2. **Treponema buccale**.

Treponema buccale is an organism which reflects the light, when seen by dark ground illumination, in such a manner that it appears to have a double contour. Its length varies from 7 to 25 micra, and its width is about 1 micron. It has from 3 to 8 regular curves according to its length. Some of these organisms are slow in their movement, while others progress rapidly, but they seldom reach the speed of the Treponema vincenti. After vibrating for a few seconds it rests for a short time and then resumes its motility. This organism progresses with a cork-screw motion, and in this respect its movements differ somewhat from those of the other spirochaetes.

As it is only the wider organisms (comparison of width made by synchronously prepared stained smears) which show a double contour under the dark field illumination, it is fairly certain that the double contour is due to the width of the organisms. Those organisms that appear to reflect only a single line, do so because the bands of reflected light on either side of the organism are so close that they are beyond the resolving limit of the microscope objective.

It is extremely unlikely, although a possibility in some cases, that the double contour and single line appearances may be due to different refractive indices of the organisms when in certain fluid media.
3. **TREPONEMA MACRODENTIUM.**

Treponema macrodentium is an organism which varies in length from 10 to 25 micra, in width from 0.25 to 0.5 micron and has from 8 to 12 coils. It lashes about very vigorously, but does not make much progress. Both ends of the organism vibrate rapidly while the centre is comparatively inert. After a period of motility it stops momentarily and straightens itself out somewhat, so that there may be only approximately six or less coils to be seen.

4. **TREPONEMA MICRODENTIUM.**

Treponema microdentium is a small spirochaete with very close coils. The organism varies from 5 to 12 micra in length, and from 0.25 to 0.5 micron in width with approximately 8 to 12 rigid coils. It is only slightly motile and makes very little progress.

Treponema mucosum is morphologically indistinguishable from Treponema microdentium.
Fig. 39 Smear taken from the gingival margin in a case of Vincent's infection and stained by Fontana's method. The following organisms are typical of the various classifications: A. Treponema buccale, B. Treponema macrodentium, C. Treponema vincenti.

All the mouth spirochaetes can be grouped according to the above morphological classes. The classification is simple yet sufficient and until much more is known about the spirochaetes it would be unwise to alter or add to the nomenclature.

Beside the spirochaetes, in Vincent's infection and most cases of gingivitis and pyorrhoea, there are to be found fusiform bacilli, spirilla, vibrios, leptothrices, streptocoeci, and various other irregularly occurring organisms.
FUSIFORM BACILLI.

There appear to be two types of fusiform bacilli found in Vincent's infection although Knighton (13) 1933, claims to have discerned three types by growing them anaerobically on blood agar plates to which gentian violet has been added.

TYPE 1. FUSIFORM BACILLI.

Type 1. fusiform bacilli are about 10 micra long and 2 micra wide, presenting a double contour and slightly tapered ends to reflected light. They are feebly motile organisms and bend a little when moving along. They are always present in appreciable numbers.

TYPE 2. FUSIFORM BACILLI.

Type 2. fusiform bacilli are non motile bacilli about 7 micra long and 0.14 micron wide presenting a single line when illuminated through the dark field condenser. This type can only be located with difficulty in certain cases.

The fusiform bacilli are more resistant to tannic acid and glycerin medication (which is very efficacious in the treatment of acute Vincent's infection) because immediately after such treatment smears show that the

spirochaetes are rendered inactive while the fusiform bacilli remain unaffected.

Fig. 40  Fusiform bacilli, A., from a case of Vincent's infection.

Fig. 41  Fusiform bacilli showing banding. Stained by gentian violet x 660.
SPIRILLA.

The spirilla which occur in Vincent's infection are more curved and slightly shorter but wider organisms than the fusiform bacilli. They are propelled with rapidity by their flagella.

VIBRIONS.

Vibrios are actively motile, small, comma-shaped organisms about 2 micra long, and are propelled with great speed by their terminal flagella.

Fig. 42 Organisms resembling A, Fusiform bacilli; B, Spirilla; and C, Vibrios.
Aisenberg (14) 1933, and von Beust (15) 1929, following on the work of Leon Williams (16) 1899, have studied the morphology of leptothrices seen in smears from the gums. Beust discovered and named the Lepto-thrix calciformis. It apparently grows from a segmented stem which is surrounded by small spherical bodies. At one end of the stem a fructifying head appears. It resembles a mass of pointed bacilli adhering by their ends to the central stem. Von Beust has called them falcate spores and believes they are the fusiform bacilli that are seen in smears from the mouth. Dean et al. (17) 1931, describe the life history of an organism, one stage of which is similar to fusiform bacilli. The pathogenicity of these organisms has not been determined.

(14) Aisenberg, M. S. "Morphologic Studies of Microorganisms of Fusiform Type." The Dental Cosmos, Vol. 75, No. 6, June 1933, Page 546.


Fig. 43  Segmented Stem of a Leptothrix which is surrounded by small Spherical Bodies.

Fig. 44  Broken off portion A, of a Fructifying Head of an Leptothrix Calciiformis.
Many workers are of the opinion that at least some of the spirochaetes are just different life cycles of certain fusiform bacilli. The very thick Treponema buccale and the equally thick Type 1. fusiform bacillus are generally the ones which are considered to be identical.

The spirochaetes appear to be the more delicate of the two groups since they die after 24 or 36 hours on a dark ground slide preparation, while the fusiform bacilli (and the spirilla) remain alive and active.

It is very difficult to say at the present time which is the most destructive of the spirochaetes. Wagener et al. (18) 1930, have not found Treponema vincenti present in smears from mouths having no abnormal gum tissue, and they consider their laboratory diagnosis of Vincent's infection positive only when Treponema vincenti is present as the predominating organism in smears from deep necrotic pockets.

The importance of the part played by fusiform bacilli is not definitely known. They are generally considered to be more facultative anaerobes than the stricter anaerobic spirochaetes. A combination of aerobic and anaerobic organisms as the fusospirochaetal group offer, especially when there are plenty of aerobic bacteria.

acting on the surface, can act symbiotically very well. The aerobic surface bacteria use up sufficient oxygen to make the deeper field, where the fusiform bacilli and spirochaetes abound, a very favourable environment for them.

Sections of tissues prepared by Tunnicliff (19) 1919, which are affected by fusco-spirochaetal organisms show an external necrotic layer, a medium layer composed of masses of fusiform bacilli sometimes in pallisade formation, and spirochaetes, and finally a deep layer of living tissue which is invaded by spirochaetes.

It is not surprising, therefore, in acute cases of Vincent's infection when the spirochaetes actually invade living tissue, to observe from a smear taken from the gum margin, a greater number of fusiform bacilli than usual. The reason is because the smear is only taken from the necrotic and medium layers where the fusiform bacilli abound. The deepest layer, which is very sensitive and vital and which the spirochaetes alone invade, is not touched when making a smear examination. This fact has led many writers to attribute greater pathogenicity to the fusiform bacilli than they deserve.

Of the other organisms occurring with consistency in Vincent's infection and pyorrhoea, one is important

and needs mention. It is a Streptococcus viridans which can always be grown on blood agar showing typical alpha haemolysis. Staphylococci can often be found, but are inconstantly present and never as plentiful as the Streptococcus viridans. Hartzell (20) 1927, firmly believes the Streptococcus to be the first and principal invader. The streptococci, however, can seldom, if ever, be diagnosed from a smear from the gums because of their scarcity compared to the fusospirochaetal organisms, and because of their similar shape to the spherical bodies which break away from the stems of the leptothrixes.

Recently attention has been drawn to the presence of Trichomonas buccalis or Trichomonas elongata (21) in the gingival pocket. Wenrich (22) 1931, has made a study of the taxonomy of this organism and has found its cultural characteristics and morphological appearance similar to Trichomonas vaginalis, but different from the intestinal form. Beatman (23) 1933, has found the

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Trichomonas buccalis present in 15% to 20% of pyorrhoea cases. He does not know if it is pathogenic or not.

That Trichomonas can become pathogenic seems probable. Judd (24) 1931, cites that during a winter he treated ten girls who were suffering from acute vaginitis. In these cases he found Trichomonas vaginalis accompanied by an extremely acrid vaginal secretion.

