PERIODONTOLOGY

SYLLABUS

UNITED STATES ARMY INSTITUTE OF DENTAL RESEARCH
WALTER REED ARMY MEDICAL CENTER
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A SERIES OF LECTURE NOTES

ON

PERIODONTOLOGY

Compiled By

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PREFACE

This syllabus was prepared for the training program in Periodontology at Walter Reed Army Medical Center. The work is not intended to be an all-inclusive doctrine of periodontia; indeed, in this rapidly changing science today, one should maintain broad concepts and avoid the pitfalls of enthusiastic conformity to those doctrines which may lack the support of critical scientific analysis.

We hope the student will be stimulated to pursue supplemental study, and encouraged to adopt a philosophy of "periodontal consciousness," which he should carry with him no matter what course in dentistry he may pursue. This concept is becoming ever more important as our knowledge in dentistry increases, and the role of periodontics becomes more positive in the overall treatment of oral diseases.

W. B. B.

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LECTURE NO. 1

CLINICAL AND HISTOLOGIC EXAMINATION OF THE GINGIVA

Wilfred B. Bell*

GINGIVA: "that part of the oral mucous membrane that covers the alveolar processes of the jaws and surrounds the necks of the teeth" (Glickman).

I. CLINICAL EXAMINATION will reveal the following features:

A. Texture

B. Color

C. Form

D. Relation to teeth and adjacent soft structures

II. GROSS CLINICAL COMPONENTS include the following:

A. MARGINAL OR UNATTACHED GINGIVA: About 1 mm wide--surrounds the teeth in a collar-like fashion, and forms the soft tissue wall of the gingival sulcus. Separated from the attached gingiva by the "free gingival groove" (Orban).

B. ATTACHED GINGIVA: Extends from the marginal gingiva to the alveolar mucosa. It is characteristically stippled, firm, resilient, and tightly bound down to the underlying cementum and alveolar bone. HIGHLY IMPORTANT as the functional "stress bearing" component of the gingivae.

C. ALVEOLAR MUCOSA: Separated from the attached gingiva by the MUCOGINGIVAL LINE. A thin, non-stippled, loose tissue that blends into the vestibular fornix.

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D. INTERDENTAL PAPILLA: That portion of the gingiva which extends into the interproximal space. The tip and lateral borders of the interdental papilla are formed by a continuation of the marginal gingiva from the facial and lingual surfaces of the adjacent teeth. Interproximal form is probably in the shape of a "U" with the base below the height of the facial and lingual projections due to the contour of the proximal surfaces of the teeth.

III. MICROSCOPIC FEATURES OF THE NORMAL GINGIVA:

A. THE MARGINAL GINGIVA consists of a central core of connective tissue which is covered by a stratified squamous epithelium. One aspect of the epithelium of the marginal gingiva forms the lining of the gingival sulcus, the other is continuous with the epithelium of the attached gingiva. The epithelium lining the inner aspect of the gingival sulcus is not ordinarily keratinized.

B. ATTACHED GINGIVA is continuous with the marginal gingiva and has a connective tissue stroma covered with epithelium characteristically the same as the marginal gingiva.

C. ALVEOLAR MUCOSA is covered with a thin, non-keratinized epithelium. It is loosely attached to the underlying periosteum by a well defined submucous layer of loose connective tissue.

IV. COMPONENTS OF THE EPITHELIUM:

A. A surface keratinized layer consisting of flattened, clearly staining strands devoid of nuclei (hornification). Occasionally this layer is missing and sometimes it is parakeratotic, but both are considered normal by Orban. The first line of defense to penetration by noxious substances.

B. Multilayered granular portion consisting of prominent basophylic granules in the cytoplasm and a shrunken hyperchromic neucleus.

C. Multilayered prickle cell portion with prominent intercellular bridges and wide intercellular spaces.

D. Cuboidal basal cell layer which affects the attachment

of the epithelium to the basement membrane of the connective tissue layer by short processes that fit into the grooves of the lamina propria.

V. COMPONENTS OF THE CONNECTIVE TISSUE LAYER:

A. Papillary layer consists of papillary projections which interdigitate with the downward epithelial pegs (rete pegs). Consists of dense connective tissue and contains the vascular supply and innervation of the gingiva. Microscopic studies show a single capillary loop in each of the papillae plus a reserve supply of capillaries.

B. Reticular layer is densely collagenous and firmly attaches the gingivae to the periosteum of the alveolar bone. This corresponds to the submucous layer which is not recognized in gingival tissue but is more prominent in the palate. In the latter we find glands, blood vessels, nerves, and adipose tissue.

VI. THE NORMAL GINGIVAL SULCUS:

"The shallow groove around the tooth bounded on one side by the surface of the tooth and on the other by the epithelium lining the free margin of the gingiva" (Glickman). This shallow V-shaped depression has its base at the most coronal level of the epithelial attachment.

VII. FORMATION OF THE GINGIVAL SULCUS:

At the conclusion of enamel matrix formation, the ameloblasts produce a thin membrane on the surface of the enamel, the PRIMARY ENAMEL CUTICLE. It is connected with the interprismatic enamel substance and is contained only on the enamel. After formation of the enamel cuticle, the ameloblasts shorten and the epithelial cells comprising the enamel organ are reduced to a few layers of cuboidal cells which are then called the REDUCED DENTAL EPITHELIUM. This remains attached to the primary cuticle and covers the entire enamel down to the cemento-enamel junction.

During the eruption of the tooth, the tip of the tooth approaches the oral mucosa and the reduced dental epithelium fuses with the oral epithelium. The oral epithelium over the tip of the tooth disintegrates in the center and the crown emerges into the oral cavity. The reduced dental epithelium is now termed the EPITHELIAL ATTACHMENT (CUFF).

As the tooth erupts, the epithelial attachment gradually separates from the tooth surface. The shallow groove which develops between the gingiva and the surface of the tooth is called the GINGIVAL SULCUS. The bottom of the sulcus is found where the epithelial attachment (formerly reduced dental epithelium) separates from the surface of the tooth. The marginal gingiva thus is coronal to the bottom of the sulcus.

VIII. SECONDARY ENAMEL CUTICLE:

Secondary enamel cuticle as described by Gottlieb is a product of hornification of the epithelial attachment and is a precursor to the separation of the epithelial attachment from the tooth. It appears on both the enamel and cementum. As the epithelial attachment moves apically, it separates from the cuticle which remains attached to the tooth surface. On the enamel it combines with the primary enamel cuticle and forms a composite hyalin structure. On the cementum it is found as a thin homogenous non-cellular structure only on the cemental surfaces traversed by the epithelial attachment. The nuclei and cell walls of the epithelial cells adjacent to the tooth disappear and the cells coalesce to form a continuous strand.

There is some evidence that the COMPOSITE CUTICLE (primary and secondary enamel cuticle combined) on the enamel surface is more resistant to caries than the single cuticle located on the cemental surface.

IX. GOTTLIEB'S THEORY OF TOOTH ERUPTION:

According to the above, we see that we have two related phenomena. The eruption of the tooth towards the occlusal line and the migration of the epithelial attachment apically. These phenomena occur together but at a reduced rate after the tooth reaches its antagonist. This is termed ACTIVE AND PASSIVE ERUPTION.

For convenience sake, these are placed in four stages:

FIRST STAGE:

When the tooth reaches the plane of occlusion, one-third to one-fourth of the enamel is covered by the epithelial attachment. (The anatomical crown is larger than the clinical crown). The bottom of the gingival sulcus is still on the enamel. The apical portion of the epithelial attachment is at the cemento-enamel junction. We would expect to find this stage in youth up to perhaps age 30, although this may vary widely.

SECOND STAGE:

The apical or bottom of the sulcus is still on the enamel but more apically than formerly and the epithelial attachment has progressed until the most apical portion is on cementum; however, the more coronal portion is still on enamel. This stage may persist until age 40 or later.

THIRD STAGE:

The gingival sulcus is briefly on the cemento-enamel junction and the epithelial attachment is entirely on cementum. This probably should not be considered a distinct phase but rather a brief interlude before progressing into the fourth stage.

FOURTH STAGE:

This is reached when the cementum is exposed by progression of the epithelial attachment apically and the bottom of the gingival sulcus is on the cementum. The clinical crown is now larger than the anatomical crown. Of course this may vary in different individuals and even on different teeth in the same individual.

Coincidentally with the above, Gottlieb envisions synchronization of the following phenomena:

A. Migration of the epithelial attachment toward the apex.

B. Atrophy of the marginal gingiva.

C. Atrophy of the alveolar crest.

D. Extension of the teeth toward the occlusal line.

E. Compensating attrition of the teeth to prevent excessive length of the clinical crown.

According to this concept, the normal position of the gingiva varies with age--so the epithelial attachment may be attached either on the crown or on the root. Therefore, recession of the gingiva is not always pathologic.

NOTE: References at end of Lecture No. 2.

LECTURE NO. 2

THE PERIODONTIUM

Wilfred B. Bell*

I. THE PERIODONTAL LIGAMENT:

"The periodontal ligament is the connective tissue which surrounds the root of the tooth and attaches it to the bony alveolus; it is continuous with the connective tissue of the gingivae" (Orban).

A. NORMAL STRUCTURAL COMPONENTS:

1. Bundles of connective tissue fibers.
2. Connective tissue cells.
3. Strands of epithelium (epithelial rests).
4. Blood vessels.
5. Lymphatics.
6. Nerves.

The most important element is the collagenous or principal fibers. They are arranged in bundles and follow a wavy course. The part of the principal fibers which is embedded in cementum or bone is termed "Sharpey's Fibers." The bundles are probably held together with a cementing substance and contain no elastic fibers, according to Orban. He believes the apparent elasticity of these structures is due to the arrangement of the principal fiber bundles in a wavy course from bone to cementum, thereby allowing slight movement of the tooth upon stress. Although the bundles appear to run directly from bone to cementum, the individual shorter fibers are apparently joined

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together or "spliced" in an area midway between bone and cementum termed the "intermediate plexus." It is believed that the constant readaptation of the periodontal ligament to the functional stresses exerted on the teeth is reflected in alterations and changes in the intermediate plexus rather than the terminal endings in cementum and bone.

B. THE PRINCIPAL FIBERS ARE ARRANGED AS FOLLOWS:

1. Gingival Group:

Pass from cementum into the free and attached gingiva. They support the gingiva and help bind it down, finally terminating in the fibrous gingival tissue.

2. Transseptal Group:

Extend interproximally over the alveolar crest and are embedded in the cementum of the adjacent teeth.

3. Alveolar Group:

Attach the tooth to the alveolus.

a. Alveolar Crest Group:

Extend obliquely from just underneath the epithelial attachment to the alveolar crest. This group resists the coronal thrust of the more apical fibers and helps retain the tooth in the socket.

b. Horizontal Group:

Extend at right angles to the long axis of the tooth from the cementum to the alveolar bone. They function the same as the alveolar crest fibers.

c. Oblique Group:

The largest group of fibers and bears the brunt of the vertical masticatory stresses. The fibers run obliquely from the cementum to the bone to support the tooth in a "sling-like" fashion.

d. Apical Group:

Radiates from the cementum of the tooth at the fundus of the socket to the bone.

e. Interradicular Group:

Extend from the interradicular septum to the bifurcation of multiradicular teeth.

There are also regularly arranged connective tissue fibers between the principal fibers--termed the interstitial connective tissue of the periodontal ligament. The blood vessels and lymphatics are enclosed within this tissue.

Cellular elements found in the periodontal ligament are fibroblasts, cementoblasts, osteoblasts, osteoclasts, tissue macrophages, and epithelial rests.

C. VASCULAR SUPPLY:

1. Apical vessels:

Pass coronally from the apical region through the periodontal ligament to the gingiva, passing off lateral branches in the direction of the cementum and bone on the way to their termination.

2. Interalveolar vessels:

In the alveolar bone, the interalveolar artery gives off small branches, the alveolar perforating arteries, which pass through channels in the alveolar bone to reach the periodontal membrane.

3. Anastomosing gingival vessels:

The vessels of the periodontal ligament anatomose with vessels passing over the alveolar crest from the gingival tissue to form a rich capillary network.

Lymphatics, nerves, and venous drainage generally accompany the arterial supply.

D. FUNCTIONS OF THE PERIODONTAL LIGAMENT:

1. Physical (Supportive):

   a. Transmission of masticatory forces to the bone.

   b. Attachment of the teeth to the bone.

   c. Maintenance of the gingival tissues in their proper relation to the teeth.

   d. Shock absorption or diminishing the impact of external forces.

   e. Soft tissue casing for the vessels and nerves to prevent injury by mechanical forces.

The arrangement of the principal fibers is similar to a suspension bridge. When an axial force is imposed on the tooth, there is a tendency toward displacement of the root into the alveolar socket. The oblique fibers undergo a transition from their untensed wavy pattern to their full length potential. This group sustains the major part of the axial masticatory force.

According to Gottlieb, two conditions govern the efficiency of the suspension apparatus: the number of fiber bundles per unit area, and the area of the root surface available for the embedding of fibers.

Boyle and others believe that forces of occlusion are transmitted first as hydraulic pressure to the alveolar bone through the semifluid periodontal ligament, before the periodontal fibers are brought into function.

When a tipping force is applied to a tooth, the tooth rotates about an axis of rotation with the apical portion of the root moving in a direction opposite to the coronal position. Some principal fibers are therefore placed under tension and others compressed.

The axis of rotation for a multi-rooted tooth is located within the interradicular septum of the alveolar bone. In single-rooted teeth, it is generally considered to be located at a point slightly apical to the middle of the root. The periodontal ligament is relatively thin in this area due to its hour glass shape.

2. Formative Function:

The osteoblasts, osteoclasts, and cementoblasts are found in the periodontal ligament and are concerned with the formation and resorption of the bone and cementum. In this respect, the periodontal ligament acts as a periosteum to these two hard tissues. This is essential for the accommodation of the periodontium to functional stresses as well as in the repair of injuries involving the calcified tissues.

Where bone formation is in progress, osteoblasts are found and are apparently important for the attachment and reattachment of the periodontal fibers to the alveolar bone.

The multinucleated osteoclasts are found only during active bone resorption. The process of osteoclastic resorption is not fully understood but apparently the cytoplasm of the osteoclast exerts some influence on the bone bringing about resorption of both the inorganic and organic components.

Cementoblasts are found between the fibers of the periodontal ligament on the surface of the cementum.

3. Nutritional and sensory function:

This includes the supplying of nutrients to, and the removal of waste products from, the other tissues of the periodontium via the blood vessels and lymphatics. The periodontal ligament also receives impulses and reacts to stimuli.

E. ADDED:

Recent work by Fullmer has revealed what he terms "oxytalin" fibers in the periodontal ligament. The function of these fibers is unknown, but they seem to possess chemical and staining characteristics not unlike elastic fibers, which, if substantiated, would indicate that they provide some elasticity to the suspensory function of the periodontal ligament. These fibers have been found throughout the periodontal ligament, but the largest fibers are located in the transseptal region, irrespective of either its position on the root or the relative health of the tissue.

II. CEMENTUM:

"A calcified mesenchymal tissue which forms the outer covering of the anatomical root" (Glickman).

A. CEMENTOGENESIS:

Hertwig's epithelial root sheath forms a mold into which the root dentin is deposited. The epithelial root sheath separates the newly formed dentin from the surrounding connective tissue--the precursor of cementum. Cementum cannot be deposited as long as the epithelial sheath remains intact. However, either through degeneration of the epithelial sheath or proliferation of the connective tissue through the sheath, the connective tissue makes contact with the dentin. The connective tissue cells then differentiate into cementoblasts with subsequent deposition of cementum. The remnants of the epithelial sheath are known as the "epithelial rests."

The activity of the cementoblasts produces first an uncalcified cementoid tissue which is later transformed into calcified cementum. Under normal conditions a thin layer of cementoid, lined with cementoblasts, is always found on the surface of previously deposited cementum. The cementoid substance is highly resistant to osteoclastic resorption, although the inorganic content of cementum is forty per cent, similar to bone but less than enamel and dentin.

B. TYPES OF CEMENTUM:

1. Cellular:

Ordinarily more prominent on the apical half of the root and contains embedded cementocytes in lacunae. The lacunae are connected with each other by canaliculi in much the same manner as the Haversian systems in bone.

2. Acellular:

Ordinarily covers the cervical portion of the root and is free of enclosed cells. It is clear and structureless except for fine fiber bundles embedded within the calcified matrix which run vertically to the surface of the root.

There is no definite pattern for the arrangement of the cellular and acellular cementum. The acellular type is generally deposited on the dentin and covered by the cellular type; however, many variations may occur.

Cementum varies in thickness with its greatest width demonstrated at the apical area to compensate for the continuous eruption of the tooth. Both types of cementum are deposited in layers as evidenced by the formation of incremental lines. The superficial layers are penetrated by the periodontal ligament fibers and thus form the tooth side attachment for this structure.

The metabolic activity of cementum is much less than bone and therefore we do not find the process of alternating resorption and regeneration of the tissue. There is a continuous deposition of cementum to compensate for loss of tissue vitality and to affect new attachment for the periodontal ligament fibers. Repair of cemental tears has also been noted but resorption of this tissue is not a physiologic process.

III. THE ALVEOLAR PROCESS:

A. ALVEOLAR BONE PROPER:

Forms the inner wall of the tooth socket and consists of bundle bone, which affects the attachment of the periodontal ligament fibers, and adjacent lamellated bone. The composite is recognized as the lamina dura radiographically.

B. SUPPORTING BONE:

Adjoins the alveolar bone and contains a network of bony trabeculae enclosing marrow spaces. The trabeculae vary in density and arrangement depending on the degree of function.

C. CORTICAL PLATES:

Dense compact bone adjacent to the supporting bone. It fuses with the alveolar bone proper at the crest of the alveolar process.

Living bone is constantly in a state of flux. In spite of the fact that this is one of the hardest tissues in the body, it is engaged in continuous resorption and regeneration. In the normal, healthy individual, a fine state of equilibrium exists between these two processes. As age progresses, the repair phase gradually falls behind the resorptive phase. This is true of other tissues as well and should be considered a natural phenomenon of tissue aging. However, precocious resorption as seen in periodontal disease is a pathologic process and may be the result of many factors, both local and systemic.

Osteoclasts are generally multinucleated giant cells which differentiate from young fibroblasts or undifferentiated mesenchymal cells of the connective tissue. Their function is the resorption of bone, and while the process is not completely understood, the cells seem to produce a proteolytic enzyme which dissolves the organic constituents, and chelating substances which render soluble the inorganic bone salts. Pure decalcification of bone, allowing an organic matrix to remain probably does not occur. The action of the osteoclasts apparently is not selective to either the inorganic or organic components but acts on both.

Osteoblasts produce new bone and also probably differentiate from undifferentiated mesenchymal cells. Osteoblasts first produce an uncalcified osteoid. The osteoblasts become embedded in the matrix and are then known as osteocytes.

IV. DISCUSSION:

The periodontal ligament and the contiguous bone and cementum depend on function as the stimulus to the maintenance of health. Remove the function and the ligament becomes thinner due to the atrophy of the fiber bundles. In addition, the growth potential of the cementum and alveolar bone will be lost. Increased function, within the adaptive capacity of the ligament, will result in well-formed fiber bundles and a thicker tissue.

A tooth which has been functionless for a period of time and then suddenly is brought into function through restorative, prosthetic, or orthodontic procedures must undergo a period of adjustment and physiologic conditioning of the supporting tissues before it can function as a healthy unit. It might be suggested that before such teeth are utilized to fulfill a particularly traumatic-functional requirement, such as a prosthetic abutment (either fixed or removable) that it be brought into function gradually with a temporary "conditioning" appliance.

The scales may tip in the other direction and due to loss of support of the tooth, through periodontal disease or other factors, the tooth may be subjected to occlusal forces which may exceed the tolerance or adaptive capacity of the attachment apparatus. This may result in a breakdown of the attachment mechanism and present a condition termed occlusal traumatism. We may consider this an important entity in itself with many ramifications and thus will be considered at a later date.

It should be obvious by now that the tooth-supporting tissues mentioned above, including spongy supporting bone, are cast in a do-or-die role with the tooth. In the absence of uncontrolled disease factors, the tooth maintains the surrounding tissues, likewise under the same conditions the surrounding tissues maintain the tooth. If one or the other is lost--both are lost. This should point out the importance of maintaining BOTH units.

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LECTURE NO. 3

THE INFLAMMATORY PROCESS

Rowland A. Hutchinson*

I. INTRODUCTION:

Inflammation is the primary defense reaction of the body and may be defined as "the reaction of a tissue to an injurious agent." Injury is the effect on cells and tissues produced by excessive stimulation. These stimulae may be physical, chemical, viable or non-viable, and vary in intensity and duration. Whenever tissue is injured, cellular metabolism is interrupted and a series of tissue reactions follow at the site of injury. These reactions tend to destroy or to limit the spread of the injurious agent and to repair or replace the damaged tissues. These local reactions to injury constitute inflammation. "Injury is followed by inflammation; inflammation is preceded by injury."(Anderson)

II. TYPE OF REACTION IN VERTEBRATES:

The basic response in man and other vertebrates is a VASCULAR RESPONSE. The inflammatory reaction in man is similar to lower forms of animal life in the way that the reaction basically behaves. Phagocytosis is a basic defense mechanism of lower forms of life, along with humoral antibodies and autotomy. The great advantage of the vascular reaction is that it hastens and intensifies the inflammatory response, enabling enormous numbers of phagocytic cells and antibodies to be delivered speedily to the injured tissues.

III. FUNCTIONS OF THE INFLAMMATORY REACTION:

A. To transport to the site of injury phagocytic cells which engulf and digest bacteria, dead cells, and other debris.

B. To transport antibodies to the injured site.

C. To neutralize and dilute the irritant.

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D. To limit the spread of infection or the irritating agent.

E. To initiate repair of the injured tissue.

IV. CARDINAL SIGNS OF INFLAMMATION:

Celsus, in the first century A.D., described the symptoms of inflammation as: rubor (REDNESS), tumor (SWELLING), calor (HEAT), and dolor (PAIN). These signs are caused by the dominant vascular response. Redness and heat being caused by an increased blood supply to the area due to the dilatation of vessels and the filling of previously collapsed capillaries. Swelling is the result of increased blood in the area and the presence of the plasma and cellular exudate. Pain is due to the products of injured cells acting upon sensory nerve endings and increased tension in the area due to the inflammatory exudate.

V. VASCULAR CHANGES:

Immediately following injury, there is a transitory vasoconstriction which is followed by a VASODILATATION. These changes are probably mediated by an axone reflex response. With the vasodilatation there is an increased capillary pressure and an accelerated rate of flow filling previously collapsed capillaries with blood. Plasma escapes into the tissues and the cells within the vessels become more concentrated. This causes the blood to become more viscous, thus explaining the next vascular change which is a DECREASED VELOCITY OF FLOW. As plasma continues to escape, the flow becomes so slow that in some vessels there is complete stasis and the vessel is solidly packed with stationary blood cells. The escape of plasma from the vessels is due to an INCREASED PERMEABILITY of the vessel walls. This increased premeability is probably due to an increased widening of the space between the endothelium cells which allows normally permeable substances to escape more rapidly and also allows substances such as colloidal proteins which normally cannot escape to diffuse out into the tissue. STARLING'S HYPOTHESIS refers to the differences in intravascular and extravascular hydrostatic and osmotic pressures which normally are responsible for the return of fluid into the venous end of the capillary. With the increased amount of plasma and plasma proteins in the tissue bed, the osmotic pressure is so changed that fluid is no longer drawn back into the venules. This results in:

A. Stasis
B. Edema
C. Shock
D. Changes in lymph flow
E. Fibrin formation

VI. CELLULAR COMPONENTS OF INFLAMMATION:

A. POLYMORPHONUCLEAR LEUKOCYTES (PMN'S, GRANULOCYTES).

1. Neutrophils - most numerous of the pmn's. Their chief function is phagocytosis, principally phagocytosis of bacteria. The neutrophils are the "first line of defense" since they can be thrown rapidly and in great number into the infected areas.

2. Eosinophils - are similar to neutrophils but they stain intense red with eosin. They are greatly increased in allergic or hypersensitive reactions and along with phagocytosis, may function to carry histamine.

