A survey of literature submitted as part requirement for the degree of Master of Dental Surgery.

G. T. HUTCHINSON B.D.S.
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Introduction

The development of periodontia as an art has progressed from earliest times. The development of periodontia as a science is of recent origin. This lack of basic knowledge has resulted in endless, and often futile, controversy with regard to all aspects of the subject.

We have endeavoured, in this thesis, to examine principles rather than techniques for, if the principles are correct, suitable techniques are readily developed.

Note:

We have used the terminology advocated by the American Academy of Periodontology (1) since it is the only logically developed system available. It is accepted by the American Dental Association and the British Association of Teachers of Periodontia.
SECTION I.
NORMAL PHYSIOLOGY AND HISTOLOGY.

1. The gingival sulcus.
   (a) Development.

   Following formation of the primary enamel cuticle the ameloblasts degenerate. The epithelial cells comprising the enamel organ are reduced to a few layers of cuboidal cells - the reduced enamel epithelium. Under normal conditions this covers the entire enamel surface, extending to the cemento-enamel junction, and remains attached to the primary enamel cuticle (2). The nature of this attachment of the epithelium is not yet settled. Orban (3) assumes that there exists a highly polymerized cementing substance, a product of the enamel epithelium. Baume (4) indicates that this attachment is due to the tono-fibrils (intercellular bridges) which penetrate the ameloblasts, fuse together and form the primary cuticle.

   At the time of eruption, the reduced enamel epithelium covering the cusps degenerates. The remaining reduced enamel epithelium is then termed the epithelial attachment.

   The fate of this epithelial attachment is the source of some controversy. Gottlieb (5) and Orban (2,5)
state that fusion of the epithelial attachment and the basal cells of the oral epithelium occurs. Then, as the tooth moves towards the occlusal plane (active eruption) the clinical crown is gradually increased by separation of the epithelial attachment from the tooth and recession of the gingiva. The base of the sulcus is to be found at the point of separation.

A slight modification of this view is held by Weski (6) who considers that there is an intra-epithelial splitting of the epithelial attachment, some of the surface cells remaining attached to the secondary cuticle.

Becks (7) presents a different explanation of this process. He demonstrated that there is a progressive degeneration of the epithelial attachment, as it is replaced by the proliferating basal cells of the oral epithelium. Baume (4) supported this view by demonstrating that it is possible to distinguish oral and attachment epithelium by a differential staining reaction. He also considered that his sections showed that the degeneration of the attachment epithelium was due to the fact that their nutrition had been cut off by the basal cells proliferating down, deep to them, through the connective tissue.
The base of the sulcus is in this case found where the degenerated epithelial attachment is replaced by the oral epithelium.

There is actually little difference between these opinions. The latter view of Becks and Baume explains the mechanism of the change described by Gottlieb and Urban.

(b) Epithelial Attachment.

We have shown in the previous section that the reduced enamel epithelium is attached to the enamel and that this epithelium degenerates to be replaced by oral epithelium.

There are opposing views as to how long this epithelial attachment persists after the tooth has erupted.

On histological evidence Gottlieb (2) and others concluded that this attachment existed until the thirtieth year under favourable circumstances. From this followed the conclusion that a sulcus depth of zero was possible and did exist. They also implied that, for many years after the eruption of the tooth, the base of the gingival sulcus could be found up to one third of the way up the enamel.
This theory, first advanced in 1921 and widely accepted since, differed from the then current opinion that, almost immediately after eruption, the base of the sulcus was to be found at the cemento-enamel junction. Black (8) first questioned this theory when he was unable to substantiate, by clinical examination the deductions Gottlieb had drawn from histological specimens. More recently, Waerhaug (9,10) has studied this problem extensively. He directly opposes the view that the epithelial attachment persists for any time at all and bases his opinion on the following points:

1. It is possible to demonstrate an "epithelial attachment" after the epithelium is completely detached and then replaced before sectioning. This demonstrates the fallibility of the microscopic evidence on which Gottlieb based his theory.

2. The gingiva may be readily lifted back from the tooth by a stream of air or water.

3. A thin steel probe may be passed to the cemento-enamel junction of all teeth without resistance.

4. It is impossible to demonstrate clinically a sulcus of zero depth, implied by Gottlieb.

A theoretical argument against the persistence of the epithelial attachment is advanced by Adler (11). He considers that, as the reduced enamel epithelium has finished its function of producing enamel, its elimination is in line with physiological principles of eliminating all
non-functional tissues. This is supported by the observation of Baume (4), that the degeneration of the reduced enamel epithelium is complete soon after the ameloblasts had disappeared. Thus we have two further indications that the demonstrated mechanism of the epithelial attachment, the reduced enamel epithelium, is unlikely to persist until anywhere near the age of thirty.

There appears then, to be no valid reason why the base of the gingival sulcus should be found anywhere but at the cemento-enamel junction.

(c) Passive Eruption.

Gottlieb (5) coined the term "passive eruption" to indicate a gradual increase in the clinical crown brought about by apical movement of the base of the gingival sulcus. He considered this a physiological process and described four stages:

1. Base of the sulcus on the enamel.
2. Base of the sulcus on the enamel, epithelium has proliferated along the cementum.
3. A transitory stage with the base of the sulcus at the cemento-enamel junction.
4. The base of the sulcus is found on the cementum.

We have considered previously whether the sulcus epithelium is over attached to the enamel. That the
bottom of the gingival sulcus can be found on the
conversum is a common observation. Whether this apical
movement is a physiological process must receive further
consideration.

The report of Evaluating Committee I of the 1951
Periodontal Workshop of the University of Michigan (13)
includes the following:-

"The epithelial proliferation is always associated
with inflammatory processes, destruction of period-
ontal fibres, presence of calculus and bacterial
films. It may be concluded that the concept of
continuous passive eruption must be re-examined.
This process of migration of the epithelial
attachment is not necessarily a physiologic
process in all instances, but may be attributed
to pathologic processes."

Williams (13) considers the proposition of involution
of the teeth as a physiological process of ageing, as
implied in this theory, untenable. He supports his
argument by citing observations on Eskimos (14)
where it was found that, despite considerable occlusal
movement of the teeth to compensate for extensive
functional wear, there was no evidence of apical movement
of either the epithelial attachment or the gingival margin.

We agree, for it does not seem reasonable that the
loss of such important functional organs as tooth should
be a physiological process.
II. Clinical and histological appearance of normal gingiva.

(a) Clinical Description.

There seems to be general agreement with the various published descriptions of normal gingiva (3,12,15,16). The results of these clinical studies may be summarized as follows:

The gingiva are pink in colour, of firm consistency with a varying degree of surface stippling. There is a steady slope toward the crown of the tooth ending in a knife-like margin of the free gingiva. The inter-proximal papillae are well formed and fill the interproximal spaces. The sulcus is of shallow depth and free of exudate.

Observations are made that the gingiva of children are redder and more shiny than the adult (17,18). On the basis of clinical and histological study we cannot accept this as normal. We have found that, if children carry out adequate oral hygiene measures, their gingival tissues differ very little from that of the adult.

(b) Histological description.

"The histological appearance of normal gingiva demonstrated by conventional techniques has been repeatedly described. Neuer methods of microscopy and histo-chemical analysis are being perfected so that deficiencies of these techniques become obvious. (19)"
The epithelial surface is intact and is keratinised in all areas since keratinisation, or more properly, hornification, is associated with the cellular changes of all stratified squamous epithelium. The product of this keratinisation – keratin, is absent in the crevicular epithelium (12,20).

All transitions from non hornified to parakeratotic and hornified epithelium of the gingiva have to be considered as within the range of normal (2).

There are epithelial pegs in the marginal gingiva. The formation of these pegs in the sulcus epithelium is considered as a sign of irritation (2,5,21).

The connective tissue fibres are well defined and a specific architectural arrangement may be distinguished. We will discuss this later under the heading of the "free gingival group" of the periodontal membrane.

Histological studies of "normal" gingiva (13,15,16) refer to a constant round cell infiltration of the connective tissue. On the basis of the physiological presence of lymphocytes and other round cells in such areas as the lingual tonsil, it has been argued that the presence of these cells is evidence of a defence reaction in response to the constant presence of bacteria in the gingival sulcus (Boyle (12)).
Bernier (12) however states:

"The presence of small lymphocytes, plasma cells, macrophages and occasional leukocytes in the gingiva at the base of the crevicular epithelium, grouped as they are, presents a histological picture indistinguishable from that seen in inflammation elsewhere."

We agree with this latter view. Serial sections of any inflamed gingiva, however normal it may look clinically, always reveals the reason for the inflammation — ulceration of the crevicular epithelium. We feel that there should be a much more critical view of so-called "normal-gingiva". What is common or even universal is not necessarily physiological.

III. The Periodontal Membrane

(a) Histology

The periodontal membrane, or more properly ligament, is part of the attachment mechanism of the tooth.

The main functional elements are the principal fibres; dense slightly kinked white connective tissue fibres which run from the cementum of the tooth to the alveolar bone, adjacent teeth and gingiva. There are no elastic fibres present in the principal fibres; tooth movement is accommodated by straightening of the principal fibres. These fibres were first divided into
various functional groups by Black. (8).

(1) Free gingival = distributed to the gingiva.

(2) Trans-septal = across the bony septum to the adjacent tooth.

(3) Alveolar crest = from cementum to alveolar crest.

(4) Horizontal = not well demarcated from the above but pass horizontally to be attached to the alveolar bone close to the crest.

(5) Oblique = predominant group, pass occlusally from the cementum to the alveolar bone.

(6) Apical = in immediate relation to the apex of the tooth and are arranged in a fan-like manner.

The function of those groups of fibres in resisting the various movements to which the tooth is subject may be discerned by observing the direction of the fibres. Two outstanding features are the predominance of the oblique group, whose function is to resist vertical occlusal stress, and the action of the trans-septal group = maintaining interproximal contact.

The gingival group have recently been more minutely examined by Goldman (22) who further emphasizes the function of these fibres in maintaining the close adaptation of the free gingiva to the tooth.
Fibroblasts, osteoblasts and cementoblasts, formative cells necessary for the constant remodelling of the principal fibres, alveolar bone and cementum, are constantly demonstrated in the periodontal membrane. The continuous deposition of cementum, stressed by Gottlieb (23) is demonstrated by the organization of the cementoblasts into a continuous line along the cementum.

Orban (24) advances the theory that an important factor in maintaining the cementum formation is the stimulation of the epithelial remnants of the sheath of Hertwig. These epithelial remnants are a constant feature of the periodontal membrane. They have been demonstrated by tangential section (24) to be in the form of a network rather than the more conventional view of separate groups of cells.

(b) Blood Supply.

The generally accepted views of the course of the blood vessels are summarized as follows:

The main artery supplying the tooth divides into the dental and intra-alveolar branches. The dental artery, the smaller, supplies the pulp of the tooth. The intra-alveolar artery enters the supporting bone and emerges at the crest of the alveolar process, inter-
proximally, to supply the gingiva. While passing through the bone, small vessels are distributed laterally through the alveolar bone (lamina dura) where they unite to form a syncitium of vessels within the periodontal membrane. There is some anastomosis with small branches of the dental artery in the apical region. Additional blood vessels arise from the sub-periosteal vessels of the labial and lingual cortical bone. These supply mainly the gingiva but may send small perforating vessels to the coronal part of the periodontal membrane. There is also an anastomosis between the gingival and periodontal groups through the superficial fibres of the periodontal membrane. (2, 25, 26, 27).

Earlier opinions that the blood flow runs vertically through the periodontal membrane (8, 18) do not appear acceptable from a functional aspect. During function the tooth is depressed in the socket; the maximum pressure from this movement is developed in the apical region (28). Thus, if the flow were vertical, functional movement would impede rather than encourage vascular exchange. On the contrary, it is readily seen that the lateral flow is encouraged by functional movement.
(c) **Lymphatic Drainage**

That lymphatic vessels exist in the periodontal membrane may be inferred from their universal presence in functional tissues throughout the body. (29)

Their demonstration, histologically, is simple, since their function of transporting foreign material such as organic dyes makes them readily visible. (2,29,30).

Due to their tortuous course it is extremely difficult to demonstrate their exact distribution.

Orban (2), James and Counsell (21) and others (27,31) consider the lymphatic vessels closely follow the pattern outlined above, for the blood vessels. This seems in line with the associated distribution found in other parts of the body.

On the basis of histological study Black (8) and the basis of histopathology Box (30) and Fish (31) state that these lymphatic vessels run from the gingival sulcus, through the periodontal membrane to the regional lymph nodes.

Study of the histological methods employed revealed that the placing of a blue dye in the gingival sulcus and the later demonstration in the regional lymphatics was considered as sufficient evidence that passage had been through the periodontal membrane.
That Box and Fish agree with this opinion is shown in the statements on the formation of necrotic tracts (Box) and the spread of infection (Fish). Neither of these opinions appear to be confirmed by other studies.

The consideration of these opinions from a functional aspect, presented in the discussion on the blood supply, seems equally applicable with regard to the lymphatic drainage.

We conclude from this discussion that vascular and lymphatic flow of the periodontal membrane is laterally through the alveolar bone, a distribution which reacts favourably during function.

IV. Normal Occlusion.

Normal Occlusion has never been defined and there is accordingly great confusion over this problem.

Langhorne (33) presents a concept of the development of normal occlusion which seems reasonable.

This may be summarized as follows:

"When the teeth erupt, they are guided into their first contact by the balanced muscle pressure of the tongue, lips and cheeks. With first occlusal
contact the tooth are guided into correct position by the inclined planes of the cusps. The interdigitation of these cusps maintains this position until there is sufficient strength and hardening of the supporting structures to maintain this correct relation without the aid of interlocking cusps.

The cusps having fulfilled their function are then worn down by gradual wear during function and finally disappear. With loss of posterior cusp height the anterior overbite is gradually reduced and with the tongue maintaining contact between the upper and lower teeth, an end to end occlusion is developed."

Gottlieb (5), "An ideal occlusal relation between anterior tooth is an end to end bite or a similar relation" and others (15,35,54) support this concept of the ideal occlusion. Studies of races noted for their good natural dentition (14) and clinical examination of patients with good dentitions indicate that healthy periodontal tissues are co-existent with such occlusions. Whether the nature of the occlusions alone, or the function necessary to obtain such occlusions, is the operative factor, remains a point for discussion.

The predominance of the oblique group of periodontal fibres, whose main function is to resist vertical stress, indicate that the increased vertical and the decreased horizontal stress created by this type of occlusion is well tolerated.
Other theoretical considerations which support this view are —

(1) Deposition of secondary dentine protects the pulp from exposure during wear of the occlusal surface;

(2) Continuous cementum formation to maintain the attachment of the tooth as it erupts to adjust the vertical dimension reduced by occlusal wear.

We feel that the concept of an end to end occlusion has a reasonable enough biological and functional basis to be considered as a normal occlusion.

V. Normal Diet.

If one accepts the concepts of normal occlusion and normal supporting structure, outlined previously, then it is reasonable that the foods of these people with such oral conditions constitute a normal diet.

Price (35) has made an extensive study of such groups of people. His outstanding observations were:
Healthy racial groups, both orally and generally, had an adequate supply of all nutrients.

Change to highly prepared "civilized" diets resulted in rapid degeneration of these people in all respects.

The importance of function has been emphasized by Baaregaard (14) and others in reports on studies of Eskimos and other native races. (34).

Those two features are correlated since it is frequently shown that unprepared foods, i.e., those requiring function during mastication, contain greater amounts of their original nutrients than those foods softened by extensive preparation.

The optimum degree of function is unknown. There is some evidence (32) that it should be sufficient to gradually reduce the cusp height but not more rapidly than the secondary dentine is deposited to protect the pulp.

VI. Normal X-Ray Appearance of the Teeth and Supporting Bone

The trabeculae of the supporting bone are of an even density throughout (56). The compact bony surface of the socket and alveolar crest visible as the "lamina
The periodontal membrane is of an even thickness throughout (36) and should be one and a half times as thick as the lamina dura (37).

That dense alveolar bone offers a better prognosis, and could therefore be considered normal, has been advanced by Fish (34) and many others. Though no definite basis has been demonstrated for this hypothesis, it is probable that the dense arrangement of the bony trabeculae, as an expression of good function, is associated with good prognosis. The function is the operative factor; the dense trabeculae the result.

Orban and Ritchie (38) have recently added certain refinements to these observations. They have shown that long narrow interproximal crests are associated with teeth having broad flat contacts and wide interproximal crests are found with teeth with extremely convex interproximal contacts.

That a tooth with a large root area has a better prognosis is recognised (39). There has, however, been no attempt to classify these varying root forms with any degree of accuracy.

These observations indicate that the normal X-Ray appearance is reasonably well defined. Future studies
will probably supply a more detailed knowledge.

Summary:

The tooth and supporting structures are a well-designed functional unit. When receiving optimum function and adequate nutrients, the maintenance of this unit throughout life is not only possible but logical.

The form of the tooth and supporting structures is such that they are self-cleansing. The gingival margins are held closely adapted to the tooth surface and are further protected during function by the convexity of the tooth.

Functional movement of the tooth increases the rate of vascular exchange and lymphatic drainage. Such function is better tolerated in a vertical direction.

Normal occlusion, normal diet, and normal function have not yet been defined. There are, however, indications of the basic requirements of these definitions.
SECTION II.

NOMENCLATURE.

A fixed nomenclature in a subject with as many unsolved problems as periodontia is impossible. It is, however, possible to establish principles of nomenclature which are plastic enough to designate new findings accurately (40, 41).

A standard classification has a decided advantage in the exchange of scientific knowledge and in teaching. As an aid in treatment planning this classification should also indicate the tissue, or tissues involved, and the nature of the change.

Early periodontal nomenclature was opynymous (40, 41). The disease processes were designated by the proper name of the clinician who first described the condition e.g. Riggs Disease. This system demanded a knowledge of the initial description, not necessarily complete, and did little to convey an understanding of the precise pathology involved.

Development of another classification to replace this obviously faulty system resulted in the symptomatic designation of the disease process (41). Examples are
pyorrhoea alveolaris, pyorrhoea simplex, which, among others, are still in use today. Much of this terminology developed when the basic pathology was imperfectly understood. It has a similar fault to the previous system; there is no method of describing the tissue changes.

The more widely accepted method of nomenclature, in fields other than periodontia, is to form a word. The prefix is the name of the tissue involved. The suffix describes the type of process.

There are two schools of thought with regard to the prefix (44). The American accepts peri; the European para. Each group claims the support of eminent classicists.

Par - at the side of, as used in parathyroids.
Peri - around as in peri-vascular. The latter appears to describe the relation of the periodontal membrane to the tooth more accurately, even if it does imply a complete enveloping. Since these terms do not form any operative part of the word when used, there seems little object in endless discussion of this matter.

The tissues involved are the gingiva and later alveolar bone, periodontal membrane and cementum. The inclusive term periodontium appears widely accepted.
The reactions in these tissues may be either, inflammation when the suffix "itis" is used, or degeneration when the suffix "osis" appears to be acceptable. The varying reactions of inflammation such as hyperplasia, ulceration and necrosis may occur. These terms are then used to indicate the type of inflammatory reaction. A further indication of the stage of inflammation is found in the terms acute, sub-acute and chronic.

A description of the position (43) and extent (44) of the lesion may further aid in the formation of a useful terminology. The basic terms generally accepted are:

- gingivitis
- gingivosis
- periodontitis
- periodontoses

There are, however, many faults immediately discernible, of even this basic classification. Schour (43) is quite correct when he suggests alveolar osteitis is a more accurate description than periodontitis.

The terms atrophy and pre-senile atrophy have been used. There seems reasonable evidence that these are only variations of the inflammatory process (45,46).

On the contrary the term "gingivosis" appears more firmly established. Recent evidence indicates that degeneration may be differentiated from inflammation in the condition originally designated "chronic desquamative
gingivitis" (47).

We do not propose to extend this survey. In the present state of knowledge an extensive discussion attempting to finalize a nomenclature for periodontal disease is illogical.
SECTION III.

THE PERIODONTAL LESION.

Clinical description, histopathology and X-Ray appearance of the various changes.

1. Gingivitis.
   (a) Histopathology.

"The inflammatory reaction seen appears to be no different to that found in other body tissues; the tissues involved are epithelium, connective tissue and bone" (Bernier).

The initial change in the gingiva is first observed in the connective tissue immediately underlying the crevicular epithelium. There is an increase in capillaries; probably due to a nervous reflex. (27).

The sulcus epithelium then reacts to the irritant by an increased mitotic activity of the basal cell layers which spreads to the prickle cell layers. This increased activity of the formative layers of the epithelium leads to increased desquamation of the surface cells within the sulcus (27,51). Early degenerative changes are then visible in immediate relation to the gingival sulcus. The connective tissue fibres are swollen and
dogenerated. The structure of the area is masked by a dense infiltration of inflammatory cells. The epithelium is ragged and degenerated, frequently ulcerated.

The increased activity of the epithelium is maintained. The basal cells of the epithelium proliferate markedly and extensions of the epithelium are found invading the connective tissue. These down growths of epithelium break up the oedematous gingival and trans-septal fibres and the inflammatory reaction spreads toward the alveolar crest. (17,20,21,27,31).

With the loss of the free gingival fibres the gingiva loses the close adaptation and the normal sulcus becomes a gingival pocket (48). For many reasons, the reaction may vary from acute to chronic. This change is reflected in the nature of the inflammatory cells present. Polymorphonuclears in acute inflammation; plasma cells and lymphocytes in the chronic condition.

(b) Clinical Description.

As may be expected in a reaction influenced by a multitude of factors, the clinical appearance, as an expression of underlying pathological changes, is subject to many variations. The biological basis for many of
those variations is not well understood.

The first clinical evidence of a change from normal is evidenced by a swelling and loss of stippling of the gum margin, almost entirely localized to the interdental papillae (49). The inflammatory reaction as demonstrated by a visible hyperaemia then involves the marginal gingiva.

The colour varies from red to blue-purple depending on the vascular state of the tissues. Red indicates an active hyperaemia of the acute and subacute conditions. The bluish changes disclose an extremely chronic state of venous stasis associated with a slowly progressing lesion. The excess vascularity of the tissues is readily demonstrated clinically when slight trauma induces free and copious bleeding.

With increase of intra-cellular fluids and inflammatory exudate within the connective tissue there is a change in form of the tissues. The normal closely adapted gingiva become rounded and enlarged. Destruction of the free gingival fibres (48) results in the loss of the close adaptation of the gingiva. The gingival pocket so formed destroys the normal self cleansing form of the area and further accumulation of irritants is encouraged. Subgingival calculus may be deposited.
The irritants become more closely placed to the deeper tissues and the lesion progresses. (10).

In the early stages the edematous nature of the enlargement may be demonstrated clinically by pitting on pressure. With continuation of the irritant but increased resistance of the tissues the reparative stages of the inflammatory reaction are manifest and the tissues become fibrosed (50). Both these changes are classified under the heading of Hyperplastic Gingivitis. There seems to be a tendency for these hyperplastic changes to be associated with hormonal changes of pregnancy and puberty (51,52). Attempts by Ziskin (53) to describe more typical changes have not been well supported. (51,52). An exceedingly vascular localized gingival hyperplasia has been described. Ziskin (53) considered this as a "pregnancy tumour", the excess vascularity being attributed to the modifying influence of the hormonal changes present at this time. The fact that a similar lesion described as "Granuloma Pyogenicum", indistinguishable histologically, has been demonstrated in both sexes, at all ages, make it doubtful whether such a specific term as "pregnancy tumour" is valid (54).

Under certain circumstances which may be due to increased virulence of organisms (55), reduced resistance of the tissues (56,57) or a combination of both, an
exceedingly acute reaction occurs. Originally classified under the heading of Vincent's infection it is more properly an acute gingivitis (58). This has been modified under American terminology to the formidable acute necrotizing ulcerative gingivitis (17).

Clinically a typical picture is found. The anaerobic nature of the organisms is emphasized by the location of the early changes in areas of reduced oxygen tension such as third molar flaps and interproximal stagnation areas (55).

The interproximal tissues show a typical punched out appearance. The mucosa and gingiva are intensely hyperaemic, swollen and bleed easily, perhaps spontaneously (17,18). The lesions are covered with a greyish yellow to white membrane. Additional signs are a foul odour and glandular enlargement. The patient may show signs of general malaise and complain of pain, metallic taste, excess salivation, wedging apart of the teeth and a feeling of looseness of the teeth.

Chronic Vincent's infection is probably no different from chronic marginal gingivitis (58). If it is a sequel to the acute stage a typical malformation of the tissues may still exist.
II. Periodontitis.

Periodontitis is the result of a progressive gingivitis; the differentiation depending on the state of the periodontal fibres. A periodontitis is considered to exist when the periodontal fibres are invaded by the inflammatory cells. An arbitrary division—

(a) Histology.

The inflammatory process extends along the interceptal vessels, directly to the narrow spaces of the supporting bone. On labial and lingual the spread is along the periodontal side of the cortical bone (12,21,31). An osteitis then develops and there is loss of cancellous bone. The view held by Fish (34) and others that the inflammation spreads directly to the periodontal membrane along the lymphatics does not appear to be supported by other histopathological studies nor the accepted distribution of the lymphatics.

The cortical bone of the socket often remains intact giving the typical notch like appearance found in X-Rays of the early stage of this disease (59). The periodontal membrane is edematous for a considerable distance down the root. The alveolar bone is then destroyed, the periodontal membrane is invaded and the principal fibres destroyed. Through lack of stimulation
and direct invasion the cementoblasts are killed and the cementum ceases to be a vital tissue. This exposed and necrotic cementum is rapidly covered by proliferating sulcus epithelium. With detachment of this epithelium from the cementum, the gingival pocket becomes a periodontal pocket.

Subgingival calculus is always present within the pocket and, as a source of bacterial accumulation and physical irritation, contributes to the extension of the lesion. Waerhaug (9) has shown that, if the calculus approaches closer than about 1.43 mm to the bottom of the pocket, there is further destruction of bone.

Gottlieb (23, 46) considers this proliferation of the epithelium along the cementum is an expression of one of the activities of epithelium; namely the exfoliation of foreign bodies of which necrotic cementum is an example. Fish (34) attributes this apical movement to the lack of physical resistance originally supplied by the periodontal fibres. Aisenberg and Aisenberg (60) claim that the epithelium exerts a more positive reaction and has some lysing affect on the tissues.

We believe that the well established fact that epithelium proliferates to maintain a surface continuity is sufficient explanation for this downgrowth. This is
well supported by the observations made when re-attachment of the gingival and periodontal tissues is attempted. The main problem is to prevent the epithelium proliferating to cover the granulation tissue before the blood clot is organized.

The theories of Gottlieb, Fish and Alsenberg appear unnecessary.

If healing of the tissues occurs, without surgical interference, it does so by the formation of granulation tissue. The epithelial continuity is re-established but there is no regeneration of the original form (27). The trans-septal fibres are formed at a lower level (5).

If a pocket forms below the level of the alveolar crest it is termed an Intrabony pocket (17). This may occur, when for various reasons only one of two adjacent teeth is affected interproximally, or, on the palatal of the anteriors where the cortical bone of the palate is not included in the inflammatory destruction (17,16).

(b) Clinical description.

A gingivitis of varying degree is always associated with periodontitis. With slowly progressive lesions this may not be obvious as the active ulceration is often
found in the depths of the pocket (34).

The main clinical feature of periodontitis is a deepening of the gingival sulcus below the cemento-enamel junction. This indicates that the essential change, differentiating from gingivitis, destruction of the periodontal membrane, has taken place.

Pus or other exudate may be expressed from the pocket. Subgingival calculus and food debris are frequently found below the gingival margin.

If there has been extensive bony destruction the teeth may be mobile. This sign is a late demonstration of this condition.

(c) **X-Ray Appearance.**

The earliest changes in periodontitis visible radiographically are destruction of the continuous outline of the lamina dura of the alveolar crest, and thickening of the periodontal membrane in the coronal part. A typical notching of the alveolar crest then appears (17,59). Miller and Pelzer (56) relate the extent of the darkening of the bony shadow to the rate of progress of the condition. A wide area of changed density indicates a rapidly progressive lesion. The bony destruction is at a reasonably uniform horizontal level throughout
the mouth—the so called periodontitis simplex.

III. Gingivosis.

(a) Clinical Description.

The term gingivosis was proposed by Schour and Massler (61) in 1945 to designate a degenerative lesion of the gingiva found in starving Italian children. The primary changes, which were cyclic, resulted in extensive tissue destruction in the absence of any marked inflammation. To quote the authors:

"The clinical impression is that the disease is of a degenerative rather than an inflammatory nature."

The non-inflammatory nature of the change is further emphasized by successful systemic therapy with niacinamide, pyridoxine and V-B complex. Local therapy was unsuccessful.

Wainwright (62) describes a similar change associated with pantothenic acid deficiency.

A similar cyclic, degenerative change of the gingiva associated with the menopause has been known for at least one hundred years. A description of this condition, under the heading of "Desquamative Gingivitis", by Sorrin (63) is very similar to that of Schour and Massler (61) for their gingivosis.
Sorrin outlines changes which may be summarized as follows:

The first symptom is a slight soreness around the necks of the teeth. The gums thicken and become red. This redness soon spreads to the remainder of the oral mucosa. Cyclic ulceration of the tissues then begins. The gums become smooth, bleed readily and the patient complains of pain and a burning sensation. To this may be added the observation by Ziskin (64) that watery blisters occur in the pre-ulcerative stage.

The degeneration associated with VIt. B deficiency apparently resulted in greater total tissue destruction but, in all other respects, the description appears remarkably similar to the above.

Reports of successful therapy using oestrogons support the view that the lesion is degenerative (64,65). This classification is supported by Orban, et al (47) who head a recent extensive contribution on this subject "Gingivosis".

(b) **Histopathology.**

The areas involved are either partially or completely devoid of epithelium. The remaining thin strips of epithelium are in active stages of degeneration
as evidenced by extensive hydropic degeneration and intercellular oedema. Vesicles, due to the accumulation of fluid in the sub-epithelial layers are occasionally visible. The corium is markedly oedematous and beneath the areas of ulceration show extensive round cell accumulation (63).

Orban (47) demarcates clearly, in his sections and observations, the inflammatory change resulting from ulceration of the sulcus epithelium and that from desquamation of the marginal epithelium. A further indication that the lesion may be differentiated from the usual course of chronic marginal gingivitis. Engel, et al (66) using newer methods of histo-chemistry have added the following observation and offer the probable reason for the epithelial desquamation:

"In desquamative gingivitis there is a disturbance of the ground substance of the gingival connective tissue. This is manifested by changes in the properties of the interstitial components.

(a) The basement membrane is either absent or partially dissolved.

(b) The ground substance contains increased quantities of water-soluble glycoprotein and water soluble alcohol insoluble glycoprotein residues."

We conclude from this that gingivosis, a degenerative lesion of the gingiva is a clinical entity. At the moment there are apparently two types -
(a) Due to lack of Vit. B group.

(b) Due to a hormonal disturbance associated with the menopause.

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IV. Periodontosis

Periodontosis is a general term which refers to "a degenerative non-inflammatory destruction of the periodontium originating in one or more of the periodontal structures characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease." (1)

(a) Histopathology.

There is little agreement on the histopathology of periodontosis. Gottlieb (46) who first described the clinical entity suggested that the primary change was that of failure of deposition of the cementum or "deep cementopathia" as he termed it. Orban and Weinmann (67) describe the following four degrees of change:

(1) Degeneration and disorganization of the principal fibres of the periodontal membrane.

(2) This leads to cessation of cementum deposition of the alveolar bone which they attribute to lack of functional stimulation, and increased tissue pressure caused by oedema and capillary pressure.
There is then rapid proliferation and migration of the sulcus epithelium along the root. There may be proliferation of the epithelial remnants in the periodontal membrane.

With development of these deep pockets and accumulation of irritants a secondary periodontitis rapidly follows."

Goldman (17) and Box (68) describe similar changes the latter using the term "rarefying pericementitis fibrosa". Although there is reasonable agreement on the histology of the change, the interpretation remains controversial. All the authors mentioned above consider the change degenerative in origin. Bernier (69) states that the change is a variation of the inflammatory reaction in which the degenerative phases of inflammation overshadow the other phases. Ransford (45) considers the change as "aseptic inflammation" of a variable nature; "Ischaemia, hyperaemia, oedema, atrophy, degeneration and even necrosis may occur" (45). The rarity of this condition, particularly when not obscured by inflammation due to local irritants, makes study difficult. At the present moment no definite conclusions may be drawn as to the correct interpretation of the histopathology of this lesion.
Clinical Description

That such a clinical entity exists is a better established fact than the histopathology of the lesion. It was first recognized by Gottlieb under the term diffuse alveolar atrophy and shortly after by Box (68) as "periodontitis complex". The term Periodontosis was developed much later, under the auspices of the American Academy of Periodontology, in an effort to emphasize the degenerative nature of the lesion.

Clinically, the outstanding early features are increased tooth mobility and tooth migration. Absence of gingival irritation with deep pocket formation is characteristic. Labial drift of the upper anteriors and the formation of adiastema occur early. The drifting of the teeth is explained as a result of pressure from the edematous periodontal membrane (5,67).

To find such a condition clinically without complicating periodontitis is rare. Lack of contact teeth and deep pocketing lead to the accumulation of irritants. Secondary gingivitis and periodontitis rapidly follows. This multiple condition of degeneration and inflammation is generally recognized under the heading "periodontitis complex".
(40)

(c) **X-Ray Appearance**

A summary of the various changes visible radiographically is given (17,18,19):  

(1) An arc-like loss of contour of the bony support of the first molar which extends from the mesial surface of the second molar to the distal surface of the second bicuspid. This is associated with bone loss in the incisor area.

(2) Thickening of the periodontal membrane with absence or haziness of the lamina dura.

(3) Bony destruction follows a vertical rather than a horizontal direction.

(4) A generalized alteration in the trabecular pattern of the alveolar bone, with increased size of cancellous spaces.

(5) Generalized loss of alveolar bone accentuated in the first molar and incisor region.

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V. **Gingival Recession**

(a) **Clinical Description**

A condition in which atrophy and recession of the gingival tissues occurs has been described. The clinical features of inflammation are not apparent; the tissues in fact appear anemic. The tissue loss is even around all teeth and there is no deepening of the sulcus nor formation of exudate. When occurring in young people the term pre-senile atrophy is used (17,18,19). Uncomplicated
cases are rare, for, with loss of gingival architecture, food retention favouring inflammatory changes is encouraged. (72).

Another type of gingival recession, associated with tooth brush trauma, may be distinguished. It is distributed in the more accessible areas and is associated with signs of abrasion on the teeth (75).

A third type of gingival recession may be noted. Healing of a gingivitis or treatment by surgery of a periodontitis often leads to the establishment of a normal gingival architecture at a lower level. Thus the appearance of a senile atrophy is simulated.

(b) Histo-pathology

There have been no conclusive studies of this condition. Generally, there are signs of decreased cellular activity such as reduction in mitotic activity, decreased vascularity and so on. It is important to note that there is a constant inflammatory reaction present. (72).

(c) X-R a y Appearance

There is a generalized loss of bone. The lamina dura of the crest may be indistinct but there is no extensive area of bone darkening (34,36). In other
words, the bone structure appears relatively normal except that it is situated at a lower level.

VI. The Periodontal Membrane

Histopathology.

The periodontal membrane as a functional mechanism reacts to the varying degrees of pressure to which it is subjected. The changes may be demonstrated histologically.

Hypofunction.

The principal fibres are reduced in number and are replaced by loose fibrous tissue. The trabeculae of the supporting bone are reduced and there is an enlargement of the marrow spaces (17,25).

Normal Function.

Normal function has never been defined. Histologically a periodontal membrane which demonstrates the acceptable architecture of the principal fibres, in the absence of signs of destruction or inflammation, may be considered in normal function.

Hyperfunction.

Increase in pressure from normal may be taken to affect the periodontal membrane in a graded series of changes.
There have been no studies of the nature of the changes found in "moderate, intermittent horizontal over-stress", (Agnew & Gratzinger (12)), though there are indications that the changes described under the heading of "periodontosis" are an expression of a force of this nature (45).

A study of the architecture of the periodontal fibres indicate that an overloading is unlikely to develop in a vertical direction. Most studies have examined the effects of increased horizontal stress.

Bax (33) and Stone (74) have demonstrated experimentally the effects of severe acute overloading. They found pressure necrosis of the periodontal membrane and surrounding alveolar bone. The necrotic tissue was removed by cellular activity of the surrounding vital tissues.

Further knowledge of the changes in the periodontal membrane have been obtained from studies of the effects of pressure by orthodontic wires.

Light Pressure.

Areas of pressure and tension develop in the periodontal membrane. There follows a remodelling of the support of the tooth and the tooth moves slightly
until the pressures are equalized (73). On the pressure side there is osteoclastic activity, from the periodontal membrane, until the normal width is re-established. Deposition of bundle bone on the tension side effects similar changes. Bone is also formed on the external alveolar surfaces, as the tooth moves, to maintain the supporting bone. (72).

Heavy Pressure

There is pressure necrosis of the periodontal membrane and alveolar bone on the side of pressure. This necrotic tissue is removed by giant cell activity originating in the alveolar bone. On the tension side the periodontal fibres may be torn or, more frequently, pieces of cementum are detached from the surface of the tooth. As a result of this pressure the tooth will re-adjust its position; but more slowly than with light pressure. (5,12,33).
SECTION IV.

ETIOLOGY OF PERIODONTAL DISEASE.

1. Bacteriology:
   (a) Bacteria.

The total number of organisms present within the mouth is enormous. Most of these seem saprophytic. Obvious and known pathogens which do not appear to be associated with any typical disease process may be transient. They are probably due to contamination from the surrounding air or the associated respiratory tract. Some of the organisms present may become pathogenic under favourable conditions (76) and be active in a type of endogenous infection (77).

Bacteriological studies of periodontal disease have not been able to implicate any one organism "but a characteristic group of micro-organisms is found in association with the lesions of all types of periodontal disease" (Rosebury)(77).

The constant species include "alpha and gamma streptococci, anaerobic streptococci, anaerobic vibrios and several varieties of spirochaetes and fusiform bacilli". These types vary quantitatively from patient to patient and
from area to area in the same mouth" (78). Most of the organisms of pathogenic significance appear to be strict anaerobes. Fosdick, et al (79,80) support this relation of anaerobic organisms to periodontal disease and give a list of seventeen different organisms which may be active.

The association of these groups of organisms with periodontal disease is apparent; whether the relation is one of cause or effect is unknown.

Rosebury (78) has been able to produce typical necrotic lesions in experimental animals by innoculating groups of organisms found in various stages of periodontal disease. Repeated transmission leads to the elimination of many extraneous organisms and the final inoculant contains one or more small spirochaetes, small fusiform bacilli, mobile vibrios and anaerobic streptococci (78). These organisms do not appear to be effective in pure unmixed culture.

Attempts to produce similar necrotic lesions in man have failed (37) but Kirkpatrick (81) reports a successful experimental inoculation of animals, particularly in those with a predisposing systemic disturbance. We feel the failure to produce the condition in man insignificant as no attempt was made to duplicate the condition.
associated with a necrotic gingivitis. The placing of these organisms on a normal gingival margin is a vastly different proposition to the masses of the organisms held in close contact with the gingival margin, protected from the oral fluids by accumulated debris, and in the presence of tissues which are metabolically sick either from local or systemic disturbances — the conditions found in association with this acute ulceration clinically.

Further evidence of an active association is given by Rosebury (37),

"The dramatic effectiveness of penicillin in abolishing infection and its immediate sequels in both oral and extra-oral fusco-spirochaetal disease supports the view that infection is an essential factor in their causation."

And Kirkpatrick (76),

"Wherever the fusco-spirochaetal organisms are found some pathological condition exists".

Although this evidence is inconclusive we feel that the passive role attributed to this group of organisms is incorrect.

(b) **Viruses**

Studies of virus activity have demonstrated no relation with periodontal disease in either the acute
or chronic forms. Viruses seem to be associated with acute herpetic stomatitis and recurrent apthous stomatitis.

(c) Bacterial Products.

Under the heading of toxins Fish (34) has often associated bacterial products with periodontal disease.

More recently there has been a study of specific substances which may have an action on the intact mucous membrane (32).

Oral bacteria known to produce such substances are:

1. "Staphylococci (pathogenic) produce a coagulase which coagulates fibrin. This may favour bacterial activity by sealing off the bacteria from the tissue defence mechanisms or vice versa, by limiting the spread of infection."

2. Group A, haemolytic streptococci produce a fibrinolysin which dissolves fibrin clots and thus aids bacterial spread.

3. Staphylococci produce hyaluronidase Streptococci which acts on Pneumococci hyaluronic acid, one of Clostridia (some) the intercellular cementing substances of the body."

This last substance has been shown to have some action on the gingiva and the periodontal membrane (33, 84). It is capable of penetrating an intact mucous membrane and initiating an inflammatory response in the connective
tissue (83).

(a) Polyopeptides, etc.

An associated, though as yet undemonstrated, hypothesis (82) is, that, during proteolysis of food debris, certain breakdown products of the proteins are formed which may be capable of damaging the tissues.

We conclude from this discussion that there is increasing evidence that certain demonstrated groups of anaerobic organisms play an active role in periodontal disease.

The mechanism of this action has received some clarification by the demonstrated effect of one of the bacterial products, hyaluronidase, on intact mucous membrane.

II. Calculus.

The formation of calculus has always been a subject for dental research. The complete mechanism for this process has not been explained.

There are three problems:

1. Precipitation of the calcium and phosphate ions.

2. Cohesion of the precipitate.
3. Adhesion of the accumulation to the tooth.

(a) Precipitation.

Precipitation will occur:

1. If calcium or phosphate ion is increased the solubility product, which is a constant for any one solution, is changed. There will then be a precipitation of calcium phosphate until equilibrium is re-established.

2. If the ability of the saliva to retain these ions in solution is altered, e.g. Change in pH.

There are various theories advanced to demonstrate methods whereby some of these changes may take place.

Adamson (35) demonstrated the enzyme phosphatase in inflamed gingival tissues. This enzyme is known to have an action on organic phosphates, that results in the splitting of the compound and liberation of the free phosphate ion. Adamson further demonstrated the ability of this substance to form a precipitate of calcium phosphate from saliva under experimental conditions.

Bibby (36) has experimentally demonstrated the necessity for bacteria in calculus formation. Many writers consider these bacteria act by changing the pH in their immediate environment. The ability of organisms to produce this change has never been demonstrated (37).

A change in pH of saliva has been attributed to many other factors. The most common one is that of loss of
carbon dioxide gas from solution. The action of carbonic anhydrase on the bicarbonate group in further aiding this evolution of gas has been described (88) and the loss of the gas has been demonstrated.

All of these theories present possible mechanisms but each may be found wanting from many aspects.

They do not, for example, explain the uneven distribution of calculus within the mouth, nor do they explain the different distribution of supra- and sub- gingival calculus (89).

(b) Cohesion and Attachment of the Precipitate.

There is general agreement that an organic matrix for the calculus is formed by the colonies of branching filamentous organisms constantly found within the calculus (53, 86, 87). Black, (8), experimentally demonstrated that a roughened surface was essential before the calculus could be deposited. The attachment to the enamel is probably associated with the secondary enamel cuticle and to the cementum by actual growth of the organisms into the surface irregularities (86).
(c) Variations in Appearance.

Clinically two types of calculus are described. A soft light coloured, quickly deposited and lightly adherent supra-gingival calculus. This is found largely on the teeth close to the openings of the salivary ducts. This is in contrast to the hard dark and firmly attached subgingival calculus found in gingival and periodontal pockets. Goldman (17) and most other writers agree that there is no difference in the method of precipitation of the different types of calculus. The difference in texture is the result of compression by the surrounding gingival tissues, the dark colour the result of staining by products of the pocket exudate and the firmer attachment is explained by the more favourable mechanical action of the roughened cementum surface.

We believe there is sufficient evidence (33,85,80) to support the proposition that subgingival calculus is a product of the pocket exudate rather than of the saliva. Nicholls (90) states that it is "unlikely that saliva will penetrate to the depths of the periodontal pocket, six millimetres deep, especially in the face of a continual flow of exudate from the pocket."
(d) Role of Calculus in etiology of Periodontal Disease.

**Supra-gingival calculus.**

The action of this material is similar to that of many other local factors in periodontal disease and will be discussed more fully under that heading.

**Sub-gingival Calculus.**

Clinically, calculus appears to play a major role in periodontal disease; if it is removed the inflammation abates (17,45). There is evidence given above, that the sub-gingival calculus is the result of gingival inflammation. Waerhaug (9) has shown that if calculus is deposited closer than about 1.4 mm to the bony crest, there is bony resorption.

The action of calculus is two-fold. The physical irritation to gingiva moved over its rough surface (34) and the action of the massive bacterial flora contained within the calculus (9).

We may conclude that, although the result of gingival inflammation, subgingival calculus plays an active role in periodontal disease.
III. The Role of Local Factors in Periodontal Disease

Many local factors have been assigned a role in the initiation and progress of periodontal disease (18,25, 90,91). Their association with the disease process is evident. A causative connection is established by the cessation of destruction when the irritant is removed. The mechanism of these varying factors is not well established.

The initial lesion is a break in continuity of the sulcus epithelium. This destruction may be due to a single traumatic incident followed by secondary bacterial invasion. Secondly the local factors may so alter form and therefore function, of the oral cavity that the accumulation of bacteria and food debris is encouraged. The products of this stagnation then may damage the epithelium.

Thirdly the alteration in function may, by decreasing the blood supply and therefore the nutrition of the surface cells, reduce the local threshold of resistance (92).

(a) Acute Traumatic Damage with Secondary Bacterial Invasion.

Fish (34) states"...the gum margins are softer and are thus constantly damaged by incidental harder fragments in the food". Examination of this author's
Illustrations show that he supports the general view that the initial lesion is within the gingival sulcus, an area protected from damage by food fragments. In addition we consider it unlikely that a patient could continue such painful trauma sufficiently long to be effective. Similar arguments may be advanced against the damage by tooth brushing and interdental stimulators. These instruments may induce gingival recession; it is extremely unlikely however that the patient would persist in producing painful traumatic ulceration of the gingiva until a chronic gingivitis develops.

Clinically, it is found that traumatic damage to the gingival mucosa heals rapidly.

We consider acute mechanical damage to epithelial continuity plays little part as an etiological factor in periodontal disease.

(b) Local Factors as they Alter Form and Function.

We have shown previously that the normal mouth is self-cleansing in form, particularly in association with the function of a normal diet.

Local factors associated with a change in form may be listed:-
1. Supra-gingival calculus.
2. Destruction of tooth structure by dental caries.
3. Poor restoration of tooth structure: more specifically overhanging margins, rough margins and unpolished surfaces of the filling, no interproximal contacts and poor occlusal anatomy.
4. Poor design of prosthetic restorations which damage by direct pressure of the appliance or by allowing food impaction.
5. Mal-position of the teeth, irregular alignment, and often interproximal contacts.

It is readily seen that each of these varying factors prevents the removal of food debris during normal function.

The materia alba and mucinous plaques so formed are protected in the deeper parts from saliva and gaseous exchange and a diminished oxygen tension is soon established (55). There is then a proliferation of anaerobic bacteria (76) and putrefaction of protein substances in the debris (82). The increase in bacterial enzymes and protein breakdown products then initiates the damage to the sulcus epithelium (82).

In addition, the constant pressure of excess amalgam, calculus, dentures and so on, by interfering with the blood supply of the tissues, reduces the supply of nutrients to the epithelial cells, and influences the ability of the epithelial cells to resist the action of the above substances (92).
Herschfeld (93) reports a similar change from food impaction, the result 
of many of the above changes in form of the teeth.

(c) Mouth Breathing.

Mouth breathing can readily be assigned a position under a functional heading. Firstly it implies the unnatural use of the mouth as an air passage. The passage of air over the gingiva has a drying effect which by changing the environment reduces the resistance to local irritants (17,94). Secondly, the imbalance of oral musculature leads to development of a typical mal-occlusion. The poor function of these teeth encourages the accumulation of local irritants.

Emalie (94) considers that, in the absence of stagnation, only a mild irritation is produced. We support this view for we have found, clinically, that an adequate regime of tooth brushing, has, by eliminating these irritants, returned the gingiva to normal.

(d) Diet - Local Changes.

Contemporary methods of food preparation result in the presentation of most foods in a state requiring little chewing. During the little mastication necessary, the food is rapidly reduced to a finely divided semi-liquid mass
After deglutition there remains clinging to the protected areas of the tooth a mass of food debris (95, 96, 97). We have already discussed the role of this materia alba under other headings.

The little function required results in reduced blood flow in the periodontum. The reduced nutrition of the gingiva reduces the local threshold of resistance to infection (97). We believe the importance of this reduced function has been insufficiently stressed. Studies of mouths in heavy function have demonstrated that, despite accumulation of debris around the gingival margins, progressive periodontal disease is not found (14). The importance of function has been further stressed by Lyons (98) and Williams (99) who consider the functional stimulation of chewing is much more valuable and important than artificial stimulation.

(e) Poor Oral Hygiene.

If we are to accept soft diets then application of artificial methods are necessary to maintain the mouth in a healthy state. Lack of application of these measures has an obvious result. Progress of the lesion is due to the continued presence of these irritants.
Destruction of correct oral form, as the result of the initial lesions, results in the accumulation of greater quantities of debris.

(f) Summary.

We have presented an hypothesis which attempts to explain the mode of action of local factors as they imitate periodontal disease.

The more usual practice is to supply extensive lists of these local factors, their part in the etiology of periodontal disease being accepted as proven by association. We believe that continual repetition of these lists adds little to the further knowledge of periodontal disease. Though much of this hypothesis remains undemonstrated there are indications that it is not without a reasonable basis.
IV. Occlusal Dysfunction

The role of occlusal dysfunction in the initiation and progress of periodontal disease is a most controversial matter. This dysfunction may be of two types, hypo- and hyperfunction.

(a) Hypofunction

We have discussed the changes in the periodontal membrane of a non-functional tooth previously. We have also indicated that function is an important factor in maintaining the health of the periodontal membrane and supporting bone. The degree of this optimum function has not yet been defined.

It appears that, apart from reduced tissue resistance, the role of hypofunction in relation to periodontal disease has not been determined.

(b) Hyperfunction

Discussion on this subject is generally limited to horizontal forces since it has been shown that vertical forces are well tolerated. Necrotic changes have been described (13,74) in the periodontal membrane as a result of severe acute traumatic occlusion. A direct
application of these results to the problem under review is difficult to find. It is unlikely that such an obviously painful condition would be allowed to persist in human subjects (12, 45). It may occur temporarily following the placing of a restoration too high on the occlusal surface. Another factor, limiting such forces, is found in the control of muscle pressure by the proprioceptive nerve endings in the periodontal membrane (75). Gottlieb (75) considers that such forces are developed during nocturnal bruxism.

Such nerve destruction occurs more frequently as a result of periodontal disease (73). Here the loss of bony support increases the "extra-alveolar lever" arm and normal forces are greatly increased in effect. Similar changes have been demonstrated by Orban and Weimann (100), where the occlusal function was confined to a few remaining teeth.

Changes in the periodontal membrane, resulting from less severe occlusal stress have not been determined. Normally the result of light pressure is the movement of the tooth until a state of equilibrium is established (75). For such forces to become pathological the teeth must be prevented from moving. Two mechanisms, capable of stabilizing the tooth against such forces, are steep interlocking cusps and muscle pressure from tongue and lips.
The forces so developed have been described by Agnew and Gratzing (12) as a "moderate intermittent horizontal overstress".

Many years ago Dax (68) described the result of such occlusal dysfunction under the descriptive title of "rarefying pericementites fibrosa", a precursor state of periodontitis complex. Rainfjord (75) recently advanced a similar opinion when he stated that the degenerative changes associated with the clinical entity "periodontosis" were the result of such occlusal dysfunction. The present lack of knowledge of this problem makes a conclusion impossible.

(c) Gingival Changes.

Although many more or less specific changes have been attributed to occlusal dysfunction (13) there seems little evidence to support these claims. All that can be said at the moment is that this dysfunction may find expression in the gingiva as a reduced resistance to local irritants (75). Fish (101) and others (12) conclude that, in the absence of gingivitis, occlusion dysfunction cannot initiate the downgrowth of epithelium. However, once inflammatory changes do occur and spread to the areas of damaged or destroyed periodontal membrane there is a
rapid deepening of the pocket (12) resulting in the vertical type of periodontal disease – periodontitis complex (75). 

V. Systemic Factors.

It seems reasonable that all systemic disturbances, which affect the metabolism of the entire organism, influence the course of periodontal disease by altering the resistance and reparative ability of the tissues.

Some of the infinite variety of disturbances possible have been studied. Unfortunately, the difficulty of assessing chronic slight alteration has resulted in these studies being confined to severe acute deficiencies. These results are mainly not directly applicable to chronic periodontal disease. They do, however, indicate the way for future study.

(a) The Vitamins.

Vitamin A.

Studies of VIT A deficiencies appear to be confined to experimental animals (102). The changes reported include epithelial hyperplasia and atrophy of the salivary
glans. The bony changes are the subject of many conflicting reports (103). Boyle (103) suggests the debris of the epithelial hyperplasia may encourage bacterial growth. The associated xerostomia may act by changing the environment of the surface epithelium and reducing the resistance to infection. This deficiency has rarely been found in the human subject.

**Vitamin B Group.**

A similar distribution of the B group vitamins in foods makes a specific shortage of any one vitamin very rare (62).

The Vitamin B Group of deficiencies has been associated with gingival lesions in many studies, both clinical and experimental (56, 57, 61, 62, 81, 104, 105).

The commonest association appears to be with acute ulcerative gingivitis (Vincent's Infection). Successful treatment of the acute stage of this condition has been reported following therapy with Vitamin B2 (yeast extract) (61, 104) and niacinamide (57). Wainwright (62) reports a "destructive lesion not associated with a marked degree of inflammation" in pantothenic acid deficiency. The description appears very similar to the "gingiviasis" reported by Schour and Maser (61) which responded to specific therapy with pyridoxine and niacinamide.
The B Group of Vitamins is extremely complex. New substances are often isolated. Demonstration of changes associated with specific vitamins, while of value in increasing our knowledge of the subject, are not of such direct clinical importance, since their separate occurrence is rare. The important point is that it has been demonstrated clinically and experimentally that this group do influence the metabolism of the gingiva.

Vitamin C.

The bleeding gums of scurvy were one of the earliest recognized vitamin deficiencies. There are indications that many of these early conditions were the result of multiple deficiencies, including the then unknown B Group vitamins.

It is now fairly well established that Vitamin C has an important role in the differentiation of connective tissue cells. The influence of a deficiency of Vit. C on the supporting structures of the teeth is obvious. The experimental evidence supporting this view is well summarized by Glickman (106) from observations on guinea pigs.

1 Periodontal membrane and alveolar bone: Destructive changes resembling those described for periodontosis.
Gingival connective tissue; collagen degeneration and haemorrhage, the deficiency itself did not increase the incidence of marginal gingivitis.

Pocket formation is not initiated in the absence of complicating local factors. When pocket formation does occur there is greater destruction with Vitamin C deficiency.

Minghorne (107) supports these general conclusions on the results of an extensive study. He found that with low Vitamin C intake the subjects were more likely to develop a gingivitis. When a gingivitis had developed Vitamin C therapy alone was insufficient to cure the gingivitis.

The increased vascularity and haemorrhage reported in experimental studies has been demonstrated on clinical material by Orban (108) and indicated by the clinical descriptions of a curvy which emphasize swollen, cherry red gingiva which bled easily. (108).

Vitamin D

This vitamin controls the calcification of tissues. Deficiency results in the formation of increased thickness of cementoid with narrowing of the periodontal membrane (74). Rickets in early childhood may result in deformed dental arches and thus may be a predisposing factor in the
(a) Less Specific Dietary Effects.

Severe acute and chronic starvation obviously must influence the course of periodontal disease. This has been demonstrated experimentally by Glickman (109,110) and described clinically by Schour and Massler (61). Protein deficiency (111) and a relative protein deficiency with excess carbohydrates (61) have also been reported to play a part in the progressive periodontal lesion. Miller (13) considers that an "acid ash" diet is a similar predisposing factor.

(b) Endocrine Disturbances.

Endocrines are chemicals secreted within the body which control the metabolism of all cells. The exact nature of these hormones and their intricate interaction has not been fully determined but it seems reasonable that all endocrinopathies should alter the course of periodontal disease (113). Hypo-insulism (Diabetes mellitus).

Glickman (109) in experimental studies on rats reported a non-specific osteoporosis unrelated to gingival disturbances and noted an increased destruction from comparable trauma.
Other studies \((51, 113)\) were able to demonstrate no more than a tendency toward more severe changes in human diabetics, but could find no positive correlation between the gingival changes and the diabetic state. Changes associated with hyperparathyroidism and hypo- and hyperthyroidism have been reported \((17, 114)\).

**Hormones.**

Hormones are endocrines associated with reproduction and sexual characteristics.

Hormones were first related to changes in the gingiva when it was noted that chronic desquamative gingivitis was associated with the menopause.

Later it was demonstrated that the changed hormonal secretion associated with development of the ovum in the female caused specific changes in the vaginal epithelium. Ziskin \((55)\) was able to demonstrate that a similar, though not as marked, change occurred in the gingiva. Although it is not known exactly which hormones are involved \((115)\) it is possible to divide them into two large groups.

1. Oestrogens, associated with
   epithelial hyperplasia
   increased keratinization and
   increased resistance to infection.
5. Pregnancy group associated with increased vascularity and decreased resistance to infection.

"In most instances, however, the answer is not a clear cut excess or deficiency but rather an altered balance" (115).

The three main hormonal changes occur at puberty, pregnancy and the menopause. A tendency to gingival hyperplasia and gingivitis has often been reported to occur at puberty. (116).

There have been no studies to attempt to explain the mechanism of this change. Poor oral hygiene of the adolescent and mouth breathing are commonly associated aetiological factors. It is assumed that the hormonal change is the modifying factor. (115).

The term pregnancy "gingivitis" is frequently used. Ziskin (53) described five distinct clinical entities ranging from a slight colour change to the so-called "pregnancy tumour". Miller et al. (51,52) in extensive controlled studies were unable to find any such specific changes either clinically or histologically. They concluded that the hormonal disbalance played only a modifying part on the course of the gingivitis.
Intensive study of hormonal gingivosis (desquamative gingivitis) has established a much more definite picture of this entity and demonstrates a possible mechanism for these changes. The reduced production of oestrogen at the menopause was considered to cause the desquamation of the epithelium. The association of this hormone has been confirmed by successful specific therapy (64). Engel (115) using newer methods of histo-chemistry has been able to further explain the mechanisms of this change. He considers that the hormonal changes activate tissue enzymes which disrupt the intercellular material. The first change is loss of the basement membrane of the epithelium (66). This explains the desquamation of the epithelium.

A similar oestrogen withdrawal at pregnancy helps to explain the changes at that time. The changes at puberty are a different proposition, for at this time oestrogen production is beginning.
Summary

All systemic disturbances, that alter tissue metabolism, must influence the course of periodontal disease.

This relation has been demonstrated with regard to certain severe acute deficiencies of vitamins and probably with withdrawal of oestrogen at the menopause.

VI. Note on aetiology of acute ulcerative gingivitis (Vincent's infection).

The unsubstantiated theories and opinions, relating to the initiation of this acute phase of periodontal disease, are legion.

There are two possibilities:

1. Increased virulence of the organisms,
2. Decreased resistance of the tissues.

Box (55) supports the hypothesis of increased virulence of the organisms. This has been demonstrated to take place with other pathogens, never with the fusospirochaetal group.
A group of four organisms, endogenous anaerobes, have been demonstrated experimentally (77) as having the capacity to induce fusco-spirochaetal lesions.

The effectiveness of penicillin therapy indicates that bacteria play an active part in the disease (37).

There is evidence that decreased tissue resistance leads to the development of an acute exacerbation of gingivitis. This has been demonstrated with Vitamin B (57, 81, 104) and hormones (53). A reduced resistance of the tissues due to local irritants and occlusal dysfunction is another distinct possibility.

There remains, of course, the reasonable proposition that both these mechanisms are active at the one time.

Summary.

There is evidence that the acute phase of gingivitis is the result of tissue destruction by an endogenous group of anaerobic organisms. Reduced tissue resistance has been shown to play an active part in the lesion.

VII. Note on etiology of Periodontosis.

The usual understanding of this condition of periodontosis or diffuse alveolar atrophy is that systemic
disturbances are the primary etiological factors. The tissue changes seen are those of degeneration. Gluckman (73) criticises this persistent attempt to implicate a "systemic factor" in one type of periodontal disease and not in the other. We agree, for it has been repeatedly demonstrated that many systemic disturbances alter the progress of inflammatory periodontal disease due to local irritants.

We consider the more logical view is that recently advanced by Ramfjord (45).

The changes found histologically, according to this author, are those of the degenerative stages of inflammation resulting from slight occlusal overloading. (See earlier section for discussion). The role of occlusal dysfunction in this disturbance was emphasised by Box (117) many years ago.

The role of a systemic disturbance in this condition is apparent. Decreased cellular metabolism would soon convert a normal occlusal function to a pathological force. Ramfjord (45) has experimentally demonstrated this fact by inserting slightly high restorations, and so increasing the force of occlusion, in monkeys suffering from various debilitating conditions. A similar state of
disrupted tissue metabolism may be assumed in Gottlieb's original patients, all of whom died from severe disturbances.

Summary.

There is evidence that occlusal dysfunction is the initiating cause of the condition known as periodontosis. Interpretation of the histopathology of this lesion as showing the degenerative stages of inflammation supports this view.

VIII. Note on the etiology of Gingival Recession.

It is difficult to accept the concept of atrophy, as implied in the general clinical description "senile atrophy," as the etiological factor in this condition, in the absence of atrophy elsewhere in the body. (45).

Gottlieb (46) considers that the etiology is the same as periodontitis except that the free gingiva recedes at the same rate as the epithelium proliferates along the root. The constant presence of inflammation, histologically, supports this view.

The narrow vertical areas of gum recession, often termed Stillman's Clefts are localized expressions of the
some process. Originally they were attributed to occlusal trauma. The mechanism is probably a rapid displacement of the tooth resulting in complete resorption of the labial alveolar plate (75). This view is supported by Fish (34), who considers such recession is due to the loss of the labial alveolar plate.

Hirschfeld (118) considers that improper tooth brushing may cause such recession. This is a possibility, particularly if a tooth is out of alignment. In those cases one generally finds another sign of toothbrush abrasion in the form of grooves on the teeth.

Summary

There is no evidence that the etiology of "senile atrophy" differs in any way from that of periodontitis or gingivitis.
SECTION V.

DIAGNOSIS OF PERIODONTAL DISEASE.

Diagnosis.

The diagnosis of periodontal disease is essentially the recognition of the varying changes from normal. A knowledge of the possible etiology is essential if one is to interpret the clinical appearance correctly. We have discussed these features in previous sections.

A history of the patient's symptoms, local and general, and utilization of the laboratory aids of biopsy, blood examination and biochemistry are as necessary in the elucidation of these factors as in any other disturbance of the human body.

Prognosis.

The essential basis of prognosis must be:

(i) Can the tooth serve a useful functional purpose?

(ii) Will the patient be able to maintain the supporting tissues in a satisfactory form after the completion of treatment?
Though basically simple, answers to these two questions require consideration of many factors, not the least of which is the patient's ability and desire to carry out the procedures necessary to maintain the mouth free of irritants.

The infinite variations of the factors affecting the prognosis of any one dentition make the establishment of definite rules impossible. We will not, therefore, consider this matter further.
SECTION VI.

TREATMENT OF PERIODONTAL DISEASE.

The object of treatment of periodontal disease is to establish normal physiological processes within the periodontal tissues.

(a) Relation with other fields of dentistry.

Basically the local treatment of periodontal disease involves all aspects of dentistry. Restorations must adequately replace lost tooth structure with regard to contour, anatomy and high surface polish. Prosthetic appliances must not directly damage the gingiva nor encourage accumulation of irritants.

Oral surgery must be performed with due regard to the reformation of correct anatomy after healing. Orthodontics should aim at producing good functional occlusion and correct tooth alignment.

We do not propose to deal with these aspects in detail for, although it is the place of the periodontist to assume responsibility for the effects of these measures, it is not specifically within the scope of periodontia to complete these treatments.
(b) Removal of Local Irritants.

There is universal acceptance that the first principle in periodontal therapy is the removal of local irritants.

Many texts (5, 17, 18, 25, 33) stress this point. We do not propose to discuss instrumentation or armentarium for we feel, that providing the objectives of the therapy are sound, implementation is largely a matter of personal choice.

Beube (119) has experimentally demonstrated the effectiveness of these measures and this effectiveness is well supported by clinical observations.

Gottlieb (5), Ramfjord (120) and Hard (121) and many others indicate that this is all the treatment that is necessary in most types of gingivitis and many early conditions of periodontitis. All are agreed that such treatment is an essential pre-requisite of more extensive treatments.

Pressure packing with wax (33) and gauze strips soaked in zinc chloride (50) and many other measures have been reported as aids in the removal of those irritants.

The surface of the teeth should be left in as highly polished condition as possible; surface irregularities
favour the accumulation of debris.

The rough porous nature of exposed cementum has long been recognized as a major source of bacterial accumulation. Instructions to remove the roughened outer layer are universally given.

Riffle (122) has recently shown that this much desired smooth surface is not obtained unless all the cementum is removed. He suggests that a deliberate technique with this as an objective is indicated.

Summary:

The removal of local irritants is an essential treatment in periodontal therapy.

(c) Home Care.

The almost universal acceptance of non-detergent diets requiring little function necessitates the development of artificial aids to add to this function and remove food debris.

The toothbrush is the only method available, at the moment, which is suitable for the necessarily frequent use by the patient. This may be supplemented, as required, by wooden toothpicks and other methods of interproximal
cleansing in areas inaccessible to the toothbrush.

(c) Type of Brush.

Broadly speaking there are two types of brushes; those with short firm spaced bristles and those with large numbers of long soft bristles closely placed. The type of brush selected depends upon the technique of tooth-brushing prescribed.

The former group find application in the vibratory techniques of Charters and Stillman (18) which are such that the stiff bristles do not damage the gums.

The softer type brushes are used in the rotary technique of Fones and the physiological technique of Smith (99) and Bell (123). In these methods the bristles are swept over the teeth and gingiva.

We support the latter techniques. The criticism of the hard types of brushes is that the techniques required are more exacting and when abused they may become extremely destructive.

Formerly natural bristles were universally used (118). The development of fine diameters of nylon bristles has removed one of the early objections to this type of brush — excessive hardness. Nylon bristles, in addition to being more readily available, have certain advantages
such as standardization of texture, greater durability, and ease of cleaning.

(e) **Objectives of Tooth brushing**

(i) **Removal of debris.**

Clinical opinion has been supported by experimental demonstrations that the toothbrush is an effective method of removing food debris and bacterial plaques (123,124). The value of removing this debris is obvious.

(ii) **Gingival stimulation.**

The value of tooth brushing as a method of supplying artificial stimulation is in doubt. It seems reasonable to this writer that alternate compression and relaxation of the gingiva must influence vascular and lymphatic flow favourably. This must, to some extent, counteract the stagnation resulting from poor function. Though undemonstrated by experimental evidence this view is widely accepted (50,99) and supported by studies of the effects of massage on other body tissues (124).

Lyons (98) and Williams (99) and others do not completely accept this hypothesis. They contend that massage is only a poor substitute for good natural function. We accept natural function as an ideal and
an aim of periodontia. We do consider it expedient, however, to utilize artificial stimulation as a means of treatment in view of the many more problems involved in propagating the knowledge of functional diets.

Lyons (98) also opposes gingival massage on two other grounds. He considers it is contra-indicated to massage the gingiva while inflamed and that massage involves a risk of bacteraemia. These objections appear more theoretical than real. Lundquist (50), for example, considers that massage is essential to remove the "lymphatic block" and induce free flow of lymph. If one considers that a bacteraemia may occur with normal chewing Lyons' second objection appears less alarming.

(iii) Keratinization

A well keratinized gingival epithelium is considered one of the prime objects of therapy by many writers (18,34,125). Robinson and Kitchin (126) concluded that toothbrushing increased the keratinization of the gingiva but a better controlled experiment by Castenfelt (127) did not obtain such positive results. General clinical opinion seems to accept keratinization of the gingiva as a result of tooth brushing, despite these
inconclusive experimental results.

We are of the opinion that it is immaterial whether or not keratinization is the result of toothbrushing. If one considers that the initial lesion is of the sulcus epithelium, and that this epithelium is rarely if ever keratinized, there appears little reason to attempt to attain a keratinization of the gingival epithelium. Further, no amount of toothbrushing could affect the sulcus epithelium.

(iv) Reduction in pocket depth.

There is some experimental evidence (137) that toothbrushing will reduce pocket depth. This is useful in the elimination of shallow pockets. Its application in the treatment of deep pockets is limited. Irritants are likely to accumulate in the inaccessible parts of the pocket and initiate a further inflammatory reaction before normal architectural form could be obtained.

(v) Intercusal stimulation.

The use of wooden toothpicks and rubber points is based on the same principles as the toothbrush. They are a method of treating specific areas inaccessible to the toothbrush.
(f) Conservative surgery.

Ideally, conservative surgery aims at reforming the lost attachment of the tooth, thus requiring the formation of new bone, periodontal membrane and cementum.

Hard tissues heal elsewhere in the body so it seems reasonable that, providing organization of the granulation tissue is not disturbed, these changes are theoretically possible around the teeth.

Repair of hard tissues and periodontal membrane has been frequently noted in radiological surveys of completed apicectomies. Beube (128) has demonstrated similar results experimentally and obtained proof of the repair histologically. It is important to note that both these findings referred to a closed bony cavity unrelated to the gingival margin. Glickman (129) and Waerhaug (9) have demonstrated that there is some re-attachment following surgical detachment of normal tissues. The former observations were on dogs; the latter following the placing of full crowns on dogs and humans. These observations, again, bear little relation to the periodontal pocket.

The only experimental studies where attempts have been made to duplicate clinical conditions have been those of Ramfjord (130). He induced typical periodontal pockets
In Rhesus monkeys and was able to demonstrate some re-attachment after treatment.

There have been no conclusive clinical studies of this treatment. Early claims by Box (168) seem to confine results to those of an epithelial re-attachment. Other clinical successes have been based mainly on radiographic evidence. This is far from satisfactory. Beebe (131) demonstrated, histologically, a minor success with one tooth. Few studies have included measurement of the height of the gingival margin, before and after treatment, and thus do not reveal how much of the claimed success in pocket elimination is due to shrinkage of the tissues, following retraction of the healing fibrous tissue.

While theoretically re-attachment may occur, and has been demonstrated under certain conditions, examination of the requirements of the operative technique reveal that it is very difficult to accomplish.

All irritants and epithelium must be removed from the area of healing. The blood clot must be protected from oral fluids and movement, for at least fourteen days (27) to allow the tissues to become organized.

Removal of all the irritants is possible though extremely difficult. Flap and semi-flap operations (18)
have been devised to facilitate the removal of calculus from root surfaces.

The major problem appears to be the removal of all the epithelium to prevent a rapidly proliferating epithelium from intervening between the granulation tissue and the tooth surface. Ramfjord (130) experimentally demonstrated that caustic removal of pocket lining and extensive curetting was successful. Others (9, 59) show that this is almost impossible. Attempts to remove the epithelial attachment may only result in a deeper pocket. Even if it is possible to remove the pocket epithelium there still remains the problem of dealing with the remnants of Hartwig's sheath and the epithelium of the marginal gingiva.

The use of splints to stabilize the tooth to prevent disruption of the blood clot by movement (132) and packs to protect the clot from oral fluids have been developed (17). Whether these procedures are effective for the fourteen or more days necessary has never been demonstrated.
Summary

Theoretically re-attachment is possible. In the absence of a definite technique based on sound clinical evidence the practical application of this theory must be approached with caution. We consider the procedure an aim of improved therapy rather than an acceptable technique.

(g) Gingivectomy

"Gingivectomy is the surgical removal of excessive free gingival tissues to enable the patient to keep the teeth clean"—Ramfjord (120).

The technical simplicity of the operation and the definite and predictable results obtained has resulted in the wide acceptance of this procedure.

First techniques advocated were radical, in the extreme use of the word. Black (8) and Ward (133) advocated removal of a soft tissue followed by curetting of the granulation tissue from the bony surface. They considered that the granulation tissue was infected. Kronfeld (134), Fish (135) and many others demonstrated that there were no bacteria in this granulation tissue. The granulation tissue should be considered as reparative body function; the result of irritation rather than a
cause of destruction,

Techniques were modified and it is now generally accepted that removal of the soft tissue to eliminate the pocket is sufficient (136). Bone is only removed when treating intra-bony pockets and, rarely, on the palatal of the upper anteriors where the cortical bone of the palate has not resorbed (135).

Easily controlled surgical excision appears the method of choice; chemical and electrical cautery may destroy more tissue than required and thus have a limited application.

Orban (136) and others (137,138) have serially studied the progress of healing after a gingivectomy.

Orban and Archer (137) studied the healing without using a surgical pack. The most important conclusion they drew was that, to expedite healing and prevent formation of excessive gingival tissue, the free blood clot should be kept to a minimum.

After surgery the wound surface is generally protected by a medicated pack, basically zinc-oxide-eugenol. The value of this pack in reducing post-operative pain is obvious. In addition Bernier and Kaplan (138) have shown that healing progresses more rapidly under a pack. They showed that the epithelium covers the surface and collagen formation has begun after six days and after sixteen days
the epithelium appears mature. Although the same principles are involved, the term gingivo-plasty (17,139) has been used to designate the contouring of hyperplastic gingival tissues as distinct from true pocket elimination.

Summary.

The operation of gingivectomy is simple and effective. Post-operative packs are indicated to relieve pain and expedite epithelization.

(h) Occlusal Equilibration.

The object of occlusal equilibration is the establishment of physiological occlusion. Although a definite concept of physiological occlusion has not been established there are numerous indications that the following criteria are acceptable.

1. Distribution of the load over as many teeth as possible during all functional movements of the mandible.

2. Reduction in lateral stresses on the teeth.

It follows that the mandible will be free to move in both lateral and protrusive excursions.

These two principles are generally accepted but there is some difference of opinion as to further objectives of treatment. Schnyler (140) has developed a method of
spot grinding which, in addition to the above, has as its goal "the reduction of the essential force applied in the act of mastication." That is to say there is an attempt made to reduce vertical as well as tangential forces. The following principles are followed to obtain this reduction:

1. Retention of the sharpness of cutting cusps.
2. Increase food exits.
3. Decrease of contact surfaces.

On the contrary Williams (39) reasons that the processes of natural wear are the guiding steps in occlusal equibration. This technique follows the principles of even distribution of occlusal loading and the reduction of lateral stress, but the occlusal surfaces developed in this technique are increased in size and the cutting surfaces of the cusps are removed. This, if anything, would tend to increase the vertical loading.

In the light of present knowledge it is difficult to evaluate conclusively these opinions. In view of the evidence, discussed previously, that it is almost impossible to overload a tooth in a vertical direction and that modern diets require little function, we are inclined to consider the "natural wear" technique more favourably.
In those cases in which a severe degree of overbite exists, grinding can do little. Orthodontic treatment is the only satisfactory approach for the reduction of lateral stress. (141).

(1) The Uses of Drugs in Periodontal Therapy.

There are few, if any, drugs of specific value in the treatment of periodontal diseases (142). They must be considered as aids to treatment, not cures.

The use of pre-operative sedation, anaesthesia during operative procedures and post-operative analgesia requires little comment.

I. Caustics.

We can find little support for the use of caustics in periodontal therapy. It has long been recognised that tissue resistance and activities of the various body defence mechanisms are the major factors affecting the outcome of any infection. Medicine has long searched for substances capable of specifically attacking invading bacteria without damaging tissues of the body. These ideals have been realised recently with the introduction of anti-biotics.
It is not therefore, current practice to use, as anti-bacterial agents, substances which indiscriminately damage both bacteria and tissue cells. This principle is supported by the latest edition of Accepted Dental Remedies (143) and experimentally by Glickman and Johannessen (144) who specifically condemn the use of chronic acid.

Disclosing solutions, such as iodides, are of value in demonstrating the presence of bacterial plaques to both the periodontist and the patient.

Various measures have been adopted for the treatment of sensitive dentine. Concentrated zinc chloride, formaldehyde and sodium fluoride paste have been advocated. We consider indiscriminate application of these medicaments unwise since it has been frequently demonstrated that such substances applied to dentine may result in pulp death.

2. Oxygen Therapy

There is evidence that application of pure oxygen to the inflamed gingiva influences the rate of healing when associated with other therapeutic measures. Box (33) outlines the procedures advocated by Dunlop in which oxygen and certain medicaments are insufflated into the gingival pocket. The rationale of this therapy is that
anoxaemia of chronic inflammation is improved and that active tissue metabolism re-commences.

Orban (145) and Kaiser (146) have obtained results indicating that the nascent oxygen of 30% hydrogen peroxide and urea hydrogen peroxide respectively do expedite tissue healing.

3. **Antibiotics.**

Since first introduced the antibiotics have been frequently advocated for periodontal therapy with particular attention being paid to the acute phase of gingivitis. *(Vincent's Infection).*

Successful treatment of this condition has been reported (54, 147) and others. We agree with Lyons (148) who considers that none of the many claims were based on well controlled experiments and none allowed for the fact that the acutely painful stage of this condition often disappears spontaneously. Problems of culturing the organisms make it very difficult to assess their susceptibility to the penicillin or other anti-biotic.

In addition to the weak clinical and experimental bases for this therapy there are three other factors to consider:
(i) Possible local reaction to the anti-biotic.

(ii) Possible sensitization of the patient to the anti-biotic concerned.

(iii) Possible production of resistant strains of organisms.

The first two considerations are likely to be more embarrassing than dangerous. Production of resistant strains is a definite possibility and in view of the success of other treatment we consider that persistent use of anti-biotics is contra-indicated.

Specific indication for systemic use of anti-biotics is found in the condition of valvular heart disease following rheumatic fever; the damaging bacteraemia incident to operative interference is thus controlled. To conclude "...*...*...*..." the introduction of this drug group has brought forth greater enthusiasm and more extensive use than the facts justify" (Ostrander) (149).

(j) Systemic Therapy

There are two aspects of systemic treatment of periodontal disease.

Specific: where clinically obvious and bio-chemically demonstrated deficiencies or disturbances exist. Non-specific in dealing with less spectacular changes of the
little understood sub-clinical deficiencies. In this latter field we are confined to prescribing diets which adequately fulfill all the known requirements of the various nutrients.

(i) **Vitamins.**

Treatment with specific vitamins results in a ready response. This has been successfully demonstrated clinically and experimentally many times.

The Vit. B. Group have been successfully applied in acute ulcerative gingivitis (Vincent's Infection) (57, 81) and the degenerative gingivosis described by Schour and Massler (61). Success with Vit. C therapy in scurvy has been reported frequently.

(ii) **Endocrines.**

Surgery and medication appear effective methods for controlling the endocrine disturbances of hyper-parathyroidism, hyper- and hypothyroidism and hypo-insulism.

The endocrines of the adrenal cortex have been tentatively applied to gingival lesions but there is insufficient evidence of any value as yet. (47).

(iii) **Hormones.**

Recognition that oestrogen withdrawal is probably responsible for certain gingival lesions resulted in
attempts at therapy with this substance. Ziskin (64,65) reported some success with local application of oestrogen in chronic desquamative gingivitis. This local therapy resulted in systemic absorption and, bearing in mind, the carcinogenic properties of these hormones, this therapy must be approached with caution. (115)

It is convenient to note here that the numerous empiric treatments of this condition were not successful (63,73); even the exponents admitted this.

(k) Dentifrices.

These substances have rarely been examined specifically as they influence the progress of periodontal disease. As aids in cleansing the oral cavity and polishing tooth surfaces they have their place.

An attempt has been made to include a mucinase in a dentifrice; the results reported were far from conclusive. (114).

(1) Mouth Washes.

It seems reasonable that vigorous use of a mouth wash would remove debris loosened by tooth-brushing (150).

The value of medicaments in the mouthwashes is unproven, and indiscriminate use may be detrimental. In the section on mouth washes in Accepted Dental Remedies there are examples, such as tooth decalcification by
persistent use of hydrogen peroxide, which contra-indicate
many of the most favoured additions.

Everott and Bettman (150) sum up the situation
fairly accurately when they conclude their recent paper
with the following:

"In the absence of any confirmation of value
of any addition to the mouthwash, it is probable that
warm water is all that is required".

(m) Treatment of Acute Phase of Gingivitis
(Vincent's Infection)

The acute phase of this condition has been success-
fully treated by countless local medicaments as well as
systemic administration of arsenenamine, vitamins, hormones
and anti-biotics. Such multiplicity of treatments
indicates that either the aetiology of the disease is
unknown or the assessment of successful treatment is
uncritical.

Lyons (148) in a critical survey of the treatment of
this condition emphasises the lack of controlled experiment
in assessing results of therapy. He presents the opinion
that the criterion of success is whether or not interprox-
imal destruction is limited by the treatment.

There are three main methods of treatment, Symptomatic,
anti-biotic and oral hygiene. The application of
substances such as chromic acid (34), Churchill's iodine and silver nitrate (33) and others depend for their action on coagulation of the bacteria and tanning of the surface of the ulcer to relieve pain. We have discussed the reasons in a previous section why we do not consider these methods acceptable.

The use of anti-biotics is more logical but we believe that they should be reserved for extremely severe conditions. We have also discussed this in a previous section.

The third group advocate immediate prophylaxis followed by measures to maintain oral cleanliness (17,73,148, 151). If one considers the activity of anaerobic organisms as an essential part of this disease then treatment to remove the anaerobic conditions engendered by accumulated debris seems logical. Fears that infection may be spread are not borne out in practice. In fact the very strict anaerobic nature of the organisms involved makes such fears baseless.

Further support for this treatment is offered by Uhn (152) who found, in a comparative study, that it was equally effective as any depending on medication. Immediate oral prophylaxis has the distinct advantage that treatment of the underlying gingival changes is actively
(100)

become much earlier.

(II) Prevention of Periodontal Disease.

There is evidence that there are two methods of preventing the loss of teeth as a result of periodontal disease.

Baregaard (14) and Price (55) and many others have shown that natural functional diets with an adequate supply of nutrients are consistent with good dentitions. These diets aid the development of normal mouths by supplying function as well as materials to maintain the supporting tissues in an optimum metabolic state. Periodontists should propagate the knowledge of these diets not only for the part they play in oral health but in the health of the whole organism.

At the present moment the implementation of this ideal seems more difficult than the teaching of artificial methods of cleansing and stimulation. The success of these methods has been proven experimentally and clinically. Failure to apply these methods during childhood, when gingival destruction begins (153) leads to extensive destruction of the natural form of the tooth and supporting structures. Measures to prevent the loss of teeth in later years are of an heroic nature (154). "The complicated techniques required to tighten hopelessly involved tooth" are the cause of much of the hopeless attitude to
SECTION VII.

ASSOCIATED ORAL CONDITIONS

There are several common conditions in which oral manifestations are of importance. Strictly speaking, they do not come under the heading of periodontal disease. These changes, however, are of importance in the differential diagnosis of certain stages of the periodontal lesion.

(a) Acute Disturbances.

(1) Acute Herpetic Stomatitis (155, 156)

The primary attack, in childhood, of the herpes simplex virus is an acute ulcerative stomatitis which must be differentiated from acute ulcerative gingivitis (Vincent's Infection).

Important points of differentiation are possible. General malaise, temperature and glandular enlargement precede the development of the oral lesion. At this time small vesicles may be found on the mucosa demonstrating the activity of the herpes simplex virus. These vesicles
break down and secondary infection of the ulceration ensues. The ulcers, which may be found anywhere on the oral mucosa, are typically described as punched out and surrounded by an inflammatory halo. The gingiva are swollen, red and haemorrhagic. Salivation is excessive.

Bacteriologically there is no typical change in the oral flora; corneal inoculation of rabbits may demonstrate the viral origin of the condition (153).

3. Acute Staphylococcal and Acute Streptococcal Gingivitis (74) — may be differentiated by the situation of the ulceration, rare with streptococcal infections, not typically on the gingival margins. The margins of ulceration are less clear cut than the acute herpetic stomatitis.

The bacteriological picture is less definite; fusospirochaetal organisms may be present but they are not a predominant feature.

3. Acute Catarrhal Gingivitis (58, 174) is usually associated with upper respiratory tract infection. The gingiva are swollen, glazed and hyperaemic. There is no ulceration and the condition rapidly subsides.
(b) *Gingival Hyperplasia.*

(1) **Leukaemia.**

Many of the blood dyscrasias of the white cells are manifest as a gingival enlargement. This applies particularly to the acute stages of monocytic and myelogenous leukaemias (74).

A typical blood picture and demonstration of the immature leukocyte forms within the gingiva are diagnostic features (157, 158).

It is important to note that a typical acute ulcerative gingivitis (Vincent's Infection) may develop during the course of this disease and should be borne in mind if the acute oral symptoms do not react favourably to local therapy.

(2) **Dilantin.**

A typical hyperplasia of the gingiva occurs in some patients undergoing systemic treatment of epilepsy with the drug dilantin. (74, 159, 160, 161).

The interdental papillae are involved first. There is an increased stippling, followed by a firm warty enlargement. The distribution varies but may be more apparent in areas of irritation. Histologically there is hyperplasia of the epithelium, increase in the epithelial pegs and massive increase in the fibrous
bundles of the connective tissue. Inflammation is not a dominant feature (17).

(s) Idiopathic Fibrous Hyperplasia.

Fibrous enlargement of the gingiva has been frequently reported (74, 160). Although the etiology is unknown, the term "gingivoma" has been used designating that the enlargement is in the nature of a new growth.

The enlargement is dense and firm and is often so extensive that the teeth are invisible. Signs of inflammation are notably absent; the patient only complains of enlargement. That the condition is essentially associated with the gingiva is demonstrated by the disappearance of the enlargement when the teeth are removed.
Bibliography


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Abbreviations

B.D.J. British Dental Journal.
O.S., O.M., O.P. Oral Surgery, Oral Medicine, and Oral Pathology.