Graham and Collins (25) 1934, have also noticed the association of Trichomonas in some inflamed conditions of the vagina, and have found that the clinical condition does not clear up until the Trichomonas ceases to inhabit the vagina.

The harm done by Trichomonas in pyorrhoea pockets is not know, but it is certain that it plays no part in the aetiology of gingivitis, because it appears only after pocket formation has commenced. Further Hegner (26) 1928, writing about Trichomonas buccalis states:—

"There is no real evidence, however, that this flagellate is in any way responsible for the pathological conditions with which it is associated."


SECTION NO. 6

PATHOLOGY

OF

GINGIVITIS.
PATHOLOGY OF GINGIVITIS.

The pathology of gingivitis is evidenced by inflammation and infection of the gingiva, especially about its margin and crevice.

The swollen and discoloured epithelium becomes stretched and thin, exposing the sub-epithelial tissues upon the slightest abrasion and causing haemorrhage.

Fig. 45  Chronically inflamed gingiva. Note the papilla, P, of Corium C, extending to the stratum corneum, S.C.
Fig. 46 Inflamed gingiva. Note cellular infiltration at C.I., and benign prolongation of epithelium at E.

Hyperaemia in the corium and nearby deep tissues occurs and persists, but the blood flow in the capillaries and venules of the epithelial papillae and surrounding sub-epithelial tissue about the gingival margin, slows up after a time and lymph exudate and phagocytic cells are attracted to the inflamed tissue. Stasis or at least congestion in the sub-epithelial tissue near the epithelial papillae then occurs. At the margin of the inflammation, and in the deeper tissues, active hyperaemia remains. The two areas, that of congestion about the gingival margin, and that of active hyperaemia in the cemental gingiva can be differentiated by pressure tests. In the former case the marginal gingiva does not show a blanched area after a release of pressure, whereas in the latter case the blood can be readily
driven out of the vessels leaving a blanched area immediately afterwards.

Although this clinical test does not apply to the deeper tissues around the alveolar crest and periodontal membrane, nevertheless, the presence therein of an abnormal amount of blood is manifested by the fact that a subjective symptom of a severe gingivitis is that the teeth feel elongated, sore to bite upon, and as though they were wedged apart.

NEW THEORY OF POCKET FORMATION.

Should the inciting cause be constantly present or infection established, the deep seated hyperaemia will result in decalcification (1) of the alveolar crest and deprivation of calcifying material for the cementoblasts and osteoblasts which are functioning in the zone of hyperaemia. The consequence is that new periodontal membrane fibres are unable to gain attachment to the cementum, and so replace the older degenerating ones.

The epithelial attachment which physiologically grows down the cementum is stimulated to greater activity because of the loss of sufficient underlying support and sends finger-like processes of epithelium over the cemental end of the periodontal membrane.

It does not obtain the firm attachment that it normally would attain, because it depends upon the reduced

periodontal membrane for its attachment, and this membrane in turn is itself defectively attached by old fibres which readily become detached from the cementum. Before a firm attachment can be obtained the epithelium has to grow down over the periodontal membrane until it covers a band of periodontal membrane which has a firm attachment to the cementum. Such an area is not found until the advancing epithelium is out of the zone of hyperaemia. The epithelial downgrowth then attaches itself upon the reduced periodontal membrane, and so gains its organic attachment once more to the tooth. The free margin of the gingiva does not progress rootwise nearly so rapidly, if at all, and therefore a periodontal pocket is formed between the deepened crevicular mucous membrane and the tooth.

Willman (2) 1933, writing on periodontal diseases states that the epithelial attachment irritated by calculus grows down upon the cementum until it comes in contact with the periodontal fibres. He adds, "As the alveolar crest is resorbed, owing to the increase of fluid pressure in the tissues about it, the upper-most fibers of the periodontal membrane are released and cease to function. When this occurs, the epithelium is enabled to grow or extend its attachment rootwise, and since it seems

bound by some obscure law to maintain a fairly uniform average width, it detaches from the tooth surface a corresponding distance above. In other words, the epithelial attachment migrates bodily down the side of the tooth as soon as a few periodontal membrane fibers are gone."
The hypothesis that the "attachment migrates bodily down the side of the tooth" is anomalous, and no evidence has ever been brought forward to substantiate it. In the light of the writer's theory of the epithelial attachment to the cementum, its downward growth is easily explained: It gains fresh attachment by sending finger-like processes of epithelium over the periodontal membrane, which contract in towards the cementum and gain attachment to the reduced cementoblastic layer. It depends upon the presence of newly formed periodontal membrane fibres for its attachment, and therefore it grows down upon the root not so much because of the opportunity afforded, as for the vital necessity for a proper attachment.

Gottlieb, whose statements on dental histopathology are much respected in scientific circles voices the opinion of most histologists when he says (3), "As long as connective tissue fibers are anchored in the surface of the cement below the epithelium attachment, a downward growth of the latter is obviously out of the question."
The writer agrees that the epithelial attachment

cannot penetrate the periodontal membrane to gain a deeper attachment; but hastens to explain that epithelium cannot itself attach to cementum at any time, but requires an intermediate layer of reduced cementoblasts to act as a cementing layer. An epithelial downgrowth, therefore, actually starts to occur when it is in contact with healthy periodontal membrane.

During the process of reattachment of the epithelium the pocket readily deepens over the area where only old degenerated periodontal fibres had existed. The prolonged pocket is readily infected and calculus formation proceeds. Again a hyperaemia is produced in the deep sub-epithelial tissues and a vicious circle of events is completed causing the pocket to progressively deepen.

The pathology at this stage has passed from that of gingivitis to complex periodontitis. Secondary infections of the pocket occur, and many leucocytes migrate by diapedesis through the capillaries into the gingival crevice and there die after fulfilling their scavenger duties. The accumulation of these white blood cells, shed epithelial tissues and bacteria, constitute the pus which is so often seen. Bacterial toxins emitted from organisms which live in such a favourable environment as is offered to them in the secluded spaces of the gingival crevice, probably have their toxicity enhanced. These toxins in turn further affect the surrounding
tissues more especially as the epithelium lining the inner side of the gingival crevice is so thin. Eventually should the irritation and bacterial infection be severe enough, the supporting structures of the tooth are so weakened that the tooth becomes loose and may migrate away from its normal position, or not get its proper physiological wear causing traumatic injury to itself and to its occluding teeth. Tooth migration with consequent irregularity of the arch formation, prevents the supporting structures of the tooth from receiving their share of normal massage arising from the mastication of food. The lack of physiological massage is likely to cause further degeneration due to dis-use; thus affording more favourable opportunities for bacterial invasion.

In acute Vincent's infection, not only is the gingival trough affected but also the external surface of the gingival papillae and surrounding gingiva, causing oedema of the corium, necrosis of the covering epithelium, haemorrhage and other cardinal signs and symptoms. The invading organisms may attack any part of the mucous membrane of the mouth or pharynx but not the exposed lips, forming a necrotic false membrane, which is accompanied by ulceration of the sub-epithelial tissues. The ulcerated surfaces contain a grayish white pseudomembrane of necrotic epithelial structures, lymph exudate, and bacterial detritus.

Considering the devastating nature of the disease
there is an extremely poor emigration of leucocytes into
the seat of infection and consequently very little, if
any, pus is seen.

Should the infection progress further the alveolus
and the deeper tissues are exposed, and the toxaemia
from the devastated areas may cause death.

Because of the accompanying pain patients seldom
allow the infection to spread too deeply without seeking
treatment which is generally very effective.

After recovery from the acute stage the tissues are
so deformed that a chronic stage of gingivitis is likely
to follow with similar pathological sequelae as described
above under gingivitis.

The pathology of hypertrophic gingivitis is mani-
fested by excessive overgrowth of connective tissue
probably due to the influence of toxins or other irrit-
ants. Muir (4) 1933, writing on hypertrophy generally
states that one effect of a toxic agent is that it leads
to a reactive overgrowth of the more resistant connect-
ive tissue cells. The process of hypertrophy may be
considered more as a favourable reaction to irritation
than otherwise. As pointed out by Kritschevsky and
Seguin (5) the dental ligament (which is a band of fib-

(4) Muir, R. "Text-Book of Pathology." Page 146, Third

(5) Kritschevsky, B., and Seguin, P. "The Unity of
Spirochetoses of the Mouth." Dental Cosmos LXVI. Pages
511 and 622, May and June, 1924.
rous connective tissue connecting the gingiva to the neck of the tooth) acts as an effective barrier to the invasion of such organisms as the spirochaetes. Such being the case a hypertrophy of the sub-epithelial tissues must act as an effective defence against the invasion of spirochaetes also.