3. Basophils - The function of basophils is not known. They do not increase in acute infections like other granulocytes.

B. MONOCYTES (MACROPHAGES, HISTIOCYTES).

Their origin and relations are rather confused and they are known as monocytes in the blood, macrophages, histiocytes, and polyblasts in the tissues. They are mononuclear and are larger than the pmn's, thus they ingest large particles which pmn's cannot phagocytize. They are the "second line of defense" appearing later in the reaction than the pmn's since they are less numerous in the blood and are less chemotactic. Along with the functions of phagocytosis and intracellular digestion, the macrophages also function to form antibodies. GIANT CELLS are formed by the fusion of many macrophages, usually about an insoluble foreign body.

C. LYMPHOCYTES

Are smaller than pmn's and appear to consist almost entirely of nucleus. They appear late in the course of inflammation and are especially prominent in chronic conditions. They exhibit neither chemotaxis or phagocytosis and their function is not definitely known, although they may function

to carry antibiodies.

D. PLASMA CELLS

Are most prominent in chronic inflammation and are believed to produce and carry antibodies. They have eccentric shaped nuclei resembling a cartwheel.

VII. CELLULAR REACTIONS:

A. MARGINATION AND EMIGRATION OF LEUKOCYTES

Normally the cellular elements of blood move in the center of the stream while the plasma flows at the periphery. In inflammation, as the circulation slows, the cellular elements move toward the periphery. The endothelium and leukocytes both become sticky and the leukocytes adhere to the vessel wall. The leukocytes then emigrate through the vessel wall by ameboid motion (diapedesis).

B. CHEMOTAXIS

A directional response. After the leukocytes pass through the vessel wall they move toward the site of injury rather than wander at random. This directional response can be either positive or negative. Leukocytes exhibit positive chemotaxis toward bacteria, injured tissue products, and some foreign bodies; however, viruses, so far as is known, do not attract pmn's--possibly accounting for the lack of inflammation in virus infections.

C. PHAGOCYTOSIS

The process of ingestion by a cell. All cells are probably capable of phagocytosis under some conditions; however, in man the two most important phagocytic cells are microphages (pmn's) and macrophages. The mechanism of phagocytosis is ameboid-like consisting of gel-sol transformation. In order for phagocytosis to occur, the phagocytes and bacteria must remain in contact (by surface tension). Certain highly virulent organisms become surrounded by a slimey capsule and thus are difficult to phagocytize.

D. INTRACELLULAR DIGESTION

After the bacteria are phagocytized, they are digested

by proteolytic enzymes within the phagocyte.

VIII. TYPES OF INFLAMMATORY LESIONS:

The pictures of inflammation vary and depend largely on predominance of one of three processes: exudation, proliferation of tissue cells, or necrosis caused by the injurious agent.

A. EXUDATIVE LESION

The dominating process is escape of fluid or cells from vessels into the lesion. This can be serous, fibrinous, purulent, or hemorrhagic depending on the composition of the exudate. An example would be the inflammatory lesion of an acute periapical or periodontal abscess.

B. PROLIFERATIVE LESION

This is particularly noticeable in chronic inflammation where the connective tissue cells tend to multiply. An example would be pyogenic granuloma.

C. NECROTIC LESION

Although some necrosis is present, at least in individual cells, in all inflammation, certain types dominate the picture. An example would be an acute necrotizing ulcerative gingivitis.

D. STAGES The nature, intensity, and duration of the irritant, the resistance of the host, and other variables influence the degree and nature of the inflammatory lesion as follows:

1. Acute:

   Lasts only a few days to a few weeks.

2. Subacute:

   Two to three weeks or longer. The histologic characteristics are that the neutrophils are replaced by eosinophils and lymphocytes and exudate is less prominent. Proliferation, especially of young blood vessels and connective tissue, is more prominent.

3. Chronic:

Months or years. This is due to the injurious agent persisting in the tissues and continuing to damage them. Usually there are repeated episodes of necrosis, and in response granulation tissue forms and becomes converted into fibrous tissue which is the most characteristic feature of chronic inflammation.

IX. THE BIOCHEMISTRY OF INFLAMMATION:

A. LEUKOTAXINE:

Lewis, in 1924, postulated that capillary premeability was caused by an "H" substance related to histamine. In 1935, Menkin isolated a specific polypeptide not related to histamine which is capable of causing increased capillary premeability. This was isolated from an inflammatory exudate and was termed leukotaxine. In addition to affecting capillary permeability, leukotaxin is the only known substance to be definitely chemotactic for leukocytes. The mechanism of chemotaxis is not known but may be concerned with electrical charges.

B. LOCALIZATION OF INFLAMMATION:

With increased capillary permeability, plasma proteins such as albumin, globulins and fibrinogen are released into the tissue. In the presence of thrombokinase, the fibrinogen is precipitated out as a fibrinous network. Fibrin plugs fill the lymphatic channels resulting in a lymphatic blockade, consequently the area becomes "walled off."

C. ALKALINE AND ACID PHASES OF INFLAMMATION:

With the inflammed area being "walled off" from the rest of the organism, it developes its own pH and metabolic pattern. The "walled off" inflammatory area becomes acid due to the anaerobic glycolytic metabolism of the pmn's and the production of organic acids such as lactic acid. This feature of an initial alkaline phase and later acid phase explains many of the sequences of events in the inflammatory reaction. Pmn's do not survive below pH 7.0 while macrophages survive fairly well at pH 6.8-6.9, thus explaining why the pmn's are replaced by macrophages as the reaction persists. Below pH 6.8, both pmn's and macrophages are destroyed and a state of

suppuration developes. LEUKOCYTOSIS, an increase in the number of circulating leukocytes and LEUKOPENIA, a decrease in the number of circulating leukocytes are both related to the inflammatory reaction. Factors released in both the alkaline and acid phases of inflammation are responsible for producing leukocytosis and leukopenia. These factors are: in the alkaline phase, LPF (leukocytosis promoting factor) causing leukocytosis and LEUKOPENIN causing leukopenia. There are two corresponding factors released in the acid phase. The level of circulating leukocytes is regulated by the interrelationship of the four mentioned factors. PYREXIN is a substance, released in the acid phase of inflammation, thought to be responsible for a systemic rise in temperature in the inflammatory reaction. NECROSIN is a substance released in the acid exudate which is thought to be one factor responsible for local tissue damage.

X. HORMONAL MODIFICATION OF INFLAMMATION:

ACTH causes liberation of hydrocortisone from the adrenal cortex (same effect as administration of cortisone). This tends to reduce inflammation, i.e., counteracts leukotaxin which reduces capillary permeability thus decreasing exudation and inhibiting the formation of granulation tissue. However, this has the detrimental effect of increasing susceptibility to bacterial and mycotic infections, possibly by decreasing phagocytosis.

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LECTURES NO. 4 and 5

THE PERIODONTAL LESIONS

Wilfred B. Bell*

I. DEFINITION:

Periodontal Pocket: A pathologically involved gingival sulcus with increased depth, the result of an increase in gingival bulk, rapid migration of the epithelial attachment with gingival detachment and alveolar bone loss, or a combination of both.

II. TYPES OF PERIODONTAL POCKETS:

A. SUPRABONY

Base of the pocket is coronal to the adjacent crestal alveolar bone.

1. Relative:

   Gingival overgrowth (enlargement) with no destruction of the subjacent osseous tissue.

2. Absolute:

   Destruction of the subjacent osseous tissue with or without gingival enlargement.

B. INFRABONY

Base of the absolute pocket is apical to the adjacent crestal alveolar bone.

1. One osseous wall.
2. Two osseous walls.
3. Three osseous walls.

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III. DEVELOPMENT OF THE POCKET

The sequence of tissue changes in the initiation and development of periodontal pockets has been described by many investigators with as many opinions. We will consider the following:

A. GOTTLIEB

The initial change in pocket formation occurs in the cementum. Nothing can induce rapid migration of the epithelial attachment in the presence of a highly developed cemental barrier. Inflammation and trauma are of secondary importance and can only do harm if there is a lowered resistance of the cemental surface or some inhibition in the deposition of cementum.

B. FISH

Destruction of the periodontal fibers is a prerequisite for the initiation of pocket formation. The periodontal fibers are a barrier to the epithelial attachment migration. Degeneration and necrosis of these fibers occur secondary to the gingival inflammation, allowing the epithelial attachment to proliferate apically until it reaches healthy periodontal fibers.

More recently, FISH describes the morphological characteristics of the gingival papillae as a ridge-shaped depression, or "col," between two peaks formed by the buccal and lingual papillae. In recently erupted teeth, the sides of the col are covered with a vestigal remnant of the enamel organ. This is normally replaced later by stratified squamous epithelium unless this vulnerable area is irritated inducing breakdown of the "col" with ulcer formation, detachment of the papillae, and progressive periodontal breakdown.

C. AISENBERG

Stimulation of the epithelial attachment by inflammation rather than destruction of the periodontal fibers is the prerequisite for the initiation of the periodontal pocket. The epithelial attachment has been observed microscopically to be attached to the cementum between individual periodontal ligament fiber bundles.

D. BOX

Invasion of bacteria at the base of the sulcus due to some defect in the epithelial attachment is followed by connective tissue degeneration and subsequent pocket formation.

E. WILKINSON

Epithelial changes initiate pocket formation. Inflammation is secondary. The sequence is as follows:

1. Thickening of the oral epithelium and epithelial attachment in the sulcus--etiology unknown.

2. Nutrition of the cells lining the sulcus is reduced with subsequent necrosis, degeneration and calcification (serumnal calculus).

3. The calcific masses separate from the underlying healthy epithelium and this produces a trough or pocket.

4. Proliferation of the epithelial attachment is followed by destruction of the connective tissue fibers and alveolar bone. This is subsequent to and dependent on the original epithelial changes.

F. WEINMANN AND OTHERS

Describe the histopathology of the periodontal pocket in terms of the several tissues involved in the extension of the inflammatory process, as follows:

1. The Epithelial Attachment

The epithelial attachment migrates apically at a rate in excess of what would be considered physiologic. Along with epithelial attachment migration, the position of the marginal gingivae will determine the extent of pocket formation. If the marginal gingivae remain in the same position, move coronally, or follow the epithelial attachment at a slower rate, then a pocket will develop. If the rate of migration of the epithelial attachment and the attached gingivae are the same, then we have recession without pocket formation.

2. Sulcular Epithelium

Extension of the epithelium into the underlying connective tissue results in a loss of the demarcation between the basal cells and the connective tissue and the formation of discrete epithelial buds. The intercellular bridges in the prickle cell layer become swollen, increase in size, and due to distension, rupture to form intercellular vesicles. Further degeneration of the lateral epithelium results in ulcer formation and necrosis with a break in the continuity of the epithelium. This allows the contents of this septic pocket area to come into direct contact with the underlying connective tissue.

3. Connective Tissue

An inflammatory response is elicited with associated tissue changes including alteration in circulation, edema, exudation, degeneration, and repair. The increase in bulk of the gingivae associated with the inflammatory response further aggravates the problem.

Since this is primarily a chronic inflammatory lesion, the cellular exudate contains predominently plasma cells and lymphocytes with few polymorphonuclear leukocytes. Combined with the leukocytic infiltration are newly-formed connective tissue cells, endothelial buds forming new blood vessels, and young fibrous tissue. This constitutes the granulation tissue found in periodontal pockets and reflects the repair phenomenon of the inflammatory process. The low intensity, constant irritation associated with local etiological factors responsible for pocket formation results in this less-than-complete effort to repair.

At the same time, areas of degeneration of the collagenous periodontal ligament fibers occur with the resultant loss of effective attachment for the fiber bundles involved.

4. Alveolar Bone

The extension of the inflammatory process into

the crestal alveolar bone results first in a marginal resorption, then subsequent extension via the perivascular spaces of the interalveolar system through the lateral extensions of the alveolus to the periodontal ligament.

The progressive marginal resorption of the alveolar bony housing results in further detachment of the periodontal fiber bundles and increased loss of tooth support.

5. Cementum

As the epithelial attachment progresses apically along the surface of the root, remnants of the embedded Sharpey's fibers on the denuded cementum are exposed to the deleterious effects of the septic pocket contents. This results in fragmentation and necrosis of the cementum. Destruction of the cementum allows exposure of the dentinal tubules of the dentine and subsequent formation of root caries.

G. GRANT AND ORBAN

Describe the presence of neutrophile granulocytes in the epithelial attachment and postulate that this may be the result of penetration of diffusible bacterial products from the depth of the pocket into and through the epithelial attachment. This could result in changes in the vitality of the cementoblasts and effect the integrity of the gingival fiber apparatus and the subsequent apical proliferation of the epithelial attachment.

H. GLICKMAN AND SMULOW

Observed in an animal study that excessive occlusal forces resulted in a direct extension of the inflammatory process into the periodontal membrane on the compression side, rather than into the interdental septum. Little change was noted on the tension side, but the authors explained that in their experiment, the tension was not severe enough to damage the periodontal membrane fibers and thereby open a pathway for the inflammation. Where the force was severe enough to cause necrosis of the periodontal membrane, this seemed to act as a barrier and direct extension into the periodontal membrane did not occur. It is interesting to note that the

artifically-induced pathologic changes noted early in the experiment, underwent repair in three to four months and the periodontium was restored. However, the periodontal injury induced by attrition tends to persist.

IV. CLASSIFICATION OF PERIODONTAL DISEASES (ORBAN)

A. INFLAMMATORY CONDITIONS: (75%)

1. Gingivitis:

Localized to free margin of gingiva.
Swelling, shallow pockets.
Acute or chronic according to duration.
Ulcerative, purulent, etc., according to symptoms.
Local or systemic according to etiology.
Local (Extrinsic); Infectious; Physical; Chemical.
Systemic (Intrinsic); Dietary deficiency; Endocrine disturbance.

2. Periodontitis:

Inflammation extends to deeper supporting tissues.
May be deep pockets, suppuration, abscess formation.
Varying degrees of alveolar resorption.

a. Simplex--following gingivitis.
b. Complex--following periodontosis.

B. DYSTROPHIC CONDITIONS: (25%)

1. Degenerative:

Qualitative changes (GREEK: Difficult nourishment).

a. Gingivosis:

(Chronic desquamative gingivitis)
Systemic etiology.
Connective tissue degeneration (others say epithelial degeneration). Possibly deficiency estrogenic hormone (female) or testosterne (male).

b. Periodontosis:

Degeneration of collagenous fibers of the

periodontal membrane with rapid migration of epithelial attachment and secondary pocket formation.
Irregular bone resorption with formation of granulation tissue.
Primarily systemic etiology--inherited inferiority of dental organ.
Early--no inflammation.
Late--deep pockets with periodontitis.

2. Atrophic: (QUANTITATIVE CHANGES)

Periodontal atrophy--bone recession.
Precocious aging. Aging. Disuse--loss of normal function.
Trauma--toothbrush, orthodontia.

3. Gingival Hyperplasia:

Overgrowth of the gingivae in varying degrees, fibrosis with lack of inflammation.
Infectious--pyogenic granuloma.
Endocrine dysfunction--pregnancy.
Drugs--Dilantin.
Idiopathic (idiopathic fibromatosis)

C. PERIODONTAL TRAUMATISM:

Pressure necrosis and its consequences.
Primary--overstress, bruxism, etc.
Secondary--loss of supporting tissue.

DISCUSSION

At the present time, it would appear that the inflammatory gingival periodontal lesion and its course into the deeper periodontal structures with concomitant tissue destruction is more readily understood as regards etiology and treatment than those periodontal conditions evidencing dystrophic manifestations. It is, therefore, not surprising that we recognize approximately 90% of periodontal disease as being inflammatory in nature.

While some investigators place a greater emphasis on one or the other of the periodontal tissues as being more vulnerable to "attack" by the etiologic factors, one should accept a broad view and realize that the tissues of the periodontium are influenced by a multiplicity of factors, many of which are unknown. Therefore, it is somewhat hazardous to predict, exactly, tissue behavior under any given set of circumstances.

The clinician may view all this as being an interesting academic problem rather nebulose, controversial, and not too practical. He wants to see a particular clinical picture respond to his specific treatment with a positive result which he can see and measure, using this criteria to establish the relative "health" of his results. This philosophy is more gratifying to the individual not in periodontia, but in other fields of dentistry where the "treatment site" is not so closely related to the sensitive balance of generalized systemic influences. In spite of this, however, experience has shown that the greatest success follows the early treatment of periodontal disease. THEREFORE THE IMPORTANCE OF EARLY TREATMENT CANNOT BE OVEREMPHASIZED.

A simple formula, which has been used many times to illustrate periodontal disease, is as follows:

Mechanical
Chemical
Bacterial

Injury X Duration X Frequency X Intensity (Modified by the resistance and reparative capacity of the host) EQUALS The Periodontal Lesion.

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LECTURE NO. 6

CLINICAL AND RADIOGRAPHIC EXAMINATION OF THE PERIODONTAL PATIENT

Rowland A. Hutchinson*

I. GENERAL CONSIDERATIONS:

A correct diagnosis of periodontal disease is dependent upon a complete dental and systemic history, detailed examination of the oral tissues, the periodontium, the occlusion, and utilization of various mechanical and laboratory tests when indicated. Examination procedures are designed to give adequate diagnostic information in order that rational therapy can be used in preference to symptomatic treatment. Rational therapy is directed toward the removal of the causative agent in addition to clinical signs and symptoms and, if properly instituted, will usually effect a cure which is preferred to the temporary improvement resulting from empirical treatment of signs and symptoms.

II. ARMAMENTARIUM AND CHARTS:

A. Necessary armamentarium includes:

1. Mouth mirror, explorer, periodontal millimeter probe.

2. Instruments or materials to determine pulp vitality.

3. Impression materials for making study casts.

4. Photographic equipment.

5. X-ray equipment.

B. The use of a standard periodontal diagnosis and treatment plan chart promotes systematic examination and helps prevent the omission of important details necessary to make a correct diagnosis. The chart is a valuable reference

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during recall visits. It should provide for the following:

1. Personal data: name, identification number, age, sex, race, marital status, and occupation.
2. Chief complaint.
3. Medical history and past dental history.
4. Dental outline to record clinical findings.
5. Examination record.
6. Analysis of case: etiology, diagnosis, prognosis.
7. Treatment plan.
8. Record of treatment.

III. CLINICAL EXAMINATION:

A complete clinical examination and charting should be accomplished at the first visit unless the patient is seen on an emergency basis. In the event the patient is seen for emergency treatment, a future appointment should be scheduled for a comprehensive examination.

A. A COMPREHENSIVE CLINICAL EXAMINATION SHOULD BE DONE SYSTEMATICALLY and should include the following:

1. Chief Complaint:

   This is the principal reason which led the patient to seek periodontal treatment. Questioning should include all events related to the complaint including a history from the origin until the present visit for treatment.

2. Medical History:

   This is very important, particularily regarding systemic conditions which affect the oral tissues and disabling diseases which lower the vitality and limit the periodontal treatment procedures, or have a bearing on prognosis.

   a. Present physical status should be ascertained.

b. Previous illnesses such as diabetes, rheumatic fever, cardiovascular disease, serious infections, and endocrine dysfunction should be noted.

c. Self-medications and allergic reactions should be elicited.

d. Abnormal bleeding or drug reactions are important.

e. Psychologic considerations should be determined. Is the patient apprehensive? does he appear interested and cooperative? how does he feel about his oral condition?

f. Brief dietary interrogation.

3. Past Dental History:

This is an important phase to consider in arriving at a diagnosis and eventual treatment plan. Patients vary greatly in their willingness to reveal facts regarding prior dental disease and experiences, and careful questioning may be necessary to elicit a complete and accurate dental history. The following items should be considered in obtaining the history:

a. Previous periodontal conditions and treatment.

b. Incidence of gingival hemorrhage.

c. History of abscesses or loose teeth.

d. Toothaches.

e. Reasons for missing teeth.

f. Previous dental experiences.

g. Oral hygiene habits.

h. Occlusal habits.

4. Facial Appearance:

Prior to examining the oral cavity, a superficial

examination should be made to observe the conformation of the jaws. Swelling or asymmetry, abnormal color of the skin, and the presence of bullae, vesicles, or pigmented lesions should be noted. Any signigicant odor of the breath should be noted; ie, odors such as alcohol, acetone, odor associated with ANUG.

5. Examination of the Oral Tissues:

Careful examination should be made of all the oral tissues and any deviation from normal should be recorded. The gingivae are closely inspected using the following as a guide:

a. Color

b. Form

c. Tone

d. Hemorrhage and exudate

e. Pain and sensitivity

f. Pigmentation

6. Study of the Occlusion:

At the initial examination, the occlusion should be inspected for gross prematurities both in centric and lateral occlusion. These gross defects should be noted and an attempt should be made to correlate the occlusal discrepancies with clinical and/or radiographic evidence of breakdown of the periodontium. If it is decided that occlusal correction is indicated, a more careful examination should be made at the time of the correction. Study casts mounted on an articulator using a wax bite transfer are an aid in diagnosing occlusal discrepancies.

7. Examination of The Teeth:

The teeth are examined with respect to each of the following:

a. Calculus--supragingival and subgingival.

b. Mobility--this should be recorded for each tooth and when mobility is present, an etiologic reason should be explored.

c. Contacts--open or defective contacts should be noted.

d. Restorative dentistry defects--amalgam and silicate overhangs; defective margins or contours of gold restorations and crowns.

e. Vitality--if periapical pathology is suspected, the vitality of the tooth should be determined.

8. Probing for Periodontal Pockets:

The location, shape, and depth of pockets are measured with a millimeter periodontal probe. This survey should be complete and should be conducted in a uniform manner. To accurately measure for pockets, the probe is gently inserted between the gingival tissue and tooth until resistance is felt. An attempt should be made to move the probe further apically by gentle manipulation. The pocket depth is noted on the facial, lingual, and interproximal areas of each tooth. Care should be taken that the probe is inserted parallel to the tooth surface to ensure obtaining an exact measurement. Any evidence of bleeding or exudation while probing the pocket should be noted. Furcation involvement should be carefully explored with an explorer and noted on the chart.

B. STUDY CASTS:

The study casts are an important adjunct to the clinical examination and are an aid in treatment planning. They afford a three-dimensional record of the occlusion, a lingual view of occlusal realtionships, and when properly mounted in an articulator, they are an aid in determining occlusal prematurities. They provide a clear view of tooth morphology, open contacts, wear facets, and tooth soft tissue relationships which are often distorted in the mouth due to the transulcency of the enamel and the presence of saliva on the teeth.

C. COLOR PHOTOGRAPHS:

These provide a record of the periodontal condition and progress during treatment. Referral to these records is valuable upon recall of the patient since color changes are a characteristic feature of many periodontal changes.

D. LABORATORY TESTS:

Various laboratory procedures may be necessary as aids in diagnosis of periodontal conditions which are suspected of being associated with systemic diseases. The tests more commonly used are as follows:

1. Red cell count.

2. Differential white cell count.

3. Determination of the serum calcium, serum phosphorous, and alkaline phosphatase.

4. B.M.R. - Basal metabolic rate.

5. Laboratory studies for the detection of diabetes mellitus: urinary glucose, blood glucose, glucose tolerance test.

6. Bacteriologic examination.

7. Biopsy.

IV. RADIOGRAPHIC EXAMINATION:

"The diagnosis of periodontal disease is made with a periodontal probe, complimented by inspection and palpation. Although the dental roentgenogram plays a significant role in periodontal treatment planning and prognosis, the patient must be examined clinically before a diagnosis of periodontal disease can be made." (Prichard). Radiographs supplement the information obtained from clinical observation and as such are an important aid in diagnosis. "The presence of radiographic changes means that the disease process has progressed to a stage sufficient to alter the structures to a radiographically detectable degree. Therefore it is important to differentiate between the earliest clinical signs of periodontal disease and the earliest radiographic signs of periodontal disease." (Glickman). It bears repetition that the radiograph is only an aid in diagnosis of

the patient's periodontal condition.

A. RADIOGRAPHS ARE AN AID IN THE DIAGNOSIS OF PERIODONTAL DISEASE BY DEMONSTRATING THE FOLLOWING:

1. Dense interproximal calculus deposits.
2. Periapical pathologic involvement.
3. Root form and shape.
4. The height of the alveolar process in one plane.
5. Loss of lamina dura.
6. Thickening of the periodontal ligament.
7. Root resorption.
8. Restorative dentistry defects.

B. RADIOGRAPHS DO NOT SHOW THE FOLLOWING:

1. Hard to soft tissue relationships.
2. Whether the case is treated or untreated.
3. Structures on the facial or lingual surfaces of the root.
4. Morphology of bone deformities.
5. The number of osseous walls present in an infrabony defect,
6. Tooth mobility.

