



THE UNIVERSITY OF
SYDNEY

COPYRIGHT AND USE OF THIS THESIS

This thesis must be used in accordance with the provisions of the Copyright Act 1968.

Reproduction of material protected by copyright may be an infringement of copyright and copyright owners may be entitled to take legal action against persons who infringe their copyright.

Section 51 (2) of the Copyright Act permits an authorized officer of a university library or archives to provide a copy (by communication or otherwise) of an unpublished thesis kept in the library or archives, to a person who satisfies the authorized officer that he or she requires the reproduction for the purposes of research or study.

The Copyright Act grants the creator of a work a number of moral rights, specifically the right of attribution, the right against false attribution and the right of integrity.

You may infringe the author's moral rights if you:

- fail to acknowledge the author of this thesis if you quote sections from the work
- attribute this thesis to another author
- subject this thesis to derogatory treatment which may prejudice the author's reputation

For further information contact the University's Copyright Service.

sydney.edu.au/copyright

PREVENTION AND CONTROL OF
PERIODONTAL DISEASE

S. KILISIMASI, B.D.Sc. (Qld.)

A Thesis submitted in partial
requirement for the
DIPLOMA IN PUBLIC HEALTH / DENTISTRY

Department of Preventive Dentistry
Faculty of Dentistry
University of Sydney

1967

ACKNOWLEDGMENTS

In this venture I have relied on the example and writings of the many workers in this field. I should like to acknowledge my debt to all of these, and particularly to Mr. S. Levine, M.D.S. (Sydney), Lecturer in Periodontics whose writings first stimulated my interest in periodontics.

I should like to express my gratitude to my mentor Mr. P. D. Barnard, M.D.S., M.P.H., F.A.C.D.S., F.A.P.H.A., Senior Lecturer in Preventive Dentistry, who pointed my feet in the right direction and also for encouragement and friendship that he has given me during this course.

I have much pleasure in acknowledging the expert help and advice which I have received from Professor N. D. Martin, M.D.S., F.A.C.D.S., F.A.C.D., F.I.C.D., F.A.P.H.A., and many friends and colleagues in Australia.

TABLE OF CONTENTS

	<u>Page</u>
1. INTRODUCTION	1
2. PERIODONTAL DISEASE	4
ANATOMY OF PERIODONTIUM	4
Gingiva	4
Periodontal Ligament	6
Cementum	9
Alveolar Process	10
CLASSIFICATION AND NATURE OF PERIODONTAL DISEASE	12
Inflammatory	13
Gingivitis	14
Acute	15
Chronic	19
Periodontitis	25
Dystrophic	29
Degenerative	29
Gingivosis	30
Periodontosis	32
Atrophic	35
Recession	36
Disuse	39
Hyperplastic	41
Gingival hyperplasia	42
Traumatic	47
Traumatism	47
Primary	48
Secondary	51
SUMMARY AND CONCLUSION	53

	<u>Page</u>
3. EPIDEMIOLOGY OF PERIODONTAL DISEASE	57
Periodontal Indices	60A
Gingivitis and Periodontitis	69
Necrotizing Ulcerative Gingivitis	
Atrophy	91
Hyperplasia	93
SUMMARY AND CONCLUSION	101
4. PREVENTION AND CONTROL OF PERIODONTAL DISEASE	107
INDIVIDUAL	109
DENTIST	112
AUXILIARY	115
SPECIALIST	121
COMMUNITY	122
SUMMARY AND CONCLUSION	135
5. TONGAN COMMUNITY	138
6. SUMMARY AND CONCLUSIONS	148
7. REFERENCES	151



INTRODUCTION

In medicine the term "disease" is not confined to the infectious diseases, but is applied to any deviation from the normal. Thus periodontal disease is any abnormal state of the periodontium. Such deviation may be either developmental, that is a growth anomaly, or it may be acquired, that is due to some environmental factor.

Like all other tissues the periodontal tissues are subjected to a number of pathological changes: inflammatory, dystrophic and traumatic.¹¹² The disease process may be restricted to the gingivae or may involve the deeper periodontal structures.

Disease of the periodontal tissues is so very common in civilized races that there are few adults who fail to exhibit evidence of either an active lesion or the ravages produced by a previous disease process.¹⁶⁵ Periodontal disease is not, however, a disease of civilization and there is little doubt that the investing tissues of the teeth have been subject to disease from time immemorial. On literatures it shows that periodontal disease has existed from the

earliest times and historical records show that there was an awareness of this disease and need for its care. Merritt¹⁰⁵ described it as the commonest of all the diseases amongst Egyptians 4,000 years ago. Evidence¹⁶¹ of destruction of the bone of the alveolar process is present in the earliest skulls which have been found, and that of Rhodesian Man, 3,000 B.C., contains the vacuity once occupied by a periodontal abscess. Weinberger¹⁶¹ describes the Babylonians and Assyrians had suffered from periodontal conditions and they had practised the use of some medicinal substances for mouth washes and local applications. He¹⁵ also found out from the oldest Chinese medical work written about 2,500 B.C., there is a description of gingival inflammation, periodontal abscess and ulceration.¹⁶¹ Hippocrates of Cos in 335 B.C. described the causes of periodontal disease and he believed that the cause was due to deposition of pituita or calculus. In Rome, Aulus Celsus (1st Century A.D.) mentioned periodontal diseases affecting the soft parts of the mouth. Chewing of unripe pears and apples was the treatment he recommended. Amongst the Arabians in 850-923 Rhazes recognized this disease and advised the use of honey and opium for its treatment. Pierre Fauchard (1678-1761),⁵⁴ the father of modern dentistry, had written a lot about periodontal disease. Chronic periodontitis was for a long time known as

Fauchard's disease. He emphasized the need to keep the teeth clean if disease were to be prevented.

The development of modern periodontology began in the last century when John Riggs recognized the great importance of subgingival calculus in the causation of chronic periodontitis. He curetted the subgingival calculus and polished the root surface. He taught his patients how to brush their teeth with a rotating motion. Before the end of the last century the value of regular prophylaxis in the prevention of periodontal disease was being recognized and practised.

The aim of this Thesis is to review the literature of the Anatomy, Classification and Nature of Periodontal Disease, its Epidemiology, Control and Prevention and to specially consider the application of preventive and control measures found in the literature to the Tongan community.

PERIODONTIUM

An appreciation and understanding of periodontium in a state of health is essential before proceeding to study the diseases of it. Without such knowledge the processes and the principles of treatment cannot be fully comprehended nor suitable preventive measures instituted.

Periodontium has been defined by Sidney Sorrin¹⁴⁶ as the tissues in immediate proximity to, and surrounding, the tooth including cementum, periodontal membrane, alveolar bone and gingiva. In the past, there has been a tendency to study the four components of the periodontium as separate tissues and so fail to appreciate that they form a biological unit within the body. Each of the four components of periodontium is concisely described:

1. THE GINGIVA

The gingiva in health is pink, firm, knife-edged, and scalloped to conform to the contour of the teeth. The colour of the gingiva may vary slightly, and in dark-skinned individuals physiological melanin pigmentation may be found as diffuse brown or grey-blue patches. The interdental papilla

completely fills the space apical to the contact area between the teeth, and its interproximal shape conforms to that of the contact area thus forming the interdental col. The shape of the papilla varies slightly according to the shape and relationship of the teeth, and its surface is grooved by an interdental sluiceway. The gingiva may be divided into the marginal gingiva and the attached gingiva.^{146, 112}

The Marginal Gingiva forms a tight collar approximately 1 mm. wide around the neck of the tooth. It is pink, firm and smooth, and as it is the soft-tissue wall of the gingival crevice it may be separated from the tooth by the insertion of a blunt probe. It is normally separated from the tooth surface by a shallow gingival sulcus of approximately 1 mm. deep. At the base of the crevice the x is attached to the tooth at the amelocemental junction.

The Attached Gingiva joins the gingival margin at a shallow groove, the free gingival groove, and extends to the muco-gingival junction where it joins the alveolar or vestibular mucosa. Unlike the gingival margin, which is protected from functional irritation, the attached gingiva is a food-shedding surface. It is pale pink, firm and stippled, and its surface of well keratinized epithium is tightly bound down to the underlying periosteum. In health it is

clearly demarcated from the alveolar mucosa which is loosely bound down to the underlying tissues and is very vascular.¹⁴⁶

The Gingival Sulcus. This is the space between the unattached free gingiva and the tooth. It is bounded on one side by the dental cuticle and on the other side by the epithelium of the sulcus. Its usual depth is 1 to 2 mm. Noye said this has a significance because the initial stages of periodontal disease usually commence here. The gingival sulcus is merely a shallow groove, the bottom of which is at the point of separation of the attached epithelium from the tooth.¹¹²

2. PERIODONTAL LIGAMENT

This ligament (alveolodental) is a dense, regular connective tissue attaching the tooth to the alveolar bone. Its primary function is to support the tooth in its alveolus and to maintain the physiologic relation between the cementum and bone. The alveolodental ligament attaches the tooth to the bone of the alveolus. It consists of five groups of bundles:^{54, 113}

- (a) Alveolar crest group: The fiber bundles of this group radiate from the crest of the alveolar process and are attached to the

cervical part of the cementum. This is the smallest group of principal fibers.

- (b) Horizontal group: The bundles run at right angles to the long axis of the tooth, from the cementum to the bone. This is the next smallest group of fibers.
- (c) Oblique group: The bundles run obliquely. They are attached in the cementum somewhat apically from their attachment to the bone. These fiber bundles are most numerous and constitute the main support of the tooth against masticatory forces.
- (d) Apical group: The bundles are irregularly arranged and radiate from the apical region of the root to the surrounding bone.
- (e) Interrandicular group: This group courses from the crest of the interrandicular septum to the furcations of multirooted teeth.

Epithelial attachment. During active eruption the tooth moves through the overlying mucosal tissue until the cells of the reduced enamel epithelium contact those of the basal layer of the oral epithelium. At this stage the outer cells of the reduced enamel epithelium and the basal cells of the oral epithelium are actively proliferating and the layers fuse.

Subsequently degeneration of the epithelial cells occurs until eventually the crown tip emerges covered only by the degenerated inner layer of the reduced enamel epithelium. This reduced enamel epithelium is now termed the epithelial attachment and is continuous with the epithelium of the gingiva.

Orban¹¹¹ describes the epithelium as a shield in biologic rather than a mechanical sense against chemical and bacterial injuries. There is a true attachment of epithelium to the tooth, although the precise biochemical nature of the epithelial attachment is still unknown. Gottlieb and others thought of the epithelial attachment as a persistence of a primary union between the ameloblasts into a reduced enamel epithelium. In contrast to the idea of the persistence of a primary attachment, it has been claimed that epithelium is attached to the surface of the enamel by a cementing substance the secondary enamel cuticle.

The length of the normal epithelial attachment of an adult tooth is about 1 mm. The distance between the apical end of the epithelial attachment and the alveolar bone crest is also about 1 mm. This represents the amount of connective tissue attachment at the dentogingival junction. Since there is continuous movement of the clinical crowns

occlusally in order to maintain contact with their anatagonists as wear takes place, the epithelial attachment also migrates apically over the root surface.

3. CEMENTUM

Cementum is a specialized, calcified connective tissue of mesenchymal origin which covers the surface of the anatomic root of tooth. Noye describes it in view of its function it becomes one of the most important of the dental tissues, for no matter how perfect the teeth may be, without firm attachment, they become useless and are soon lost. Its primary function is to attach the fibers of the periodontal ligament to the surface of the tooth shown by Stern.

The formation of cementum begins during the early stages of tooth eruption and is accomplished by differentiated mesenchymal cell, the cementoblasts. Formation of cementum is a continuous process of apposition. The surface of cementum is covered by the most recent layer which is uncalcified (cementoid) and is lined by cementoblasts.

It reacts to inflammation and functional stress, either favourably by means of hyperplasia of cellular cementum or unfavourably by means of resorption and loosening of the tooth.^{108, 113}

4. THE ALVEOLAR PROCESS

The alveolar process is that part of the jaws which supports the teeth. It is generally described as consisting of two parts; "the alveolar bone proper" which forms the socket wall, and the "supporting bone" made up of the buccal and lingual cortical together with the cancellous trabeculae between these plates and the socket walls.

Alveolar process commences approximately 1 mm. below the apical portion of the epithelial attachment (Sidney Sorrin).¹⁴⁶ The alveolar bone proper (the inner wall of the socket) is also called the lamina dura because of its appearance as a roentgenopaque line in roentgenograms. Since it is perforated by many openings for entrance and exit of blood vessels and nerves to and from the periodontal ligament, it is also called the cribriform plate. Variations in bone morphology are of significance in the progress and manifestations of periodontal disease.

The supporting bone also will adapt itself to functional requirements. Bone is resorbed if functional requirements are reduced, and additional bone is formed if functional influences demand it.

The loss of occlusal function will lead to osteoporosis,

a disuse atrophy of the supporting bone, whereas increased functional demands will produce denser bone.

Pathological processes, both local and general, may also alter bone morphology and these changes may be observed on radiographic examination. The bone in the alveolar processes is in a constant state of flux, influenced primarily by functional stimuli and also by intrinsic factors.

Proper understanding of the biologic principles of the periodontium is basic to understanding the principles of the practice of periodontics.

CLASSIFICATION AND NATURE OF PERIODONTAL DISEASES

Through the years, innumerable classifications of periodontal diseases have been suggested and no single one has received universal acceptance. Much of the confusion has arisen from a failure to agree upon a basis for nomenclature, aetiology and pathology having been used both independently and combined. Orban¹¹² says "A classification is not a permanent structure. It must be adaptable to change and discovery".

Glickman⁵⁴ based his classification on the nature of pathologic changes, aetiological factors, and clinical features. Goldman⁵⁸ and Marshall-Day⁴³ have suggested a basis of differentiation of these pathological processes, which are Degenerative, Neoplastic and Inflammatory. Caranza²³ has suggested the classification, where terminology of the periodontal disease is based on aetiological factors:

- (i) Determining factors: Inflammation, Trauma, and Combination of Inflammation and Trauma.
- (ii) Predisposing factors: A deficiency in the mechanism of bone formation, and an exaggeration in the mechanism of bone resorption.

Schour¹¹² based his classification on anatomical features of the periodontium.

Orban¹¹³ classified periodontal diseases based upon the nature of the pathological changes and clinical features according to anatomical site; it is simple and useful for everyday practice.

CLASSIFICATION OF PERIODONTAL DISEASES

<u>Inflammatory</u>	<u>Dystrophic</u>	<u>Traumatic</u>
Gingivitis	Degenerative	Traumatism
Periodontitis	Gingivosis	Primary
	Periodontosis	Secondary
	Atrophic	
	Recession	
	Disuse	
	Hyperplastic	
	Gingival Hyperplasia	

Inflammatory Diseases of Periodontium

An inflammatory process develops whenever a given factor attacks the integrity of the tissues by physical or chemico-infectious action. It is type of reaction and repair involving the non differentiated connective tissue like bone and cementum the parenchymatous tissues and the neighbouring epithelium may also undergo secondary changes.¹¹³

Nature of Periodontal Disease

Inflammatory: Periodontal disease may be inflammatory and involve only the gingiva and this is a gingivitis and may involve the papilla, the marginal area or the attached gingivae or a combination of these. The gingivitis may be discrete, diffuse or generalized throughout the mouth in varying degrees of inflammation. There may or may not be an exudate; the inflammation may be acute or chronic and both may exist together in different areas of the mouth. A discrete lesion may become diffuse and generalized and the acute stage may become chronic, and a lesion of long standing suddenly develop an acute purulent state.

Gingivitis leads to periodontal disease. Periodontal disease causes tooth loss.

GINGIVITIS

Gingivitis^{146, 58, 98} is an inflammation of the gingiva. It is only a gradual change from the clinically normal gingiva to gingivitis. Clinically, gingivitis is recognized by the usual signs of inflammation, which are:-

1. Redness
2. Swelling
3. Bleeding
4. Exudate
5. Infrequently pain

The outstanding clinical changes of gingivitis are inflammation (changes in tissue colour and form) and haemorrhage (bleeding).

The inflammation may be acute or, more often, chronic, and the clinical features may include hyperplasia, ulceration, necrosis, formation of pseudomembranes, and purulent and serious exudation. The lesions may be localized or generalized and may be distributed in the papillary, marginal or attached gingiva.

1. Acute Gingivitis

Acute gingivitis^{158, 154, 98} is usually associated with specific infection or injury. It is usually a painful inflammation arising in the papillary or marginal gingiva as a result of infection or accidental injury.

Acute inflammation of the gingiva may be caused by a variety of physical and chemical agents, by specific micro-organism, or by non-specific members of the oral flora. The outer layer of epithelium is normally well hornified and resistant to mild injury that frequently occurs in the mastication of food.

Cause. Physical and chemical agents:

1. Any hot food or drink taken injudiciously.
2. Escharotic drugs such as silver nitrate, phenol, even hydrogen peroxide used at strong dilutions and too frequently.
3. Asprin burns resulting from a misguided attempt to eradicate toothache by resting an aspirin on the gingiva.
4. Toothbrush bristle or a woodstick lacerating the gingiva.
5. Tobacco burns.

Signs and Symptoms. The patient may suffer severe pain for a short time after which the affected area is sore and extremely sensitive to any further irritation. The wound is usually seen as a bright red area denuded of epithelium and with a ragged edge of loose necrotic tissue that can be felt by the tongue. Sensitivity lasts for a few days until the wound is completely covered.

An acute gingivitis caused by any of the local factors just mentioned is a localized lesion. The vascular and cellular response is that of all inflammatory reactions, though the amount of vasodilatation is particularly pronounced.

Treatment. The wound usually heals without active interference, providing secondary infection does not occur. The patient must avoid hot and spicy foods and will probably do this automatically. Drinking cold water or any cold bland fluid will soothe.

Mouthwash of glycerin and thymol may be advisable. Tabs. Codein Co. should be given if the pain is marked. Triamcinolone in Orabase will control the inflammatory reaction, diminish symptoms and shorten the healing time. Corticosteroids should be restricted to those cases in which injury is fairly extensive or the pain is severe. If there is secondary infection a systemic antibiotic should be prescribed: Caps. Pencillin V. 250 mgm., 4 caps. per day for five days. Hot salt water mouth washes may be irritant and should not be prescribed.

Necrotizing Ulcerative Gingivitis

Necrotizing ulcerative gingivitis^{158, 113} is an acute infection of the gingiva. It is also known as Vincent's gingivitis because of the description of the organisms associated with the disease by Vincent; and as Trench mouth

because of the heavy incidence during World War I.

Its signs and symptoms are ulceration of the tips of the interdental papilla, pain, bleeding, and apparent sudden onset, and foul odour. There may be malaise, loss of appetite, and elevation of temperature but these symptoms are not always present.

The disease is confined to the gingiva; it does not affect the tongue or the alveolar mucosa. It does not occur in small children and is primarily a disease of the adolescent and young adult, although it does occur in older people.

The interdental papilla may appear eroded, punched out, or clipped off because of the ulcerative destruction. The ulcerations may progress to include the marginal and, rarely, the attached gingiva. The lesions may be covered with a whitish pseudomembrane. The gingiva around the ulcerations is an angry red colour. Bleeding occurs upon touch.

The aetiology of the disease is not fully understood. Bacteria, other local factors, systemic factors, and psychogenic factors have all been incriminated. The disease is accompanied by an increase in the numbers of spirochetes and fusiform bacilli and is an infection. Attempts to transmit

the disease in human beings have failed almost without exception. The disease is not communicable.

Treatment. The objectives of treatment is the elimination of the necrotizing process, the restoration of the tissue to health, and the correction of tissue deformities. Initial treatment may be varied and be supplemented according to the needs of the patient.

2. Chronic Gingivitis

Gingivitis^{158, 98} is an inflammation of the gingiva. Usually, chronic gingivitis is a generalized condition and, therefore, factors which are widely distributed, such as bacterial plaques and calculus, are most frequently responsible. Neglect is a principal cause of gingival and periodontal disease.

Cause. The basic aetiological factor in chronic marginal gingivitis is irritation of local origin, generally both mechanical and bacterial. These factors are responsible for gingival irritation and inflammation:

1. Bacterial plaque
2. Materia alba
3. Dental calculus

4. Food impaction
5. Faulty restorations
6. Poorly designed and constructed dentures and bridges
7. Orthodontic appliances
8. Lack of adequate function
9. Malalignment of teeth
10. Direct trauma
11. Failure to replace missing teeth
12. Mouth-breathing
13. Carious teeth
14. High frenum, shallow vestibule.