The epithelium covering the hypertrophied fibrous tissue becomes hypertrophied itself and shows signs of chronic irritation. There is seldom any true pocket formation and the periodontal membrane is rarely severely affected. The hypertrophied tissue, because of its bulk and its apposition to the crown of the tooth, may form a false pocket which readily becomes infected and foul with decaying foodstuff.

A form of trauma worthy of mention, because of its indirect bearing on gingivitis, is that due to an excessive strain on a tooth whereby the periodontal membrane and alveolus (usually at its margin) are injured to such an extent that they necrose.

The necrotic area may be either undermined, absorbed by osteoclasts and replaced by new tissues, or may become infected, and later produce a periodontal abscess. Such infection invariably produces a nearby gingivitis.
SECTION NO. 7

THE BLOOD, AGE AND SEASONAL CHANGES

IN RELATION TO

GINGIVITIS.
THE BLOOD, AGE AND SEASONAL CHANGES

IN RELATION TO GINGIVITIS.

It is hard to ascribe any particular blood picture in cases of gingivitis or complex periodontitis. Many blood counts and analyses have been made by the writer and others, but the counts vary so much that a blood examination seems to be of little value unless either a blood dyscrasia is suspected or a thorough medical examination of the patient can be made to eliminate the possibility of other systemic factors influencing the blood.

To confirm these statements a resume of some opinions concerning the blood conditions in gingivitis and complex periodontitis are given below.

Hartfall (1) 1934, states that bacteriologists are reluctant to attribute any specific blood changes to Vincent's disease.

Many cases of Vincent's infection accompany blood conditions such as agranulocytosis and leukemia, but it is generally considered that the infection is secondary to the blood dyscrasia. This view is held by Thieda (2)

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1934, Vastine (3) 1928 and others.

On the other hand Smith (4) 1932, has produced in guinea-pigs the blood picture of both infectious mononucleosis and agranulocytic angina with fusospirochaetal material. It seems very suggestive, therefore, that the fusospirochaetal organisms may influence the blood in human beings.

Further it is to be remembered that although the fusospirochaetal organisms invade the tissues during acute attacks, producing inflammation at the same time, they apparently exercise a repellant influence upon the leucocytes so that very few of them appear on the surface and consequently there is very little, if any, pus.

Should the fusospirochaetal infection be severe enough, it is quite logical to expect the repressive influence on the leucocytes (which apparently exists) to extend to and influence the haematopoietic organs producing, for example, such a condition as agranulocytosis. Schaefer (5) 1934, from a study of cases in the literature shows that the mucous membrane infection is primary in some cases of agranulocytosis, while in others the


blood condition appears first.

Davis (6) 1934, gives a case history of a patient 65 years of age suffering from agranulocytosis and Vincent's infection where apparently both conditions were noticed about the same time. Dean et al. (7) 1931, report three cases of Vincent's infection where the associated blood dyscrasia in each case may be due to a fusco-spirochaetal blood stream infection.

Rosenthal (8) 1929, has noticed that where Vincent's infection has affected such deep structures as bone, both the red and white cell counts drop.

Appleton (9) 1932, analysing a large number of cases of agranulocytosis concludes that ulcerative lesions are secondary lesions accompanying or following agranulocytosis.

Hume (10) 1932, in the course of an examination of many hundreds of blood films has noticed that the blood


shows a leucopenia with relative lymphocytosis in patients suffering from inflamed gums, gingivitis, pyorrhea and rubber-sore mouth.

Goadby (11) 1931, confirms this statement.

Gingivitis is frequently noticed in patients suffering from leukemia. Cooke (12) 1931, gives a case history of monocytic leukemia where the patient had among other symptoms, bleeding gums.

Vastine (3) 1928, has examined a few cases that were supposed to be acute Vincent's disease, but actually were reactions associated with a late stage of lymphatic leukemia. Pilot (13) 1928, describes the associated stomatitis in patients suffering from acute lymphatic leukemia as being "most remarkable." Davis (14) 1933, studied seven cases of leukemia that came to autopsy. Six showed changes from petechial haemorrhage to marked ulcerative stomatitis.


Hartfell (1) 1934, writing on blood conditions in Vincent's infection says in the majority of cases a mixed lymphomonocytic reaction is encountered which disappears when the Vincent's infection is treated and cured.

Reichmann (15) 1926, writing on Vincent's infection has noticed in a few cases that the number of leukocytes passed 20,000 with 7% to 10% eosinophils. He has also noticed the coagulation time to take from 6 - 12 minutes.

Smith (4) 1932, dealing with fusco-spirochaetal angina gives the following varied blood conditions accompanying it: In the usual case the white cell count is from 8-15 thousand with an increase of polymorphonuclears, but in other cases the blood presents the picture of glandular fever, infectious mononucleosis, agranulocytic angina or pseudo leukaemia.

Kreiger (16) 1928, noticed that every one of a number of cases of Vincent's infection had a peculiar blood picture. One case showed leukaemia. Pettit (17) 1933, studied a case of chronic hypoplastic anaemia which responded with decided improvement to treatment aimed at


the cure of Vincent's infection of the mouth.

Hartzell (18) 1927, dealing with periodontoclasia says "as staphylococci gain in number, the number of polymorphonuclear leukocytes increase. The polymorphonuclear leukocytes are always present with the plasma cells and lymphocytes in purely streptococcal infections, but not in such great numbers as we find them where the staphylococcus is also at work."

Grove and Grove (19) 1933, have found blood serum in cases of pyorrhoea definitely low in phosphorus with a concomitant rise in calcium content. Read (20) 1933, gives tables showing five cases where the blood sedimentation times have improved after the treatment of periodontitis.

AGE AND SEASONAL CHANGES.

Patients of all ages and either sex, providing there are teeth present, may be affected with Vincent's infection. When the term Vincent's infection is used, it is understood to be the acute form of this disease.

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However, there are periods during life when patients become more susceptible to the disease, or at least when the affliction is more likely to occur.

Rosenthal (21) believes Vincent's infection to be a disease of childhood, and Smith (4) gives the periods two to ten, and eighteen to twenty-five years of age as being the most susceptible times. Reichmann (15) presents similar periods shown graphically by a curve which has its peak at about the age of twenty-three with a subsidiary peak at about three and a half years of age. The peak of another curve for chronic cases occurs at about the age of 50. Wright (22) believes there is no age immunity for Vincent's infection, but has found it to exist more often between the ages of eighteen and forty years, and to be more prevalent in the spring and fall than at any other time of the year. Reichmann (15) claims that it is more common during the fall and winter than at other times.

Seasonal changes suggest that the predisposing cause may be dietary because foods and their vitamin content vary so much during the four seasons (23).

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Puterbaugh (24) 1934, quotes Reckford and Baker as having found Vincent's infection most prevalent in the month of February. Puterbaugh thinks it is more prevalent in males than females, and that cigarette smoking plays an important role in providing suitable "soil" for the development of fusospirochaetal organisms.

Of 412 patients treated at the Dental Hospital of Sydney for gingivitis during the years 1933 and 1934, 186 or 45% were males. Taking each year separately the percentage is the same.

These findings are not directly comparable to those of the above mentioned writers, because their inferences concern acute Vincent's infection more so than just gingivitis. Nevertheless, the relationship is close because the urge which prompts almost every patient presenting for the treatment of gingivitis is pain, and pain is an indicative symptom of acute Vincent's infection.

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Fig. 47 Graph showing the number of patients presenting for the treatment of gingivitis each month at the Dental Hospital of Sydney. It appears that the number of patients treated each month, for gingivitis, during the period from May until November is greater than for the remainder of the year.
SECTION NO. 8

DIET

IN RELATION TO GINGIVITIS.