V. DISCUSSION:

The diagnosis of periodontal disease is predicated upon a complete comprehensive examination of the patient and correct interpretation of the case history and clinical findings in terms of the operators knowledge of the periodontium. Diagnosis should also include an understanding of the underlying disease processes and their etiology. It is well to remember that we are treating a patient with a disease, NOT a disease with a patient.

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PERIODONTAL DIAGNOSIS AND TREATMENT PLAN

NAME (Last, First, Middle Initial)				GRADE	SERVICE NR	CASE NR	DATE
JONES, John				PFC	1234562890		8 Apr 63
AGE	**SEX**	**RACE**	**MARITAL STATUS**	**OCCUPATION**		**PHOTO NR**	**CASTS NR**
19	Male	Caucasian	Single	Clerk		12-16	---

CHIEF COMPLAINT: Bleeding and tenderness on toothbrushing.

MEDICAL HISTORY:

Are you presently under the care of a physician? ☐ YES ☒ NO

Are you taking any medications? (Laxatives, pain or headache relievers) ☐ YES ☒ NO

Have you ever been treated for heart disease? ☐ YES ☒ NO

Are you allergic to any drugs? (Antibiotics, aspirin) ☐ YES ☒ NO

Have you ever had abnormal bleeding? ☐ YES ☒ NO

Have you ever had any ill effects from local anesthetics? ☐ YES ☒ NO

REMARKS:

Patient recently recovered from acute upper respiratory infection of 2 weeks' duration

CLINICAL AND RADIOGRAPHIC EXAMINATION

CHARTING INSTRUCTIONS: Mark gingival height in red; bone height in blue; X missing tooth; ⌧ tooth to be extracted; record mobility as 1, 2, 3, and write in on crown; record prognosis as G: F: Q: P: H, and mark letter on crown; record periodontal abscesses as PA written on root; record x-ray changes on root or in apical area by drawing; record vitality by + or − written above the crown.

CHART A - Mark in black contacting teeth in working, balancing and protrusive position.

CHART B - Mark centric contacts in black; circle prematurities in red.

CHART A

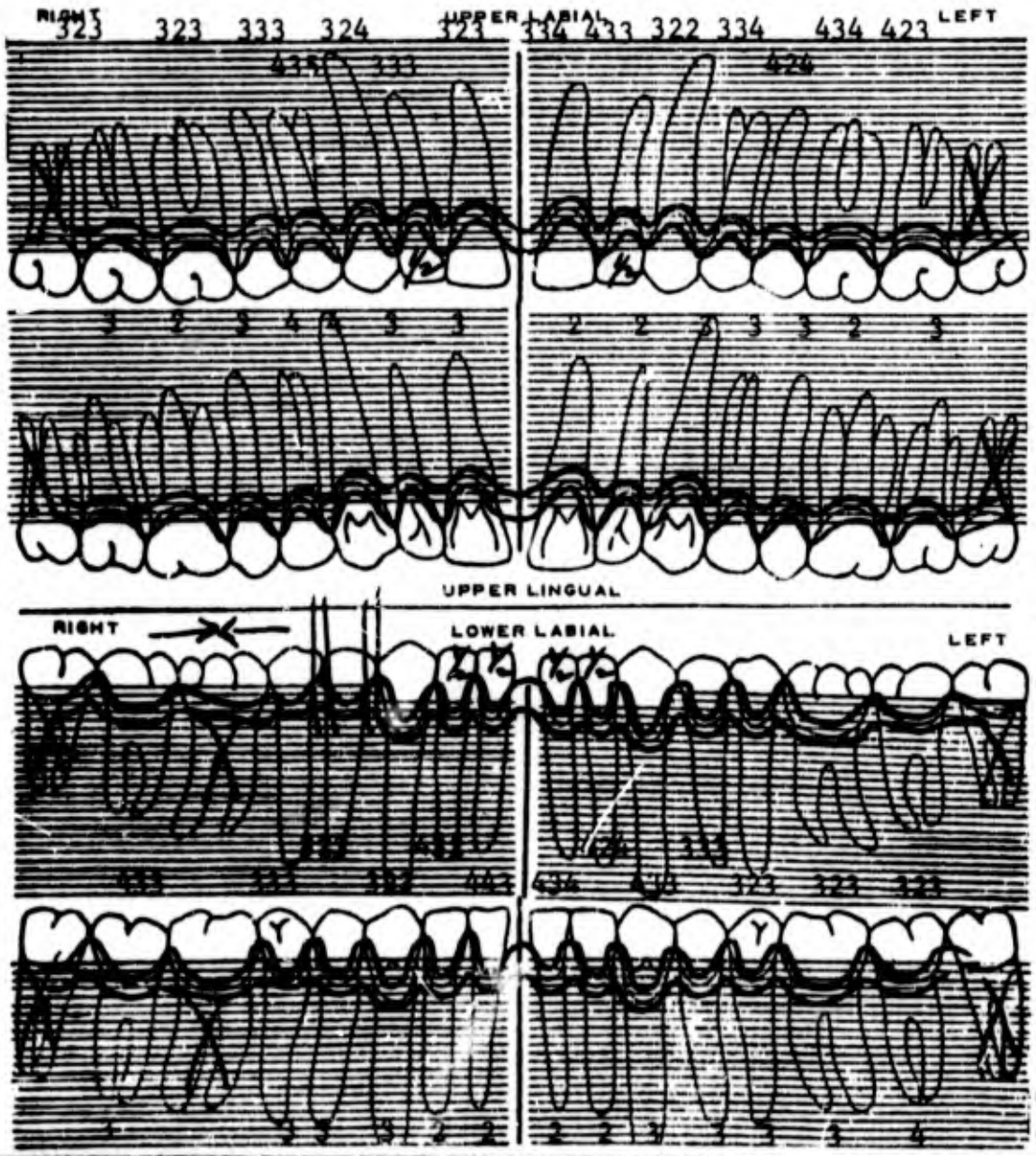
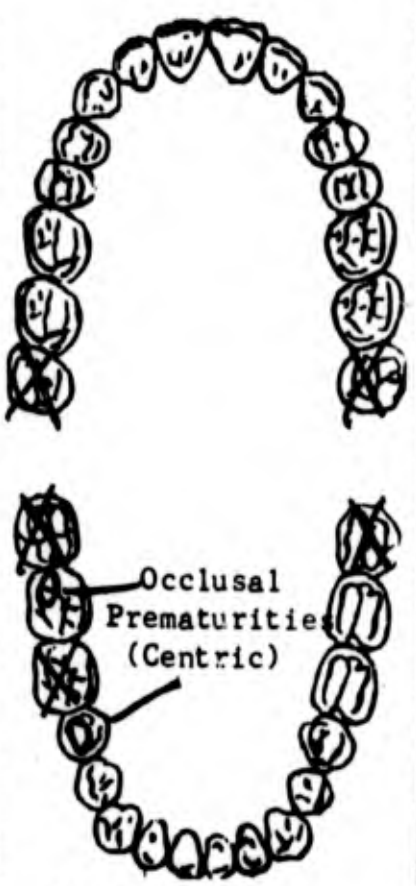


CHART B



REMARKS
Diastemata
Malpositioned teeth, etc.

#29 distally inclined
#31 mesially inclined
Open contact area #27-28
Open contact area #28-29

WRAMC FORM 77
18 AUG 1952

(Continue) CLINICAL AND RADIOGRAPHIC EXAMINATION

ORAL MUCUOSA
Abrasions
Growths — None

TONGUE
Coating
Color
Epithelial Covering
Growths
Shape
Size — Normal

FLOOR OF MOUTH
Lesions — None

ROOF OF MOUTH
Torus — None

THROAT — Normal

LIPS — Normal

SALIVA — Serous

OCCLUSAL HABITS
Bruxism - No
Fingernail Biting - Yes
Pencil Biting - No
Tongue Thrusting - No
Mouth Breathing - Yes
Soft Tissue Biting - No
Others - None

PSYCHOSOMATIC FACTORS
None

ORAL HYGIENE HABITS
Dentifrice Paste
Frequency once a day,
Method misses week-
Brush ends.
Effectiveness Poor

CALCULUS-Mod. supra- and subgin. mostly materia alba & debris.

PERIODONTIUM

GINGIVAE -Hyperplastic & edematous

ALVEOLAR BONE
Good

CEMENTUM
Good

TEMPOROMANDIBULAR JOINTS
Normal

LABORATORY STUDIES INDICATED

URINALYSIS
C.B.C.
GLUCOSE TOLERANCE
OTHER

None

NUTRITIONAL ASSAY

Adequate

PAST DENTAL HISTORY

PREVIOUS PERIODONTAL TREATMENT

Scaling and prophy. 4 years ago.

OTHER DENTAL TREATMENT

Extraction #30 - 9 years ago. No Dental care for 2 years.
Routine operative dentistry.

ANALYSIS OF CASE
(Etiology, Diagnosis, Prognosis)

ETIOLOGY: Local: Poor Oral Hygiene - materia alba, debris, lack of gingival stimulation
Calculus, supra and sub., moderate.
Food impaction due to poor contacts #27, 28, 29 areas.
Caries and poor restorative dentistry.
Systemic: Resistance probably lowered by recent URI.
DIAGNOSIS: Marginal gingivitis, generalized. Inflamed, hyperplastic and edematous marginal gingiva. No involvement of alveolar mucosa or supporting bone.
PROGNOSIS: Excellent following therapy and patient maintenance.

TREATMENT PLAN
(To Include Recommendations for Replacements and Best Sequence of Treatments)

1. Scaling
2. Oral hygiene instructions.
3. Subgingival curettage.
4. Occlusal equilibration (centric prematurities #29 and #31).
5. Re-evaluation.
6. Replacement of #19 and correction of open contacts (#27, 28, 29 areas) by fixed prosthesis.

DATE	TREATMENT RECORD AND PROGRESS NOTES *(Continue on Reverse Side)*	
8 Apr 63	Examination, preop. photos taken, charting completed.	
	//Scaling.	DPM
15 Apr 63	Cavitron scaling - upper and lower quadrants.	
	//Oral hygiene instructions.	DPM
22 Apr 63	Oral hygiene instructions - disclosing solution - use of stimudents,	
	rubber interproximal stimulator, dental floss, etc.	DPM
29 Apr 63	Subgingival curettage and root planing. Upper and lower right	
	quadrants.	
	//Curettage, left side.	DPM
6 May 63	Subgingival curettage - left side, upper and lower.	
	//Occlusal equilibration.	DPM
13 May 63	Occlusal adj. Reviewed oral hygiene. Re-emphasized lingual uppers	
	where slight debris noted. Referred to operative for replacement	
	#19 and correction of contact areas #27, 28, 29. Polished.	
	//Recall 3 months	DPM

PERIODONTAL DIAGNOSIS AND TREATMENT PLAN

NAME (Last, First, Middle Initial)				GRADE	SERVICE NR	CASE NR	DATE
BROWN, Edward				Capt	077765		7 May 63
AGE	**SEX**	**RACE**	**MARITAL STATUS**	**OCCUPATION**		**PHOTO NR**	**CASTS NR**
29	Male	Caucasian	Married	Helicopter pilot		#6-10	--------

CHIEF COMPLAINT: NONE

MEDICAL HISTORY:

Are you presently under the care of a physician? ☐ YES ☒ NO

Are you taking any medications? (Laxatives, pain or headache relievers) ☐ YES ☒ NO

Have you ever been treated for heart disease? ☐ YES ☒ NO

Are you allergic to any drugs? (Antibiotics, aspirin) ☐ YES ☒ NO

Have you ever had abnormal bleeding? ☐ YES ☒ NO

Have you ever had any ill effects from local anesthetics? ☐ YES ☒ NO

REMARKS

Good health.

CLINICAL AND RADIOGRAPHIC EXAMINATION

CHARTING INSTRUCTIONS: Mark gingival height in red; bone height in blue; X missing teeth; ‖ teeth to be extracted; record mobility as 1, 2, 3, and write in an crown; record prognosis as G: F: Q: P: H, and mark letter on crown; record periodontal abscesses as PA written on root; record x-ray changes on root or in apical area by drawing; record vitality by + or − written above the crown.

CHART A - Mark in black contacting teeth in working, balancing and protrusive position.

CHART B - Mark centric contacts in black; circle prematurities in red.

CHART A

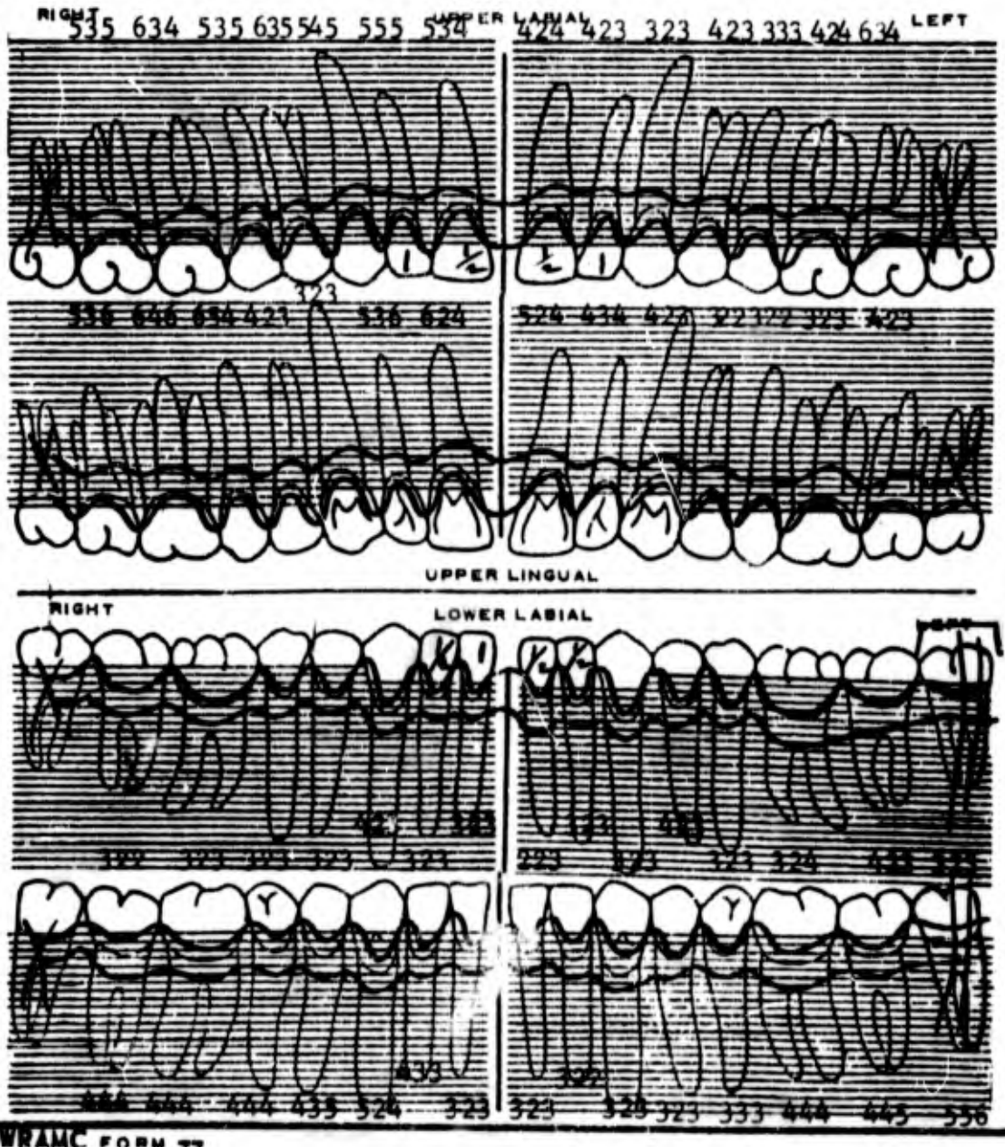
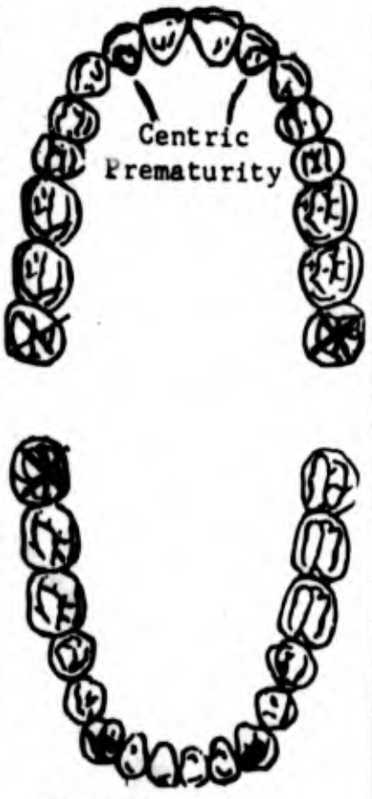


CHART B



REMARKS
Diastema
Malpositioned teeth, etc.

#17, pericoronitis, 1960

(Continue) CLINICAL AND RADIOGRAPHIC EXAMINATION

ORAL MUCUOSA Hyperkeratosis, left tuberosity area of palate.
Abrasions
Growths

TONGUE Normal
Coating
Color
Epithelial Covering
Growths
Shape
Size

FLOOR OF MOUTH
Lesions — None

ROOF OF MOUTH
Tone — None

THROAT Normal

LIPS Normal

SALIVA Normal

OCCLUSAL HABITS None
Bruxism
Fingernail Biting
Pencil Biting
Tongue Thrusting
Mouth Breathing
Soft Tissue Biting
Others

PSYCHOSOMATIC FACTORS

ORAL HYGIENE HABITS
Dentifrice Paste
Frequency 2 x daily
Method
Brush
Effectiveness Fair

CALCULUS
Heavy supra- & mod. subgingival.

PERIODONTIUM
Mostly fibrotic.

GINGIVAE Little inflammation. Pink color.

ALVEOLAR BONE Horizontal bone loss. Loss of interprox. septa.

CEMENTUM
Normal.

TEMPOROMANDIBULAR JOINTS
Normal.

LABORATORY STUDIES INDICATED

URINALYSIS
C.B.C.
GLUCOSE TOLERANCE
OTHER
None

NUTRITIONAL ASSAY
Adequate

Smokes 1 pack per day plus pipe.

PAST DENTAL HISTORY

PREVIOUS PERIODONTAL TREATMENT

1959 - scaling and prophy. & OHI

OTHER DENTAL TREATMENT

1960 - Pericoronitis #17 - #16 extracted.
1958 - #1 and #32 extracted.
1957-1959 - Operative - Caries on #2, 3, 28 - resotred with amalgam.

ANALYSIS OF CASE
(Etiology, Diagnosis, Prognosis)

ETIOLOGY: Mostly local factors - Calculus (supra-and subgingival), inadequate oral hygiene and lack of gingival stimulation, amalgam restorations with poor contact areas and rough margins, history of pericoronitis, #17.
DIAGNOSIS: Periodontitis, moderate, generalized; slow progression of the inflammatory process has caused formation of fibrotic, somewhat hypertrophied gingiva with pocket depth varying from 3 to 6 mm. Pocket depth does not extend into alveolar mucosa, therefore, the surgical aspect of therapy will be confined to gingivectomy--no muco-gingival surgery necessary.

PROGNOSIS: Good for all teeth.

TREATMENT PLAN
(To Include Recommendations for Replacements and Best Sequence of Treatments)

1. Extract #17 - nonfunctional and extruded.
2. Scale and root plane all quadrants.
3. Oral hygiene instructions.
4. Gross occlusal equilibration to remove centric prematurities.
5. Gingivectomies - all quadrants.
6. Occlusal evaluation and equilibration.
7. Replacement of defective amalgam restorations.

DATE	TREATMENT RECORD AND PROGRESS NOTES *(Continue on Reverse Side)*	
7 May 63	Examination, photos, appt. with Oral Surgery (Ext. #17)	
	//Scale right side.	DPM
14 May 63	Scaled upper and lower right side, polished Class V amalgams, right side.	
	//Scale left side.	DPM
21 May 63	Scaled left side, polished amalgams	
	//OHI & occl. equil.	DPM
28 May 63	OHI & disclosing sol. & stimudents.	
	//Gtmy - upper right.	DPM
5 Jun 63	Gingivectomy - upper right quad.	
	//Gtmy - lower right, and POT	DPM
12 Jun 63	Gingivectomy - lower right quad. POT, upper right.	
	//Gtmy, upper left.	DPM
19 Jun 63	Gingivectomy - upper left quad. POT, lower right.	
	//Gtmy, lower left.	DPM
26 Jun 63	Gingivectomy, lower left. POT, upper left.	
	//POT	DPM
3 Jul 63	POT, lower left	
	//Occl. equil.	DPM
10 Jul 63	Occlusal equilibration; review of oral hygiene instructions.	
	Patient placed on 2-month initial recall. Referred to Operative.	DPM

PERIODONTAL DIAGNOSIS AND TREATMENT PLAN

NAME (Last, First, Middle Initial)				GRADE	SERVICE NR	CASE NR	DATE
POTTS, Robert				Col	033445	3	11 April 1963
AGE	**SEX**	**RACE**	**MARITAL STATUS**	**OCCUPATION**		**PHOTO NR**	**CASTS NR**
45	Male	Caucasian	Married	JAG		#7-11	#5

CHIEF COMPLAINT: Dull vague pain--upper right side--#5-6 area--food impaction.

MEDICAL HISTORY:

Are you presently under the care of a physician? [X] YES [ ] NO

Are you taking any medications? (Laxatives, pain or headache relievers) [X] YES [ ] NO

Have you ever been treated for heart disease? [ ] YES [X] NO

Are you allergic to any drugs? (Antibiotics, aspirin) [ ] YES [X] NO

Have you ever had abnormal bleeding? [ ] YES [X] NO

Have you ever had any ill effects from local anesthetics? [ ] YES [X] NO

REMARKS:

Patient has duodenal ulcer--controlled by diet and amphogel.

CLINICAL AND RADIOGRAPHIC EXAMINATION

CHARTING INSTRUCTIONS: Mark gingival height in red; bone height in blue; X missing teeth; H tooth to be extracted; record mobility as 1, 2, 3, and write in on crown; record prognosis as G: F: Q: P: H, and mark letter on crown; record periodontal abscesses as PA written on root; record x-ray changes on root or in apical area by drawing; record vitality by + or − written above the crown.

CHART A - Mark in black contacting teeth in working, balancing and protrusive position.

CHART B - Mark centric contacts in black; circle prematurities in red.

