean percentage per man. While this score may be inadequate or misleading as a description of the status of any one individual, group gingival recession scores are closely correlated with group bone resorption scores as determined by X-ray examination.

This simple count of teeth with exposed cementum is readily learned by inexpert examiners and between-examiner variation can be reduced almost to zero. It can be difficult to make such counts in the mouths of persons with heavy supragingival calculus, or in betel-chewers. This index yields little information in young population groups.

4.3 **Composite indices**

Not all indices are based wholly upon reversible, or upon irreversible, signs of disease. Some make use of both types of sign. The index proposed by Ramfjord appears to belong in this category.

4.3.1 *Ramfjord Index*

Procedures and criteria for the Ramfjord Index are mandatory. Six teeth are examined in each mouth: the maxillary right first molar, the maxillary left central incisor, the maxillary left first bicuspid, the mandibular left first molar, the mandibular right central incisor, and the mandibular right first bicuspid. The area around each of these teeth is dried with

cotton wool and observed and tested for "deviations from health in colour, form, density, and bleeding tendency". Inflammation is scored in three degrees: a score of 1 for mild to moderate inflammatory gingival changes not extending all around the tooth; a score of 2 for mild to moderately severe gingivitis extending all around the tooth; or a score of 3 for severe gingivitis characterized by marked redness, tendency to bleed, and ulceration. This gingivitis score is the periodontal score for the tooth if, on probing, the epithelial attachment is found to be on enamel.

Measurements with a University of Michigan No. 0 pocket probe are then made on the mesial, buccal, distal, and lingual aspects of each of the six teeth examined. Where the epithelial attachment is on enamel, the depth of the crevice is noted but not used in the tooth score. If the gingival crevice in any of the four measured areas extends apically from the cemento-enamel junction but not more than 3 mm (including 3 mm) in any area, the gingival score is ignored and the tooth is assigned a periodontal disease score of 4. If any crevice extends 3-6 mm (including 6 mm) below the cemento-enamel junction, the tooth score is 5; and if any extends more than 6 mm below the cemento-enamel junction the tooth score is 6, the maximum on the scale. The average tooth score is the score for the patient. Where one or more of the specified teeth are not present in the mouth, the score is based on the remainder.

The distance from the cemento-enamel junction to the depth of the crevice is not measured directly. Independent measurements are made for the distance from the cemento-enamel junction to the gingival margin, and for the distance from the gingival margin to the bottom of the crevice. These are added or subtracted to arrive at the tooth score.

The bottom of the gingival crevice seems to be a remarkably stable indicator of the height of the adjacent alveolar bone. If acceptably accurate measurements can be made on the living person, this dimension might be as informative as the X-ray films regarding the gross amount of bone remaining, and might give an even better description of its architecture. Consistent measurements with the probe require considerable skill on the part of the examiner.

The first three grades on the Ramfjord scoring scale refer to gingivitis, which is a reversible sign. The last three grades refer to depth of the gingival crevice below the cemento-enamel junction. This is irreversible to all intents and purposes; highly-successful treatment, for example, could be expected to make no change at all in the scores of most patients.

The potential value of the Ramfjord Index is great enough to warrant giving it adequate trials. A series of crevice measurements compared with independent readings of radiographs for the same patients would clarify the relation between these two types of estimation, and indicate whether the six teeth chosen are fairly representative of the whole mouth, particularly in adults with extensive tooth loss. Tests of comparability between

examiners should be made with adequate numbers of patients under field conditions, after the examiners have had enough experience with the method to overcome the "learning" effect.

If proved valid within reasonable bounds, the index would furnish an estimate of bone loss in younger individuals such as is not provided by the index of gingival recession, and would be usable in field situations where X-ray examination is impracticable.

4.3.2 *Periodontal disease rate*

Recently an index has been suggested called the "periodontal disease rate". After examination, the number of teeth affected with periodontal disease in a given mouth is determined and computed as the percentage of all teeth present in that mouth. This is taken as the score for the individual. The group score is the mean of the scores for the individuals comprising the group. It has been reported that about the same conclusions were reached with this method as when the same patients were scored under PI.

The value of this finding as an indicator of total status rests upon the relation between the severity of disease and its extent throughout the mouth.

A tooth is scored as positive for periodontal disease if there is (a) gingival necrosis, or hypertrophy, or inflammation encircling the tooth, or a purulent exudate from the gingival crevice; or (b) a gingival crevice having a depth of 3 mm or more; or (c) tooth mobility greater than 1 mm in any direction; or (d) radiographic evidence of resorption of alveolar bone extending more than 3 mm apically from the cemento-enamel junction. In this form the index includes both reversible and irreversible signs, and requires the use of both the pocket probe and X-ray equipment.

Advantages of this index are simplicity in summation, and the expression of disease prevalence in readily-understandable terms.

4.4 **Auxiliary information**

The interpretation of data gathered by any of these indices is often facilitated if other pertinent information can be considered at the same time. Data on mean numbers of missing teeth by age, and sometimes by tooth type, and the percentage of the population which is edentulous, may be an essential complement to data on disease prevalence.

The relation between periodontal disease and calculus or oral debris is so marked and uniform that no consideration of other factors can be undertaken unless the effect of these conditions can be held constant in some manner. To this end some evidence of their prevalence or extent must be considered. In populations where a substantial number of mouths are free from debris or calculus, simply noting the presence or absence of

these factors may give information adequate for group comparisons. When debris and calculus are found in practically every mouth examined, it is necessary to resort to some estimate of their relative extent or severity. One such measure is the Oral Hygiene Index (OHI).

For this index the condition of the mouth is judged by the condition of six tooth surfaces. Examination is made of two incisors and of the first fully erupted teeth distal to the second bicuspid on each side of each arch. These are ordinarily first molars. The buccal surfaces of the upper molars, the lingual surfaces of the lower molars, and the labial surfaces of the upper right central and lower left central incisors are included in the survey.

Oral debris is detected by running the side of an explorer across the surface being examined. The area is scored 0 if no debris or stain is present, 1 if soft debris is found covering not more than the gingival third or if extrinsic stain is present, 2 if soft debris covers from one-third to two-thirds of the tooth surface, or 3 if more than two-thirds of the surface is covered. The surface is considered to encompass half the circumference of the tooth.

Calculus is scored on the same scale: 0 if no calculus is present; 1 if supragingival calculus covers not more than one-third of the tooth surface; 2 if supragingival calculus covers between one-third and two-thirds of the surface, or if individual flecks of subgingival calculus are present; and 3 if supragingival calculus covers more than two-thirds of the tooth surface, or if a continuous, heavy band of subgingival calculus surrounds the cervical portion of the tooth.

The score for the individual is the average score for the surfaces examined. It may be calculated separately for debris or for calculus, or as a composite score for the two.

The routine examination procedure of the Ramfjord Index includes a series of indices which are supplementary to the periodontal disease score. Occlusal and incisal attrition, tooth mobility, lack of contact, plaques (after use of a disclosing solution), and calculus, are scored on specified scales running from 0 through 3, corresponding in general to the clinical descriptions of none, mild, moderate and severe.

4.5 Discussion

While many other devices have been employed in reports of population findings of periodontal disease, they are, for the most part, arithmetical or statistical variations of the indices discussed here.

All these measures are amenable to analysis by modern statistical methods. This is one of the minimum requirements for a population index, because sound statistical design should be followed in every field study.

In young children, all the indices are measures of simple gingivitis and the choice of index depends upon the object of study. The more stringent

is the definition of normal used, the more useful is an index like P-M-A in separating out individuals with complete freedom from disease. The difference between the absolute normal and persons with disease is accentuated by such devices as taking the worst possible score for a quadrant or other anatomical area. Improvement in sensitivity at this level of disease, however, is compensated for by a loss of sensitivity at higher levels; such devices frequently obscure very real differences between moderate and severe stages of disease. Where distinctions of this sort are required, an index such as PI is more appropriate.

The simplest measure to apply under primitive conditions is P-M-A. The examination can be made with the tongue blade without contamination of the examiner's fingers. Hand instruments with minimal provision for sterilization are adequate for PI, the Ramfjord Index, and the count of teeth involved in gingival recession.

If X-ray equipment is not available on the spot, it may be very difficult to set up, and not all fixed equipment is fully dependable under field conditions. X-ray indices take several times as long as other indices to determine. On the other hand, X-ray examination is the best means of detecting periodontitis in incipient stages or in very young individuals. The count of teeth involved in gingival recession is least sensitive to incipient disease. The count of overt pockets in PI is less sensitive than radiographic examination for the detection of incipient disease. As far as the Committee was aware, the sensitivity of the Ramfjord Index to incipient disease has not been determined.

All these measures are subject to error. Error that is random and equally distributed about the true finding is readily compensated for by increased sample size. Compensation is not possible for "learning effect", or the gradual shift toward higher or lower estimates with time and experience. This effect tends to be greatest in those procedures requiring the most skill, much accessory equipment, or the greatest judgement on the part of the examiner. It can be controlled with X-ray films, provided that the readings are all made at one time at the conclusion of a study, rather than at intervals during its course.

Estimation of bone resorption from X-ray films is facilitated by use of a measuring device scored into equal divisions in such a manner that resorption may be estimated to the nearest tenth of the tooth root regardless of the length of its shadow. When such a device is employed reading error is reduced in magnitude and tends to be random about the true value.

The principal use of a population index is to provide comparison between two or more population groups, or subgroups within a population, or between group status at two or more points in time. Of the indices described, comparability between independent examiners is best with the count of teeth showing gingival recession. Comparability between examiners can be very high in the PI examination and in reading X-ray films, if they are

first calibrated against each other. Comparability between examiners for the Ramfjord Index, and between radiographs produced by different techniques, remains to be determined.

Any of these indices should provide useful information when applied in appropriate situations by competent examiners. The choice of method should largely be dictated by the type of information desired and the conditions under which the survey has to be made.

Because of its simplicity and ease of application and the comparability of the findings, the PI index is recommended as the basic measure for determination of relative group prevalence. Where additional information is required other indices may be added to the examination procedure as appropriate. Every effort should be made to develop indices for such factors as traumatic occlusion for which no population data are at present available.

5. GEOGRAPHICAL DISTRIBUTION AND EPIDEMIOLOGY OF PERIODONTAL DISEASE

Prevalence estimates for periodontal disease have been based either upon the proportion of persons showing some deviation from an ideal condition of the tissues, or upon the proportion exhibiting gross and radical tissue destruction. When the former criterion is applied to North American populations, the invariable finding is that nearly every adult shows some sign of periodontal disease. When the second criterion is applied, there is substantial agreement that about half the adults who still retain some teeth at the age of 50 years show evidence of gross and extensive tissue destruction.

Survey data gathered by the American Dental Association indicate that, in men over the age of 35 and in women over the age of 40 years, periodontal disease is responsible for between two and three times as many extractions as dental decay.

Where quantitative comparisons can be made from available data using North American findings as a measuring rod, both the prevalence and severity of periodontal disease seem, with few exceptions, to be as great or greater in other parts of the world.

5.1 Periodontal disease throughout the world

India and Asia. On the basis of radiographic examination, police constables in India seem to exhibit more extreme bone resorption than males of the same ages in Boston. The clinical condition of boys in Bombay was judged to be significantly worse than that of boys in Atlanta, Georgia. Periodontal disease was held responsible for the loss of 79% of teeth

extracted for persons older than 30 years in Bombay. By clinical standards, national servicemen in Singapore (including Chinese, Malays, Pakistanis and Indians) showed slightly more disease than the Indian boys mentioned above. Advanced disease was reported as affecting about half the adults surveyed in Chieng Mai province of Thailand. Even higher disease levels were reported from Viet Nam, particularly in tribesmen of the hill areas.

Pacific area. Moderate to extensive bone loss has been reported in natives of New Guinea, Polynesians on Puka Puka atoll, and villagers on Raratonga. The general picture is one of a widespread prevalence of destructive disease.

Africa. In surveys of schoolchildren in Egypt, disease levels were found comparable with those of Indian boys as contrasted with very low levels in the Sudan. In Ethiopia the prevalence reported was midway between that for Egypt and that for the USA. A high prevalence has been reported for children in Nigeria.

Eastern Mediterranean. Scores nearly as high as those in Egypt were reported from Syria, and scores nearly as low as those in the Sudan from Iran.

South America. Peruvian servicemen showed about the same levels of disease as servicemen in Singapore. Ecuadorian servicemen showed somewhat less disease, while among civilians in that country the prevalence seemed comparable to that among civilians in Ethiopia.

Europe. Bone resorption in Oslo industrial workers was much the same as, or slightly higher than, resorption in Boston males, but was distinctly less than in Indian police constables.

5.2 Variability of populations

Because of the extreme variations observed within and between populations none of these reports, describing the condition of specific groups, may safely be taken as indicative of the status of a whole nation or region. Much more information must be gathered before such generalizations are justified.

5.3 Epidemiological characteristics

All epidemiological reports agree that the number of persons with gross and destructive disease increases systematically as the population ages, and that this increase in the number of persons affected is accompanied by an increase in the average severity of disease as well. There is similar agreement that the onset of the disease may be as early as puberty and that many adolescents show extensive destruction of tissues before the age of 20 years.

The true meaning of some associations has not been established. Some ancient Eskimo skulls show evidence of extensive disease ; in others such signs are absent. Among living Eskimos, some have been found quite free from disease ; others no less primitive, eating the same foods and living, in fact, in adjacent villages, showed a high prevalence of destructive disease. It seems unlikely that ethnic or dietary factors are responsible for these differences.

Variations in oral hygiene practices or tobacco consumption have been shown to be the basis of the differences found among European industrial workers, and differences in mouth cleanliness fully explain crude differences between Negro and white persons in the USA. It may eventually be demonstrated that similar differences in habits provide the true explanation of the differences in disease between other population groups. These are the most consistent and convincing associations so far developed in epidemiological research. Every observer who has taken note of calculus or oral debris in any way has reported at least a qualitative relation with the levels of periodontal disease in the groups he has studied.

5.4 Summary

(1) In the USA some degree of periodontal deterioration can be demonstrated in virtually every adult. Gross tissue destruction can be seen in about half of all persons studied in middle age or later. At these ages periodontal disease is responsible for the extraction of two or three times as many teeth as is dental caries.

(2) Where comparisons have been possible, few have indicated any lower prevalence in other parts of the world. In most areas, and particularly in Asia, disease seemed to be much more prevalent and severe than in the USA.

(3) The onset of disease may be as early as puberty in susceptible individuals. In population groups both the prevalence and severity of disease increase with increasing age.

(4) While other factors may be responsible for some observed differences between populations, within populations a positive correlation is invariably found between mouth cleanliness and the health of periodontal structures.

6. PREVENTION AND TREATMENT OF PERIODONTAL DISEASE

As periodontitis leads to permanent destruction of the supporting structures of the teeth, its prevention is probably more important than that of those diseases in which repair can take place. Periodontal disease is a product of (1) predisposing local factors, (2) systemic factors, and

(3) exciting local factors. Any step should be taken that can improve periodontal health by changing these factors.

6.1 Control of predisposing local factors

Any local or systemic condition interfering with the growth and development of the periodontium is liable to predispose to periodontal disease. Therefore all measures which contribute to the normal development of these structures will contribute to the prevention of periodontal disease.

Chronologically, the first factor to be considered is a balanced diet for the pregnant woman. In this sense different factors may play an essential role, as for instance: (a) a sufficient supply of proteins of good quality, (b) a correct quantity and ratio of mineral salts (especially calcium and phosphorus), and (c) an adequate intake of vitamins.

Secondly, the same considerations must also be given to the diet of the growing child. Particularly, a lack of vitamin D interferes with the growth and development of the jaws and teeth, but other vitamins may also be involved.

6.2 Systemic factors

The Committee agreed that most of the systemic factors in periodontal disease are unknown. However, it is known that vitamin A plays a role in the metabolism of epithelium and indirectly in resistance against infection. Some of the components of the vitamin B complex are of importance for the resistance both of epithelium and connective tissue.

Vitamin C is essential for the formation of collagen. An adequate intake of vitamins should therefore be advocated whenever a deficiency is suspected.

In some economically less-developed countries correction of the diet, including vitamin deficiencies, may be a valuable adjunct in the preventive work. In the economically more-developed countries most people have an adequate diet as far as vitamins and nutrients are concerned.

6.3 Local exciting factors

It was agreed by the Committee that bacterial plaque and calculus are the most common direct causes of periodontal disease. Any measure able to prevent the formation of such deposits should be undertaken.

6.3.1 Control of local factors by the individual

Consistency of foods. Raw and fibrous foods help to clean the teeth and gingiva during mastication, and should be introduced into the diet at as early an age as possible.*

* This recommendation is not, of course, intended for areas where contaminated raw foodstuffs may present a grave public health problem.

Tooth-brushing. Brushing the teeth and gums, using a suitable technique, immediately after every meal, or at least twice a day, is an important part of the treatment and prevention of periodontal diseases. The aims of tooth-brushing are : to remove plaque from the teeth, gingival margins and gingival crevices, and, in doing so, to prevent calculus formation and to stimulate the blood supply and increase keratinization of the gingiva by massage.

Mouth-rinsing. Thorough rinsing with plain water helps to remove any debris loosened by the tooth-brushing. The addition of some weak antiseptic or flavouring agent may make this procedure more acceptable but not more efficacious.

Interdental cleaning. Debris between the teeth can seldom be removed by tooth-brushing or mouth-rinsing. In children where the interdental papillae are usually still firmly adapted to the teeth, special interdental cleaning is rarely necessary. However, in older children and adults when there is evidence of inflammation, use of dental floss is indicated. In persons with recession of the papillae, interdental cleaning is always indicated and it is probably the most important part of tooth cleaning. Special tooth-brushing methods can be used, but in most cases such aids as interdental sticks, elastic bands, or dental floss are indispensable.

6.3.2 Control of local factors by the dentist

Ideally, from an early age every person should have regular and periodical oral examination, preferably twice a year, for checking whether any disease is present, for treatment if necessary and for ascertaining the effectiveness of oral hygiene habits.

Oral prophylaxis. Even when thorough tooth-cleaning is maintained by the patient there will usually be regions in the mouth where the cleaning is not sufficiently effective. Regular visits to the dentist for oral prophylaxis are therefore necessary. Any deposits of calculus, supragingival as well as subgingival, should be removed with proper instruments. Particular emphasis should be given to the interdental areas. Attention should also be paid to the fact that not all accretions are calcified to form calculus.

Malocclusion. The prevention or correction of malocclusion will help to prevent periodontal diseases by : (a) eliminating gross stagnation areas, (b) providing sufficient room between the teeth for well-formed interdental papillae, (c) preventing food-packing, (d) making a lip-seal possible, (e) preventing direct damage to the gingiva by teeth of the opposing jaw, (f) preventing occlusal trauma.

Proper restorations that maintain the normal contour of the teeth and provide adequate contacts between them are important in preventing localized periodontal disease. Every care should be taken that overhanging

margins are avoided or corrected, and that removable appliances are carefully designed to prevent periodontal damage.

6.4 Prevention—a personal responsibility

Although all the above-mentioned measures may contribute to the prevention of periodontal disease, they are not equally applicable in all areas and to all individuals.

Changing dietary habits is a slow process, even in countries where all the prescribed nutrients are available and within economic reach of most people.

Treatment of malocclusion may be feasible in most cases in some countries but in others such treatment cannot be provided.

Unfortunately there is no public health measure, like fluoridation or vaccination, that can be applied for the prevention of periodontal disease. The only measure that is within reach for practically all people is improvement in oral hygiene. Systematic efforts to improve oral hygiene will, therefore, probably be the public health measure that will give the greatest improvement. Thus, each individual holds the key to his own periodontal health. It is the responsibility of the dental health service and the dental profession to arouse awareness of this problem in the individual.

6.5 Treatment of periodontal disease

In the treatment of all types of inflammatory periodontal disease, thorough scaling and polishing of the teeth and the maintenance of perfect oral hygiene by the patient are the fundamentals of treatment. However, additional procedures are often indicated, e.g., root and soft tissue curettage; gingivectomy; gingivoplasty; flap operations; frenectomy and mucobuccal fold extension; selective tooth grinding; and the fitting of temporary, removable or fixed splints, or other appliances.

7. ROLE OF THE DENTAL HYGIENIST IN THE PREVENTION AND CONTROL OF PERIODONTAL DISEASE

The WHO Expert Committee on Auxiliary Dental Personnel defined the functions of a dental hygienist as follows:¹

“Persons in this category should work under the close supervision and direction of a dental practitioner. For certain public health aspects of the work, he or she may have some independence of action provided that this is carried out as part of the public health team work. The Committee

¹ *Wld Hlth Org. techn. Rep. Ser.*, 1959, 163, 13

was satisfied that dental hygienists are most valuable in the field of preventive dentistry and by virtue of the practice of simple dentistry can extend the benefits of oral health to larger sections of the community. It was also considered by the Committee that recruitment of dental hygienists need not be limited solely to females but that male dental hygienists might also be employed where adjudged necessary.

“ An enumeration of the functions of a dental hygienist would include :

- (1) The cleaning of teeth.
- (2) The removal of calculus.
- (3) Individual and group instruction in oral hygiene.
- (4) The cleaning of mouths, on the orders of the dentist, before treatment is instituted.
- (5) The topical application of fluorides or other prophylactic solutions.
- (6) The screening or primary examination of groups, such as school-children or factory employees, for dental defects, in order that they may then be referred to qualified dentists for treatment.
- (7) In the case of schoolchildren, liaison with local public health nursing services to ensure effective follow-up of recommendations or treatment. (To make this follow-up truly effective, public health nurses should have some elements of instruction in dental hygiene and the part played by diet in relation to dental disease included officially in their training curriculum.) ”

7.1 Prevention of periodontal disease

The most important local etiological factor in nearly every mouth is irritation from bacterial plaque, part of which is often calcified as calculus. The widespread use of dental hygienists would appear to be a practical and relatively inexpensive method of controlling this factor.

Periodontal disease is rare in children before the eruption of the permanent dentition but often appears with the eruption of the permanent incisors. Thus, for the prevention of periodontal diseases alone, it should not be necessary for the dental hygienist to see children under six years of age. For the inculcation of good oral hygiene habits, however, instruction of younger children, say from the age of two years, is desirable. From school age, the following services can be performed by the hygienist.

7.1.1 Group instruction in oral hygiene

Instruction of classes of schoolchildren in the technique of oral hygiene, with the aid of models, slides and films can be quite effective. It would probably be still more effective if the parents could also be given instruction.

Tooth-brushing and mouth-rinsing are the only forms of artificial oral hygiene that should be taught to groups of children.

Advice on the types of food that are good for gingival health, and when to eat them, can be given at the same time. In children, the natural form of oral hygiene resulting from the chewing of raw, cleansing foods at the end of meals has been shown to improve gingival health.

7.1.2 Individual instruction in oral hygiene

This is more effective than group instruction but is probably necessary only for those children in whom group instruction has failed to produce a clean mouth. Such children would be referred to the dental hygienist by the dentist making routine inspection for caries, periodontal disease or malocclusions. In the event of such an inspection service not being available, the hygienist should inspect the children's mouths in order to assess the oral hygiene and general oral health. In selected cases, the use by the hygienist of a disclosing solution and a hand mirror can have a big effect on intelligent children. Checking on their performance with their toothbrush and also in rinsing their mouths thoroughly afterwards is often salutary.

7.1.3 Scaling and polishing the teeth

Scaling is usually not necessary until adolescence or adult life. Regular scaling then constitutes an important preventive measure. Polishing to remove stains on children's teeth is a vital adjunct to oral hygiene instruction when accompanied by advice to maintain the teeth in sparkling condition.

7.1.4 Reference back to dentist

If, on follow-up, the hygienist finds that the gingival inflammation is still present in spite of the apparent co-operation of the patient in regard to oral hygiene, the patient should be referred to a fully qualified dentist or periodontal specialist for diagnosis and treatment plan.

7.2 Control of periodontal disease

The dental hygienist can play an important role in the control of periodontal disease. Every case of periodontal disease requires thorough scaling and polishing of the teeth.

7.2.1 Supragingival scaling and polishing

The removal of supragingival calculus is an important part of the control of all types of periodontal disease. This scaling and the subsequent polishing can be done as efficiently by a dental hygienist as by a fully trained dentist.

7.2.2 *Subgingival scaling and polishing*

The removal of subgingival calculus is essential in the control of chronic periodontal disease. The dental hygienist can become extremely proficient in this tedious and time-consuming procedure. As many etiological factors, both local and general, may be involved, the treatment of cases with subgingival calculus should be under the supervision of a dentist or periodontal specialist. Subgingival scaling by the dental hygienist should be directed to the removal of deposits on the teeth, and the planing and polishing of cementum, but should not include curettage of soft tissues of the periodontal pocket.

7.2.3 *Application of drugs*

Astringent antiseptic drugs may be applied routinely by the hygienist after scaling. Treatment of acute conditions such as acute ulcerative gingivitis (Vincent's disease) should be under the direct supervision of the dentist.

7.2.4 *Oral hygiene instruction and tooth-brushing*

(a) *Tooth-brushing (or mouth-brushing)*. The particular technique of tooth-brushing most suitable to the case will depend on the periodontal disease present and should be prescribed by a dentist.

(b) *Interdental cleansing*. Regular cleaning of the teeth interdentally is most important in the control of established periodontal disease but can cause damage to the normal interdental gingiva. The technique to be taught to each patient by the hygienist should be prescribed by a dentist. It may be aimed not only at improving interdental cleansing but also at re-shaping the interdental "col" and perhaps at reducing the depth of the pocket by the application of intermittent pressure to the gingiva.

8. PERIODONTOLOGY AND DENTAL EDUCATION

The responsibility for introducing and improving prevention and treatment of periodontal disease rests with the dental profession. Only the dental profession can evaluate the impact of this disease on the individual and on the community, and only the dental profession can advise the health services how to manage this problem on a broad basis. Finally, the treatment has to be carried out and individual instruction in prevention has to be given by the dentist. Is the dental profession educated to do all these things? The answer will probably be that it is not.

In countries where the main principles of dental education have evolved, the major dental health problem was originally caries and its immediate

effects. As a consequence, training was concentrated on operative and prosthetic dentistry. This training may have been adequate half a century ago. Today, in some countries, caries has to a certain extent been brought under control and a new problem has arisen—the need to save teeth from the ravages of periodontal disease in later life. In most cases, this change in the type of dental treatment needed has not been met by a corresponding change in the curriculum of the dental schools. In one country (USA) from which data are available, the total time for the teaching of periodontology in the curriculum averaged 83 hours in 1958. This was only 2.1% of the total curriculum. Prosthodontics and operative dentistry together received about 22 times as much attention as periodontology. In the same country, a committee of teachers in periodontology considered that a minimum requirement of 350 hours, or a four-fold increase, was necessary for the adequate teaching of this subject.

It is quite obvious, therefore, that the dental curriculum must be radically altered in most countries before the dental profession can be educated to control periodontal disease in a satisfactory way.

In some countries periodontal disease poses a greater problem than caries even in young people; where this is so due consideration should be given to this situation in the training of the dental students.

In many dental schools there are no departments of periodontology, which may be given more or less attention in some other departments as a minor subject. Thus it seems highly desirable that separate departments and chairs of periodontology be established in all dental schools and that increased time be devoted to this field in both clinical and preclinical courses.

9. PUBLIC HEALTH ASPECTS OF THE CONTROL OF PERIODONTAL DISEASE

Whether periodontal disease is a manifestation of general disease or is itself the cause of other diseases, it must be given attention in any public health programme. Such attention should cover the maintenance or, if necessary, the restoration of the healthy functioning and appearance of the oral cavity and its associated parts.

9.1 Dental health education

As periodontal disease is incurable in its late stages, and as simple treatment can only be effective when it is instituted early, every endeavour should be made to educate the public and draw attention to the need for prevention and early care. Education should start as part of the training programme given to school-teachers, especially those of the elementary and intermediate schools. It should also be given in some detail to all

health personnel and particularly to those concerned in any way with school health. All the known audio-visual means for educating the public should be used. Programmes for instruction in oral hygiene should be developed for the schools and carried out in hygienic conditions under the supervision of teachers and health visitors. There should be facilities for supplying toothbrushes and adjuncts such as toothpastes and mouthwashes.

Advice and talks to the students and their parents during the meetings of the parent-teacher associations are of great value.

Health education programmes should aim to rouse public consciousness to the facts that periodontal disease affects general health, that it is similar to other health problems which require early recognition and treatment, and that prevention is much more to be desired than treatment, which is time-consuming and costly.

9.2 Dental health services

A dental health service must be an integral part of any public health administration and must receive appropriate consideration in the development of the national health service. Programmes should therefore be developed for the establishment of dental health services, including the provision of personnel needed for the prevention and treatment of periodontal disease.

In countries with well-developed dental health services, this problem should be considered in future planning. Where dental treatment is paid for either by the state or by health insurance companies, adequate remuneration should be provided for preventive and curative treatment of periodontal disease. In countries where periodontal disease is the main problem, the attention of the dental health service should be directed towards its control from the beginning.

Most of the preventive work could be done by dental hygienists trained to carry out periodical prophylactic treatment. The use of these dental hygienists saves both time and money and they can be attached to nurseries, child welfare centres, schools, factories, dental clinics in hospitals, and the private clinics of dental surgeons.

9.3 Management of periodontal disease

Periodontal disease is an important public health problem but it is not a spectacular one. It does not usually affect very young persons, nor does it as a rule cause pain or death ; but it does make life miserable for many middle-aged and old people. However, consideration must be given to all age-groups by the health services.

It was the unanimous opinion of the Committee that prevention is the method of choice in the management of periodontal disease and that

preventive measures should be instituted early in life before serious damage and loss of teeth have been caused.

10. RESEARCH

Among problems that may be of direct interest to WHO there are several the elucidation of which could greatly advance understanding of periodontal disease. Of these, the question of racial incidence is a conspicuous example. It is, of course, difficult to view the effect of racial factors without the intrusion of geographical, socio-economic or dietary influences. Nevertheless, there remains considerable evidence for the existence of certain undetermined factors as a result of which periodontal disease is more prevalent in some ethnic groups than in others. There is no doubt that the resolution of these problems would broaden our understanding of etiological factors and contribute to improved methods of therapy as a consequence.

Since a study of this nature calls for epidemiological investigations on a comparatively large scale, it would be desirable to devote attention to other important factors that may have a bearing on the etiology and pathogenesis of periodontal disease and that could be assessed concurrently, such as nutrition, dietary habits and systemic disease.

It is suggested that WHO is suitably placed to sponsor investigations of this type and it is therefore recommended that WHO conduct and stimulate long-term (i.e., longitudinal) studies in areas where the prevalence and severity of periodontal disease are unusually high or low, and where a variety of possible etiological factors are available for study.

11. CONCLUSIONS

The Committee decided that the following four main principles are basic to the control of the world-wide problem of periodontal disease :

(1) continued efforts to gather more information about the basic nature of the disease and its prevalence in populations throughout the world ;

(2) health education to convince people that pain and loss of teeth from periodontal disease is not inevitable and to inform them about hygienic and other preventive measures of proved efficacy ;

(3) expansion and improvement of instruction in methods of treatment given in dental schools ;

(4) reorientation of public dental health services to give prevention and control of periodontal disease an adequate place in all future programme planning.

Annex

LIST OF WORKING PAPERS *

- WHO/DH/32 Biochemical aspects of periodontal disease, by S. D. Schultz-Hautt
- WHO/DH/33 Indices for recording periodontal disease, by A. L. Russell
- WHO/DH/34 The geographical distribution and epidemiology of periodontal disease, by A. L. Russell
- WHO/DH/35 The education and functions of the dental hygienist in the prevention and treatment of periodontal disease, by H. A. Zander
- WHO/DH/36 The role of local factors in the etiology of periodontal diseases, by R. D. Emslie
- WHO/DH/37 The role of the dental hygienist in the prevention and treatment of periodontal disease, by R. D. Emslie
- WHO/DH/38 The education and functions of the specialist dental practitioner in the treatment of periodontal disease, by H. A. Zander
- WHO/DH/39 The education of health personnel and the general public in the prevention of periodontal disease, by S. Sweilim
- WHO/DH/40 The role of the general dental practitioner and the periodontal specialist in the clinical control of periodontal disease, by J. Kostlán
- WHO/DH/41 The anatomy, histology, biology and histopathology of the periodontium and associated structures, by J. Waerhaug
- WHO/DH/42 Avenues for future research in periodontal disease, by B. Cohen
- WHO/DH/43 Memorandum on periodontal disease, by P. O. Pedersen
- WHO/DH/44 Undergraduate periodontology (précis of *Proceedings of a workshop for teachers in periodontology*, ed. Harold G. Ray)
- WHO/DH/45 Periodontal diseases: definitions, classification, nomenclature, by A.-J. Held
- WHO/DH/46 The importance of early dental care programmes in the prevention of periodontal disease, by K. L. Shourie
- WHO/DH/47 Etiology of periodontal disease, by F. A. Carranza

* A limited number of mimeographed copies of these papers are available for distribution and can be obtained free of charge on application to the Chief Dental Health Officer, World Health Organization, Palais des Nations, Geneva.