LITERARY SUMMARY.

EPIDEMIOLOGICAL SURVEY.

EXPERIMENTAL INVESTIGATION.
LITERARY SUMMARY.

A vast amount of work has been carried out in the investigation of nutritional deficiencies in relation to dental diseases. Most of the investigations, however, have aimed at the problem of the elucidation of dental caries while comparatively little has been accomplished in the field of dietetics as related to gingivitis.

The constituents of a normal diet are as follows:

1. Proteins.
2. Fats.
3. Carbohydrates.
5. Vitamins.
7. Roughage.

All are necessary for the maintenance of health and for the supply of energy and warmth. A deficiency of any one of the items results after a time in some systemic disorder or cachexia.

From a study of the collected dietetic evidence to date it would appear that the vitamin deficiencies participate to a great extent in predisposing the gingival tissues to infection. Their deficiency may render inert or unassimilable many of the necessary foods which contain compounds, such as those of calcium and phosphorus, or may have a selective pathogenic effect upon certain tissues such as the gingiva.

That a defective diet is in some way responsible for gingival lesions is the belief held by, amongst
others, Goadby (1), Alkin (2) and (3) 1934, Scholes (4) 1916, Cloutier (5) 1933, and McLaurin (6) 1918. In fact Hanke (7) 1932, finds that under a certain dietary regime in which inter-alia, large amounts of orange juice, milk and vegetables are given, most cases of gingivitis which are not associated with calculus, disappear fairly rapidly in from 30 to 60 days. From a clinical aspect, cases of scurvy at least, show an improvement of the infected gingival condition. Falconer (8) 1934, who


is a South African and probably sees more cases of scurvy than we do in Australia says that anyone who has seen the immediate and remarkable improvement in the infected gums of a scorbutic subject following the administration of Vitamin C, cannot have failed to be impressed by it.

It appears that a marked deficiency in any essential ingredient of the diet does not lead to a corresponding compensatory increase in food intake (9). As a result people who eat a lot of concentrated foods or extracts such as sugar, have their hunger appeased without obtaining a natural proportion of mineral salts or food activators which would have been consumed had the food been eaten in a raw state.

The result may be at least a chronic vitamin deficiency.

At the same time the requirements of the body for different proportions of food ingredients varies according to circumstances.

A diet which is adequate at one time may be inadequately balanced under other circumstances with the result that manifestations of food deficiency diseases may appear. Many instances (10), (11), have been


reported where groups of men have contracted scurvy or
Vincent's infection after being put to unaccustomed hard
work, even though their supply of anti-scorbutic foods
has been maintained or increased.

That pregnancy causes a similar dietetic stress as
just instanced in cases of overstrain is suggested by
the work of Day (12) 1933, upon pregnant and non-pregnant
guinea-pigs. He concludes "The changes in the teeth of
the pregnant group were much more pronounced, pointing
to an intensified effect on the teeth of pregnant animals
with comparatively slight deficiencies of Vitamin C."

It may reasonably be concluded that fatigue, exhaust-
on or pregnancy, which calls for the deposition of new,
or the repair of old tissue, tends to precipitate a vita-
min deficiency disorder which would not have occurred
under normal circumstances.

The mineral and alkaline-earth salts are of import-
ance because they play a major part in the formation of
the dental tissues themselves. Should some of the salt
supplies be insufficient, as Lennox (13) believes phos-
phorus to be in certain countries, no amount of food

(12) Day, C. D. M. "The Effect of Anti-Scorbutic
Deficiency on the Pregnant Organism and Dental Tis-

(13) Lennox, J. "Observations on Diet in its Relation
to Dental Disease: How Environment and Civilized
Methods of Cooking Affect the Phosphorus Content of
a Civilized Diet." The Dental Magazine and Oral
activator can rectify the deficiency. On the other hand, should an excess of body building salts, especially calcium and phosphorus, be present in the diet they will not be absorbed unless the necessary and correct amount of vitamins be also present.

The problem remains to ascertain what factors are responsible for the initiation of pathological tissue changes in the gingivae.

A brief resume of some of the work carried out on the vitamin deficiencies is as follows:--

VITAMIN A DEFICIENCY.

Mellanby and King (14) 1934, have demonstrated that Vitamin A deficiency effects the periodontal tissues by producing epithelial hyperplasia, gingivitis and periodontal disease. Degeneration of the maxillary and mandibular divisions of the trigeminal nerve has been noted. By the addition of Vitamin A or carotene, the authors have prevented these conditions. However, they could not correlate the epithelial and nervous lesions resulting from the Vitamin A deficiency. They suggest that the loss of neurotrophic control owing to the vitamin deficiency may be responsible for pyorrhoea, and other diseases of the periodontal tissues.

Nicholls (15) 1934, describing the manifestations of Vitamin A deficiency in the albino rat says that the lining epithelium of the upper respiratory tract shows transformation into a stratified epithelium of flattened cells which undergo keratinization. That a Vitamin A deficiency results in a local infection of the mucous membrane due to the failure of mucus secretion is the conclusion drawn by Cramer and Kingsbury (16) 1924. Goldblatt et al. (17) 1927, have found that metaplastic changes in epithelium to the squamous keratinizing type are as frequent and as great in rats fed on a diet deficient in Vitamin A alone, as on a diet deficient in Vitamins A and D.

McCollum (18) 1932, summarising his knowledge of foods and accessory factors that effect the teeth and their attached tissues, says that lack of Vitamin A results in keratinized epithelium which tends to desquamate.

This factor would lessen the resistance of the


gingival mucous membrane which normally depends upon its presence (19).

VITAMIN B2 DEFICIENCY.

Inflammation of the mouth with sloughing of the mucosa, diarrhoea and skin changes of a nature regarded as analogous to those seen in pellagra have been produced in dogs fed on a diet deficient in Vitamin B2, (18).

VITAMIN C DEFICIENCY.

Scurvy which is due to a total absence of Vitamin C in the diet and appears after about 40 days (18), is manifested (1) in the mouth by haemorrhages and changes in the periosteum of the bones, hypertrophy of the gums which are turgid and bleed easily, generalised periodontitis and loose teeth. Spontaneous haemorrhages occur from the mouth, nose, throat and other mucous membranes of the body. Pilot (20) 1928, has noticed marked changes in the gums of patients with scurvy and says that the stomatitis was the initial symptom noticed in several cases, and that usually the patients first consulted the


dentist on account of the foulness of the mouth and the loose teeth. Further, Vincent's organisms were found in abundance and in one case Streptococcus viridans was recovered from the blood. The antiscorbutic diet given led to a marked improvement in the state of the gums.

Hirschfeld (21) 1931, cites a case of periodontoclasis occurring in a boy 14 years of age. His medical history showed that he had been mal-nourished since birth and showed several systemic symptoms of the scorbutic syndrome. This lad recovered after dietetic treatment coupled with instrumentation.

It has been found by Zilva and Wells (22) 1919, amongst others, that in scurvy occurring in guinea-pigs, well defined changes appear, without exception, in the structure of the teeth before they appear in other organs.

Many microphotographs showing the changes occurring in the pulp on partial and complete scurvy in guinea-pigs, are presented by Hojer and Westin (23) 1925, but they do not show any change in the periodontal tissues.


EPI DEMI O LOGI CAL

SURVEY.

In July 1934, the writer accompanied Dr. F. W. Clements of the School of Public Health and Tropical Medicine, Sydney University, to the island of Manus, Territory of New Guinea, to investigate a suggested relationship of diet to gingival disease.

It was known that the diet of the natives on this island varied in different districts, and that each type of diet was comparatively simple compared with the diets of civilisation.

The main variation of food in the different districts consisted in the presence of either taro or sago in the diet. Seldom were both carbohydrates eaten in the same district.

Taro is the bulb of a lily-like plant of the cassava family, and sago is a food obtained from the pulp of the stem of the sago palm. It is not to be confused with domestic sago which is manufactured from manioc.

The itinerary along the coast and through the inland districts was planned so that contact would be made with natives who lived in the taro and in the sago districts.

To avoid obtaining results from selected groups, word was always sent forward to each village a day before it was to be examined so that all the inhabitants would
Fig. 49  A native taro garden.