CHART A

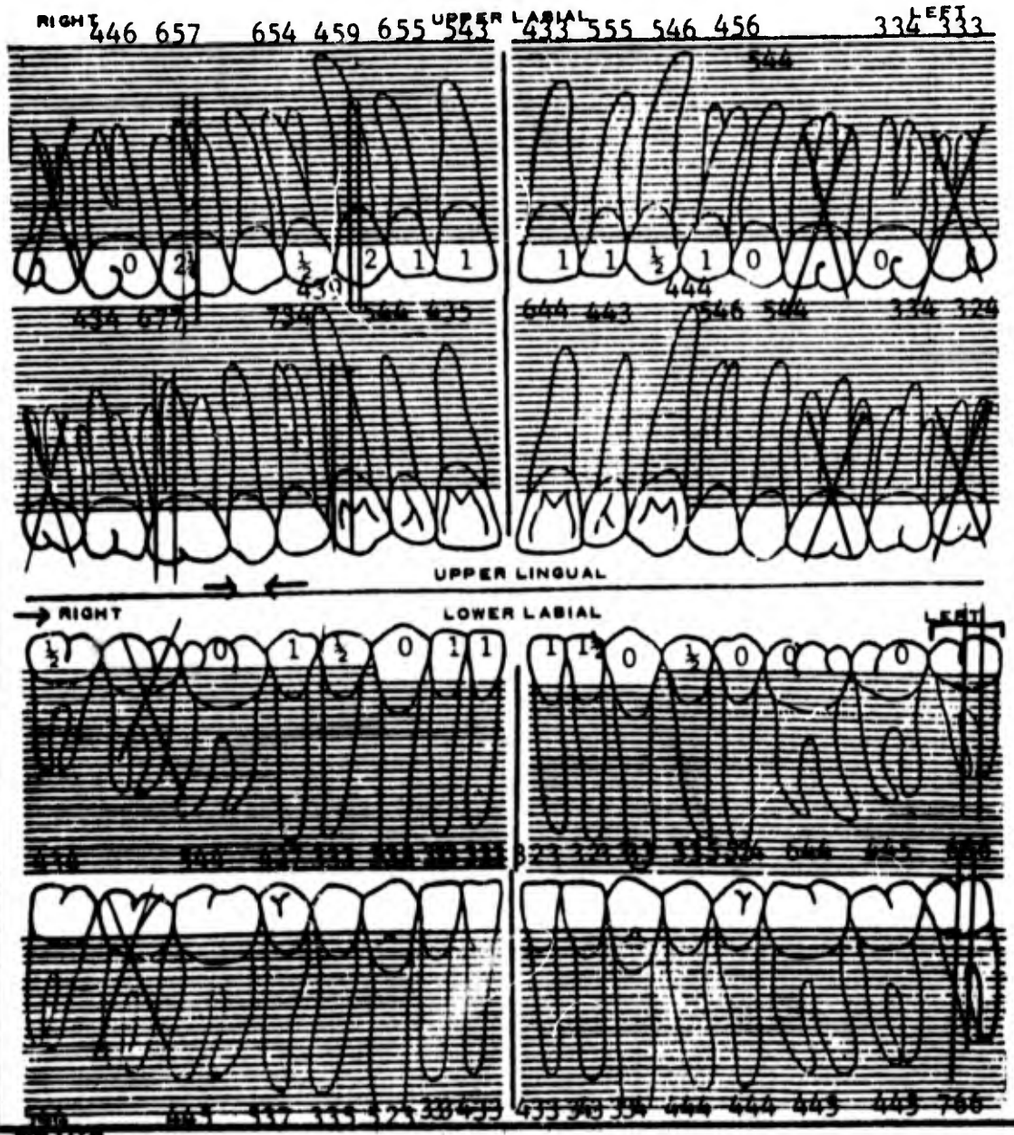
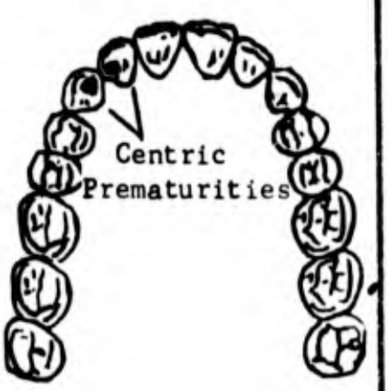
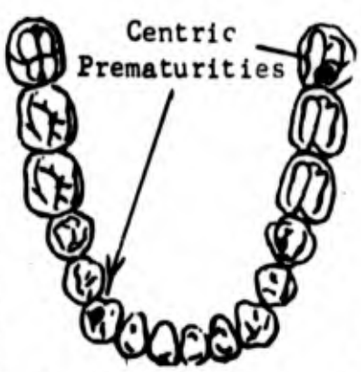


CHART B





REMARKS
Diastema, Malpositioned teeth, etc.

#32 - Mesially inclined
#17 - extruded
#5 & 6 - Open contact area.

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(Continue) CLINICAL AND RADIOGRAPHIC EXAMINATION

ORAL MUCUOSA
Abrasions, Growths: None

TONGUE
Coating, Color, Epithelial Covering, Growths, Shape, Size: Normal

FLOOR OF MOUTH
Lesions: None

ROOF OF MOUTH
Torus: None

THROAT
Normal

LIPS
Diffuse hyperkeratosis

SALIVA
Normal

OCCLUSAL HABITS
Bruxism --------Yes
Fingernail Biting, Pencil Biting, Tongue Thrusting, Mouth Breathing, Soft Tissue Biting, Others: No

PSYCHOSOMATIC FACTORS
None

ORAL HYGIENE HABITS
Dentifrice--Paste
Frequency-2 x's daily
Method, Brush
Effectiveness-Fair

CALCULUS
Moderate supra- and heavy subgingival

PERIODONTIUM

GINGIVAE-Mostly fibrotic with edematous interdental papilla.
ALVEOLAR BONE
Horizontal & vertical bone loss.
CEMENTUM Normal

TEMPOROMANDIBULAR JOINTS
Normal

LABORATORY STUDIES INDICATED

URINALYSIS - Yes
C.B.C. - No
GLUCOSE TOLERANCE-Yes
OTHER
Possible diabetes

NUTRITIONAL ASSAY
Adequate

Smokes 2 packs daily

PAST DENTAL HISTORY

PREVIOUS PERIODONTAL TREATMENT

1958, 1959, 1960, 1961, 1962 - Periodontal scaling and possible soft tissue curettage

1958 and 1960 - Occlusal equilibration

Routine prophylaxis periodically.

1961 - Periodontal Abscess #5-6 area, incision and drainage

OH Instructions - 1958

OTHER DENTAL TREATMENT

1940 - #1, 16 extracted--caries, nonrestorable.

1950 - #14 extracted - Perio. abscess.

1951 - #31 extracted - Perio. abscess.

ANALYSIS OF CASE
(Etiology, Diagnosis, Prognosis)

ETIOLOGY: Patient presents with advanced periodontal disease due to usual local factors probably superimposed on poor systemic resistance and repair factors--calculus, inadequate oral hygiene, malposed teeth, open contacts, food impaction areas, poor margins on restorations, caries #6, 9, 13 and 19. Wear facets and mobility indicative of occlusal trauma.
DIAGNOSIS: Periodontitis with occlusal trauma (primary and secondary occl. trauma)

PROGNOSIS: #3, 6, 17 - Hopeless
#5, 7, 12, 29 - Poor
Remaining teeth good pending patient cooperation following therapy.
Generally more favorable for remaining lowers.

TREATMENT PLAN
(To Include Recommendations for Replacements and Best Sequence of Treatments)

1. Extract #3, #6, #17 (periodontally hopeless).
2. Periodontal scaling and root planing--all remaining teeth.
3. Oral hygiene instructions.
4. Gross occlusal equilibration.
5. Mucogingival surgery--all quadrants beginning with--lower right--#29 distal infrabony pocket - 3-walled.
6. Final occlusal equilibration.
7. Re-evaluation.
8. Replace #31 by 3-unit fixed bridge; replace #3, 4, 6 and 14 with removable prosthesis.

DATE	TREATMENT RECORD AND PROGRESS NOTES *(Continue on Reverse Side)*	
18 Apr 63	Examination, charting, photos, study models. Blood work negative.	
	//Perio. scaling--upper and lower right	DPM
25 Apr 63	Scaled and root planed - right side	
	//Scale & root plane - left side	DPM
2 May 63	Scaled and root planed - upper and lower left side	
	//OHI and gross occl. equil.	DPM
9 May 63	OHI and gross occl. equil. Appts made with Oral Surgery for extractions.	
	//Mucogingival surgery--lower right quad.	DPM
23 May 63	Gingivectomy - lower right quadrant. Reverse bevel and repositioned	
	flap. Sutures. IBP #29 mesial - 4 mm. 3-walled.	
	//Gtmy - upper right quadrant	DPM
30 May 63	Gingivectomy - upper right quadrant. Reverse bevel and repositioned	
	flap. Sutures. POT - lower right quad. Removed sutures & replaced	
	periodontal dressing.	
	//Gtmy - lower left quad. & POT - right side.	DPM
7 Jun 63	Gingivectomy - lower left quad. - removed dressing lower right quad.	
	repacked IBP #29.	
	//Gtmy - upper left.	DPM
14 Jun 63	Gingivectomy - upper left quad. - repositioned flap, sutures, etc.	POT
21 Jun 63	POT Removed sutures, changed dressings. //POT	DPM
27 Jun 63	POT - Removed all periodontal dressing. // Final occ. equil.	DPM
12 Jul 63	Final occl. equil. //Prophylaxis	DPM
19 Jul 63	Prophylaxis, re-evaluation. Recall--3 months. Referred to Crown	
	and Bridge and Prosthetics.	DPM

PERIODONTAL DIAGNOSIS AND TREATMENT PLAN

NAME (Last, First, Middle Initial)				GRADE	SERVICE NR	CASE NR	DATE
ANDERSON, Susan				Dep			19 Jan 63
AGE	**SEX**	**RACE**	**MARITAL STATUS**	**OCCUPATION**		**PHOTO NR**	**CASTS NR**
16	F	Negroid	Single	Student		12-15	7

CHIEF COMPLAINT: Loosening and migration of teeth, gingival bleeding.

MEDICAL HISTORY:

Are you presently under the care of a physician? [XX] YES [ ] NO

Are you taking any medications? (Laxatives, pain or headache relievers) [ ] YES [XX] NO

Have you ever been treated for heart disease? [ ] YES [XX] NO

Are you allergic to any drugs? (Antibiotics, aspirin) [ ] YES [XX] NO

Have you ever had abnormal bleeding? [ ] YES [XX] NO

Have you ever had any ill effects from local anesthetics? [ ] YES [XX] NO

REMARKS:

Presently under examination by Medical Clinic to rule out systemic factors; i.e., diabetes, blood dyscrasias, vitamin deficiencies.

CLINICAL AND RADIOGRAPHIC EXAMINATION

CHARTING INSTRUCTIONS: Mark gingival height in red; bone height in blue; X missing teeth; M teeth to be extracted; record mobility as 1, 2, 3, and write in on crown; record prognosis as G: F: Q: P: H, and mark letter on crown; record periodontal abscesses as PA written on root; record x-ray changes on root or in apical area by drawing; record vitality by + or - written above the crown.

CHART A - Mark in black contacting teeth in working, balancing and protrusive position.

CHART B - Mark centric contacts in black; circle prematurities in red.

CHART A

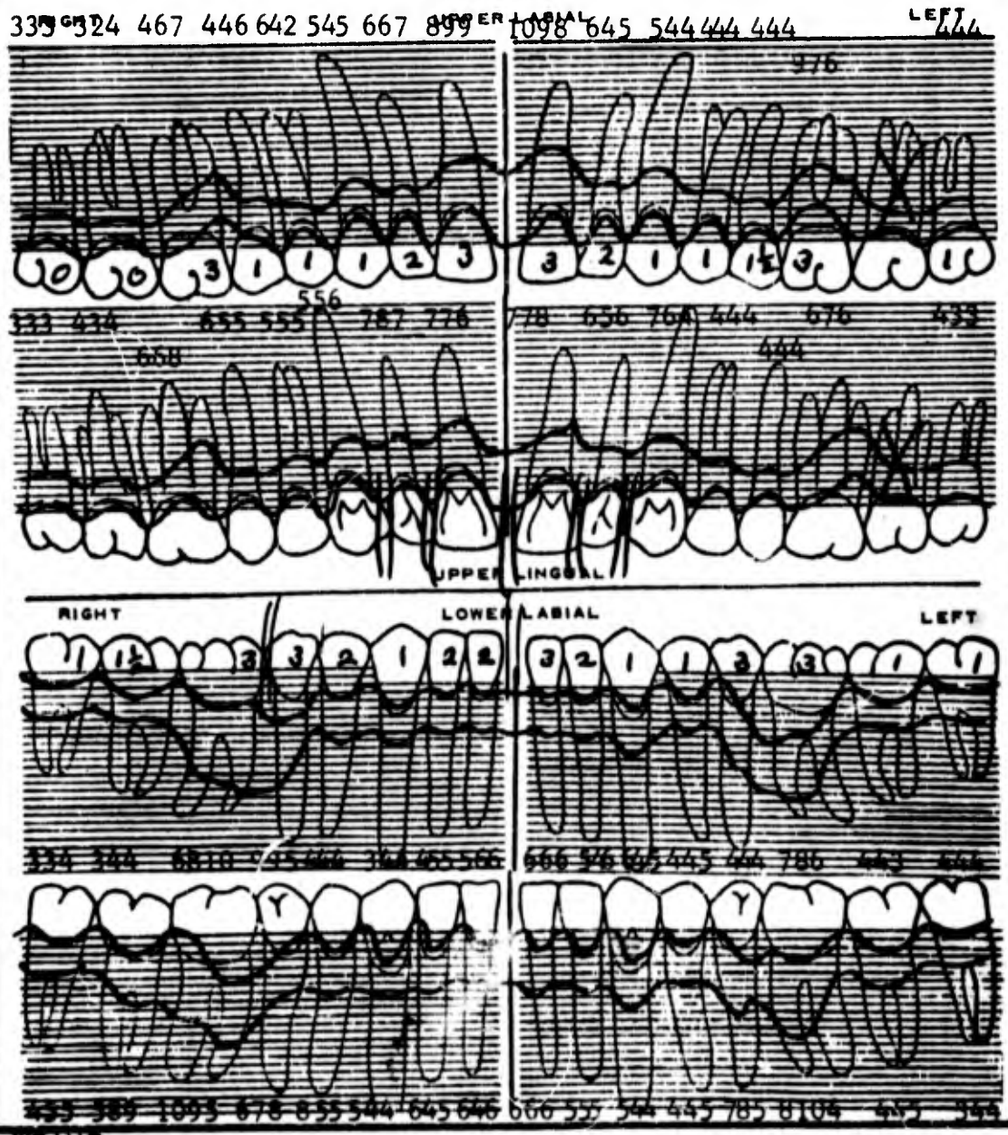
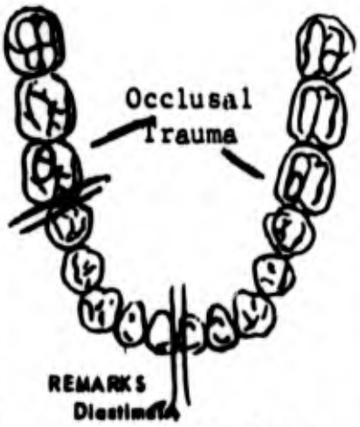


CHART B



REMARKS
Diastemata
Malpositioned teeth, etc.

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(Continue) CLINICAL AND RADIOGRAPHIC EXAMINATION

ORAL MUCUOSA
Abrasions, Growths - None

TONGUE
Coating, Color, Epithelial Covering, Growths, Shape, Size - Normal

FLOOR OF MOUTH
Lesions - None

ROOF OF MOUTH
Torus - None

THROAT
Normal

LIPS
Normal

SALIVA
Normal

OCCLUSAL HABITS
Bruxism, Fingernail Biting, Pencil Biting, Tongue Thrusting, Mouth Breathing, Soft Tissue Biting, Others - None

PSYCHOSOMATIC FACTORS
None

ORAL HYGIENE HABITS
Dentifrice - Paste
Frequency - 3 x daily
Method - after meals
Brush
Effectiveness - Good

CALCULUS
None

PERIODONTIUM

GINGIVAE edematous, hyperplastic, areas of ulceration.
ALVEOLAR BONE-Widening of P.D. ligament; vertical resorption.
CEMENTUM
Normal

TEMPOROMANDIBULAR JOINTS
Normal

LABORATORY STUDIES INDICATED

URINALYSIS Yes
C.B.C. Yes
GLUCOSE TOLERANCE Yes
OTHER hemoglobin
Blood phosphorus
Blood serum calcium
17-ketosteroids
NUTRITIONAL ASSAY
Adequate

PAST DENTAL HISTORY

PREVIOUS PERIODONTAL TREATMENT

1961 - Therapy consisted of scaling, occlusal equilibration ("one or two teeth") and vitamin therapy.

OTHER DENTAL TREATMENT

Low caries index
3 occlusal amalgams - #19, 21 & 29 - 1960
#15 extracted 1961
(History of mobility and abscess)

ANALYSIS OF CASE
(Etiology, Diagnosis, Prognosis)

ETIOLOGY - Very few local factors present to account for rapid and severe periodontal pathology which is manifested by extreme mobility, deep pockets and migration of the teeth (these signs are most severe in first molars and anterior teeth.--Oral hygiene good, caries index low, calculus, slight--no bruxism, etc. Occlusal traumatic areas are secondary to the migration and mobility.--Systemic factors of diabetes, vit. deficiency, blood dyscrasias ruled out by medical examinations.--Probable degeneration of the periodontal membrane fibers and the alveolar bone.
DIAGNOSIS: Periodontosis.
PROGNOSIS: Poor for first molars and anterior teeth; other teeth questionable.

TREATMENT PLAN
(To Include Recommendations for Replacements and Best Sequence of Treatments)

1. Extract #3, 7, 8, 9, 10, 14, 19 and 30.
2. Temporary immediate P/dtr.
3. Gross occlusal equilibration.
4. Splint mobile lower anteriors with steel wire and acrylic splint, #22-27.
5. Kadon intra-coronal splint, upper and lower bicuspids, upper and lower molars.
6. Perio. scaling and curettage.
7. Oral hygiene instructions.
8. Occlusal equilibration.
9. Re-evaluation.
10. Gingival surgery - all remaining teeth.
11. Fixed upper and lower prosthesis to splint remaining teeth and replace missing teeth.

DATE	TREATMENT RECORD AND PROGRESS NOTES *(Continue on Reverse Side)*	
19 Jan 63	Examination, photos, study models, etc.	
	//Continue exam.	DPM
26 Jan 63	Exam completed - consult with Surgery & Prosthetics. Referred to	
	Medical Clinic for evaluation. Appt. with Surgery and Prosthetics	DPM
5 Feb 63	Lower anterior stainless steel and acrylic splint.	
	//Kadon splint, upper and lower right bicuspids	DPM
6 Feb 63	Kadon Splints, upper and lower, right side.	DPM
10 Feb 63	Kadon splints, upper and lower, left side.	DPM
14 Feb 63	Scaling & curettage, left side.	DPM
17 Feb 63	Scaling & curettage, right side.	DPM
21 Feb 63	Oral Hygiene Instructions	
	//Occlusal equilibration	DPM
24 Feb 63	Occlusal equilibration	
	//Re-evaluate - Possibility of gingival surgery.	DPM
	Will reappoint in two weeks.	

LECTURE NO. 7

CASE ANALYSIS

Dominick P. Mandracchia*
and
Samuel Kobrinsky**

I. INTRODUCTION:

Case analysis connotes a thorough understanding of a particular case whether it be a patient with carious teeth which need restorations, impacted teeth which require removal, missing teeth which need replacements, or gingivitis which needs to be treated.

Case analysis requires the knowledge and understanding of the causative factors (etiology); the ability to distinguish one disease process from another (diagnosis); and the ability to forecast the course and outcome of a disease either with or without treatment (prognosis).

Etiology, diagnosis, and prognosis are evolved following a thorough examination of a patient, and the resulting case analysis gives us the ability to formulate the correct treatment plan.

Patients should not be treated without the proper examination, case analysis, and treatment plan.

II. ETIOLOGY:

In considering the etiology of the various manifestations of periodontal disease, it must be remembered that there are many causative agents and that the same etiologic factor may produce disease of different intensity in different individuals; i.e., food impaction, causing an inflammation of the interdental papilla may result in slight damage to one person, while in another the gingival tissues may be markedly affected.

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**Major, United States Army, Dental Corps, Walter Reed General Hospital, Washington 12, D. C.

The causative agent may alter in intensity and frequency and the repair ability can change depending on the physical health of the individual, thus, the same causative agent may cause disease of varying intensity. The following formula used elsewhere is important enough to repeat here.

> Local etiologic factor x severity x frequency of injury x duration of disease process modified by the resistance and reparative capacity of the host equals periodontal manifestations.

Etiologic factors may be roughly classified into local irritants and systemic or qualifying factors. The local agents act directly on the periodontium, while the systemic factors (the resistance to breakdown and the repair potential) modify and qualify the effect of local irritants. The result of this interaction is the clinical picture of the disease as we see it.

A. <u>Gingival Irritants - Primary Factors</u>:

1. Calculus - most important of gingival irritants; formation not fully understood. Effective oral hygiene helps keep teeth free of adhering material and calculus. Abrasive and detergent foods keep teeth cleaner.

    a. Accumulates on hard tissue surfaces, not the soft tissues.
    b. Clean, smooth area presents a more difficult area for deposits.
    c. Remnants of a nidus of calculus remaining after a prophy is quickly covered by more calculus--more so than a highly-polished surface.
    d. Adheres to cervical areas, almost never at the height of contour of a functioning tooth.

2. <u>Mucinous Plaques</u> - soft, mucillaginous, adhering substance which accumulates at the cervical portion of the tooth. Composed of mucin, bacteria and their products, and adhering foodstuff.

3. Materia Alba - soft, white deposit of food debris, purulent materials and mucinous plaque underneath. These deposits usually cause at least a mild gingival inflammation.

4. Bacteria - intact mucous membrane presents a barrier to infection, however, recent studies demonstrate presence of micro-organisms on the surface rather than within the gingival tissues. These bacteria produce toxins which diffuse into adjacent tissues and produce inflammation.

5. Food Impaction - the forceful impaction of food against the gingiva creating pressure on the tissue, or a direct thrust of material against the gingival margin. May be the result of the following:

   a. Faulty contacts and malposition of marginal ridges.
   b. Action of plunger cusps.
   c. Faulty contour of tooth, i.e., missing cingulum or under-developed cingulum.
   d. Anterior overbite: in a deep overbite, the lower incisor may impinge on the palatal gingiva.
   e. Caries resulting in faulty contact.
   f. Improperly contoured restorations--no bulge to protect gingival margin.

6. Food Retention - food does not traumatize the tissue, but is retained in contact with the gingiva because of poor anatomy of tooth; i.e., over-contoured crowns which become inaccessible to the normal physiologic action of the passage of food, such as the mesial margin of a mesially tilted tooth adjacent to a saddle area. This is often seen around a tooth which is not in function.

7. Irritation by Overhangs and Faulty Margins - these irritations are the cause of gingival inflammation and resorption of the alveolar crest.

8. Mouth Breathing - its action is believed to cause dehydration of the gingiva which leads to a loss

of tissue resistance; may also be due to a drying of the entire oral cavity, resulting in a loss of the protective factors of saliva or an upset in the bacterial flora.

9. Faulty Toothbrushing - may cause abrasion or recession of the gingiva and may, in addition, foster an inflammation already present by causing further injury.

   According to some, it may cause abrasion of the tooth surface itself and may cause gingival clefts.

10. Irritations due to Habits - due to faulty use of toothpicks, fingernails, pencils, instruments, etc., which may cause gingival damage. In most instances, the lesion is localized to a definite area and often patients are unaware of these habits.

B. SECONDARY FACTORS:

As a result of the local environmental factors (primary factors) a gingival disease process may be initiated and may progress to a stage where the tooth-gingival relationship changes. Recession may occur and frena, muscle attachments, and deviations of the mucogingival boundary may become important factors. Results of these secondary factors may be:

1. Shallow vestibular sulcus.
2. Narrow band of attached gingiva.
3. Further recession of the gingiva due to pull of the frenum.

C. ALTERATIONS IN FUNCTION:

1. Functional Insufficiency:

   This may be the result of the following:

   a. Infra occlusion.
   b. Loss of antagonists.
   c. Open bite.

2. Non-occlusion will result in the following histological changes in the periodontium:

(1). The P.D.M. becomes thinner.
(2). Loss of most of the principal fibers.
(3). Loss of most of the lamina dura.
(4). Supporting bone is diminished in quantity with fewer trabeculae.
(5). The marrow space is markedly increased in size.

D. OCCLUSAL TRAUMATISM:

The result of repeated traumatic insults to the periodontium, the intensity and frequency of which is beyond the normal adaptive capacity of the supporting tissues.

1. Effect on the Attachment Apparatus:

a. Widening of the periodontal ligament.
b. Resorption of the root surface.
c. Cemental tears.
d. Thrombosis of blood vessels.
e. Hyaline degeneration.
f. Necrosis and hemorrhage of the periodontal ligament.

According to Goldman, the gingival tissues are not affected in occlusal traumatism. Despite the lesions of the attachment apparatus, the gingiva remains intact with no change in the epithelial attachment or gingival fiber apparatus.

Since gingival lesions are so commonly found, lesions both there and in the attachment apparatus coexist. Except for the occasion when an infrabony pocket occurs, the two processes remain distinct. If the marginal (gingival) lesion extends deep enough to reach an area affected by traumatism, (the attachment apparatus at the alveolar crest), then an infrabony pocket may develop.

Clinical diagnostic signs of occlusal traumatism would include wear patterns on the teeth (facets), mobility and migration of the teeth.

2. Causes of Occlusal Traumatism:

a. Cusp interferences, grasping contacts,

uneven wear, faulty restorations, excessive wear, disharmonies in mandibular movements in relation to the static maxilla, and compulsive occlusal habits.

b. Loss of marginal tissue in periodontal disease causes changes in the clinical crown--root ratio, and these are distinct contributing factors in the generation of traumatism.

c. Disharmony in centric closure--sometimes premature contacts are found which will not allow for the simultaneous contact of the teeth in centric closure, and the mandible may have to shift to close completely. These are called slides. The resulting accentric closure often causes trauma. In some cases, the traumatic blow is executed at the end of the slide upon a tooth far removed from the premature contact.

