SOME OBSERVATIONS

ON THE

PATHOGENESIS OF GINGIVITIS.
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by

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PREFACE.

In the past, findings which have been of immense value to dental science have not been luckily stumbled upon as one might discover a nugget of alluvial gold in a useful state, and of immediate value, but they have been the end result of countless carefully planned experiments, each serving a purpose and acting as a foundation for greater discoveries. There are times, however, when one has a lucky find, but usually the "luck" is proportional to the skill and labour previously expended, and occurs according to the law of average.

It is hoped that this investigation will prove to be at least a stepping-stone for more valuable work to follow.

The aim of the thesis has been to elucidate some of the problems confronting the dental profession concerning the pathogenesis of gingivitis.

The biological laws governing the initiation of such a disease as gingivitis are extremely complex and multifarious, requiring the co-operation, help, and advice of others who specialise in various branches of science.

Accordingly my thanks are due to the staff of the Dental Hospital, Sydney, and especially the technicians of the Research Department of the above institution, whose untiring efforts to perfect new technical requirements have been very encouraging.
I wish to thank those members of the University staff who have tendered me advice and particularly Professor Harvey-Sutton and his staff at the School of Public Health and Tropical Medicine who have co-operated with me in some of this work.

I would here intimate that I would never have been able to carry out these investigations without the financial aid of the Australian Dental Association (N. S. W. Branch) and the Walter and Eliza Hall Trust, whose aim has been to further Dental Research, and so to help relieve humanity of such burdens as gingivitis.

Finally, I wish to express my gratitude to Professor A. J. Arnott, Dean of the Faculty of Dentistry, Sydney University, whose encouragement and unceasing help in all matters of research has been most inspiring.

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SECTION NO. 1

INTRODUCTION.

DEFINITION.

HISTORY.
INTRODUCTION.

Health, the necessary factor for the maintenance of life, is the ideal one hopes to attain and maintain. It is an intangible phenomenon which is under our control but which may be lost through neglect or misadventure, and once lost is hard to regain. Sound teeth and healthy supporting structures aid immensely the health of the whole body.

A great number of pathogenic micro-organisms, which cause systemic infections, gain entrance to the body through the mouth, and their continued presence is rendered more frequent by unhealthy oral tissues. The mucous membrane of the mouth is very resistant to germ life, but its junction with the teeth at times offers a weak point which unfortunately readily breaks down.

Gingivitis with its accompanying infection and probable consequent body ailments, is a menace to the health of our people. For instance, apart from causing inefficiency in mastication and general metabolic changes, acute gingivitis may result in a secondary infection of the throat and lungs, especially if a general anaesthetic be given for some other condition. An unhealthy condition of the gingival trough, even if it does not cause pain or act as a focus of infection, prevents the full and proper function of the teeth in mastication and nullifies the hygienic action of the tongue and cheeks. Insufficient
mastication leads to a train of digestive troubles which in turn reflect on the general health in an undesirable manner.

That we have drifted from the normal, as a result of our civilized habits or for other reasons, is sure, but where the fault lies is not so certain. Once the fault is known we can adjust our lives accordingly and prevent the ill effects from which we now suffer.

It is realised that if the cause of a disease be known, a method of treatment is more likely to suggest itself which may result in a permanent cure.

To know the aetiology of a disease is the greatest aid to its eradication and paves the way for its prevention. The symptomatic treatment of the various other manifestations of gingivitis is generally only a matter of the application of accepted common therapeutic measures.

With a view to supplementing our knowledge on the pathogenesis of gingivitis, the writer has undertaken this investigation.

The study of gingivitis is extremely complex and it would be impossible to adequately deal with all the phases of the disease in a work of this nature, but if some further light can be thrown on its pathogeny the undertaking will not have been in vain.

This presentation deals mainly with the most common type of gingivitis, which has been termed chronic Vincent's infection, and which occurs in over 90% of
gingivitis cases.

Gingivitis in most of its forms is but a stepping stone to suppurative periodontitis (pyorrhoea).

The change is a slow but progressive one. The two affections are intimately associated and may be considered as the primary and secondary manifestations of the same disease.

Observations from private and hospital practice covering a period of many years and the conclusions drawn after a review of the literature, all point to the fact that there is no one single direct or predisposing cause of gingivitis.

Because of its complexity, its dependence on many biological laws and as yet unknown phenomena, the problem of the aetiology of gingivitis, like that of caries, is a difficult one to elucidate.

Dental caries, occurring as it does in a hard structure of the body, can be arrested and the teeth restored to their original shape by mechanical restorative means, such as metallic fillings.

Gum diseases on the other hand do not lend themselves to such treatment; indeed up to the present there is no evidence to show that gum tissue once lost has been restored to its normal shape. The writer does not mean to insinuate, however, that diseased conditions of gums cannot be arrested or cured, nor that it would be an impossibility to restore the gums to their normal shape,
including at the same time a good attachment to tooth and bone. Gum restoration if achievable will have to be done by grafting. A cure merely implies a restoration to health. It is, therefore, quite feasible to expect a cure of gingivitis or pyorrhea if treated properly. In fact a cure is often seen.

Besides dealing with other causes, it is hoped that this work will show the part which diet plays in the aetiology of gingivitis.

Any treatment of the common gingival diseases may be beneficial to a greater or lesser extent, but it will never completely make good the harm wrought by dietetic errors; the correction of which in each individual case is of paramount importance.
DEFINITION.

Gingivitis is derived from the Latin word "gingiva", meaning "the gum", and the Greek "itis" denoting inflammation.

The term includes any inflammation occurring in the soft tissues about the necks of the teeth resulting in either atrophic or hypertrophic degeneration, and in consequent loss of the tooth supporting alveolus. It is intimately associated with acute Vincent's infection and Complex Periodontitis (pyorrhoea).

It may be an idiopathic entity in which severe bacterial infections damage the gingival tissues, or a symptom of constitutional disturbances such as are commonly described as being associated with scurvy, agranulocytosis or leukemia. It may also be a symptom of poisoning or over medication with such drugs as mercury or bismuth. Gingivitis may occur in an acute form as acute Vincent's infection or in a chronic form when it is usually the forerunner of so called pyorrhoea.

Acute Vincent's infection is a contagious disease characterised by inflammation of the gingivae resulting in necrosis of the surface epithelium and ulceration of the sub-epithelial tissues. Necrosis, however, is not seen in chronic Vincent's infection.

Gingivitis is called by some writers paradentitis, which denotes an inflammatory process of the tissues
round the tooth, to distinguish it from paradentosis, a condition symbolised by deep vertical pockets which have been brought about by supposed non-inflammatory degenerative changes. Stillman and McCall call all forms of gingivitis, except acute Vincent's infection, Simplex Periodontitis or Horizontal Pyorrhea, and call the condition with vertical pockets Complex Periodontitis. The writer has used this commonly accepted classification in this work, but has included acute Vincent's infection under the heading of Simplex Periodontitis.
HISTORY.

Gilmer (1) 1906, was one of the first to describe Vincent's infection. He (2) 1928, says "It was forty-three years ago that I first saw a case of the disease which I named acute ulcerous gingivitis. I saw very few patients suffering from it until the return of our soldiers from Europe after the close of the World War, when it was widely disseminated in this country."

Nevertheless, it is likely that Vincent's infection is an age old disease. Hardgrove (3) 1928, states that the infection has passed unnoticed for many years being regarded as a symptom of syphilis, tuberculosis, scurvy or other diseases.

Hippocrates referred to aphthous affections of a malignant nature that often developed early in the Spring. This is suggestive of the infection, the seasonal aspect of which will be discussed later.

Topley and Wilson (4) 1931, state that Van Leewen-


hock who was the first man to actually see micro-organisms, in the year 1683 observed, drew, and measured, large numbers of living organisms including bacterial and protozoal forms. It is likely that the first microorganisms he saw were the fuso-spirochaetal ones which accompany gingivitis, since he obtained his material from round the gum margins.

Pyorrhea is considered to be an age old disease because of its many manifestations shown in very ancient skulls.

Since a chronic Vincent's infection (common gingivitis) is probably always the forerunner of pyorrhea, it must be assumed that it is as old a disease as pyorrhea.
SECTION NO. 2

HISTOLOGY.
HISTORY

So that a proper understanding of the pathology of gingivitis may be gained it is advisable first to study the normal histology of the gums, its surface and deep structures, its nutritional supply, and its attachment to the teeth.

As the gum is organically attached either to the enamel or to the cementum or both at the one time, a brief study of the histological structure of enamel and cementum will be dealt with as well as the embryology of the developing tooth to show how the connection of the gum to the tooth originates. Further since the periodontal membrane and the alveolus are affected by gingivitis a short description of these structures will be given.

Box (1) states that the part of the oral mucous membrane called the gingivae, constitutes all the soft tissues about the necks of the teeth, down to the level of the crest of the alveolus.

The marginal gingiva is that part of the gingiva above the level of the deep end of the epithelial attachment; and the remainder of the gingiva between the levels of the deep end of the gingival attachment and the crest of the alveolus is called the cemental gingiva.

Fig. 1 Diagram illustrating the anatomical structures of the gingiva: A, Free Margin or Crest of Gingiva; B, Gingival Crevice or Trough; C, Crevicular Epithelium; D, Bottom of Gingival Crevise; E, Deep end of Epithelial Attachment; F, Crest of Alveolus.

The crest of the gingiva where the crevicular epithelium joins the oral mucous membrane epithelium is called the free gingival margin or the gingival crest.

The gingival crevice (or trough) is the space between the enamel cuticle and the gingival epithelium, the deepest part of the space being the bottom of the crevice. The epithelium bordering the gingival crevice is called the crevicular epithelium.

Those epithelial tissues adhering to the surface of the tooth (from D to E, Fig. 1) and forming a band of attachment around it, constitute the epithelial attachment.
Orban (2) 1929, gives a good description of dental histology and embryology relevant to this investigation and much information has been gathered from his work. Mention must be made of Gottlieb who in 1921 first showed the organic attachment of the gingivae to the teeth.

THE MUCOUS MEMBRANE.

The mucous membrane of the gingivae and mouth acts as a protective covering for the deeper structures such as the alveolus and periodontal membrane. It consists of several layers, the outermost covering the gingiva (3) being a keratinous layer called the stratum corneum. Beneath this is a thin layer, the stratum lucidum and then another thin layer, the stratum granulosum. Beneath these thin layers is the stratum germinativum which is relatively thick and forms the bulk of the epithelium. At the base of the stratum germinativum the cells are more cylindrical in shape and form the stratum cylindricum. Finally beneath the epithelium lies the corium which contains the nutritional supply for the squamous epithelium.

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The corium is composed of connective tissue which carries blood vessels, lymphatics, and mucous glands; and it forms papillae which run into the epithelial layer. Each papilla contains one or two capillary loops which adequately supply the epithelium with nourishment. The nourishment is conveyed between the epithelial cells by the lymph exudate from the blood vessels and is absorbed into the cells. The blood supply to the gingivae comes from the periodontal, periosteal, and the adjoining mucous membrane, blood vessels.

Because of the loose connective tissue forming the corium, the outer epithelial layers of the oral mucous membrane with the exception of the gingival mucous membrane can move freely in any direction over the deeper structures such as bone or muscle.

The gingival mucous membrane is firmly attached to the alveolus and periodontal membrane, being rendered immovable by many interlacing strands of fibrous tissue in the corium.

It will be shown below how the epithelium of the mucous membrane is attached to the tooth through the "epithelial attachment" of the gingiva.
Fig. 2. Gingival Mucous Membrane; S.C., Stratum Corneum; S.L., Stratum Lucidum; S.Gr., Stratum Granulosum; S. Ge., Stratum Germinativum; S. Cy., Stratum Cylindricum; C., Corium; B.V., Capillary Blood Vessel supplying a Papilla of the Corium.

ASPECTS OF ENAMEL DEVELOPMENT
AND OF THE ERUPTION OF THE TEETH,
WHICH CONCERN THE EPITHELIAL ATTACHMENT.

The first sign of tooth development is demonstrated by a thickening of a small band of oral epithelium. Later there is an apparent downgrowth of the epithelium and an organisation of the nearby connective tissue corresponding to each tooth.

The enamel is one of the first parts of a tooth to be formed and it owes its origin to the enamel forming organ which is derived from the epithelium A, Fig. 3.
Fig. 3  Toothgerm. A, Apparent Downgrowth of Epithelium; B, Organisation of Connective Tissue.

Fig. 4  Showing A, Oral Cavity; B, Dental Lamina; C, Outer Enamel Epithelium; D, Stellate Reticulum; E, Stratum Intermedium and Ameloblasts; F, Dental Papilla; G, Developing Bone.
Fig. 5  Enamel Forming Organ. A, Outer Enamel Epithelium; B, Stellate Reticulum; C, Stratum Intermediate; D, Ameloblasts; E, Odontoblasts; F, Dental Papilla.

The ameloblasts are long columnar enamel forming cells, each one of which forms an enamel prism.

Fig. 6  Ameloblasts A, torn away from the Enamel B; C, Terminal Bars; D, Tomes Processes; E, Stratum Intermedium.
When the enamel formation is complete, the constituents of the enamel forming organ, consisting of ameloblasts, stratum intermedium and the outer enamel epithelium (the stellate reticulum having by this time become part of the outer enamel epithelium), unite together.

![Diagram](image.png)

**Fig. 7** The Enamel Forming Organ reducing in size A, as the Enamel increases in thickness B.; C, Dentine.

The ameloblasts lose their columnar shape, change into a cuboidal form, and become part of a single more or less homogeneous stratum of cells, consisting of the remains of the enamel organ.
Fig. 8  Partly Reduced Enamel Organ. A, External Enamel Epithelium merging towards the Stratum Intermedium B; C, Stellate Reticulum; D, Ameloblasts becoming cuboidal in form; E, Enamel; F, Dentine pulled away from the Enamel.

The last product of the ameloblasts is a thin enamel layer or cuticle called Nasmyth's membrane, or the primary enamel cuticle, which although only about one micron thick, serves as an organic connection between the cells which deposit it and the enamel.

This enamel cuticle is called the primary enamel cuticle by Gottlieb to distinguish it from the secondary enamel cuticle which is formed later at the time of the eruption on the tooth. The secondary enamel cuticle is considered to be a keratinous layer adherant to the enamel and deposited by the epithelial attachment; and because it extends over the cementum when the epithelium gains attachment there, it is also called the dental...
cuticle or cuticula dentis.

The reduced enamel epithelium consists of squamous epithelium similar to the epithelium of the mucous membrane of the mouth. When, during eruption of the tooth, the reduced enamel epithelium comes in contact with the mucous membrane epithelium of the mouth, it unites with the latter, and then becomes an extended part of it and is called the epithelial attachment.

Fig. 9 Tooth of Rat showing the Epithelial Attachment E.A., in organic connection to the Enamel E; D, Dentine.

The epithelial attachment which is actually a band of epithelium organically attached to the enamel at first, does not remain in the same position for any length of time, but extends in the direction of the root apex and becomes again organically attached to the cementum. Approximately, at the same time and rate as it loses attachment from the direction of the occlusal surface, it
gains attachment along the root, so that the width of the band of attachment is fairly constant.

The progression of the epithelial attachment from the enamel to the cementum may be illustrated by Gottlieb's descriptions of the four stages of tooth eruption. The first stage is where the erupting tooth comes in contact with its antagonists of the other jaw, when the lower third of the anatomical crown is not erupted, and the epithelial attachment ends at the cemento-enamel junction.

Fig. 10  Diagrams illustrating four stages of the relationship of the epithelial attachment to the tooth, and representing the four stages of tooth eruption (Gottlieb).  En., Enamel;  D., Dentine;  C., Cementum;  C.E.J., Cemento-Enamel Junction;  Ep., Epithelium of Gingiva.

The second stage of eruption is marked by the downward growth of the epithelial attachment beyond the cemento-enamel junction, so that the attachment is upon both enamel and cementum. In the third stage the bottom of the gingival crevice is at the cemento-enamel junction, and the epithelial attachment is entirely upon the cementum. The fourth stage is similar to the third stage
except that cementum is exposed to the saliva.

Fig. 11 Epithelial Attachment to tooth corresponding to the first stage of eruption. D, Dentine; E.S., Enamel Space (the enamel has been lost through decalcification of specimen); E.A., Epithelial Attachment; S.E.C., Secondary Enamel Cuticle; A, Alveolus.
Fig. 12  Epithelial Attachment E.A., to the Cementum C., in the fourth stage of eruption when Cementum C', is exposed to the saliva; D, Dentine.

The manner in which the epithelial attachment extends on to and along the cementum has never been satisfactorily explained. It has been considered previously to just grow down upon and adhere to the cementum by means of a hornified layer of the epithelium. Later, after detachment of the epithelium from the tooth, this so called hornified layer is considered to be left attached to the tooth and is then called the dental cuticle. However, Weski, Meyer, and Lund (4) have not been able to obtain the staining colour characteristics for horn when staining the cuticula dentis. If the dental cuticle were a hornified layer produced by the epithelium it

would be represented by a continuous layer covering the enamel and the cementum, but in photomicrographs illustrating the dental cuticle (5) and (6), a junction is always noticed opposite the cemento-enamel junction which suggests that the cuticle is not only divided into two parts, but because of the different thickness and density, each is of different origin; the one adhering to the enamel is probably the reduced ameloblastic layer, while the other adhering to the cementum is probably the reduced cementoblastic layer.

The writer has sought the reason for the firm epithelial attachment because:

(a) It would constitute an anomaly if the mucous membrane epithelium could attach itself to cementum, a bone-like structure, without some intermediary connective tissue cementing substance to afford an organic attachment and

(b) A knowledge of its attachment would greatly influence and suggest logical methods for the treatment of gingivitis and complex periodontitis.

Accordingly a study of continuously growing teeth, such as are seen in certain animals, and which would

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show a rapid continuous downgrowth of the epithelial attachment, was made. From these suggestive observations an analogous, but much slower process was observed in human specimens.

NEW THEORY OF THE EPITHELIAL ATTACHMENT TO THE CEMENTUM.

Finger-like processes from the epithelial attachment grow down into the corium and cover the cementum end of the periodontal membrane fibres which slant upwards (in the lower jaw) from the alveolar crest to the neck of the tooth. The periodontal membrane fibres and cementoblasts thus invaginated become reduced until the epithelial projection reaches and unites with the cementoblasts which subsequently have their function changed from a parenchymal to a connective tissue one. The cementoblasts are already attached to the cementum by their recently deposited collagenous cemental fibres.

The reduced cementum-forming cells then act as the intermediary cementing substance between the epithelial attachment and the cementum, just as the reduced enamel epithelium and the primary enamel cuticle act as the connecting tissues between the gingival epithelium and the enamel.
Fig. 13  Epithelial Attachment to Tooth of Guinea-Pig. E.A., Epithelial Attachment; P.E., Finger-like Process of Epithelium extending over the Periodontal fibres P.; C, Cementum; D, Dentine; A, Alveolus.

Fig. 14  Epithelial Attachment E.A., torn away from Cementum C, in the process of section making; P.E., Prolongation of Epithelium over the Periodontal Membrane fibres P.; A, Alveolus.
Fig. 15  Epithelial Attachment extension Process P.E., covering the Periodontal Membrane P.; D, Dentine; G.E., Gingival Epithelium; A, Alveolus.

Fig. 16  Epithelial Attachment E.A., partly torn away from the Cementum C, showing a recently deposited collagenous cemental fibre F, connecting the Epithelium to the Cementum.
Fig. 17 Human Tooth and Gingiva. Due to a technical fault the Epithelial Attachment E.A., is torn away from the Cementum, C. The Epithelial Extension Process, P.E., has apparently become attached to the Cementum C at C'.

In normal cases there is seldom any clinical crevice or pocket between the free margin of the gum and the tooth. Skillen (7) 1931, in a study of human material found that "The gingivae in some cases had crossed the enamel cemental junction without the development of a pocket or of any appreciable pathology."

Microscopically a crevice from 0.0 to 1.0 millimetre is seen in normal healthy gingivae.

CEMENTUM.

The cementum is a bone-like structure covering the root of a tooth and affords anchorage for the periodontal membrane fibres which connect the tooth to the alveolar bone. It is laid down in lamellae which run parallel to the surface of the root.

Cementum differs from bone in that its collagenous fibres run at right angles to its surface and the lamellae, whereas in bone the fibres run parallel to the lamellae.

There are two kinds of cementum:—

(1) Primary cementum.
(2) Secondary cementum.

(1) Primary cementum is built by the cementoblasts and does not contain any embedded cells in its matrix, but encloses the ends of the fibres of the periodontal membrane. It covers all but the apical third of the root, and is often itself covered by secondary cementum.

(2) Secondary cementum resembles the structure of bone more closely than primary cementum. It contains in its matrix, embedded cement corpuscles and the ends of the fibres of the periodontal membrane.
THE PERIODONTAL MEMBRANE.

The periodontal membrane consists of collagenous connective tissue fibres which connect the tooth to its alveolar bone, its neighbouring tooth or teeth, if present, and to the gingiva. It is about a quarter of a millimetre in thickness, but this average measurement varies a good deal. By means of the cementoblasts and osteoblasts which are contained in the periodontal membrane, fresh cementum and alveolar bone are continually being deposited and needed for the attachment (8) and even the existence of the periodontal fibres themselves which are constantly being renewed.

The periodontal membrane derives its blood supply principally from the adjoining medullary spaces of the alveolar bone, but the blood vessels to the pulp, branch at the tooth apex and supply it to some extent.

Fig. 18  Periodontal Membrane P., of Guinea-pig; Principal Fibres P.F., entering Bundle Bone B; Indefinite Fibres I.F., supporting blood vessels and nerves; C, Cementum.

Fig. 19  Osteoclasts, O, resorbing Alveolus, A, prior to the deposition of bone for the anchorage of new fibres in the periodontal membrane, P.
Alveolar bone is bone which is deposited by the osteoblasts next to the periodontal membrane and is itself supported by other "supporting bone."

There are two types of bone in the jaw—lamellated and bundle bone.

Lamellated bone is made up of collagenous fibres forming lamellae which are embedded in a calcified cementing substance. The lamellae for the most part either lie parallel to the bone surface or are concentrically arranged around Haversian canals. Connective tissue cells (osteoblasts) which deposit bone are included into the bone substance and are called bone corpuscles. The latter lie in lacunae or cell spaces in the bone.

Bundle bone (9) is similar to lamellated bone, into which bundles of fibres become embedded. The periodontal membrane gains its firm attachment by bundles of its fibres entering such bone.

SECTION 3.

AETIOLOGY.
AETIOLOGY.

The histology of the periodontal tissues have been dealt with to help in the explanation of some of the aetiological factors.

Usually in cases of gingivitis, an immediate cause, such as an invasion by micro-organisms, appears evident. Upon careful investigation, however, it is found to be but a secondary aetiological factor, since the organisms are present at times in all mouths, and may be conveyed from one individual to another, perhaps by droplets of saliva during conversation, yet not give rise to any clinical symptoms of gingivitis.

The aetiology of gingivitis may be divided into two classes:--

A. Systemic Factors.

B. Local Factors.

A. It is not definitely known how systemic factors influence the gingivae but such conditions as blood dyscrasias, scurvy, and diabetes, are generally considered to affect the gingivae deleteriously.

B. The following is a classification of some local aetiological factors causing gingivitis:--

1. Mechanical irritants and local abnormalities.
2. Disuse.
3. Trauma.
4. Infections.
Fig. 20  Inflamed gingiva about an ill-fitting gold crown.

Fig. 21  Case showing a gingivitis beneath an ill-fitting bridge pontic. The upper left lateral incisor is a pontic attached to the upper left canine. A smear taken from beneath the pontic showed infection with Vincent's organisms.
1. MECHANICAL IRRITANTS AND LOCAL ABNORMALITIES.

(a) Irritating dental restorations such as overhanging rough fillings, ill fitting crowns and bridge-work, lack of contact points in restorations, and artificial dentures which impinge upon or irritate the gingival margins.

(b) Defective oral conditions such as gingival opercula covering partly erupted third molars, crowding and elongation of the clinical crowns of the teeth, which hinder the normal massage of the gingiva by food, calculus (which will be dealt with fully in Section 9).

Rommel (1) 1929, has drawn attention to a pocket formation between the enamel and gingiva in erupting teeth, which acts as a nidus for micro-organisms.

To confirm this, the writer examined about a hundred teeth in all stages of eruption from the time the cusps just appeared through the gingiva until the tooth reached the occlusion. It was found that in all cases there was at least a deep crevice on one side of the tooth, and in the majority of cases a pocket from two to five millimetres existed.

Although, as pointed out in the Histology Section, there is an organic attachment of the gingiva to the

enamel, it is surprisingly easy to break asunder, especially near the cusps or incisal edges of the teeth. As a consequence it is conceivable that the slightest trauma about the gingival margin, at the time of early eruption, may cause a pocket.

Pocket formation during the eruption of teeth probably explains in part at least, the ready susceptibility of young patients to acute Vincent's infection.

(c) Wrong type of diet (2) whereby only soft foods are eaten which do not stimulate and massage the gums during mastication as would crisp cellular foods.

(d) Lack of or defective artificial oral hygiene.

Fig. 22 Case showing infection of gum operculum overlying the distal surface of the lower left third molar. This site is a common primary incubation zone for the Vincent's organisms.

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Fig. 23. Case showing gingivitis around the lower incisors only. Smears showed only fuso-spirochaetal organisms.

2. DISUSE.

Wear on the incisal and occlusal surfaces of the teeth is normal. In aged persons where the gingivae are healthy, the enamel is almost completely worn off the occlusal surfaces of the posterior teeth so that when the mouth is closed, the gingivae in each jaw are quite close to each other. Clinically, however, in old age it is usually found that insufficient wear has occurred upon the occlusal surfaces of the teeth, and that inflamed gingivae are present.

This means that the teeth are not used sufficiently in everyday life, or that the consistency of the food eaten is such that too little wear results from
its mastication.

The bad habit acquired early in life of eating only soft or liquid foods, whereby little masticatory effort is required, is rarely overcome, even though the consistency of the food eaten later in life may be better. The art of proper mastication never having been learnt, is not usually acquired at this later period. In addition the rush and worry associated with civilised habits are frequently responsible for insufficient use of the teeth.

Often the result is that the jaws are not properly developed resulting in crowding and malposition of the teeth.

Lack of mastication further robs the gums of their normal amount of use and passive exercise.

Gottlieb and others have shown how teeth normally erupt during life. If the physiological wear upon the teeth is not sufficient, therefore, they appear elongated.

Elongated crowns of teeth further help to hinder food from massaging the gingival tissues.

Mechanical devices such as artificial dentures and bridges often cover up the gum margin in such a way that it cannot get its necessary stimulation from the mechanical action of food rubbing over it during mastication or from the action of the tongue and cheeks at all times.
Fig. 24  Diagram (a) illustrates teeth with a piece of food upon their occlusal surfaces. The food is of crisp consistency with a tough crust. Diagram (b) shows the same teeth and food as in Diagram (a) after closure of the jaws. Note the stimulating effect the food must have when it passes over the gingiva.

Fig. 25  Diagram (a) shows the same teeth and food as in Figure 24 (b), but after gingival recession has occurred. Note that the food does not reach the gingiva. Diagram (b) illustrates the same teeth and food as in Diagram (a) after the same amount of gingival recession, but also after attrition of the occlusal surfaces. Note that the food must have the same stimulating effect when it passes over the gingiva, as it has in Fig. 24 (b).
These factors, namely lack of mastication, the wearing of certain dentures, and elongation of the clinical crowns of the teeth, lead to the non-use of the periodontal tissues resulting in food stagnation around the necks of the teeth and other unfavourable sequelæ.

Intimated by analogous phenomena occurring in other bones and their surrounding tissues, some sequelæ of disuse that may arise are decalcification of the alveolar bone adjacent to a gingivitis and detachment of the periodontal membrane from such decalcified alveolus. Greig (3) states that decalcification unrelated to injury is most common in the cervical portion of the spine resulting from immediately adjacent inflammatory processes such as ulcers of the pharynx and suppurating tonsillitis. "Added to this, the inflammation restricts the use of the vertebrae and we have seen that disuse favours decalcification, by not fully utilizing the normal blood supply." Further Greig relates (4) instances where following decalcification of bone, ligaments attached to it yield readily.

The alveolus is meant by nature to withstand a certain amount of intermittent pressure, and the proper amount and frequency may be roughly judged as being correct when the wear upon the teeth is commensurate with

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(4) Idem, Page 228.
the age of the individual.

Where deficient wear is marked it is almost certain the supporting alveolus is not functioning sufficiently within physiological limits, and therefore, decalcification and degeneration of the calcified supporting tissues may be expected.

3. **TRAUMA.**

Some factors leading to trauma of the gingivae and investing tissues of the teeth are as follows:

(a) Abnormal biting habits such as biting cotton with the teeth.

(b) Incorrect tooth brushing, use of tooth picks and floss silk.

(d) Injury due to accidents, operations, mechanical devices such as matrix bands or separators.

Orban et al. (5) 1933 have found that in almost all cases where teeth had been extracted, there were signs of trauma in the neighbouring teeth.

(c) Uneven elongation of the clinical crowns of one or more teeth, due to lack of normal wear upon the occluding surfaces.

This last factor may produce "actual" traumatic occlusion which is the condition existing when the cusps

of the teeth are so interlocked that during mastication, excessive strain is placed upon one or more of them, with the result that an injury occurs in the periodontal membrane and sometimes also in the alveolus. Stillman and McCall (6) 1927 call this trauma which is manifested in the supporting tissues, "occlusal trauma", and state that traumatic occlusion is a functional incoordination of the teeth relating wholly to a mechanical disharmony.

The pathological, periodontal effects usually attributed to traumatic occlusion are in most cases due to lack of proper mastication which is difficult when the occlusion is defective. The beneficial results arising after the correction by grinding of interlocking cusps, is due, for the most part, not so much to the relief of occlusal trauma, as to the greater efficiency accomplished in mastication, resulting in greater stimulation of the gingivae by detergent food.

4. INFECTION.

Infection of the gum tissue will occur providing (a) the infecting organisms are present in virulent form and (b) the tissues are so degenerated as previously described under heading No. 1, that (i) their defence is lost and (ii) they can be readily reduced of oxygen thus rendering them favourable pabulum for anaerobic

(6) Stillman, and McCall. Dental Items of Interest. April, 1927.
organisms (7).

The two factors, degenerated gingival tissue and infecting organisms, must be present before infection can occur. The severity of the infection depends on the virulence of the organisms and the degree of katabolism which has resulted in the tissues.

Once infection becomes established (and it is usually fuso-spirochaetal in nature) inflammation sets in.

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Upon consideration it is seen that the above mentioned aetiological factors cause an inflammatory reaction in the gingivae. In most cases the causes operate for a considerable time and, therefore, the inflammatory reaction becomes chronic. However, the inflammation in the gingiva begins by exhibiting, according to its severity, the usual cardinal signs of inflammation such as swelling, redness and pain.

One of the effects is a hyperaemia of the gingiva and a slowing of the circulation. Later, amongst other inflammatory phenomena, congestion and perhaps stasis occur in the capillary loops and venules near the seat of irritation which is usually in the marginal gingiva.

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(7) Box, H. K. "Necrotic Gingivitis (Trench Mouth)." The University of Toronto Press, Toronto, 1930. Page 34.
Surrounding this area there is a zone of active hyperaemia.

Both these zones, that of congestion in the marginal gingiva and that of active hyperaemia in the contiguous deeper tissues, play a very important part in gingivitis and later on, in complex periodontitis.

The effect upon the deeper adjacent tissues where an active hyperaemia is present may be implied by analogy with commonly accepted laws governing hyperaemia elsewhere in the body.

It is to be remembered, however, that the blood vessels supplying the marginal gingiva come from the periodontal membrane which derives some of its blood supply from the medullary spaces of the alveolar bone, the periosteum of the alveolus and the adjoining mucous membrane: A part of each of these tissues are usually in the zone of hyperaemia.

Greig (8) 1931, states "as manifest truth" capable of recognition by every operating surgeon that hyperaemia in bone produces rarefaction, decalcification or osteoporosis.

During the progress of hyperaemia, calcifying salts of the bone are dissolved and removed, while new bone and cementum formation in the same zone are impossible. As a result the strength and support of the periodontal

membrane in the hyperaemic area suffers, because it depends upon the continual deposition of cementum and alveolar bone for the attachment of its fibres. Consequently the fibres become elongated and lose their attachment, paving the way for pathological pocket formation.