Fig. 50  The taro plant is a tuber belonging to the lily family.
Fig. 51  Sago-palm. The stem is about 25 feet high.

be present for inspection.

An examination consisting of the following observations was made: -- Sex, age, condition of the teeth, formation of the arches, state of the gingivae especially observing,

(a) gingivitis in all its forms,
(b) recession of the gingival attachment,
(c) suppurative periodontitis,
(d) presence of tartar and other oral abnormalities.

Those villages in which the natives lived mainly on sago were classed together, as were the villages where
the natives depended on taro for their main sustenance. The natives were closely questioned concerning their foods, and the weights of the constituents of average meals obtained.

The proportion of animal or fish protein to the carbohydrates in the diet varied somewhat as would be expected, but on the average there was a bad balance towards an excess of carbohydrate.

Either taro or sago is eaten twice a day, as a rule, and is frequently supplemented at least once a day with some fish or opossum. Even then the weight of protein is only about one twenty-eighth that of the carbohydrate.

As far as possible samples of the principal foods were obtained, preserved and sent to the School of Public Health and Tropical Medicine, Sydney University, for analysis.

Two factors complicating the results of the oral investigation were the presence of calculus arising from the chewing of betel nut, and the effect of smoking. As far as could be observed these habits were indulged in to the same extent by the various age groups of natives throughout the island.

On the coast the natives spend their time fishing, and barter for either sago or taro from the inland natives who exchange their products for fish. In those districts which are suitable for the cultivation of taro the natives are fully occupied with its production and do not bother about working sago since the sago palm
grows mainly in swampy country which is owned by less fortunate natives. It seems unusual that the natives do not tire of the one carbohydrate. Sago is relished as a food just as much as taro and where the one is plentiful, the other is not bothered about. Those villages which are too far from the coast to barter for fish depend for their animal proteins by catching and eating opossums and marmots.

Other foods which are eaten in very scanty quantity are (a) fruits, such as paw-paw, bananas, lemons, sour-sap, (b) betel nut with daka leaf, and sugar cane, (c) pig (festival time). The occurrence of all these items of diet is more or less universal even though the supply is very limited.

For periods of months on end during certain monsoonal seasons, when no fish can be caught, the food supply is restricted to the staple foods such as sago or taro, as the case may be.

Taro is dug up, its roots cut off, scraped a little and then steamed. It forms a sticky glutinous mass which when cool is picked up by hand and eaten as one would eat an apple.

Its composition which has been estimated at the School of Public Health and Tropical Medicine, Sydney, is as follows: --
**STEAMED TARO.**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>63.6</td>
</tr>
<tr>
<td>Protein</td>
<td>1.70</td>
</tr>
<tr>
<td>Fat</td>
<td>0.41</td>
</tr>
<tr>
<td>Ash</td>
<td>0.94</td>
</tr>
<tr>
<td>Phosphorus (P2O5)</td>
<td>0.119</td>
</tr>
<tr>
<td>Calcium (CaO)</td>
<td>0.032</td>
</tr>
<tr>
<td>Starch and Sugar</td>
<td>33.199</td>
</tr>
</tbody>
</table>

100.000

---

**Fig. 52**  Taro ready to be steamed.

The vitamin content of taro has been estimated at the Bernice P. Bishop Museum, Honolulu, and is stated to contain "Twice as much Vitamin A as white potatoes, and as much as cabbage; and the Vitamin B content is half that of whole grain (including embryo), and twice as much as milk."

This means that taro is rich in Vitamin A and fairly so in Vitamin B.
Sago is obtained by pounding up the pulp of the stem of the sago palm, washing and straining the fibre so obtained with water and collecting the sediment which is dried, wrapped in green palm leaves and stored away.

Fig. 53  The stem of the sago palm is split open and the pulp is pounded into small fragments.
Fig. 54 The comminuted pulp is carried to a washing, filtering and sedimentation apparatus (all made from parts of the palm) where the coarse fibres are filtered away from the finer particles.

Fig. 55 The filtrate, collected in a reservoir, is allowed to settle and the supernatant liquid is decanted off.
Fig. 56  The collected sediment (sago) is dried, tightly wrapped in leaves, and stored away. When needed it is mixed with about one-sixth of its weight of ground up cocoanut, cooked by mixing constantly over a fire and when cool enough eaten by taking a handful, compressing it into a lump, and placing it in the mouth.

Fig. 57  A lump of sago.
Fig. 58  The sago is broken up.

Fig. 59  Cocoanut is ground up and about one part of it is mixed with five parts of sago.
Fig. 60  The sago and cocoanut are stirred together and roasted over a fire.

Fig. 61  When cool, a handful of sago mixture is taken and placed in the mouth.
Sago consists essentially of starch. Its occurrence in the stem of the palm rather precludes any possibility of its possessing vitamins, because in plantlife vitamins are usually confined to the germinating centres and leaves.

Fig. 62  Granules of sago resembling starch; x 100 approx.

The composition as analysed at the School of Public Health and Tropical Medicine, Sydney, (24) was found to be protein 1.2%, fat 0.6% and carbohydrate 83.5%. The biological test for vitamins failed to reveal any vitamins in measurable quantities.

Natives living on a sago diet consume from 1½ to 2 ozs meat or fish to every 28-30 ounces of sago at a meal. There are two meals a day so that from 3 to 4 ozs. meat are consumed with 56 to 60 ozs. of sago. Assuming that 33% of the weight of dried fish or meat is protein, then the protein:sago ratio would be approximately 1:56. Dried sago is about half the weight of prepared sago so that the protein:carbohydrate ratio would be 1:28. The amount of fat consumed is about the same as the amount of protein, making the protein:fat:carbohydrate ratio 1:1:28 or 100:100:2,800. A committee of the Royal Society has laid down as a proper diet for an average man the following ratio:-- protein:fat:carbohydrate :: 100:100:500.

Since taro contains some protein the ratio would be a little better for those natives living on it, but even so it may be safely estimated that all the natives eat at least five times more carbohydrate in proportion to protein than they should.

A comparison of the two classes of carbohydrate diet eaten by the islanders, shows that although both are badly balanced because of a deficiency of protein, the one in which taro forms the basis, contains amongst other vitamins a sufficiency of vitamins A and B, whereas the other in which sago forms the basis, appears deficient in vitamins A and B, except for some contained in the ground-up cocoanut eaten with the sago, along with a very scanty amount contained in other foods occasionally
eaten. As the coconuts only constitute about one-sixth of the total amount of the sago, the sago diet must be considered to contain an insufficient amount of vitamins.

From amongst other sources, Vitamin C is obtained from pepper leaf which is very rich in the vitamin, and is eaten with betel-nut.

Mineral salts are supplied in sufficient amounts in the fish or flesh eaten, and in the calcined coral which is chewed with the betel nut.

**ANALYSIS OF CALCINED CORAL.**

<table>
<thead>
<tr>
<th>Substance</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium oxide (CaO)</td>
<td>62.88</td>
</tr>
<tr>
<td>Phosphorus (P2O5)</td>
<td>0.56</td>
</tr>
<tr>
<td>Carbon dioxide (CO2)</td>
<td>20.90</td>
</tr>
<tr>
<td>Water</td>
<td>16.26</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100.00</strong></td>
</tr>
</tbody>
</table>

The significant difference of the two classes of diet appears to be in the different vitamin contents of taro and sago. This does not mean to imply that the natives living on sago do not get any similar vitamins to those living on taro, but rather that the amount of vitamins they consume are deficient when compared with the amount ingested by the natives living on taro.

Table No. 1 presents the occurrence, in cases examined during the investigation, of the oral conditions pertaining to the gingivae, in absolute numbers and in percentages.

There are about 15,000 natives living on the island
<table>
<thead>
<tr>
<th>DIET</th>
<th>Total Number</th>
<th>Recession of Gingival Attachment</th>
<th>Crowding of Teeth</th>
<th>Traumatic Occlusion</th>
<th>Calculus Formation</th>
<th>Chronic Gingivitis</th>
<th>Acute or Sub-Acute Vincent's Infection</th>
<th>Suppurative Periodontitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAGO</td>
<td>1020</td>
<td>290=28.43%</td>
<td>236=23.14%</td>
<td>47=4.61%</td>
<td>222=21.76%</td>
<td>221=21.67%</td>
<td>52=5.10%</td>
<td>278=27.25%</td>
</tr>
<tr>
<td>TARO</td>
<td>956</td>
<td>255=26.67%</td>
<td>160=16.74%</td>
<td>8=0.84%</td>
<td>183=19.14%</td>
<td>236=24.69%</td>
<td>14=1.46%</td>
<td>145=15.17%</td>
</tr>
</tbody>
</table>

**Significant Difference**
- No
- Yes

**NOTE:** For an analysis of this table see appendix at end of section.
of Manus, and of these about 2,000 were examined. As far as possible such villages as would afford average conditions of living and an equal number of taro and sago eating natives, were visited. Accordingly some villages on each of the coasts and inland were inspected. It is, therefore, thought that the natives who were examined offer a good sample of the total population. Particular care was taken to examine every native who lived in each village visited so that a selected grouping would not enter into the problem.

On the evidence produced it may be taken that the percentages stated in the table show the probability of each of the conditions mentioned, occurring in all the natives throughout the island.

The results have been tabulated into two main groups, viz., those natives living on taro as their staple diet, and those living on sago.

Applying the statistical Chi squared test, advised by Pearl (25) for similar tables, to the distribution of mal-conditions of the gums, in the sago and taro eating natives, it is found that the distributions are significantly different because such an occurrence would happen fewer times than 1 in 1,000,000 by chance alone.

The most apparent differences are in the incidence of crowding of the teeth, traumatic occlusion, acute or 

sub-acute Vincent's infection and suppurative periodontitis. The occurrence of each of these conditions is significantly different in the taro eating and the sago eating natives.

**Fig. 63**  A typical mouth showing normal healthy gums.

**Fig. 64**  Case showing crowding of the teeth and traumatic occlusion. There is a slight gingivitis present.
Crowding of the teeth and traumatic occlusion are factors which may predispose to any gingival disturbance, and the higher incidence of these phenomena may be due, in part at least, to the different consistency and manner of eating taro and sago. Taro has approximately the consistency of a firm pear and is eaten by holding it in the hand and biting pieces out of it. Thus the incisor teeth are exercised and the gingivae are stimulated each time a bite is taken. Sago is prepared in such a manner that it consists of very small particles, and is placed into the mouth by the hand as one would place a heap of bread crumbs, so that there is no resulting incisal action of the anterior teeth. Perhaps this may seem a minor point, but when it is remembered that the major function of the incisor teeth is to incise and bite into food, the importance of this exercise must be realised.
Fig. 66  Taro is held in the hand and eaten like an apple.

Fig. 67  Sago is placed in the mouth, like a heap of bread crumbs and does not need to be bitten into.
Fig. 68  Acute Vincent's infection about the deciduous incisors. Note the necrotic interdental papilla of gum between the upper central incisors.

Fig. 69  Sub-acute Vincent's infection of the upper incisors.
Fig. 70  Crowding is seldom marked in the deciduous teeth, but at this child's age the upper teeth should have separated to allow room for the larger permanent teeth. Note crowding of lower permanent incisors.

Fig. 71  A chronic form of Vincent's infection showing hypertrophied marginal gingivae.
Although sago, which is mixed with a little ground-up cocoanut during its preparation prior to roasting, is in a comparatively fine state when placed in the mouth for mastication, nevertheless, because of the cocoanut present, it is tough and fibrous, and requires a side to side masticatory movement for its further comminution prior to swallowing. Taro, on the other hand, is not tough and requires only a more or less up and down crushing movement of the teeth for its mastication prior to deglutition. Pickerill (26) 1923, has shown that a tough diet requiring the excessive use of the internal pterygoid and mylo-hyoid muscles early in life, produces a narrow arch and causes crowding of the anterior teeth. This phenomenon probably explains the reason for the greater incidence of crowding seen in the sago eating, than the taro eating natives.

Crowding of the teeth and traumatic occlusion are often closely related and the latter is usually the result of such crowding, more especially, as is the case with these natives, traumatic occlusion is in almost every case only present in the anterior teeth where crowding occurs. At the same time it must be remembered, that traumatic occlusion, even in the presence of a crowded arch, cannot be present if a sufficient amount of wear has occurred.

As regards traumatic occlusion, especially in the older natives, a complicating factor of betel-nut chewing arises. In some of these cases traumatic occlusion of the anterior teeth occurs even though these teeth are regular. The reason for the occurrence of such traumatic occlusion is that lime (calcined coral) is placed, by means of a spatula, on to the occlusal surfaces of only the molar and premolar teeth while the betel-nut is being chewed. The lime is used to neutralise the acidity of the nut juices, but it is an abrasive and wears the posterior teeth down leaving the anterior teeth comparatively long so that they clash during mastication and cause occlusal trauma.

It is noticed that in both groups there is a high incidence of gingivitis, although the difference is not significant. Several hundred smears were made from the gum margins of these cases, and the fusco-spirochaetal organisms were invariably found, although there was no evidence that acute symptoms of Vincent's infection had ever occurred. It is almost certain that these cases of gingivitis supply the source of the organisms which accompany and probably produce acute Vincent's infection when the dietetic and local conditions are such that the tissue resistance is lowered.

From the table it will be noticed that there is a far greater incidence of acute or sub-acute Vincent's infection in the sago eating natives than in the others.
The significant difference in the occurrence of the infection cannot be fully explained by the different physical structures of the foods, because cures of the infection can be effected by the administration of large doses of orange juice, and therefore some dietetic deficiency must be present. Its sporadic occurrence in isolated areas indicates that it may be due to a constitutional disturbance, which is probably dietetic in origin. The writer does not know of any other changing influence which can be correlated with the infection. The causative organisms are present in quite a high percentage of mouths, and apparently await a favourable opportunity for invasion when a dietetic insufficiency occurs which is of sufficient gravity under the circumstances to undermine the resistance of the periodontal tissues.
APPENDIX.

STATISTICAL ANALYSIS OF TABLE NO. 1.

The tests of significance between two different proportions have been calculated according to suitable formulae given by Yule (27). When a difference between two proportions has been three times its own standard deviation, it has been considered significant.

1. Recession of Gingival Attachment: The difference between the percentages is 1.66% which is 0.83 times the standard deviation of the difference, and therefore is not significantly different. Such a different percentage could have occurred from errors of simple sampling.

2. Crowding of Teeth: The difference between the percentages is 6.4% which is 3.55 times its standard deviation, and therefore significant.

3. Traumatic occlusion: The difference between the percentages is 3.77% which is 5.09 times its standard deviation, and therefore significant.

4. Calculus Formation: The difference between the percentages is 2.62% which is 1.44 times its standard deviation, and therefore not significant.

5. Chronic Gingivitis: The difference between the percentages is 3.02% which is 1.59 times its standard deviation, and therefore not significant.

6. Acute or Sub-acute Vincent's Infection: The difference between the percentages is 3.64% which is 4.50 times its standard deviation, and therefore significant.

7. Suppurative Periodontitis: The difference between the percentages is 12.08% which is 6.54 times its standard deviation, and therefore significant.
EXPERIMENTAL INVESTIGATION.

A resume of the literature on dietetics in relation to gingivitis and the result of the epidemiological survey carried out in New Guinea by the writer, point to the fact that if there is a dietetic deficiency responsible for katabolic changes occurring in the gingivae, then the deficiency is probably due to either a partial lack of vitamins A, B2 or C, or some combination of these when possibly the protein carbohydrate ratio is badly balanced towards an excess of carbohydrates.

Accordingly some animal experiments to ascertain or suggest which of the above deficiencies might be responsible for the predisposition towards gingivitis were commenced.

Up to the present the following deficiencies have been worked upon at the Dental Research Laboratories of the United Dental Hospital, Sydney:--

A.

(i) Vitamin C deficiency in guinea-pigs.

(ii) Vitamin C plus vitamin B2 (partial) deficiency in guinea pigs.

(iii) Vitamin B2 (partial) deficiency in guinea-pigs.

B.

Vitamin B2 deficiency in rats.
(i) Vitamin A (partial) deficiency in rabbits.

(ii) Vitamin C deficiency in rabbits.

(iii) Vitamin A (partial) plus vitamin C deficiency in rabbits.

Owing to the small number of animals used, the results obtained in the following experiments are only suggestive of what may occur normally under such conditions. For that reason the experiments have not been dealt with at length.

......

A.

Dietetic experiments were performed upon guinea-pigs to ascertain whether any change occurred in the periodontal tissues, when a diet deficient in vitamin C, vitamin B2, or a combination of vitamins C plus B2, was fed to them.

(i) A group was fed the following diet used by Eddy (28), which is deficient in vitamin C:

Baked skim milk . . . . . 30%
Butter fat . . . . . 9%
Sodium Chloride . . . . 1%
Rolled oats and wheat bran of each, equal parts . . . . 59%
Cod Liver oil . . . . . 1%

Plus a certain amount of yeast fed separately.

.............

In place of the yeast whole "Vita B" which is a preparation containing an abundance of vitamins B1 and B2, was substituted.

(ii) A group on a vitamin C plus B2 deficient diet received the above diet except that 1 c.c. of an extract of "Vita B" containing no vitamin B2, was substituted in place of the whole "Vita B".

(iii) A group on a vitamin B2 deficient diet received a similar diet to the above (Eddy) except that no "Vita B" but 1 c.c. of extract of "Vita B" together with 3 c.c.s. of orange juice per day (to supply vitamin C) were administered.

A control group received a similar diet to the Eddy one except that 3 c.c. of orange juice per day were administered (by pipette).

Because of a slight amount of vitamin B2 contained in wheat-bran it must be understood that only a partial

.................

* The Vita B extract is prepared by placing 200 G of "Vita B" in about 250 c.c. of 92% (by weight) of alcohol. This is shaken for half an hour and then filtered. Another 250 c.c. of 92% alcohol is added to the residue and shaken for a quarter of an hour and then filtered. This latter procedure is repeated once again. The filtrates are added together, and the alcohol evaporated off over a hot water bath. The residue is collected and sufficient distilled water is added to make 200 c.c. of extract. 1 c.c. of extract is therefore equivalent to 1 G of Vita B.


It may be concluded, therefore, that the above extract of "Vita B" contains vitamin B1, but no vitamin B2.
deficiency of vitamin B2 is implied when that deficiency is mentioned.

The experiment lasted for 22 days in the cases of groups 1 and 2, and for 9 weeks in the case of group 3. A control guinea-pig was used for groups 1 and 2, and another for group 3.

The effects of the experimental diet upon the periodontal and contiguous tissues were that:

1. Vitamin C deficiency produced a scar tissue instead of enamel.
Fig. 72  Dental and Periodontal Tissues of Guinea-pig on the Vitamin C deficient Diet showing Scar Tissue S.T., where Enamel should be. The Ameloblastic layer has disappeared and the Periodontal Membrane, P., is attached to the Scar Tissue; D, Dentine.

Fig. 73  Similar tissues to those shown in Fig. 72 of control Guinea-pig. Note that the Enamel must have been properly calcified, because it has been entirely decalcified during histological preparation leaving a clear Enamel Space at En; D, Dentine; Am, Ameloblasts attached to Periodontal Membrane, P.
2. Combined vitamin C (complete) and vitamin B2 (partial) deficiency produced changes in the squamous cell layer of the gingival mucous membrane so that large vacuoles appeared near the stratum corneum.

Fig. 74 Guinea-pig, Vitamin C (complete) and B2 (partial) deficiency, showing large Vacuoles, V, in the superficial parts of the Epithelium near the Gingival Crest; D, Dentine.

Fig. 75 Guinea pig, control diet, showing normal gingiva.

Fig. 76 Guinea-pig, Vitamin B2 partial deficient Diet, showing sub-periosteal Haemorrhage, H; D, Dentine; F, Periodontal Membrane.

B.

Vitamin B2 deficiency has been investigated in rats. A group of rats were fed on a vitamin B2 deficient diet as follows:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Purified casein</td>
<td>18 parts</td>
</tr>
<tr>
<td>Osborne and Mendell</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>salt (29)</td>
<td>8 &quot;</td>
</tr>
<tr>
<td>Butter fat</td>
<td>2 &quot;</td>
</tr>
<tr>
<td>Cod-liver oil</td>
<td>1 c.c.</td>
</tr>
<tr>
<td>Corn starch</td>
<td>68 &quot;</td>
</tr>
<tr>
<td>Extract of &quot;Vita B&quot;*</td>
<td>1 c.c.</td>
</tr>
</tbody>
</table>


*See foot note on Page 151.
As a control another group was fed a similar diet to the above, except that the extract of "Vita B" was replaced with whole "Vita B" fed separately.

The rats were maintained on this experimental diet for 17 weeks.

Those rats on the vitamin B2 deficient diet showed (1) a large number of interglobular spaces in the dentine (which signifies irregular calcification) and (2) fewer osteoblasts present in the periodontal membrane, at its connection with the alveolus.

The rats in the control group did not show any of the above changes, and the periodontal tissues appeared normal.
Fig. 77  Rat, Vitamin B2 deficient Diet.  D, poorly calcified Dentine;  P, Periodontal Membrane fibres adjacent to Alveolus A showing absence of Osteoblasts.

Fig. 78  Rat, Vitamin B2 control Diet.  D, well calcified Dentine;  P, Periodontal Membrane fibres adjacent to Alveolus A showing a normal number of Osteoblasts.
C.

Experiments in which it was hoped to produce a vitamin A, a vitamin C, and a combined vitamin A and C deficiency, in rabbits were begun. It was found that the rabbits would not eat an experimental diet similar to that recommended by Findlay (30) namely,

<table>
<thead>
<tr>
<th>Basal Diet:</th>
<th>Casein</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Starch</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>Yeast ext.</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Salt mixture</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Olive oil</td>
<td></td>
</tr>
</tbody>
</table>

unless some oats equal in amount to about a third of the total diet were added.

The above diet would therefore only supply a partial deficiency in vitamin A because oats contain a small amount of this vitamin.

The rabbits were divided into four groups:

Group 1 -- vitamin A (partial) deficient diet, received the above basal diet together with onions to supply vitamin C.

Group 2 -- vitamin C deficient diet received a similar diet to the above basal one, except that cod liver oil was substituted in place of olive oil.

Group 3 -- combined vitamin A (partial) and vitamin C deficient diet, received the basal diet alone.

............... 

Group 4 -- control diet, received a similar diet to the basal one, except that cod liver oil was given in place of olive oil, and onions were added to the diet.

Two of three rabbits in the vitamin C deficient group died after five weeks, and the experiment was ended at the close of the seventh week.

In this experiment the only changes observed occurred in those rabbits which belonged to the vitamin C deficiency group, where a slight degeneration of the crevicular epithelium shown by vacuoles appearing in the cells, was noted.
Fig. 79 Rabbit, Vitamin C partial deficient Diet. Ep, Epithelium showing Vacuoles in the outer layer; D, Dentine; En, Enamel Space; D.C., Dental Cuticle.

Fig. 80 Rabbit control diet, showing normal gingival tissues; Ep, Epithelium; D, Dentine; D.C. Dental Cuticle.
SECTION NO. 9

THE SALIVA AND CALCULUS

IN RELATION TO GINGIVITIS.
SALIVA.

The saliva is nature's mouth wash and liquid cleansing agent. It consists mainly of water and contains two valuable ferments—ptyalin and maltase. The ferments act upon starch converting it into maltose (a disaccharide) which is then hydrolysed to form glucose or dextrose. The action of the ferments upon food during mastication is very slight, because of the limited time available for fermentation before the food passes to the gastric juices. From a dental aspect, however, the ferments are important mainly because of their cleansing function after a meal when they convert otherwise insoluble food-stuff which adheres to the teeth and gums into soluble substances which are washed away. Bennett (1) 1914, gives one analysis in 1,000 parts by weight of saliva as follows:

<table>
<thead>
<tr>
<th>Component</th>
<th>Parts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>994.203</td>
</tr>
<tr>
<td>Mucin</td>
<td>2.202</td>
</tr>
<tr>
<td>Ptyalin</td>
<td>1.390</td>
</tr>
<tr>
<td>(Carbonates</td>
<td></td>
</tr>
<tr>
<td>)Phosphates</td>
<td></td>
</tr>
<tr>
<td>Salts</td>
<td></td>
</tr>
<tr>
<td>(Chlorides</td>
<td>2.205</td>
</tr>
<tr>
<td>)Sulphates</td>
<td></td>
</tr>
<tr>
<td>(Nitrites</td>
<td></td>
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</tbody>
</table>

Pickerill (2) 1923, states, "The chief constituents (of


saliva) from a dental protective aspect are:—water, the alkaline salts of calcium and sodium, the phosphates of calcium and magnesium, ptyalin, sulphocyanate of potassium, sodium chloride and mucin." He concludes (3), "That practically all the normal constituents of saliva are, if present in sufficient amount, of value and importance in protecting the teeth against the occurrence of dental caries, and in maintaining the health of the oral mucous membrane."