3. Systemic Influcences:

The effects of systemic disease on the periodontium are too numerous to list, but some of the most important are: diabetes, nutritional deficiencies, gastrointestinal disorders (which may be complicated by nutritional changes), and allergies. Drug assimilation may have effects on periodontal tissues. An example of this is Dilantin Sodium which causes a characteristic fibrous hyperplasia. This phase will be treated more completely in a later lecture.

III. DIAGNOSIS:

Chronicity is one of the main characteristics of the inflammatory periodontal lesion. Most of the causative factors are constantly present or repetitious and therefore complete healing cannot occur. This leads to a chronic inflammatory process which persists in spite of any attempt at repair. Therefore, most of the diagnostic signs are indicative of chronicity.

A. Color Change:

Normal color of gingiva--pink to coral. In early gingival inflammation, color assumes a deeper shade. In the long-standing inflammatory conditions, the corium of the gingiva tends to become fibrotic, and there is a return to the pink. Color change may be limited to the gingival margin or it may spread diffusely involving the attached gingiva and the alveolar mucosa.

B. Loss of Stippling:

In gingivitis, the gingival fiber apparatus is destroyed and with the edema and cellular infiltration and swelling, the surface stippling is lost. The gingiva takes on a shiney, edematous appearance. In later stages where fibrosis has occurred, stippling may return.

C. Contour and Position:

Loss of knife-edge margins which become thicker and festooned, gingival recession of gingival hyperplasia.

D. Clefts:

Caused by many of the etiologic factors affecting gingival tissue. May be associated with pocket formation.

E. Changes in the Interproximal Papilla:

This area seems to be more vulnerable--involved more often than the marginal gingiva. Calculus heavier here.

F. Pocket Formation:

Due to the pathologic process in the gingival tissues.

G. Bleeding and Exudation:

Denotes an involvement of the sulcular epithelium. An intact lining will NOT allow for passage of blood and exudate.

H. Bone Involvement:

Changes in the bone may occur in the crestal region or the attachment apparatus. Recognition of where the involvement is, is a very important diagnostic aid. (Occlusal trauma or periodontitis).

I. Mobility:

Generally, the more root surface for periodontal attachment, the firmer the tooth. Mobility is often seen when no pocket formation is present (changes in attachment apparatus).

J. Migration:

For the most part, migration is associated with pocket formation, food impaction, wedging, periodontal traumatism and/or habits.

K. Disturbances in Occlusion:

A faulty centric relation with a positive habit pattern may be the initiating factor in a disturbance causing mobility.

L. Interradicular Involvement:

Bifurcation or trifurcation involvement occurs when a marginal periodontitis advances to such a degree that the interradicular area is denuded of tissue.

M. Periodontal Abscess:

Forms in pockets or between the roots of multi-rooted teeth. There may be a direct fistula to the outer surface of the gingiva, or the abscess may drain directly into the periodontal pocket.

IV. PROGNOSIS:

A. Definition:

The prognosis is the prediction of the duration,

course, and termination of a disease as well as the reasonable expectation of its response to treatment. Prognosis should be determined before the treatment is planned.

B. Prognosis in Patients with Gingival Disease:

When periodontal disease is limited to the gingivae, the determination of prognosis requires consideration of the presence, nature and distribution of local etiologic factors, the nature of the associated gingival response and the possibility of systemic etiology.

If the gingival response is relative to the local etiologic findings, it may be assumed that there is probably no systemic involvement. Elimination of the local causative factors should alleviate the condition. Therefore, provided all local etiologic factors have been recognized and complete cooperation of the patient has been elicited, the prognosis would be favorable.

When the response of the gingiva to local irritants is unusually severe, a systemic complication should be suspected. Prognosis will vary depending on the role of the systemic factor.

1. If the systemic involvement is modifying the gingival reaction to local irritants, then removal of the local irritants will often alleviate the clinical disease. Examples of this situation are pregnancy and leukemia.

2. If the systemic involvement is capable of producing gingival disease in the absence of local irritants and local irritants are merely complicating the clinical picture, then correction of the systemic factors and removal of the local factors are essential for alleviation of the gingival disease. Examples of this situation are chronic desquamative gingivitis and gingival enlargement associated with dilantin therapy.

C. The Prognosis in Patients with Chronic Destructive Periodontal Disease:

1. General Prognosis:

This is based on an assessment of the past behavior of the periodontal tissues and can be used as a guide for anticipating the response to treatment. It is important because it is the basic determinant as to whether or not treatment should be undertaken. Only when a general prognosis is determined should the prognosis of individual teeth be considered.

The basis for determining a general prognosis lies in the evaluation of the response of the alveolar bone to local factors in the oral cavity. Since cessation of bone loss is the ultimate aim of treatment, an understanding of the nature of the response of the alveolar bone is most important in determining the likelihood of attaining the desired result. The degree to which local factors contribute to bone loss will be the determinant as to whether local treatment will be effective in arresting the loss of bone.

2. Considerations in Determining a General Prognosis:

a. Radiographic findings:

The loss of bone as seen in the X-ray may be compared with the average bone loss for a subject with comparable local factors. Height of alveolar bone cannot be, by itself, a determinant of prognosis except where there is extreme bone loss or where there is little bone loss. The significance of comparable radiographic findings varies, depending upon the clinical conditions with which they are associated.

b. Age of the patient:

All other factors being equal, prognosis is better in the older of two patients with a

comparable degree of periodontal disease.

c. Systemic background:

Where there is periodontal disease not explainable by local factors, one may assume that there exists a systemic influence. Detection of these disturbances is often difficult and their exact connection with the periodontal problem is frequently unknown. When the medical problem cannot be found, prognosis is poor. Removal of local factors will, however, retard the destructive process. In patients with potentially destructive systemic disorders such as diabetes, nutritional deficiency, hyperthyroidism, or hyperparathyroidism, correction of this disorder will have a helpful effect upon the periodontium, and the prognosis will be correspondingly improved.

d. Patient attitude:

Lack of cooperation and interest by the patient precludes a good prognosis.

e. Periodontal pocket:

The presence or depth of periodontal pockets cannot be used as sole means in determining prognosis. Obviously the paient with deep pockets and little bone loss should have a better prognosis than the patient with shallow pockets and advanced bone loss. Pocket depth may be the result of gingival hyperplasia or tissue edema and the alveolar bone height may be normal. On the other hand, advanced recession may produce negligible pocket depth and yet may result in alveolar bone height which is inadequate to support the teeth.

f. Severity of Gingival Inflammation:

All other factors being equal, the more severe

the inflammation the better the prognosis will be.

g. Number of remaining teeth:

If there are too few remaining teeth to satisfactorily support a prosthesis, the prognosis will be bad. Prognosis is based partly on the ability to establish a satisfactory functional environment.

h. Malocclusion:

Malalignment of teeth, malformation of the arches, and disturbed jaw relationships are an important etiologic factor in periodontal disease. Alleviation of these conditions by orthodontic or prosthetic means is necessary to improve the prognosis.

i. Tooth morphology:

All other factors being equal, likelihood of a favorable prognosis is diminished when roots are short and tapered and crowns are relatively large and well formed. This gives inadequate root surface available for support by the periodontium and also results in a crown root ratio which improperly distributes functional forces on the periodontal tissues.

3. <u>Determining the Prognosis of Individual Teeth</u>:

a. Mobility:

May be due to loss of alveolar bone, inflammatory changes in the periodontal ligament or trauma from occlusion. When bone loss is the prime cause, the prognosis is poor.

b. Abutment teeth:

Increased functional demands on these teeth, particularly when there is no support distal

to them in removable prosthesis, requires the use of more rigid standards in evaluating the prognosis of such teeth.

c. Bone support:

When the bone level varies in different areas of the root, one should take into consideration the heights of bone on the less involved surfaces before making a prognosis because of the effect this bone has in raising the fulcrum and providing more faborable leverage upon the periodontium.

d. Relation to adjacent teeth:

Teeth with questionable prognoses should be evaluated in the light of how much benefit their extraction would bring to adjacent teeth. Unsuccessful treatment of a tooth with a doubtful prognosis may render irrepairable damage to neighboring teeth.

e. Bifurcation and trifurcation involvement:

These must be taken into account in evaluating prognosis of individual teeth. The presence of furcations does not necessarily indicate a hopeless prognosis. The added support of a multi-rooted tooth is an advantage over single-rooted teeth with comparable bone loss.

f. Caries, apical disease, and tooth resorption:

Determination of prognosis must take into account all other aspects of the teeth.

V. TREATMENT PLANNING:

A. Rationale:

After diagnosis and prognosis have been established, the treatment is planned. This should include a sequential

order of all treatments and although subject to change due to unforeseen occurrences, no treatment should be instituted until the plan is outlined in its entirety.

Our plan should be concerned with dentitions rather than heroic attempts to save individual teeth. Periodontal treatment requires long-range planning if it is to be taken out of the realm of temporary relief measures.

B. The Master Plan for Periodontal Treatment:

The master plan for periodontal treatment is predicated upon the nature of the pathologic procedures responsible for the destruction of the periodontium. It consists of the following four phases:

1. Soft tissue phase:

   Elimination of gingival inflammation and periodontal pockets, and the factcrs which cause them.

2. Functional Phase:

   Establishment of optimal functional relationship for the entire dentition.

3. Systemic Phase:

   Provision for necessary consultation with the patient's physician, and treatment of systemic disorders when indicated.

4. Maintenance Phase:

   This entails the maintenance of a local environment conducive to periodontal health. It includes instruction in oral physiotherapy, recall at regular intervals, yearly follow-up radiographs, and further treatment as indicated.

   In presenting the treatment plan to the patient, it should be pointed out that retention of periodontally involved teeth without necessary treatment may result in the following:

a. Impairment of masticatory efficiency with resultant gastrointestinal disturbances due to the poorly masticated food.

b. Exudation from pockets can spoil the taste of the food as well as cause irritation to the mucosa of the stomach.

c. Change to diets heavy in carbohydrates and starches which have "mushy" texture.

d. Bacteremia due to periodontal infection.

e. Inability to wear prosthetic replacements necessary for adequate oral function.

f. Shortening the span of usefulness of less severely involved teeth.

Treatment should not be forced on a patient who does not have the sincere desire to retain his natural dentition and who will probably not provide the oral physiotherapy required for the maintenance of the dentition after treatment. Success of periodontal therapy, more so than any other dental treatment, is based on the complete cooperation of the patient.

VI. SUMMARY:

The careful analysis of each individual case of periodontal disease is a mandatory prerequisite to successful therapy. Only an uncovering of the prime etiologic factors and their elimination will bring a cessation of the disease process. Without correct determination of the prognoses, countless hours will be wasted by the periodontist and needless expenses will be inflicted on his patients.

Technical procedures constitute the simplest phase of treatment. Only an understanding of specific etiologic factors, a sequential plan for their elimination, and a determination of prognosis for the entire case and for individual teeth comprise complete case analysis and total treatment.

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LECTURE NO. 8

THE RATIONALE FOR PERIODONTAL TREATMENT

Samuel Kobrinsky*

I. INTRODUCTION:

Periodontal treatment is aimed at the elimination of disease, the restoration of the periodontal tissues to a healthy state, and the maintenance of the health of the periodontal tissues. Clinically, we would like to see cessation of bone loss, decrease in tooth mobility, the elimination of pockets, the cessation of gingival bleeding, where possible, the regeneration of destroyed tissue, and the prevention of the recurrence of the disease.

Although a multiplicity of factors, both local and systemic, may contribute to periodontal disease, our therapy is directed mainly at the local factors. These may be easily observed and corrected whereas there are yet many gaps in our knowledge regarding the influence of systemic factors. Whenever indicated, systemic therapy is used as an adjunctive measure.

II. PRINCIPLES OF HEALING:

The Clinical results of treatment are an expression of microscopic changes occurring in the tissues and the understanding and interpretation of these changes is essential for intelligent patient management.

A. REPAIR AND INFLAMMATION

There is evidence of direct relation between inflammation and the initiation of repair. Experiments have shown that wounds protected by a dressing have shown no sign of repair for several weeks; when a mild irritant was introduced, there was prompt contraction and granulation.

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B. FACTORS WHICH INFLUENCE HEALING (AREY)

1. Infection--retards healing when present either at site of wound or in remote areas of the body.

2. Foreign bodies--retard healing due to inflammation induced by their presence and also due to their contamination.

3. Mechanical pressure--aids healing by

   a. Eliminating dead space.
   b. Controlling oozing.
   c. Limiting stasis.
   d. Limiting amount of plastic substance entering the wound.

4. Temperature--Increase hastens healing.

5. Chemical irritation--slight topical irritation with a substance such as turpentine shown to hasten healing in experimental animals.

6. Age--healing ability decreases with increasing age.

7. Disease--a detriment to general metabolism retards healing.

8. Nutrition:

   a. Protein diet stimulates healing.
   b. Fat diet inhibits healing.
   c. General avitaminosis retards healing.
   d. Vitamin A deficiency delays healing; administration of moderate doses aids healing; small doses have no effect; large overdoses retard healing.
   e. Vitamin C deficiency retards healing.
   f. Vitamin D overdose shown to retard healing in experimental animals and to cause calcification in the arterioles of the deep granulation tissue.

9. Hormones. Systemically:

   a. Male hormone accelerates healing in both

males and females.
b. Estrogen hormone hastens healing in the female, has no effect in the male.
c. Thyroid extract aids healing.
d. ACTH or Cortisone retards granulation tissue formation and wound healing.
e. Severe stress inhibits healing.

C. HEALING MECHANISM

1. Epithelial Regeneration:

Epithelium is capable of swift and extensive regeneration. Unlike connective tissue whose fibroblast derivation is in dispute, it is generally agreed that epithelium arises from existing epithelium.

a. There is a tenfold increase in mitotic figures at the wound edge which represents an increase in normal mitotic figures rather than a qualitative alteration in cell metabolism.
b. Mitotic metabolism has been shown to be aerobic.
c. Migration begins within a few hours and proceeds at approximately .5 mm per day.
d. Mitotic figures are seen primarily in a 1 mm. zone at the wound edge.
e. Epithelium migrates rapidly toward the depth of the wound until it reaches a point of tissue continuity and proceeds beneath the clot.

2. Connective Tissue Regeneration

a. Cells:

1). The dominant cell in the healing area after the cellular exudation of inflammation subsides is the fibroblast. The origin of proliferating fibroblasts is widely disputed. Two main theories cite its origin from

A blood element.
Perivascular tissue (Immature fibroblasts present)

There are three stages of fibroblast activity in healing:

First Stage: Proliferation.
Second Stage: Collagen fiber formation.
Third Stage: Scar formation.

2). The next most dominant cell seen is the endothelial cell. There is rapid capillary proliferation from the peripheral inflamed vessels.

b. Ground Substances--synthesized by the fibroblast; serves an important function in interfibrillary cementing of fibrous connective tissue.

D. HEALING IN THE ERADICATION OF THE PERIODONTAL POCKET

It bears emphasis that healing is taking place even during active periodontal disease. Our treatment is pointed at creating an environment conducive for the progress of this inherent healing process. The events of healing may be grouped as follows:

1. Removal of noxious substances:

   a. Scaling and root planning to remove irritants, mechanical, chemical, and bacteriological, which provoke inflammation.

   b. Curettage--removes degenerated and necrotic debris. Use of dull instruments causes some of this debris to be left behind as tabs, etc., and these must be eliminated by enzymes or phagacytotic action before healing can take place. Another purpose of curettage is to eliminate the epithelial barrier to reattachment.

2. Recovery of "sick" cells.

   Cells in which degeneration has not progressed beyond the reversible state undergo recovery.

3. Regeneration--growth and differentiation of new cells and intercellular substance to form functional

units (Glickman).

E. GRANULATION TISSUE IN HEALING

The granulation tissue in the pocket wall is the same type of tissue that replaces the clot after healing. It differs only in that the pocket wall granulation tissue does not mature due to the presence of the irritants which caused the pocket. Removal of these irritants will allow it to mature.

III. CLINICAL AND LABORATORY STUDIES OF HEALING FOLLOWING PERIODONTAL TREATMENT:

A. SCALING AND CURETTAGE

Box - Clinical and microscopic studies of periodontal tissue showed that healing was complete in nine days or less.

Blass and Lite - Histopathologic examination following subgingival curettage showed that healing was complete in ten days.
Goldman - In a histopathologic study with 15 comparative areas found that healing following ultrasonic curettage is more rapid than with hand instruments.
Glickman - In two weeks following treatment by scaling and curettage, with proper oral physiotherapy by the patient, the gingiva will be slightly reduced in height and will have normal color, consistency, surface texture, and contour. There will be no notable gingival bleeding.

B. GINGIVECTOMY

Bernier and Kaplan - found that following gingivectomy and covering with a pack consisting of zinc oxide, eugenol, and oil of bitter almond, epithelization was complete in six days and the tissue appeared clinically normal in thirty days.

C. MUCOGINGIVAL SURGERY

Grant and Ivancie - Alveolar mucosa with its elastic fibers were replaced with collagenous connective tissue in eight months.
Wilderman - found that bone denuded in mucogingival

surgery suffered complete resorption. Interproximally there was total regeneration, but facially only fifty per cent regeneration. There was an increase in fibrous connective tissue attachment which partially compensated for the lost bone. This constituted functional repair with an anatomic deformity.
Staffileno, Wentz, Orban - found that when a split thickness flap was used in mucogingival surgery, there was only a small amount of bone resorption which completely regenerated during healing. There was a functional repair with no anatomic deformity.
Bohannan - found that clinical healing was completed in 26 weeks following complete denudation of bone in mucogingival surgery.
Corn - reported clinical healing complete in three months following mucogingival surgery in which a split flap technique was used.

D. REATTACHMENT

1. Definition: The attachment of gingival epithelium connective tissue to tooth surfaces denuded by periodontal disease. Thus, reattachment is a re-establishment of the epithelial attachment and of Sharpey's fibers to an area of cementum which was adjacent to a pathologic pocket.

2. Reattachment Studies:

Reattachment has been described frequently in the literature in animal studies where artificial periodontal lesions have been produced. Skillen and Lundquist stated that they found evidence of slight reattachment on dogs' teeth, but found no evidence of reattachment in human material.
Beube reported healing and reattachment of periodontal ligaments in artificially established pockets around the roots of dogs' teeth. He also reported similar findings in human material from several cases of periodontosis. Linghorne and O'Connell reported successful repair of surgically produced lesions in the periodontium of dogs. They found new cementum and reattached periodontal connective tissue on injured root surfaces.
Ramfjord reported successful experimental reattachment of connective tissue and epithelium to root surfaces of monkeys' teeth which had been previously

exposed to inflammatory periodontal disease. He found reattachment only where inflammation was mild and no reattachment in areas of severe inflammation.

Sicher states that reattachment is a possibility, even probability, because epithelium displays the ability to attach itself to tooth surfaces where an epithelial attachment had never before existed (as seen in recession). He also noted that epithelium will even attach itself to inert substances such as implants.

Experimental evidence proving reattachment to be a possibility in human subjects with periodontal disease is scanty. However, most clinicians with opinions based on clinical observations agree that reattachment can occur. Glickman states that following a thorough scaling and curettage, there is some degree of reattachment above the area of previous attachment.

3. Method for obtaining reattachment

   a. Removal of calculus and necrotic cementum by scaling and root planning.
   b. Removal of immature granulation tissue and ulcerated crevicular epithelium by curretage.
   c. Fixation of mobile teeth to promote healing.

4. Factors which may deter reattachment

   a. Epithelial proliferation may occur prior to fiber and cementum formation and thus prevent reattachment.
   b. Cementum may have an accumulation of bacteria and bacterial products in defects caused by degenerating remnants of Sharpey's fibers.
   c. Age--with the physiologic tendency toward recession there would be less likelihood that reattachment could occur in an older patient to the degree you might expect in the younger patient.
   d. Systemic disease--if the major etiologic factor of periodontal disease is a systemic disorder, reattachment would probably not occur by treatment of only local factors.

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LECTURE NO. 9

SCALING AND SUBGINGIVAL CURETTAGE

Wilfred B. Bell*

I. SCALING

For many years the practice of "apoxesis" or root surface treatment was considered by many learned periodontists to be the most definitive clinical procedure in the treatment of "periodontoclasia." The other two legs of the tripod supporting "conservative" periodontal treatment were nutritional considerations and adequate maintenance therapy. The fact that the present day treatment of periodontal disease compliments to a high degree this basic rational, attests to the validity of this early philosophy.

Scaling is basically the removal of both the hard and soft accretions from the coronal and radicular surfaces of the tooth. Instrumentation to accomplish this procedure must be done in an exacting and skillful manner. To achieve definitive results, particularly in the relatively inaccessible subgingival areas, requires more than a little tactile skill and practice.

The surfaces of the tooth not visible to the eye must be carefully explored to seek out and carefully remove all of the calculus, followed by a complete planning of the tooth surface to remove any irregularities including fragments of partially detached cementum.

The anatomy of the teeth should be constantly borne in mind during the scaling procedure. Many teeth present concave root surfaces near the cemento-enamel junction and if fastidious exploration and instrumentation is not accomplished in these areas, of course, treatment will be ineffectual.

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There are few, if any, instances in the treatment of periodontal disease when scaling is not a necessary prerequisite to other therapeutic measures. In many cases, scaling itself is sufficient to effect definitive soft tissue changes in the gingiva which result in shrinkage and resolution of the pocket. This is more often achieved when the pocket is shallow, edematous and hyperemic (see subgingival curettage).

The instruments used in scaling are numerous, but they all generally may be classified as chisels, hoes, curettes, or sickles as determined by the shape of their cutting blade. Each type presents certain variations which necessitates altering the stroke used in manipulation in order to obtain maximum effectiveness. Alterations in the angle of the shank of the instruments are made to obtain increased accessibility in specific areas of the mouth.

Recently the use of ultrasonics in scaling has been investigated and advocated by many clinicians. This has brought forth a cabinet-type instrument with various hand tips capable of emitting vibrations up to 29,000 per second. Clinical experience has demonstrated that this instrument is highly valuable for the rapid removal of heavy accumulations of hard calculus, particularly the supragingival type. If properly used, it is atraumatic to the gingival tissues and has the added benefit of a constant lavage which aids in the rapid removal of detached calculus and soft debris from the operated area. The use of this new principal of ultrasonics represents an important contribution to the field of periodontal therapy. However, it should be realized that it only compliments and not supplants our existing armamentarium.

II. SUBGINGIVAL CURETTAGE

The scaling procedure as previously discussed emphasized the complete removal of extraneous substances from the tooth side of the gingival sulcus. We must realize that even though the emphasis is placed on only this "hard wall" of the sulcus, that inadvertently there will be some removal of the soft tissue wall due to the mechanical manipulation of the instrument in the subgingival area. This may not be deliberate, but nevertheless it occurs and probably with some therapeutic effect, particularly if the gingival inflammatory lesion is in an early stage of development.

Now, in the discussion of subgingival curettage we will confine our remarks specifically to the soft tissue side of the sulcus (or pocket) even though we realize that the term in its broadest definition includes similar treatment of the tooth surface as well. In other words, pure scaling by itself without deliberate curettage of the soft tissue lining of the pocket may be effective therapy in some cases; however, pure soft tissue subgingival curettage in itself will not be effective unless the adjacent tooth structure is also treated.

Curettage is indicated for the treatment of gingival pockets which are amenable to shrinkage; that is, when the increased gingival bulk is due to an increased number of inflammatory elements. It is a surgical procedure for the removal of the epithelium, epithelial attachment, and a portion of the adjacent connective tissue of the hyperplastic gingival pocket. Removal of this soft tissue pathology with sharp curettes will result in a fresh bleeding connective-tissue surface capable of regeneration.

Whether or not subgingival curettage will be effective in the remission of the pocket depends on the character of the soft tissue lining the pocket. In a low-grade, chronic inflammation as is commonly found in periodontal pockets, there is a tendency for fibrosis to occur if the process has been in operation for a considerable length of time. This is merely an attempt at healing on the part of the involved tissue, and is a physiologic attempt at repair of the injury. This may be masked in the generalized enlargement of the gingival tissues due to edema and attendant phenomena. It is only when these factors are resolved that the residual fibrosis or scarring becomes

manifest. This fibrosis does not disappear when the irritant is removed, but must be corrected by surgery. Also, the breakdown in the gingival lining in the pocket with attendant ulceration and necrosis of the epithelium and inflammatory response in the connective tissue, if allowed to remain long enough, sets up dynamics of its own which are not resolved with only removal of the subgingival irritants.