Bacterial plaque is the film which collects on teeth not properly cleaned. It is composed of partially digested food debris, epithelial cells, mucin and micro-organisms, and it is in the plaque that enzymes capable of tissue destruction are formed.

Material alba is a soft white deposit of broken down food debris which tends to collect about the gingival margins. As a good culture medium for the oral flora its presence may lead to both gingival inflammation and decalcification of teeth substance.

Dental calculus (synonym - tartar) contains about 80 per cent of inorganic material, most of which is calcium phosphate as apatite. There are two kinds of dental calculus:

- (1) Supra-gingival calculus,
- (2) Sub-gingival calculus.

The presence of calculus causes gingival irritation.

Food impaction: Food retention and impaction in the interproximal spaces caused by a plunger cusps of various teeth traumatise gingiva.

Faulty restorations: Overhanging margins will collect plaque which is irritant. Badly contoured restorations cause food stagnation and may result in damage to the gingival margin.

Poorly designed partial dentures may cause gingival damage in several ways. They tend to collect food debris, and calculus forms which mechanically irritates the gingiva.

Orthodontic appliances. Without diligent attention to maintain good oral hygiene, food debris and plaque formed may set up gingivitis.

Malalignment of teeth makes natural cleansing and efficient toothbrushing difficult and sometimes impossible. Because of this debris may be retained and eventually form calculus.

Direct trauma to the gingivae may be caused by hard or sharp particles in food, fish bones. Gingival

damage and chronic inflammation may result when teeth make contact with gum in the opposing jaw. This is quite common in cases Angle Class 11 malocclusion with a deep overbite so that the lower incisors strike the lower gingivae.

Failure to replace missing teeth: (a) the opposing teeth overerupt; (b) teeth on either side of the gap tilt; (c) tooth contacts in both jaws become abnormal, food-shedding becomes inefficient and food-impaction and stagnation results; (d) in the absence of the cleansing action of normal function unopposed teeth collect debris; (e) with tooth movement occlusal disharmonies are produced with result an abnormal occlusal stresses. The net result is gingival irritation and inflammation.

Mouth breathing causes gingivitis restricted to the upper anterior region. Either in the mouth breather or the patient with short-upper lip the gingiva in the front of the mouth is not continually bathed in saliva. The effects are gingival inflammation due to drying out of the epithelium lowers the resistance of the tissue (dehydration).

The irritant produces all the phenomena of inflammation, namely the initial vasoconstriction followed immediately by vasodilatation, a slowing of the blood-flow in the capillaries, fluid exudation from the vessel walls. The leucocytes are chiefly lymphocytes and monocytes, and many plasma cells are usually present.

Signs and Symptoms. In disease states the gingiva may be swollen, receded or retracted. Normal gingiva is firm, but it may become turgid, boggy, or dense in the presence of inflammation or enlargement.

One of the first signs of disease is the loss of stippling owing to destruction of the gingival fibers. The surface may become glossy, pebbly, or roughly granular. The gingiva may be red if it is acutely inflamed, or, if the inflammation is of long standing, it may have a magenta hue. However, the absence of classic symptoms does not rule out the presence of disease. Symptoms are: Gingival bleeding - Unpleasant taste - Halitosis - Discomfort and pain - Altered gingival appearance and pockets.

Treatment. Treatment planning demands an awareness of the many aetiological factors that might be involved. It must not only be recognized that systemic complications, if any are present, must be diagnosed and, if possible, brought under control, but that all other known factors, local, anatomical and functional, must be given consideration. In addition, a definite effort should be made in making oneself aware of the possible daily emotional trials besetting the patient. The purpose of treatment is to arrest the advance of the disease and to maintain the teeth and the periodontium in function repair for as long as possible.

1. Remove the immediate cause of disease.
2. Correct predisposing factors.
3. Take preventive measures to avoid recurrence.

Scale and polish teeth. Give instruction in oral hygiene techniques. Correct faulty restorations, prosthetic appliances and any other predisposing factor. Perform gingivoplasty where the gingival contour is poor and not improved by scaling. Re-appraise oral hygiene. Recall regularly for inspection and scaling.

PERIODONTITIS (PYORRHOEA)

McCarthy⁹⁸ defines periodontitis or pyorrhoea as suppurative inflammatory condition of the supporting tissues of the teeth. Orban¹¹² describes periodontitis as inflammatory disease of the gingiva and of the deeper tissues of the periodontium.

Periodontitis is considered a direct extension of gingivitis that has advanced or has been neglected.

Weinmann¹⁶³ classified periodontitis from a topographical point of view according to the initial attack as:

1. Apical periodontitis and
2. Marginal periodontitis.

Marginal⁵⁷ periodontitis is termed by many "periodontitis simplex", to differentiate it from "periodontitis complex". The latter is a secondary inflammation following the degenerative changes of periodontosis. Periodontitis simplex, or marginal periodontitis, on the other hand, develops as a penetration of a gingivitis. It has also been differentiated according to the duration of its occurrence:

1. Acute. Such as periodontal abscess, or developing from acute ulcerative gingivitis. Mostly it is due to local tissue injury, followed by healing or further destruction.

2. Chronic. Which is subdivided into:

- a. Periodontitis simplex and
- b. Periodontitis complex.

Aetiology

Periodontitis, according to Orban,¹¹² is caused by:

1. Extrinsic etiology. It is presumptive that local irritation is the primary aetiologic factor in periodontitis.
2. Intrinsic aetiology. Local factors are of paramount aetiologic importance. Systemic factors also play an important role in the development, form, and severity of some cases of this disease. In short we can say Periodontitis is the result of an untreated chronic marginal gingivitis or inadequate control of chronic marginal gingivitis.

Clinical Features. Manson⁹² says in chronic periodontitis the clinical features of chronic gingivitis are present to some degree, associated with pocketing tooth mobility, tooth migration and, occasionally, sensitivity to percussion.

McCarthy⁹⁸ said it is characterized by the formation of pathologically deepened gingival crevices around the involved teeth, resorption of supporting alveolar bone with loss of periodontal membrane fibres, suppuration from deep crevices,²⁹ and eventually mobility or loosening of the involved teeth.

Treatment. Periodontitis is not a disease from which a patient can be considered cured following a course of therapy unless that therapy be the extraction of the teeth. It is a disease which must be continually kept in abeyance, and, therefore, one which must demand continued prophylactic measures. Because the periodontist is faced with a problem of maintaining a periodontium at optimal function and repair, he necessarily must adhere to sound biological principles in his treatment, utilizing the accepted studies on tissues metabolism in health and disease.⁵⁴

The objectives of the treatment of chronic periodontitis are:

1. To eliminate the periodontal pocket.
2. To remove the immediate cause of the disease, i.e. calculus and plaque.

3. To achieve a tissue morphology conducive to efficient food-shedding and the maintenance of cleanliness.
4. To correct predisposing factors as in the treatment of chronic gingivitis, e.g. badly contoured fillings, gum-stripping dentures etc.
5. To correct the occlusion if it is thought that occlusal stresses may be a contributing factor in the progress of disease.
6. To gain the patient's co-operation so that recurrence is avoided.

Eradication⁵⁴ of the periodontal pocket is the keystone of the overall treatment regimen. Just as the removal of carious structure is essential in the restoration of a tooth, so too the successful outcome of all procedures involved in the total treatment of periodontal disease depends upon complete eradication of the periodontal pocket. Without pocket eradication the periodontal benefits provided by occlusal adjustment and restorative procedures are curtailed. On the other hand, treatment limited to pocket elimination without regard for other conditions contributing to the destruction of the periodontium also falls short of the desired goal.

Total eradication of the periodontal pocket is

imperative, anything less is a compromise inconsistent with the preservation of the health of the periodontal tissues. Partial reduction in pocket depth is not enough even if it is attended by improvement in the clinical appearance of the gingiva and the cessation of pus formation.

DYSTROPHIC

Orban¹¹² gives the definition of Dystrophic as a term used to designate pathologic conditions produced by abnormal tissue nutrition leading to disturbance in cells metabolism and manifested by degeneration, atrophy, or hyperplasia.

Many writers have used the titles as degenerative conditions or processes, regressive changes, dystrophic disease, atrophic conditions, and degenerations, while many European authorities used the words "paradentoses" or "paradentose" to signify this group.

Wade¹⁵⁸ describes it as a passive deterioration of a cell, tissue, or organ.

According to Orban degeneration is a morbid process in which vital cells undergo retrograde processes, changing eventually into nonvital substances. These nonvital substances can be fatty in nature (fatty degeneration) and can

be protein substance (hyaline, mucinous, or amyloid degeneration). The nonvital substance may also be of mineral nature (calcareous degeneration) or consist of serous fluids accumulating in the cytoplasm (hydropic degeneration).

There are two types of degeneration of periodontium: The first one is gingivosis which is a non-inflammatory change occurring in the gingiva. Clinically there are two types of gingivosis; one in which the lesion is diffuse, the other where there is a mottled appearance. The second type is called periodontosis because the deeper periodontal tissues are affected by the degenerative process and it is a non-inflammatory destruction of the periodontium.

When degenerative and inflammatory lesions coexist the condition is named periodontitis complex to differentiate it from periodontitis simplex. Certain authorities would prefer to call this mixed condition "periodontosis complex".

Gingivosis

The term gingivosis¹⁴⁰ was first used to designate degenerative changes in the gingiva observed in starving children in post-war Italy. Earlier, gingivosis had been described in the literature as desquamative gingivitis.⁹⁷

Gingivosis⁵² is a rare dystrophic condition characterized by degenerative changes of gingival connective tissue, involving the papillary, marginal, and attached gingiva. Irregular areas on the entire gingiva are fiery red, smooth, and shiny. Stroking the gingiva with a finger, cotton roll, or a blast of air tends to peel away the surface epithelium, exposing the painful bleeding surface of the connective tissue.

In more severe cases, the gingiva is covered by multiple row bleeding areas on background of intense erythema. These lesions begin as blisterlike eruptions which break. Generally, the lesions do not occur in edentulous areas.

Etiology. The etiology of this disease is unknown. The probable association with disturbances in sex hormones is based on theoretical assumptions and on some clinical observations. Systemic hormonal therapy has failed to cure the disease.

Treatment. Treatment of gingivosis has been a problem from time to time that it was first recognized clinically. Most procedures have afforded only temporary relief. The topical application of estrogenic hormones¹⁷³ in the form of ointment has brought temporary improvement in some cases, but no

permanent results have been observed. Some cases have improved with application of corticoids.¹⁷¹ The best result in treatment to date was achieved by the use of 10 mg tablets of hydrocortone acetate which were chewed into a mash and retained in the mouth for half an hour, repeated once or twice daily. The patient pushes the material to the various affected areas with the tongue. The material then is expectorated. This continued for one month, or until improvement is noted, Corticoid ointments (2.5 per cent) in an adhesive base have also been recommended for the treatment of this condition.³⁶

Periodontosis

In the past periodontosis has been known as diffuse alveolar atrophy, rarefying pericementitis fibrosa, by Box, and cementopathia by Cottlieb. According to Orban¹¹⁰ periodontosis is a degenerative disease that affects the deeper periodontal tissues.

Aetiology. The aetiology of periodontosis¹⁸ is not known. It is believed that some systemic factor is affecting the process of tissue replacement, but laboratory tests rarely reveal figures

which are considered to be outside the normal range. It has been suggested as the result of animal experimentation, that periodontosis is due to protein deficiency, and this might well be so in races such as the Hindus, where little animal protein is contained in the diet as reported by Marshall-Day.³⁹

Clinical Pathology. A patient suffering from periodontosis usually presents complaining of spacing, increasing prominence, elongation, or loosening of one or more of the upper front teeth. The change has usually been noticed for some time, often as much as a year, before the patient seeks advice and has become progressively worse during this period.

Clinical examination substantiates the patient's complaint and critical examination may reveal similar changes in other parts of the mouth.

The favourite sites for periodontosis are around the first molars and maxillary incisors.

Frequently the state of oral hygiene is excellent, there is no food debris and very little calculus. The gingivae appear uniformly healthy

around both the affected and non-affected teeth, unless the condition has become a periodontitis complex. Inflammation must be regarded as a secondary feature in periodontosis.

Treatment. The prognosis of periodontosis is, in most cases, poor, as is evident from the study of its pathology and its lack of known aetiological factors. Therefore only the symptoms present can be treated. Local factors such as poor oral hygiene, food impaction, traumatic occlusion (possibly with bruxism) must be identified, and, if present, corrected. The patient should be placed on a well-balanced diet, and general hygiene should be good.

In most cases no abnormal systemic factors are evident, and the treatment takes the following form: 145, 146, 152

1. Scaling and instruction in oral hygiene techniques. If patient co-operation is poor any further periodontal treatment will be a waste of time.
2. Extracting teeth with poor bone support.
3. Adjusting occlusion so that multiple contacts are made in all positions and excursions.

4. Splinting teeth which have drifted and are mobile but where bone support is still fairly good.
5. Providing a bite guard for wear at night if there is a possibility of bruxism.
6. Treating pocketing as one would routinely, close supervision during the post-operative period is essential.
7. Providing correctly designed partial dentures.
8. Ensuring a regular three-monthly recall and closely supervising the case with emphasis on assiduous attention to good oral hygiene.

ATROPHIC PERIODONTAL CONDITIONS

Atrophy is a diminution in size due to shrinkage of cells and/or fibers which have reached full development. It is therefore different from hypoplasia which is a failure of the tissue constituents to reach maturity. As compared to degeneration, it is more or less a quantitative tissue change (degeneration is a qualitative one).

Periodontal atrophy is far from being a rare condition, though it is not common in children. It is a rather common problem in the geriatric patient.¹²⁶

Aetiology

The cause of atrophy is defective nutrition¹³⁴ of the cells. There are then many different aetiological factors, both local and systemic, which may rise to atrophic changes in the periodontium.

The two commonest causes are faulty brushing and loss of function due to extraction. Some of the other causes are toxic and chemical influences. All of these factors will cause an alteration in cellular metabolism.¹²³

Recession of the Gingiva

The effect of atrophy¹⁰¹ upon the gingiva often is expressed in gingival recession. The gingiva might not show any other sign of pathology. It often is thin and finely textured. It may be pale pink with a thin gingival margin and relatively elongated and pointed papillae. There may be no pocket present and the gingival sulci are usually very shallow and without exudate.

Recession¹²² may be generalized, or it may be localized to a single tooth, or to a group of teeth. Gingival recession may be an expression of disease, or it may be physiologic and an expression of aging. The distribution of the recession is of considerable significance in determining

the aetiological factor responsible for the condition. When generalized, a systemic factor is indicated, whereas a localized distribution signifies either disuse or pressure.

Over-eruption of the associated tooth immediately focuses attention in the case of disuse, while recession whether lingually or labially on the anterior teeth suggests an examination of the occlusal relationship of these teeth. Recession confined to the labial aspect of the mandibular incisors and lingual aspect of the maxillary antagonist denotes an excessive overbite. Areas subject to severe tooth-brush abrasion are buccal surfaces of upper canines and premolars.

The aetiology of gingival recession, whether localized or generalized, is not easily determined.

1. Toothbrush trauma.
2. Excessive orthodontic forces.
3. Local irritations - chemical and physical.
4. Occlusal trauma due to excessive overbite.
5. Faulty tooth alignment.
6. Anatomic abnormalities such as thin bony plates and high frenum attachments.

Radiographs may reveal the impaired trabecular pattern; loss of bone at alveolar crest, particularly if it has occurred circumferentially and so can be observed in the

interdental septa; narrowing of the periodontal ligament; and increase in the thickness of the cementum, demonstrated by the width of the apical part of the root, in an example of atrophy due to disuse.

Treatment. The treatment of gingival recession is difficult, since there is no known way to increase the height of gingival tissues toward the occlusal surface. Treatment should include measures to prevent continuation of the recession; all local irritating factors should be removed or minimized. The most important therapeutic measure is the institution of proper oral hygiene (correct brushing, soft brush, nonabrasive dentifrice) adapted to the specific patient. No two cases may require the same care. The basic principles are:

1. meticulous cleanliness without injury and
2. frequent careful root planing without injury to the gingiva.

Restorations may be necessary to restore or to create proper deflecting tooth contours for soft tissue protection.

Periodontal Atrophy Due to Disuse

This form of atrophy occurs in the periodontal tissues as a result of lack of function. Goldman⁵⁸ uses the term "functional insufficiency" to describe the form of atrophy and lowered tissue resistance resulting from lack of function. He differentiates between a total insufficiency of the jaws resulting from lack of adequate masticatory function, and a localized insufficiency of the supporting tissues react to change in function by constant adaption. All tissues of the periodontium are influenced by functional changes. It has been claimed by Orban that the free gingival groove at the border of free and attached gingiva is an expression of such influences.

Preissecker¹²⁰ concluded his investigation on young albino rats after six months saying that the width of the periodontal membrane around the nonfunctioning teeth was approximately half that of the functioning teeth. He also distinguished between the physiologic periodontal membrane width in nonfunctioning and that in functioning teeth.

Coolidge³¹ measured the width of the periodontal membrane in 172 teeth from 15 human jaws. He concluded that the thickness of the periodontal membrane in each jaw was in direct proportion to the intensity of masticatory function.

Clinical findings:

- (a) Loss of tone of the gingivae due to lack of functional stimulation;
- (b) recession of the gingivae may or may not occur;
- (c) extrusion of the tooth occurs and may have an increase in thermal sensitivity.

Wade¹⁵⁸ says that Disuse Periodontal Atrophy is usually a sequela of tooth extraction, but may result from some developmental anomaly, such as an anterior open bite or a severe Class III malocclusion, or occur after malunion of a fractured jaw.

Miller¹⁰⁶ listed the following causative factors in nonocclusion:

- (1) habits,
- (2) anomalies of development,
- (3) disease,
- (4) injury to oral structures, and
- (5) faulty restorations.

Histopathology:

- (1) Thinning of the periodontal ligament;
- (2) thinning and reduction in the number of periodontal fibers;
- (3) thickened cementum; and

- (4) reduction in the height of the alveolar bone, and osteoporosis.

Treatment. The aim should be to prevent the development of pathologic conditions. Replacement of missing or lost teeth.

HYPERPLASTIC PERIODONTAL CONDITIONS

Hyperplasia is the term used for the increase in the size of an organ or its parts. In hyperplasia, this increase is due to an increase in the number of cellular elements of the organ and does not serve a functional purpose. Hypertrophy is defined as an increase in size due to enlargement of the individual cells and/or fibres. Whether an enlargement of the tissue results from hypertrophy or hyperplasia it is determined by microscopic examination of its cells. Orban¹¹² says there is no gingival hypertrophy. As far as the overgrowth of the gingiva is concerned, this definitely is a hyperplasia. As far as the other structures of the periodontium are concerned, hypertrophy may take place in response to an increase in functional demand, e.g. cemental hypertrophy, periodontal hypertrophy. Any increase in the thickness of cementum may be due to an increase in the number of its elements (hyperplasia), as in the nonfunctioning tooth, or to increased function, in which case it is a hypertrophy.

Gingival Hyperplasia

Gingival hyperplasia is an overgrowth due to increase of the fibrous tissue elements of the gingiva, a fibrous hyperplasia. It is not an inflammatory condition, although inflammation might affect the hyperplastic tissue secondarily.

Clickman⁵³ classified gingival hyperplasia from clinical point of view into two general types:

1. Inflammatory hyperplastic gingivitis.
2. Fibrous hyperplasia of the gingiva.

Orban¹¹² claims that in true hyperplasia, the overgrowth is due to increase in number of the local cellular elements and fibers and thus is termed gingival hyperplasia or fibrous hyperplasia of the gingiva. The suffix "itis" which connotes inflammation, is not used here.

Hyperplasia might occur as a delimited localized overgrowth limited to a certain area. Microscopically an increase of the fibrous and cellular elements is well marked. If calcification and ossification may take place in this localized overgrowth it is then called ossifying fibromas. The aetiology of these fibromas is unknown.

Gingival hyperplasia is manifest clinically as enlargement of the gingivae. This enlargement may be localized or generalized and may be confined to the marginal gingiva or affect the entire gingiva.

Generalized gingival hyperplasia may be of genetic origin or caused by any of the systemic factors which have been mentioned.

Mild hyperplasia causing enlarged rounded gingival margins produces a festooned appearance. Gingival festoons usually are caused by chronic irritation. The irritation is frequently the result of traumatic use of a toothbrush.

Dilantic Sodium-Induced Hyperplasia

Gingival hyperplasia often is associated with Dilantin sodium therapy in the management of epilepsy. In such instances the hyperplastic gingiva is firm, resilient, and painless unless there is also inflammation from local irritation. The enlargement seems to be an overgrowth of the papillae. In this case the interdental papillary gingiva is often affected to a great extent leaving the margin unaffected or less affected.

Not all patients taking Dilantin sodium show gingival

hyperplasia. Local etiologic factors may influence the amount and location of the enlargement.

Microscopically there is an increase of the fibrous tissue elements, proliferative tendency in the epithelium, and increased accumulation of inflammatory cells.

It may be possible for the physician to change to other drugs (Mesantoin, Mysoline) which can control convulsions and apparently do not induce hyperplasia. At other times, the dosage of Dilantin sodium can be reduced and combined with other drugs.

Overgrowth can be prevented in some cases by good oral hygiene. Removal of all local irritations. Gingivectomy.

Hyperplastic Gingivitis

Hyperplastic gingivitis is an inflammatory disease characterized by overgrowth of the gingiva and is due to accumulation of inflammatory products, edema, and cellular elements.

In inflammatory hyperplastic gingivitis, the enlarged gingiva is soft, edematous, hyperemic or cyanotic, sensitive to touch, not stippled and can be made to bleed quite easily.

1. Gingivitis in Puberty

Hyperplasia of the marginal gingiva occurs most often at puberty when there may be a temporary endocrine imbalance. The severity of gingivitis may vary with menstrual periods. However, it is seldom possible to demonstrate signs of endocrine imbalance except during pregnancy. All of these periods parallel times of increased emotional stress. The inflammation often may be characterized by infiltration of plasma cells alone.

Local treatment sometimes is not sufficient to cure gingival inflammation completely because of the intrinsic factors which may be present.

2. Gingivitis in Pregnancy

During pregnancy pre-existing gingivitis tends to become more severe and it may change in character.

Systemic factors play an important part in the etiology of gingivitis, not as a primary but rather as a secondary or modifying influence. Gingivitis during pregnancy becomes aggravated by the proliferative tendencies in the epithelium, connective tissue, and endothelial cells. Proliferation of the capillaries is one of the most characteristic microscopic features of gingivitis during pregnancy.

The papillae are prominent and are sharply demarcated from the attached gingiva. The margins often are irregular and appear fringed. Individual hyperplastic papillae may become extremely large and then are called pregnancy "tumours". Both pregnancy tumours and the aggravated state of the gingivitis will have a tendency to disappear after parturition but may recur during subsequent pregnancies.

The treatment most often consists of root planing, curettage and the establishment of good oral hygiene, including stimulation of the tissues.

3. Scorbutic Gingivitis

It is a systemic influence upon inflammation of the gingiva. One of the most characteristic features of gingivitis in scurvy is the disappearance of collagenous, fibrous elements from the connective tissue.

Usually gingivitis was present prior to the scorbutic symptoms.

The gingiva appears bright red. It is hyperplastic, ulcerative and haemorrhagic.

Therapy is to give a high dosage of vitamin C, 1,500 mg per day for a week. This then reduces to 500 mg

per day for the next four weeks; then 300 mg per day for a month, and finally 100 mg per day thereafter.⁵³

TRAUMATIC PERIODONTAL DISTURBANCES

Traumatism is a morbid condition produced by repeated mechanical forces. Tissue trauma can be produced by forces which exceed the physiologic limits of tolerance.

Stillman introduced the term "Traumatic occlusion" in 1917.¹⁵¹ Box¹⁷ called it "Traumatogenic occlusion". Pressure pericementitis by Black. Rule named it "Occlusal overstress", and "Dynamic irritation" by Gratzinger.⁶¹ There are more or less synonymous with traumatic occlusion.

Traumatic occlusion is defined as an abnormal occlusal stress which is capable of producing or has produced an injury to the periodontium. Experimental studies and histopathologic studies of section of human jaws at autopsy have proved that occlusal trauma is a definite cause of certain pathologic disturbances in the periodontal membrane. The observations in human autopsy material which substantiate the presence of occlusal trauma include abrasion, cemental tears, root and alveolar bone resorption, periodontal membrane, injuries and fracture of the teeth.

Stillman¹⁵¹ is credited with being the first to

emphasize traumatic occlusion as a cause of periodontal disease. McCall supported Stillman in the belief that, once traumatic occlusion is established, irritation and periodontal congestion result.

Orban and Weimann¹⁰⁹ found signs of trauma in every case of missing teeth in their studies of human teeth.

Trauma from occlusion may be caused in three ways:

1. Alteration in the occlusal force.
2. Alteration in the capacity of the periodontium to withstand occlusal forces.
3. A combination of both.

Clinically, periodontal traumatism may be classified as primary and secondary. In primary traumatism the force is pathologic; in secondary traumatism the force is physiologic but the resistance of the periodontium has been reduced by loss of attachment apparatus. Traumatopathy is the same whether the trauma is primary, secondary, or orthodontic.

1. Primary Traumatism

Orban^{109, 112} defines primary traumatism as the production of mobility in a tooth with normal support subjected to excessive force. Periodontal disease and

occlusal disharmonies so commonly occur together that it is difficult to determine which comes first.

Rule insisted that occlusal overstress is not a primary factor in the initiation of periodontal disease. He considered it a secondary predisposing factor, operating almost exclusively in cases in which the foundational structures have become weakened through dystrophic or other predisposing factors.

Merritt¹⁰⁴ stated that trauma is a symptom of periodontal disease and not the cause of it. MacMillan^{90a} disagreed with the opinion that traumatic occlusion is the predominating causative factor in periodontal disease. He said that the degenerative process attributed to traumatic occlusion could not be produced on a perfectly healthy tooth by masticatory forces.

Trauma¹²² from occlusion may properly be considered the primary aetiologic factor, if destruction of supporting tissues occurs when the only local alteration to which a tooth is subjected is one of occlusion. The "high filling" gives rise to occlusal disharmony that results in acute symptoms such as sensitivity to percussion, tooth mobility and pain. Frequently, the

tooth adjusts itself to dissipate the force. If this does not occur, it may result in periodontal abscess.

Prosthetic replacements,^{121, 122} constructed with insufficient attention to the direction of functional forces, can injure the supporting tissues of abutment teeth previously uninvolved by periodontal disease.

Trauma from occlusion may also be considered as a primary causative agent when destruction of supporting tissues occurs in relation to teeth that have drifted or extruded following the extraction of adjacent or opposing teeth.

The periodontal tissues of teeth moved into unfavourable positions in the course of orthodontic treatment are also subject to injurious changes from occlusion.

Treatment.

1. Find the cause and remove it. Correct any premature contact due to faulty restorations.
2. Replace any missing tooth or teeth by either well designed partial denture or crown and bridge.
3. Carry out occlusal rehabilitation. Use bite guard to prevent bruxism.

2. Secondary Traumatism

When the environment or position of a tooth is altered by periodontal disease so as to make it subject to trauma from occlusion, the latter assumes the role of a secondary disease-causing factor.

In the course of pathologic migration or extrusion associated with periodontal disease, teeth reach a position in which they become subject to excessive occlusal forces.

Glickman⁵⁴ says "trauma from occlusion is a secondary factor when loss of alveolar bone and periodontal ligament fibers create increased functional demands upon the remaining portion of the periodontium".

Accelerated tooth eruption and extrusion that result from a disparity in the physiologic balance between active and passive eruption may similarly place a tooth in an unfavourable position, so that it becomes affected by trauma from occlusion.

Cause.

Coollidge³² has summarized the local causes of occlusal trauma as following:

1. Unrestored loss of posterior teeth with tooth drift;
2. Malposition of the teeth;
3. Cuspal interference;
4. Uneven wear of the teeth;
5. Atrophy of the supporting tissues of the teeth;
6. Excessive overbite;
7. Uneven eruption rate;
8. Abrasion with loss of vertical dimension;
9. Defective occlusion as induced by ill-constructed artificial restorations and orthodontic appliances; and
10. Injurious oral habits.

Subjective complaints. Orban¹¹² gives the following:

1. Awareness of occlusal disturbance.
2. Soreness on percussion.
3. Premature contacts.
4. Bruxism.
5. Itchy sensation and desire to rub affected area.
6. Pain or discomfort (due to the pulpal irritation).
7. Soreness in the area of the temporomandibular joint.
8. Soreness in the muscles of mastication.
9. Cracking noise in the temporomandibular joints.

Treatment. Wade⁵⁸ gives a sound order of treatment:

1. Relief of acute pain.
2. Removal of acute infection.
3. Eradication of gross sepsis.
4. Removal of direct irritants.
5. Elimination of harmful habits.
6. Correction of systemic factors.
7. Elimination of pockets.
8. Attainment of satisfactory gingival contour.
9. Elimination of deep lesions.
10. Occlusal rehabilitation.
11. Home care.

SUMMARY AND CONCLUSION

Inflammatory disease of tissues and structures that invest and support the teeth probably occurs almost universally, for with the exception of persons who no longer have any of their own teeth, no one is fully immune to attack.

It is widely recognized that the major cause of tooth loss beyond the age of 30 is periodontal disease. We realize the chronic nature of most periodontal disorders; the inception occurs long before it is necessary to remove a tooth. The predominating early symptom is a gingivitis,

usually caused by a local irritant which triggers a chain reaction resulting in tooth loss. The local condition is usually one that is amenable to treatment if it is diagnosed early and if proper therapy is applied. Yet, the surveys that from 60 to 90 of children have some form of gingivitis, most of which goes unrecognized and untreated.

Several conflicting opinions exist concerning the aetiology of periodontal disease. There are those who say that the cause is unknown. Others say that the cause is known but is never single, involving usually a combination of dysfunctional, irritational, and systemic factors in varying proportions. Diet may affect the periodontal tissues both by its direct oral action and through its systemic effect.

Knowledge of the etiology and the methods of treatment of periodontal disease has increased rapidly in recent years, so much so that control is possible in most individuals willing and able to carry out the necessary but sometimes tedious routine of oral hygiene. Nevertheless it seems likely that research may produce some further solutions to the problems of this disease and thereby have a further effect upon treatment.

It appears to be general agreement that gingival deposits such as calculus and plaque constitute the most important local aetiological agent for many periodontal diseases ranging from simple gingivitis to severe periodontitis. The opinion is supported by both clinical and experimental observations.

Neglect is a principal cause of gingival and periodontal disease; neglect of the healthy mouth which permits disease to occur; neglect of early disease which permits to destroy the tooth supporting tissues and neglect of the treated mouth which permits disease to recur.

The chief reason for treating periodontal disease is to preserve the natural teeth in the mouth. Without treatment there is a continuation of the destruction of the supporting tissues of the teeth to a point where the teeth loosen and fall out. Also, from the point of view of general health, it is understood that gum disease may act as a focus from which disease may spread to other parts of the body. Treatment will prevent this. For other reasons such as bleeding, bad breath, and poor esthetics, periodontal treatment should be performed.

Early diagnosis, proper treatment, and periodic

checking on results of therapy are very important.

The treatment of periodontal disease is accomplished by a variety of treatment methods such as scaling, curettage, periodontal surgery, occlusal adjustment, etc.

At the conclusion of periodontal therapy the patient is placed on home care routines and periodic office treatments. This control phase is essential to the ultimate objective of periodontal treatment which is a healthy periodontium.

EPIDEMIOLOGY

Periodontal disease is a widespread¹⁶⁶ disease of mankind and it becomes a public health problem at present. This is well established by epidemiological studies. The introduction to the 1961 W.H.O. Report on Periodontal Disease contains the following statement:

"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 the natural dentition and deprives many people of all their teeth long before old age. The total effect of periodontal disease on the general health of populations is unassessable."

This statement shows that periodontal disease is one of the most common diseases to which man is prone, and is probably responsible for the loss of more teeth than is dental caries. As methods of caries control become more

effective periodontal disease will be clearly seen as the major dental problem. If we have a straightforward and comprehensive account of the theory and practice of periodontology we can, with the most recently devised techniques, attack this problem or control the periodontal disease through the agency of the dental practitioner and also on public health level. According to Englander,⁵⁰ "The fundamental function of epidemiology is to determine the occurrence and distribution of mass disease in order to define problems and formulate objectives". Marshall-Day⁴³ says "that periodontal disease constitutes a serious and universal health problem. This is true because in some degree and in one form or another it affects large population groups at all ages and so frequently remains undiagnosed and untreated until disease is well advanced".

Epidemiological studies have been carried out in many countries of the world and shown that periodontal disease exists everywhere. It is found in primitive societies, as well as in advanced modern societies, where it is found in urban groups as well as rural groups. It occurs in all races of mankind, and at all ages. It also occurs in both male and female. The World Health Organization summed up the whole status of periodontal disease as a public health problem as follows:

- "1. In the U.S.A. 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 dental caries.
2. Where comparisons have been possible, few have indicated any prevalence in other parts of the world. In most areas, and particularly in Asia the disease seems to be more prevalent and severe than in the U.S.A.
3. The onset of disease may be as early as puberty in susceptible individuals. In population groups both the prevalence and severity of disease increased 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."

In 1945, King⁷⁶ demonstrated for the first time that it might be possible to define mild, moderate or severe gingivitis in objective and qualitative terms and figures.

Schour and Massler,¹⁴⁰ from the results of their study of post-war Italy in 1947, the first quantitative method of assessing gingival disease was formulated and referred to as the P-M-A Index.

Work upon the epidemiology of periodontal disease has been retarded by the following factors:

1. Two areas are involved in most instances: the gingiva, where gingivitis is usually a reversible process, easy to observe; and the alveolus, where bone loss is the best measure of damage and is almost always chronic, cumulative, and hard to observe.
2. Periodontal disease, unlike caries, has its greatest incidence late in life.
3. Subjective measurement of the severity of the disease is usually necessary. The only real quantitative measure - an estimation of the severity of chronic periodontal disease in terms of linear bone loss - can often be misleading and is extremely difficult to obtain without taking X-ray.

Several of the early epidemiological investigations surveyed the incidence of gingivitis in children, before 1945, but did not clearly define the criteria and method of evaluation employed. The findings simply reported the

presence or absence of gingival disease and its degree of severity if present.

Periodontal Indices

1. PMA index for gingivitis. For work among children, Schour and Massler¹⁴² have developed what they call the PMA index for gingivitis. The existence of gingivitis is noted in each of the three areas relative to a given tooth: the gingival papilla mesial to the tooth (P), the buccal or labial gingival margin (M), and the gingival attachment (A). Cases are called "mild" if one to four papillae and 0 to two margins are affected, "moderate" if four to eight papillae and two to four margins are affected, "severe" if more than eight papillae and more than four margins are affected. Involvement of attachments is associated with severe cases. The average PMA for the group is determined by totaling the number of gingival units affected and dividing by the number of cases under study. In practice this method is proving an unsatisfactory measure of severity, since papillae and margins often become inflamed about the same time and considerable change in severity is possible after this point.

2. Periodontal index. This index was developed for the assessment of chronic destructive periodontal disease by Russell,¹³¹ but there is provision for recording gingivitis also. Here each tooth studied in the mouth is assigned a score between zero and eight. Zero describes a tooth negative for all criteria and eight describes a tooth with advanced alveolar bone loss, gingivitis, and loss of masticatory function. Intermediate numbers describe various combinations and degrees of gingivitis. The values for all teeth in the mouth are then averaged to give the patient's score.
3. Radiographic indices of bone loss. When chronic destructive periodontal disease is present, the loss of alveolar bone which results is an irreversible change.⁴⁴ Resorption of alveolar bone is scored on a scale from zero through ten, with a grade of ten indicating complete loss of alveolar bone. A grade of five is scored when half of the alveolar support is lost, and a grade of one is scored when there is just bone loss to be visible in the film. Thus the average score for the areas read in the mouth is taken as the resorption figure for that person.
4. Gingival recession index. Stahl and Morris¹⁴⁸ in

study carried out for the American Army. They counted the number of teeth in which gingival recession had exposed the cementoenamel junction, and the total teeth present in each mouth. Then they expressed these values as a percentage score of recession for an individual. The whole group score was computed as the mean percent per man.

5. Periodontal disease rate index. This includes both reversible and irreversible signs and requires the use of both pocket probe and X-ray equipment. All the teeth and surrounding tissues are examined in a given mouth. A tooth is scored as positive if there is (a) gingival necrosis, or hypertrophy or inflammation encircling the tooth, or a purulent discharge from the gingival crevice, or (b) a gingival crevice having a depth of 3 m.m. or more or (c) tooth mobility greater than 1 m.m. in any direction or (d) radiographic evidence of resorption of alveolar bone extending more than 3 m.m. apically from the cementoenamel junction. Thus number of teeth affected with periodontal disease is determined and computed as the percentage of all teeth present in the mouth. This is the score for individual and group score is the mean of the scores for individuals comprising the group.¹³⁸

6. Oral hygiene index. This index was developed by Green⁶⁴ et al and covers all the teeth fully erupted in the given mouth, and is based on simple clearly defined criteria which reduce examiner variations to a minimum.

This index is composed of the combined "Debris Index" and "Calculus Index", and each in turn is based on 12 numerical determinations, representing the amounts of debris or calculus present on the buccal and lingual surfaces of each of three segments of each dental arch, (i) the segment distal to the right cuspid, (ii) the segment distal to the left cuspid and (iii) the segment mesial to the right and left first bicuspids. Separate recordings are made for the buccal and lingual surfaces to differentiate oral hygiene status between these surfaces.

Each surface is scored for oral debris and calculus independently on a scale from zero through three. For each individual an average debris and calculus score is computed which is combined for the oral hygiene score. Group values for the modified (or simplified OHI-S) range from zero to six.

7. Gingival bone count index. This index has been developed

by Dunning,⁴⁷ and permits differential recording of gingival and bone conditions. An explorer is used in exacting specific pockets and a bitewing X-ray procedure is applied for loss of bone. Subjective measurements are made on a zero to three scale, for gingivitis of each tooth and proportional measurements of bone loss is made on a zero to five scale.

Gingival score. If there is no deviation from normal gingiva then a tooth is scored zero or negative. For mild gingivitis involving free gingiva (margin and papilla) it is score one, for moderate gingivitis involving both free and attached gingiva it is scored two, and for severe gingivitis with hypertrophy and easy bleeding it is scored three.

Bone score. One score is assigned for each tooth studied. For no bone loss the tooth is assigned a score zero, for incipient bone loss or notching of alveolar crest it is scored one, for bone loss approximating one fourth of root length or pocket formation - one side not over half root length, it is scored two, for bone loss approximating one half of the root length or pocket formation one side not over three fourths root.

Length with slight mobility it is scored three, for bone loss approximating three fourths of root length or pocket one side to apex with moderate mobility it is scored four, and for complete bone loss with marked mobility it is scored five. Thus maximum possible gum-gone count per person is shown by score eight, and for obtaining G.B. count in the individual the whole mouth mean scores are added.

8. Ramfjord index.¹²⁴ This index is based upon reversible and irreversible signs of disease, which can be recorded by its application. Six selected teeth are examined in order to save time and effort, and to indicate the periodontal condition of the whole dentition. These are maxillary right first molar, maxillary left central incisor, maxillary left first bicuspid, mandibular left first molar, mandibular right central incisor and mandibular right first bicuspid. The area around each of these teeth is dried with cotton wool and is observed and tested for deviation from health and colour, form, density and bleeding tendency. The inflammation is scored in three degrees:

- (i) a score of one for mild to moderate inflammatory gingival changes not extending all around the tooth;

- (ii) a score of two for mild to moderately severe gingivitis extending all around the tooth; and
- (iii) a score of three for severe gingivitis, characterized by marked redness, tendency to bleed and ulcerate.

This gingivitis score is the periodontal score for the teeth, if on probing the epithelial attachment is found to be on the enamel. Then a pocket probe (number zero) is used for measuring the depth of the periodontal pockets - on mesial, buccal, distal and lingual aspects of each of these six examined. When 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 cementoenamel junction, but not more than 3 m.m. (including 3 m.m. measurement) in any area then gingival score is ignored and the tooth is assigned a periodontal disease score of four. If any crevice extends to 6 m.m. (including 6 m.m.) below the cementoenamel junction, the tooth score is five, and if any extends more than 6 m.m. below the cementoenamel junction, the tooth score is six, which is the maximum on the scale. When one or more of the

specified teeth are absent then the score is based on the remainder of the teeth. The distance from the cementoenamel junction to the depth of the crevice is not measured directly. Independent measurements are made for the distance from the cementoenamel junction to the gingival margin, and for the distance from the gingival margin to the bottom of the crevice. These are then added or subtracted to arrive at the tooth score. Thus the first three grades on the Ramfjord scoring scale refer to gingivitis which is a reversible sign. The last three grades refer to the depth of the gingival crevice or of periodontal pocket, below the cementoenamel junction. This is an irreversible sign of disease.

1. PMA index which records the amount of inflammation in the papillary, marginal and attached gingivae and is very useful for children.
2. Russell's periodontal index (PI), by which a numerical value is given for the different degrees of periodontal breakdown with a greater value for the more severe conditions.

3. Radiographic indices. It is the best means of detecting periodontitis in incipient stage. When an irreversible population index is desired, the X-ray examination is the best method of choice.
4. Gingival recession index. This is a second irreversible lifetime measure of periodontal destruction. This index seems to be very simple as simple counts of the teeth with exposed cementum can readily be learnt by any examiner and examiner variations can be reduced almost to zero. This index would be difficult to apply and to make counts in mouths with heavy supra gingival calculus or in betel chewers would be a problem.
5. Sandler Stahl index (PDR) which is a simplified method of noting the percentage of teeth affected by periodontal disease.
6. The oral hygiene index (OHI) and the simplified oral hygiene index (OHI-S), both of which record the amount of debris and calculus.
7. Gingival bone count index. This index seems to be useful because subjective measurements of gingival condition is combined with simple proportional measurements of bone loss in order to produce a composite score.

8. Ramfjord's index. This index also scores periodontal disease with higher scores for the more severe conditions and also scores certain aetiological factors such as calculus, plaque, occlusal attrition, tooth mobility and open contacts.

Gingivitis

In 1913 Black^{14a} carried out possibly the first epidemiological survey when he analyzed statistics obtained from 500 persons who ranged in age from twenty to thirty-five years. Ninety-five per cent had some degree of gingivitis.

About five per cent were due to carious cavities on the axial tooth surfaces, 18 per cent to bad margins on restorations, 10 per cent to lack of contact and seven per cent to improper contact of restorations. Approximately 40 per cent of gingivitis was associated with the carious process or its repair; but it must be emphasized that the gingivitis resulted from the previous activity of caries and does not constitute proof that the two processes were active side by side at the same time.

In 1925, Ainsworth and Young, ~~they~~ assessed the occurrence of gingivitis in 4,063 children from 2-14 years of

age, residing in England and Wales. They observed no gingivitis in 2,438 children (60%), slight gingivitis in 1,439 (33%), intermediate gingivitis in 244 (6%) and severe gingivitis in 36 (0.8%).

They suggested that boys had a slightly higher incidence than girls in the later age groups. A definite increase in the number of children with gingivitis was evident with increase in age, from 18.5% at five years to 53% at 14 years. Such increase was found not only in the number of children affected but also in the severity of the gingivitis. Quoted in Dunning⁴⁸ and Glickman⁵⁴.

In 1929 Anderson examined 100 China male workers in Peiping ranged in age from 15 to 33 years. He reported that 90 per cent had gingivitis. Quoted in Dunning.⁴⁸

In 1936, Massner et al, reported the result of a questionnaire type of survey of the occurrence of gingivitis in children. Figures were collected and submitted by dentists from 26 states in United States and data published on 1,438,318 children (boys and girls) from six to fourteen years of age. They made no attempt to differentiate among the various forms or degrees of gingivitis. Their data indicated that in white children (i) the incidence of

gingivitis increased with age from 3.5% affected at 6-8 years, 5.6% at 9-11 years, to 8.6% affected at 12-14 years, and (ii) the occurrence of gingivitis was higher in boys than in girls (6.4% and 4.8% respectively). Quoted in Glickman⁶⁴ and Dunning.⁴⁸

Westin made an investigation into questions of social hygiene in the counties of Vasterbotten and Norbotten, Sweden in 1937 and reported 86.5 per cent of 1,141 schoolchildren examined had gingivitis. Quoted in Glickman⁶⁴ and Dunning.⁴⁸

In 1938 Clements²⁶ and Kirkpatrick examined 530 European pre-school and schoolchildren of New South Wales. They found 63 per cent with gingivitis.

In 1939 Clements²⁷ and co-workers report 50 per cent had gingivitis after examining 350 aborigines, all ages.

Williams,¹⁶⁷ in 1939, examined 243 Samoan children, European and mixed decent 5-16 years of age, 53 per cent showed gingivitis. He then examined 712 Samoan children, Polynesian, 5-20 years of age and found 54 per cent showed gingivitis.

In 1940 Day^{44a} examined 203 children of one Kasur school in India, which was in a fluoride endemic area. He

no gingivitis in 82 (40.4%), mild gingivitis in 74 (36.4%), moderate gingivitis in 44 (21.7%) and severe gingivitis in 3 (1.5%). The incidence of gingivitis increased with age from 30% at 6-7 years to 69.51% at 9-12 years and 69.01% at 13-28 years. He concluded that the occurrence and severity of gingivitis was greater in the Kasur fluoride-endemic area than in the non-endemic Lahore area, a conclusion which, while supported by his figures quoted above.

In 1940, in India, Day⁴⁴ noted the occurrence of gingivitis in 756 children of one school in Lahore. The average age was 13.58 years. He distinguished only between slight and severe gingivitis. He observed that 38.69% showed slight gingivitis and 30.69% showed severe gingivitis. Approximately 31% are presumed to have exhibited no gingivitis.

In Queen Mary College, which is attended mostly by girls from five to 24 years and some boys five to 11 years (the sons and daughters of ruling princes and well-to-do families), 323 subjects were examined, the group being made up of 261 girls and 62 boys. Day⁴⁰ noted that only six of the 1,054 children (0.57%) examined of Islamia High School (middle-class persons) showed an absence of gingival disease. In all groups, the incidence of gingivitis was practically 100% (94.7-100%). Day^{44d} also showed that 73.62% of the

cases showed either severe or very severe gingival disease, 25.81% showed slight to moderate gingival disease and only 0.57% of the children could be classified as having normal gingiva. In Queen Mary College where 261 girls from the wealthy class were examined, both the incidence and severity of the disease were significantly less than in the Islamia High School. When the same age groups (9-17 years) from both places were compared, it was observed that the incidence of disease was 99.43% for the 1,054 boys and 73.74% for the 179 girls. On the other hand, if the 58 boys of the same social and economic status were compared with comparable 106 girls from the same class, they found that in the same age range (6-11 years) both the incidence and the extent of the disease were greater in the boys than in the girls, the incidence of gingival disease being 77.59% in boys and 70.75% in girls.

Schour and Massler (1947)¹⁴⁰ conducted a survey of the occurrence of gingivitis in post-war Italy in various age groups. The prevalence was determined from the examination of 3,905 persons aged 6-60 years in four communities. The incidence increased with age and ranged from 40% in the group 6-10 years old to 98% in the group 51-60 years old. The severity increased with age, starting with a mild type in the group 6-10 years old, extending through the marginal

gingivae in the attached gingivae in the 51-60 years old group.

Bruckner (1943)⁴⁸ examined 1,634 American school-children 4-16 years of age. Of the 819 boys examined, 735 (89.7%) showed healthy gingivae, 72 (8.8%) slight gingivitis and 12 (1.5%) severe gingivitis. Of the 815 girls examined, 757 (92.9%) showed healthy gingivae, 47 (5.8%) slight gingivitis and 11 (1.4%) severe gingivitis.

King and Franklyn (1944)^{76, 77} observed in the Gibraltar children the incidence of gingivitis and compared with the incidence in English boys of almost the same age and similar basal diet. Gingivitis, in general, occurred with similar frequency in English and Gibraltar children (77-79% and 85% respectively). However, when types were differentiated, a striking contrast between the English and the Gibraltariands was noted: only 1-2% of the former suffered from papillary lesions compared with 40% of the Gibraltar children. Of the English children, 75-86% suffered from marginal gingivitis, as compared with 45% of the Gibraltar children.

King (1945)^{76, 77} observed in the 90 children 11-14 years of age at the Harpenden Institution, gingivitis was present in 70% and absent in 30%. The incidence and extent

of disease were significantly greater in the children from lower economic levels than in those of higher economic levels. He observed, too, that boys in either category had more gingivitis than did the girls.

Day (1944)^{44c} also surveyed the prevalence of gingivitis in five villages near Hissar City. These villages were affected the worst in the famine district. The average age of the 314 children examined was slightly under 12 years. He observed a high incidence of gingivitis. Eighty-five per cent of the children showed mild to severe gingivitis of the hypertrophic and non-hypertrophic types. Seventy-eight per cent of the children suffered from hypertrophic gingivitis. A generalized hypertrophic gingivitis is rare in children of the United States. In these malnourished children of India it occurred usually as a hard, fibrous and painless swelling characterized by more or less uniform thickening hyperplasia of the marginal gingivae. From his observations it appeared that the occurrence of gingivitis increased greatly with undernutrition and malnutrition.

Day and Shourie (1944) examined 613 healthy Indian schoolchildren 5-15 years of age. In the group 5-10 years of age, 74.4% showed gingivitis, and 19.8% were free of gingivitis. In the group 11-15 years of age, 73.2% showed

gingivitis and 20.8% were normal. Quoted by Glickman.⁵⁴

In 1945, Schwartz¹⁴⁴ reported the following after examining Masai natives in Kenya, East Africa:

89 examined, age 4-19 years, 18% had gingivitis.
214 examined, age 20-39 years, 27.1% had gingivitis.
105 examined, age 40-up years, 61.9% showed gingivitis.

Dawson,⁴⁵ in 1946, examined 423 hospital patients in Cairo, ranged in age from 15-25 years. Ninety-five point nine per cent had gingivitis. 351 patients, aged 26-35 years had showed 99.6% had gingivitis. 200 patients, aged 36-up years and reported 100% had gingivitis.

In 1945 Shay and Smart^{145a} reported indications of a positive correlation between caries and gingivitis. They examined 98 airmen and found that two-thirds of those with caries had gingivitis while only one-third of those with no active caries had gingival inflammation. They offered statistical analysis to show that the difference was significant and they concluded that gingivitis was more prevalent in a carious mouth.

Day,^{44b} in 1944, and King,⁷⁶ in 1945, on the other hand, reported no relationship between caries and gingivitis. Day examined 200 malnourished boys averaging 14 years of age

in the Kangra Valley of India and observed a remarkably low incidence of caries. Thirty-six per cent of the subjects had no caries; the others averaged one and a half cavities per child and a high percentage of the lesions recorded were defective fissures not actual cavities. Gingivitis, however, was very prevalent; 81% showed from light to very severe conditions. He concluded that the high incidence of gingivitis in these children had no relationship to dental caries.

King^{76, 77} reported on 387 persons ranging in age from 12 to 30 years. He examined the gingivae approximating 165 upper incisors with proximal or cervical caries. These gingivae were found to be normal or only slightly diseased in 54% or a little more than one half of the areas examined and, therefore, King stated that there is no difference in the gingivae surrounding carious and non-carious teeth. He further noted that the frequency of caries in the upper anterior teeth increased with age while there is a tendency for the extent of the gingival lesions to diminish. These observations led him to conclude further that there is no relation between caries and gingivitis.

Thus we see the confusion that exists about an association between these two conditions even though here a casual relationship might be expected.

In 1957 Greene⁶³ examined 1,613 schoolchildren, Bombay, ranged in age from 11-17 years; and 557 males of the same ages in Atlanta. In addition 63 Indian males, 18-30 years of age, were examined in the rural area near Bombay. He concluded that 96.9% had gingivitis. Periodontal disease was highly prevalent in both groups but was significantly more severe in Indian than in Atlanta. Oral calculus was more abundant in the Indian group. The rural persons studied in India had more severe periodontal disease, more calculus, and more debris than those in urban India. When groups of similar oral hygiene status were compared, there still was a statistically significant between the periodontal disease scores in India and Atlanta, suggesting the presence of other altering factors.

The periodontal scores remained relatively stable for ages 11-17 years. However, the scores were considerably higher for ages 19 and above. The periodontal disease scores recorded for the low socio-economic group in urban Bombay were consistently lower than for the same ages studied in the nearby rural area of Basein.

Parfitt¹¹⁴ in studying the factors which affect gingivitis, emphasized the necessity for assessing severity as well as occurrence. He examined 255 boys and girls (with

ages ranging from 2-17 years) living in an institution outside of London. He reported that (1) prevalence and severity of gingivitis that reached a peak for boys at 13 years and for girls at 11 years, and (2) a seasonal incidence of gingivitis which showed slightly lower values for August, September, October and November than for other months.

In 1959 Russell¹³⁴ and co-workers studied 2,474 persons aged from under one to 86 years in South Vietnam. Civilian males and females scored substantially higher than their contemporaries in military service. There was a tendency for disease indicators to be higher in villages where typical diet was relatively low in total calories, protein, carbohydrate, riboflavin and iron; and relatively high in niacin and calcium.

Russell¹³² examined 28,926 white persons and 1,018 negroes ranging from five to more than 80 years of age. The great majority of the persons studied were 20 years of age or younger.

Both the prevalence and the severity of disease increase with age. At age 60 and over, about three-quarters of the white persons and about nine-tenths of negroes examined showed some overt evidence of tissue damage.

Negroes show a higher prevalence and a greater severity of disease than white at all ages. This difference may be due more to educational background than to race. But this one study so far seems to shed real light upon race factors.

Disease is slightly more prevalent in males than in females. When present at all, it may tend to be somewhat more severe in young girls than in young boys. It is more severe in males than in females after the age of 20 or 30 years.

A short-lived gingivitis may complicate comparisons between groups of children if they are examined at the peak or early in the declining slope of an outbreak of an upper respiratory infection.

Benjamin¹⁰ and co-workers in their study in rural children of 25 Indiana countries show that gingivitis was distinctly more prevalent in the younger rural children than in children in a series of urban groups. It is doubtful whether the observed differences can be attributed to rural life as such. Clear indication here that the prevalence or severity of periodontal disease may be influenced by the social or economic conditions obtaining in the communities in which they live. This possibility should receive further study.

In 1950 Day and Shourie⁴² examined 823 boys and girls, 91% Negro population, in Virgin Islands, ranged in age from 6-18 years. They reported that 57.1% had gingivitis.

In 1953 Davies³⁸ examined 497 Cook Islands, Polynesian, ranged in age from 1-40 years. He showed that 45% had gingivitis.

Dental examinations were recorded for 103 individuals of various age groups of the Australian aborigines by Cran.^{35, 34} In the survey carried out in May, 1955, an incidence of 58.5% of gingivitis was observed. This was mild in nature and consisted of two types: one a hyperplastic gingivitis associated with crowding of the lower anterior teeth in young subjects, and the other a very limited marginal gingivitis associated with a small amount of calculus formation around the few teeth, especially the cuspids.

In the 1956 survey,⁵ an incidence of 42.7% was observed. No significant change has taken place in the gingival conditions since the previous survey.

During these examinations by Cran, careful observations have been made on the depth of the gingival sulcus. Recession of the gingivae is uncommon in these subjects and is not generalized; when it occurs it is confined to isolated teeth, in particular the cuspids.

Barnard³⁴ revealed in his study of schoolchildren of New South Wales that 75% seen had some degree of gum infection.

In 1960 Rosenzweig^{128a} studied the prevalence of gingivitis in a sample of 4,500 children 13-14 years of age in Israel using P.M.A. index. He presented this finding, 43.11% to 82.62% of the children had gingivitis. Children born in Israel showed the least amount of gingivitis while the North African Jews and the Arabs showed the highest. In all groups gingivitis was more prevalent in males than in females. It may be that these sex differences in gingivitis are due to hormonal factors operating at 13-14 years of age. No significant differences were observed in gingivitis between children living in cities or villages. A positive correlation between gingivitis and malocclusion was established in both Jews and Arabs.

McCombie⁹⁹ and Stothard examined 1,501 metropolitan Vancouver schoolchildren, revealed positive relationships between poor oral hygiene and gingivitis. Children with more professional dental care had less gingivitis. 1,501 children ranging from the age of seven to fifteen years. They concluded (a) a positive statistically significant relationship between poor oral hygiene and the presence of

gingivitis has been re-affirmed. (b) A positive statistically significant relationship has been demonstrated between malocclusion and the presence of gingivitis, and specifically between crowding of any teeth, overjet and overbite and the presence of gingivitis. (c) Children with gingivitis have a significantly higher mean number of restored permanent teeth than do children classified as not having gingivitis. (d) A statistically significant relationship between the prevalence of gingivitis and the level of professional dental care, has been demonstrated.

✓ Ramfjord¹²⁵ and co-worker carried out a study of the periodontal status of boys 11-17 years old in Bombay, India. The main purpose of this investigation was to study the initial phase of destructive periodontal disease in the permanent dentition. The main survey included 827 boys from Bombay City and 791 boys from the Basein Road district. In addition, 59 males, 19-30 years old, were examined at a teachers' college in the Basein.

✓ They show that almost 100% of the individuals examined had periodontal disease. True periodontal pockets did not occur to any significant extent before the age of 15 years. The periodontal disease increased slowly in severity with increasing age, although gingival inflammation decreased

slightly in the middle teens. The amount of calculus increased with age. The amount of bacterial plaques decreased from 11 to 15 or 17 years of age; later it increased. A direct relationship was observed between the amount of calculus and the severity of periodontal disease. The most severe gingival inflammation and pocket formation occurred around the lower incisors and the maxillary first molars. These teeth had the highest calculus scores. The lowest periodontal disease index score was for the mandibular first bicuspid. All periodontal disease was clearly of inflammatory nature (gingivitis and periodontitis).

✓Russell¹³² related education levels to the prevalence of periodontal disease. Those men attaining higher levels of formal education had less periodontal disease. Periodontal disease is also less severe in individuals with higher general educational backgrounds.

Gingival inflammation appears to be less frequent and less severe in occupations which require greater skill and more educational background. The incidence of gingivitis is lower in office personnel than in factory workers.^{1, 93}

Stammeyer¹⁵⁰ found that gingivitis was negatively correlated with the frequency of effective toothbrushing.

Shklair and co-workers found that institution of regular toothbrushing reduced the incidence of gingivitis in a recruit population. Davies and co-workers related education levels to the prevalence of periodontal disease. Those men attaining higher levels of formal education had less periodontal disease. Van Der Aue, and Cullen reported greater incidences of gingivitis in the submariners and further found that the crews exhibiting poorest oral hygiene had the greatest incidences of gingivitis.

✓ Mehta¹⁰³ and co-workers carried out a study of prevalence of periodontal disease in India child population in relation to their socio-economic. A selection of secondary boys' schools, age 11 to 17 years. Their findings show a very high incidence of periodontal disease in children between the ages of 11 and 16. There is no significant variation in the incidence of disease in the different socio-economic groups. The severity of the disease increases with low socio-economic group and decreases with the higher socio-economic group. It is pointed out that it would be dangerous to use a particular section of the mouth to denote the disease experience of the entire mouth.

Vivone¹⁵⁷ carried out observations on persons of

different ages in South America, members of each group being subject to similar environmental conditions. Average ages were as follows: girls 16.3 years; males 14.9, 17.8 and 21.1 years. Both sexes: between 23 and 30 years. From this study the following points are:

1. The lesions of the paradentium are very frequent and varied.
2. ~~Early~~ diagnosis is indispensable.
3. It is possible to discover systemic states by means of the study of parodontal disease must be undertaken by the specialist and by the general practitioner. It benefits the health of the mouth and teeth and contributes to the shaping of Public Health Dentistry.
4. Those who are careful about oral hygiene have fewer parodontal manifestations than those who neglect this prophylaxis.

✓ Massler⁹⁴ reported a study based on the examination of 804 children; 381 from the Melrose Park Luterhan School and 423 from Mooseheart. The children ranged in age from 5-14 years and included an approximately similar distribution of boys and girls. It showed that 64.3% of the 804 children examined showed one or more of their gingival papillae affected by gingivitis, 35.9% showed mild, 12.8% moderate,

9.2% severe and 6.5% very severe gingivitis. The percentage of children affected with gingivitis increased significantly with increasing age. It increased very rapidly from 9.1% at five years to 69.3% at seven years. The percentage rose slowly thereafter to a peak of 80% of children affected at 11 years. The character of the inflammation as observed clinically left us with the impression that the sharp rise in gingivitis observed at 7-8 years of age was caused primarily by the eruption of the four incisor teeth and the first permanent molars. The slower eruption of the bicuspid teeth and the fact that these latter teeth appear over a much longer period of time (9 to 12 years).

Bossert¹⁶ and Marks carried out intensive statistical study of the prevalence of periodontal disease among 12,800 employees at the home office of the Metropolitan Life Insurance Company in New York City. They show that periodontal disease was increasingly common with advancing age. By the age of 48 more than half of all persons were affected, and in most instances the process was active. The prevalence was slightly lower than in men up to age 55, and much lower after that age. Extractions for periodontal disease occurred in about one out of every five persons by age 45. A somewhat greater number of lower teeth were affected than

upper teeth, but many more upper teeth had been extracted for periodontal disease than lower teeth. In the upper jaw the teeth most susceptible to periodontal disease were the left molars, particularly the left first molars, the least susceptible teeth were the cuspids. In the lower the most susceptible teeth were the incisors, particularly the central incisors, the least susceptible teeth were the first bicuspids.

Belting⁸ and Gupta surveyed the periodontal conditions of 105 patients receiving psychiatric treatment and 117 patients receiving medical care other than psychiatric treatment at the Outpatient Clinic of the Veterans Administration West Side Hospital. All patients were male veterans between the ages of 20 and 25. The periodontal lesions were evaluated by the method of Russell without X-rays. The experimental group of psychiatric patients showed a higher incidence of periodontal disease than did the control group of non-psychiatric. The increase was highly significant in each age group up through age fifty. Among those persons brushing their teeth twice a day, the incidence of periodontal disease also was significantly higher in the experimental than in the control group, with the exception of the years between 31 and 40. This eliminates the variable factor of oral

hygiene care as a possible cause affecting the difference in incidence.

Cohen²⁹ and co-workers evaluated periodontal conditions in 300 male and female institutionalized, mentally subnormal children aged three to eighteen. This group consisted of both mongoloid (100 cases) and non-mongoloid patients (200 cases). All the mongoloid children were found to have periodontal involvement varying in degree from moderate to severe. In addition to periodontitis, a superimposed necrotizing gingivitis was noted in many cases. There was radiographic evidence of bone loss in all cases studied. This was characterized by horizontal resorption and loss of crestal lamina dura. Oral hygiene was poor, and calculus, materia alba were abundant, but it was apparent that tissue breakdown was considerably more severe than expected in this age group despite the local factors present. Nutrition in this group was excellent and has been carefully evaluated. Periodontal disease was more prevalent and more severe in the mongoloid group than in the non-mongoloid, mentally subnormal patient.

Necrotizing Ulcerative Gingivitis (Vincent's)

Sorrin¹⁴⁶ carried out a study at New York University College of Dentistry; it was found that the greatest incidence

occurred between the ages of 19 and 30 years and (in New York) during the month of October.

Grupe⁶⁵ and Wilder observed acute necrotizing ulcerative gingivitis occurred in 2% of newly inducted army personnel in the age group of 17-21 years. During the initial examination of 2,622 trainees 58 cases or 2.2% of those examined had Vincent's infection. They say a psychosomatic factor seems likely as the predisposing agent, acting primarily on the papillary arterioles that resemble end arteries.

Pindborg¹¹⁸ and co-workers (from April 10, 1964 to August 10, 1964) examined 10,000 patients coming to the Admission Clinic at the Dental College, Bangalore India. Among the 10,000 examined a number of 236 patients (2.36%) with acute necrotizing gingivitis were diagnosed. Four of these patients had simultaneously a herpetic gingivostomatitis. Furthermore, ten cases of acute herpetic gingivostomatitis were seen. It is striking that 58% of these patients were below the age of 10. The male:female ratio for patients with acute necrotizing gingivitis was 1:1 below the age of 10, and 3.7:1 above the age of 10. Distribution according to religion 2.7% among the Hindus suffered from acute necrotizing gingivitis, 4.75% among the Moslems had the disease.

Atrophy

Miller¹⁰⁶ described three types of precocious advanced alveolar atrophy:

- (a) early (5 to 7 years of age, involving incisors and molars);
- (b) late (11 to 13 years of age, involving cuspids, bicuspid and second molars); and
- (c) combined (early involvement of all teeth).

Miller and Seidler¹⁰⁷ suggested that developmental aberrations and nutrition and metabolic diseases were causative. Hulin ascribed the condition to inherited pluriglandular dysfunction. Vincent pointed out that the stepped up nervous tension and excitement in children and the emotional and psychologic imbalances are factors not to be neglected in the study of causes of early retrogressive changes, since they do produce emotional maladjustments in adolescence that are reflected in physical disorders.

Russell¹³⁴ and co-workers examined over two thousand Vietnamese aged up to 86 years. They said extensive pocket formation and destruction of supporting tissues were seen in 56% of civilian females and 67% of civilian males 25 years of age or older. The mean gingival recession scores for military personnel and civilians were not markedly different

from military males in the United States but showed much lower scores than their civilian counterparts. Scores for females were slightly lower than scores for civilian males. There was a general tendency for the higher P.I. and recession scores to occur in villages with the lower intakes of calories, protein, carbohydrates, riboflavin, and most particularly, of iron and relatively high in niacin and calcium.

Stahl and Morris¹⁴⁸ in a study carried out for the American Army used Gingival Recession Index. They counted the number of teeth in which gingival recession had exposed the cemento-enamel junction, and the total teeth present in each mouth. Then they expressed these values as percentage score of recession for an individual. The whole group score was computed as the mean percent per man.

Ramfjord¹²³ in his studies with Indians concluded that with increase of periodontal disease and increase of gingival recession takes place.

Marshall-Day³⁹ and co-workers study of total recession of gums without pocket formation shows a fairly good correlation with bone resorption.

Recession of gingiva and resorption of alveolar bone are closely associated as shown by Sandler and Stahl.¹³⁸

Hyperplastic Gingivitis

Ziskin¹⁷² and co-workers have supported the theory of a gingivi is due to pregnancy and caused by hormonal changes; they carried out studies in 1933, 1937, 1938 and 1941. They observed that the condition usually improves so rapidly after parturition without any treatment being carried out, and without any extra care being exercised by the patient, one must look for systemic causes, possibly hormonal, particularly as the local conditions remain the same except that the body has reverted to its nongravid state.

Maier and Orban⁹¹ in a study of 530 pregnant women from third to ninth month of pregnancy, divided the patients into the following groups:-

1. No pathology	236 or 44.6%
2. Mild inflammation	190 or 35.9%
3. Moderate inflammation	93 or 17.5%
4. Severe inflammation	8 or 1.5%
5. Tumour formation	3 or 0.5%

They said there is no significant differences in the incidence of gingivitis in pregnant and non-pregnant individuals. They also mentioned that there are some clinical features which at least point to an endocrine unbalance.

Hilming⁶⁶ in a survey of 203 pregnant women, found that

100% of them had gingivitis in some form and 47% had a definite specific form of gingivitis gravidarum and 53% a non-specific gingivitis gravidarum. The latter referred to a gingivitis which was present during the pregnancy, but not due to it, and was not ameliorated by the termination of the period of gestation. As he points out, it is impossible to have accurate criteria for a pregnancy gingivitis beforehand, and it is logical to expect that a gingivitis which is causally connected with pregnancy would be ameliorated after delivery. This is the basis of his determining whether the patient had a gingivitis gravidarum, or not.

Reed¹²⁷ favours a dietary cause. He exaggerated his statements of possible end results and his method of overcoming it with diet.

Gridley,⁶² in 1954, carried out a survey of 1,002 gravid women in Egypt. These were mostly of poor and lower middle class aged between 17 and 38 years, in their fourth to ninth month. He found the following:-

- N. Norman gingivitis: 14%.
- C. Common gingivitis without hyperplastic signs: 60%.
- H. Hyperplastic gingivitis: 23%. (Among this group 30 cases, or 30% showed accentuated hyperplasia.)

T. Pregnancy tumour: 2.7% or 27 cases showed pregnancy tumours. Most of the pregnancy tumours occurred at the upper right lateral incisor region, where gold crowns are inserted as ornaments amongst the lower classes in Egypt. The hyperplastic gingivitis and pregnancy tumours were considered as definitely due to pregnancy.

In New Zealand, Wilson and Skinner¹⁶⁸ examined 321 pregnant women and 38 women seen one month post partum. A group of 80 non-pregnant women was also examined. Comparing the incidence of gingivitis in pregnant and non-pregnant control groups (Europeans only) they summarised:

	<u>Pregnant</u>	<u>Non-Pregnant</u>
Number of cases	174	50
Class I Normal	50%	76%
Class II Mild Gingivitis	37%	16%
Class III Moderate Gingivitis	8%	8%
Class IV Severe Gingivitis	5%	Nil

They calculated the difference between the figures for Class I and there was significant

Rohackova and Ticha¹²⁹ in Czechoslovakia, in examining 187 pregnant women, found about 80% temporary genuine hypertrophy of gingival tissues. In the control group of non-pregnant women, different types of gingivitis with symptoms

less severe than those of pregnant group, occurred only in 25%. In 1.8% of patients with pregnancy gingivitis, pregnancy tumours were observed.

Gonzales⁶⁰ examined 379 pregnant women. 71.5% of these had gingivitis. He classified them thus:-

Erythematous	36.4%
Hypertrophic	13.5%
Ulcerative	2.1%
Gravidic	19.5%

A survey⁸³ of 684 pregnant women varying in age from 13-45 years, married and single from all social strata with the majority coming from the lower income. Three hundred and thirty-three were diagnosed as having gingivitis gravidarum according to Ziskin's classification and scored by the Russell Periodontal Index. This is a high incidence of gingivitis in the pregnant group in Sydney. In those diagnosed as gingivitis gravidarum, no statistical significance could be attributed to age, number of pregnancies or weeks of gestation, although there was some diminution of the condition in the ninth month. The frequency of brushing was directly related to the incidence of the gingivitis, showing that those who brushed more frequently were less inclined to suffer from the condition. The Greek, Maltese and Italian groups exhibited the highest incidence of gingivitis and

these were the groups which brushed less frequently. No clinical test carried out as to the part played by hormonal changes during pregnancy. There were no obvious cases of nutritional deficiency amongst these patients, but the highly farinaceous diet of some groups did appear to cause irritation due to deposited debris.

The importance of local factors in gingivitis in pregnancy cannot be minimized but the role played by systemic disturbances due to changes in hormonal balance must be considered.

Prichard¹²¹ says "hormonal disturbances are known to affect the periodontium. During pregnancy pre-existing gingivitis tends to become more severe and it may change in character. Hyperplasia of the marginal gingiva occurs most often at puberty when there may be a temporary endocrine imbalance. The severity of gingivitis may vary with menstrual periods".

Erb and Brazezinsky⁵¹ examined 146 pregnant and 123 non-pregnant women; 53 (36.3%) of these pregnant women were born in Israel and 93 (63.7%) were newly arrived immigrants. Among the non-pregnant group, 44 (35.7%) were born in Israel and 79 (64.3%) were immigrants. They found that 70.7% of

the 146, those pregnant (gravidae) showed gingival disorders of varying degrees. 60.3% of the 53 Israel born pregnant women suffered from gingivitis while 76.3% of the 93 new immigrants were thus affected. A greater prevalence of gingivitis gravidarum was in the 26-30 age group (34.3%) whereas among the immigrants it was in the 21-25 age group (38%). These differences were not statistically significant. In general the age of the pregnant women was not correlated with the incidence of gingival disorders. Immigrant women showed poor oral hygiene and had more frequent gingivitis. The greater number of gingival disorders occurred in the anterior segments of both the maxilla and mandible.

Lighterman⁷⁹ and Lees stress the hormone and vitamin factors as the cause, and place irritation as a secondary factor, because many patients with gingivitis have no observable irritation, and others who are pregnant, with periodontal irritation, show no signs of the condition.

They include other etiological factors, such as alteration of endocrine function, increase dietary demands of the body, a complex interaction of diet and hormones, periodontal irritation, and neglected oral hygiene, all acting directly on the patient and being counteracted by the patient's resistance.

Bain² suggested that the prolonged stress of pregnancy may cause, in some women a mild adrenal cortical insufficiency and because of local or systemic factors, an inflammatory progress may develop in the gingivae. As there is little or no cortisone or hydrocortisone available in the circulation to combat the inflammation, the process goes unchecked.

Sidney Sorrin¹⁴⁶ says that gingivitis associated with pregnancy is an exaggerated inflammatory response to local irritants modified by the systemic hormonal changes. With good oral hygiene and no local irritants, the gingivae will remain normal and the gravid state alone will not cause inflammatory changes.

Compertz³⁰ found in a survey of 200 pregnant patients, that general nutrition and dietary habits seems to make a difference in the mouth conditions. Women who were under-nourished had more periodontal pathology.

Levy⁸² states that scurvy manifests itself in pregnant women as a result of various faddist diets, distaste for food containing Vitamin C, inability to keep food in the stomach due to hyperemesis gravidarum and diets prescribed by attending physician for gastro-intestinal disorders.

Cross^{34a} says that gingivitis gravidarum and pregnancy

tumours, the most important factors are Vitamin C deficiency, hormonal alteration and trauma. He gives large doses of ascorbic acid daily as treatment and thorough antenatal prophylaxis.

Reviewing all of the studies employing the P-M-A Index on subjects from 2-50 years of age Schour and Massler⁹³ reported that the analysis of all their data revealed that the gingivitis attack rate is characterized by two phases.

The first phase demonstrates a rapid rise from a low point at five years of age (9% of children affected) to a peak at 11-13 (65%). This is followed by a decline to a plateau at about 16 years of age, after which little to no increase between ages 16-25 occurs.

At age 25, the second phase begins with a slow increase in gingivitis in contrast to the first phase. On a clinical basis, the two phases can be readily distinguished with an acute inflammatory response characterizing the first phase, and considered to be self-reparatory since it is related to eruption of the permanent teeth and crowding and shedding conditions associated with the years of mixed dentition.

Phase two, beginning after age 25, is characterized by chronic inflammation and is not self-corrective, but, rather

tends to be accumulative. In all the studies evaluated by the investigators, gingivitis was found to progress usually from the papillae to the gingival margins and finally to the attached gingivae, with the upper and lower anterior region bearing the brunt of the gingivitis attacks.

SUMMARY AND CONCLUSION

Up to the present time, there have been no uniform criteria of assessment for the measurement of periodontal diseases, and until there is agreement on this basic consideration, little or no reliable statistical data on the epidemiological characteristics of periodontal diseases can be ascertained, nor can the effects of any control procedures adopted, be measured. Advancement in our knowledge of the periodontal disease process has now reached a point where the development of standardized criteria for its measurement is critically needed. Epidemiological research is necessary for a further understanding of this disease process and will open the way to new methods of investigation.

There is wide variation in the reported prevalence of periodontal diseases due primarily to lack of uniformity in the methods of assessment used, and secondarily to inherent

differences in the sample populations examined. The studies cited in this review have established the high world-wide prevalence of periodontal disease.

There are two patterns of consistency in the reported morbidity rates of periodontal disease:

- (1) the age of onset occurs between 10 to 15 years, and
- (2) the peak incidence is reached between 40 to 50 years of age.

Periodontal disease is slightly more prevalent in males than in females. It is more severe in males than in females after the age of 20 or 30 years. At age 60 and over, about three-quarters of the persons examined showed some overt evidence of tissue damage. When present at all, it may tend to be somewhat more severe in young girls than in young boys.

Since periodontal disease constitutes a major public health problem, the public health dentist is the logical specialist to conduct further research on its epidemiology. Further useful epidemiological research on periodontal disease is dependent upon the development of a standardized method of assessment.

Evidence that periodontal disease is less prevalent and

less severe in people of the more favoured social status has been adduced from such diverse populations as rural children in Indiana, workers in a manufacturing plant in Oslo, and schoolchildren in Bombay. These differences in the prevalence of active disease are reflected in higher rates for teeth loss from periodontal destruction in persons from less favoured status groups.

There are many local etiologic factors for gingival and periodontal disease, but poor oral hygiene overshadows all others. There is a high correlation between the presence of plaque, calculus, oral debris, and poor oral hygiene and the prevalence and severity of gingival and periodontal disease.

Clinical studies of causes of tooth loss, show that low-income patients lost more teeth to periodontal disease than to dental caries. More than 80% of these patients were Negroes. Studies have shown that Negroes had consistently scored higher than whites in the same localities when examined for active periodontal disease.

Females were consistently found to have less periodontal disease than males, though the differences are not very great. Females show a significantly lower bone loss of the periodontitis variety than do males, but a relatively higher

incidence of loss from periodontosis.

A number of studies employing various evaluative procedures found periodontitis increasing with age, as did gingivitis, in both prevalence and severity.

Degree of urbanization appears to be related to periodontal disease. In two studies of U.S. children, rural children were found to have significantly higher group scores than did urban children, particularly at the younger ages.

Degree of education is inversely related to the severity of periodontal disease once it has started.

Clinical impression has existed that periodontal disease is more common in persons with emotional disturbance.

With increase of periodontal disease an increase of gingival recession takes place.

A great many of the studies reviewed considered gingivitis alone and a summary of the findings is as follows:

Incidence of gingivitis: A comparison of data on the percentage of children with gingivitis in England, Wales, India and America showed that approximately 40% to 70% of the children were affected by gingivitis.

Studies showed that the percentage of children affected with increasing age. The increase was found not only in the number of children affected, but also in the severity of gingivitis.

Many studies indicate that the occurrence and severity of gingivitis tends to increase with an increase in malnutrition. But there is little indication in data that periodontal diseases can be prevented to any important degree by the improvement of nutrition, unless accompanied by a thorough scaling and polishing of all the teeth (dental prophylaxis).

The frequency of brushing was directly related to the incidence of the gingivitis, showing that those who brushed more frequently were less inclined to suffer from the gingivitis. People exhibiting poorest oral hygiene had the greatest incidence of gingivitis.

The severity of the disease increases with low socio-economic group and decreases with the higher socio-economic group.

Most of the studies show a positive correlation between gingivitis and malocclusion.

Gingivitis was more prevalent in a carious mouth.

The chief lesson to be learned from epidemiological surveys such as these, is that periodontal disease is at least as important as dental caries as a dental health problem. And if this is true of the younger age groups, by the time early middle age has approached, caries have virtually ceased to be a problem and periodontal disease has assumed major proportions.

PREVENTION AND CONTROL OF PERIODONTAL DISEASES

Prevention of periodontal diseases is based on the knowledge of aetiology and pathogenesis. If the cause of disease is known, it usually can be prevented.

Marginal gingivitis and periodontitis are the most common of periodontal disease, and fortunately are preventable ones. They are caused by local factors which are accessible, correctable and controllable.¹³³ In most gingival and periodontal disease inflammation is the predominant if not sole pathologic process and local irritants combined with micro-organisms are its principal causes.

Periodontosis is not preventable because the aetiology is still unknown.

It is axiomatic that gingival and periodontal disease must be recognized early and treated as soon as they are discovered.¹⁵⁷ It is simpler to treat slight gingivitis than severe gingivitis, to eliminate shallow pockets rather than deep pockets and to prevent bone destruction and osseous defects rather than correct them.

It is essential that all oral lesions be recognized and treated as early as possible,¹⁵⁷ and that the dental care for every patient be as comprehensive as practicable. Sydney Levine⁸¹ says "if we can understand the nature of periodontal disease and recognize its aetiology, then, and only then, can we formulate treatment plans and attack this problem both at the dentist level and at the public health level". Early recognition and treatment ensure a maximum opportunity for success with a minimum expenditure of effort. It promises greater satisfaction for all. The opportunity for early detection is most apt to occur in the dental office.

A great deal has been written on gingivitis stating the earliest manifestations of the disease process appear in the marginal and interdental gingival tissues and progressively involve the attached gingiva. Since the untreated or unnoticed gingival pathology will eventually involve the supporting structures of the teeth, the recognition and treatment of the early gingival lesion seems to be the most practical solution to reduce the incidence of the more advanced form of periodontal destruction. To detect the incipient changes from a normal gingiva to a tissue that is pathologically involved demands the ability of an astute diagnostician, since the alteration is often quite subtle.

INDIVIDUAL

Person's Role in Prevention and Control of Periodontal Disease

In this aspect we are concerned with (a) one person with a complaint to treat and, (b) a community composed of many persons, some with complaint, some undiscovered disease, and some healthy.

Why the patient seek dental treatment or dental advice? The answer may be one main symptom, such as bleeding gums, or it may be a variety of complaints, all of which should be noted carefully for future reference.

The patient should realize that the major part of the success of the treatment depends on his or her co-operation to give proper history, symptoms and other relevance of his complaint. This helps a lot for the dentist to establish correct diagnosis and proper treatment planning. It is accepted generally throughout the health professions that which is not diagnosed cannot be treated, while that which is diagnosed incorrectly cannot be otherwise than treated wrongly.

The patient should confide in the general practitioner or the dentist who treats him. Thus creating a good relationship and a better mutual understanding between the dentist

and the patient. The patient should be given full instruction of post-operative and home care to carry out. Treatment is in vain unless the patient is instructed in methods designed to maintain adequate oral health.

Person or persons may minimize periodontal disease by dental home care (chiefly tooth brushing), and the systemic disease. Home care for the periodontal patient is somewhat more exacting than home care for the control of caries.⁶

Tooth brushing must be carried out with a particular effort to reach those interproximal embrasures which so often accompany periodontal damage.

Person or persons should visit the dentist at least twice a year.

Toothbrushing

Hoover⁷⁰ has demonstrated that the toothbrushing retards the onset of gingivitis.

Studies^{85, 86, 89} have shown that toothbrushing reduces the incidence of gingivitis. Lovdal⁸¹ and others have shown that subgingival scaling followed by controlled oral hygiene reduced the incidence of gingivitis by from 50% to 90% depending upon the individual oral hygiene habits previous to treatment.

Glickman and co-workers have indicated that interdental gingival inflammation can be reduced by as much as 26.3% by combining interdental cleansing with toothbrush.⁵⁶

Braudtzaeg and co-workers showed that improved oral hygiene alone suffices to improve periodontal health.

Studies have shown that cessation of oral hygiene measures leads to gingivitis which is eliminated when they are reintroduced.⁵⁶

Toothbrushing reduces plaque and accumulated debris and reduces calculus.⁶⁹

The proper use of a suitable toothbrush may be considered the alpha and omega of periodontal management, as upon it depends not only the correction of many common forms of gingival inflammation, but also the successful post-operative outcome of any mode of periodontal treatment. If the patient is not instructed as to the proper type of toothbrush and its correct application to meet the particular requirements of the case, failure may then be expected, regardless of the mode of therapy that was employed.^{68, 89}

DENTIST

The first step in prevention and cure of periodontal disease is to recognize and eliminate local and systemic conditions. The dentist who detects and treats periodontal disease in its earliest stages, and for a much larger number of patients, renders a far greater service to the public.

It is possible that the general practitioner sees almost as many periodontal as carious lesions. It has been shown, however, that only a small percentage of the affected patients are aware of the presence of periodontal disease, and that an even smaller percentage have received any treatment. The need for adequate examination, investigation and recognition is imperative.

Early identification and classification of all periodontal lesions is imperative. Examination procedures, oral diagnosis and treatment planning should all be as comprehensive as practicable for each patient. Periodontal diseases are extremely prevalent, and because there are relatively few certified specialists in periodontics, the role of a well-oriented, conscientious general practitioner in dealing with the problem is extremely important. He has a real opportunity available to render a well balanced program of treatment for the individual patient.

Dunning⁴⁸ gives three main methods by which periodontal disease may be minimized. These involve (i) dental prophylaxis, (ii) dental home care (chiefly toothbrushing), and (iii) the systemic strengthening of gingivae through proper nutrition and the elimination of systemic disease. Diagnosis is usually followed by systematic local treatment, and thorough general physical check-up with treatment of all systemic conditions.

The dentist must educate the patient to the point of view that teeth are worth preserving for life, and that they can be preserved for life if he is prepared to co-operate fully in maintaining good oral hygiene. This is the responsibility of the dental profession and while there are other forms of patient education, that which rests on the personal relationship of the practitioner and his patient can be the most effective and is the most important.

It must also be emphasized that so much of chronic periodontal disease is iatrogenic,⁹⁹ that is, produced by incorrect dental treatment. The practitioner's obligations are therefore twofold; he must practise sound dentistry and he must educate his patients in oral hygiene techniques.

If treatment has been carried to a successful conclusion, the responsibility for maintenance rests largely with the

patient. No effort should be spared in making the patient aware of this vitally important phase of therapy.

If co-operation is poor the benefits of treatment will not be maintained, and no matter how skillfully treatment has been rendered, it will fail. It is the dentist's duty to ensure that the patient has been instructed in a correct program of oral physiotherapy. It is also necessary to supervise this program, checking progress periodically.

The patient should be recalled as often as is necessary to maintain satisfactory oral hygiene, and to arrest the incidence of fresh caries. If the patient will not, or cannot, keep the mouth clean and healthy, treatment may have to be discontinued or drastically revised. At these recall appointments the patient's co-operation should be reviewed, and treatment procedures re-evaluated.

An efficient recall system is a fundamental factor in this program. On recall, cavity detecting radiographs are made, and the health of the periodontal structures is checked. The teeth, the periodontal tissues, and the occlusal function should now conform to the ideals described earlier.⁹⁹

AUXILIARY1. Dental Hygienist

The role of the hygienist assumes growing importance as dentistry becomes increasingly oriented toward preventive services, particularly those directed toward disease of the gingiva and supporting tissues in the mouth. The hygienist also extends the services of the dentist to more patients by relieving him of those professional duties for which she has been trained and licensed, and allowing him to devote all his time to those services which only he is trained to do. It has been estimated that the amount of dental care could be increased by 25% if dental hygienists were utilized to the maximum extent.

W.H.O. gives the definition and functions of the dental hygienist: "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 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".

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 of or primary examination of groups, such as schoolchildren 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 nursery services to ensure effective follow-up of recommendation 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 disorders included officially in their training curriculum.

If we are to broaden the scope of periodontal service and improve the health of our population, we must depend more

on auxiliary personnel, particularly the dental hygienist. Since much periodontal disease begins at an early age and is caused or aggravated by neglect, the role of the hygienist in health education and preventive treatment assumes increasing importance. More and more people, particularly children, should receive careful training in proper oral hygiene. The hygienist is qualified to undertake this important task.

Stoll^{152a} gives the main responsibility of the dental hygienist as follows:

1. To build confidence in the patient that the dental services he receives are the best for his individual needs.
2. Dental care is a continuous process. Regular dental appointments are to be expected and the patient will be notified for periodic dental prophylaxis, x-rays and examination by the dentist at specific intervals.
3. The patient is motivated to carry out his part of the dental care program, by assuming responsibility for his own dental cleanliness through toothbrushing, gum massage and flossing.
4. The dental hygienist is capable in preparing comprehensive dental histories, thus saving

much time for the dentist and the patient. Her understanding of total health in relation to dental health provides a background for obtaining facts that might otherwise be overlooked.

5. The dental hygienist can allay fears, misapprehensions and misunderstanding in reference to the dental services to be undertaken.
6. Most important is her ability to teach with scientific accuracy, dental health, personal nutrition needs and recommended procedures.

Hygienists are trained to teach the principles of dental health to individual patients and to group audiences, and the few practitioners who employ such auxiliaries for this purpose speak highly of their work. It is not suggested that this work should no longer be the responsibility of the dentist in general practice, but that wherever appropriate it should be delegated to suitably trained persons.

2. New Zealand Dental Nurses

They are trained to carry out a very clearly defined and limited range of dental operations on pre-school and schoolchildren, including teaching them good oral hygiene habits, and they are under the supervision of fully qualified

chief dental officers of the Division of Dental Health of the Department of Health.

The dental nurse aims to prevent dental disease by early and regular inspection, by dental health education - both of the children and their parents, including the encouragement of local support for fluoridation - and by the systematic early treatment of carious lesions which, in turn, contribute to the prevention of malocclusion and periodontal disease.

3. Dental Assistants

The Expert Committee on Auxiliary Dental Personnel of the World Health Organization, surveying current utilization of dental assistants, lists these duties:

1. Reception of the patient.
2. Preparation of the patient for any treatment he or she may need.
3. Preparation and provision of all necessary facilities, such as mouthwashes, napkins, receivers.
4. Sterilization, care and preparation of instruments.
5. Preparation and mixing of restorative materials, including both filling and impression materials.

6. Care of the patient after treatment until he or she leaves, including clearing away of instruments for re-use.
7. Preparation of the surgery for the next patient.
8. Presentation of documents to the surgeon for his completion, and filing of these.
9. Assistance with x-ray work and the processing and mounting of x-rays.
10. Instruction of the patient, where necessary, in the correct use of the toothbrush.
11. Aftercare of persons who have had general anaesthetics.

Flein found that the addition of one dental assistant increased the number of patients treated by a dentist by 33% if he were using one chair and by 62% if he were using two chairs. Later studies made by the U.S. Public Health Service in Richmond, Indiana, show that a dentist can double his output with the help of one full-time and one half-time assistant. Increases of this sort depend upon standardized operative techniques which use a minimum number of instruments, and careful indoctrination of the assistant to anticipate the operator's needs. Quality of service and control of patient are both improved under such a system.

4. Dental Laboratory Technicians

This group has little impact in the fields of preventive dentistry and dental care for young children, but affects in a very important way the efficiency of dental treatment for older patients. In Tasmania, dental mechanic deals direct with public which may be likely to cause iatrogen as a result of their work.

SPECIALISTS

Unfortunately, those who perform elaborate periodontal therapy and equally elaborate full coverage rehabilitation for cases of advanced periodontal disease are too often mistaken as being the ultimate experts of the profession. These procedures are of scant significance from a public health standpoint since probably less than 1% of the public have the fortitude or the funds to avail themselves of this type of service.

In the medical and dental professions the specialists' activities are today based largely on the hospital service; a certain amount of consulting and specialist work is carried out under private contract, a large proportion of which is financed by the insurance societies.

Specialists in periodontology are confined to the teaching hospitals; only a very few are found in private practice.

Specialist services should always improve the service rendered by a profession to the public; to the individual professional man the sharing of problems with colleagues who have a special interest or who have made a special study in particular aspects of a subject, must be to their immense mutual benefit. It is important for the profession to recognize that it is within the precincts of the hospital that the professions of medicine and dentistry come into their most intimate contact.

COMMUNITY

In dental public health, education of the public is as important as curative services. Dental health education is therefore a major endeavour for the public health dentist. It should be aimed not only to motivate people for desirable dental health practices but also to influence the public to participate in and support dental health programs.

A dental public health program should be adequately staffed, financed, and tailored to fit the needs and resources of its locale. It should be planned to (a) apply

community methods for the prevention of dental diseases; (b) carry out effective education in dental health through the schools and the community organizations; and (c) attempt to ensure that all members of the community receive dental care.

The periodontics department in the hospital would be in the position of continuing, under the most ideal conditions, its primary functions of examination, diagnosis, and treatment of the disabilities, diseases and destructive processes which affect the oral mucosae and the investing and supporting tissues of the teeth. One of the drawbacks of periodontal practice in the private office is the relative difficulty of expeditiously procuring the laboratory examinations which can contribute so much to the diagnosis of systemic factors in periodontal disease. Because of the normal existence of these facilities in the standard approved hospital, this drawback is eliminated.

Preventive dentistry is identified with personal preventive practices, with community efforts and with public agency-sponsored programs. It is dynamic, inclusive and clinical. The hospital is an ideal place to implement preventive dental philosophies.

Manpower

It is necessary to determine those factors which will influence the dental manpower requirements of the future.

Some of these can be detailed as:

1. An alteration in the size of the population. The ratio of dentists to population.
2. An increase in national productivity. With this the standard of living of the community rises which in turn means an increase in the demand for dental care.
3. An increase in dental awareness. This will result in prolongation of the life of the natural dentition. The overall increase in the need for treatment and the more time-consuming nature of the advanced forms of therapy associated with this should be remembered.
4. A decrease in the average working week of the profession. In this connexion the professions always lag behind the primary producers of wealth, but there is no doubt that the trend is for all individuals to work whorter hours. A reduction in the dentist's working week of one day would for example necessitate an additional more dentists to maintain the present-day standards of the service alone.

5. Advances in research and operative techniques. Many of these, as yet unknown and unpredictable, could have a marked effect on the future trends of practice, as well as the incidence of disease.
6. The use which is made of ancillary help of all types. These can be broadly classified into those who by direct assistance to the operating dentist enable him to do more work in the same amount of time, or those who by relieving him of some of the simpler forms of treatment enable him to undertake other responsibilities.
7. Fluoridation of water supplies. If this is carried out on a national scale it would produce a reduction in the need for treatment of caries in these individuals.
8. Improvements in habits of diet and oral hygiene. This could reduce the incidence of dental disease in all age groups.

Those items which are related to a need for an improvement in the public appreciation of dental health will be slow to develop because of the inevitable delay in the effect of dental health education, and because they are dependent largely upon the priority and emphasis given to any program of dental health education.

In many countries where the dentist population ratio is not favourable priorities are usually made for:

1. Relief of pain.
2. Conservative treatment.
3. Tooth replacements.
4. Preventive procedures.

The ratio of population varies considerably from country to country, even from state to state. The most favourable ratio is found in Sweden (1:1,000), closely followed by Norway (1:1,800).

In many countries the increase of dental manpower has not kept pace with the need and demand of a rapidly growing population. To meet this expanding need some countries have introduced the dental nurses as in New Zealand, dental hygienists in United States of America, and assistant dental officers in the Pacific Islands.

The graduating dentist has a particular interest in the study of dental manpower resources, since he must choose a location to establish practice. The relative supply of dentists varies widely from state to state and from city to city. As a result, some areas consistently suffer a lack of

dentists; others have more dentists available than are needed to satisfy public demand regardless of regional or national shortages. The selection of a location to establish practice should be guided partly by a knowledge of the relation between the demand for service and the supply of professional personnel.

Treatment Needs

In spite of the high prevalence of periodontal disease, the American Dental Association in its 1952 study of dental needs finds only about 8% of United States dental patients in need of periodontal treatment. This may reflect a lack of alertness on the part of the average dentist to the diagnosis and treatment of periodontal disease, a difference in interpretation between practitioners and researchers upon this matter, or, more likely, a feeling on the part of dentists that much periodontal disease can be kept under control through dental prophylaxis without more specific treatment. Whatever the answer, a discrepancy exists here which is a challenge to public health dentists.

About three out of four of the entire adult population at risk - the 90 million men and women who had at least one permanent tooth - had periodontal disease, and about one out of four had destructive periodontal disease. Two out of

four had gingivitis ranging from mild inflammation involving a few teeth to severe inflammation involving all their teeth. This estimation was made in 1960 in America.

Education

With the need for more effective prevention of periodontal disease so apparent, we must recognize that the practice of prevention is based on lay education. The public must have more understandable information about the prevalence, the cause, the treatment and the prognosis of periodontal conditions. There has been a dearth of such information directed into lay channels in comparison with the amount disseminated concerning dental caries.

Periodontal disease unlike dental caries, has no general measure like fluoridation available for the prevention of periodontal disease. It is hoped that an enzyme can be incorporated in a dentifrice that will inhibit the deposition of calculus on teeth. This will be a definite aid to the periodontal patient. It would appear, however, that the major part of periodontal disease can be prevented and controlled by efficient oral hygiene. This is an important responsibility of the patient, the dentist and his supporting staff.

Any sound program of dental health education must include:

1. The study of methods directed to changing the habits or attitudes of the public, for example in relation to the intake of refined carbohydrates or the habits of oral hygiene.
2. The application of appropriate therapeutic measures by community action - for example fluoridation of water supplies.
3. The education of the individual in the need for recognition and treatment of the early lesion, be it caries, periodontal disease, or a developmental or acquired dento-facial abnormality.
4. The dental health education of the individual by experience at the hands of the dental profession.

A good dental service provides opportunities for proper education. In communities where dental disease is common, three points should be stressed:

First, that prevention is better than cure; regular attendance at a dental clinic for a prophylactic inspection will save much future trouble.

Secondly, it should be known that the teeth and gums ought to be kept clean at all times. For

the modern city dweller the regular use of a toothbrush properly applied as soon as possible after meals, should be recommended.

Thirdly, it should be stressed that sticky sweets or soft starchy foods between meals are harmful, since they lodge between the teeth and encourage dental decay. Heavy tax on sweets may help to reduce excessive consumption.

Lack of knowledge also plays its part in the parents' attitude to treatment and perhaps this may be the greatest problem of all that one has to contend with in Tonga. It is often noticeable that when parents are invited to discuss a particular relating to the child's dentition, their knowledge of the importance of retaining deciduous teeth until the child attains a certain age is lamentable; and as regards the intake and control of carbohydrates it is non-existent. Apathy certainly plays its part in the attitude of parents and even today with radio propaganda and dental health education, the care of the teeth in many instances would appear to have the least claim on a child's personal needs. Whilst children of today dare not attend school without having their faces washed, yet their teeth may not have been brushed for weeks on end; solely due to indifference on the part of the parents rather than lack of knowledge.

Most people are conscious of the need for sound dental

care, yet, so often do not seek an early treatment for their dental troubles. We may ask ourselves, why is there such a gap between dental information and dental practice and how can we best motivate the individual not merely to wish for healthy teeth, but also to act on his wishes? We should indeed do well to remember that "health is not something which can be imposed by authority; it begins and flourishes only as it is practised". If attitudes towards dental treatment can be measured, we shall then be in a better position to assess the current dental health picture.

The prime object of any campaign for the prevention of dental disease should be the health education of the public. The possibilities in the field of dental health education in Tonga at the present time are largely unexplored. Success in this field will depend upon the availability of authentic and up-to-date scientific information, and the availability of sufficient funds to enable the profession to guide the public towards a proper awareness of the importance of dental health, together with the availability of treatment. This will be a most important factor in regulating the growth of demand for dental care.

The most effective dental health education is undoubtedly carried out at the chairside by the dental surgeon together

with his staff. The profession on the whole recognizes this, but is discouraged from spending any length of time on this problem by the fact that daily patients required urgent relief of pain are more than we can handle.

Nutrition and Diet

Nutrition is essentially an environmental problem. The milder vitamin deficiencies associated with modern civilization are also conducive to periodontal disease over longer periods of time. Avitaminosis C manifested itself as scurvy and produced acute periodontal disturbance accompanied by loss of teeth. Vitamin A deficiency may possibly show a positive correlation, though firm evidence is lacking. Niacin deficiency has been found to produce a severe type of necrotic gingivitis with pseudomembrane formation and associated sloughing along the buccal mucosae. Beyond these recorded results in humans, experimental work in animals has shown other types of nutritional deficiency to affect the periodontium, notably protein starvation and magnesium deficiency. Profound changes can be induced in periodontal tissues of some laboratory animals by altering their nutritive status or dietary patterns.

Certain chemical and physical hazards are known for their effects upon the periodontal tissues. Mercury, lead,

and thallium, in decreasing order of frequency, have been reported to produce gingivitis accompanied by a dark line parallel to the gingival margin. In long standing cases alveolar resorption occurs. Radium and other sources of ionizing radiation produce alveolar damage and loosening of the teeth. Study should be made with the possible relation of fluoride in the drinking water to periodontal disease.

Dietary Control

It requires the co-operation of the public and it is necessary to initiate protraction and expensive propaganda designed to discourage the indiscriminate eating of refined carbohydrates, and to promote good dental hygiene.

The systemic control of periodontal disease lies chiefly in adequate nutrition with vitamin C and in the control of blood dyscrasias, heavy-metal poisonings, and other conditions which impair the health of the gingivae.

In many underdeveloped countries such foodstuffs as refined wheat flour and sugar and sweetened soft drinks nowadays form a regular part of the diet of the indigenous people, particularly in the towns. The incidence of caries and periodontal disease among the children of such communities, where the facilities for dental care and treatment are

still limited, is often very high. Their parents frequently have lost many teeth, a large number of those remaining being loose and embedded in pools of pus.

Physical Effects

It is noteworthy that periodontal disease is rare among primitive people who make full use of their teeth. The massaging effect of rough, tough foodstuffs no doubt quickly removes the earliest deposits of tartar. As an instance, children in Tonga have excellent gums, perhaps because of their habit of chewing pieces of sugar-cane between meals; the cane, despite its sugar content, seems to keep the teeth and gums healthy by its local abrasive action.

Foods that need to be chewed not only massage the gums but perhaps prevent the teeth from developing "disuse odontoporosis". It is unlikely, however, that the modern man will ever be persuaded to return to the gross, bone-chewing habits of the Tudor kings. Consequently the modern man with his polite dietary habits must perforce see his dentist regularly.

Coarse fibrous foods may improve the gingiva by mechanical surface stimulation, but their major benefit may be derived from cleansing away irritating food accumulations.

Slack^{145a} and Martin carried out their studies on

detergent properties of apple. They found that the gum condition of the children was significantly better in the apple group. The action of the apples appears to be two-fold.

- (i) Firstly, the low pH of the apple stimulates salivary flow, which is also increased by mastication of fibrous apple fragments.
- (ii) Secondly, the mechanical cleansing action of the apples sweeping over the teeth and gums in the presence of increased salivary flow removes debris and stimulates the gum tissue.

Antibiotics

Studies⁷⁵ have shown that plaque accumulation and gingival infection have been controlled by antibiotics in the diet and healing following local periodontal treatment has been improved by systemic antibiotics. Systemically administered drugs which modify the saliva so as to inhibit calculus formation could be a logical outgrowth of current research on anticalculus agents.

SUMMARY AND CONCLUSIONS

Clinical experiments incontrovertibly indicate that toothbrushing is a most important patient administered

preventive and adjunctive therapeutic procedure. In no other field of medicine and in the control of no other chronic disease can the patient so effectively assist in preventing and reducing the severity of a disease as can be done with the toothbrush in relation to gingivitis.

The regular, careful use of the toothbrush prevents the retention of soft debris at the junction of gingiva and the teeth. Maximum effectiveness in this regard is obtained by brushing as soon as possible after meals.

It has been demonstrated that toothbrushing retards the onset of gingivitis, reduces its incidence, improves gingival health and gingival stippling, reduces plaque and accumulated debris and reduces calculus.

Most periodontists agree that proper toothbrushing is an effective, simple and inexpensive measure in maintaining the oral cleanliness so essential to oral health.

Prevention is far more realistic than futile attempts to keep pace with destruction. Prevention is more sensible than dental repair.

The known methods whereby dentists can prevent periodontal disease are more effective than those available for

any other chronic affliction of the human body, even more effective than caries preventive measures. But they are not widely used.

TONGAN COMMUNITY

Tonga has a population of 77,429 revealed in 1966 census. There are 39,157 males and 36,964 females. Thirty per cent of the population (or 27,955) are children under ten years old. Tongans belong to the Polynesian Race.

Dentistry came to exist in Tonga in the last two decades. Its main concern was to relieve the pain mainly by extraction and dental prosthesis. In those days most of the extractions were done without local anaesthetic which created an dental phobia to some of the Tongans.

There have not been any dental surveys of periodontal diseases carried out in Tonga. The explanation which I am giving here is merely an observation of prevalence and severity of periodontal diseases treated in the dental clinic in the last four years.

Periodontal disease is one of the great public health problems of this time in Tonga. It causes a considerable loss of teeth in Tongan adults in the middle and older age groups. Some deviation from the ideal condition of the periodontium has been seen in virtually every adult.

Periodontal disease is the main cause of teeth mortality in adults, in fact, the adult population has an almost universal requirements for periodontal care. In 1965, dental extractions due to periodontal disease were 6,309 teeth compared to 6,078 teeth extracted because of dental caries. The total number of patients treated in 1965 was 22,119 of which 12,419 were treated as periodontal disease.

Certainly, attention to periodontal disease constitutes one of the major responsibilities of the dental profession in Tonga. It has been noticed that rural children have less gingival disease than urban children of comparable age. This is probably due to the fact that the rural children live mostly on native staple diet like manioc, yams, bananas, green leaves and vegetables, plenty of fish and many sorts of sea foods, pork and meat. Whereas the urban children have more access to European foods, and the tendency is greater for them to eat sugary refined, sticky foods.

With a rising population and a measure of economic advancement there has developed a greater demand for more and more refined carbohydrate foods with the inevitable result that a greater burden is thrown upon the dental service.

Originally, the dental service was a relief-of-pain service. People came for treatment only when compelled by

pain, and there were many fears and prejudices in the minds of these patients. It was, therefore, desirable to extend the service and to work towards prevention. To achieve this more buildings, staffs, equipment, and transport appeared to be necessary. Unfortunately provision of these essentials required finance which was not readily available. The problem had therefore to be viewed in the light of the limited financial resources available, the scattered nature of the various islands, the level of literacy of the inhabitants, and other factors necessitating the adoption of a more flexible approach.

Hitherto, the necessity for relieving pain and eliminating septic and other urgent conditions had restricted our activities in schools to annual visits. On such occasions efforts had been limited to inspection of pupils' teeth, issuing of notices to parents informing them whenever urgent treatment was necessary, toothbrushing demonstration, short lectures on health education, and few operative works done.

Majority of the Tongans are poor people with limited educational status and the opportunities are less likely to understanding the reasons for personal hygiene and for the avoidance of sources of infection. Through ignorance many adults and almost all the old aged people do not clean their

teeth. Inflammatory periodontal disease is universal and strongly associated with the presence and amount of oral debris and calculus in the mouths of these Tongans, who seldom or never practise oral hygiene. The adult population, male and female, suffers the same from periodontal disease.

At the moment, of course, only a minority appreciate the benefits of regular dental care and estimates suggest that not more than 30% do so. More rapid improvement depends not merely upon the public at large being better informed about dentistry, but even more upon their regarding dental health as something desirable in itself. This in turn can come about by more effective dental health education in its broadest sense.

It is self-evident, for instance, that people do not look after their teeth firstly because they do not regard their teeth as being of sufficient importance to make the necessary effort at oral hygiene; secondly, dentistry is generally regarded as being a painful and frightening procedure. As a consequence, perhaps, anything to do with the teeth is best ignored. Many look forward to the day when all the teeth can be extracted and replaced by a denture which to them is an acceptable alternative, even perhaps an improvement upon their own teeth. Indeed there are many

otherwise well-informed who would argue on this basis that there is little point in preserving the natural teeth. Yet this is a backward-looking attitude.

If people do not accept that their teeth are important, no amount of propaganda will make them brush their teeth, eat fruit, or what we will. Nor is it an easy task to convince them, and the arguments produced by the profession, however sound they may be, do not impress the laymen. So many young Tongans know very well that their teeth are often a nuisance to them, while their parents manage perfectly well with artificial dentures. Modern advertising does its best to make them eat more and more sweets and they are by no means reluctant to conform.

Simple Treatment

By the Dentist: Prophylaxis:⁷⁰ Patient's teeth should be cleaned at regular intervals. Prophylaxis insures the removal of stains and deposits of tartar that the patient is missing with his daily home care. It helps him to maintain a healthy mouth but does not in any way take the place of his daily home care routine. He is the only one who can prevent a recurrence of periodontal disease. How often he will need an oral prophylaxis depends on how well he practises his daily home care. Dunning emphasises the three main methods

by which periodontal disease may be minimized. These involve dental prophylaxis, dental home care (chiefly tooth-brushing), and the systemic strengthening of gingivae through proper nutrition and elimination of systemic disease.

Dental prophylaxis⁶⁸ has even more importance in the control of periodontal disease than it does in the control of dental caries. This is because deposits of salivary and serumal calculus are highly conducive to periodontal disease and cannot be removed by the patient in the course of home care. Patients with a tendency to gingivitis must be observed until it is known in how many months the accumulation of hard deposits upon the teeth will pass beyond the control of home care. Many periodontal patients must receive dental prophylaxis every three months or every four months in addition to whatever more extensive treatment may be necessary at the hands of the dentist.

The dentist or dental hygienist performs the services for the patient as a direct, purposeful experience. As this type of experience is considered to be most productive of permanent learning, every effort should be made to provide authentic information concerning the immediate treatment being performed and its application for better dental health. Research indicates that most of the information which adults

retain in reference to the health of their teeth is first heard or experienced in a dental office. Having the scientific knowledge, the dentist and his staff should consider themselves as teachers in the fullest sense. Unfortunately, too few dentists know how to impart this knowledge to lay people by using sound teaching techniques. Not only is it necessary to teach the facts, but it is also necessary to correct wrong impressions about dentistry.

The people should be taught that in the absence of adequate oral hygiene procedures, the bacterial plaque in the gingival crevice areas increases in volume until this inner layer frequently mineralizes to form calculus. Such mineralization strengthens the attachment of the bacterial mass to the tooth surface, while the gradation from mineralized to viable plaque ensures that a constant source of irritation is provided for the gingival tissues.

Simple Home Prevention

The family should, either individually or as a group, ask adequate instruction from the dentist in home care of the mouth. Thorough tooth cleaning is a difficult procedure for an adult; children require adult supervision. This is essential below the age of six and highly desirable until adolescence.²⁷ It is often noted that children who come

from homes where the standard of living is high and whose parents instruct them in oral hygiene and regular toothbrushing, unfortunately do not have correspondingly good teeth, and I am of the opinion that this is due to lack of intelligent supervision. Undoubtedly such children brush their teeth with regularity but make a very poor job of it and it often happens that the first permanent molars are completely missed and, in consequence, develop dental caries, in many cases soon after eruption. Regular checks on the efficiency of cleaning should be sought during routine dental examinations. Correct toothbrushing is a most effective aid in keeping both hard and soft tissues of the mouth in a healthy condition.^{9, 24} Other cleaning aids such as the toothpick, dental floss and rubber stimulator are very helpful when used under professional supervision.

The contact surfaces and embrasures between the teeth must be cleansed with dental tape and other special instruments.¹¹⁵ Dental tape should be used at least after the evening meal. It passes between the teeth through the contact surfaces and helps to prevent decay as well as periodontal disease.²²

Round toothpicks or soft wooden wedges called Stim-U-Dents can be used in small spaces in the front of the mouth,

but rubber points are necessary for large areas and for spaces between the back teeth.

Contact with oral tissues must be firm but gentle. These tissues cannot stand rough treatment. Periodontal disease is caused by local irritation, and improper use of instruments for oral hygiene can be severe irritant.

Preventive measures are directed to the removal of the preliminary soft deposits of early calculus by vigorous chewing of hard foods, supplemented with regular and thorough toothbrushing.

Proposed Dental Program

Any dental program designed to meet the needs of the Tongan community must be adapted to fit in with the economic and educational development of the country. Quite apart from the main difficulties created by having to treat dental disease in a population living on islands scattered over an area of several thousands of square miles, there are problems of remoteness of supply, a comparatively low dentist population ratio (1:11,061), lower levels of literacy, inadequate buildings and equipment, restricted finance and transport. Activities are limited by the amount of money available.

The public must be confronted with the facts about

periodontal disease and must be educated in its prevention.

The periodontal status of Tonga should be explored in terms of what it will mean in tooth loss, patient and dentist time, and in terms of private and government funds to treat the existing periodontal disease and to replace the teeth lost because of it.

To motivate the Tongans to take advantage of available methods of preserving oral health, the public must recognize in its own interest that the mission of dentistry is to keep healthy mouths healthy rather than repair damage done by preventable disease.

SUMMARY AND CONCLUSION

Periodontal disease probably occurs almost universally and no one is fully immune to attack.

It is widely recognized that the major cause of tooth loss beyond the age of 30 is periodontal disease. It constitutes a major public health problem.

Most periodontal diseases are caused by local factors which are accessible, correctable and controllable.

Neglect is a principal cause of gingival and periodontal disease; neglect of the healthy mouth which permits disease to occur; neglect of early disease which permits to destroy the tooth supporting tissues and neglect of the treated mouth which permits disease to recur.

Early diagnosis, proper treatment, and periodic checking on results of therapy are very important. The treatment of periodontal disease is accomplished by a variety of treatment methods such as scaling, curettage, periodontal surgery, occlusal adjustment, etc.

The chief reasons for treating periodontal disease is to

preserve the natural teeth in the mouth.

Periodontal disease is slightly more prevalent in males than in females. It is more severe in males than in females after the age of 20 or 30 years.

Prevention of periodontal disease is based on the knowledge of aetiology and pathogenesis. Periodontal disease can be prevented by elimination of local irritation, patient education, and avoiding the creation of disease-producing conditions. Systemic disorders capable of initiating gingival and periodontal disease or aggravating locally caused disease should also be corrected.

Clinical experiments incontrovertibly indicate that toothbrushing is a most important patient administered preventive and adjunctive therapeutic procedure.

Prevention is far more realistic than futile attempts to keep pace with destruction. Prevention is more sensible than dental repair.

The periodontal status of Tonga should be explored in terms of what it will mean in tooth loss, patient and dentist time, and in terms of private and government funds to treat the existing periodontal disease and to replace the teeth lost because of it.

Any dental programme designed to meet the needs of the Tongan community must be adapted to fit in with the economic and educational development of the country.

To motivate the Tongans to take advantage of available methods of preserving oral health, the public must recognize in their own interests that the mission of dentistry is to keep healthy mouths healthy rather than repair damage done by preventable disease.

REFERENCES

1. Arno, A., et al. - Incidence of gingivitis as related to sex, occupation, tobacco consumption, tooth-brushing and age. Oral Surg., 11:578 (June) 1958.
2. Bain, A. - Desquamative and pregnancy gingivitis. N.Y.S. Dent. J. 20:18-20 (Jan.) 1954.
3. Barnard, P. D. - Dental survey of State schoolchildren in New South Wales. Jan. 1954-June 1955. Spe. Rep. Ser. Nat. Health & Med. Res. Counc. Aust., No. 8, Canberra, 1956.
4. Barnard, P. D. - Dental conditions of senior dental students. Austral. J. Dent., 11:344 (Oct.) 1966.
5. Barrett, M. J. - Dental observations on Australian aborigines: Yuendumu, Central Australia. Austral. J. Dent., 57:127-138 (June) 1953.
6. Bell, D. G. - Teaching home care to the patient. J. Periodont., 19:140 (Oct.) 1948.
7. Belting, C. M., and Gupta, O. P. - Incidence of periodontal disease among persons with neuropsychiatric disorders. J. Dent. Res., 39:744-745 (July) 1960.
8. Belting, C. M., et al. - Prevalence and incidence of alveolar bone disease in men. J.A.D.A., 47:190 (Aug.) 1953.
9. Beube, F. E. - Gingival massage. J. Periodont., 19:66 (Apr.) 1948.
10. Benjamin, C. M., et al. - Periodontal disease in rural children of 25 Indiana counties. J. Periodont., 28:294-298 (Oct.) 1957.

11. Berman, C. L., et al. - Observations of the effects of an electric toothbrush. *J. Periodont.*, 33:195 (Apr.) 1962.
12. Bernier, J. L. - Role of inflammation in periodontal disease. *J. Oral Surg. Oral Med. and Oral Path.*, 2 (5): 583 (May) 1949.
13. Bernier, J. L. - Changes in bone and cementum in periodontal disease. *J. Periodont.*, 22 (1):29 (Jan.) 1951.
14. Bhaskar, S. N., and Frisch, J. - Occlusion and periodontal disease. *Internat. Dent. J.*, 17 (2): 251-260 (June) 1967.
- 14a. Black, G. V. - *D. Cosmos* 60:1219 (July) 1913.
15. Blake, G. C. and Trott, J. R. - *Periodontology*. London, Butterworths, 1962 (pp. 1-3).
16. Bossert, W. A., and Marks, H. H. - Prevalence and characteristics of periodontal disease in 12,800 persons under periodic dental observations. *J.A.D.A.*, 52:429-442 (Apr.) 1956.
17. Box, K. - Traumatic occlusion and traumatogenic occlusion. *Oral Health*, 20:642 1930.
18. Boyle, P. - Experimental evidence of systemic influence on the development of periodontal disease. *J. Periodont.*, 22:195 (Oct.) 1951.
19. Brucker, M. - Studies of the incidence and cause of dental defects in children. *J. Dent. Res.*, 22:309 (Aug.) 1943.
20. Brucker, M. - Studies on the incidence and causes of dental defects in children. III gingivitis. *J.A.D.A.*, 45:706 (Apr.) 1943.
21. Campbell, H. G., and Cook, R. P. - Incidence of gingivitis at Dundee Dental Hospital. *Year Book of Dentistry*, 1942.
22. Campbell, J. M. - Toothpicks and Toothbrushes. *Dent. Mag. and O. Top.*, 81:26-24 (Feb.) 1964.

23. Carranza, F. A., and Carranza, F. A. Jr. - A suggested classification of common periodontal disease. *J. Periodont.*, 30 (2):140-147 (Apr.) 1959.
24. Carter, S. B. - The masticatory mucosa and its response to brushing. *Brit. Dent. J.*, 101:76-78 (Aug.) 1956.
25. Charters, W. J. - Home care of the mouth. *J. Periodont.*, 19:136 (Oct.) 1948.
26. Clements, F. W., and Kirkpatrick, R. M. - Medical and dental surveys of school and pre-school children of N.S.W. Nutrition in relation to dental health. *Austra. Dent. J.* 10:339, 418 (Oct.) 1938.
27. Clements, F. W., and McCloskey, B. P. - Child Health. London, Edward Arnold Ltd., 1964 (p. 328).
28. Cohen, B. - Morphological factors in the pathogenesis of periodontal disease. *Brit. Dent. J.* 107 (1): 31-39 (July) 1959.
29. Cohen, M. M., Winer, A. and Shkler, G. - Boston dispensary Rotch Research Laboratory. Periodontal disturbances in the group on mentally subnormal children. *J. Dent. Res.*, 39:745 (July) 1960. *D. Clin. N. America* pp. 483-489 (July) 1960.
30. Compertz, J. M. - Factors that influence mouth conditions during pregnancy. *Dent. Abst.*, 7:345 (June) 1962.
31. Coolidge, E. D. - The Thickness of the human periodontal membrane. *J.A.D.A.*, 24:1260 (Aug.) 1937.
32. Coolidge, E. D., and Hine, M. K. - Periodontology. Philadelphia, Lea and Febiger, 3rd ed., 1958 (pp. 86-93).
33. Cran, J. A. - Notes on the teeth and gingivae of Central Australian Aborigines. *Austral. Dent. J.*, 56: 356-361 (Dec.) 1955.
34. Cran, J. A. - Notes on the teeth and gingivae of Central Australian Aborigines. *Austral. Dent. J.*, 2: 277 (Oct.) 1957.

- 34a. Cross, W. G. - Gums tumours in pregnancy and gingivitis gravidum. Brit. Dent. J. 75:85-89 () 1943.
35. Cunningham, W. M. - Prevalence of periodontal (parodontal) disease II, In Australia. Internat. Dent. J., 5 (2):200-208 (June) 1955.
36. Dahl, L. O., and Davis, B. B. - Oral hygiene habits of young children. J. Periodont., 25:209 (July) 1954.
37. Davis, E., Ray, F. G., and Farleigh, C. M. - A study of the need for periodontal treatment in a group of naval personnel with a comparison of periodontal indices. J.A.D.A., 64:503 (Oct.) 1961.
38. Davis, G. N. - Dental conditions among the Polynesians of Pukapuka (Danger Island). J. Dent. Res., 35:734-741 (Oct.) 1956.
- 38a. Davis, G. N. - Social customs and habits and their effect on oral disease. J. Dent. Res., 42:209-232 (Jan.) 1963.
39. Day, C. D. M., and Shourie, K. L. - Roentgenographic survey of periodontal disease in India. J.A.D.A., 64:503-512 (Oct.) 1961.
40. Day, C. D. M., and Shourie, J. L. - Hypertrophic gingivitis in Indian children and adolescents. J.A.D.A., 39:572-588 (Nov.) 1949.
41. Day, C. D. M., et al. - Periodontal disease: prevalence and incidence. J. Periodont., 26:189 (July) 1955.
42. Day, C. D. M., and Shourie, K. L. - Gingival disease in the Virgin Islands. J.A.D.A., 40 (2):175-185 (Feb.) 1950.
43. Day, C. D. M. - Epidemiology of periodontal disease. J. Periodont., 22:13-22 (Jan.) 1951.
44. Day, C. D. M., and Tandan, G. C. - The incidence of dental caries in the Punjab. Brit. Dent. J., 69:389 (Dec.) 1940.

- 44a. Day, C. D. M. - Chronic endemic fluorosis in Northern India. Brit. Dent. J., 68:409 (May) 1940.
- 44b. Day, C. D. M. - Nutritional deficiencies and dental caries in Northern India. Brit. Dent. J., 76: 115, 143 () 1944.
- 44c. Day, C. D. M. - Oral conditions in the famine district of Hissar. J.A.D.A., 31:52-58 (Jan.) 1944.
- 44d. Day, C. D. M. - Periodontal disease: Present status and interpretation of epidemiological research. In Muhler, J. C., and Hine, M. K. - A symposium on preventive dentistry. St. Louis, Mosby 1956 (p. 174).
45. Dawson, C. E. - Dental defects and periodontal disease in Egypt 1946-1947. J. Dent. Res., 27:512-523 (Aug.) 1948.
46. Dodds, A. E. - The prevalence of parodontal disease. I. in Africa. Internat. Dent. J., 5 (3):319-326 (Sept.) 1955.
47. Dunning, J. M., and Leach, L. B. - Gingival bone count: a method for epidemiological study of periodontal disease. J. Dent. Res., 39 (3):506 (May-June) 1960.
48. Dunning, J. M. - Principles of Dental Public Health. Cambridge, Massachusetts. Harvard University Press, 1962 (pp. 151-164).
49. Engel, M. B., et al. - The pathogenesis of desquamative gingivitis is disturbance of the connective tissue ground substance. J. Dent. Res., 29: 410-418 (Aug.) 1950.
50. Englander, H. R. - Epidemiology, a fundamental discipline in dental research. J.A.D.A., 65 (6): 755 (Dec.) 1962.
51. Erb, A., and Brezezkinsky, A. - Gingivitis in pregnant Israel women. J. Periodont., 34:447 (Sept.) 1963.

52. Foss, C. L., Grupe, H. E., and Orban, B. - Gingivitis: J. Periodont., 24:207-219 (Oct.) 1953.
53. Glickman, I. - Acute vitamin C deficiency and periodontal tissue II. The effect of acute vit. C. deficiency upon the response of the periodontal tissues of the gums to artificially induced inflammation. J. Dent. Res., 27:201 (Feb.) 1948.
54. Glickman, I. - Clinical periodontology. London, Saunders, 2nd ed., 1958 (pp. 4,253-256) Chapters 25 and 26. (pp. 4,442-443).
55. Glickman, I. - Inflammation and trauma from occlusion co-destructive factors in chronic periodontal disease. J. Periodont., 31:5-10 (Jan.) 1963.
56. Glickman, I. - A blueprint for the periodontal health of the American public. J. Periodont., 38 (5): 361-368 (Oct.) 1967.
57. Goldman, H. M. - Extension of exudate into supporting structures of teeth in marginal periodontitis. J. Periodont., 28:175 (July) 1957.
58. Goldman, H. M. - An introduction to periodontia. St. Louis, Mosby, 2nd ed., 1962 (p. 34).
59. Goldman, H. M., et al. - Periodontal therapy. St. Louis, Mosby, 2nd ed., 1960 (p. 25).
60. Gonzales, O. S., De La Fuente, R. M., and Chacon, U. B. - Gingivitis of pregnancy. Dent. Abst., 7:345 (June) 1962.
61. Gratzinger, M. - Dynamic cause of periodontal disease. J.A.D.A., 37:294 (Sept.) 1948.
62. Gridley, M. S. - Gingival conditions in pregnant women. Oral Surg. Oral Med. Oral Path., 7:641-646 (June) 1954.
63. Green, J. C. - Periodontal disease in India, report of an epidemiological study. J. Dent. Res., 39: 302-312 (Mar.) 1960.
64. Green, J. C., and Vermillion, J. R. - The oral hygiene index. J.A.D.A., 61:172-179 (Aug.) 1960.

65. Grupe, H. E. - Acute necrotizing ulcerative gingivitis. J. Periodont., 27:255 (Apr.) 1956.
66. Hilming, F. - Gingivitis gravidarum. Oral Surg. Oral Med. Oral Path., 5:734-751 (July) 1952.
67. Hine, M. K., and Hine, C. L. - Classifications and aetiology of periodontal disturbances. J.A.D.A., 31 (19):1297-1307 (Oct.) 1944.
68. Hine, M. K. - Prophylaxis, toothbrushing and home care of the mouth as caries control method. J. Dent. Res., 27:225 (Apr.) 1948.
69. Hine, M. K. - Use of toothbrush in the treatment of periodontitis. J.A.D.A., 41:158 (Aug.) 1950.
70. Hoover, D. R., and Lefkowitz, W. - Reduction of gingivitis by toothbrushing. J.A.D.A., 65:361 (Sept.) 1962.
71. Jamison, H. C. - Prevalence and severity of periodontal disease in a sample of a population. Thesis, University of Michigan, School of Public Health 1960.
72. Jenkins, G. N. - The physiology of the mouth. Oxford, Blackwell Scientific Publications, 2nd ed., 1960 (p. 312).
73. Johnson, N. P., and Young, M. A. - Periodontal disease in Mongols. J. Periodont., 34:41-46 (Jan.) 1963.
74. Kauffman, J. H. - A study of the toothbrush. J.A.D.A., 26:32 (Jan.) 1939.
75. Keyes, P. H., and Jordan, H. V. - The effect of two diets and antibiotics on periodontal disease. Abstr. I.A.D.R. 10:53 (Jan.) 1965.
76. King, J. D. - Gingival disease in Dundee. Brit. Dent. J., 77:241 (Nov.) 1944.
77. King, J. D. - Dental disease in the Isle of Lewis. Brit. Dent. J., 68:349 (May) 1940.

78. Ladavala, M. R. N., and Harris, R. - A study of the gingival and periodontal conditions of a group of people in Chiang Mai Province. J. Periodont., 30 (3):219-222 (July) 1959.
79. Lighterman, I., and Lees, S. - Study of aetiology of pregnancy gingivitis with special references to hormonal and diet influences. Dent. Abst., 3:356 (June) 1958.
80. Leigh, R. W. - Dental pathology of the Eskimo. Dent. Cosmos, 67:884-898 (Sept.) 1925.
81. Levine, S. - Periodontal disease: a public health problem. Austral. Dent. J., 10 (1):69-74 (Feb.) 1965.
82. Levy, A. T. - Monograph on gingivitis gravidarums. Dent. Items. Interest, 63:1114 (Dec.) 1941.
83. Lewis, H. K. - A study of gingivitis in pregnancy. M.D.S. Thesis. University of Sydney.
84. Littleton, N. W. - Dental caries and periodontal disease, among Ethiopian civilians. Pub. Health Rep., 78:631 (July) 1963.
85. Lobene, R. R. - Effect of automatic toothbrush on gingival health. J. Periodont., 35:41 (Apr.) 1964.
86. Loeb, V. - Gingival massage. J. Periodont., 19:70 (Apr.) 1948.
87. Loos, S. - Prevalence of periodontal (parodontal) disease. Internat. Dent. J., 5 (3):319 (Sept.) 1955.
88. Lovdal, A., et al. - Incidence of clinical manifestations of periodontal disease in light of oral hygiene and calculus formation. J.A.D.A., 56:21 (Jan.) 1958.
89. Lyons, H. - Gingival massage. J. Periodont., 19:71 (Apr.) 1948.
90. Lyons, H. - Fiction and fact in periodontology. J.A.D.A., 39:513 (Nov.) 1949.

- 90a. MacMillan, H. W. - The case against traumatic occlusion. J.A.D.A., 17:1996 (Aug.) 1930.
91. Maier, A. W., and Orban, B. - Gingivitis in pregnancy. Oral Surg. Oral Med. and Oral Path., 2:334-373 (Mar.) 1949.
92. Manson, J. D. - Periodontics for the Dental Practitioner. England, Henry Kempton, 1966 (p. 29).
93. Massler, M., and Schour, I. - The P-M-A Index in gingivitis. J. Dent. Res., 28:634 (Dec.) 1949.
94. Massler, M., et al. - Occurrence of gingivitis in suburban Chicago schoolchildren. J. Periodont., 21:13-22 (Jan.) 1951.
95. Massler, M., et al. - Epidemiology of gingivitis in children. J.A.D.A., 45:319 (Sept.) 1952.
96. Massler, M., et al. - Gingivitis in young adult males. Lack of effectiveness of permissive program of toothbrushing. J. Periodont., 28:111 (Apr.) 1957.
- 96a. McCall, J. O. - The periodontist looks at children's dentistry. J.A.D.A., 20:1518-1521 1933.
97. McCarthy, F. P., et al. - Chronic desquamative gingivitis. Oral Surg. Oral Med. and Oral Path., 13:1300 (Nov.) 1960.
98. McCarthy, P. L. - Disease of mucosa. New York, McGraw Hill, 1964 (p. 220).
99. McCombie, F., and Stothand, D. - Relationship between gingivitis and other dental conditions. J. Canad. Dent. A., 30 (8):507 (Nov.) 1966.
100. McCombie, F., and Chua, S. C. - Dental epidemiology in Malaya. J. Canad. Dent. A., 23 (12):687 (Dec.) 1957.
101. Mehta, F. S., et al. - Mouth hygiene habits and their relation to periodontal disease. J. Dent. Res., 35:645 (Aug.) 1956.

102. Mehta, M. M., et al. - Periodontal disease among adults. J. Canad. Dent. A., 21:617 (Nov.) 1955.
103. Mehta, F. S., et al. - Prevalence of periodontal disease 5 epidemiology in India child population in relation to their socio-economic status. Internat. Dent. J., 6 (1):31-40 1956.
104. Merrit, A. H. - Periodontal disease. New York, Macmillan Co., 3rd ed., 1945 (p. 43).
105. Merrit, A. H. - The historical background of periodontology. J. Periodont., 10:7-25 (Jan.) 1939.
106. Miller, S. C. - Non-occlusion as a factor in periodontal disease. J. Dent. Res., 8:602 (Oct.) 1928.
107. Miller, S. C., and Seidler, B. B. - A correlation between periodontal disease and caries. J. Dent. Res., 19:549 (Dec.) 1946.
108. Noyes, F. B. - Oral histology and embryology. Philadelphia, Lea and Febiger, 8th ed., 1960 (pp.230-240).
109. Orban, B. - Tissue changes in traumatic occlusion. J.A.D.A., 15:2090 1928. Amer. Dent. Assn. Jnl. XV, 2090-2106.
110. Orban, B., and Weimann, J. P. - Diffuse atrophy of the alveolar bone (periodontosis). J. Periodont., 13:13 (July) 1942.
111. Orban, B. - Clinical and histologic study of the surface characteristics of the gingiva. Oral Surg. Oral Med. and Oral Path., 1:827-841 (Sept.) 1948.
112. Orban, B., et al. - A concept theory and practice. St. Louis, Mosby, 2nd ed., 1963 (pp. 61, 87, 152).
113. Orban, B., and Sicher, H. - Oral histology and embryology. St. Louis, Mosby, 6th ed., 1966 (pp. 213-264).

114. Parfitt, G. J. - A five-year longitudinal study of the gingival condition of a group of children in England. *J. Periodont.*, 28:26-32 (Jan.) 1957.
115. Parfitt, G. J. - Cleansing the subgingival space. *J. Periodont.*, 34:133-138 (Mar.) 1963.
116. Patton, J. L. - Ineffective toothbrush. *Dent. Abst.*, 6:175-176 (Mar.) 1961.
117. Pelzar, R. - A study of the local oral effect of diet on the periodontal tissue and gingival capillary structures. *J.A.D.A.*, 27:13-24 (Jan.) 1940.
118. Pindborg, J. J., et al. - Occurrence of acute necrotizing gingivitis in South India children. *J. Periodont.*, 37:15-17 (Jan.-Feb.) 1966.
119. Posselt, U. - The physiology of occlusion and rehabilitation. Oxford, Blackwell Scientific Publication, 1962 (p. 80).
120. Preissecker, O. - Influence of experimental disease on the periodontal membrane. *J. Dent. Res.*, 1: 518 (June) 1931.
121. Pricard, J., and Lorimer, J. W. - Periodontal prostheses in occlusal trauma. *J. Periodont.*, 28:26 (July) 1957.
122. Prichard, J. L. - Advanced periodontal disease: Surgical and prosthetic management. Philadelphia, Saunders Co., 1965 (pp. 358-457).
123. Ramfjord, S. P. - Local factors in periodontal disease. *J.A.D.A.*, 44:647 (June) 1952.
124. Ramfjord, S. P. - Indices for the prevalence and incidence of periodontal disease. *J. Periodont.*, 31:50 (Jan.) 1959.
125. Ramfjord, S. P. - The periodontal status of boys 11-17 years old in Bombay, India. *J. Periodont.*, 32 (3):237-248 (July) 1961.

126. Ritchey, B., and Orban, B. - The periodontal pocket. J. Periodont., 23:199 (Oct.) 1952.
127. Reed, C. B. - The mouth in pregnancy. J.A.D.A., 20: 1631 1933.
128. Robinson, B. G., and Kitchin, P. - The effects of mass- age with toothbrush on the keratinisation of the gingiva. Oral Surg. Oral Med. and Oral Path., 1:1042 (Nov.) 1948.
- 128a. Rosenzweig, K. A. - Gingivitis in children of Israel. J. Periodont., 34:404-405 (Sept.) 1963.
129. Rohackova, J., and Ticha, B. - Pregnancy gingivitis. Dent. Abst., 3:357-358 (June) 1958.
130. Rovelstad, G. H., et al. - Survey of dental health of the naval recruit. I. Status of dental health. J.A.D.A., 58:60 (May) 1959.
131. Russell, A. L. - A system of classification and scoring for prevalence surveys of periodontal disease. J. Dent. Res., 35 (3):350-359 (June) 1956.
132. Russell, A. L. - A social factors associated with the severity of periodontal disease. J. Dent. Res., 36:922 (Dec.) 1957.
133. Russell, A. L. - Some epidemiological characteristics of periodontal disease in a series of urban population. J. Periodont., 28:286 (Oct.) 1957.
134. Russell, A. L., et al. - Periodontal disease and nutri- tion in South Vietnam. J. Dent. Res., 44:775- 782 (July-Aug.) 1965.
135. Russell, A. L. - Epidemiology of periodontal disease. Internat. Dent. J., 17 (2):282-289 (June) 1967.
136. Sandler, H. C. - Testing the uniformity of the P-M-A Index as a measure of periodontal disease. J. Dent. Res., 31:323 (June) 1952.
137. Sandler, H. C., and Stahl, S. S. - The influence of gen- eralized disease on clinical manifestations of periodontal disease. J.A.D.A., 49:656 (Dec.) 1954.

138. Sandler, H. C., and Stahl, S. S. - Measurement of periodontal disease prevalence. J.A.D.A., 58:83 (Mar.) 1959.
139. Schei, O., et al. - Alveolar bone resorption as related to oral hygiene and age. J. Periodont., 30:7 (Jan.) 1959.
140. Schour, I., and Massler, M. - Gingival disease in post-war Italy (1945). J.A.D.A., 35:475 (Oct.) 1947.
141. Schour, I., and Massler, M. - Gingivosis in hospitalized children in Naples. Am. J. Orthodont. and Oral Surg. (Oral Surg. Sect.), 33:756 (Oct.) 1947.
142. Schour, I. - Terminology and classification of gingival of periodontal disease. J. Periodont., 22 (1): 10 (Jan.) 1951.
143. Schour, I., and Massler, M. - Prevalence of gingivitis in young adults. J. Dent. Res., 35:475 (Oct.) 1947.
144. Schwartz, J. - Teeth of Masai. J. Dent. Res., 25:17 (Feb.) 1946.
145. Shaffer, E. M. - Periodontal instrumentation scaling and root planning. Internat. Dent. J., 17 (2): 297-314 (June) 1967.
- 145a. Shay, H. and Smart, G. A. - The association of local factors with gingivitis. Brit. Dent. J., 78:135-137 (Mar.) 1945.
- 145b. Slack, G. L., and Haynes, S. - Preaching and practice of oral hygiene. Brit. Dent. J., 104, 171, 158.
146. Sorrin, S. - The practice of periodontia. New York, McGraw Hill Book Co., 1960 (pp. 158-159).
147. Stahl, S. S., et al. - The effect of protein deprivation upon the oral tissues of rat and particularly on periodontal structures under irritation. Oral Surg. Oral Med. and Oral Path., 8:760 (July) 1955.

148. Stahl, S. S., and Morris, A. L. - Oral health conditions among army personnel at the army engineer centre. *J. Periodont.*, 26:180 (July) 1955.
149. Stahl, S. S., et al. - Effects of toothbrushing on the keratinisation of the gingiva. *J. Periodont.*, 24:20 (Jan.) 1953.
150. Stanmyer, W. R. - Measure of tissue response to frequency of toothbrushing. *J. Periodont.*, 28:17-22 (Jan.) 1957.
151. Stillman, P. R. - Management of pyorrhoea. *Dent. Cosmos.*, 59:405 (Apr.) 1917.
152. Stern, I. B. - The statue of temporary fixed-splinting procedures in the treatment of periodontally involved teeth. *J. Periodont.*, 31:217 (July) 1960.
- 152a. Stoll, F. A. - Dental health education. Philadelphia, Lea and Febriger, 2nd ed., 1960 (pp. 47-48, 234).
153. Strandle, M. W. - The blessed twig: a historical talk with Japanese toothpick. *Dent. Abst.*, 6:175-176 (Mar.) 1961.
154. Tiecke, S. C. - Pathologic physiologic of oral disease. St. Louis, Mosby Co., 1959 (pp. 405-419).
155. Thoma, K. - Oral Surgery. Vol. I. St. Louis, Mosby, 1963 (p. 196).
156. Toto, P. - Clinical evaluation of chewing gum in gingivitis and caries. *J. Dent. Res.*, 39:750 (Aug.) 1960.
157. Vivone, R. A. - Prevalence of periodontal (parodontal) disease. *Internat. Dent. J.*, 6 (1):26-29 (Mar.) 1956.
158. Wade, A. B. - Basic periodontology. Bristol, John Wright & Son Ltd., 2nd ed., 1965 (pp. 208, 285-293).

159. Waerhang, J. - Current basis for prevention of periodontal disease. *Internat. Dent. J.*, 17:2 (June) 1967.
160. Waerhang, J. - Current basis for prevention of periodontal disease. *Internat. Dent. J.*, 17:267-277 (June) 1967.
161. Weinberger, B. W. - An introduction to the history of Dentistry. *J.A.D.A.*, 34:170-184 (Feb.) 1947.
162. Weinmann, J. P. - Progress of gingival inflammation into the supporting structures of the teeth. *J. Periodont.*, 12:71 (July) 1941.
163. Weinmann, J. P. - Periodontitis: aetiology, pathology, symptomatology. *J.A.D.A.*, 44:701 (June) 1952.
164. W.H.O. - Expert Committee Report, Technical Report Series No. 207, on periodontal disease, 1960. See 166.
165. W.H.O. - Program for preventing and treating periodontal disease. *Dent. Abst.*, 6:606 (Oct.) 1961.
166. W.H.O. - Technical Report Series, No. 207, periodontal disease. 1961 (p. 3).
- 166a. White, C. L., and Russell, A. L. - Some relations between dental caries experience and active periodontal disease in two thousand adults. *New York J. Dent.*, 32:211 1962.
167. Williams, S. F. - Dental service in Western Samoa. *N.Z. Dent. J.*, 35:115-131 (May) 1939.
168. Wilson, R. S. - Teeth and gums in pregnancy. *N.Z. Dent. J.*, 47:138-147 (July) 1951.
169. Winer, R. A., et al. - Antibiotic therapy in periodontal disease. *I.A.D.R.*, 43:72 1965 (Abstracts). Effect of _____ on gingival disease. *Oral Surg. Oral Med. and Oral Path.*, 9:615-616 (June) 1956.

170. Zegarelli, W. V., et al. - Prednidolone in the treatment of incapacitating desquamative stomatitis. Case report. J. Periodont., 28:310 (Oct.) 1957.
171. Zegarelli, E. V. - Triamcinolone in the treatment of acute and chronic lesions of the oral mucosis membrane. Oral Surg. Oral Med. and Oral Path., 13:170 (Feb.) 1960.
172. Ziskin, D. E. - Effect of male sex hormones on gingivae and oral mucousmembrane. J. Dent. Res., 20:419 (Oct.) 1951.
173. Ziskin, D. E., and Zegarelli, E. V. - Chronic desquamative gingivitis. Amer. J. Ortho., and Oral Surg. (Oral Surgery Sect.), 31:1 (June) 1945.

UNIVERSITY
OF SYDNEY
DENTAL LIBRARY