**LITERARY REVIEW.**

Broderick (9) 1928, has written extensively on the systemic metabolic changes that may occur during life and which are likely to upset the acid-base balance of the tissues. He writes (10) that the causes of pyorrhoea alveolaris "lie in a tendency to an alkalaemia, in the compensation of which, conditions will become favourable for the deposition of calculus around the necks of the teeth in the subgingival trough. That as the result of the irritation which this calculus brings about, there will occur a marginal gingivitis that will predispose these tissues to infection by any of the micro-organisms that inhabit the mouth, and that the clinical condition so easily recognised, is the normal result of this infection."

Kritschevsky and Seguin (11) 1924, Belding and ..........


(10) Idem. Page 204

Belding (12) and (13) and Keilty (14) are of the opinion that an infection with spirochaetes is the forerunner of almost all gingival ailments. Kritschevsky and Seguin (11) mention that an outbreak of acute spirochaetosis may be traced to a decrease in the vitality of the tissues by the action of attrition, elimination of poisonous medicaments, and irradiations.

Merritt (15) writing on the etiology of periodontoclasia gives the main factors as follows:--

(a) A systemic metabolic disturbance usually represented by an excess of basic elements in the body fluids.

(b) A subnormal metabolism in the periodontal tissues by which their susceptibility to infection is increased and

(c) Bacterial infection.

All these writers' views are very similar, in that each implies that a predisposing metabolic disturbance of

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the host, a local disturbance, and then bacterial infection, represent the sequence of events.

Hartzell (16) 1927, says that providing the soft tissues are daily stimulated by friction and pressure, no periodontoclasia or pyorrhoea can exist.

This is somewhat in agreement with the other views since by maintaining the local resistance, and preventing blood stasis, infection is not so likely to occur even in spite of, as Hartzell says, the worst possible diet.

It is extremely unlikely, however, that patients on the worst possible diet would have a perfect local resistance and so it is equally unlikely that they would have normal gingivae.

Grieves (17) 1919, says "oral filth, not oral sepsis, appears to be the real factor," whereas Hardgrove (18) 1928, says Vincent's infection is contagious and clean mouths seem to be not much more immune than mouths poorly cared for.


Moore (19) also considers Vincent's infection to be a "filth" disease.

This appears to be so in a few instances but in most cases the disease appears despite apparently fair oral hygiene. Reichmann (20) agrees with this latter view, and has found only a very small percentage of Vincent's infection in dirty mouths.

The difference of opinion regarding cleanliness and filth are a matter of degree of "filth" only. Cleanliness is judged as a rule by the appearance of the teeth after tooth brushing etc. This kind of cleanliness is very deceiving. The parts of the teeth and gums that are seen may look clean, but in between the teeth and the areas near the gingival trough which are hidden from sight and which are the areas that really matter, may be quite unclean.

Physiologically clean teeth and gums are a different matter altogether. For the teeth and gums to be physiologically clean the gums would have to be normal, in which case infection could not take place. Little importance can be placed, therefore, on discussions of cleanliness and "filth" in relation to gingivitis unless physiological cleanliness, and "filth" are the two


criteria under discussion.

Prinz (21) acts as a mediator by stating that in a perfectly clean mouth the disease is rarely observed. He explains the reason why Vincent's infection is found on one side of a mouth and not the other at times. It is because the side that is used for mastication is cleansed and so no infection occurs, while upon the other side chewing is not performed (because of some pathological defect) and as a consequence infection ensues due to the collected sordes.

Willman (22) states that "marginal pyorrhoea," which constitutes 90% of all clinical cases of pyorrhoea, is usually the result of calculus.

Fones (23) summarises the opinions of many workers in periodontooclasis by stating that the underlying factor in the aetiology of all forms of periodontooclasis is low resistance to bacterial invasion on the part of the cells of the supporting tissues of the teeth.


Blackberg and Berke (24) demonstrated in animals, periodontal lesions with epithelial proliferation occurring when there was a disturbance in calcium and phosphorus metabolism.

Richards (25) discussing pyorrhoea maintains the view that an inherent lack of resistance either mechanical or physiological in the investing tissues is a predisposing cause.

Rosenthal (26) and Prinz (21) 1927, writing on Vincent's infection say the predisposing cause of the infection is apparently an avitaminosis.

Wright (27) 1930, says, "One of the chief predisposing factors in Vincent's infection seems to be low resistance from malnutrition, disease, lack of proper rest and exposure."

Puterbaugh (28) 1934, states that malnutrition, 


avitaminosis, severe metabolic disturbances and diseases of the oral tissues are important predisposing conditions in Vincent's infection.

Reichmann (29) 1926, and Withycombe believes the aetiological factor to be purely local. Smith (30) 1932, thinks overcrowding poor hygiene and improper diet, especially when there is a deficiency of vitamin C, are predisposing causes.

Sorrin and Miller (31) 1928, say a lowered general resistance plus suitable pabulum are necessary for the Vincent's organism to propagate. Fitzgerald (32) thinks there is a predisposing cause due to some debility or malnutrition and a local cause of some irritation. Merritt (33) 1933, stresses poor oral hygiene as the predisposing factor. He thinks the capacity of the organism to cause disease depends on 1. Their number. 2. Their


virulence, and 3. The resistance of the host.

Smith (34) summarises a very common contention regarding the pathology of periodontoclasia. The factors are as follows:

1. Disturbance of function, i.e. traumatic occlusion.
3. Infection by bacteria so held.

Bonney (35) 1924, gives debilitating diseases, excessive use of tobacco, excessive fatigue, exposure and lack of proper food as the principal causes of Vincent's infection.

Almost all writers on acute Vincent's infection including Rosenthal (26), Smith (30), Merritt (33), Pilot (36) and Wagener (37) believe that the disease is contagious and infectious, but most of them believe

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\text{(35) Bonney, T. C. } "\text{Acute Ulcerous Gingivitis: Its Prevalence in the United States, and the Importance of its Early Recognition and Treatment." } \text{The Dental Cosmos. Vol. 66, No. 12, Page 1281, 1924.}
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\text{(36) Pilot, I. } "\text{Fusospirochetal Infections of the Mouth." } \text{Journal of the American Dental Association. Vol. 15, Page 1763, 1928.}
\]

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\text{(37) Wagener, Edna H., McEvoy Elmer, Taber Loren, and Curley Martin, } "\text{The Laboratory Diagnosis of Vincent's Infection." } \text{Journal of Dental Research, Vol. 10, Page 591, 1930.}
\]
the "soil" must be suitable before infection can take place.

Corby (38) 1933, thinks that periodontoclasia is a symptom of abnormal complex nerve reflexes, caused by a disturbed mental state altering the quantity and the chemical composition of the fluids of the body, and under these adverse conditions micro-organisms, traumatic occlusion, etc., carry on their destructive action.

Haley (39) 1933, thinks the contributing causes of paradentosis are in part at least "filth" collections, weather changes, inefficient circulation and exercise, and perhaps diet.

Larson and Barron (40) 1913, give some very interesting experimental proof that there must be some predisposing factor present before a fusospirochaetal infection can occur. They found that in no case could hospital gangrene, which was shown to be a fusospirochaetal disease, be produced experimentally in inoculated patients. Apparently Vincent inoculated himself, but only produced an insignificant pustule. Continuing


they say, "Gemy inoculated three persons with material from severe cases of hospital gangrene without success. It is to be assumed, therefore, that certain conditions must be fulfilled before an infection can take place."
SECTION NO. 4.

DIAGNOSIS.
DIAGNOSIS.

As explained in the introduction, gingivitis is intimately associated with complex periodontitis and these conditions may be considered the primary and secondary stages of the one disease. Therefore, as might be expected, the transition from one stage to the other is a gradual one, calling for an intimate knowledge of the symptomatology of both conditions.

Accordingly a classification of periodontal diseases and the symptoms of each class are presented to show the category to which gingivitis belongs and for its diagnosis.

In writing on periodontal diseases, acute Vincent's infection is usually dealt with as a separate entity, but because it is a form of gingivitis and is considered by the writer as just an acute form of common gingivitis, it is dealt with (for the want of a better classification, realising that it becomes phagedenic in nature at times) under the heading of Marginal Periodontitis.

Periodontitis may be conveniently divided into three main classes:

(A) Simplex or Marginal Periodontitis.
(B) Complex or Vertical Periodontitis.
(C) Atrophic Periodontitis.

(A) Marginal Periodontitis may be divided into the
following sub-headings:—

(1) Acute Vincent's Infection.

(2) Common Gingivitis or Chronic Vincent’s Infection.

(3) Other types of Gingivitis.

As this thesis deals principally with the most common form of gingivitis called by the writer chronic Vincent's infection, Class A of the classification of periodontitis will be dealt with more fully than Classes B or C.

(A) SIMPLEX OR MARGINAL PERIODONTITIS.

Marginal periodontitis is characterised by inflammation of the gingivae accompanied by discomfort and haemorrhage.

As a rule it is not suppurative, but may become so after pocket formation when it gradually turns to vertical periodontitis.

(1) ACUTE VINCENT'S INFECTION.

Acute Vincent's infection (synonyms:—ulceromembranous stomatitis, necrotic gingivitis) is an infectious disease affecting the gums and oral mucous membrane. It usually starts in some area where conditions are favourable for anaerobiosis. Such areas are (a) gingival flaps overlying erupting teeth, (b) pockets existing between the gums and teeth, (c) fissures formed by the deposition of calculus which adheres to the necks of the teeth and which lies in contact with the gums, (d) spaces beneath fillings which protrude beyond the cavities at the
gingival margin, (e) bands, crowns and bridge pontics, and (f) folds in the cheek caused by loss of tone of the cheek muscles, which consequently become traumatized during mastication.

The free margin of the gum is then attacked by the fusco-spirochaetal organisms and becomes necrotic, leaving a light greyish slough on its surface. The infection spreads and forms patches upon the cheek, palate, fauces and tonsils but not upon any exposed parts of the lips.

Necrotic mucous membrane is easily wiped away leaving a bleeding surface underneath. A gnawing pain is experienced in the gums, while the teeth feel numb or as though they were wedged apart or elongated. Mastication is difficult, and tooth-brushing is impossible, because of their discomfort and pain. The saliva is plentiful, thick and ropy. Halitosis is intense, the odour being characteristic of the disease. A metallic taste is sometimes noticed. Cervical lymph glands are often swollen and sore. The patient feels depressed, complains of malaise, runs a temperature of about 100°F., has an increased pulse and respiration rate, and often cannot sleep at night. The gastro-intestinal track is sometimes deranged, constipation being usually present together with anorexia. The tongue is heavily coated and occasionally swollen.

The disease may be only manifested on one side of the mouth, in one jaw, or in defined areas. A character-
istic feature of Vincent's infection is the disappearance of the interdental papillae, which results in the gum margin ending in a fairly straight line. The infection never spreads on to the lips, but may appear anywhere in the mouth and spread to the tonsils, throat or lungs. In many cases, however, infection apparently starts, or certainly shows exacerbated signs in the throat and lungs, before it does in the mouth. Smith (1) gives some case histories one of which indicates that fusco-spirochaetal disease can start in the lungs without acute symptoms first occurring elsewhere.

Rosenthal (2) 1929, classified the disease into three groups:--The first class is called primary Vincent's Infection and affects the superficial tissues of the mouth and throat, the second class is called secondary Vincent's infection and affects the deeper structures e.g. bone, and the third class or tertiary Vincent's infection is where invasion occurs in other parts of the body as in the lungs.

Finally a diagnosis of Vincent's infection would not be complete without examining under the microscope a suitably stained smear from the gums. Spirochaetes and


fusiform bacilli should predominate the field. By this method Vincent's infection may be differentiated from diphtheria and thrush.

Clinically acute Vincent's infection may be differentiated from diphtheria in that,

1. Its onset is sudden whereas that of diphtheria is slow.

2. Its associated constitutional disturbances are slight compared with those of diphtheria.

3. The mucous membrane is necrotic in Vincent's infection whereas in diphtheria a false membrane composed of fibrin is present.

4. Its necrotic mucous membrane is much easier to remove than the fibrinous false membrane of diphtheria.

5. There is much more pain associated with Vincent's infection than with diphtheritic lesions.

6. The lymph glands draining necrotic areas are usually enlarged and tender, while the glands draining diphtheritic areas are seldom apparent.

Acute Vincent's infection differs from thrush in that its lesions are never as extensive as in thrush where they may cover the whole of the mucous membrane of the mouth and lips at the one time. The halitosis arising from Vincent's infection is more marked and different from that arising from thrush, where it is of a sweetish acid character. The microbic flora is not the same, the Oidium albicans being the causative
organism found in thrush.

Secondary lesions of syphilis are sometimes confused with acute Vincent's infection. The "snail track" ulcers of syphilis differ by being more destructive and more prone to invade the cheek and palate than do the lesions of acute Vincent's infection. The Wasserman test in these cases may be an aid, a negative reaction indicating that the ulceration is not due to syphilis.

Fig. 26  Case of Acute Vincent's infection showing the necrotic mucous-membrane which may be seen around the lower premolars, loss of the apex of the interdental papillae between the lower right lateral incisor and canine, swollen papillae about the lower incisors and inflamed haemorrhagic areas around the upper right incisors.
Fig. 27  Case of acute Vincent's infection showing necrotic gingival margin (vide upper right second incisor), the loss of the interdental papillae and slight general hypertrophy.

Fig. 28  Case illustrating Vincent's infection of the oral mucous membrane near the lip, but not upon any exposed portion of it. Smears taken from the necrotic membrane showed Gram positive diplococci and Vincent's organisms.
(2) CHRONIC VINCENT'S INFECTION.

The following are symptoms which occur in chronic Vincent's infection, most of which disappear when suitable treatment is instituted.

(a) Inflamed, tender, haemorrhagic gums.

(b) Absence of stippling due to the distended tissues.

(c) Loss of the interdental papillae which gives a straight line appearance to the gum margins.

(d) Loss of the bucco or labio-lingual or palatal connections of gum tissue, causing a depression between the teeth where normally an elevation is present, and an apron-like appearance of the outer or inner gum margins.

(e) Hypertrophy of the papillae which cover the crowns of the teeth.

(f) Flat summits of the oedematous papillae presenting the appearance as though half of each papilla had been cut off with a sharp knife.

(g) Altered appearance of each gingival margin from a concave to a V shape exhibiting a zig-zag appearance of the margin of the gingivae.

(h) A semi-circular iris appearance of the gum margin.

(j) Glets along the margin which appear as though the gingiva had been cut through at an angle with its border.
Hirschfeld (3) 1934, describes many symptoms of Vincent's infection, most of which are similar to the above and are of the chronic type.

Fig. 29. Case of gingivitis showing a straight line appearance of the gingival margin between the lower lateral incisors.

(3) Hirschfeld, I. "Vincent's Infection of the Mouth: Clinical Incidents in its Diagnosis and Treatment." Journal of the American Dental Association, Vol. 21, Page 768, No. 5, May 1934.
Fig. 30. Case of chronic gingivitis in the upper jaw. There were no crevices deeper than 2 millimetres.

Fig. 31. Enlarged X-ray photograph of upper left incisors and canine of patient shown in Fig. 30, exhibiting a normal periodontal membrane at A, and a thickened periodontal membrane due to the long standing gingivitis at B.
(3) OTHER TYPES OF GINGIVITIS.

As over 90% of gingivitis cases may be classified as acute or chronic Vincent's infection, this presentation is concerned mainly with these, and only touches on the other types of gingivitis which are less frequently met with and which are usually symptoms of profound constitutional disturbances.

GINGIVITIS OF BLOOD DYSCRASIAS.

Blood dyscrasias are important to dentists because the early manifestations of these abnormalities, which occur in the mouth, may influence the afflicted ones to seek dental rather than medical aid. One of the symptoms of some of the blood dyscrasias is an acute gingivitis, which exhibits all the signs and symptoms of acute Vincent's infection.

For the elimination of the possibility of a blood dyscrasia being present, a blood examination is indicated in acute Vincent's infection cases, especially where fever, malaise, and mental confusion exist and where the condition does not respond readily to local treatment. Under such circumstances it would not be impossible for the patient to be suffering from such a condition as agranulocytoysis, where delay in the proper treatment of the blood condition would inevitably prove fatal.
The important blood dyscrasias are:
(a) Agranulocytic angina.
(b) Pernicious anaemia.
(c) Haemorrhagic purpura.
(d) Leukaemia.
(e) Leishmaniasis.

To diagnose and differentiate each condition, a blood examination is necessary; however, a brief description of the clinical signs and symptoms, as gathered from the writings and case histories of Mark (4) 1934, Cody (5) 1934, Lichtwitz (6) 1934 and others are as follows:

(a) Agranulocytic angina is manifested by swollen haemorrhagic gums infected with the fusobacterial organism, and exhibiting all the characteristic symptoms of acute Vincent's infection, or Vincent's angina when a dysphagia is evident.

(b) Pernicious anaemia causes a burning feeling of the mucous membrane of the cheeks and gums. Red


spots appear on the tip and edges of the tongue accompanied by dryness and cracking. Swelling and bleeding from the gums is noticeable, especially in the final stage.

(c) Haemorrhagic purpura shows somewhat a similar oral condition to that of agranulocytic angina, such as haemorrhages from the gingival margins, and from the nose.

(d) Leukaemia has the same oral entities as agranulocytic angina.

(e) Leishmaniasis and other parasitic blood conditions produce anaemia and exhibit similar mouth manifestations to the latter.

THRUSH.

Thrush is an infection of the gums and mucous membrane of the mouth which at its inception is painless, but becomes painful if desquamation of the white diseased plaque occurs leaving an excoriated haemorrhagic surface. The plaque, which may cover the whole of the mucous membrane of the mouth, tongue, and lips is due to a parasitic fungus, Oidium albicans.

It occurs mainly in children under the age of five, but may appear in adults, especially if they are suffering from some debilitating disease.
PREGNANCY GINGIVITIS.

Ziskin et al., (7) 1933, summarising their observations on 416 cases of pregnant women, found 158 or 37.9% of them had some form of gingivitis. The commonest site was in the anterior region, the next most frequent site being the premolar region. The authors describe four forms of pregnancy gingivitis. Each may be a progressive stage of the same underlying factors.

1. The first form is characterised by bleeding of the gums when slightly traumatised by the toothbrush or by food, etc.

2. In the second form the free gum margin is involved, and takes on the colour and appearance of a raspberry. It bleeds easily, and is more common in the anterior region.

3. The third form is manifested by generalised hypertrophy of the gingivae. The papillae grow out and push the normal gum back forming a straight blanched line immediately above the proliferative tissue. It is usually confined to one section of the mouth and is called "Hypertrophic Gingivitis of Pregnancy."

4. The fourth stage is characterised by the so-called "Pregnancy Tumour" which is usually confined to

a single growth arising somewhere along the gum margin. At the beginning the tumour appears to be an overgrown gum papilla. It may be either sessile or pedunculated, and it appears cyanotic in colour with a bright red border.

The authors state that "Pregnancy Gingivitis" may or may not be accompanied by a subacute or chronic Vincent's infection, suppurative periodontoclasia, or by the presence of fungi and bacteria in the tissue. They summarise by saying, "The significant change in the gums in 'Pregnancy Gingivitis' is hyperplasia of the epithelium."

**TRAUMATIC GINGIVITIS.**

There is a type of gingivitis caused by the traumatic action of the toothbrush which deserves mention. It is caused by the too vigorous action of hard toothbrushes or by the use of abrasive dentifrices in conjunction with the brush. In right handed patients the teeth and gums of the left side are abraded to a much greater extent than those of the right side where a "back-hand" stroke of the brush is required. The mucous membrane becomes hornified in parts and worn away in areas near the necks of the teeth. The lingual surface of the gum as well as the interdental papillae are not, as a rule, altered to any great extent.
Fig. 32 Case of gingival recession due to toothbrush trauma. The abrasion marks on the gum correspond to those on the teeth. Patient thought his shoulder pain was relieved when he made his gums bleed by vigorous brushing.

**CATARRHAL GINGIVITIS.**

Catarrhal gingivitis is an inflammatory type of ulitis usually associated with acute coryza. The gums become shiny, red, and swollen and the teeth feel uncomfortable and elongated due to the hyperaemic periodontal membrane.

The complaint is only transitory, but the congested or hyperaemic gingivae readily succumb to a chronic fusospirochaetal infection, and later develop suppurative periodontitis.

**GENERALISED HYPERTROPHIC GINGIVITIS.**

Generalised hypertrophic gingivitis is a relatively
painless condition characterised by hard, fibrous, red turgid gums. The enlargement may extend outwards from the alveolus, or towards the incisal, or occlusal surfaces of the teeth when a false pocket is formed. In certain cases the swelling, which is hard and of a fibrous nature, may reach to the muco-labial or mucobuccal fold.

In mild cases the anterior teeth are affected to a greater extent than the posterior ones. Irregularities of the hypertrophic gum occur presenting a nodular appearance over the alveolus, or enlarged and sometimes club-shaped papillae.

The condition is often associated with mouth breathing, enlarged tonsils, and adenoids, but cases present that are not connected with these conditions.

In the latter instance some chronic irritation, such as that which arises from the existence of a chronic Vincent's infection, is present. One of the ways in which the body reacts in defence of a fusospirochaetal infection is by the production of fibrous tissue (8) and some cases of hypertrophic gingivitis may well be due to such a reaction.

Fleming (9) 1933, and Goadby (10) 1931, describe the condition very well and the former writer gives the histories of some cases which he has treated.

Fig. 33 Case showing hypertrophic gingivitis. Note the apron effect that the gum presents over the upper left lateral incisor, also the ulceration on the lower gum and haemorrhagic area around the lower left lateral incisor. A smear from the gingival margin showed an infection with the fusospirochaetal organisms.

LOCALISED HYPERTROPHIC GINGIVITIS.

Localised hypertrophic gingivitis is a pathological state of the gums occurring around one or at least a limited number of teeth.

It is due to some irritation which is primarily

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mechanical and secondarily of microbial origin. Ill
adapted dentures or bridges and sharp edges of broken
teeth or fillings are the usual sources of mechanical
irritation.

The appearance of the hypertrophied gums is essent-
ially the same, but perhaps not so pronounced as prev-
iously described under Generalised Hypertrophic Gingivi-
tis.

Fig. 34  Case showing hypertrophy of the gingivae
on the palatal surface of the upper molars in a male
aged 40. The hypertrophic gums almost met at the median
sagittal plane and presented the appearance of a cleft
palate. The condition had been present since childhood,
but the patient had not worried about it as he thought
the two growths were his tonsils. The gums on the
palatal surface of the upper molars were heavily infected
with fusco-spirochaetal organisms, but elsewhere, although
the gingivae showed infection with the same organisms, no
hypertrophy was evident.
A chronic type of Hypertrophic Gingivitis is described by Goadby (11) 1931, which is really a sclerosing hyperostosis of the alveolar bone. In this affection nodules of bone protrude from the alveolus, and the covering mucous membrane appears stretched and thin, to such an extent that the colour of the bone can be seen beneath. Goadby states that histological examination of the enlarged bony masses, shows irregular bone hypertrophy resembling osteoma.

Fig. 35  Case showing hyperostosis of the alveolar bone. A fold of gum tissue is seen at A above the osteophyte B which extends to the first molar region.

(B) COMPLEX OR VERTICAL PERIODONTITIS.

Complex periodontitis is manifested by pocket formation and suppuration, and for this reason it is sometimes called suppurative periodontitis. When an infected pocket over about three millimetres deep is found and the signs of atrophic periodontitis are not present, the condition comes under this classification.

In cases where the pockets are deep, serumal calculus can usually be found adhering tenaciously to the sides of the teeth opposite the crevicular epithelium. The teeth become loose, drift out of occlusion and appear elongated because of the accompanying recession of the gingivae. Usually there is no pain, but halitosis is often present together with an early morning bad taste. Pus is present and there may or may not be a superimposed marginal inflammation.

X-ray photographs show a rarefying osteitis about the margin of the alveolar bone, which presents a diffuse appearance merging from the totally decalcified alveolar crest to the normally calcified deeper bone sometimes a centimetre away. The periodontal membrane appears thickened, usually for a distance of a few millimetres deep from the alveolar crest, but sometimes entirely around the tooth.
Fig. 36. Case of complex periodontitis and X-ray photograph of upper right lateral incisor and canine area. Note rarefying osteitis at the margin of the alveolar bone and thickened periodontal membrane.

(C) **ATROPHIC PERIODONTITIS.**

Atrophic periodontitis is evidenced by areas of sharply demarcated bone deficiency at the alveolar margin, and loose teeth which drift out of occlusion. There may be little or no visible inflammation of the soft tissues.

Examination by X-ray photographs show large clearly defined areas of bone loss involving the crest of the alveolus.

The rarefaction starts at the crest of the alveolus and proceeds slowly towards the level of the tooth apices without affecting the deeper bone.

The loss of alveolus so maims the support of the teeth and gums that the former drift out of occlusion, and the latter cleave away from the sides of the teeth.
causing deep pockets which readily become infected. Drift-
ing of the teeth results in traumatogenic occlusion which
in turn causes further loss of alveolar bone and so a
vicious circle of pathological conditions is set up. The
anterior teeth often protrude in a fan-like manner.

It is difficult to attribute any one factor as the
cause of atrophic periodontitis. The fault is apparently
due to the non-regeneration of the periodontal fibres at
the crest of the alveolus which atrophies when its con-
nection with the tooth, via the periodontal membrane, is
severed. The non-regeneration of the periodontal fibres
approximating the crowns of the teeth may be due to some
local nutritional deficiency or halisteresis such as
would occur in a hyperaemic area.

It would appear that a general lack of bone forming
elements in the body cannot be the cause of the alveolar
atrophy because the deeper alveolar bone always appears
radiographically normal.

Willman (12) 1933, describing Diffuse Atrophic Pyorr-
hoea says it is possibly due to a failure of the cemento-
blasts to deposit layers of cementum to anchor new fibres
of the periodontal membrane as they are developed to re-
place old ones.

(12) Willman, W. "Periodontal Diseases." Journal of
the American Dental Association. Vol. 20, No. 9,
Page 1617, 1933.
Fig. 37  Case of male, aet. 20, showing diffuse atrophy of the alveolus. Although the gums look relatively normal, very deep vertical pockets are present around all the incisors. Note the good teeth, lack of wear, traumatic occlusion and the drifting of the anterior teeth in a fan-like manner causing a very wide diastema between the upper left central and lateral incisors. All the incisors except the upper right central and lateral, were very loose.
Fig. 38. X-ray photographs of case shown in figure 37. Note the extensive bone loss around the upper left lateral incisor and around the lower incisors. The deeper bone appears normal.
SECTION NO. 5

BACTERIOLOGY.
Bacteriology.

The mouth harbours a tremendous number of microorganisms, most of which are saprophytes, or at least non-pathogenic to the host. It is extremely hard to know which of them are the normal inhabitants. Any organisms which cause harm or disease by their presence cannot be classified as normal, because a normal mouth does not show disease. An examination of a healthy mouth reveals organisms such as Streptococcus, Staphylococcus, diphtheroids, Coliform bacilli, Micrococcus, catarrhalis, and filamentous forms of the Leptthrix class.

Normally most of the organisms inhabiting the mouth remain non-pathogenic, but some may become pathogenic under favourable conditions.

Kritschevsky and Seguin (1) 1924, who have done a lot of work on fusco-spirochaetal diseases, believe that these organisms are the causative agents of the affections associated with their presence, such as Vincent's infection, that the spirochaetes are the principle invaders, and that the fusiform bacilli play only a secondary part which consists in completing the digestion of the tissues. In severe fusco-spirochaetal infections they state that the leucocytes are twisted and torn into

fragments by the spirochaetes. Cahn (2) 1925, thinks the fusiform bacillus is the specific organism in Vincent's infection and that it lowers the resistance of the gums allowing the spirochaetes to invade the area. He further thinks the number of spirochaetes present is directly proportional to the amount of inflammation.

Garvin (3) 1927, believes that the future will show that the spirochaete plays a much greater part in the pathology of periodontoclasia than it has been given credit for. He questions the importance of the statement, so often made, that this disease can exist only as the result of the presence of the Streptococcus and Staphylococcus.

Pilot (4) 1928, does not attach any importance to the mere presence of Vincent's organisms in the mouth and considers them part of the normal flora. Nevertheless, in the same article he mentions that in smears made from the mouths of medical students only 50% showed spirochaetes. If spirochaetes are normal inhabitants of the mouth they might be expected to be found in a

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majority of cases.

On the other hand Appleton (5) 1929, holds the view that the presence of Vincent's organisms is indicative of some pathogenic condition.

Belding and Belding (6) 1933, are of the opinion that the spirochaetes are the primary invaders in periodontal infections, and the pyogenic cocci secondary ones. They later (7) 1934, state: "For all practicable purposes, as far as dentistry is concerned, every infection in or about the mouth, with the exception of caries and an occasional case of focal infection, is caused by the activity of the fusco-spirochaetal organisms, the fusiform bacilli and streptococci, both generally innocuous, and the pathogenic spirochetes." They have found that the deeper layers of the dental plaque is composed almost exclusively of fusiform bacilli and their products. Quoting them further, they say "This film permits the development of gingivitis and pyorrhoea; for its mass is easily penetrated by the motile spirochete and provides the food and anaerobic habitat which is essential for its initial invasion and continued existence in the oral


cavity." In this way only, they believe, is the fusiform bacillus in symbiotic relationship with the spirochaete.

The writer has found the presence of Vincent's organisms in almost every case of gingivitis and periodontitis examined, and would like to stress the fact that wherever the fusospirochaetal organisms are found some pathological condition exists. The fusospirochaetal group of organisms are never found in a normal healthy mouth.

Although there is an abundance of evidence to show that the fusospirochaetal group of organisms are likely to be the causative agents of the diseases with which they are associated, yet it is submitted that they have never been definitely proved to be the causative organisms.

In order to ascertain if the comparative number of spirochaetes to other organisms present, had any relationship to the clinical appearance of the gingivae, the writer conducted a weekly examination for a period of four months, of the gums of fourteen patients who presented gingival conditions ranging from normal to that of complex periodontitis. Smears upon microscope slides were obtained and stained by Fontana's method, and the average percentage of spirochaetes to the total number of organisms in several fields was tabulated.

It was found that the percentage of spirochaetes to the total number of organisms present was only related
to the clinical appearance of the gingivae when the percentage was over eighty or below fifty. In the former case the gingivae were very inflamed and in the latter case the gingivae were only very slightly inflamed. When there was no apparent inflammation usually no spirochaetes could be found.

The bacteriology of Vincent's infection, especially the chronic form when pus is present, and that of pyorrhoea are very similar.

In pyorrhoeic pockets, at times, the debris and pus present may be sufficient to overshadow the fusospirochaetal organisms, but if the pus is wiped away and a smear taken from the bottom of the pocket generally only the fusospirochaetal organisms are seen. Their presence deep in the pockets is significant that they are at least present where tissue destruction is progressing.

An interesting observation is that the more chronic a gingivitis becomes the more the secondarily infecting cocci appear in the gingival pocket.

A great number of writers have written on the organisms associated with Vincent's infection and pyorrhoea. The spirochaetes are the most frequently discussed and nearly every writer has given his own name to the various ones seen, with the result that the nomenclature to a great extent, is completely incoherent and useless.
Hindle (8) 1931, and Appleton (9) 1934, have endeavoured to correlate the various names and the organisms, and their work has simplified the previous confusion. Smith's classification (10) 1932, of the fusospirochaetal organisms based on Noguchi's work has been found the most useful by the writer whose observations of the mouth organisms correspond exactly with the morphology given by Smith.

The morphology and motility are best examined by dark-ground illumination. Where stained specimens are relied upon the writer agrees with Wilson (11) 1933, that Fontana's stain gives the most satisfactory results. However, for rapid diagnostic purposes more simple stains such as gentian violet or weak carbol-fuchsin as recommended by Wadsworth (12) 1927, are very convenient and effective. Examination of stained specimens has the disadvantage that in most cases when the spirochaetes loose


their motility or die they alter in shape to such an extent that some or all of their coils may be lost. It is, therefore, difficult to differentiate the various types of spirochaetes by this method.

Dealing with the spirochaetes first, there are to be found four morphological types. The generic name Treponema is used instead of Spirochaeta.

1. **Treponema Vincenti**.

*Treponema vincenti* is a spirochaete about 20 micra long, and 0.3 micron wide, and appears as a single line when viewed by the dark-ground illumination. It has about 20 coils or spirals which are small when compared with some other mouth spirochaetes; it is actively motile, propelling itself by a snake-like movement whereby only two or three long secondary body waves are used for locomotion. The twenty odd small spirals do not assist in the progressive movement. When examined by dark ground illumination in a wet slide preparation, the *Treponema vincenti* have a great tendency to adhere to the glass slide or debris; they lash violently to free themselves and usually succeed in doing so after a few seconds. Their progression across the field is extremely rapid at times, and for apparently no reason they will stop suddenly, reverse their movement, and travel in the opposite direction. Dexterous manipulation of the mechanical stage is necessary to keep the organisms under observation.