Broderick (4) stresses the importance of the hydrogen ion concentration (pH) of saliva as being a good index of the tendency of the body tissues to become either alkaline or acid. The blood pH remains remarkably constant, but should there be a tendency for it to become acid it is first shown in the saliva. The blood contains buffer salts to keep it at a constant hydrogen ion concentration of about 7.2; should, however, these salts become saturated, either on the acid or alkaline side, and the blood pH alter at all as a consequence, death would occur. Broderick and others have found that the saliva in cases of gingivitis or pyorrhoea gives an alkaline reaction, whereas in cases of rampant caries the reaction is acid.


Normal patients give either a neutral reaction for their saliva or a pH about 6.8. The writer (5) 1932, examined the salivary pH of 140 patients with the following results:—Average pH of saliva in cases of suppurative periodontitis 7.325, average pH of saliva in normal mouths 6.35, and average salivary pH of patients showing tendency towards dental caries 6.37.

Hawkins (6) 1931, has found that active pyorrhoea presents an acid saliva and anaemic pyorrhoea an alkaline one.

The pH reaction of the saliva is important in that its influence may enhance the growth of various types of bacteria or protozoa, or precipitate certain calcium salts to form salivary calculus.

Price (7) describes a case of pyorrhoea where the saliva absorbed calcium and phosphorus when mixed with powdered bone. After nutritional treatment with fat soluble activators the saliva actually gave up calcium and phosphorus to the powdered bone.

..............


Turner (8) 1934, thinks that mucus is the secretion of importance in pyorrhoea, and that the saliva is of little consequence.

The writer was very hopeful at one time of obtaining favourable results in the treatment of periodontitis by altering the reaction of the saliva from an alkaline to a neutral or slightly acid one. The method employed was that advocated by Broderick (9) who administered ammonium chloride internally. In the limited number of cases that were treated, no change, which could be attributed to the altered pH value of the saliva, was noticed in the condition of the gingivae.

Although an alkaline saliva under certain circumstances is still considered a possible contributing aetiological factor in gingivitis and periodontitis, the writer has abandoned the treatment for the correction of the salivary reaction because of its insignificant effect.


CALCULUS.

Calculus consists essentially of calcium phosphate and calcium carbonate, food debris, micro-organisms and epithelial scales.

Bennett (10) gives the following analysis for soft molar calculus:

- Water and inorganic matter ........ 21.48 parts
- Magnesium phosphate ............ 1.31 "
- Calcium phosphate with a little carbonate and fluoride ........ 77.21 "

There are two varieties of calculus:

1) Salivary
2) Serumal

1) Salivary calculus is soft, chalky, and a light yellow to grey in colour. It forms principally upon the lingual surfaces of the lower incisors and the buccal surfaces of the upper molars. Generally its removal is easy. It has the tendency to cling to the crown and neck of a tooth and to cover over the gum which it irritates but slightly, rather than to precipitate and form in the gingival trough.

Because of its bulk it tends to push the gum away from its attachment to the crown and root of the tooth.

until such time as the tooth becomes loose and is exfoliated. During the process, however, there is seldom any pocket formation. The mode of its formation is not definitely known, but it would appear that calcium phosphate is held in solution in saliva by the latter's carbonic acid content. After the secretion of the saliva from the ducts, some carbon dioxide gas is given off with the result that calcium phosphate is thrown out of solution. During meal time the precipitation of salivary calculus does not matter as it is washed away and swallowed with each bolus of food, but during the interval between meals it combines with mucin and micro-organisms and adheres to the exposed surfaces of the teeth.

![Fig. 81 Decalcified section of salivary calculus. Calculus consists of an organic matrix composed of fungi, leptothrisses and detritus, and is deposited in layers forming irregula rly stained lamellas.](image)
Filamentous forms of leptothriices are always found amongst the bacterial flora where calculus exists, and this organism, which is about 30 micra long at times, probably aids calculus formation by being entangled in the precipitate. It then acts as a binding agent.

(2) Serumal calculus is a hard, brittle, dark brown to greenish black coloured mass. The dark colour is due to its being stained with haemoglobin. It adheres very tenaciously to the roots of the teeth, and always forms in a pocket between the gum and tooth.

Goadby (11) describes it as a deep-seated blackish and greenish salivary calculus, which is definitely associated with suppurative periodontitis. He states that the greenish colour is partially due to derivatives of blood pigment and partly to a mixture of iron sulphide.

Bunting (12) 1929, gives the analysis of subgingival calculus estimated by J. H. Salisbury as:--

<table>
<thead>
<tr>
<th>Component</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water and organic matter</td>
<td>32.24</td>
</tr>
<tr>
<td>Magnesium phosphate</td>
<td>0.98</td>
</tr>
<tr>
<td>Calcium phosphate</td>
<td>63.08</td>
</tr>
<tr>
<td>Calcium carbonate</td>
<td>3.70</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>100.00</strong></td>
</tr>
</tbody>
</table>


If this calculus is placed upon a piece of cotton wool, and sufficient pressure with an instrument brought to bear upon its centre, a distinct crack can be heard.

Serumal calculus forms upon the roots of almost any teeth, but especially the upper incisors and the lingual surfaces of the lower molar teeth. It chronically irritates the gums and is regarded by some as the cause of pocket formation.

Its formation is considered by the writer, to originate by the deposition of calcium and other salts from blood which lies in the gingival pocket (due to minute gingival haemorrhages), and from lymph which effuses into the pocket, together with some of the mouth organisms and debris; therefore, it is a result rather than the cause of pocket formation.
SECTION NO. 10

SUMMARY

AND

CONCLUSIONS.
SUMMARY

The following important factors have been dealt with:—

1. The histology of the dental tissues concerned with gingivitis. A new theory has been suggested regarding the gingival attachment to the cementum.

2. Local and systemic factors concerned with the aetiology of gingivitis.

3. An adequate symptomatology of gingival and periodontal diseases; so that an intelligent diagnosis of gingivitis may be made.

4. A study of the apparently important microorganisms concerned in gingivitis and associated conditions.

5. The pathology of gingivitis: A new conception of pocket formation has been presented.

6. The hydrogen ion concentration of the saliva in cases of periodontitis and the formation of salivary and serumal calculus.

7. The aetiological aspect of diet and the two ways in which it may affect the gingivae, viz., (i) mechanically, (ii) biochemically.

8. An epidemiological survey, wherein data on gingival conditions and foods eaten were obtained.

9. Some experimental vitamin deficiencies in guinea-pigs, rats and rabbits.
CONCLUSIONS.

1. Gingivitis is usually caused by a syndrome of factors acting in harmony with each other.

2. It is a very common affection caused by local, as well as in some cases, systemic factors.

3. The organic attachment of the gingival epithelium to the tooth for the most part is a strong one, but during tooth eruption before a given tooth reaches its plane of occlusion the attachment is easily broken asunder.

4. There is no accepted significant blood change attributable to gingivitis.

5. Infection by micro-organisms is probably secondary to katabolic changes first occurring in the gingivae.

6. The fusco-spirochaetal group of organisms are always present in cases of the common form of gingivitis.

7. Figures in an epidemiological survey suggest that acute Vincent's infection is intimately associated with a defective diet.

8. Dietetic experiments on some animals indicate that certain vitamin deficiencies affect dental tissues deleteriously.

FINIS.