III. SUGGESTIONS FOR INSTRUMENTATION IN SCALING AND SUBGINGIVAL CURETTAGE:

1. Instruments must be sharp.
2. Use direct vision whenever possible.
3. Use warm compressed air frequently to improve vision.
4. Irrigate working area frequently to remove loose calculus and other debris.
5. Insure positive control of the instrument by employing firm finger rests.
6. Employ proper strokes when the instrument is activated to insure the correct position of the blade to the operative site.
7. A topical or local anesthetic should be used as needed.
8. Extreme care should be taken to make the procedure as atraumatic as possible.
9. The procedure should be done systematically in a predesignated area without unecessary repeated manipulation to a site already treated.
10. Reevaluation of the case should be accomplished periodically to assess the effectiveness of the treatment.

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LECTURE NO. 10

ACUTE NECROTIZING ULCERATIVE GINGIVITIS

Wilfred B. Bell*

I. INTRODUCTION:

Historically this disease may be traced back to the Greek Armies around 400 B.C. In the 19th century, Napoleon's army suffered from "sore mouth" in epidemic proportions. A century later in World War I, "trenchmouth" was a familiar disease among the troops of the A.E.F., as it was still later in World War II, and the Korean conflict. Dentists assigned to our large service basic training areas today recognize ANUG as a rather common disease entity.

II. CLINICAL SIGNS AND SYMPTOMS:

The marginal gingivae present punched out, eroded, crater-like depressions. The superficial border of these depressions is formed by a gray, pseudo-membranous slough which is demarcated from the remainder of the gingival mucosa by a pronounced linear erythema. The characteristic lesion is the crater-like interproximal depression covered with a gray surface slough. Fetid odor, increased salivation and spontaneous gingival hemorrhage are present in varying degrees in most cases. Acute pain is the most pronounced subjective symptom. Extra oral symptoms may include regional lymphadenopathy, elevation of temperature and general malaise.

III. ETIOLOGY:

Local factors such as calculus, poor oral hygiene, overhanging margins of restorations, ill-fitting prosthetic devices and traumatic occlusion have been reported as predisposing agents.

Even since Plaut and Vincent in the 1890's described the oral lesion and attributed its origin to the fusiform

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and spirochete organisms, the role of these bacteria has been studied to determine whether they are actually the primary invaders or secondary to invasion by other organisms not excluding viruses.

Box lists certain areas in the oral cavity such as gingival flaps over partially erupted third molars and the tonsils as "primary incubation zones." He believes the disease spreads from these zones to other areas of lowered tissue resistance, the "secondary incubation zones," and from there to other areas in the mouth.

Various systemic factors have been reported in the literature which may, in some degree, exert a predisposing influence on the gingiva rendering it more vulnerable to the disease. Such conditions as nutritional deficiency, gastrointestinal disturbances, alcoholism, blood dyscrasias, and even the common cold have all been cited as possible complicating secondary factors.

Recently, Moulton and others have reported the high prevelance of emotional illness in patients with ANUG. Schluger has stressed the importance of fatigue and nervousness in precipitating the onset of the disease while others believe a "stress factor" may be an important inciting agent.

Communicability of the disease has not been established although it has been observed to occur in groups living under the same environmental circumstances.

IV. TREATMENT:

A. First Visit:

1. Apply topical anesthetic.
2. Remove supragingival calculus and gross subgingival calculus.
3. Irrigate copiously with warm water and/or diluted 3% hydrogen peroxide.
4. Polish teeth with rubber cup and fine pumice.
5. Instruct patient to rinse the mouth frequently with warm water.
6. Placement of a periodontal dressing may be indicated in some cases.

B. Second Visit: 24 - 48 hours

1. Same as first visit.

2. Instruct patient in proper oral hygiene procedures using a soft, multituffed brush.

C. Third Visit: 48 - 72 hours.

1. Check oral hygiene effectiveness with disclosing tablets.
2. Complete subgingival scaling, if not previously accomplished.

D. Fourth Visit: 10 days to 2 weeks.

1. Check oral hygiene effectiveness with disclosing tablets.
2. Accomplish surgical procedures as necessary to remove interproximal craters.

Subsequent visits should be planned following the completion of the surgical phase to further check patient's oral hygiene effectiveness.

"Shotgun" treatment using penicillin is hardly justified when one considers the serious side effects possible with its use. In the rare cases when antibiotics might be indicated, one of the tetracyclines may be used with effectiveness and relative safety.

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LECTURE NO.11

GINGIVECTOMY (GINGIVECTOPLASTY)

Wilfred B. Bell*

I. INDICATIONS AND RATIONALE FOR TREATMENT

Basically the gingivectomy is a procedure for the surgical resection of the diseased soft tissue wall of supra bony periodontal pockets.

The pockets may be "relative" or "absolute" but in both cases the soft tissue wall is of fibrous texture with relatively little inflammation as observed clinically; in other words, we would not expect therapy such as subgingival curettage directed towards shrinkage of the tissues to be successful. Thus, surgical resection is the treatment of choice.

A secondary benefit to be derived from the gingivectomy is the exposure of surfaces of the tooth inaccessible to the eye and relatively inaccessible to tactile instrumentation by the operator. The importance of complete removal of subgingival calcarious deposits cannot be over emphasized; however, the gingivectomy should not be thought of as a procedure solely designed to facilitate scaling for the operator.

II. CLINICAL OPERATIVE PROCEDURE

A. Planning

The preoperative diagnosis and treatment plan should have been previously accomplished along with a complete and thorough scaling of the operative area. Extent of the area to be surgerized may vary according to individual circumstances, but generally the case is operated by quadrants.

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B. Anesthesia

Conduction anesthesia may be used on the lower arch particularly when the right and left halves are to be operated individually. The facial aspects of the upper arch are adequately anesthestized with local infiltration into the muco buccal fold placing the needle in a horizontal position and injecting the anesthesia while the needle is being passed mesio-distally. After the facial aspect is anesthesized, the needle is passed through the facial interproximal papillae and the anesthetic solution deposited into the lingual papillae mesial and distal to each tooth. Some areas such as the disto lingual tissues of the last remaining molar may demand a direct lingual injection, but for the most part the above should supply sufficient anesthesia, thus obviating the necessity of the painful palatine injections. The added benefit of local infiltration is, of course, the hemostasis acquired through the epinephrine component of the local anesthetic solution. It should be noted that a minimum anesthesia is all that is necessary for soft tissue work.

C. Determining the Pocket Depth

Delineation of the pocket depth is accomplished with pocket markers or some other suitable means, whereby a series of bleeding points is created on the attached gingivae of the operative site. The more puncture points placed, the better outline of the pocket morphology we have, but the minimum would be one point on the facial and lingual gingival aspects of each tooth.

D. Surgical Excision

A narrow, pointed, and sharp knife such as the Orban #1 or #2 is used first to make a beveled incision disto-mesially to the depth of the pocket distal to the last tooth. This is carried mesially as far as possible on the facial and lingual gingival aspects of the tooth. Then a palate-shaped knife such as the Kirkland #15 or #16, or Goldman-Fox #17 is used to extend the incisions to their mesial terminal points. The incisions are made at approximately a 45-degree angle at a point approximately 2-3 mm. apical to the previously-placed puncture points and carried to the facial and lingual aspects of the teeth. This creates the desired bevel with positive eradication of the pocket. The sharp, pointed knife is then utilized

in the same fashion to effect the interproximal incisions, after which the surgerized ribbon of tissue is detached and removed from the area.

E. Scaling and Curetting

All surfaces of the teeth are examined for the presence of calcarious deposits which may have inadvertently been missed during the scaling procedure. All such deposits are carefully removed with suitable scalers. Soft tissue tags and granulomatous tissue are removed carefully and thoroughly with sharp curettes and the area irrigated to remove the debris.

F. Soft Tissue Contouring

Notwithstanding the fact that the initial incision in most cases produces the desired bevel, there may be some cases which need further embellishment to attain the desired physiologic contour of the gingiva. While it may be true that "natural" contouring may be expected to occur within several months postoperatively, it seems desirable to build it into the initial surgical procedure, thus reducing the sharp shelf-like margins to obviate food accumulation and to faciliate oral hygiene measures in the early healing stages. Special soft tissue high speed rotating diamond stones have been suggested for this purpose of gingivoplasty, or the gingivectomy knife manipulated in a scrapping motion may be used effectively in achieving the desired results. Whatever the means utilized, the procedure should be carried out as atraumatically as possible and the end result should closely resemble the natural physiologic contours of normal, healthy tissue.

G. Placement of the Surgical Dressing

Various types of periodontal packs have been suggested and until recently they were generally of the zinc oxide and eugenol type with individual additions such as asbestos fibers, tannic acid powder, and the like, to enhance their effectiveness in one way or another. Recently a zinc oxide-dehydrogenated fat (Crisco) mixture with zinc bacitracin has been developed and used with excellent clinical results. Since this dressing does not contain eugenol, which is somewhat irritating to the tissues, it has a wider application of use. The

anti-microbial effect of the antibiotic seems to prevent the rather unpleasant odor usually found under a pack and clinical experience so far seems to indicate faster healing and less postoperative patient discomfort. (The formulae for both types of packs are at the end of this section).

After the pack has reached the proper putty-like consistency, it is fashioned in a small roll of sufficient length to cover the surgerized area and then placed on the facial and lingual operated areas working from the posterior to the anterior terminus. With finger pressure and instruments, the two segments are joined interproximally. Some prefer to place small wedges of dressing into the interproximal areas at the outset and then join them to a facial and lingual strip in order to obtain positive retention.

The packs should protect the wound surface from traumatic effects of mastication, result in less postoperative pain to the patient, aid in rapid hemostasis, and facilitate healing. Care should be taken to see that the pack does not create problems. It should be "muscle trimmed" while still soft to insure that normal movements of the tongue and cheek are not interfered with. The occlusion should be checked in the various excursions to avoid impingement and possible loss later due to fractures. The pack and the exposed tooth surfaces may be covered with adhesive foil to help prevent dislodgement before adequate "setting" has occurred.

After one week, the dressing is removed and the wound irrigated with warm water and "debrided." The teeth are checked once again for calcarious deposits and the wound surface checked for redundant overgrowth of granulation tissue. In both cases the extraneous material is removed.

In most cases, repacking for another week is indicated, but this depends on the case and is done at the discretion of the operator. (Postoperative instructions to the patient will be found at the end of this section).

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LECTURE NO. 12

OSSEOUS SURGERY

Dominick P. Mandracchia*

I. THE PERIODONTIUM - NORMAL

A. The Gingival Unit

1. Consists of free gingiva (keratinized), gingival margin, sulcular epithelium, epithelial attachment, and the underlying lamina propria (connective tissue) which consists of fibroblasts, vascular elements, lymphocytes and plasma cells especially subjacent to the sulcular epithelium and collagen fibers of the gingival unit which can be subdivided into:

   a. Group A, Group B, and Group C gingival fibers.
   b. Transseptal fibers.
   c. Circular fibers and alveolar crestal group.

As long as these fibers are attached to the cementum, the free gingiva is well attached to the tooth and not retractable.

B. The Attachment Apparatus

Separated from the gingival unit by the overlying transseptal fibers.

1. Consists of the periodontal membrane with its principal fibers and interstitial tissue; the alveolar bone proper (lamina dura and trabecular supporting bone) and cementum.

II. GINGIVITIS

A pathologic change which occurs in the gingival unit

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and not in the attachment apparatus.

A. Gingival Pocket

1. Pocket - space bordered by tooth, ulcerated sulcular epithelium and the coronal portion of epithelial attachment. Depth is not a necessary criteria in differentiation of a pocket from a healthy gingival sulcus.

2. In a gingival pocket, the increase in depth is due to an increase in size of the gingival tissue, not to the apical migration of the epithelial attachment (also called a pseudo pocket).

3. Subjacent to the ulcerated sulcular epithelium the inflammatory process causes edema, swelling, reddness, increase in cellular elements, more polys into area, increased vascularity, lysis of the Group A collagen fibers and resultant loss of stippling and rectractability of the free gingiva.

III. PERIODONTITIS

A progression of the pathologic process beyond the gingival unit and into the underlying attachment apparatus.

A. Periodontal Pocket

1. With the progression of the inflammatory process, Groups B and C gingival fibers are destroyed and with the removal of this barrier and also the destruction of the transseptal fibers, the epithelial attachment can migrate apically, the coronal portion of the epithelial attachment detaches from the enamel and we have the beginning of a periodontal pocket.

2. The inflammatory process generally spreads along the outside of the alveolar process and also through the alveolar crestal bone into the marrow spaces.

   a. Round cells are observed in the marrow spaces.

b. Fatty marrow is replaced by fibrous marrow.
c. Crest of alveolar process may show horizontal cup-like resorption.
d. Resorption occurs from the marrow spaces to the periodontal ligament (rear resorption), widening and eventually destroying the vascular channels and then the lamina dura to which the Sharpey's fibers are attached.

3. Suprabony Pocket--associated with horizontal bone resorption where the base of the pocket is coronal to the bone.

4. Infrabony Pocket

a. Associated with a vertical (or combination of both) pattern of bone resorption, where the base of the pocket is apical to the crest of bone.

5. Transseptal fibers--seem to be important in their attempt to wall off the inflammatory process. In an attempt at repair, these fibers reform at a lower level than originally.

IV. CLASSIFICATION OF IBP'S

A. Three Osseous Walls

1. Proximal, buccal, and lingual walls (interproximal IBP).
2. Buccal, mesial, and distal walls.
3. Lingual, mesial, and distal walls.

B. Two Osseous Walls

1. Buccal and lingual (crater).
2. Buccal and proximal.
3. Lingual and proximal.

C. One Osseous Wall

1. Proximal
2. Buccal
3. Lingual

D. Combinations

1. 3 walls and 2 walls.
2. 3 walls and 2 walls and 1 wall.
3. 3 walls and 1 wall.
4. 2 walls and 1 wall.

Combinations are found because it is common to find IBP's where the apical part has more walls than the coronal part.

E. Four Osseous Walls

Buccal, lingual, mesial and distal.

V. CLINICAL EVALUATION OF IBP'S

The choice between a reattachment attempt with its attractive rewards and an osteoplastic reshaping of the bony defect can only be made on the basis of careful clinical evaluation of the IBP.

A. Reattachment

Reattachment procedures attempt to induce a new growth of bone and periodontium to fill in the infrabony defect.

B. Osteoplasty

1. Osteoplasty procedures attempt to remove enough of the crestal bone to eliminate the defect by creating a new level consistent with the general attachment level.

2. Osteoplasty is often combined with osteoectomy in the treatment of IBP's in order to achieve a proper physiologic contour of the bone (as opposed to reverse architecture).

3. Osteoectomy is the removal of firmly attached bone with the resultant loss of some of the support around the tooth in order to achieve the correct physiologic form in the area.

4. The Osteoplasty - Osteoectomy method is more certain and predictable than the reattachment

method, but it sometimes is too destructive to the adjacent periodontium to be useful.

C. Qualifying Factors

1. Number and location of osseous walls.
2. Root anatomy--length and shape.
3. Width of interproximal alveolar bone.
4. Depth of pocket--deep and narrow versus shallow and wide.
5. Mobility of teeth.
6. Occlusal forces.
7. Remaining alveolar bone support.
8. Contact relationships.

D. Topography of IBP's

1. Three-walled IBP's--these are trough-like defects usually seen in the interdental areas. Also occasionally seen on lingual surfaces of maxillary and mandibular teeth, less often in other areas.

2. Two-walled IBP's--seen in the interdental areas as osseous craters.

3. One-walled IBP's--usually in the interdental area, where the proximal wall is the one remaining. Often seen in mandibular molar region, mesial aspect of a molar adjacent to an edentulous area.

E. Clinical Examination to Determine Topography

1. Radiographic examination of the IBP discloses a vertical resorptive lesion, but gives no information concerning base of pocket. A pocket cannot be recognized by X-ray alone, because a vertical lesion may be present, but the gingival tissue may be intact with no pocket formation.

   Radiopaque materials can be used; also, Hirschfeld points, gutta percha points, and bismuth solutions impregnated with cotton.

2. Probing of Pocket

Periodontal probe, explorer, 25-gauge needle, etc.

Radiographic examination will not reveal the number of walls in the particular IBP. The topography of the bony portion of the pockets is more important than the soft tissue part, and this can be determined only by accurate probing of the base of the pocket and also the bony crests.

VI. ETIOLOGY

A. Superimposed on the diseased gingival unit, certain factors may induce the formation of IBPs.

1. Uneven levels of adjacent marginal ridges.

2. Some degree of tilting of teeth.

3. Root anatomy--the indentation of the mesial aspect of the roots combined with bucco-lingual movement of tooth in trauma.

4. Distance between roots--narrow interproximal area disposes to horizontal resorption and supra bony pockets.

5. Food impaction areas, poor margins on restorations, etc.

6. The occlusal traumatic lesion--(1) cannot per se cause pocket formation, but is so often found in association with the soft tissue lesion or periodontitis. Cohen calls this Occlusal Periodontitis (occlusal traumatism and periodontitis) this is a lesion of the gingival unit and the attachment apparatus. (2) Occlusal periodontitis leads to:

   a. Widened P.D.M. space.
   b. Resorption of alveolar bone and tooth, cemental tears, tooth mobility, drifting, wear facets.
   c. Diseased gingival attachment.
   d. Infra bony pockets.
   e. Can also cause supra bony pockets.

VII. RATIONAL OF THERAPY

The most favorable type for reattachment is the 3-walled IBP. With 2 walls a slanting fill-in of bone may be obtained; whereas with one-wall, no additional attachment is to be expected and osseous surgery is indicated.

VIII. CORRECTION OF ETIOLOGIC FACTORS

A. Correction of factors responsible for initiation of pocket:

1. Reshaping to correct food impaction, wear facets, marginal ridges.

2. New restorations with good margins, etc., close contact points, restore lost anatomy; ie., lingual of maxillary central incisor.

3. Correct occlusal trauma due to facets, plunger cusps, bruxism, and other habits; ie., tongue, pipes, pencils.

4. Scaling of tooth surfaces to remove all deposits prior to operative procedure. Better postoperative healing when gingival inflammation has been resorbed.

5. Immobilization of mobile teeth--temporary splints.

IX. THERAPY

A. Reattachment--for the 3-walled IBP's.

1. Curettage--gingivectomy procedure.

   a. Gingivectomy to level of bone crest.
   b. Removal of contents of pocket with curette--prescaling of root surface prior to surgical procedure advocated by many. Remove entire contents including transseptal fibers and epithelial attachment. Transseptal fibers run from tooth to tooth and would prevent cementum to bone reattachment if left.
   c. Formation of clot, covered with tin foil and perio pack--or place pack directly

into bony trough as advocated by some. Clot only acts as a surgical dressing and is not really organized by proliferating fibroblasts. Perio pack (according to some advocates) also serves as a surgical dressing.

d. Weekly pack changes for about three weeks.

2. Flap approach

a. Where access is inadequate, a flap may be made.
b. Disadvantage--more tissue injury and possible faulty gingival form after healing.
c. Debridement of inner wall of gingival flap.
d. Flap is replaced and vertical incisions are sutured. Suture thru interproximal papilla to hold tissue close to tooth surface.
e. Modified flap--can be used instead of making vertical incisions mesial and distal to operative site.

B. Therapy for Two- and One-Walled IBP's

1. Osteoectomy--osteoplasty to eliminate lesion.

That procedure which trims the bone crest so that the base of the resultant sulcus will be occlusal to the new bone crest is termed osteoectomy. When recontouring is necessary, the term osteoplasty is used.

a. Contra-indications:

1). The excessive weakening of the support of an adjacent tooth.
2). Creation of a gingival form not conducive to self-cleansing (improper physiological form).
3). Difficulty in maintaining cleanliness by oral hygiene.

b. Indications:

1). Broad, shallow defect.
2). Not too much loss of bone support.

3). When resulting architecture is acceptable.

c. Procedure

1). Flap or gingivectomy plus flap.

Followed by trimming of the bone crest with small chisels or by a bur in dental engine. Bone files can be used. Diamond stones to contour the crest. Debris is flushed with warm saline. Suture flap carefully.

2). Two-walled craters

Can be ramped to the buccal or lingual. Allow lingual crest to remain and ramp to the buccal or leave buccal crest and ramp to the lingual or palatal. This helps prevent esthetic deformity and yet provides a cleanseable interdental area.

C. Therapy for Combination IBP's

Frequently three-walls observed in apical part of pocket with two and/or one wall at coronal part.

The one- and two-walled part eliminated by osseous surgery and the three-walled part is prepared and treated for reattachment.

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LECTURE NO. 13

MUCOGINGIVAL SURGERY

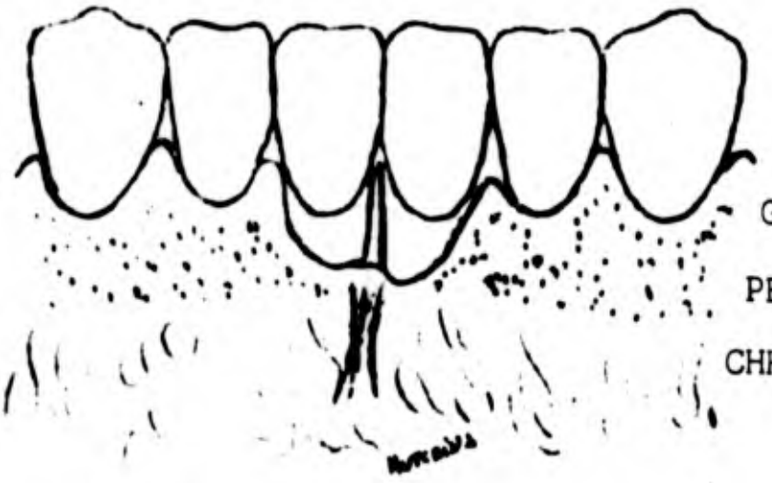
Dale Hutchins*

I. INDICATIONS:

A. INSUFFICIENT ATTACHED GINGIVA

The band of attached gingiva may become insufficient or non-existent due to one of several causes. As gingival recession occurs, regardless of the cause, the width of the band of attached gingiva narrows. It may eventually migrate to the area of the frenum attachment. In this event, tissue tension will constantly be applied to the gingival margin, and an otherwise physiologic gingival crevice will become pathologic.

FRONTAL VIEW OF LOWER ANTERIOR REGION



FRENUM AND MUCOSA CONTINUOUS WITH GINGIVAL MARGIN

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If a periodontal pocket or cleft terminates at/or coronal to the mucogingival line, curettage will be ineffective since the entire pocket wall is attached to a mobile tissue. A surgical procedure designed for pocket elimination would leave a surgical margin of mucosa which will heal slowly and soon retract and permit food retention with further complication.

B. SHALLOW VESTIBULAR FORNIX

In some patients, the vestibular fornix, or trough, is normally shallow. This may be due to high, broad frenum and muscle attachments providing little room during eating and oral hygiene procedures. This situation endangers periodontal health in mouths in which the base of the gingival sulcus is approaching the mucogingival line.

II. REVIEW OF THE LITERATURE

Mucogingival surgery is a relatively new concept in dentistry. Sufficient time has not elapsed for adequate study of recent techniques, theories, and clinical observations. As late as 1952, Schluger observed those cases which involve the alveolar mucosa due either to the extreme depth of the pocket or the coronal position of the mucogingival junction were poor surgical candidates. He admitted that objectives and goals must be lowered in these instances. Goldman, in 1953, stated that periodontal incisions must be limited to attached gingiva and that when alveolar mucosa was involved, the tissue curled and became undermined.

Extension of the mucobuccal fold, or increasing the vestibular depth must first be credited to Kazanjian. In 1924, he described a method of deepening the vestibule, increasing the ridge height in edentulous patients. His technique involved a rather complicated incision through the buccal mucosa, sharp dissection under the mucosa back to the ridge, sharp dissection apically to the desired vestibular depth and fixation of the tissues by extra-oral sutures. This technique was basically unchanged when he again described it in 1935 and 1940.

Cooley described "A Method for Deepening the Mandibular and Maxillary Sulci to Correct Deficient Edentulous Ridges." He applied the following basic fundamentals of plastic

surgery to plastic modification of the periodontium:

A. The raw surfaces created on soft tissue will contract steadily and heal with a dense mass of scar, but if the raw surfaces of the soft tissue are surgically covered by epithelium, then this epithelium will undergo only a minimal contraction.

B. Raw surfaces that are created on bony surfaces heal with acceptable soft tissue covering because the bone cannot contract during healing due to its rigidity.

C. If epithelial flaps are to be moved to a new location, they must be sufficiently undermined by sharp dissection to permit retention, without tension, in their new location.

D. There is a strong tendency for soft tissues undergoing plastic repair to return to their previous location. This tendency must be planned for by over-correcting and using adequate fixation of the tissue to a fixed base.

Many authors have described techniques for frenectomy, and some for vestibular remodeling, to enhance denture retention; but, prior to 1953 no reference is made to mucogingival surgery as either periodontal therapy or preventive technique. In his 1953 text, Goldman relates vestibular depth and frenum attachment to periodontal health. He describes a procedure for severing mandibular labial frenum attachments and simultaneously increasing the depth of the vestibule. His original technique, sometimes called a "pouch operation," involved an incision across the frenum, followed by a blunt dissection of connective tissue and muscle fibers from the periosteum, which were left in place over the bone.

1954 saw the publication of several papers introducing mucogingival surgery. Nabers, in his paper Repositioning The Attached Gingiva, discusses the histologic differences between gingiva and mucosa, and the inherent inability of mucosa to function as gingiva. The cases where routine gingivectomy incisions would involve the mucosa, he made vertical incisions and created

a flap which he sutured further apically after trimming papillae and epithelial remnants. He noted that after healing, a wider band of attached gingiva had been formed including normal healthy gingiva over the denuded bone.

Nabers' work represented a breakthrough and the years since 1954 have seen much developmental and investigational work in this field.

Gottsegen noted that in cases where there was insufficient gingiva, toothbrushing was interfered with, tension opened the gingival sulcus trapping food, and finally that following scaling and curettage, an area of this type would not return to health. His treatment was to incise through the mucosa to bone and bluntly reflect the tissue apical to the incision. He then placed an acrylic stent to prevent primary healing. He noted that incisal migration of the gingival attachment occurred following surgery.

Waltzer and Halik investigated the relationship between frenum attachment and gingival health. They believed their technique, similar to Gottsegen's, resulted in a widened zone of attached gingiva and deepened vestibular fornix.

Stewart, using a modification of the original Kazanjian technique, made a shallow incision in the mucosa paralleling the ridge and as far away from the ridge as it was desired to lower the level of final attachment. He then separated, by blunt dissection, the mucosa from the underlying connective tissue in the area apical to the incision. This operation was done in conjunction with routine marginal periodontal surgery.

Hileman in 1957 described a method of relieving the tension produced by attachment of the labial frenum near the gingival margin. An incision was made at right angles to the underlying bone following the mucogingival line, at least two teeth to each side of the area in question. The connective tissue and muscular attachments were then released from the periosteum by blunt dissection. They were separated sufficiently deep to displace the vestibule one to two centimeters lower. The area was then covered by an over-extended acrylic splint which was lined with periodontal pack and wired to the teeth.

Ivancie conducted a histologic study of repositioning the attached gingiva in periodontal therapy. In this study

a mucoperiosteal flap was raised by blunt dissection. The flap was carried apically, sutured and covered with an over-extended dressing. Biopsy specimens were obtained at one, six, and eight months postoperatively. He found that the resulting gingiva at one month was composed of immature connective tissue without good orientation of collagenous fiber bundles. Connective tissue was more cellular than normal and was covered by a thin layer of stratified squamous epithelium. The epithelium showed evidence of epithelial ridge formation and beginning keratinization. Six months sections revealed nearly complete reassumption of the characteristics of normal gingiva. His investigations led him to conclude that complete regeneration of healthy normal gingiva had occurred within eight months.

Freidman attempted to standardize terminology in mucogingival surgery. He felt that instead of terms such as "push back" and "pouch operation" the nomenclature should be based on the objective of the operation. Thus he proposed the following terms:

A. REPOSITIONING THE ATTACHED GINGIVA: An operation which attempts to retain the zone of attached gingiva rather than excise it by gingivectomy. The entire band of attached gingiva is repositioned as a mucoperiosteal flap.

B. GINGIVAL EXTENSION: When the object is to extend the zone of attached gingiva remaining after surgery by gingivoplasty or gingivectomy.

C. GINGIVAL REPLACEMENT: When the object is to create a new zone of attached gingiva where none existed before pocket elimination or after gingivectomy.

D. FRENOTOMY: When the frenum is incised or partially removed in order to reduce tension on the gingival margin.

E. FRENECTOMY: When the frenum is completely excised.

F. VESTIBULAR EXTENSION OR MUCOBUCCAL FOLD EXTENSION: To denote an operation, the object of which is to deepen the buccal vestibule.

Also, in 1957 Ariaudo and Tyrrell reported an evaluation of the technique presented by Nabers. They advocated, in addition to a horizontal incision through the attached gingiva, vertical incisions at the ends of the proposed flap.

These incisions were carried to, but not through, the periosteum. The mucosa was then reflected leaving the fibrous periosteum intact on the bone.

Following necessary osseous remodeling and root planning, the flap was positioned apically, being held in place by finger pressure until the clot structure was sufficiently strong to maintain the flap in position. This operation resulted in a narrow margin of exposed bone, a band of periosteum exposed, and finally the repositioned flap. They recommended the use of sutures, periodontal pack, and, when necessary, a compound stent to maintain the flap in its desired position.

Nabers published another paper on gingival repositioning in 1957. He stated that in most cases involving mucogingival surgery, if there is gingiva present, gingival repositioning is the treatment of choice.

In 1958 Ochsenbein discussed the relationship of underlying bone to tissue being repositioned over it. He suggested the use of small V-shaped incisions in the margin of the flap to relax circumferential tension and enable the tissue to conform to any undulating contours of the underlying bone.

Patur and Glickman in 1957 attempted to reattach gingiva to the denuded roots of lower anterior teeth by making a horizontal incision at the mucogingival junction, elevating the attached gingiva and suturing it in place coronally. They found that the flap became necrotic, was sloughed, and its original site healed with granulation tissue and new attached gingiva.

Pfeifer in 1959 compared the results of gingival repositioning as described by Nabers, and the replacement of gingiva by a method similar to that used by Bork and Weiner. He concluded that of the two techniques, gingival repositioning resulted in more comfortable healing with a superior postoperative clinical result. He recommended the use of an inert plastic film called Telfa to cover denuded bone prior to application of periodontal pack.

Many methods have been described for maintaining the soft tissue flap in its apical position. Many have used sutures covered by periodontal pack; sometimes reinforced by tinfoil. Other authors use acrylic or compound stents ligated to the teeth. Ariaudo and Tyrell suggested a

suspension suture placed around a tooth to prevent apical displacement of the flap. They sutured the flap to the periosteum by a mattress suture to prevent occlusal displacement. They also point out that unless the flap conforms intimately with the underlying bone, the spaces between the flap and the bone fill in with fibrin which becomes organized into dense connective tissue. To prevent this, they recommend the use of V-shaped incisions in such areas to allow the tissue to collapse into the depressions in the bone.

Siebert describes a method of stabilizing the tissue flap in its apical position by the use of Cobalt alloy tissue tacks. These tacks were fabricated by the author and inserted in holes drilled in the bone at the level at which he wished to maintain the mucoperiosteal flap.

Wilderman, Wentz, and Orban described their studies of the histology of regenerated tissues in 1960. Their experimental mucogingival surgery was performed on dogs Specimens of tissue were obtained periodically following surgery and the healing process analyzed. Their findings correspond to those of Ivanchie. They also state that in their subjects, some permanent bone loss occurred on the buccal root surface where bone was denuded, whereas none occurred in the interproximal surfaces.

Textbooks published by Orban, Wentz, Everett, and Grant in 1958 and Goldman, Schluger, Fox and Cohen in 1960, contain sections on mucogingival surgery which contain complete descriptions of theories and techniques available at that time.

Of particular interest is Goldman's description of "creeping attachment" or physiologic regeneration. It has been discovered by several clinicians that following mucogingival surgery there is often coronal migration of the marginal gingival tissue over a previously denuded root. This is especially evident in the lower anterior segment where a deep cleft involving one tooth is present preoperatively.

Goldman's text contains pictures of three cases which demonstrate this graphically. He observes that physiologic regeneration seems to occur most often when periosteum extends from the incised gingival portion into the new, deeper vestibular fornix. Regeneration continues until

a consistent marginal level is approached and reveals no clinical sulcular depth.

In Dental Clinics of North America, Practical Periodontal Therapy, 1960, Ochsenbein published a paper which was essentially the same as his 1958 paper, as did Hileman. In the same volume, Ariaudo and Tyrrell introduce the term "periodontoplasty" to describe surgical modification of the periodontium and closely associated anatomic structures. They describe a technique for gingival repositioning, leaving the periosteum on the bone.

Ochsenbein published another paper in 1960 which features excellent drawings and photographs and descriptions of various techniques. In this publication, he observes that it is impossible to separate the attached gingiva from its periosteum by sharp dissection. He describes the healing process of exposed alveolar bone noting that at first clumps of granulation tissue form at the cervical area, forming a ring which encircles the neck of the tooth. This tissue proliferates apcially over the denuded bone until it reaches the cut edge of the alveolar mucosa. He states: "The new zone of attached gingiva develops from the apical extension of the cervical granulation tissue and not from coronal proliferation of the alveolar mucosa."

He also describes a "double flap" procedure which denudes marginal bone and leaves a zone of periosteum from which the mucosa has been reflected to deepen the fornix.

Also in Dental Clinics of North America, Grupe describes a method of horizontally sliding a flap of attached gingiva and mucosa to cover a root denuded by a gingival cleft.

The "periosteal separation" as described by Corn attempts to obtain maximal attached gingiva and vestibular extension while denuding a minimal amount of bone. This is done by leaving the periosteum in place except in the cervical area and in a narrow band of denuded bone at the base of the fornix.

The success of most of the techniques mentioned have been judged on the basis of their clinical appearance postoperatively. Evidence has been contradictory in some cases, however. Bohannen, in 1961, presented the

results of his study of various techniques for vestibular extension in the lower anterior segment. He studied three techniques which he described as:

A. Complete denudation of labial alveolar bone.
B. Reflection of the mucosa but with periosteal coverage retained over the labial plate.
C. Vestibular incision into the base of the fornix.

He evaluated the success of the vestibular extension by positioning a radiopaque lead shot at the depth of the vestibular fornix and making serial cephalometric radiographs which he compared with the preoperative radiograph. He found that in all techniques, the six-months result was less extended than was the immediate postoperative result.

Six months postoperatively, the various techniques had resulted in the following mean depth of vestibular extension:

Series	Mean gain in vestibular depth
Denudation	5.1 mm.
Retained Periosteum	1.0 mm.
Vestibular Incision	1.5 mm.

This study and its successors may have an important effect on the techniques now being advocated by some authors.

III. CURRENT CONCEPTS AND TECHNIQUES

The discipline of mucogingival surgery is currently in a state of great progress. Many operations have been devised and studied. Some have been discarded; new ones are currently being evaluated to judge long-term effectiveness. Some basic concepts which have developed relative to mucogingival surgery are:

A. Attached gingiva will be formed on that bone which is completely denuded of periosteum.

B. The healing of denuded bone is slower and more painful than that of bone on which a mucoperiosteal flap or periosteum remains.

C. Denudation of bone may result in some destruction of bone, particularly over the labial aspect of the root.

D. Physiologic regeneration is a clinical entity and follows removal of irritants (including tension) and surgical removal of existing epithelium.

E. Simple frenectomies, even coupled with dissection of attachments and postoperative packing, result in only temporary vestibular extension.

F. Four techniques, which differ only slightly, now being used quite extensively are the sliding flap, gingival repositioning, double flap, and periosteal separation.

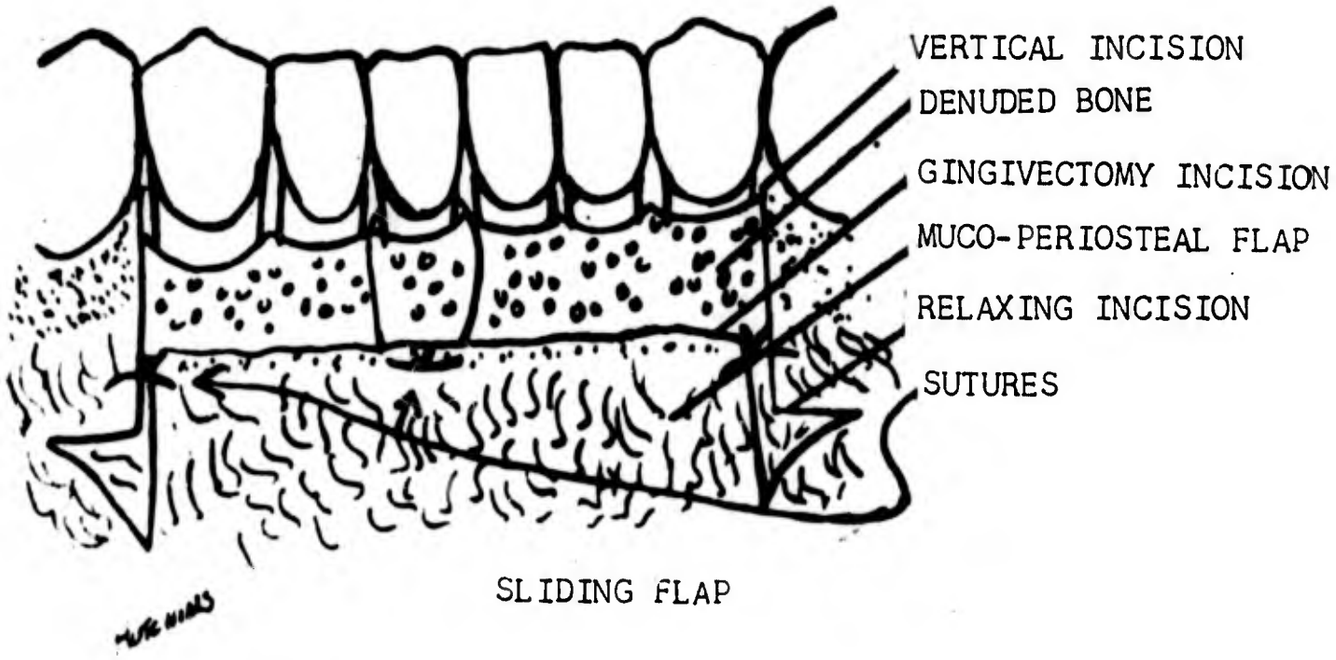
1. Sliding Flap ("Push Back")

a. Indications:

1). Lack of sufficient attached gingiva following marginal surgery.
2). Slightly, but not severly, shallow vestibule.
3). Presence of solid labial plate of bone.

b. Technique:

Following gingivectomy, the remaining attached gingiva and mucosa are elevated as a modified muco-periosteal flap, denuding bone. In the lower anterior segment, this flap should extend from cuspid to cuspid. The flap, which may require both vertical incisions at its ends and further horizontal relaxing incisions distal to this, is freed from underlying bone by blunt dissection, freeing frenum and muscle attachments. It is repositioned apically and stabilized either by sutures, tissue tacks, or splints.



SLIDING FLAP

2. Gingival Repositioning

a. Indications:

1). Periodontal pockets, the base of which extend to or below the mucogingival junction.
2). Adequate vestibular depth is present.
3). A band of attached gingiva is present (although not attached to the teeth).

b. Technique:

Gingival repositioning is a modification of the sliding flap. It differs only in that the marginal incision is designed to conserve gingival tissue. Instead of a gingivectomy, a "repositioning incision" is made; that is, a reverse level incision. Below this, the flap is blunt-dissected and repositioned as in a sliding flap technique.

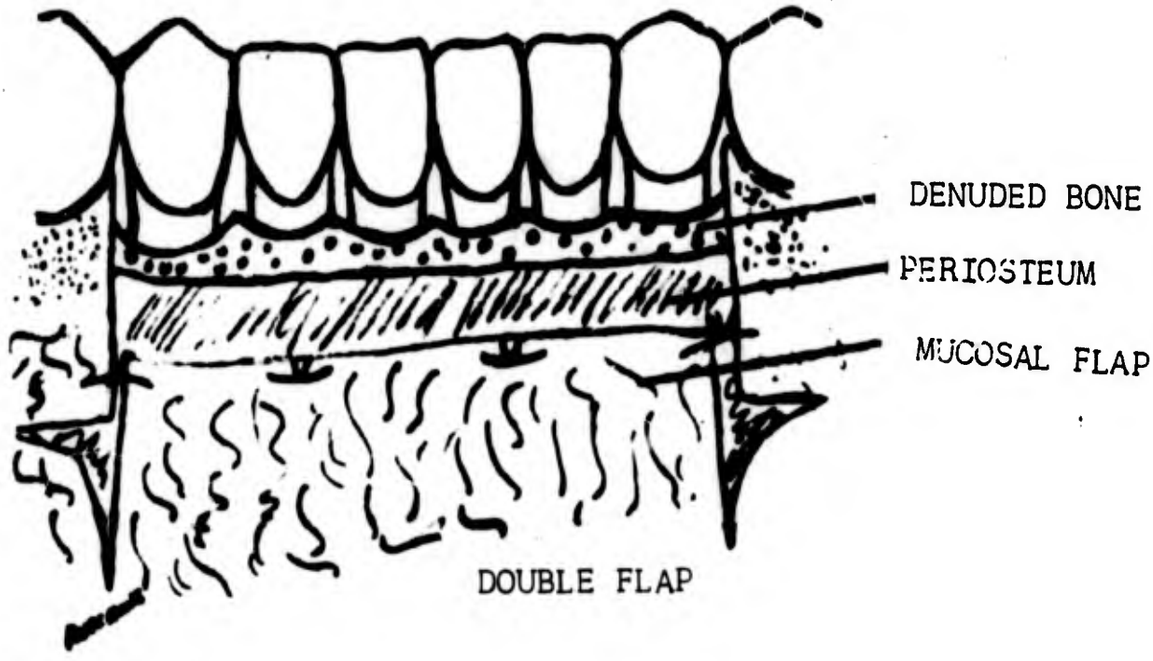
3. Double Flap

a. Indications:

1). Shallow vestibule
2). Lack of sufficient width of attached gingiva.

b. Technique:

Routine marginal surgery is performed, using a gingivectomy incision. Osteoplasty is performed where necessary, and a narrow band of crestal bone is denuded Vertical incisions are made at the cuspid area, through the mucosa, but not through the periosteum. The mucosa is separated from the periosteum by sharp dissection, severing frenum and muscle attachments. The mucosal flap is positioned further apically, sutured to underlying periosteum and packed in place.



DOUBLE FLAP

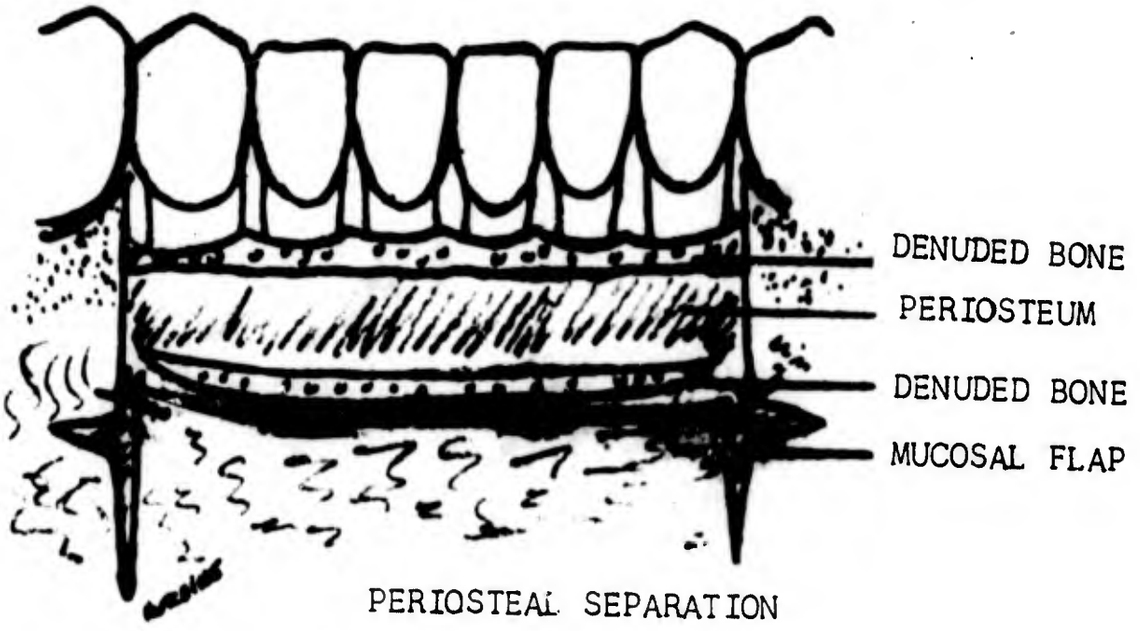
4. Periosteal Separation

a. Indications:

1). Insufficient attached gingiva.
2). Shallow vestibule.
3). High muscle and frenum attachments.

b. Technique:

Periosteal separation varies from the double flap only in that a band of bone is denuded at the base of the fornix to "tack" the vestibule at that level. Following routine surgery, sharp dissection of the mucosa from periosteum is accomplished. Muscle and fibrous attachments are severed to a depth considered necessary for vestibular extension. A horizontal incision is made through the periosteum at the depth of the retracted alveolar mucosa. A narrow band of bone is denuded at this level. The mucosal flap is sutured to periosteum apical to this area and surgical dressing applied.



PERIOSTEAL SEPARATION

If a suitable band of attached gingiva is present, and the only objective is the extension of the vestibule, this operation can be initiated with a horizontal incision at the muco-gingival junction and then completing the procedure as described above.

IV. DISCUSSION:

The objective of all four techniques described is the same.

A. Removal of marginal pathology.
B. Creation of acceptable width of attached gingiva.
C. Creation of a physiologically-acceptable vestibular fornix.

Of the four techniques, the gingival replacement is probably the most conservative in that it preserves the existing gingival tissue. This provides a tissue dressing for most of the denuded bone. This technique is limited, however, in that it requires presence of attached gingival tissue preoperatively, and cannot be used when the vestibule must be extended to a depth such that the attached gingiva of the flap would be positioned on the mucosal aspect of the fornix.

The "push back" results in the broadest band of attached gingiva and the most successful for deepening the fornix. Due to the large area of denuded bone, however, the postoperative course is likely to be stormy. Pain can usually be controlled by a sedative periodontal dressing.

The "Double Flap" is somewhat more difficult to manipulate. There is some danger of perforating the mucosa during sharp dissection. Leaving the periosteum intact provides more comfortable healing, but may result in only temporary deepening of the fornix.

For creation of a new zone of attached gingiva and deepening of the vestibule in the lower anterior segment, the "periosteal separation" currently seems to be the treatment of choice. The area of denuded bone at the base of the fornix serves to "tact" the fornix at that level, while leaving the periosteum in place coronally results in more comfortable healing.

More studies of the long-term results of this treatment are necessary before any valid judgments can be made.

It must be emphasized that the field of mucogingival surgery is constantly changing and should continually be re-evaluated in light of new evidence and long-range studies of healing and results obtained.

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LECTURE NO. 14

PATIENT HOME TREATMENT

Wilfred B. Bell*

I. INTRODUCTION:

The ultimate success of the treated periodontal case depends to a large extent on the ability of the patient to understand and carry out proper oral hygiene measures. The periodontist and the patient are partners in the business of the eradication of periodontal disease and maintenance of the oral tissues in a state of health thereafter. Their respective individual responsibilities are definitive only when they are accomplished collectively. Failure of one will neutralize the other one's effort.

II. OBJECTIVES:

Orban lists the main objectives of home care as follows:

A. To reduce microorganisms by removal of all debris, materia alba, and plaques from the teeth, especially from the interproximal spaces, and thus eliminate irritation to the gingiva and combat halitosis.

B. To promote blood circulation.

C. To promote keratinization of gingival tissues, rendering them more resistant to mechanical and bacterial irritation.

III. THE TOOTHBRUSH:

Literally scores of different toothbrushes are on the market, each purporting to offer certain advantages over a competetor's product. Formerly, we were only faced with the selection of the hardness of the bristles, type of bristle, number of tufts, shape of the handle, and the like. Now today, in this mechanical age, in addition to the

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conventional toothbrush selection, we are faced with the evaluation of the automatic toothbrush and must decide whether the self-charged, battery-operated, or plug-in kind is best for our patients. As a matter of fact, maybe the toothbrush is outmoded and perhaps what we really need is one of the new high-pressure water jet appliances, the use of which may be further enhanced by the addition of a comfortable stool by the sink where the patient may sit in comfort while practicing his oral hygiene. Additional embellishment may include a control panel attached to the sink at which the patient could play with an impressive array of tubes, valves, bottles of various colored "therapeutic agents," and the like.

It should be obvious that we will defeat our purpose if we set up an oral hygiene regimen for the patient that is too complicated for him to understand, too expensive for him to purchase, and too time-consuming for him to perform. Furthermore, it is highly unlikely that the subjective gratification of the patient following oral physiotherapy will ever reach that of a steam bath and massage. In other words, we have a certain lack of motivation on the part of the patient.

Selection of the toothbrush should be determined by the particular brushing technique to be recommended, which in turn may be predicated on several individual factors. This is to say that one particular toothbrush and one particular technique may not be indicated for all patients.

Generally speaking, however, we have found that a soft nylon multi-tufted (48 tufts), medium-sized, 4-row type may be used with acceptable results by most patients. We feel that it has the following advantages:

A. The soft texture precludes the possibility of damage to the soft oral tissues.

B. The individual bristles number some 1800 which, when compressed, form a compact, dense mat and cover a relatively large area.

C. It is relatively inexpensive.

D. Patient acceptability is good.

IV. ORAL HYGIENE INSTRUCTIONS:

A. Some type of disclosing solution, or wafer, is given to the patient to determine the present level of oral hygiene. This evaluation is made with the patient and areas of neglect are pointed out.

B. The patient is then instructed to use the brush in his own manner, after which the mouth is again examined for remaining areas of food retention made more obvious by the still-present disclosing dye.

C. The recommended brushing technique is explained to the patient, and demonstrated first on models and then in the patient's mouth.

D. The patient practices the technique in his mouth and receives corrective assistance as needed from the dentist.

E. Auxiliary hygienic aids are explained and demonstrated.

V. THE BRUSHING TECHNIQUE:

Various methods have been described over the years, all of which are effective in varying degrees if done properly.

A. Charters: On the facial surfaces, the brush is positioned with the bristles pointing coronally, the tips on the teeth and the side on the gingiva. A circular, vibratory motion is used in such a way that the brush bristles remain relatively stationary in a rather narrow area. The "toe" of the brush is used for the lingual surfaces and is rather difficult to negotiate without some modification of the technique.

B. Stillman: Bristles are placed at about a 45-degree angle with the tips of the bristles pointing apically and positioned on the gingiva. This is the reverse of the Charters method. Light pressure is applied and the brush brought coronally with a sweeping motion, but without twisting the brush. This method works equally well on the facial and lingual surfaces.

C. Fones: The brush is placed in the mouth and the teeth occluded. With the brush at about right angles to the facial surfaces of the upper and lower teeth, a scrubbing circle is described, negotiating both the upper and lower segments. The lingual surfaces are done similarly with the teeth parted.

D. Another method, which we have found to be quite effective using the above-described brush, is the placement of the brush at approximately a 45-degree angle with half the bristles covering the attached gingiva and the other half on the occlusal or incisal surfaces of the teeth. The action is a rapid, short stroke, back-and-forth motion with the bristles spread and pressed against the gingiva, the facial (or lingual), and proximal surfaces of the teeth.

The time spent for each brushing period should be individually recommended depending on the requirement for the case. Emphasis should be placed on the importance of reaching all areas in the mouth, particularly the distal surfaces of the last posterior teeth. Special care is also needed for isolated and malposed teeth as well as post-surgical gingival areas which may present special oral hygiene problems.

VI. AUXILIARY AIDS:

Adjunctive aids should be selectively prescribed in cases where they are needed to fulfill a certain requirement in order to further elevate the oral hygiene level. They should not be recommended merely as an additional exercise to satisfy the dentist who believes that if a "little is good--more is better."

Some of the more useful aids are:

A. Cone-shaped rubber tips: Reasonably effective for proximal cleansing and gingival massage.

B. Triangular-shaped balsa wood points (STIMUDENTS): Excellent for interproximal cleansing and gingival stimulation, especially following periodontal surgery. They are designed to fit the interproximal space and their softness allows for a change in dimension to conform to most areas.

C. Dental tape and floss: Good for the removal of soft accretions in relatively inaccessible areas including fixed prosthesis. Should be used carefully to avoid soft tissue damage.

D. Pipe cleaners: Most effective for cleansing post surgical furcations.

E. Others:

1. Small rubber sponges on a hemostat-type instrument for gingival massage.
2. Gauze strips used primarily to clean proximal areas of isolated or spaced teeth.
3. Knotted nylon yarn used in the same manner as dental tape.

VII. THE AUTOMATIC TOOTHBRUSH:

The various automatic toothbrushes on the market all have one thing in common--they substitute electrical energy in one form or another for muscular energy. The oscillation of the brush, whether in the vertical or horizontal direction, does reduce the patient's role in the operation to merely placing the brush in the proper region.

Various reports comparing the automatic and hand brushing effectiveness have been published recently. Sanders and Robinson, using rapid calculus formers as subjects, found hand brushing (nylon bristles) three times a day for one month, did not significantly reduce calculus when compared with no brushing at all. Brushing with an automatic toothbrush, however, three times a day for one month significantly reduced formation of calculus when compared with the groups using the hand brush or no brush at all.

Chilton, et al., selected a group of cooperative students, all of whom demonstrated some gingival inflammation; and by random selection, divided them into two groups. One group used an electric toothbrush on their upper arch and a standard toothbrush on the lower arch, while the other group did the reverse. Both groups brushed two times a day for eight weeks. They found no statistically significant difference, using the PMA and cleanliness index, between the electrically-brushed and hand-brushed areas.

Another study, using the same kind of electric toothbrush as described in the preceding paragraph, was conducted by Toto and Farchione. Twenty patients were selected with a diagnosis of periodontitis. They were all given routine periodontal treatment, but ten patients were given oral hygiene instructions using the electrically powered toothbrush while the other ten patients were instructed in the use of the conventional brush. The patients returned every week for continued treatment and were scored as to deposits of materia alba, color of papillary and marginal gingivae, and degree of stippling. In all the patients, there was a parallel improvement in gingival health through the first four weeks. After this period, the investigators report that the gingival stippling was more marked in the group that used the electrical instrument.

It would seem that the electrically-powered toothbrush could be used with greater advantage by handicapped persons and those individuals who for some reason or another are unable to manipulate the hand brush in the recommended manner. Patients who maintain a high standard of oral hygiene with the conventional brush probably would find no additional advantage to the electrically powered device.

VIII. SUMMARY:

Our responsibility to the patient does not end when we remove the periodontal dressing for the last time; on the contrary, our most difficult job is just beginning. We must educate the patient to appreciate what has been done to establish a state of health in the oral cavity, and impress upon him the absolute necessity for his cooperation in following our instructions to maintain that which we have given him. The education of the patient to new habit patterns in his oral hygiene procedures may be frustrating and tiresome to the practitioner and patient alike, but patience must be exercised and repeated visits may be necessary before the subject is able to completely comprehend and physically accomplish that which is necessary.

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LECTURE NO. 15

PRINCIPLES OF OCCLUSION AND TRAUMATISM

Samuel Kobrinsky*

I. INTRODUCTION:

In 1901, Karolyi, an Austrian dentist, described occlusal trauma as a factor in the etiology of pyorrhea alveolaris. Later, Maurice Roy, in discussing treatment of pyorrhea alveolaris, concluded that teeth in normal function would have a more favorable prognosis. An evolution of these early clinical impressions, the role of trauma from occlusion as an important etiologic factor in the causation of periodontal disease has been well established on an experimental and histopathologic basis.

II. IDEAL FUNCTIONAL RELATIONSHIPS:

Breitner speaks of the ideal tooth-supporting apparatus being in functional equilibration--forces in the masticatory skeleton--the muscles, tongue, growth impulses--counteracting one another in perfect balance.

Teeth are in a delicately balanced, functional relationship to each other within maxillary and mandibular, as well as in occlusal and articular arrangement. They are constantly growing in an occlusal direction accompanied by growth of alveolar bone at the margin as well as at the fundus to compensate for attrition. Another growth process is mesial migration to compensate for proximal attrition.

In addition to these active movements, there is the passive shift of the epithelial attachment and the coordinated shift in the relation of the intra-alveolar and extra-alveolar parts of the teeth. Biologically we have the deposition of new cementum, insertion of new fibers, and the constant apposition and resorption of bone.

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The muscular balance as outlined by Breitner is as follows:

Lips----------Tongue
Cheeks----------Tongue
Eruption of teeth----Masticatory muscles
Masseter----------Periodontal ligament elasticity

Internal pterygoid

In vertical movement---------Periodontal ligament elasticity
In lateral movement----------Internal pterygoid, opposite side

External pterygoid

In anterior movement---------Posterior third of temporalis, suprahyoid, digastric
In lateral movement----------External pterygoid, on the other side

Schuyler describes occlusal harmony as:

A. Maximum distribution of stress in centric relation
B. Retention of vertical dimension.
C. Harmony of guiding inclines and, therefore, distribution of eccentric occlusal stresses.
D. Harmony of guiding inclines so that occlusal stresses may be favorably applied to the supporting tissues.
E. Sharpness of cutting cusps.
F. Adequate food exits.
G. Decrease in contact surfaces.

Under ideal conditions, there is a constant and even wear on the occlusal surfaces and incisal edges and also at the contact areas, compensated for by vertical eruption and mesial shift. Sicher states that this constant shift in relation between bone and tooth is possible because of the rather short life span of the principle fibers of the periodontal membrane. Recent findings of great numbers of mitoses in the periodontal membrane as compared to other ligaments give evidence of the continual rebuilding and rearranging of the fibers.

III. ETIOLOGY OF MALOCCLUSION:

Orban feels that the delicacy of the balance of the masticatory apparatus is its main weakness. Complications by intrinsic factors such as metabolic disturbances and endocrine imbalance may impair the normal adaptability of the tissues.

According to Sicher, it is the rare individual who shows the ideal attrition necessary to maintain this balanced occlusion. The average person shows a lack or abnormal pattern of wear with concomitant breakdown of the delicately correlated and balanced changes in the functioning tooth. The basic problem created by abnormal wear is the conversion of well-tolerated axial-occlusal stresses to a poorly tolerated lateral-occlusal stress. Breitner points out that only a small number of the principal fibers of the periodontal membrane are designed to resist lateral stresses.

A. Lateral forces may be created by:

1. Discrepancy between centric and habitual occlusion with prematurities in the final millimeter of closure.

2. Balancing side prematurities in lateral excursion.

3. Excessive overbite with incisal guidance which is not harmonious with the posterior guiding incline, and the slope of the anterior wall of the mandibular fossa.

4. Bruxism: In addition to harmful lateral forces, there may be excessive wear and reduction of vertical dimension if inadequately compensated for by active eruption. Ramfjord, in a study of 34 adults with a habit of bruxism, found certain similarities of symptoms. He found they all had discrepancies between centric relation and centric occlusion, and he found that many had disturbed muscle contraction patterns in swallowing, and that many had severe balancing side prematurities.

B. There are many iatrogenic causes of occlusal trauma:

1. Extractions with no replacement create problems of tipping and extrusion of neighboring and opposing teeth.

2. Restorations which are high or which have no occlusal contact.

3. Improper location of proximal contact can cause vertical and lateral tooth displacement.

4. Increase of the normal vertical dimension. Breitner states this will cause increased tension of the internal pterygoid, masseter, and temporalis muscles which will, in turn, cause occlusal and temporomandibular ligament problems.

C. Periodontal Disease: The potential for trauma from occlusion increases following periodontal disease. Gingival recession and loss of alveolar bone associated with periodontal disease creates a condition unfavorable to the stress bearing potential of the periodontal membrane. With recession, there is a decreased area available for fiber attachment; this results in an increased stress being placed on the remaining fibers. Loss of alveolar bone also results in more stress on fewer fibers. There is also more force exerted on the remaining bone.

D. Habit Neuroses: Sorrin has enumerated many habits and improper practices which may damage the periodontal tissues. Some of these are:

1. Lip biting

2. Cheek biting

3. Bruxism

4. Clenching

5. Tongue thrusting

6. Fingernail biting

7. Pipe smoking

8. Unilateral mastication

IV. EFFECT OF TRAUMATISM ON THE PERIODONTIUM:

Since the early experiments of Gottlieb and Orban in 1932, the effects of occlusal stresses on the periodontium have been well documented and this, perhaps, is the most lucid phase of the study of occlusion.

Gottlieb and Orban found that by placing high crowns on dogs' teeth they could create excessive lateral forces on the periodontium, and study the effect by means of microscopic sections. They found bone resorption in areas of pressure and apposition in areas of tension. In areas of excessive pressure, there was necrosis and undermining resorption. In most instances, the teeth were displaced to new positions and the periodontal tissues returned to normal.

Kronfeld studied an autopsy specimen which contained teeth in heavy, light, and no function. He found that heavy, axial function produced hypertrophic periodontal tissues and teeth with little or no function produced reduction of the periodontal soft tissues and atrophy of bone. He said that no conclusions could be drawn concerning the influence of over-function on the teeth and he described this particular jaw as "well-reacting."

Orban repeated his and Gottlieb's high crown experiment and reported on some additional findings. He found that the axis around which the tooth tipped was always slightly apical to the middle of the root. He found that teeth on the control side showed traumatic changes due to cusp interference and to a shift of the jaw to the control side. Areas of bone necrosis resorbed very slowly by undermining resorption because the dead tissue could not produce osteoclasts. Bone formed on the outside of the alveolar ridge to compensate for bone resorbed on the periodontal ligament side of the ridge. This prevented shortening of the ridge. He found no gingival changes except in some areas where there was food impaction.

Bhaskar and Orban repeated the experiment on monkeys by placing high mesial-occlusal-distal inlays. They paid particular attention to the soft tissue reaction and reported that occlusal trauma did not produce any changes in the soft tissue. The pressure side findings were necrosis, bone resorption, root resorption, and thrombosis in the

periodontal ligament. On the tension side, there was widening of the periodontal ligament, thrombosis, and bone formation.

Glickman and Weiss also reported that trauma from occlusion did not initiate periodontal pocket formation in experimental animals.

Wentz, Jarabek, and Orban performed the following experiment in response to criticism that the previous works did not duplicate actual cuspal interference because the teeth were able to move out of traumatic occlusion. In their "jiggling experiment," they placed high crowns which forced the teeth bucally; then, by means of an orthodontic appliance they placed lingual tension on the crown so that there would be a buccal movement when the teeth were occluded, and a lingual movement when the teeth were out of occlusion. They concluded that although the teeth loosened and the periodontal ligament was three times the normal width, this did not connote a sign of inflammatory periodontal disease, but rather a tissue adaptation to an increased functional demand. They found an additional functional compensation in the new bone formation on periosteal and endosteal surfaces of the bone around these teeth.

These experiments would seem to indicate that trauma from occlusion does not initiate periodontal pockets.

Many researchers became interested in what effect trauma from occlusion would have on pre-existent periodontal disease.

Weinmann reported that the inflammatory pathway in periodontal disease was from the gingiva through the perivascular space of the intra-alveolar vessels. After reaching the marrow spaces of the alveolar bone, it extended laterally into the periodontal ligament. However, he did report that in several cases the inflammation seemed to spread directly into the periodontal ligament from the gingiva.

Suspecting that trauma could be causing the inflammation to spread directly to the periodontal ligament, the following experiment was performed by Macapanpan and Weinmann:

By placing rubber dam material between rat molars, a simultaneous gingival inflammation and a traumatic injury were produced. Histologic sections revealed necrosis on the pressure side, and the direct course of inflammation into the periodontal ligament on the tension side. They concluded that damage to the periodontal ligament on the tension side diminished the normal periodontal ligament resistance to direct infiltration from a gingival inflammation with a resulting periodontitis.

Glickman repeated the above experiment because he suspected that necrosis on the pressure side of the periodontal ligament could have prevented the entry of gingival inflammation. He found that where there was no periodontal ligament necrosis, the gingival inflammation pathway was altered primarily to the pressure side of the periodontal ligament, and that angular bone deformities were produced there.

One might speculate that if this were true, we could predict the direction of the traumatic forces and thus easily correct them.

The effect of trauma on the roots of the involved teeth has been observed and discussed by the following:

A. Bernier reports that cementoid deposition on the root prevents resorption. Like osteoid, it is highly resistant to resorption.

B. Coolidge states that cemental fracture is a common traumatic injury.

C. Ramfjord and Kohler in their study of periodontal reaction to functional stress found little cementoid or new cementum. The striking feature of their study was the stability of Sharpey's fibers in spite of increased functional stress.

D. Kronfeld states that the thickness of cementum showed inverse ratio to the thickness of the periodontal ligament, and cementum was formed excessively in functionless teeth.

E. Weinmann, reporting on the pattern of resorption and repair of human cementum, found that 90 per cent of the

teeth studied showed some root resorption. He found most resorption in the apical third of the root, usually on the mesial and buccal. He concluded that trauma appeared to be the most important factor.

V. TREATMENT - THE ADJUSTMENT OF THE OCCLUSION:

A. In the correction of Occlusal disharmony, Schuyler presents the following objectives:

1. To correlate centric occlusion with unstrained centric relation.

2. To obtain maximum distribution of occlusal stress in centric relation.

3. To retain correct vertical dimension.

4. To equilibrate the steepness of tooth inclines, thereby distributing eccentric occlusal stresses.

5. To reduce the incline of guiding tooth surfaces so that occlusal stresses may be more favorably applied to the supporting tissues.

6. To retain sharpness of cutting cusps.

7. To increase food exits.

8. To decrease contact surfaces.

B. Occlusal adjustments in centric relation:

1. When a cusp interferes in centric relation and also in excursive movements, it should be reduced.

2. When a cusp interferes in centric but is in proper contact in the excursive movements, the fossa should be reduced.

3. When a cusp does not contact the opposing fossa in centric, but intereferes in excursive movements, by corrective grinding the guiding plane of the opposing fossa should be reduced.

4. After harmony has been established in centric, all corrective grinding to relieve premature contact in lateral and protrusive positions must be done upon the guiding planes.

5. If in corrective grinding it becomes necessary to relieve a centric holding cusp, the other cusp must be carefully retained to maintain centric occlusion.

6. Premature contact between anterior teeth in centric should be relieved by reducing the edges of the lower teeth.

C. Occlusal Adjustment in Lateral Movements:

1. In reducing the inclines of the guiding planes, the areas where mandibular and maxillary cusps rest in opposing fossae should not be relieved, but the guiding planes of the cusps which do not maintain centric may be relieved.

2. When the lingual cusp or the maxillary molar is in excessive balancing contact with the buccal cusp of the mandibular molar, correction may be made by slight grinding on the guiding plane of the mandibular molar. Correction is never made simultaneously on the maxillary molar, as this would eliminate occlusion in centric.

3. Premature contacts between anterior teeth in excursive movements should be relieved by corrective grinding on the lingual inclines of the maxillary incisors being careful to retain the centric stops.

D. Correction of Protrusive Excursion:

An ideal relationship in protrusive where all incisal edges contact in the anterior and all buccal cusps contact in the posterior is rarely encountered; and in fact, we are rarely able to correct to this ideal. It is possible to give considerable relief by bringing at least the anterior incisal edges into uniform contact. Relief is made by reducing the lingual of the maxillary anteriors being careful to

maintain centric. It is sometimes possible where there is excess contact of the lower incisor to reduce this contact to its lowest point and thus materially reduce the amount of grinding necessary on the maxillary opponent.

E. Although Schuyler's principles have fairly wide acceptance, there are some men who differ with his rationale. The basic differences arise in a new concept that no corrections at all may be needed in the excursive movements. The basis for this concept comes from experimental work done by McCall (Electronics) and Jankelson (Cinefluoroscopy) who show that teeth rarely come into contact when excursive movements are ordinarily made. These men advocate correction only of the centric prematurities.

Another new concept of limited excursive correction has been proposed by D'Amico who, with the photographic evidence of many fossil skulls, shows our antecedents had massive canines which were, in fact, the only teeth which came into contact in excursive movements. These teeth are still best-able to stand the lateral stresses of the excursive movements; therefore, D'Amico advocates bringing these teeth into prominence in excursive movements by: (1) building them up with restorations or (2) flattening the guiding planes of the posterior teeth, or (3) a combination of both methods.

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LECTURE NO. 16

THEORY AND PRINCIPLES OF PERIODONTAL SPLINTING

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I. INTRODUCTION

Periodontal disease causes both loss of attachment apparatus and pathologic changes in the remaining attachment apparatus. In such instances, occlusal forces which were once normal and exerted a favorable influence upon the periodontium can become traumatic (secondary occlusal traumatism) and cause tooth mobility. Many times normal periodontal tissues cannot withstand parafunctional occlusal forces; i.e., those encountered in bruxing, clenching, or in occlusal habits such as cheek biting, lip biting, tongue or foreign object biting. One or several teeth in traumatic occlusal relation may show breakdown of the periodontium (primary occlusal traumatism) and exhibit abnormal mobility. The reduction of abnormal tooth mobility is one of the objectives of periodontal treatment. Several methods of accomplishing this are available to the operator, some or all of which may be employed in a given case.

II. METHODS OF REDUCING MOBILITY

A. Eliminate inflammatory periodontal disease (improves the quality of the attachment apparatus).

B. Raise the height of alveolar attachment (increases the quantity of periodontal support; i.e., "fill-in" or reattachment in infra-bony pockets.

C. Eliminate or reduce occlusal trauma by:

1. Reducing the crown-root ratio.

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2. Occlusal equilibration to eliminate trauma on individual teeth and to obtain favorably distributed occlusal contact over a maximum number of teeth in centric and to obtain favorable relations in eccentric movements.

3. Eliminate parafunctional trauma; i.e., bruxism, clenching, abnormal biting habits, etc.

D. Use of periodontal splinting.

III. PURPOSE:

Splinting is an adjunct used in periodontal treatment. A dental splint is primarily used for reducing tooth mobility. It may also be a fixed or removable replacement for missing teeth. The purpose of splinting is to redistribute occlusal forces so that the adaptive capacity of the remaining supporting structures is not exceeded.

IV. THEORY:

Goldman, et al, state the principles of splinting are predicated on the fact that teeth ligated together create a multi-rooted unit. Thus, if two single-rooted teeth, each with a center of rotation apical to the middle third of the root, are splinted together (1) the area of root resistance is increased, and (2) the center of rotation is changed to a location somewhere in the interseptal area between them. This would give a much greater resistance to mesio-distal thrust and this resistance would increase with the number of teeth incorporated into the splint. Glickman, et al, reported on the distribution of occlusal forces on teeth included in fixed splints. They studied histologic changes in periodontal tissues and found that when excessive occlusal force was placed upon one tooth in the splint, (1) all the teeth in the splint suffered comparable injury, (2) when excessive occlusal force was applied to one non-splinted tooth, the injurious effect was not transmitted to adjacent teeth. The authors feel that, similarly, normal occlusal forces in splinted teeth would be shared by all teeth included in the splint. Another finding in this study was that furcation areas of the periodontium are the areas most susceptible to excessive occlusal forces applied in a mesio-apical or disto-apical direction.

V. PRINCIPLES OF SPLINTING:

A. Ideally, the terminal abutments should be firm. If a terminal abutment is mobile, the mesio-distal component is

relatively easily reduced, but the facio-lingual component is not and the number of other abutments must be increased to reduce it.

B. A sufficient number of firm teeth must be included in the splint--according to Glickman--the total functional root surface of the firm teeth should exceed that of the mobile teeth included.

C. Since mesio-distal mobility is more easily reduced than facio-lingual mobility, it may be possible to stabilize one mobile tooth with a single firm tooth if the major part of the mobility is mesio-distal. Usually, this will not be the case. If the mobility is facio-lingual, several firm teeth in the same segment may be required to reduce it or, better yet, the splint should go around the arch and include teeth of another segment or even both other segments. Bilateral splinting will resist movement in any direction.

D. The splint should be properly contoured to provide adequate interproximal embrasures and protect the gingiva from food impaction at the facial and lingual surfaces. Needless to say, it should not impinge upon or irritate the gingiva or mucous membranes.

E. