A CRITICAL SURVEY OF THE LITERATURE IN PERIODONTIA.

R. L. Mobbs, B.D.S.
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P R E F A C E.

The aim of this work is to present a Summary of the current literature relating to Periodontia, both from text books and the more recent dental periodicals.

Where possible different theories and viewpoints will be compared.
# Contents

<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>The Supporting Structures, Glands of the Mouth, Saliva</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>Histology</td>
<td>10</td>
</tr>
<tr>
<td>III</td>
<td>Etiology</td>
<td>25</td>
</tr>
<tr>
<td>IV</td>
<td>Development of the Periodontal lesion, Gingivitis, Periodontitis, Periodontosis</td>
<td>45</td>
</tr>
<tr>
<td>V</td>
<td>Occlusion and its relation to Periodontal Disease</td>
<td>81</td>
</tr>
<tr>
<td>VI</td>
<td>Dangers of Periodontal Disease</td>
<td>90</td>
</tr>
<tr>
<td>VII</td>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>VIII</td>
<td>Other oral lesions</td>
<td>127</td>
</tr>
<tr>
<td>IX</td>
<td>CONCLUSION</td>
<td>134</td>
</tr>
</tbody>
</table>
CHAPTER I.

THE SUPPORTING STRUCTURES, GLANDS OF THE MOUTH, SALIVA.
CHAPTER I.
GENERAL DESCRIPTION OF THE ORAL CAVITY.

GLANDS OF THE MOUTH, SALIVA.

The oral cavity may be considered to consist of:—

(a). The vestibule and

(b). The oral cavity proper.

(1) Orban divides the oral mucosa into three different types.—

1. The masticatory mucosa which consists of the gingiva and hard palate.

2. Lining mucosa consisting of the mucosa of the lips and cheeks, the vestibular fornix, mucosa of the alveolar processes adjoining the gingiva; the floor of the mouth, inferior surface of the tongue and the soft palate.

3. Specialized mucosa of the dorsal surface of the tongue.

The oral mucous membrane is kept permanently moist by the secretions of the salivary glands.

(2) GLANDS OF THE MOUTH.

Some secrete into the vestibule, others into the oral cavity proper. The smaller glands function continually and keep the mucous membrane moistened and lubricated. The larger glands - the salivary glands proper - secrete by various stimuli. Certain stimuli act on peripheral endings of sensory nerves of the mouth and produce secretion (unconditioned or inherent reflex). Other stimuli act on the organs of special sense such as sight and smell, (conditioned or acquired reflex). All glands of oral cavity are of the compound type i.e., have several or many lobules.

SALIVARY GLANDS.

I. Of Vestibule.
Two parotid glands and numerous small labial and buccal glands in the mucous membrane of the lips and cheeks.

The parotid gland is the largest of the salivary glands and is a serous gland. The secretion is thin and watery usually, but is occasionally turbid. The secretion enters the vestibule via the ducts of Stenson which open opposite the upper second molars.

Parotid saliva contains salts, proteins and ptyalin (salivary amylase). It is well adapted to cleansing the mouth, moistening dry food, and dissolving substances so that taste buds are stimulated.

Reaction of Parotid Saliva given by Box in a study of 200 different Parotid Salivas.—

In 89%, Acid reaction.

7%, Alkaline reaction.

4%, Neutral.

The labial and buccal glands are mixed glands i.e., both mucous and serous.

2. In the Oral Cavity Proper, Maximow and Bloom list the following glands:—

a. GLOSSO — MANDIBULAR GLANDS.

1. Mandibular (submaxillary gland), large gland with its duct opening at the side of the tongue.

2. Sublingual glands beneath the mucous membrane at the side of the frenulum of the tongue.

   a. The large sublingual gland with its duct opening into the duct of the mandibular gland.

   b. Several small glands opening along a fold of mucous membrane — the plica sublingualis.

b. GLANDS OF THE TONGUE.

1. Anterior lingual glands situated at each side of the median line under the apex of the tongue.
2. Posterior lingual glands.
   a. Albuminous or gustatory glands opening into the circumvallate grooves and connected with the circumvallate papillae.
   b. Mucous glands of the root of the tongue.

3. GLANDS OF THE PALATE.

Each submaxillary duct (Wharton's Duct) opens into the mouth through a soft tissue eminence, the salivary caruncle, at the side of the frenulum of the tongue. Usually, each duct of the large sublingual gland enters the mouth through the same orifice. The small sublingual ducts — (8-20 in number) open along the plica sublingualis.

The sub-maxillary gland is a serous and mixed gland, i.e., the majority of the gland contains serous cells while other parts are mixed. Its secretion is clear, rather thin and rather viscid, and contains mucin, salts and ptyalin. Depending upon the nature of the secretory stimulus, the saliva may be thick or thin.

Food stimulates a thick flow, rich in mucin, while irritants cause a flow of thin saliva.

The sub-lingual glands are mixed glands with mucous cells predominating. The secretion is clear and mucilaginous and contains mucin, ptyalin and salts.

The glossopalatine glands at the root of the tongue are mucous glands. The posterior lingual glands are serous and the anterior lingual glands are mucous glands.

Box tested 200 different sub-maxillary salivas (actually mixed sub-maxillary and sub-lingual) and found:

- 44.5% Alkaline.
- 37% Acid.
- 18.5% Neutral.

He says Brawley found an average pH of normal resting saliva of 6.75 in a study of 3,405 cases.
Coolidge and Hine give the following description of saliva:

Specific Gravity. 1.005

99-99.5% water.

0.5-1% solids in suspension or in solution.

Inorganic solids 0.4-0.6% made up of chlorides, carbonates and phosphates of Na, K, Ca and Mg and a small amount of potassium sulphocyanate.

Organic solids 0.1-0.4%. These are:— Mucin, Albumin, Globulin, Urea, Ptyalin.

The reaction of saliva in a resting gland is often weakly acid, the pH range being 6.2-7.4. During stimulation pH rises slightly and after eating a light meal it falls.

Bibby lists these properties of saliva:

1. Demulcent effect.
2. Washing effect.
3. Moistens food.
4. Chemical properties.
5. Hormonal effect.
6. Compatible with the tissues.
7. Enzyme action.
8. Aids clotting time of blood and rapid healing of wounds.
9. Antibacterial effects
10. Selective excretory effect.

The buffer action of saliva enables it to absorb a certain amount of acid or alkali with little noticeable change in chemical reaction. A stimulated saliva has a greater reserve of neutralising power than resting saliva, due to increased flow and amount of inorganic salts excreted. If it is stimulated to maximum flow e.g., in the presence of many carious or sensitive teeth, there is little reserve power for additional
demands when stimulated by substances taken into the mouth. Coolidge and Hine think this may be related to lack of neutralizing power in those susceptible to caries, and also related to freedom from calculus often observed in these.

Defence factors of the mouth.

1. Normal intact mucous membrane.
2. Excellent blood supply and so a good supply of white blood cells.
3. Cleansing effect of mastication and the movements of tongue, lips and cheeks.
4. Swallowing and air currents carrying bacteria out of the mouth.
5. Flushing action of saliva.
6. Possible antibiotic effect of certain oral bacteria against others.
7. Normal anatomy of the mouth.

THE GINGIVA.

The Gingiva is the mucous membrane surrounding the teeth and is subjected to the forces of friction and pressure during mastication. Labially and buccally it is sharply limited at the muco-gingival junction where the alveolar mucosa begins.

Normally the Gingiva is pink and has sometimes a greyish appearance. It is firmly attached to the Periosteum of the alveolar bone. The Gingiva consists of free and attached gingiva, divided by the free gingival groove running parallel to the gingival margin.

It is usually stippled but the degree of stippling may vary from person to person. Absence of stippling would indicate oedema.

The alveolar mucosa is red and small blood vessels are seen near the surface.

On the palate there is no line of demarkation due to the firm attachment of the palatal mucosa.

Corresponding to depressions on the alveolus between
the teeth, the attached gingiva also shows depressions.

In youth the interdental papillae fill the spaces between the teeth.

The gingival sulcus is normally of shallow depth,—the more shallow it is the less likelihood of pathological conditions developing. Orbán says that every sulcus may be termed normal, regardless of its depth, if there are no signs of pathologic conditions in the investing tissues.

(6)

Goldman describes these changes in normal gingivae at different ages.

In childhood — uniform hue, reddish and shiny and only part of the anatomical crown is exposed. The gingiva round a tooth is thick and rounded with a crevice several millimetres deep. The crevice becomes shallower as the tooth erupts.

In youth, occlusion is established and the alveolar process is fully developed. The gingiva ends in a fine margin pinker than childhood and with a somewhat stippled surface.

In middle age there is further eruption of the teeth and some recession of the gingivae. The gingiva is pink and firm and has a shallow crevice.

In old age the teeth are worn down and an edge to edge bite is featured and part of the root has now become clinical crown.

There is a shallow crevice but the gingival margin is more rounded.

(7)

The radiographic appearance of normal tissue would be as follows:— Periodontal membrane very thin and hardly discernible radiographically. In young patients it is thicker. There is an even white line of laminadura and the cancellous or supporting bone is of even density throughout the entire alveolus.
During life the following changes take place:
1. Migration of the epithelial attachment towards the apex of the tooth.
2. Simultaneous atrophy of the alveolar crest.
3. Protrusion of the teeth towards the occlusal line.
4. Mesial displacement of the teeth.
5. Compensating abrasion of the teeth at occlusal surfaces and points of contact.

These are continuous but at a varying rate.
When the rate of wear balances the rate of eruption, the clinical crown remains constant in height.

Linghorne submitted this theory for nature's plan for the dentition.

1. Erupting teeth are guided into position by the muscles of the lips, cheeks and tongue till contact is made.
2. Then the cusps guide the teeth into relationship.
3. Cusps maintain relationship till supporting bone is sufficiently hard and strong to maintain teeth in contact relation without the aid of interlocking cusps.

Box says that in general, modern dentitions show a retarded degree of occlusal wear in early adult life, and that the ideal adult stage of end to end occlusion in the anterior teeth, is seldom achieved. He describes studies by Stein, Cameron, Campbell and Keith who all found early wear of the teeth in prehistoric or primitive men. Both Stein and Cameron describe a typical angle of wear in first molars, the upper first molars being worn down to the gum lingually, and corresponding wear on the buccal aspect of lower first molars. He quotes Stein as saying "sturdy well worn dentitions are as rare among civilized races as they are common among the uncivilized."
It is interesting to read reports of the development of pathological states arising in primitive tribes when they are introduced to a more civilized diet.

The gingivae have a very rich blood supply from periodontal and periostial blood vessels and from vessels in the adjacent mucosa.

The main branch-arteries supplying the tooth and supporting structures divide into two groups, a. the dental arteries and b. the interalveolar arteries. The latter are the larger. The dental arteries are very delicate, and before entering the apex to the pulp, give off small branches which supply the immediate neighbourhood of the apex.

The interalveolar arteries are much larger and pass through the septa to the alveolar crest giving off branches — the alveolar perforating branches, which reach the periodontal membrane through many openings in the lateral wall of the alveolus.

The interalveolar arteries penetrating the alveolar crest give off gingival perforating branches where they divide into capillaries which supply the connective tissue papillae in the gingivae.

Besides this, the lingual and buccal gingivae are supplied by vessels between the epithelium and periosteum of the maxilla and mandible.

The venules from the gingivae drain into the pterygoid plexus, the pharyngeal plexus and the internal maxillary vein; and the lymphatics run to the submaxillary, submental, and deep cervical nodes.
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CHAPTER II.

HISTORY.

During the eruption of the teeth, the connective tissue between the reduced enamel epithelium and the oral epithelium disappears and the two layers of epithelium fuse.

As the crown emerges into the oral cavity the epithelium degenerates. The reduced enamel epithelium remains in organic connection with the unerupted part of the crown.

Having formed the enamel matrix the ameloblasts produce the primary enamel cuticle which is about 1 μ thick, usually becomes calcified, and is connected with the interprismatic substance.

The ameloblasts then shorten and the cells of the enamel organ are reduced to a few layers of cuboidal cells - this is the reduced enamel epithelium and covers the entire crown and extends to the cemento-enamel junction. When the tip of the crown has emerged, however, it is then called the epithelial attachment.

During eruption, the epithelial attachment gradually separates from the tooth surface. Before this separation occurs the secondary enamel cuticle is formed - a hornified layer 2-10 μ thick. Kronfeld says this may be looked upon as a continuation of the keratinous layer of the oral epithelium and though formed in most cases, may be completely missing if the gingival epithelium does not tend to produce a keratinized layer.

The original concept was that the gingival sulcus extended to the cemento-enamel junction. Since Gottlieb's work in 1921 it has been generally accepted that in the young tooth, the bottom of the crevice is not at the cemento-enamel junction but on the enamel.
He described the "epithelial attachment", a band of epithelium surrounding the tooth and in organic connection with Gassymth's membrane. Thus, when the tooth reaches the occlusal plane, about 1/3 or 1/4 of the enamel is still united with the epithelial attachment and this extends to the cemento-enamel junction. Gradually more enamel is exposed by a separation of the epithelial attachment, and the tissue of the free gingiva is reduced by atrophy.

The gingival crevice or sulcus is a shallow groove bounded by the tooth on one side and gingiva on the other. The bottom of the sulcus lies where the epithelial attachment is separated from the tooth. The marginal gingiva is that part lying crownward from the bottom of the sulcus.

That portion of tooth above the base of the gingival sulcus is called the clinical crown and that below, the clinical root. The junction between epithelial attachment and connective tissue is usually smooth. Ortban says it may be considered a sign of irritation if it sends epithelial pegs into the connective tissue.

The attachment of the surface cells to the enamel or cementum is firmer than the attachment of these cells to the deeper (i.e., basal) layer of the epithelium and so tears often occur within the epithelium.

(3)

Studies by Baume with Phase Contrast Microscopy, which allows observation without fixing or staining the section, showed connection between epithelial cells and the primary enamel cuticle by means of tonofibrils.

He points out that with ordinary fixation techniques the tonofibrils disappear. The existence of the tonofibrils depends upon the vitality of the epithelial
cells and if the cells die, the fibrils disappear. Baume also states that in fixed sections a mechanical connection between crown and gingiva could not be demonstrated.

This seems to be the most convincing study yet in favour of the "epithelial attachment".

(4)

On the other hand Waerhaug in his work on the gingival pocket recorded these findings:

1. In autopsy material, the distance from the gingival margin to the deepest point of the epithelial attachment corresponded generally with the clinically measured crevice.

2. After loosening the gingivae in dogs with steel instruments down to the cemento-enamel junction, prepared sections looked exactly the same as a normal "epithelial attachment".

3. By passing a fine, steel blade into crevices round erupting teeth in children and then taking X-rays it was found that the instrument extended to the cemento-enamel junction.

4. Other authors have found remnants of the epithelial attachment on extracted teeth. Waerhaug found that, by pressing a glass slide against the lips, cells adhered to the glass as strongly as they do to extracted teeth. Also he found similar cells attached to artificial crowns which were in contact with the gingiva and a similar phenomenon as has been called the secondary cuticle.

5. He found that the secondary cuticle was easily penetrable by acids and not as protective as is commonly thought.

Waerhaug suggests then, that instead of an organic connection, there is an adhesion of cells to the enamel. He suggests it should be named the "epithelial cuff". The detachment of epithelium from the tooth crown
is by a peeling-off process from the enamel surface. Some think it is caused by tears in the epithelium itself, others, that the sulcus forms at the line of union of enamel and oral epithelium. This would imply that the oral epithelium proliferates at the connective tissue side of the epithelial attachment replacing the enamel epithelium, which degenerates.

Kronfeld says that the gingival crevice is formed by degeneration of the cells of the epithelial attachment and their detachment from the dental cuticle.

The epithelial attachment is composed of stratified squamous epithelium. The basal cells are regular cuboidal cells with round, dark nuclei and these are covered by several layers of flat, squamous cells. Next to the tooth the nuclei are irregular and light.

Before the bottom of the crevice reaches the cemento-enamel junction, the deepest point of the epithelial attachment proliferates and grows down the cementum.

The downgrowth of the epithelial attachment along the root surface is impossible as long as the gingival and transeptal fibres are intact. It seems that the proliferating epithelial cells dissolve the collagen by enzyme action. Another explanation is by destruction by bacterial toxins.

Fish says the epithelial cells themselves cannot destroy these fibres.

The bottom of the crevice is often shown microscopically by the presence of a few inflammatory exudate cells.

The gingival epithelium is stratified squamous type, featuring columnar cells in the basal layer (Orban says they are cuboidal) cuboidal prickle cells in the middle layer and flattening cells in the outer layer.
Here the nuclei shrink, and a vacuole appears around the dead nucleus. Heidin granules appear in the cytoplasm and this is the precursor of keratin. As the cell recedes further from its source of nourishment, the whole cell shrinks, and by a process of dessication becomes converted into a flat scale of keratin on the surface.

According to Robinson, the epithelium lining the gingival sulcus (or a gingival pocket) shows neither keratinization nor parakeratinization.

Orran describes keratinous, granular, prickle cell, and basal layers in the epithelium. The cells of the granular layer contain kerato-hyalin granules, which are basophil, and frequently have pyknotic nuclei. The keratinous layer is acidophil and the nuclei have disappeared. Normally a well hornified layer and a granular layer is seen, but there may be no granular layer and the flat surface cells may contain nuclei which often show pyknosis. There may be little keratinisation here and this is called parakeratosis. All stages between these may be seen.

The lamina propria features long papillae in the gingiva but not in the alveolar mucosa. Corresponding with the free gingival groove, (.5-1.5 mms. from the margin) is a heavy epithelial ridge, and below this groove and down to the muco-gingival junction the epithelial surface is stippled. Absence of stippling may indicate oedema.

**GEMENTUM.**

Gementum is a calcified tissue which covers the root surface. It is produced by the connective tissue of the periodontal membrane by the formation of an organic matrix into which inorganic salts are precipitated.

After the dentine of the root is formed it lies in contact with connective tissue. It is then covered by
a layer of cementum in which fibres of collagen are embedded, the other ends of these fibres are attached to bone. When the tooth erupts there is a certain thickness of cementum and the fibres extend only to its surface. They are arranged in bundles and at about 45° to the root surface.

(10) Box describes non-cellular cementum in the gingival third, and part of the middle third of the root, as a thin layer arranged in concentric lamellae which consist of a calcified matrix, sometimes apparently structureless, and at other times granular or globular. It contains Sharpey’s fibres and it is thought that these fibres become calcified by a globular deposition of lime salts, as in bone.

Cellular cementum (described by others as secondary cementum) is arranged in lamellae also and contains lacunae from which canaliculi radiate. These canaliculi freely anastomose with each other. The lacunae contain cement corpuscles which have fine projections into the canaliculi.

The corpuscles of the lacunae near the surface of the cementum seem to join, by thin processes, with protoplasmic bodies in the periodontal membrane.

So in cellular cementum there is a continuous network of living protoplasm considered to be continually bathed in lymph.

Tests conducted recently with patients receiving radioactive iodine for thyroid disease, showed permeability of cementum, both cellular and acellular, and also of primary and secondary dentine.

Kronfeld describes the cement corpuscles or cementocytes as being cementoblasts derived from the periodontal membrane which have become embedded in the matrix.

The cementoblasts first form a non-calcified
substance called cementoid, which then calcifies into cementum.

Cementum cannot be deposited on the root surface while there is epithelium present, so, when Hertwig's sheath starts to break up, cementum may then form on the dentine of the root. Remnants of Hertwig's sheath often persist and these are called epithelial rests of Malassez. (12)

Goldman considers secondary cementum an abnormality, though quite common. (13)

Miller lists these functions of cementum.

1. Attaches fibres of the periodontal membrane to the tooth.
2. Repairs dentine and cementum in fracture of the root.
3. Isolates filled pulp canals and occasionally dead pulps.
4. Heals over resorbed areas on the root surface.

He also describes these variations in the cemento-enamel junction.

1. Where an area of dentine lies exposed between enamel and cementum.
2. Where enamel overlaps cementum
3. Where they meet edge to edge.
4. The cementum overlaps the dentine.

The superficial layer of cells in secondary cementum are normal and well stained. Deeper they show degeneration with smaller nuclei and smaller cell bodies. Deeper still the lacunae are empty.

The space necessary for extra cementum in the root is formed by minute bone resorptions which are constantly occurring in the socket of every functioning tooth. With a slight excess of functional stress some resorption of bone takes place thus causing a wider periodontal space, and this is partly compensated for, by an increase in deposition of cementum.
Hypercementosis is a diffuse thickening over the whole root. It may occur in all or some teeth in a mouth and is often found in non vital teeth. Here it is a defense reaction due to a low grade irritation of the periodontal membrane over a long period. The etiology of generalised hypercementosis is unknown but there is a hereditary tendency in some people.

Gementum is the most variable of all dental structures and one individual may have cementum three or four times as thick as another. (15)

Gottlieb states that changes in function do not interfere with the deposition of cementum. He also says that immediately below the epithelial attachment, the cementum remains in an uncalcified state for a longer period than it does in more apical areas. This uncalcified layer is thought by him to constitute a barrier against apical proliferation of epithelium.

PERIODONTAL MEMBRANE.

(16)

The periodontal membrane is the connective tissue which surrounds the root of the tooth attaching it to the alveolar bone.

The main tissue elements are white connective tissue fibres called principal fibres. There are no elastic fibres present. Most cells of the membrane are fibroblasts which lie at the surface of the fibre bundles and probably form and maintain the principal fibres.

If bone formation is in progress, osteoblasts will be found along the socket surface between the fibres of the membrane. If active bone resorption is taking place, multinucleated osteoblasts will be found, these are thought to originate from undifferentiated mesenchyme cells in the membrane. It is thought that
these cells produce a substance which dissolves the organic components of bone, and the mineral contents are either removed in the tissue fluid or engulfed by macrophages.

Cementoblasts are found on the surface of the cementum between the fibres.

The blood vessels are from three sources—
1. From the periapical area.
2. Branches from the inter-alveolar arteries passing through openings in the wall of the alveolus. (Main supply).
3. Anastomosis with vessels over the alveolar crest.

Lymph vessels follow the path of the blood vessels (17) and so also do the nerves. Box says the lymph vessels from the connective tissue papillae of the labial and lingual surfaces of the gingiva, pass over the periosteal side of the bone. Lymph vessels from under the epithelium of the sulcus penetrate the alveolar crest fibres and enter the periodontal membrane.

Also in the membrane are found epithelial cells (of Malassez) which are remnants of Hertwig's sheath and which under pathologic conditions may proliferate and give rise to epithelial masses, cysts, or tumours of dental origin. (18)

Orban says these epithelial masses are arranged in cords and have a life span of 20-30 days, new cells being formed all the time. He says there is a possibility that they may stimulate function of cementoblasts.

Cementicles are sometimes found in older people. It is thought that these may be degenerated cells, probably epithelial, which have calcified.

These may be free or fused to the cementum.

The principal fibres are of white connective tissue and the indifferent fibres are of loose connective tissue
Kronfeld describes six groups of principal fibres.—

1. The free gingival fibres which run from the alveolar margin to the gingivae and do not contribute to the retention of the tooth in socket.

2. The trans-septal fibres which are confined to the area between the teeth, and run from cementum of one tooth to that of the next.

3. The alveolar crest fibres run from the crest of the alveolus to the area of root between the deepest part of the epithelial attachment and the crest of the alveolar bone. They may be well developed or may be missing completely.

4. Horizontal fibres run horizontally around the entire tooth (circular ligament) and their function is to prevent displacement of the tooth by lateral stress.

The location of the trans-septal, alveolar crest, and horizontal fibres moves rootward as the epithelial attachment proliferates apically.

5. The oblique fibres, which are the main ones, are arranged in bundles and lie at about 45° to the root surface. There are spaces between the bundles for blood vessels and nerves. In a horizontal section the oblique fibres are seen radiating fan-wise from prominent points or single trabeculae of bone to the cementum. Their function is to attach the root to the socket and counteract vertical stress. They suspend the root in the alveolus and so occlusal pressure is transformed into pull on the alveolar bone, bone being able to withstand pull better than pressure.

6. Apical fibres. There are two groups of these.—

a. Horizontal,
b. Vertical.
These fibres, like the alveolar crest fibres, are variable and may be missing. They are only found in adult teeth with fully developed root ends. All fibres are of collagen and there are no elastic fibres. In complete relaxation they assume a wavy course.

The functions of the periodontal membrane according to Miller are:

(20)
a. Physical.
   1. Attaches tooth to bone.
   2. Absorbs shock.
   3. Layer of soft tissue for nerves and blood vessels.
   4. Keep gingivae in proper relation to the tooth.
   5. Transmits functional stimuli to bone.

b. Sensory Function — gives the teeth the sense of touch.

c. Nutrient Function — for nutrition of cementum and bone.

d. Formative Function — formation of cementum by cementoblasts on the inner surface and formation of bone by osteoblasts on outer surface

The periodontal membrane in a functioning tooth has strong, well developed fibre bundles, especially the oblique group, with narrow spaces for blood vessels and nerves.

Due to mesial movement of the teeth, the interstitial spaces in the periodontal membrane on the distal may be compressed due to stretching of the periodontal fibres.

(21)

In a non-functioning tooth the membrane is narrower, has less fibres and has strands of loose connective tissue in it. It may be replaced in areas by fat tissue. Box calls this adaptive fibropenia.
In an unerupted tooth the membrane is of uniform thickness. In a functioning tooth it is thickest near the alveolar margin. With more function it becomes thicker, and with less function it becomes thinner. It also becomes thinner with age.

The biological thickness or width of the periodontal space in an erupted tooth is .06-.1 mm and the physiological average is .18-.25 mm.

**ALVEOLAR AND SUPPORTING BONE.**

The jaws contain cortical bone, supporting bone and alveolar bone. The alveolar bone proper, consists of the outer surface of the alveolus and the wall of the tooth socket. The space between contains the supporting bone which is arranged in trabeculae. The alveolar bone remains fairly constant but the supporting bone varies with changes in functional conditions, featuring more or less trabeculae.

Under normal conditions there is continual resorption and formation of new bone. Resorption takes place in old bone that has reached the limit of life (senescence). This is then replaced with new bone. Increased function means increased stimulation of osteogenic cells (i.e., osteoblasts) and the resorption is overcompensated, and so we get an increase in the total mass of bone. This is called functional hypertrophy. In decrease function there is still resorption but less formation of new bone—called disuse atrophy.

According to Goldman, bone is built up on the distal aspect and removed mesially — this is associated with mesial physiologic movement of the teeth towards the mid-line. He describes lamellated bone and bundle bone. The latter appearing on the distal surface of the roots where islands of bone are found on the mesial of root surface, no movement has taken place for a long time.
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CHAPTER III.
ETIOLOGY OF PERIODONTAL DISEASE.

This may be divided into:-

a. Local causes and
b. Systemic causes and also a combination of
   the two.

LOCAL CAUSES:-

1. Mechanical irritants.
2. Calculus and other deposits on the teeth.
3. Bacterial causes.
4. Overfunction or lack of function.
5. Chemical irritants, mouthbreathing and
   abnormal habits.

1. Mechanical Irritants:

(1) Food impaction is a common cause of periodontal
disturbances. Fibrous food is forced between the teeth,
and in the early stages, the patient will experience
discomfort and remove it, later, the contacts become
weakened and less discomfort is felt and the patient
may neglect to remove it. This injures the interproximal
crest which may bulge out buccally and lingually in
the embrasures.

When meat fibres are impacted, putrefaction follows
and the toxins formed act then as chemical irritants
and so increase irritation.

Lack of contacts or poor contacts where restorative
work has been done, malposition of teeth, and excessive
occlusal wear resulting in loss of marginal ridges may
also give rise to food impaction.

Miller describes Vertical and Horizontal impaction
of food. -

Vertical. The forceful wedging of food between
the teeth by occlusal pressure.
Horizontal. By the action of tongue or cheeks during mastication.

Faulty margins of restoration, faulty dentures, carious lesions with rough margins and improper use of the toothbrush, floss silk or toothpicks are all possible sources of mechanical irritation.

2. Calculus.

Two types of calculus are described:
a. Supra-gingival.
b. Sub-gingival.

Kronfeld says calculus is the main source of gingival irritation; Coolidge and Hine and Goldman also consider it a cause. However, Miller, Stillman and McCall say that deposits on the teeth are results of functional or hygienic disturbances and are not the initial cause of disease.

Calculus contains desquamated epithelial cells, food debris, salivary mucin and bacteria.

These are first deposited on the tooth and then hardened by deposition of calcium salts from the saliva. Glock and Murray give this composition:

Inorganic materials \( \text{CaCO}_3 \) - 3.17%, \( \text{Ca}_3 (\text{PO}_4)_2 \) - 75.97%, \( \text{Mg}_5 (\text{PO}_4)_2 \) - 3.77%

Protein. - 8.24%
Fat. - 2.71%
Water etc. - 6.04%.

The protein consists of keratin, mucin and nucleoprotein. Hard calculus contains a slightly higher percentage of inorganic salts, and soft calculus a slightly higher percentage or organic matter and water.

Calculus can form on any tooth surface, but occurs mainly near the openings of the salivary ducts from the parotid, submaxillary and sublingual glands, and
so it is usually first seen on the buccal surfaces of the upper molars and the lingual surfaces of the lower anteriors.

Subgingival calculus can form in any sulcus and is mainly found interproximally.

Deposits of soft calculus which contain a larger percentage of organic matter and food debris quickly ferment and decompose, and substances are gradually loosened by friction of food and disappear. However, in most mouths a residue remains which gradually hardens in protected places.

Supra-gingival calculus is light yellow in colour or may be stained brown by smoking.

Sub-gingival calculus is dark brown or greenish and is hard and brittle.

The physical differences are probably due to differences in location. Supra-gingival calculus forms more rapidly and being constantly bathed in saliva loses pigments. Sub-gingival forms slowly under protective cover of the gingiva and small haemorrhages from the ulcerated gingiva add blood pigment.

Box describes three zones in a microscopic examination.

1. A cellular zone next to the epithelium containing leucocytes, epithelial cells and a few red blood cells.

2. A zone of homogeneous material possibly made up of disintegrating cells from zone 1.

3. The zone of hardened calculus.

On the surfaces of Zone 2 calculus is forming and is filled with thread-like organisms.

**FORMATION OF CALCULUS.**

The formation of calculus involves—

a. Precipitation of inorganic salts.

b. The binding together of the precipitate and its attachment to the tooth.
The three main theories for precipitation are:

a. Bacteriological.
b. Enzymatic.
c. Physico-chemical.

The bacteriological theory is that micro-organisms produce conditions which lead to precipitation. Klebs, Godfrich, Moseley, Soderlund and Naeslund attributed the cause of calculus formation to leptothrix and actinomyces organisms. Bibby showed by experiment (in vitro) that without the presence of organisms calculus will not form. He found leptothrix organisms predominant but points out that the bacteriological theory does not explain all aspects of calculus formation.

Box says that vegetations of leptothrix microorganisms serve in some manner to fix the precipitates to the tooth surface.

The enzymatic theory is based on the presence of phosphatase in gingival tissue and in desquamated epithelial cells.

Wilkinson said that the formation of serumal calculus may take place in degenerated cells of the sub-gingival epithelium before any separation from the tooth occurs... "excluding the possibility of the precipitation of salts from the saliva". He says this is probably due to the action of a phosphatase on organic phosphates or phosphoric esters found in serum. He says that G. H. Smith demonstrated that desquamated epithelial cells liberate a phosphatase.

Miller considers this a plausible explanation. Hodge and Wah Leung however, think the theory should be discarded.

The Physico-Chemical theory involves decreased solubility of the calcium salts in saliva. Prinz suggested that the protective action of colloidal proteins which hold the crystalloids in solution is lost as they become concentrated at the surface during saliva stagnation, but it has been pointed out that this does
not seem applicable to the heavy deposits often found opposite the salivary ducts where one would least expect saliva stagnation.

The presence of carbon dioxide in saliva increases its solvent power for calcium salts and it has been suggested that a loss of carbon dioxide would lead to precipitation. (20)

Kessel et al suggests that the organism, lactis aerogenes, breaks down amino acids of saliva into ammonia and nitrogen thus creating an alkalinity which would tend to precipitate calcium salts. (22)

Tenenbaum, Karshan(21) and Rosebury found the calcium and phosphorus content of saliva in calculus positive patients to be higher than in those free from calculus. (23)

Rapp analysed calculus positive and calculus negative saliva for calcium and phosphate content and also for pH. He found that all salivas had a pH higher than a calculated critical pH.

The critical pH of the calculus positive saliva was lower than that of the calculus negative, and there was a greater difference between actual pH and critical pH in the calculus positive. He suggests that this considerable difference between critical pH and actual pH of the calculus positive saliva makes it rather highly oversaturated with calcium and phosphate and that it would only need a little additional alkalination to precipitate large amounts of calcium phosphate salts. (24)

In a further study he demonstrated the presence of carbonic anhydrase in saliva which can speed up the liberation of carbon dioxide. So this may cause loss of carbon dioxide near the salivary ducts causing alkalination and hence precipitation.

Leonard says that the gingival exudate during the inflammatory process is a rich source of calcium and phosphate ions and this may be an explanation of sub-
gingival calculus.
(25)
Box says these factors seem favourable to deposition of calculus.

1. Calcium salts in a solution of colloids.
2. Adherent vegetations of leptothrix organisms on the tooth surface.
3. A rise in pH of the pocket solution.
He says that the fluid in the majority of crevices and pockets is slightly alkaline.
Coulidge and Hine list these necessary conditions for deposition.

1. The presence of a hard substance in the mouth. It will not form on soft tissue.
2. The presence of a starting point, usually a rough surface or a protected surface unexposed to friction.
3. There must be a film of organic matter spread over the hard surface.
4. The presence of an unstable colloidal solution from which insoluble calcium salts are released.

G. V. Black observed that deposition occurred from one to three hours after eating and was more abundant after heavy meals.

He found he could lessen deposits by reducing the quantity of food at meal times and by the use of saline purgatives. He found that deposits could be removed by brushing during the first twelve hours, after this they could not.

The uneven distribution of calculi is explained by small amounts of calculus spread over many tooth surfaces leading to further, and probably more rapid depositions from saliva. The greatest effect of this is near the ducts of the glands and as the saliva is then partially depleted in calcium and phosphate ions, less deposition occurs elsewhere.
Zander describes four possible ways in which calculus is attached to cementum:

1. The organic matrix of calculus is attached to the secondary cuticle.
2. In the absence of a cuticle, the calculus matrix is attached to irregularities in the cementum.
3. Organisms are continuous from calculus to cementum.
4. Mechanical locking of calculus in undercuts of cementum.

Other deposits on the teeth are:

1. Materia alba - a soft white deposit on the necks of the teeth consisting of food debris, dead tissue elements and purulent matter.
2. Mucinous plaques, consisting of mucin precipitated from saliva, bacteria and other products.

Goldman says both of these may cause gingival inflammation.

Kimball mentions these effects of bacteria in plaques and materia alba:

1. The breakdown of proteins by bacteria to indole, skatol, phenol and paraacresol which are definitely toxic.
2. Exotoxins produced by some bacteria.
3. Extracellular enzymes produced by bacteria. One of these she mentions is hyaluronidase, produced by staphylococci, streptococci, pneumococci and some clostridia.
4. Haemolysins and cytolysins.
5. Endo-toxins of gram negative bacilli.
6. Polypeptides produced by bacterial metabolism which may be harmful to the gingivae.

Hyaluronidase is considered a possible "spreading factor" which permits diffusion and absorption of fluids by hydrolysing hyaluronic acid, a constituent of the ground substance in connective tissue.
It is interesting to note the apparent lack of relationship between the severity of periodontal inflammation and the amount of deposit on the teeth. One sometimes sees heavy deposition of supra-gingival calculus with only superficial epithelial ulceration and mild inflammation. The other extreme shows little calculus and a more severe inflammatory process. This seems to indicate the complexity of some of these conditions where several factors may be operating at the same time and also the importance of the tissue reaction of the individual.

A survey among Greenlanders showed common materia alba and calculus in primitive Eskimos but only mild gingivitis, rare periodontitis and no periodontosis. Civilized Eskimos, however, featured 100% gingivitis and common pocket formation.

The role of calculus plays in the etiology of periodontal inflammation is not quite definite.

Some suggest the calculus causes ulceration of the epithelium which is then followed by bacterial invasion. Ritchey & Orban, say either calculus or materia alba is an almost constant finding and when calculus is present it always extends to the bottom of the pocket.

Wæhrhaug concluded that generally the calculus causes an increase in pocket depth by retaining bacteria close to the tissues. Goldman however, does not consider it a direct cause of pocket deepening.

Gottlieb does not agree with this and stated that formation of sub-gingival calculus is secondary to pocket formation and cannot be held responsible for deepening of the pocket, nor for the inflammation at its base.


The oral flora consists of:

1. Transient bacteria which are brought into the mouth by air, food and water and which persist for only a few hours.
2. The resident bacteria. -

a. Cocci - these are the most common and make up almost 50% of the total bacteria.

- Streptococcus viridans.
  " salivarius.
  " Haemolyticus (occasionally)

- Staphylococcus albus.
  " aureus.
  " citreus.

- Micrococcus catarrhalis.
  " flavus.
  " pharyngis siccus.

b. Bacilli,

- Lactobacillus acidophilus.
- Leptotrichia.
- B. Hoffmani and other diphtheroids.
- B. Proteus.
- Lactis aerogenes.

Members of the Friedlander group.

c. Fusiform Bacilli. - At least three varieties have been described.

d. Actinomyces.

e. Vibrios.

f. Spiral organisms. Treponema Microdentium.
  " Hacrodentium.
  " Borrelia Vincenti.
  " Buccalis.
  " Leptospira.

g. Yeast and yeast-like fungi.

h. Complex organisms.

i. Protozoa - Endamoeba gingivalis is found in filthy mouths.

- This list contains many species which are potentially pathogenic. They are held in check by a group of defense factors and only invade the oral tissues when there is
lowered tissue resistance or local trauma.

The role of bacteria in periodontal disease is still rather indefinite. Merritt says that Vincent's infection, caused by fusco-spirochetal organisms in symbiosis, is the only gingival disease caused by a specific organism. Crollidge and Hine say that mixed infections frequently occur in gingival pockets but that this is secondary to pocket formation rather than the etiological factor. Box suggested that actinomyces-like organisms may be responsible for the deepening of pockets. Miller says that it is doubtful that any one influence could, by itself, produce periodontal disease and that a combination of factors is responsible.

Fish describes the deepening of pockets by the formation of minute abscesses. Monahan-Lewis says the etiology of pyorrhea is destruction by infective organisms in the pocket. Kirkpatrick found fusco-spirochetal organisms in almost every case of gingivitis and periodontitis but never in a healthy mouth. Rosebury submits this evidence that infection is an indispensable factor in all forms of periodontal disease.

1. Exudates or gingival scrapings from lesions of periodontal disease, when inoculated subcutaneously into rabbits and guinea pigs produced a necrotic lesion with foul suppuration and a fibrinous exudate.

2. Through repeated transfers many extraneous organisms were eliminated and finally, a stabilized strain which contained small spirochetes, small fusiform bacilli, motile vibrios and anaerobic streptococci, resulted. It was only possible to reproduce lesions with pure cultures of these if all four were present.
3. These organisms were obtained from gingival scrapings from cases of Vincent's infection, pyorrhea and marginal gingivitis, and it seems that in pathogenic capacity the flora does not differ among the clinical entities of the disease.

He said the organisms are normally present in the mouth but in small numbers, so there must be precursory tissue damage which is usually non-infective.

Other infections such as catarhal gingivitis, Herpetic gingivitis, membranous stomatitis are described as a result of infection by certain organisms. Thrush is an infection by the yeast-like organism, Monilia Albicans. The above will be described in a later Chapter.


Overfunction may be caused by the masticating forces being in excess of those which the supporting tissues can withstand. This may be brought about by cusp inclines being too steep, badly contoured restorations, insufficient bony support, too powerful musculature.

Kronfeld mentions tipped teeth and loss of teeth resulting in a greater burden being placed upon the others.

Any of the above may result in loosening of the teeth due to pressure atrophy of the alveolar bone.

However it is dependent upon the tissue reaction of the individual. Some overloaded teeth will feature reinforced alveolar bone and periodontal membrane while in another patient the tooth may become loose and finally lost.

This will be discussed more fully under "Oclusion". Lack of function will result in atrophic change in the alveolar bone, loss of principal fibres of the periodontal membrane and then replacement by loose connective tissue.

Lack of function may be brought about by teeth loss
when a tooth loses its antagonist, crowding of the arch and various malocclusions, indolent mastication, non-
occlusion, premature wear.

The physical character of the diet can have quite a profound effect upon the periodontium. Obviously, soft pappy foods will lead to less functional stimulation (both occlusal via the teeth, and frictional on the gingivae). On the other hand tough, fibrous foods are more conducive to gingival stimulation and occlusal function.

5. Chemical Irritants, Mouthbreathing, Abnormal Habits.

Chemical irritants:

(52) Smoking may, by its irritating effect, lower tissue resistance and increase susceptibility to periodontal disease. (53)

Merritt says the long-continued application of nicotine to oral mucosa has the effect of restricting the blood supply due to a mild angiostenotic effect. So this could lead to undernourishment of the gingival tissues.

Alcohol may also have local effects. Sodium perbonate mouthwashes, if used too frequently, can produce a gingivitis. (54)

Mouthbreathing:

(55) According to Goldman the continual drying effect lowers the tissue resistance and so allows bacterial invasion of the gingival crevice. Usually only the anterior teeth are affected.

Abnormal Habits:

Faulty toothbrushing, biting pipe stems, opening hair pins, bruxism, unilateral mastication and other habits can all influence the health of the periodontal tissues.
SYSTEMIC CAUSES:

1. Faulty nutrition.
2. Blood dyscrasias.
3. Endocrine dysfunction.
5. Allergies, Drug Idiosyncrasies, Metallic Poisoning.
6. Psychosomatic factors.

1. Faulty Nutrition:

An adequate diet includes proteins, carbohydrates, fats, vitamins, inorganic elements, water and roughage in their correct proportions. There may be a deficiency in one or more of these, or, on the other hand, an adequate diet may be available but for some reason the individual is unable to assimilate the food.

Avitaminosis causes many cases of gingivitis and usually the patient is found deficient in more than one vitamin.

(56) A vitamin A deficiency can lead to increased susceptibility to infection and increased hornification of epithelial surfaces. The daily requirement is 4,000-6,000 i.u.

Lack of vitamin D₁ can cause Beri-Beri, with oral symptoms resembling Scurvy. Daily requirement of thiamin is 1-5 mg. Riboflavin deficiency results in glossitis, stomatitis and cheilosis. Daily requirement is 3 mg.

Nicotinic acid deficiency may cause glossitis and stomatitis. Daily requirement is 18 mg.

Of the other B group vitamins a great deal is still unknown. Ziskin et al produced gingival necrosis and bone rarefaction in rats deficient in pantothenic acid.

It has also been shown (57) that a high carbohydrate diet combined with partial deficiency of vitamins A and E predisposes to necrotic gingivitis and periodontitis.
Complete deficiency in Vitamin C causes scurvy though this is not common these days. When a gingivitis fails to improve with local treatment a deficiency in Vitamin C is often the cause.

Radusch says it has been shown that no change occurs in the gingiva with a deficiency in Vitamin C before petechiae appear on the skin.

Daily requirement of vitamin C is 75-100 mgms.

During formation of the teeth a lack of vitamin D will produce poor calcification. In children it causes Rickets and in adults, Osteomalacia with concurrent alveolar bone resorption. Daily requirement is 400-500 i.u.

Diet deficiencies are most likely to be found in the following:

1. Low income group.
2. Erroneous food habits.
3. Alcoholics.
4. Diseases altering nutritional requirements.
5. Inefficient dentures.
6. Food faddists.

Apart from systemic effects of a diet, the local effect is also important.

Such items as consistency, residue that may be left on the teeth, the number of meals and in-between-meal snacks that are eaten during the day, must all be taken into consideration.

2. Blood Dyscrasias.

Since the oral symptoms of blood dyscrasias are frequently the first lesions to appear, the recognition of these is important.

Pernicious Anaemia, thrombocytopenic purpura, the leukemias and agranulocytosis may all feature deviations from normal oral mucous membrane.

3. Endocrine Dysfunction:
In hyperparathyroidism, marked resorption of the alveolar bone may occur. The teeth may become loose and gingival inflammation may be present.

Goldman says hyperthyroidism may cause periodontosis. Periodontal disease has also been found associated with hypopituitarism, hypothyroidism, menstruation, menopause, male climacteric, puberty, diabetes mellitus, hypoadrenalism, (Addison's Disease).

That diabetes mellitus has characteristic oral symptoms is still a point of controversy. Collidge and Hine, Hirschfeld and Goldman say that there are definite oral symptoms associated with the disease.

Miller says that accompanying diabetes or a prediabetic state there is a tendency toward alveolar bone loss greater than one would expect from local irritants.

Glickman produced diabetes in rats and concluded that the nature and incidence of gingival disease are not particularly altered in diabetes, and that notable pathologic changes in the periodontal membrane or cementum are not associated with diabetes.

Thoma says that if a gingivitis is present in diabetes it is probably due to lessened resistance of the oral tissues to irritation and infection. Stahl found that alveolar bone resorption tended to increase with an increase of the severity of diabetes. Desquamative gingivitis and some hyperplasias are also thought to be associated with hormone disbalance.

4. Pregnancy:

Miller says that susceptibility to gingivitis is more marked in pregnancy and that it is probably associated with endocrine adjustments taking place.

Ziskin made a classification of gingival conditions associated with pregnancy. Maier and Urban studied 530 pregnant women and concluded that there is no
significant difference in the prevalence of gingivitis in pregnant and non-pregnant women. They consider the systemic disturbance a conditioning factor and not the primary etiological factor.

5. Allergies.

Due to a condition of exaggerated susceptibility to certain substances, lesions of the oral mucosa may appear. It may occur after absorption of drugs or because of contact with various substances.

Dentifrices, denture bases, sodium perborate, lip stick, phenolphthalein, barbiturates, sulphonamides and different foods have all been found causative.

Drug idiosyncrasias:

A fairly common example of this is a marked gingival hypertrophy due to the use of sodium diphenyl hydantoinate (dilatin sodium) in the control of epilepsy.

Metallic Poisons:

Prolonged administration of heavy metals such as bismuth and mercury in the treatment of syphilis, and the absorption of such metals as lead, copper, silver in a particular occupation may give accumulative effects in gingival tissue.

The effects of drugs and heavy metals may be brought about by:

1. Direct contact.
2. Chemicals absorbed by the intestines and later excreted by the salivary glands.
3. Indirectly the chemicals may cause disturbances of the intestinal tract which lead secondarily to gingival disturbances.

Cahn suggests that metal deposition in the gingiva is caused by the formation of hydrogen sulphide in an area of inflammation. This reacts with the circulating bismuth or lead and forms the metallic sulphide. So in a well-kept mouth it does not occur.
6. Psychosomatic Factors:

Miller says that severe nervous shocks or nervous tension over a long period can lead to recession of the gingival tissues, a tendency towardropy saliva, decalcification of teeth and alveolar bone. He says that whether this is neural in origin, due to metabolic disturbance or some effect on the trophic nerves is not yet known.

(73)

Psychosomatic factors may interfere with periodontal health by:-

1. Reduction of local nutrition by vasospasm.
2. Development of habits detrimental to the health of the oral tissues.
3. By inducing excessive chewing, clenching or grinding of the teeth.
4. By creating taste perversions.
5. By permitting insufficient food intake by limiting gastro-intestinal function.
6. By producing neglect of the oral tissues.
7. By causing systemic conditions detrimental to the health of the oral tissues.

(74)

Ruth Houlton et al made a study of emotional factors and say they may not directly or indirectly.

Direct effect may be by:-

1. Neglect of oral hygiene through anxiety, depression or rebellion against authority.
2. Poor choice of foods.
3. Oral habits like thumb sucking and biting of various objects.
4. Bruxism.

Indirectly:-

1. Endocrine disturbance. Evidently emotions can affect carbohydrate and calcium metabolism and also pituitary function.
2. Resistance to infection may be lowered in states of fatigue and nervousness.

3. Through the autonomic nervous system the circulation may be impaired.

4. Salivary change. Less saliva is found in those depressed and inhibited and an abundance of saliva in those emotionally upset.

Hereditability may also play some part in periodontal disease as several members of one family are frequently found suffering from periodontal aberrations.

One can conclude from the above that there are many etiological factors which can cause deviations from normal. Also quite frequently more than one factor is operating, which can further complicate a diagnosis and treatment planning.
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CHAPTER IV.

DEVELOPMENT OF THE PERIODONTAL LESION.
GINGIVITIS, PERIODONTITIS, PERIODONTOSIS.

Quite a number of different classifications have been applied to periodontal lesions — Fish describes:

1. Degenerations and abnormalities of growth.
   a. Senile alveolar resorption.
   b. Odontoclasia — a condition characterized by resorption of the apices of the roots of some or all of the teeth, not necessarily associated with resorption of the alveolar crests.
   c. Hypercementosis — an opposite condition to odontoclasia.

2. Inflammation.
   a. Traumatic.
   b. Acute ulcerative stomatitis or Vincent's Infection.
   c. Sub-acute marginal gingivitis.
   d. Chronic marginal gingivitis. — Special varieties of this being —
      1. Chronic hypertrophic marginal gingivitis.
      2. Chronic marginal gingivitis senilis.
   e. Pyorrhea.
      1. Simplex,
      2. Profunda;

Merritt gives what he calls a "Clinical Classification":

1. Gum recession.
   { abrasive, atrophic, inflammatory.

2. Gingivitis.
   { marginal hypertrophic, metallic poisons, systemic.

   { acute, sub-acute.

4. Periodontoclasia.
   { local, systemic.
5. Diseases of Systemic origin.

(3) Goldman classifies:—

1. Atrophy. (local trauma.
   senile and pre-senile atrophy.
2. Functional. (disease, occlusal trauma.
5. Periodontosis.

Coolidge and Hine, group the various conditions into:—

1. Inflammatory.
2. Degenerative.
3. Progressive tissue changes.

(4) Orban also describes the above three groups as follows:—

1. Inflammatory. (Gingivitis.
   Periodontitis.
2. Regressive changes. (degenerative — Gingivosis.
   atrophic.
   traumatic.
3. Productive — gingival hyperplasia which is non-inflammatory.

Fish describes "odontoclasis" (his own word) as a disease of unknown etiology in which loosening and wandering of the teeth occurs. This is suggestive of Periodontosis described by other authors, however, according to Fish odontoclasis is characterized by root resorption and the bone which replaces it is usually radiographically normal.

On the other hand, Periodontosis as described by others, affects the investing structures of the teeth and features replacement of alveolar bone by loose connective tissue. (5)

Box describes Simplex and Complex Periodontitis.

The Simplex type being a progressive gingivitis which features broad, shallow pockets, and mobility of the teeth is a late symptom.

Complex Periodontitis shows little inflammation clinically, rapid progress, deep narrow pockets and there is often early mobility.
Mucholte & Dienstein give this description of the development of the Periodontal lesion.

Whether the periodontal lesion is the result of local factors or caused by systemic dysfunction, the cell reaction is the same. The difference being, however, that with local causes only the structures in the immediate vicinity are affected but in general disorders of metabolism every cell may be directly or indirectly disturbed to varying degrees.

Irritants which cause inflammation may be physical, such as calculus or chemical such as by-products of organisms or the toxic products of protein breakdown.

The initial response is alteration in the fluid exchange between the capillaries and the intercellular fluid, brought about by a capillary spasm, followed by dilatation and increase in capillary pressure. If the cells are injured an H+ substance, or leukotaxine, makes the capillary walls more permeable. The exudate is at first serous but later the protein content is increased when the capillary walls are seriously damaged.

Next the small vascular channels are occluded with fibrin. If the lymphatics are occluded early, there is early oedema. This is a means of localising the inflammation, but deprives the area of its normal oxygen supply and this disturbs the metabolism of carbohydrate and causes formation of lactic acid from pyruvic acid, instead of carbon dioxide and water. Thus a local acidosis results. When the pH is lowered to 7.0 the polymorphs become fewer in number and degenerate. Most of the cells are now mononuclears which seem normal at pH 6.9 to 6.8. If the pH drops to 6.5 all cells except the lymphocytes are injured and frank pus develops.

The mineral salts in the bone are only stable at pH 7.4 and with lowering of pH they become more soluble and so the matrix is denuded of mineral salts.
When pH drops to 4.5 proteolytic enzymes reduce the organic matrix to solution.

Weinmann & Sichler disagree with this idea of Halisteris — see below under Periodontitis.

If the reaction is acute and severe, the periodontal fibres become oedematous and with accompanying osteitis there is increased tooth mobility. If more slow and chronic the teeth may be firm in their sockets even though more supporting structure is lost.

The inflammatory reaction progresses along the gingival perforating arteries to the interalveolar arteries and then to the periodontal membrane by the alveolar perforating arteries, thus explaining why mild inflammation can be present for such long periods with little effect on the membrane.

The first perceptible change occurs in the connective tissue papillae, then, a characteristic change takes place in the surface epithelium of the gingival sulcus and may extend to the epithelial attachment. Increased activity of the basal cell layer and in severe inflammation, mitosis in the prickle cell layer. Also, increased production of keratin is marked (this seems questionable as Histologic sections usually show lack of keratin in the involved area. Robinson states that the epithelium lining the gingival sulcus or a gingival pocket shows neither keratinisation nor parakeratinization) and desquamation of cells in the sulcus and in the epithelial attachment towards the tooth occurs, which tends to deepen the sulcus. The connective tissue nearby is oedematous with pyknotic nuclei and swollen collagen fibres. Inflammation cells are present. At the bottom of the sulcus the cells of the basal layer proliferate towards the alveolar crest. The first sign of bone involvement is rarefaction at the tip of the alveolar crest and later the supporting bone.
The connective tissue of the alveolar crest and the trans-septal fibres eventually go into solution and an open tissue lesion is evident. Goldman points out that as this is happening, new trans-septal fibres are formed below the lesion. At this stage it becomes infected and bacteria hasten the whole process.

In transition from acute to chronic the inflammatory cell picture changes from polymorphs and mononuclears to plasma cells and lymphocytes. The diffuse rarefaction of alveolar bone in the acute phase changes to a rather definite marginal rarefaction. The tissue changes from red to bluish red and has a more fibrous appearance.

The natural healing tendency results in formation of granulation tissue which replaces the damaged connective tissue and epithelial cells proliferate across this new tissue. In this description the authors state that epithelium does not proliferate or creep down the root surface.

They also say that the demineralised bone matrix may be remineralised and therefore it should not be disturbed surgically. If removed, no bony repair can take place beyond the height of the operated crest (C.F. Weinmann & Sicher - See below).

(9)

Box considers the mechanism of periodontitis is the production of a necrotizing agent in the pocket which is carried by the lymphatics. He has found necrotic tracts in the periodontal membrane which he recognises as lymphatics distended by a coagulum - like material, and by loss of their endothelial lining by transport of a necrotising agent. The adjacent connective tissue is also damaged. As these necrotic tracts approach the cementum the cementoblasts die and there is also destruction of the crest of the alveolar bone.

He describes other tracts which originate from clefts in the gingival tissues, and some extend along the periosteal side of the bone. Some tracts contained
He describes other tracts which originate from clefts in the gingival tissues, and some extend along the periosteal side of the bone. Some tracts contained epithelial bodies, fungus-like organisms and coccoid bodies and there was no associated inflammatory cellular infiltration.

Fox considers that the necrotizing agent may contain proteolytic enzymes and also that it may be the same as "neecrin" (Menkin) - a toxic material liberated by injured cells. (10)

Ray & Orban found deep areas of necrosis in the connective tissue in cases of gingivitis which were not associated with lymphatics. They say that this is evidence that toxins penetrate deeply into the tissues. The areas they described consisted of degenerating and necrotic plasma cells, necrotic fibroblasts and disintegrating collagen fibres.

Tissue fluids and extravascular cells can only be returned to the blood via the lymphatics. They are also the pathways for the removal of foreign material. A partial or complete blockage of the regional lymphatics is described thus allowing accumulation of waste products which are not drained. This is said to tend towards fibrosis of the area. (11)

Ramfjord says necrotic foci which may be found in the periodontium may be a sequel to marginal inflammation or caused by aseptic traumatic inflammation which follows dysfunctional irritation. (12)

Fisch describes the formation of chronic ulcers on soft gums which allow absorption of toxic products which diffuse among the paradental fibres and destroy them. This allows the sulcus to deepen into a pocket.

According to him, the toxins are from three sources:-

1. A group of M - substances produced by damaged cells
2. Proteolytic ferments produced when cells (especially leucocytes) die.
3. Toxins produced by the organisms.—
   a. Bacterial metabolites.
   b. Disintegration products of dead bacteria.

He describes marginal gingivitis leading on to pyorrhea simplex and, then, in certain cases, pyorrhea profunda by the formation of minute abscesses—generally in individuals with delicate connective tissue.

Fish stresses the point that organisms can not survive below the floor of an ulcer unless they produce an acute paracutal abscess. Otherwise they are destroyed at once locally, or carried away by the blood stream. (14)

In an article by Leonard, however, he states that strep. viridans may be found penetrating deeply into the tissue. (15)

Goldman also says that breaks in the epithelial lining allow bacteria to enter the sub-epithelial tissue and cause further inflammation.

In 1946 Costtloeb put forward a theory that both gum recession and pocket formation are due either to lack of cementum formation or a disturbance in the regular deposition of cementum. In his opinion inflammation is secondary except when caused by some systemic condition or secondary to pocket formation. He describes "marginal periodontia" which seems the same condition as marginal periodontitis, and "cementopathia profunda" (or diffuse atrophy).

Referring to the theory that inflammation causes apical migration of the epithelial attachment he says "In the presence of a highly developed cementum barrier, nothing can cause downgrowth of the epithelium along the cementum."

He considers that the etiology of gingival recession and pocket formation is the same, namely, lack of cementum formation and that in the case of pocket formation, the attachment to a neighboring tooth prevents recession of the gingiva.
Gingivitis: (16)
Kronfeld says it is difficult to distinguish between normal tissues and early pathological conditions. The presence of a small number of inflammatory cells in the sub-epithelial tissues occurs in almost every individual, so he suggests that intact, well cornified epithelium be considered normal.

Fish describes sub-acute marginal gingivitis where the gums bleed freely but there is no marked tenderness. It is common in young people, especially women and starts soon after puberty.

The gum margins are soft, bright red, ulcerated and bleed very easily. No marked deepening of the sulcus occurs but there is oedema and the papillae become detached.

There is usually a bad taste in the mouth in the mornings. Gradually this bright red colour changes to dull maroon and the gum does not bleed quite as easily. Fish mentions a possible softening affect of the oral epithelium caused by a sex hormone which would explain the frequent association of this type of gingivitis with adolescent women.

If this condition remains untreated it develops into pyorrhrea, often of "Profunda" type.

Chronic marginal gingivitis, which may have started as an acute or sub-acute type or may have been chronic from the onset, shows dull red gingival margins sometimes covered with a yellowish exudate. According to Fish this is most likely to develop into "Pyorrhrea Simplex".

He mentions differences in tissue structure from person to person, some having more tendency to form keratin than others and some having stronger and more dense connective tissue.

Kronfeld also remarks that some tend to develop a proliferative inflammation with tissue formation while others are more likely to develop an ulcerative form.
with tissue destruction.

So we see cases of hyperplasia with deepening of the sulcus ("false pocket") and other cases where ulceration and detachment occurs but no increase in pocket depth. (19)

Microscopically, breaks in the crevicular epithelium are seen and inflammatory cells discharging into the crevice. Below the ulcerated areas is a barrier of leucocytes and an infiltration of lymphocytes. If the inflammation is acute, the polymorphs predominate but if chronic lymphocytes are in abundance.

According to Goldman this infiltration extends down to the transverse fibres which remain intact. The capillaries are enlarged and hyperaemic. The alveolar bone is not affected.

If hyperplasia is present, the microscopic picture may show marked chronic inflammation with many round cells in the sub-epithelial tissue, increase in blood vessels, and very little connective tissue. On the other hand there may be a predominance of connective tissue and few inflammatory cells in which case the appearance is more like a fibroma.

Marginal gingivitis may exist throughout the mouth or be confined to single areas.

A common cause of slight gingivitis in young people is a narrow band of sub-gingival calculus and which is often hard to detect as there is only a slight chronic inflammation.

With mouthbreathing the gingivae are swollen and red and usually only the anterior region is affected. Goldman says that calculus is not generally found but that materia alba may be present. Lack of function probably plays some part here also. (20)

Kronfeld says that if hyperplastic gingivitis persists, fibrosis of the tissue occurs. Fibroblasts rapidly multiply and connective tissue is formed. The
inflammatory cells disappear and finally it looks like a fibroma microscopically. One form of localised hyperplastic gingivitis due to chronic mechanical irritation is the gingival polyp - a small pedunculated growth that proliferates from the interdental papilla and rarely from any other part. It is often associated with a deep carious lesion.

It consists of granulation tissue covered with stratified squamous epithelium and it is attached to neighbouring teeth by an epithelial attachment. The amount of vascularity and cellular infiltration depends upon the intensity of the inflammation and so it may be ulcerated, with the sub-epithelial tissue hyperaemic and densely infiltrated, or well hornified with fibrous sub-epithelial connective tissue and poorly vascularised. Hyperplasia is also found with systemic disturbances, - this will be discussed later.

Necrotic Gingivitis - Vincent's Infection.

Collidge and Hine say that it is generally accepted that this infection is caused by the symbiotic action of two organisms, a fusiform bacillus and a spirochete, Borelia Vincenti.

Merritt says it is the only gingival disease caused by a specific organism and is caused by fusospirochetal organisms in Symbiosis.

Other authors are rather doubtful that these are the primary etiological factors.

Fish says that as they are present in every mouth where stagnation and chronic ulceration occurs it is difficult to accept them as causative.

Probably the other theory that due to some predisposing factor such as lowered tissue resistance, the organisms become pathogenic and invade the tissues is more correct. Predisposing factors listed by Collidge & Hine are:—
Influenza, Severe Colds, Diabetes, Blood Dyscrasias, Diet Deficiencies, Overwork, Loss of Sleep and Poor Oral Hygiene.

Merritt considers smoking and oral sepsis the two most important predisposing factors locally.

(24) Studies of Papuan natives suggested that a high carbohydrate diet together with partial deficiency of Vitamins A and B₂ predispose towards Vincent's Infection and Periodontitis.

Smears always show large numbers of the two organisms. The Spirochetes are 8-20 \( \mu \) long with 3 to 6 spirals. The bacilli are 3-8 long, straight or slightly curved with round or pointed ends. Also a few cocci and other bacteria are found in the smears. Fish points out that the organisms never appear on the surface of the slough but deeper, occur in enormous numbers. They are anaerobic and difficult to cultivate. Research by Tunnicliff led her to believe that the bacilli and spirochetes are two forms of the same organism.

(25) Kirkpatrick described four types of spirochetes and two types of fusiform bacilli as well as spirilles and vibrios. Francis considers the condition is often complicated by fungi.

(27) Rosebury obtained a strain containing small spirochetes, small fusiform bacilli, motile vibrios and anaerobic streptococci from gingival lesions. With this he produced lesions in rabbits and guinea pigs with necrosis and fibrinous exudate. He found it possible to reproduce lesions with pure cultures only if all four were present.

He also says that the organisms are normally present in the mouth in small numbers and there must be precursory tissue damage before they become pathogenic.

These symptoms are described:

1. A gray or greenish slough on the marginal gingivae
2. A foul breath.
3. Rapid onset.
4. Spontaneous bleeding.
5. Pain is experienced.
6. Salivation is increased and the saliva is frequently blood stained.
7. A metallic taste is usually experienced.
8. There is a wedging sensation about the teeth and there may be anaesthesia of the periodontal membrane.
9. There may be glossitis.
10. Enlarged sub-maxillary and sub-mental lymph glands.
11. General symptoms of toxaemia and the temperature is usually above normal. In severe cases up to 104°F.
13. Exanthematous eruptions may occur.

Box describes primary and secondary incubation zones:

a. Primary.
   1. Gingival flaps on lower third molars.
   2. Palatal margins of the interproximal gingivae of upper central incisors where there are deep crevices.
   3. Buccal gingival margins of molars in crowded contact with folds of the cheek.
   4. The tonsils.

b. Secondary.
   2. Gingival tissues which have a pre-existing inflammation.
   3. Periodontal pockets.

The acute phase follows an incubation period of 24-48 hours and lasts for 4-7 days. (In some cases it may last for several weeks). Then it becomes sub-acute or chronic. With favourable conditions for growth of the organisms the acute form may return.

In acute, at first the gingivae are acutely inflamed, swollen and oedematous. Soon, sloughing of the crests
of the papillae occurs and the tissues between the teeth look punched out.

There is an acute polymorph infiltration scattered through the interstices of the covering epithelium and in the supporting tissues.

There is necrosis and desquamation of superficial epithelium accompanied by a marked inflammatory exudate which is made up largely of fibrin and this forms a firmly adherent pseudo-membrane which sends projections of fibrin into the neighboring tissues.

Bacteria are found in the membrane and it will be noted that although there is an acute polymorph reaction there is no pus or local abscess formation.

The lesions are relatively localised and do not tend to extend further than a few millimetres from the crest of the gingival margin. In the more remote areas there is infiltration of plasma cells. Cahn says an increase in monocytes is typical in a blood count.

(29) Hampp says spirochetes are to be found deep in the sub-epithelial connective tissue in advance of the necrotic tissue and fusiform bacilli.

In sections studied by Schaffer however he found that bacteria do not penetrate vital tissues but infiltrate the areas of necrosis.

In the sub-acute form, which may be a mild form or follow an acute attack, there are no systemic disturbances and little discomfort of the gingivae.

Goldman describes two forms of sub-acute.——

1. Destructive with ulceration and sloughs.

2. A hypertrophic form with or without necrosis.

Miller describes a chronic form. This may be comparatively painless and may appear in cases that have been treated surgically. In these cases the disease is slow but gradually destroys the tissue.

There is still some argument as to whether Vincent's
Infection is contagious. At one time it was commonly thought to be transmitted from person to person by way of common eating and drinking utensils, toilet articles etc. Also it has occurred where large numbers of people are grouped together (e.g., Trench Mouth). It has never been proved, however, that it is contagious.

It may affect both sexes and mostly between 16-35 years of age.

It usually only affects the gingivae but occasionally involves the whole mouth (Vincent's stomatitis) or the throat (Vincent's angina) and if it spreads to the lungs may cause death.

Rowe-Smith reported a particularly virulent case which involved the eyelids, conjuntion, penis, scrotum, anus and hips.

Findborg studied Danish soldiers and found that with men in different camps but on identical diets and living conditions there was an increase in ulceromembraneous gingivitis in one camp but not in the other and he is of the opinion that this would indicate communicability. He points out that a pre-existing gingivitis and tobacco consumption are important factors.

Kronfeld says Vincent's disease shows a seasonal incidence, being less common in summer and highest in midwinter.

Fish, in discussing contagion, mentions the possibility of the fusio-spirochetes being saprophytic invaders of the slough and that a virus is responsible. There is no proof to substantiate this conjecture however.

IDIOPATHIC FIBROMATOUS HYPERPLASIA.

This is a hyperplasia of the gingivae and connective tissue with very little evidence of inflammation. It is rather uncommon and of unknown etiology, but is thought to be hereditary. Sometimes it appears in childhood and if removed, usually reappears as the child gets older. In other cases it may appear in adolescence.
Both labial and lingual gingivae are involved, and the tissue may almost cover the crowns of the teeth.

Histologically, there may be mild inflammation in the superficial connective tissue with scattered plasma cells and lymphocytes in the deeper layer. The connective tissue shows many well defined and enlarged fibre bundles and only a few blood vessels. There is projection of proliferated connective tissue into the epithelium which consists of only a thin layer.

Cahn says the epithelium may show atrophy due to pressure from the overgrowth of connective tissue or may show hyperplasia also.

**Atrophy of the Gingiva.**

This may be caused by injurious toothbrushing which results in pressure atrophy. The gingival margin is thickened, hard and festooned and the colour and crevice depth remain normal.

Hirschfeld says toothbrush trauma may be due to:
1. Dental arch abnormalities.
2. Improper technique, or
3. Faulty toothbrush.

He thinks that Stillman's clefts are due to faulty toothbrushing rather than traumatic occlusion as suggested by Stillman. An abrasive dentifrice would also hasten this process.

Senile atrophy is a normal physiologic process. In this there is gingival recession and atrophy of the alveolar bone.

Dummett describes the ageing of the periodontal membrane, with slower mitosis, lessened capacity for cellular accretion, cellular atrophy, fatty degeneration of nerve cells, degeneration of cells and increased pigmentation, less elastic blood vessels due to hardening fibrous tissue and calcium deposition thus leading to a decreased blood supply.
In ageing of the alveolar bone he describes increased fragility, decreased vascularity, lower metabolic rate, fibrosis of the marrow and resorption of the alveolar crests.

The epithelium proliferates apically and is more prone to injury. These retrogressive changes of the changes of the alveolar bone are usually symmetrical but certain areas - labial surfaces of the cuspids and lower central, and lingual surfaces of the upper first molars - are more susceptible.

Gottlieb suggested that gingival recession could be due to lack of cementum formation which allows the migration of the epithelial attachment. He called this "Marginal Cementopathia".

Pre-senile atrophy is a similar process which occurs at an early age and so far is of unknown etiology. Clinically the soft tissues look anaemic and glisten. Recession is symmetrical and there is no inflammation. Some think that a deficiency in some sex hormone may be involved.

GINGIVITIS DUE TO SYSTEMIC INFLUENCE.

Goldman says that many cases of gingivitis are due to a lack of vitamins in the diet and that usually the patient is deficient in several vitamins rather than a single one.

Little mention is made of Vitamin A in current text books but Sinclair considers a deficiency of Vitamin A an important factor in periodontal diseases.

Kirkpatrick says that patients with scurvy would also in all probability suffer from partial deficiency of A and B and it is quite likely that these are responsible for the gingivitis associated with scurvy. He also suggests that a combined Vitamin A and B deficiency would have a more detrimental effect on the gingiva than lack of B alone.
Mellanby in 1923 produced defective bone, gingivae and periodontal membranes in puppies fed on a diet deficient in Vitamin A.

Increased hornification of epithelium, and increased susceptibility to infection are other features of a Vitamin A deficiency.

Lack of Vitamin B₁ can cause Beri-Beri, a tropical disease common among rice-eating people. This has two oral symptoms similar to scurvy, hyperplasia and oedematous rose-pink gingivae.

Riboflavin deficiency may show dry, slightly oedematous, tender gums and ulceration with a gray exudate may occur. There is often a glossitis and cheilosis.

Nicotinic acid deficiency features a stomatitis and glossitis and often a necrotic membrane containing Vincent's organisms is found. So it may happen that a diagnosis of Vincent's Infection is made while the systemic condition is overlooked.

Several authors suggest administration of nicotinic acid in cases of Vincent's Infection.

Complete deficiency of Vitamin C causes scurvy but this is fairly rare now. In adults there is hyperplasia of the gingivae and the colour varies from red to purple. Haemorrhage is frequent and the hyperplasia may be so extensive as to cover the crowns of the teeth. The teeth may become loosened.

Prinz and Greenbaum say there are no oral symptoms in an edentulous mouth.

Miller says a Vitamin D deficiency will show a bright red line on the marginal gingiva which probably indicates alveolar resorption.

Becks and Simmonds experimented on dogs and showed that a lack of Vitamin D caused loss supporting bone which was replaced by fat marrow.
BLOOD DYSCRASIAS AND GINGIVITIS.

In pernicious anaemia the gingivae may have a pale waxy colour and ulceration can easily occur. The most characteristic symptom is an atrophic glossitis.

The tongue is bright red with a smooth glistening surface and spiced foods and acid fruits may cause considerable pain.

Merritt says a sore mouth is practically continual accompaniment of pernicious anaemia.

The leukaemias often show oral symptoms before any other symptoms. Gingival hyperplasia and spontaneous bleeding is featured in the acute leukaemias and according to Merritt fusco-spirochetal organisms are always to be found. This is evidently a superimposed Vincent's Infection.

Wentz investigated gingival changes in leukemia and found that the oral symptoms were more serious in the acute forms and those of the monocytic type most prominent.

Agranulocytosis may show haemorrhage from the oral mucosa, ulceration is common and the tongue becomes red and swollen. The clinical picture may closely resemble Vincent's Infection and may be mistaken for it according to Merritt but as fusco-spirochetal organisms are usually abundant it is probably a true Vincent's Infection superimposed upon already weakened tissues. Thomä, however, states that though a Vincent's-like Infection may occur, the typical organisms are not always present.

Thrombocytopenic purpura is characterized by haemorrhages into skin and mucous membrane. Orally, areas of petechiae are seen, which are at first bright red and later change to a purple colour.

GINGIVITIS DUE TO ENDOCRINE DISTURBANCES.

Ziskin classified five types of gingivitis associated with pregnancy.

1. Bleeding from the gums with eating or toothbrushing.
If any such symptoms were present before pregnancy they are exaggerated after conception.

2. The papillae lose their stippled appearance and becomes glassy-looking. The colour changes from pink to old rose and there is some oedema. This state may persist right throughout pregnancy and return to normal after parturition.

3. Raspberry-red gums, highly inflamed free gum margin which bleeds easily.

4. Hypertrophic gingivitis. Here he describes five varieties with varying forms of hyperplasia. In all these forms the gums bleed easily but are seldom painful.

5. Pregnancy Tumour.

This is usually a single growth from any point which bleeds easily. At first it looks like an overgrown papilla but may enlarge till it is 1-2cms. in diameter and usually has a pedunculated attachment. It is cyanotic in colour with a bright red border. The tumour may begin early in pregnancy and grow rapidly. It either disappears or greatly diminishes in size after parturition.

(53) Thomas says it is most likely to occur in the 3rd month of pregnancy.

(54) Maier and Orban, however, studied 530 pregnant women and concluded that there is no significant difference in the prevalence of gingivitis in pregnant and non-pregnant women. They considered the systemic disturbance a conditioning factor and not the primary etiological one.

(55) Mosteller is also of this opinion and says puberty, pregnancy and menopause periodontal disease is a myth but that any of these may predispose the gingival tissue to proliferation in response to a local irritation.

(56) Hilming, however, says that there is a specific gingivitis of pregnancy of probable hormonal origin.
Merritt says that pregnancy seems to predispose towards gingivitis but there is probably no type of gingivitis that is peculiar to pregnancy. If a gingivitis is present it tends to assume a hypertrophic form.

Goldman considers that gingivitis is most likely to develop during the second three months of pregnancy and is characterized by bleeding and capillary hyperplasia.

Miller says that susceptibility to gingivitis is more marked in pregnancy and that it is probably associated with endocrine adjustments taking place.

It has been suggested that other forms of endocrine disturbances may affect the gingivae. Miller says that mild uniglandular or polyglandular aberrations may increase susceptibility to periodontal lesions. He quotes Wolf as saying - "the nutrition, growth and repairs of all tissues, including those of the oral cavity, are dependant upon normal metabolic functions, and they, in turn, depend upon normal and correlated activities of the endocrine glands".

Fairly frequent hyperplasia is seen in girls and boys in the adolescent period and there seems to be association between this abnormal gingival proliferation and a systemic factor e.g., hormone influence, since often the mild local irritation by itself would not cause the lesion. It may appear as the sessile or the pedunculated type and affects the anterior gingivae mainly.

Microscopically those proliferations show oedema, congestion of the capillaries and infiltration of plasma cells and lymphocytes in the sub-epithelial tissue and polymorphs in the superficial layers.

Engel in an article on hormonal gingivitis describes a carbohydrate-protein-complex ground substance in connective tissue, the state of polymerization of this being altered by enzyme action and also by hormones.
He concludes that gingivitis associated with pregnancy and menstruation and also desquamative gingivitis may be due to the effect of hormones on the ground substance.

**GINGIVITIS ASSOCIATED WITH DIABETES MELLITUS.**

As has been mentioned in Chapter III there is some controversy over the relationship between Diabetes Mellitus and Periodontal Disease.

(61) Hirschfield described these characteristic mouth symptoms:

1. The sudden appearance of acute gingival swellings resembling abscess formation.
2. After discharge of an exudate there is considerable loss of tissue.
3. These swellings are often accompanied by polyps which proliferate from the sulci. The incisor area usually being affected first.
4. The teeth become loose and sore and there is a lot of calculus formation.
5. The tongue is thick and flabby and shows imprints of the teeth along the edges.

(62) Miller states that alveolar resorption and periodontal abscesses are common.

(63) Goldman describes swollen, red, spongy gingivae, a dry mouth, enlargement of the tongue and rapid formation of supra-gingival calculus. Tissue resistance is lowered and infection occurs easily.

(64) Ray describes these changes in the gingiva:

1. Lymphocyte infiltration of the papillary layer which is independent of pocket inflammation.
2. No characteristic change in the surface epithelium but some hydropic degeneration in the prickle cell layer.
3. Degeneration of collagen fibres.
4. Frequent thickening and hyalinization of vessel walls.
5. Frequent appearance of hyaline and calcified bodies.
He says that the diabetic state may be directly responsible for the degeneration of the collagen fibres, and that it seems to modify the reaction of the gingival tissues. He also says that the increased protein breakdown which occurs in diabetes may cause a local acidosis and so explain the increase in lymphocyte and plasma cell infiltration. (65)

Stahl found that the alveolar bone resorption tended to increase with an increase of the severity of diabetes.

**DRUGS, METALLIC POISONS, ALLERGIES.**

The use of sodium diphenyl hydantoinate (dilantin sodium) in the control of epilepsy may produce a marked gingival hyperplasia.

The nature of the hyperplasia is influenced by local factors. In clean mouths the hyperplastic tissue is firm, smooth, not very tender and does not bleed easily. Where oral hygiene is poor the proliferations show inflammation and bleed more freely. Usually hyperplasia does not become evident before three months of administration of dilantin.

It has been found over a large age range and, according to Goldman, is generalized in distribution. A study of 244 patients taking dilantin, showed hyperplasia in 54% with most severe reaction in 15-30 years age group. Histologically there is proliferation of epithelium and marked hyperplasia of the connective tissue. According to Thoma there may be formation of heteroplastic bone. (66)

Mesantoin, a new drug suggested for epilepsy apparently does not cause a hyperplasia but is not quite as good as anticonvulsant.

Poisoning by lead may occur in those who handle white lead. It may be absorbed through the skin, respiratory tract or the digestive system.

The symptoms are metallic taste, sweetish breath and numerous small dots of lead sulphide around the gingivae
in areas of inflammation. They are seldom seen around healthy gingivae.

Bismuth gives a similar bluish, black line and here again the deposition (Bismuth Sulphide) occurs in inflamed areas.

Mercury is excreted through the saliva and gives rise to a stomatitis or gingivitis usually in patients with poor oral hygiene. Coolidge & Hine, say that a mercurial gingivitis is seldom seen in a clean mouth and not in edentulous patients.

There is a metallic taste, swollen red or blue papillae pocket formation, frequent gingival haemorrhage, greenish gray ulcerations, increased flow of saliva, loosened teeth and enlarged lymph nodes.

Some authors refer to a line of pigmentation in mercury poisoning but Coolidge and Hine state that Bismuth and lead are the only metals that produce these lines.

Allergic reactions are occasionally found in some individuals. Foods, drugs and certain proteins may cause the reaction. It may be caused by absorption of the substance or by direct contact. Usually there is hyperaemia of the gingiva and a blood count shows an increase in eosinophils.

**DEQUAMATIVE GINGIVITIS.**

This is an unusual disease of chronic nature and of uncertain etiology. (70)

It is said to be more common in women at the time of menopause. Most authors now consider it due to some hormone disbalance.

The areas of desquamation are seen buccally and labially but seldom lingually and do not appear in edentulous mouths.

**Symptoms:** (71)

1. Irregular inflammatory zone involving the gingivae and extending for several millimetres.
Most common round maxillary incisors.
2. Colour varies from scarlet to bluish-red and is often mottled.
3. There is a clear demarkation between the affected area and healthy tissue.
4. The area may be of glistening appearance.
5. Strips of epithelium slough off and leave a raw, sensitive surface.
6. Desquamation may be preceded by blister formation.
7. Occasionally there is a "pedunculated fungoid appearance" interproximally.
8. Very chronic and always a complaint of soreness is made.

(72)
Microscopically denudation of epithelium is seen and any which remains shows intra-epithelial vascularization. Cell outlines are blurred and the underlying tissue has little collagen. The tissue appears generally oedematous and is flecked with chronic inflammatory cells.

(73)
Cahn has found fungi deep within the tissue. He also remarks that from his observation, the epithelium seemed to be destroyed from beneath.

(74)
Schour & Massler described a gingival condition in post-war Italy which they called "gingivosis". This was more of a degenerative than inflammatory nature and in the early stages it resembled Vincent's Infection, though there were no great numbers of Vincent's organisms in the smears, and in the later stages it resembled periodontosis. They said it seemed related to impetigo-like lesions on the face. Therapy with Vitamins A, C and D had no effect, but with B complex it showed a marked improvement.

(75)
Orcan classifies desquamative gingivitis as an adult form of "Gingivosis".

PERIODONTITIS.

Most authors consider that periodontitis simplex, (marginal periodontitis, pyorrhea simplex etc.,) is the
stage following marginal gingivitis.

Fish explains pocket formation by the action of toxins in the gingival sulcus which gradually destroy the fibres of the periodontal membrane. He describes downgrowth of epithelium over the cementum surface and also proliferation of epithelium at right angles to the lining of the pocket.

Wærhaug has a somewhat similar explanation of pocket deepening. He also suggests that the infiltration of plasma cells and lymphocytes causes dissolution of the periodontal membrane fibres.

Other authors do not agree with this. Weinmann has shown that invasion of the periodontal membrane is a late symptom and says—... "only after the destruction of the bone separating the inflammatory focus from the periodontal membrane proper, is the latter involved". He says that due to the resistance offered by the periodontal membrane fibres, the inflammation follows the line of least resistance and progresses to the periosteal surface of the alveolar bone or into the marrow spaces of the septum. However, Weinmann considers that if periodontosis or occlusal trauma is also present to complicate the picture, then the inflammation can extend directly into the periodontal membrane.

Box suggests there may be a form of periodontitis caused by a specific infection which is characterized by necrosis of the epithelial attachment and rapid apical shifting of the pocket base.

Again referring to Weinman, the entire depth of the gingiva has to be involved before a marginal periodontitis can develop. He says that the epithelial downgrowth causes pocket formation and asks whether the detachment of the fibres occurs before this downgrowth or does the epithelium grow down around the fibres which are then destroyed by desmolytic enzyme action.

Histologically the picture is one of chronic inflammation,
showing plasma cells (very prominent), lymphocytes and macrophages.

When the inflammation progresses into the most superficial marrow space of the septum, the bone at the alveolar crest is removed by osteoclastic action and this is the first sign of periodontitis radiographically.

The fatty marrow is replaced by fibrous marrow and so the inflammation eventually reaches the periodontal membrane after destruction of the alveolar bone which, at the same time, causes detachment of the principal fibres.

(83) Robinson states that the pressure of edematous tissue round the alveolar bone crest and also enzyme activity cause resorption of the crest. This causes loss of the alveolar crest, free gingival and trans-septal fibres.

He says that bone reduction in periodontitis usually is the result of maintaining a normal or slightly reduced osteoblastic rate while the osteoclastic activity is increased.

Fish describes pyorrhea simplex and pyorrhea profunda and suggests that pyorrhea simplex is likely to follow a chronic marginal gingivitis whereas pyorrhea profunda frequently follows a sub-acute marginal gingivitis.

In pyorrhea simplex the pocket depth is fairly regular and radiographs show resorption of the alveolar crests and there may also be areas of sclerosis around the apices. Bone loss is regular and calculus abundant.

Pyorrhea profunda has little or no calculus associated with it at first but later a deposit of hard, green sub-gingival calculus forms. It follows a fairly rapid course and leads to loosening and wandering of the teeth. Bone loss is irregular and there are abnormally deep pockets here and there.

This condition seems analagous to periodontitis complex
as described by other authors though they suggest that it is a condition caused by both local and systemic factors.

Merritt in reference to "horizontal pyorrhea" observes that in his experience there are very few cases which show any uniformity in pocket formation. Weinmann and Sicher raise the point that removal of bone always entails the simultaneous disappearance of the organic and inorganic components. Several other authors describe the loss of the inorganic content and the retention of an organic matrix which may later be remineralized.

PERIODONTOSIS.

This is now recognised as a particular condition of the periodontium which features non-inflammatory degeneration of supporting structures. It was first described by Gottlieb in 1920 as "diffuse atrophy of the alveolar bone". A similar condition was described in 1924 by Box who named it "rarefying peri cementitis fibrosa", which he considered due to an over-stress in the periodontum.

According to Glickman it is a chronic disease occurring more frequently in females, especially in the puberty-to-forty age group. It may involve all the teeth or only one tooth. It occurs firstly and is usually most severe, in the incisor and molar areas. An early sign is mobility in the absence of any notable gingival inflammation, and early migration labiodistally of upper incisors is typical. The greatest resistance to bone loss seems to be in bicuspids areas - especially the lower.

Two stages are recognised:

a. The primary or degenerative stage and
b. Secondary or inflammatory stage.

The primary stage is caused by some systemic factor.
trauma and inflammation are ruled out. This stage is rarely seen, as complication by local factors occurs early with the formation of deep pockets. These tend to form on the side of the tooth away from which the tooth is moving.

Radiographically he describes an area of bone loss with an arc-like contour in the incisor and first molar regions, a thicker periodontal space, absence of, or haziness of the lamina dura and vertical destruction of the septa.

Also there is an increase in the size of the cancellous spaces. Orban and Weinmann describe three stages in the histopathology of this disease.

1. Degeneration of the principal fibres of the periodontal membrane and resorption of the alveolar bone. Proliferation of capillaries and the development of loose connective tissue. At this stage there is no inflammation and no proliferation of the epithelial attachment.

2. Proliferation of the epithelial attachment along the root and slight cellular infiltration of plasma cells and polyblasts.

3. Separation of the epithelial attachment from the tooth with formation of deep crevices. This then produces a pocket which becomes infected.

Stage 1 is short and soon is obscured by secondary inflammation.

The fibres of the periodontal membrane become disorganised and degenerate. The periodontal space is widened by loss of stimulus via the fibres and also by increased pressure produced by oedematous hydropic swelling of the fibrous tissue and capillary proliferation. This also brings about the migration which always occurs away from the diseased surface.

In the second stage the migration of the epithelial
attachment occurs after the loss of fibres attached to the cementum. The authors say ... "due to some toxic effect, degeneration of the connective tissue of the periodontal membrane is the primary pathologic condition in the case of periodontosis".

They explain occasional root resorption which may be found, by the lack of a new cementum layer. Old cementum which becomes calcified completely is more susceptible to resorption.

Goldman says it is essentially a disease of the periodontal membrane and alveolar bone which later affects the gingiva.

It may appear in mouths with good hygiene and no caries and remain undetected until marginal inflammation becomes apparent. The normal fatty marrow of the supporting bone is replaced by loose fibrous connective tissue with numerous dilated blood vessels.

Box describes "peri-cementitis fibrosa", a chronic and progressive disease of the periodontal membrane which results in rarefaction and destruction of alveolar bone. This new tissue is a fibrillar connective tissue and a proliferative change occurs in the blood vessels whereby the endothelial cells cause partial or complete blockage of the lumen. Occasional lymphocytes and plasma cells are seen but no significant development of new capillaries. He associates this condition with traumatogenic occlusion. In his article in 1943 however, he indicates that he accepts the classification of a purely systemic form of periodontal disease (i.e., periodontosis).

The etiology of periodontosis seems rather obscure. All the following have been suggested as possible factors, though Glickman points out that the assumption that periodontosis is a specific disease that always originates in one tissue, is wrong.

Gottlieb considered pocket formation a result of a
disturbance in the deposition of cementum and describes "cementopathis profunda" or diffuse atrophy as a condition where this cementum disturbance occurs away from the epithelial attachment of the tooth with consequent migration of the epithelium and its detachment from the tooth surface.

Alveolar resorption has been found in more individuals due to defective absorption of calcium salts through gastric hypo-acidity. Kerr suggests that it may be a manifestation of some collagen disease (e.g., periarteritis nodosa, lupus erythematosus etc.)

Of auto-intoxication, which has been mentioned by others, Kerr says that as there is no uniform agreement that it actually exists, it should not be listed as an etiological factor. Blood studies by Karshan et al indicated that deviations from normal ranges of certain blood constituents were present in periodontal patients and also that the plasma Vitamin C was low in many of the periodontosis cases. So there may be some interference in the utilization of Vitamin C, apart from this however there were no regular typical aberrations from normal ranges.

Similar lesions have been produced in animals with excessive alkaline diet, protein deprivation, diabetes (produced by administration of alloxan), Vitamin C deficiency, hyperthyroidism, hyperparathyroidism, cortisone administration.

Weinberger says we must consider alveolar bone more susceptible to dietary changes than long bones and that a high calcium and low phosphorus diet is the cause of periodontosis.

Ramfjord does not accept an explanation of periodontosis by systemic factors. He says systemic factors only influence the result of local injury and that "an unfounded
relationship has been reported to exist between the periodontal changes and systemic diseases". He explains periodontosis by local circulatory disturbances due to dysfunction, especially trauma.

Leonard takes this line and says the cases of periodontoclasia which cannot be explained by infection, or infection in conjunction with occlusal trauma, or atrophy due to lack of function, are so few as to be negligible.

Orban studied the epithelial network of the periodontal membrane and points out that there may be a connection between cessation of cementum formation and degeneration of epithelial cells in the membrane. Is it possible that these cells stimulate cementoblast activity?

A complex periodontitis is fairly generally accepted where both local and systemic factors are functioning. The course is more rapid than the simplex type, depth of pockets irregular and mobility may occur in the early stages.

Goldman points out that this form probably explains why some cases which are diagnosed as a simplex periodontitis do not respond to local treatment.

When one sees a young patient with advanced pocket formation and tooth mobility with only small evidence of local etiology it does tend to arouse suspicions of systemic factors. However, it seems quite possible that in these cases, instead of systemic disturbances actually causing the lesion, it may be that little resistance is offered to a seemingly slight local factor or factors.

In other words tissue resistance of the patient has much bearing on the progress of periodontal disease. Though quite a few cases have been produced of a pure periodontosis with no inflammatory change these seem to be very rare.
<table>
<thead>
<tr>
<th>Reference Number</th>
<th>Author(s)</th>
<th>Title</th>
<th>Publisher/Year</th>
<th>Pages/Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Fish, E.W.</td>
<td>Parodontal Disease, (Bye &amp; Spittiswoode 1946)</td>
<td></td>
<td>50-2</td>
</tr>
<tr>
<td>2</td>
<td>Merritt, A.H.</td>
<td>Periodontal diseases (Macmillan 1945)</td>
<td></td>
<td>25-6</td>
</tr>
<tr>
<td>3</td>
<td>Goldman, H.M.</td>
<td>Periodontia (Mósby 1949)</td>
<td></td>
<td>318-9</td>
</tr>
<tr>
<td>4</td>
<td>Coolidge, E.D. &amp; Hine, H.K.</td>
<td>Periodontia (Lea &amp; Feb. 1951)</td>
<td></td>
<td>40</td>
</tr>
<tr>
<td>13</td>
<td>Fish, E.W.</td>
<td>Parodontal Disease. p. 18.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Goldman, H.M.</td>
<td>Periodontia. p. 111.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Kronfeld, R.</td>
<td>Histopathology of the Teeth. (Lea &amp; Febiger, 1949)</td>
<td></td>
<td>328</td>
</tr>
<tr>
<td>17</td>
<td>Fish, E.W.</td>
<td>Parodontal Disease. pp. 51, 57-8.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>Goldman, H.M.</td>
<td>Periodontia. pp. 101-9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Fish, E.W.</td>
<td>Parodontal Disease. p. 22.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>Kronfeld, R.</td>
<td>Histopath. of the Teeth. p. 335.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>Fish, E.W.</td>
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CHAPTER V.

OCCLUSION AND ITS RELATION TO PERIODONTAL DISEASE.

(1)

Strang defines normal occlusion as normal relationship of the occlusal inclined planes of the teeth where the jaws are closed, accompanied by the correct proximal contacts and axial positioning of all teeth, and the normal growth, development, location and correlation of the various associated tissues and parts.

The alveolar arches are in a state of constant alteration and reformation. Wear of occlusal and proximal surfaces, and deposition of cementum allow changes in position to occur.

The forces which tend to balance or stabilise occlusion listed by Miller are:

1. Inclined planes of the teeth.
2. Interproximal contact.
4. A slight axial inclination of the teeth which causes a forward propelling force when the upper and lowers articulate.
5. Action of jaw muscles.
6. Atmospheric pressure.
7. Force of growth.

Interference with any of these factors will destroy the balance and cause abnormal migration.

During mastication the teeth are exposed to a resultant of a vertical and horizontal force. Whether the horizontal or the vertical component prevails depends upon the masticatory movements and the conditions in the mouth. Factors which would influence this are:

1. Height and steepness of cusps.
2. Interlocking cusps.
3. Extent of lateral excursions of the mandible.

Schwartz showed that with a horizontal stress in the crown of a tooth, the fulcrum lies between the middle and the apical third of the root in single rooted teeth, and in teeth with more than one root the fulcrum is between the roots in the bone of the inter-radicular septum.

In a fully developed tooth there is less apical movement than at the alveolar margin, due to the presence of apical fibres, but in young teeth more movement will occur at the apex due to lack of fibres there.

In tipping action about half the fibres are stretched and the other half relaxed or even compressed.

In vertical stress such as in teeth with flat surfaces, the only relaxed fibres are the apical and perhaps the alveolar crest group.

The soft tissues of the periodontal membrane act as a cushion so that the force of occlusion is received by a hydraulic shock absorber. The force is first transmitted as hydraulic pressure to the walls of the periodontal space and as fluid is displaced - mainly in the apical region, due to more communication with the marrow-spaces, the tooth moves further into the alveolus thus allowing gradual transference of the force to the fibres of the membrane.

Under normal conditions there is continual resorption and formation of new bone, and under uniform function the total mass of bone remains unchanged. Increased function means increased stimulation of osteogenic cells and the physiologic resorption is overcompensated hence an increase in the total mass of bone occurs - this is called functional hypertrophy. With decrease function, resorption still goes on but there is less formation of new bone, resulting in disuse atrophy. These changes occur mainly in the supporting bone, and the alveolar
bone proper and the outer wall of the alveolus and wall of the socket remains fairly constant.

Whether occlusal trauma is a primary etiological factor or a secondary contributing factor has been the subject of much debate. As was mentioned in Chapter IV. Ramfjord attaches much importance to occlusal trauma and even explains periodontosis on this basis.

Leonard says faults in occlusal equilibrium are an important cause of vascular changes which lead to periodontoclasia.

He describes excessive tooth movement causing crushing of areas of periodontal membrane which, if mild, leads to inflammation and resorption of bone and loss of periodontal membrane fibres. With greater force necrosis and haemorrhage occurs.

Pocket formation may occur by loss of fibres leading to apical proliferation of the epithelial attachment and its subsequent detachment from the tooth. More likely however, (according to Leonard) bacteria from any gingival infection invade the areas of damaged periodontal membrane. He says that this explains why we nearly always find pockets most advanced on the side toward which the tooth is moved during stress. (C.F. Periodontosis Chap.IV.) Also he makes a statement that, as there is no to-and-fro movement in Orthodontic treatment there is no periodontoclasis.

Leriche and Policard say intermittent pressure favours bone formation and constant pressure favours resorption. Leonard says the most frequent cause of occlusal trauma is loss of a posterior tooth which is unreplaced. The tendency for adjacent teeth to drift puts the periodontal membrane in an unstable state and during this, the structures become more susceptible to periodontal disease.

These are the diagnostic features he lists for traumatic occlusion:

1. Thicker periodontal membrane.
2. Destroyed Lamina Dura or
3. Thicker Lamina Dura.
4. Abnormal mobility.
5. The occurrence of a deep pocket when the rest of the mouth is free of pockets.
6. Pockets that fail to heal.
7. Cold air causing pain when there is no exposed dentine.
8. Tooth or teeth tender to biting stress.
9. Hypersensitivity to heat.
10. Toothache at night.

(6) (7) Orban agrees that lateral forces, if moderate, can cause inflammation and resorption and if severe and persistent can cause thrombosis, haemorrhage, necrosis and extensive resorption. He has also shown the presence of cartilage in the periodontal membrane in cases of traumatic occlusion. If traumatic occlusion acts in one direction resorption of bone occurs in the pressure zone and bone deposition in the traction zone. If in several directions bone destruction will occur all around the root which leads to a thicker periodontal space and a loose tooth.

However, he (Orban) queries the claims made (Hill etc.,) that a traumatic occlusion can impair the circulation of the gingiva, since the blood vessels to this area are in bone, and outside the periosteum, but not in the membrane.

(8) Merritt considers traumatic occlusion more often a symptom than a cause of periodontoclasis.

(9) According to Goodidge and Hine these conditions can lead to traumatic occlusion.

1. Malposition of the teeth.
2. Cusp interference,
3. Mesial drifting or tipping.
4. Loss of bony support resulting in reduced capacity for normal function.
5. Excessive overbite causing increased horizontal stress.
6. Uneven rate of tooth eruption.
7. Uneven occlusal wear.
8. Excessive wear and loss of vertical dimension.
10. Habits such as, nail-biting, bruxism etc.

Miller says premature wear will lead to lack of stimulation thus causing disuse atrophy, and also involves the necessity for excessive pressure for mastication and this may cause occlusal trauma. Box, however, describes studies by others on wear of teeth in primitive people. He says that premature wear resulting in disuse atrophy does not seem to be based on sound clinical examination. He also questions the idea that cusp height and steepness of inclined planes are necessary to periodontal health in youth.

He defines "traumatogenic occlusion" as an occlusion which, under biting pressure, produces an injury in the periodontal tissue, and refers to traumatic occlusion when the contact relation of the masticatory surfaces of the teeth occurs as the result of trauma. Factors he gives that may lead to trauma are:-

1. Excessive magnitude of occlusal force.
2. Excessive angle of contact.

These may cause excess pressure or defects of pressure in the periodontal membranes. He mentions the case of a tooth which has little function and therefore is weakened, but which in certain movements of the mandible experiences a severe form of traumatogenic occlusion.

In an investigation by Orban and Weinmann in human jaws they observed that in every jaw, which exhibited loss of teeth which had not been replaced, injuries were to be found in the periodontal tissues.

Miller lists these diagnostic signs of traumatic occlusion:-
1. Traumatic crescents.
2. Congestion of marginal gingivae. 
3. Recession.
4. McCall's festoons.
5. Stillman's clefts.
6. Absence of stippling.
7. Increased depth of sulcus.
8. Epithelial nodules.
9. Varying degrees of mobility.
12. Distended veins in the mucosa.
13. Pus in the crevicular exudate.

Other possible sequelae are:

1. Cemental tears. Fragments of cementum are torn off due to violent occlusal trauma. The tear may be reunited to the root by secondary cementum or drift away.

2. Root resorption. Teeth resist resorption much better than bone but it does occur in some patients (14). Weinsmann and Sichin describe increased forces that are within the limits of tolerance leading to deposition of new bone, if beyond these limits then resorption occurs. Also if pressure is applied to an area which is normally under tension, then rapid resorption takes place. (15)

Gottlieb stresses the importance of the position of the mandible being free of occlusal contact. He points out that a maximum of one hour is involved for actual mastication of food during a twenty-four hour period and that this is not sufficient length of time to create damage. Therefore, occlusal trauma occurs between meals. (16)

Gratzinger refers to dynamic irritation as a chronic irritation caused by lateral forces. These may be induced where there is cusp interference by the muscles of mastication during sleep.
Also if a lack of balance exists within the musculature of the mouth, and this would be acting all the time, "dynamic irritation" can result.

Radiographically an early sign of occlusal trauma may be a widening of the periodontal membrane at the apex of the tooth. With tooth contact mesially and distally, no correlation between occlusal disharmonies and the X-ray appearance of the interproximal crests is possible but if bucco-lingual movement has existed for a long time, a widening of the periodontal space may be seen round the entire tooth. According to Orban and Ritchey the most frequent occlusal disharmony occurs when the mandible must be moved anteriorly to true centric to give maximum inter-cuspation - "functional mesial displacement of the mandible". In this relationship stresses are induced distally in the upper teeth and mesially in the lowers.

NON-OCCCLUSION.

Definition.- "Non-occlusion is a condition in which one or more teeth cannot be brought into contact with their opponents in any position which the mandible may assume."

It may be caused by congenital factors but more commonly by factors which influence growth as the teeth are erupting.

As function diminishes there is degeneration of the periodontal membrane and rarefaction of the alveolar bone (disuse atrophy).

The principal fibres become irregular in their arrangement. The periodontal membrane is reduced in thickness. The cementum becomes thicker but is lacking Sharpey's fibres and there are less trabeculae in the supporting bone.

The periodontal membrane of a functionless tooth of long standing consists largely of a very loose tissue with less cells and decrease in the number of fibres. Box
calls this "adaptive fibropenia".

Also due to lack of stimulation there is loss of tissue tone and so increased susceptibility to disease.

(18)
Dummett lists these features of discus atrophy.-

1. Extrusion of teeth.
2. Increased thermal sensitivity.
3. Increased cementum which is lacking in Sharpey's fibres.
4. Reduced bulk of bone.
5. Supporting bone replaced by fatty marrow.
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CHAPTER VI.

DANGERS OF PERIODONTAL DISEASE.

(1)

Fish says there are three possible ways in which Periodontal Disease may lead to sequellae.

1. By direct spread and by possible inhalation or swallowing of pus. He mentions these possibilities: streptococcal fissure of the lips, gastric ulcer, sinus infection, lung infection, extension into the bone causing osteomyelitis, extension of infection along facial planes.

2. Bacteraemia with possible infection of damaged heart valves, or puerperal sepsis. He says that a constantly recurring bacterial shower of haemolytic streptococci may produce an anaemia by haemolysis, even though the organisms are destroyed.

3. Toxic absorption.

Fish points out the extensive surface area concerned with periodontal disease and says it is unlikely that the waste products of the organisms' metabolism are wholly innocuous to man.

FOCI OF INFECTION AND FOCAL INFECTION.

(2)

Focal infection is a secondary infection in a distant tissue or organ caused by an invasion of the blood stream by micro-organisms from a primary focus.

According to Appleton the development of secondary lesions traceable to the primary focus constitutes focal infection. He also said: "any localized infection is potentially a focus of infection".

In 1935 O'Kell & Elliott demonstrated the presence of streptococcal bacteraemia following tooth extraction. Most of the organisms were streptococci viridans.

Bacteraemia was also shown by Round et al following
mastication in septic mouths.

Burket & Burn demonstrated bacteraemia by suspending a non-pathogen in the gingival crevice. After extraction, the organism was found in the circulating blood.

In 1951 a complete issue of an American Journal was devoted to an evaluation of the effect of dental foci of infection on health.

In this the following points were made:

By means of the blood and lymph streams, spread of bacteria and bacterial toxins is possible.

The theory of "elective localization" put forward by Rosenow in 1930 has not sufficient scientific support. The defences of the body against bacterial infection are anti-bacterial and anti-toxic and in most patients a transient bacteraemia would be eliminated within ten minutes. There is considerable evidence that sensitization of the tissues to specific bacteria occurs. This may be the explanation of rheumatic fever and rheumatoid arthritis.

The bacteria in infected pulp canals are mainly strep. viridans, strep. anhaemolyticus and staph. albus. Rarely are haemolytic strep. to be found. It is strange that in a comprehensive article as this, more mention is not made of the importance of periodontal pockets, their surface area, blood supply and various bacteria. It would seem likely that with tooth mobility, jagged edges of calculus and the presence of many blood vessels, bacterial showers would occur more readily than from apical infection.

(5)

Denton found slightly more than half the granulomas he studied were infected.

(7)

Sands found these organisms in deep pockets:

Strep, viridans, strep. haemolyticus, staph. aureus and staph. albus and amoebae.

Referring again to the evaluation of dental foci.

Joint Diseases.

1. In Arthritis due to specific infection oral sepsis plays no part.
2. In Arthritis due to rheumatic fever further research is necessary to correlate with oral sepsis.
3. Rheumatoid Arthritis is of unknown etiology.
4. In Osteoarthritis infection plays no part.
   Likewise with Traumatic and Gouty Arthritis,
5. In non-articular rheumatism there is no evidence of correlation with oral sepsis.

HEART DISEASE:

Heart valves may be damaged by:
1. Congenital defects.
2. Rheumatic fever. This is probably the most common cause.
3. Initial bacterial injury, mainly by haemolytic streptococci.
4. By secondary bacterial infection, usually by strep. viridans.

So, since transient bacteraemia has been shown to occur, and strep. viridans organisms are common residents of the mouth and infected pulp canals, sub-acute bacterial endocarditis resulting from an oral septic focus is a very real danger in patients with a rheumatic heart.

RENAL DISEASE:

There seems to be little to support the claims that dental focal infection plays a role in urinary tract infections.

OCULAR DISEASE & DERMATOSIS:

Here again more scientific evidence is required to correlate dental focal infection with these lesions.

Summing up the above it would appear that earlier claims re focal infection were exaggerated, however, this should not imply that oral foci can be dismissed as harmless as they do represent a potential hazard to the patient's health.
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CHAPTER VII.
TREATMENT.

Thorough examination and recording is essential to a correct diagnosis and treatment planning.

The face should be studied for contour and any swellings or lip lesions noted. Intra- orally the cheeks, palate, fauces, tongue, gingiva, occlusion and arch formation should be examined. Colour changes, ulcers, fistulae, swellings, calculus, pus and any other abnormality must be recognised and noted. Mobility of the teeth may be observed by percussion and during masticatory movements; hidden calculus can often be discovered by trans-illumination and probing.

Radiographs are essential and usually fourteen films plus two bite-wings is indicated. Plaster models are helpful for the study of occlusion.

Systemic and dental history should be obtained from the patient and noted as well as age, sex, occupation, general hygiene and temperament. An inquiry into the diet is often of assistance in diagnosis. (Collaboration with a Physician is indicated when systemic etiology is suspected.

Other steps that may be necessary are:

1. Blood examinations (cell count, haemoglobin estimation, bleeding and coagulation times, sedimentation tests, blood pressure, blood chemistry and Wasserman and Kahn tests.

2. Biopsy of suspicious tissue.

3. Bacteriological tests.

4. Urinalysis.

For recording pocket depths the radiograph is misleading unless gutta-percha points are inserted in the pockets beforehand and the use of graduated probes is suggested by most authors.
The treatment for gingivitis caused by local irritants is based on firstly, the removal of the source of irritation, then by improving the tissue tone till a healthy condition is achieved, and thirdly, to instigate procedures to maintain healthy gingivae.

Removal of the Cause—

Calculus must be removed by the use of scalers, chisels and planing instruments (hoes and curettes). (1) Miller gives four rules for instrumentation.—

1. Use a pen grasp.
2. Have a definite finger rest - preferably a tooth.
3. With the hoe scalers have two point contact, i.e., the blade and shank in contact with the tooth simultaneously.
4. Use a full arm movement and not just finger movement.

He stresses the need for development of tactile sense, especially for calculus which is not visible.

Scaling is followed by careful polishing with rubber caps or brushes and then by using a portee-polisher with pumice and water.

Talbott's solution of iodine is used as a disclosing solution for mucinous plaques. It is swabbed over the crowns of the teeth and the mouth is rinsed once.

For topical anaesthesia before scaling 10% Butyn may be used (flow into the sulcus and leave for 5 minutes), or a Benzocaine solution, or 5% Zyclacaine ointment.

Drugs which may be used for gingival oedema are:—
1. 50% Tannic acid in glycerine. Apply for ten minutes and then rinse the mouth with 5% saline for ten minutes.
2. 5% saline used as a mouthwash four times daily.

Overhanging margins of restorations, cavities, loss
of marginal ridges and badly fitting prostheses must all be corrected. Any unsuitable toothbrushing technique must be eliminated and the patient must be instructed now to massage the gingivae with the toothbrush by one of the recognised techniques (see below).

To assist in the prevention of further calculus deposits it is suggested that the food intake be reduced, the food be thoroughly masticated, and fruit be taken after meals to stimulate flow of saliva.

Fish says there is a definite type of chronic marginal gingivitis where only the interproximal spaces in the posterior region are affected. Here it is often necessary to excise the lingual papillae if they are detached. This is done with a gingivectomy knife after local anaesthetic infiltration and the area is then packed with a zinc oxide, eugenol and cotton wool pack and left for one week. Removal of pack is followed by immediate stimulation with a wood point — this will be discussed later with gingivectomy.

Several authors advocate oxygen therapy to improve tissue tone. Box favours the Dunlop insufflation technique. Several other authors consider oxygen therapy quite helpful but Rosenthal says the action of oxygen liberators is purely mechanical. Leonard says the use of Superoxol (30% H2O2) only affects local anaesthesia and that better results are obtained by the use of toothbrush massage.

Gingival Massage —

Lyons says there is no scientific proof that massage is of benefit in periodontal treatment and that spectacular results can be obtained without massage. Few agree with this theory.

Hirschfeld says that toothbrushing plays a major role
as a preventive and therapeutic measure in chronic marginal gingivitis, chronic gingival hyperplasia and periodontitis.

He lists as contra-indications:–
Acute ulcerative gingivitis, tuberculous lesions, aphthous stomatitis, chemical burns, allergic reactions, chronic desquamative gingivitis, scurvy, pemphigus and certain blood dyscrasias.

There is also difference of opinion as to whether hornification of the gingivae does occur. Pelzer, Orban, Rodecker, Hoyes, Schour and Fish all agree that it does while Dremer, Weatherford, Maximow and Bloom do not. (12)

Stahl et al found that increased massage led to an increase in keratin and that hard natural bristles produced more keratin than soft multitufted nylon brushes.

Gingival massage is contra-indicated in Vincent's Infection and other acute conditions such as acute periodontal abscess. It is also ineffective if sub-gingival calculus is present. Otherwise massage is desirable for these reasons:–

1. To improve the circulation of the area thereby removing waste products and improving the tissue metabolism.

2. To increase the keratinization of the gingivae.

Aims of Toothbrushing:–

1. To remove all debris from teeth.
2. To gently massage gingival tissue.
3. Should not irritate or lacerate the tissue.

Coolidge and Hine recommend a straight, semi-rigid handle about 6 inches long, the head 1 inch long and ½ inch bristles arranged in two rows, with six tufts in each of uniform length.
They prefer bristle to nylon although the stiffness of nylon would be more easily standardised, they last longer and do not become soggy. They classify .010 diam. bristles (nylon) as soft and .016 extra stiff with .012 medium.

Phillips says that the abrasiveness of dentifrices on enamel is accentuated by nylon.

Goldman and Miller say the bristles should be stiff enough to transmit pressure through to the gums.

Since the introduction of nylon brushes there has been a distinct tendency for patients to choose a very hard type of brush, and it is rather questionable whether this is more suitable than a soft brush. Recently brushes with much finer nylon strands have been available and these seem better for gingival massage and also remove food debris quite effectively.

TECHNIQUES.

1. Scrub Brush.

The brush is grasped firmly and ends of bristles are rubbed vigorously over all surfaces of the teeth and gums -- this is recommended only for occlusal surfaces.

2. Fong's Technique.

The teeth are held in occlusion and the brush revolved in circles of large diameter. This is useful for children but does not massage interproximal areas very well.

3. Roll Technique.

The brush is placed as far apically as possible with the sides of the bristles touching the tissues and then using lateral pressure the brush is rolled slowly occlusalward. This gives quite good massage and also cleanses the teeth adequately.

4. Vibratory Techniques.

a. Charters'.

The ends of the bristles are in contact with the enamel and at about 45° pointing occlusalward. Using downward (in the case of lower) and lateral pressure, the brush is vibrated thus massaging the gum with the
sides of the bristles. Do three or four movements then remove brush, then repeat. Move one embrasure at a time.

5. Stillman and McCall.

The brush is placed with the ends of the bristles partly on the teeth and partly on the gingivae and pointing apically. Enough pressure to bend the bristles slightly is then given, which causes blanching of the tissue. The brush is then lifted, allowing rapid inrush of blood. This is repeated several times and the handle may be given a slight rotary motion but not enough to move the ends of the bristles.

A modification of this is to move the brush occlusalward, thus the stroke starts in the gingivae and ends at the incisal edge.


The bristles are placed almost at right angles to the long axis of the tooth but pointing slightly towards the incisal edges with some bristles on gingivae and some on tooth. The brush is then rotated in tiny circles ten times in each position. Occlusal brushing by scrub technique and the bristles removed from contact at the end of each stroke.

7. Physiologic Technique.

Using a soft, camel hair brush with 76 tufts arranged in four rows, the teeth are brushed from crown to root in a gentle sweeping motion.

8. A combination of the "Roll" technique and vibratory.

Coolidge and Hine recommend firstly the scrub brush technique on the occlusal surfaces.

For children Fone's technique.

For young adults - the "roll" or if periodontal conditions present, the "combination".

It should be done systematically and thoroughly for 3-5 minutes after each meal.
Goldman stresses the importance of thorough rinsing afterwards for its cleansing effect and also slight stimulation.

Rubber cones and wood points may be used for stimulation interproximally only when there is sufficient space. Miller considers the rubber brushes or the fingers for massage quite useless.

Leonard considers benefit is only obtained by a pressure and release technique and that any scrubbing action with the ends of the bristles should be avoided.

Lundquist uses 8% zinc chloride on cotton wool which is packed along the gingival crevices till blanching of the tissues occurs. This is followed by saline mouthwashes and massage. He claims this will eliminate the lymphatic block and promotes a good flow of lymph.

TREATMENT OF PERIODONTITIS.

According to Fish there are two possibilities here:-

1. Heal the ulcers at the base of the pocket and keep the new epithelial lining intact, or,

2. Obliterate the pockets.

Reviewing the literature, it seems that possible effects of the different forms of treatment are as follows:-

1. Treatment based on prophylaxis and sub-gingival curettage whereby all deposits are removed from the teeth. The possible results from this are:-

   a. The formation of an intact epithelial lining of the pocket or,

   b. Close adaptation of the soft tissues about the neck of the teeth which results in a clinical closure of the pocket, or,

   c. Organic reattachment of the periodontal tissues to the teeth.

   d. Recession of the gingival margin which results in either elimination of or decrease in depth of the pocket.
2. Removal of soft tissue (and in certain cases bone) to the base of the pocket hereby affecting pocket elimination. This may be done by surgical means, electro-surgery or by chemical action.

Sub-gingival Curettage.

Miller's routine for this consists of removal of gross calculus with sealers, prophylactic chisels, then small remaining particles of calculus are removed using the hoe planers and curette, using less pressure than for gross deposits.

Next the hoes and curettes are re-sharpened, and, using light pressure and long up and down strokes the root surface is planed. Strokes should overlap and the direction should be changed from time to time. This is followed by the use of the porte-polisher with pumice and water, after disclosing solution has been swabbed around the teeth.

The soft tissue must also be thoroughly curetted to remove all epithelium and granulation tissue using very sharp curettes. Topical anaesthesia may be used for this. In pockets deeper than 3 mms. Miller advises using sodium sulphide as an epithelial solvent.

Upon completion of curettage and when a blood clot has formed the area is dried and periodontal varnish is applied and allowed to dry for one minute.

(18)

Goldman suggests that the use of the toothbrush be prohibited for 5-7 days till new tissue is formed. He says the main difficulty lies in cleansing the cementum to the entire depth of the pocket.

The "Flap" and "semi-flap" operations are essentially the same as above but the field is laid open. In the full flap operation incisions are made from the gingival margin to the muco-buccal fold. The interproximal papillae are incised mesio-distally and, using a periosteal elevator, the flap is detached from the alveolar bone. All deposits
granulations and epithelial lining are then removed using curettes, scalers. After irrigation the tissue is readapted and sutured interproximally. Goldman says adhesive tinfoil may be used to protect the area. Ingle also uses this technique but he also removed the marginal edge with scissors before readapting the soft tissues.

The semi-flap operation is used where sufficient access can be obtained without involving vertical incisions.

Box uses a paraffin packing as an adjunct to curettage and also oxygen insufflation. These are the advantages he gives for the paraffin pack.

1. Separates the inflamed soft tissues from the dental wall with its calculus and septic surfaces.
2. Through pressure it reduces gingival swelling.
3. It facilitates scaling.
4. Prevents saliva and food entering pocket.
5. Its medicaments tend to inhibit anaerobic bacteria, are soothing to the tissues, and stimulate circulation.

Cripps uses a pack of zinc oxide, resin, tannic acid, asbestos and eugenol which he changes every 48 hours.

Reattachment.

This has been a much debated aspect of periodontal treatment whether the supporting tissues of teeth with periodontal pockets can be induced to reattach themselves by an organic union to the tooth. Some evidence has been put forward that reattachment can occur.

Fish says reattachment of epithelium is a physiological impossibility and that if a healthy gum margin be surgically detached from a tooth some degree of reattachment can be secured in the deepest part of the lesion but it is impossible to secure reattachment of inflamed gum to the infected surface of a tooth, even if that surface could be completely curetted, which is impossible.
Merritt says it is probably true that reattachment and complete elimination of the pocket is the exception rather than the rule but that excellent results are obtained by sub-gingival curettage though as a rule a pocket remains which may possibly be reinfected later.

Orban says that reattachment apically to the bottom of the pocket is a probability after detachment, but that results do not justify the efforts required in attempting reattachment of the pocket wall. Though the cementum above the pocket base is non vital this does not eliminate the possibility of reattachment.

Goldman does not think reattachment possible in a gingival pocket, but says that it is possible in an intra-bony pocket. He advocates removal of the epithelial attachment and says that there is then a race between the epithelium and the connective tissue, and that if the epithelium wins no attachment is possible.

Kronfeld considered reattachment of the detached gingival tissues very doubtful "since the process is of limited extent in experimental animals where the tissues were normal before operation, it appears unlikely to be successful in the clinical pyorrhcea pocket where the exposed cementum has become penetrated by the products of bacterial invasion".

Beube demonstrated reattachment in dogs' gingivae after interference with normal gingivae and also reattachment in humans with periodontal pockets after curettage and introduction of boiled cow bone powder before readapting the tissues.

Leonard says that in treating a pocket without disturbing the epithelial attachment, one of these may result.

1. The pocket remains as a deepened sulcus.
2. The gingiva shrinks till a normal depth is reached.
3. The epithelial lining reattaches itself to the tooth.
In regard to 3, he states that photo-micrographs by Box show this histologically, and that others have observed it clinically. (It should seem difficult to differentiate between connective tissue reattachment and epithelial reattachment in a clinical examination).

Blass states, in a discussion on this, that crownward growth of epithelium following surgical removal of the soft tissue side of a pocket is a constant observation.

Goldman says this will need substantial histological evidence and points out the difficulty of examination (histologic), both before and after treatment.

Friedlander says there are two possible types of reattachment, one by epithelium and one by fibrous connective tissue. He questions whether epithelial reattachment is acceptable as successful treatment and says that connective tissue reattachment is more desirable.

Orban, in reply to Leonard's statement that Box had shown reattachment of epithelium, pointed out that this was not the original pocket epithelium, but proliferating epithelium of the wound surface. He (Orban) also doubts that epithelium can grow crownward. He evidently does not accept the possibility of reattachment of the original epithelium lining the pocket. Kirkland was in full agreement with this view. (32)

Bucholls and Dienstein describe this healing process:-

After establishment of a blood clot on the pocket there is a lowered pH which causes rarefaction of the alveolar bone near the pocket and the surface of the cementum. The clot becomes organised and fibroblasts and endothelial sprouts grow from the soft tissue side. The elementary fibroblasts on the bone side become osteoblasts and those approximating the cementum become cementoblasts. (33) (34)

Studies on dogs showed deposition of new cementum either on to old cementum or dentine, which effects new attachment of connective tissue fibres. These new fibres tended to run parallel to the tooth axis. Some regeneration of the
alveolar bone also occurred. There are two theories of how this can occur. According to one the new bone cells come from existing bone, and the other suggests that cells from connective tissue become osteoblasts. The authors of this article think the evidence is in favour of the former.

(33) In an experiment by Ramfjord on Rhesus monkeys, he produced periodontal pockets by adapting copper bands to the teeth. After removal of the bands and curettage of the pockets, attachment of both connective tissue and epithelium was found. One interesting finding was that in some cases, alternating zones of connective tissue and epithelial attachment were seen. The new connective tissue attachments to the root were always associated with cementoblastic activity.

He concluded that:

1. Both connective tissue and epithelial reattachment was observed on root surfaces that had been exposed in an inflamed periodontal pocket.
2. Removal of the epithelial lining was necessary to obtain reattachment.
3. The degree of inflammation in the healing area seems to determine whether an epithelial or connective tissue reattachment would occur. Less inflammation seemed to favour connective tissue reattachment.

(36) His routine after prophylaxis and a period of home massage, is to pack the pocket with 75% phenol and 25% camphor for five minutes, which is then followed by 50% alcohol and then warm water irrigation, after curettage and warm water irrigation the area is isolated for ten minutes before exposing to the saliva. He does not use a varnish as it may tend to flow along the tooth surface, thus separating it from the blood clot.

Ramfjord says any granulation tissue present in a pocket probably loses its inflammatory character after prophylaxis and massage therapy, and therefore considers
it should not be disturbed during curettage. He seems unsupported in this idea as other authors mostly suggest removal of all granulations.

Another epithelial solvent used by some is a solution of sodium sulphide. Lyons, Leonard and Merritt consider epithelial solvents are contra-indicated in that they may do more damage than good. Ramfjord considers a closure of 2-3 mms. a good result, but Shapiro has demonstrated pocket closures of up to 7 mms.

His technique was to remove up to 1/3 of the gingival tissue in conjunction with curettage, which extended a little below the base of the pocket. He applied a soft pack to the area for a fortnight. He submits evidence that a possible 83% (of 36 cases) showed some reattachment.

Barkman, Goldman and Schofield also excised some of the papillae with an inward bevel. The reason for this is to retard the proliferation of epithelium towards the tooth, to allow a better chance of connective tissue reattachment.

Waerhaug's work on the epithelial attachment seems to indicate that sub-gingival curettage is sound practice and that if calculus and other debris is removed from the root surface (and he admits that it is a very difficult procedure) a "readhesion" of the gingival tissues to the tooth can take place.

In attempting reattachment there is also argument whether the epithelial attachment should be interfered with. Kirkland, Ingle, Glickman, Friedlander and Ramfjord all advocate complete removal of the epithelial attachment. The reason being that proliferation may take place along the wound surface before the connective tissue can become attached. On the other hand Orban and Ritchey disagree with this on the grounds that if a failure results, the tooth will then have a shorter clinical root.

Riffle says that in sub-gingival curettage of the tooth
surface it is impossible to plane to a smooth glassy surface without removing all the cementum. He has achieved better results by deliberately removing the exposed cementum.

**SURGICAL ELIMINATION OF THE PERIODONTAL POCKET.**

Gingivectomy is the treatment of choice of some operators, while others make it a last resort after other methods fail. (52)

Miller favours radical surgery when there is bifurcation involvement or when the pockets do not reduce to less than two millimetres depth after conservative treatment. (55)

Coolidge and Hine advocate gingivectomy where the sulci are so deep that suppuration persists after conservative treatment.

Most authors prefer to establish a more healthy condition in the mouth by prophylaxis and toothbrush stimulation before attempting gingivectomy. This seems a sound scheme as areas of oedema will reduce and less extensive tissue removal will result. Box stresses the establishment of a widespread sanitary influence in the mouth before any attempt is made to treat pockets.

In techniques described by Ward, Crane & Kaplan and Merritt, removal of interproximal tissue down to the alveolar crest and subsequent curettage of the bone is advised. Orban, Ramfjord, and Goldman say this is contraindicated. Orban does not disturb the epithelial attachment but Ramfjord removes it completely. (57)

Ward prepares the patient by swabbing the face with 70% alcohol, places a large apron over the body and a towel over the head. The mouth is sprayed with a mild antiseptic and the mucosa painted with weak iodine solution. After removal of tissue (using instruments of his own design) all operated surfaces are cauterised with pure phenol which is then neutralized with alcohol. Wonderpak is then placed in position. No irrigation or spraying is done during the operation.
Fish removes the gingivae to the depth of the pockets and bevels the labial surfaces. He aims at producing a scalloped effect for aesthetic reasons. He also suggests that in certain cases a millimetre or two of detached labial gum margin may be left—also for aesthetics—which the patient must later keep stimulated with wood points. Fish says that the presence of calculus, unless very gross, makes no difference to the operation. Others, however, consider that thorough scaling should be carried out before attempting gingivectomy (not the same appointment). Fish warns against any instrumentation before injection of local anaesthetic.

After surgery, Crane & Kaplan swab the area with $\text{H}_2\text{O}_2$ and then spray with a powerful atomizer spray. Black fills the interproximal spaces with pellets saturated with 8% zinc chloride.

Kirkland applies hot saline pads before the pack, to improve circulation. Different packs which may be used are:

- Wonderpack. (Ward incorporates an equal bulk of tannic acid into the pack.

- Zinc oxide and resin (equal parts) mixed with heavy mineral oil and eugenol (equal parts).

- Sterile white beeswax.

- S.S. White Impression Paste.

Fish uses zinc oxide and oil of cloves into which wisps of cotton wool are incorporated and for the end teeth of a series, or isolated teeth, scarves of gauze impregnated with the zinc oxide and oil of cloves mixture.

Linghorne & O'Connell in a study of the therapeutic properties of zinc oxide and eugenol packs concluded that-

1. Such a pack is an effective bacteriostatic agent against staph. aureus, strep. viridans, strep. haemolyticus and micrococcus catarhalis.

2. The diffusion of bacteriostatic agent from the pack into the surrounding area lasts longer than
is required in case management.

3. The pack acts as a local analgesic and stimulant.

4. The addition of 5% sulpathiazole or penicillin to the pack does not seem to increase its bacteriostatic effectiveness.

5. It is an effective fungicide against thrush organisms. Packs are left for 10-14 days and longer if necessary. Goldman changes the pack every 3 or 4 days.

After removal of dressings any granulation tissue which may have formed is removed by cauterising with 35% trichloroacetic acid or 25% silver nitrate.

As causes of excess granulation tissue formation (62) Ramfjord lists:-

1. Operating on a severely inflamed gingiva.

2. Ragged surgery.

3. Early loss of the pack.

4. Leaving the pack in position too long.

After removal of the pack polish all teeth and instigate stimulation by toothbrush and wood points. Crane and Kaplan advise recalling the patient every second day after removal of packs to spray the mouth and gently massage the new tissue with Talbot's iodine on cotton pellets.

They then suggest the patient should rinse several times daily with mild saline and massage with their fingertips or cotton rolls and soapy toothpaste. After 7-10 days of this, instruct in the use of brush and interproximal stimulators and dismiss for one month.

Kirkland advises immediate brushing and inter-dental stimulation after pack removal with frequent polishing at intervals during the treatment.

Fish also advises immediate massage with a soft badger hair brush and soapy toothpaste. He also says that it is possible in some cases to remove the lingual wall of a pocket by making a V cut so that access is obtained to the pocket from behind and a wood point may be employed for stimulation. He warns, however, that in these cases where
some pocket is left a 2 or 3 month check is imperative.

Fish considers it worth doing gingivectomy first and removing seemingly hopeless teeth later.

Goldman also says that doubtful teeth may be left but any obviously hopeless teeth should be immediately extracted. He lists these factors as suggestive of a poor prognosis.

- Wide periodontal membrane.
- Rarefaction areas around the apex.
- Much loss of alveolar bone.
- Tooth resorption.
- Bone resorption in bifurcation.

Merritt says that often molars with bifurcation involvement can be saved by curetting and later filling the space with black orthophosphate of copper.

Hirschfeld thinks the use of a water syringe by the patient a good scheme if any pockets are left for aesthetic reasons and also if there has been bifurcation involvement.

Fish advocates the use of 2 million units of penicillin intra-muscularly before gingivectomy for promotion of comfortable and rapid healing. He also suggests that after insertion of the pack the patient rinse the mouth every few minutes till the next meal with sodium bicarbonate, to prevent ulceration by eugenol.

Goldman says some removal of alveolar bone may be necessary in the case of intra-bony pockets. He also thinks that this is the only type of pocket where reattachment is possible.

While discussing intra-bony pockets, Gottlieb stated that the alveolar bone or periodontal membrane of a tooth are never present in the area of pocket formation and that the intra-bony pocket is not an intra-alveolar pocket. The alveolar bone of one tooth disappears but the alveolar bone of a neighboring tooth is still in tact. He also states that the alveolar margin moves apically before the downgrowth of epithelium.
Urban & Archer describe this reparative process after gingivectomy - without a protective pack. -

1. The formation of a blood clot.
2. Necrosis of surface of the clot by bacteria and an accumulation of leucocytes below the surface which prevent penetration of bacteria and their toxins.
3. Penetration of capillaries into the fibrin by mitosis of endothelial cells.
4. Rapid multiplication of fibroblasts.
5. A few hours after the operation, polymorphs appear and increase in numbers till the 9th. day, when they decrease.
6. Undifferentiated mesenchyme cells become migratory phagocytes.
7. By the fourteenth day the polymorphs have gone and are replaced by polyblasts. Epithelium has covered the granulation tissue by this time. They found that large clots hindered healing and delayed epithelialization.

With a protective pack, Bernier & Kaplan found the new epithelium had covered the wound in six days and so suggest that packs may be removed after ten days.

**Electrosurgical Pocket Elimination.**

This is done by the coagulatory effect of a high frequency current. General considerations listed by Webb are:

1. If 30% or more attachment is present and no bifurcation involvement, prognosis is favourable.
2. It is contra-indicated in acute infections and in highly inflamed areas.
3. Use a general or local anaesthetic.
4. The operative work in each area is completed in each operation.
5. Divide the mouth into halves or quadrants and do only one section at a sitting.

6. The after pain and discomfort is negligible.

Webb uses a flexible electrode with two silver prongs as the contacts. This, he claims, allows accurate management of coagulation which occurs only between the two poles and in immediately adjacent areas.

Before surgery he has a ten day test period of the patient's ability with a brush using Charter's method, and in the use of interdental stimulators.

An astringent is applied to the area. The electrode points are placed in contact with the tissue in the selected area and the current is then switched on (using a foot switch) just long enough for coagulation.

Interproximal tissue is done by putting a prong on each side of the papilla both buccally and lingually.

After removal of tissue pack for 5-10 days.

Other advantages given by Beatty are:

No bleeding, no swelling, no surgical shock, little granulation, teeth become less sensitive, no packing required (some advise use of packs), a beneficial hyperaemia follows, minimum loss of tissue, no loss of bony tissue, favourable prognosis for deep or shallow lesions, the operation is easy to control, it is a sterile operation, scaling may be done at a later appointment when the calculus is in clear view.

Goldman thinks the wire loop electrode the most efficient and that this may be used for cutting, scalloping, planing or incising. The extent of coagulation depends upon the resistance of the tissues to the passage of current, the current density per square centimetre, and the length of application of the electrode. (71)

Sugerman says "electrosurgical gingivoplasty" is of help in reducing thickened margins after sub-gingival curettage, to eliminate craters after Vincent's Infection, to reposition the frenum, to facilitate brushing, and to achieve better aesthetic effects.
He applies a pack for one week.

Trotter uses short wave electrosurgery for gingivectomy. He uses a smooth nerve breach for an electrode needle and also advises packing the area afterwards.

Merritt thinks electrocoagulation has little place in the treatment of periodontosclerosis as pockets up to 3 millimetres in depth can be treated more satisfactorily by curettage and in cases of deeper pockets they can be done with less pain and more rapidly by surgery.

A gingivectomy may be also performed by chemical cauterization. Pure phenol may be applied for 2 or 3 minutes.

The excess is then wiped off and the area swabbed with a saturated solution of sodium carbonate. Packs are not necessary and in 3-5 days a strip of necrotic tissue comes away. The operation is then repeated until a satisfactory result is obtained. Ramfjord does not think this can be controlled accurately.

Other drugs which may be used for this are trichloracetic acid and a mixture of sodium hydroxide and calcium oxide.

Further Treatment which may be necessary.

Occlusion must be corrected if necessary. Firstly, study models are made and mounted on an articulator. These are then correlated with the radiographs. Mouth checks are made with articulation paper and wax.

Centric occlusion is first corrected for premature contact. Then check protrusion. If it is necessary to increase horizontal overjet the lingual of the upper anteriors should be ground.

In the lateral excursions grinding is only done on the buccal cusps of the upper teeth and on the lingual cusps of the lowers as this will not alter the vertical dimension. Grinding should be done slowly on a moist tooth and the surface is later smoothed with cuttlefish discs.
It is generally agreed that it is better to do too little grinding than too much. Any obvious or gross occlusal defect should probably be corrected as early as possible.

Simple orthodontic procedures may be indicated and partial restorations either of fixed or removable types may be required.

Teeth with short clinical roots or very loose teeth may require splinting either by a fixed or a removable splint.

A temporary splint is often of assistance and can be made from steel ligature wire, waxed silk, grassline, continuous cast splints and acrylic splints.

Fixed splints include ¾ veneers for anteriors which are soldered together. Posterior teeth may have interlocking inlays or inlays soldered together—the whole occlusal surfaces should be covered to avoid dislodging the teeth from the splints.

Permanent removable splints are also of help if teeth are missing. All the teeth in the arch are splinted so that the weaker teeth are supported by the stronger ones.

Merritt thinks splints have only limited place in treatment of periodontal disease except when used for temporary stabilization.

(73)

Periodontosis.

Prognosis here is not very good but sometimes some favourable systemic change may occur and the condition will clear up.

Firstly remove hopeless teeth, then control any infection, conduct prophylaxis and correct occlusion.

A careful check of diet is necessary and co-operation with a physician to check possibility of general disturbance is advised.

The patient is instructed in home stimulation techniques. Pockets may need surgical removal. Goldman
found sub-gingival curettage unsuccessful.

Splints are helpful as often there is a tendency for
the teeth to wander. Vitamin therapy may also be attempted.
(78)

Lakosky has used sodium fluoride systemically
(0.01 gm/day) and says it probably contributes to the
binding of calcium by the tissues, especially bone.

Vincent's Infection.

The plan here is to firstly eradicate the acute
symptoms and then eliminate the factors responsible for
the condition.

Removal of as much debris as is possible without
traumatizing the tissues is recommended by most authors
and this is then followed by the use of various drugs.

Debris may be removed by wiping with cotton wool
saturated with 3% hydrogen peroxide, spraying with hydrogen
peroxide or iodo-saline. Some suggest that a little
scaling may be done but the general opinion is that a
danger of bacteremia indicates cautious manipulation of
the inflamed area. Some deaths have been reported
following extraction of teeth in the presence of acute
Vincent's Infection.

Many drugs have been advocated for elimination of the
acute phase and good results claimed by the different
authors.

Oxidation treatment using hydrogen peroxide, superoxol,
(79) (80) (81) (82) (83)
urea hydrogen peroxide sodium perbonate zinc peroxide
(84)
potassium permanganate and insufflation of pure oxygen is
commonly used.

The basis for the use of oxygen liberators is that the
suspected organisms are anaerobic. Rosenthal, however,
says that their action is a purely mechanical one and
that tests (in vitro) with higher concentrations of
oxygen than could be expected clinically showed no diminution
of motility or growth of organisms.

The use of these drugs does not seem helpful for home
treatment. Caution must be exercised with sodium perbonate
as it is considered rather irritating if used too frequently.

The dyes; methyl violet, gentian violet, brilliant green etc., have been used in aqueous solutions but their effect is a bit uncertain. It has been said that they are more toxic to tissue than to organisms and also that the fusco-spirochetes are resistant to dyes.

Mercury preparations.—metaphen and phenyl-mercuric nitrate are sometimes used — mainly for home mouthwashes.

Perhaps the two most widely used methods are:


Chromic Acid Treatment.

This is used in different strengths varying from 5 to 20% solutions and even crystals of chromic anhydride. The chromic acid is applied to the ulcers which are isolated by using cotton rolls. After a few minutes the mouth is sprayed or swabbed with hydrogen peroxide (5%). Probably the greater dilutions of chromic acid are to be preferred as it has been found that an 8% solution will etch enamel and also that the more caustic a drug is, the less the penetration.

Silver Iodide Treatment. (Adams).

Churchill's or Talbot's solution of iodine is first applied to the ulcers and then allowed to dry. A solution of silver nitrate (10-25%) is then carefully applied to the same area. The mouth is then rinsed well.

The above treatments are all usually necessary daily for a few days, and in severe cases twice daily would seem advisable.

The use of arsenicals such as arsphenamine, neosalvarsan and mapharsan is advised by some, others consider they are contra-indicated. Fish and Box suggest that a pack of zinc oxide and eugenol or paraffin is useful for the relief of pain and quicker tissue repair. Fish applies a pack if ulceration
has not gone after the third day.

The antibiotics have been used quite a lot in recent years. Goldman, in his textbook, advocates penicillin troches or in the form of an adhesive paste. Goldman & Bloom also used 250 milligrams of aureomycin every four hours and reported that after the first day all subjective symptoms had gone and healing was well under way at the end of the second day.

MacGregor & Long using penicillin lozenges each containing 250-500 units, concluded that this is the easiest and safest treatment of Vincent's Infection.

Fairchild reported an average of forty-six hours required to cure thirty-two cases. He used a spray containing 300 units/cc of water and a topical application of 100,000 units/cc.

Daniels considers there is little to support the use of sulphonamides. He gives two disadvantages for penicillin:

1. Sensitivity to topical application occurs in about 10% of cases.
2. It prolongs the clotting time of blood.

Tyrothricin is a useful substitute for penicillin in cases of sensitivity.

Another factor that should be considered is that over the period during which penicillin has been used, drug resistant strains have been developed. It seems desirable therefore, to use it only if positively indicated and then to use large doses.

Rosenthal says that streptomycin (1/10 gm./cc) applied topically with a syringe permeates debris better than penicillin.

Bacitracin, a new antibiotic, seems to have less tendency to develop drug resistant strains.

Hirschfeld stresses the importance of thorough application of the drugs. He says that probably the particular drug used is less important than the manner in which it is applied.
Lyons also attaches more importance to debridement than to the use of drugs. He says it changes the environment of the organisms.

Intravenous injections of neo-xanomacine, mapharman and bismuth in oil have been used for acute Vincent's infection.

Some consider injections tend to produce a chronic form.

In contrast to the above reports on the use of antibiotics, Yeretsky says that sulfa drugs and penicillin have no effect on the Vincent's organisms. However, she considers that often a mixed infection is the causative agent and advises the use of sulfanearazine.

The discovery of modern antibiotic drugs led to rather indiscriminate use for a time, but with the realization of some disadvantages and potential hazards they are now being used somewhat more reservedly.

Ostrander mentions these points which should be kept in mind before prescribing certain drugs.

1. With penicillin, epithelial structures are more readily sensitized than deeper surfaces. Painful oral reactions can occur, and resistant strains of organisms -- especially staphylococci -- may develop.

2. Tyrothricin is inhibited somewhat by saliva.

3. Chloromycetin may lead to aplastic anæmia.

Francis suggests that the condition is often complicated by fungi and uses a mouthwash of copper sulphate 1/1000 for this reason, following the use of chromic acid and peroxide.

Home treatment is most important, light diet, plenty of liquids including diluted orange juice, mild cathartics, plenty of rest, elimination of smoking and taking of alcohol, are all advised.

No toothbrushing should be attempted during the acute stage and eating and drinking utensils should be boiled.
as a precautionary measure.

Bliss & Goldman recommend hot irrigations to be conducted by the patient.

Vitamin therapy, mainly B and C may be of value. If, after inquiring into the patient's diet, a deficiency is suspected, multi-vitamin therapy would probably be sound treatment.

Follow up treatment consists of a definite massaging technique, balancing of occlusion if necessary and the elimination of pockets, flaps and hopelessly diseased teeth.

CORRECTION OF NUTRITIONAL DEFECTS AND VITAMIN THERAPY.

Sinclair advocated administration of vitamin A and B complex in all cases of periodontitis and Vincent's Infection. (108)

Campbell attaches much importance to Vitamin C and claims he has cured cases of gingivitis using this alone without any local treatment. He says 2000 mgs. are required to produce saturation.

Therapeutic doses of Vitamins when a deficiency exists suggested by Radusch are:

Vitamin C - 300 mgms. for 4-14 days, then reduce to 50-100 mgms.

Nicotinic Acid - 300 mgms/day for 5-7 days, then reduce to 100 mgms.

Riboflavin - 15 mgms/day reducing to 3 mgms.

Vitamin D - 600-1000 i u /day plus milk.

She also advises supplementing doses of Vitamins in cases of unsatisfactory or restricted diet, food fads, hyperthyroidism, pregnancy, lactation and periods of unusual physical exertion.

The Calcium and Phosphorus content of the diet is also important, daily requirements being about 70 mgms. of calcium and 140 mgms. of phosphorus.
Brauer says a growing child needs 1gm. of calcium/day which may be obtained from a quart of milk. This is probably the best source of calcium. Deficiency in protein with excess carbohydrate seems a fairly common dietary fault. As Leonard points out, an excessive consumption of sugar increases bacterial action and reduces the appetite. Also extra phosphorus and thiamin is required for metabolism. Herzog and McCall advise the use of thiamin chloride (100 mgs in 1cc solution intramuscularly) to reduce pain and promote healing after gingivectomy.

Home Care after Periodontal Treatment.

Home care by the patient is an important part of treatment stressed by all authors. This includes regular and efficient toothbrushing and gingival massage. Wooden points, floss-silk, dental tape, pipe-cleaners, rubber stimulators, mouth sprayers, all have their application in keeping the oral tissues in a state of health. Regular checkups are indicated, some cases will need re-calling more often than others.

As has been mentioned dietary deficiencies of the patient if they exist, should be corrected. Not only the chemical consistency of the food but also the physical nature of it is important. Soft sticky foods should be avoided and an attempt made to include more fibrous food in the diet. Calculus may be controlled by regular brushing after meals. Other suggested methods of prevention are vigorous mastication, brushing with a saline cathartic, eating fruit after meals and if indicated reduce the quantity of food taken at each meal.

Sodium Hexameta phosphate has been used (as a dentifrice 15 parts to 85 parts tals) to remove calculus deposits and prevent plaque formation.

Ammonium hydrogen fluoride has also been found to soften calculus but is now considered too dangerous a drug for this purpose.
Desquamative Gingivitis.

Surgery has been recommended by some and the affected tissue is all removed by cutting back to the healthy tissue and exposing alveolar bone.

After this a protective pack is applied for a week or two. After removal of the pack a technique of gum stimulation is commenced.

Beechwood creosote and iodine (Sorrin) metamphen and Vitamin therapy (Yeretsky) have also been used. Ziskin (116) and Silvers cured a case by the use of oestrogens (Progynon ointment) which was applied on a splint for periods of forty-five minutes.
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<table>
<thead>
<tr>
<th>Reference</th>
<th>Author(s)</th>
<th>Title</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
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</tr>
<tr>
<td>(113)</td>
<td>Gollidze, E.P. &amp; Mine, M. K.</td>
<td>Periodontia</td>
<td>pp. 70-2</td>
</tr>
<tr>
<td>(114)</td>
<td>Prinz, H. &amp; Greenbaum, S. B.</td>
<td>Diseases of the Mouth and Their Treatment</td>
<td>pp. 169-70</td>
</tr>
<tr>
<td>(115)</td>
<td>Merritt, A.H.</td>
<td>Periodontal Diseases</td>
<td>p. 62</td>
</tr>
</tbody>
</table>

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CHAPTER VIII.
OTHER ORAL LESIONS.

THRUSH:

This is caused by an infection by the yeast-like organism, Monilia Albicans most commonly occurring in children or in adults late in life.

According to Woodburne, Monilia grow best in an acid medium so an acidic saliva favours it. It is a weak pathogen but is very persistent when once established. Vitamin B complex deficiency and chronic debilitating diseases are predisposing factors.

In children an acute form occurs and white flakes of membrane covering the tongue, lips, gums and buccal mucous membrane are to be seen.

Coolidge & Hine describe soft, white, dense, elevated plaques which, when wiped away leave a tender, coining surface.

It is sometimes hard to diagnose and prolonged bacteriological tests may be necessary.

In adults subacute or chronic form occurs. The subacute type shows white or grey membranes on the buccal mucous membrane and palate. These begin as small dots and later increase in size. The surrounding mucous membrane is erythematous and when the adherent membrane is removed, a brightly inflamed area is seen which oozes blood. There is rapid regeneration of the membrane.

In the chronic form there is a diffuse redness and dryness of the oral mucous membrane and occasional flecks of white membrane.

The tongue is dry and fissured, the saliva sticky and there is a dry, burning sensation.

Treatment:-

Vitamin administration may be required, prophylaxis and alkaline mouth washes are desirable. Gentian violet 1-4%, Lugols iodine, Silver Nitrate 2-10% and merthiolate 1:1000 have all been used topically.
Internally, 2-4 grains of potassium iodide daily has been used in more severe cases.\(^{(4)}\)

Hardgrove says the longer a yeast infection has been in the mouth the harder it is to cure. He says the Monilia Albicans can penetrate into connective tissue and muscle and considers potassium iodide the drug of choice\(^{(2 \text{ grains four times daily})}\), gradually increasing the daily dose.\(^{(5)}\)

**LEUKOPLAKIA:**

This is a chronic inflammation of the mucous membrane most frequently seen in middle aged men. It develops slowly and painlessly and starts as a red, granular, sharply defined area which becomes whitish-grey and later bluish-white. At this stage it is sharply outlined from normal mucosa. Later the surface may become elevated and warty in appearance. Dryness of the mouth and excessive thirst is common.\(^{(6)}\)

There is a danger of malignancy, Kronfeld says 20-30% of oral squamous carcinomata come from a pre-existing leukoplakia.\(^{(7)}\)

The disease may occur without any obvious local irritating factors. Goldman says there is evidence that it may be caused by general metabolic disturbances such as endocrine dysfunction and Vitamin deficiency and that he has observed similar lesions during the menopause.

Other etiological factors are, excessive tobacco smoking, spicy foods, alcohol, oral sepsis, poorly fitting dentures, syphilis and tuberculosis are predisposing factors.\(^{(8)}\)

Histologically there is thickening of the epithelium, with hyperkeratosis or parakeratosis, and moderate accumulation of lymphocytes and plasma cells in the underlying connective tissue. As the condition advances the rete pegs become irregular and longer and if there is tendency toward carcinoma the rete pegs begin to invade the connective tissue.\(^{(9)}\)
Treatment consists of the removal of all irritants, avoidance of spicy food, mild alkaline mouthwashes and strict oral hygiene. Vitamin A therapy is sometimes (10) successful. Oestrogens have been used with partial success. Smearing with petroleum jelly and in some cases, surgical removal have also been suggested. The use of caustics is contra-indicated.

(14) (15) (16)

**LICHEN PLANUS:**

This may affect the mucous membrane and is usually associated with characteristic skin lesions along the flexor surfaces of the arms and legs.

Orally, the lesions are most common on the cheeks and tongue. Slightly raised, whitish-grey spots connected by lase-like lines or papules with a reddish halo are seen. Most common site is opposite the occlusal line.

Etiology is unknown but is it thought to be related to nervous disturbances.

(17) Treatment is rather uncertain, Vitamin A, arsenic, mercury and bismuth preparations have been used Vitamin B complex and hormone treatment have also been tried. Irritants must be avoided and oral hygiene maintained.

**HERPES LABIALIS:**

This is an acute, infectious disease caused by a filtrable virus and is slightly contagious. It suddenly appears on the lips usually beginning at the vermillion border. There is a short period of tension and slight soreness, then small vesicles appear on a reddened spot. These contain a colourless or yellowish liquid and after rupturing, the lesion dries up leaving yellow adherent crusts. After a week or two it heals without a scar. According to Kronfeld, sometimes it occurs on the mucous membrane of the tongue or cheek and usually accompanies some general illness or disturbance such as a cold, menstruation, gastro-intestinal disturbances and emotional strain.
Kerr describes herpetic gingivostomatitis as an acute infection caused by Herpes Simplex. He says in 50% of cases there is a history of contact and therefore it is communicable. Also he states that 75% of the population are infected at some time or other, the highest evidence being in the 1-3 age groups. The virus is intracellular and so is protected from the antibodies of the blood. After infection it becomes a "residual virus" and may become activated at a later date by slight trauma, sunlight, menstruation etc.

These symptoms are described for Primary herpetic gingivostomatitis:

Sore throat and neck and increased temperature for 3-5 days. With the appearance of oral lesions this drops.

The gingivae are red, swollen and painful and bleed easily. The oral mucosa is involved to a varying degree.

Vesicles form, then ulcerate giving painful ulcers with a red halo. There is fetor oris.

In 7-14 days the mouth is again normal and there is no scarring.

Histologically there are characteristic inclusion bodies in the epithelial cells, slight necrosis of the sub-mucosa and many leukocytes.

During convalescence, antibodies are formed in the blood and remain permanently.

Secondary Herpetic Stomatitis is caused by activation of the residual virus and gives these symptoms:

A localized burning sensation and slight swelling and then a small vesicle or group of vesicles. Early rupture occurs resulting in a shallow ulcer with an irregular undermined border. Secondary infection occurs and the ulcer becomes painful, forms a yellow membrane and a red halo. In 7-10 days it heals without scarring.
Treatment of this Herpes Infection is not very satisfactory but B Complex in large doses, aureomycin and smallpox vaccine have been used. Topically caustics and antiseptics are of some value.

Several other types of aphthae are described, canker sores, Mikulicz's aphthae. Miller refers to Periodenitis Mucosa Necrotica Recurrens as chronic aphthae yet Kerr differentiates between this and secondary herpetic stomatitis. He describes periodenitis necrotica recurrens as similar lesions occurring on gland bearing mucosa but which are often larger, heal more slowly and produce scars.

**PERLECHE.**

This is an infection occurring at the angles of the mouth and is seen in childhood and occasionally in adults, especially in those whose bite is closed by unsuitable prostheses. Fissures and crust formation are seen. Etiological factors listed are streptococci, staphylococci and monilia and also a riboflavin deficiency.

Treatment by Vitamins, topical application of 10% silver nitrate or copper sulphate is suggested.

(21) (22) (23)

**PEMPHIGUS.**

This is a rather uncommon, but serious disease of uncertain etiology. The skin and often the oral mucous membrane is involved and the typical lesions are bullae and blebs which form continuously. It is said to occur more commonly in women. Orally, there may be dryness in the early stages and later, copious blood-stained saliva. It is easily confused with erythema multiforme, however, here the lesions are of shorter duration and not as severe as in Pemphigus and there are definite periods of remission.

Treatment of pemphigus is not very satisfactory.

(24) (25)

**LUPUS ERYTHEMATOSUS.**

This condition is thought to be associated with tuberculosis. Miller says it is usually caused by streptococci. Sharply defined elevated patches occur in
the mouth with superficial vascularity at the edges. The lesions often resemble tuberculous lesions. Intravenous injections of bismuth and gold sodium thiosulphate have been used in treatment.

Syphilis may show a primary chancre in the mouth—usually on the lips, secondary lesions mucous patches, snail track ulcers and rashes and in the tertiary stage gummas. Tuberculosis may occur in the mouth as a primary lesion or secondary to lung infection. The mouth lesion may be superficial or deep seated.

There are many other diseases which feature oral symptoms but it is beyond the scope of this paper to deal with them all.

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A summary of the literature dealing with periodontal disease has been presented. Considerable advance has been made in this subject during the last twenty years, and where formerly a case of "pyorrhea" was usually deemed incurable with the consequent sacrifice of many teeth, now however, many of these cases are being successfully treated. More important however is the knowledge that the vast majority of gingival aberrations can be prevented by adequate home care, correct diet and regular visits to the dentist and there is still much to be done in educating the public along these lines.

Wærhaug's thesis indicates that possibly a wrong interpretation has been given to the attachment of the supporting tissues to the teeth, namely, the epithelial attachment.

An old concept that the alveolar bone was infected is now discounted thus leading to more conservative surgical treatment.

Etiological factors are still debatable and even now the role bacteria and calculus play is not entirely known. Similarly, the cause of calculus deposition is rather uncertain.

Systemic factors have received considerable study and more research is indicated here to further classify such entities as periodontosis, desquamative gingivitis and also the mechanism of hormone affect on the periodontal tissues.

The tendency seems towards conservative treatment by sub-gingival curettage though many still prefer surgical elimination of pockets. Both have their application and though the conservative method is a more tedious and
lengthy process good results can be achieved. In any case it seems a very sound scheme to attempt conservative therapy first as any future surgical treatment is rendered easier and more satisfactory if the mouth is brought to a more healthy state beforehand.

Terminology is becoming more standardized which is fortunate as the host of expressions used in the past made a study of this subject rather confusing.
INDEX.

Allergy. .. 38.
Alveolar Bone. .. 21.49.71.
Atrophy. .. 59.85.87.

Bacteria in plaques. .. 29.
" oral flora. .. 31.
" as etiological factors. 32, 31, 54.
Blood dyscrasias. .. 36.62.
Bone resorption. .. 47.70.84.

Calculus. .. 24.96.
Cementum. .. 14.
Classification. .. 45.
Complex Periodontitis. .. 75.

Gurettage, sub-gingival. .. 101.

Desquamative Gingivitis. 65, 67, 121.
Development of Periodontal Lesion. .. 47.83.
Diabetes. .. 37.65.
Diet. .. 34.120.

Electrosurgery. .. 111.
Endocrine effects. .. 37.62.
Epithelial attachment. .. 10.
Epithelial rests in Periodontal Membrane. 18.

Focal infection. .. 90.
Food impaction. .. 23.
Fusospirochetes. .. 54.57.
Function, lack of .. 33.

Gingiva. .. 5.
Gingivitis. .. 52.95.
<table>
<thead>
<tr>
<th>Condition</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gingival massage</td>
<td>68</td>
</tr>
<tr>
<td>Gingivosis</td>
<td>68</td>
</tr>
<tr>
<td>Gingivectomy</td>
<td>51</td>
</tr>
<tr>
<td>Gum Recession</td>
<td>54</td>
</tr>
<tr>
<td>Gingival Polyp.</td>
<td>10.14</td>
</tr>
<tr>
<td>Gingival Sulcus.</td>
<td>34</td>
</tr>
<tr>
<td>Habits</td>
<td>34</td>
</tr>
<tr>
<td>Herpes</td>
<td>129</td>
</tr>
<tr>
<td>Hormone effect.</td>
<td>37.62, 63</td>
</tr>
<tr>
<td>Hyperplasia.</td>
<td>53.58, 63, 66</td>
</tr>
<tr>
<td>Idiopathic Fibromatous Hyperplasia</td>
<td>58</td>
</tr>
<tr>
<td>Keratin</td>
<td>11.97</td>
</tr>
<tr>
<td>Leukoplakia</td>
<td>128</td>
</tr>
<tr>
<td>Lichen Planus.</td>
<td>129</td>
</tr>
<tr>
<td>Lupus Erythematosus</td>
<td>131</td>
</tr>
<tr>
<td>Metallic Poisons</td>
<td>38, 66</td>
</tr>
<tr>
<td>Mouthbreathing</td>
<td>34, 53</td>
</tr>
<tr>
<td>Necrotic Tracts</td>
<td>49</td>
</tr>
<tr>
<td>Occlusion</td>
<td>81</td>
</tr>
<tr>
<td>&quot; Correction of</td>
<td>113</td>
</tr>
<tr>
<td>Pemphigus</td>
<td>131</td>
</tr>
<tr>
<td>Periodontal Pockets</td>
<td>48, 110</td>
</tr>
<tr>
<td>Perlöche</td>
<td>131</td>
</tr>
<tr>
<td>Periodontitis</td>
<td>68, 100</td>
</tr>
<tr>
<td>Periodontosis</td>
<td>71, 114</td>
</tr>
<tr>
<td>Periodontal Membrane</td>
<td>17</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>37, 62</td>
</tr>
<tr>
<td>Psychosomatic Factors</td>
<td>39</td>
</tr>
<tr>
<td>Reattachment</td>
<td>102</td>
</tr>
<tr>
<td>Condition</td>
<td>Code</td>
</tr>
<tr>
<td>---------------------------------</td>
<td>--------</td>
</tr>
<tr>
<td>Resorption of Bone</td>
<td>47.70.84.</td>
</tr>
<tr>
<td>&quot; of Roots.</td>
<td>73.86.</td>
</tr>
<tr>
<td>Saliva.</td>
<td>2.4</td>
</tr>
<tr>
<td>Smoking.</td>
<td>34.55.</td>
</tr>
<tr>
<td>Thrush.</td>
<td>127.</td>
</tr>
<tr>
<td>Traumatic Occlusion</td>
<td>33.83.</td>
</tr>
<tr>
<td>Vincent's Infection</td>
<td>54.115.</td>
</tr>
<tr>
<td>Vitamins.</td>
<td>35.60.119.</td>
</tr>
</tbody>
</table>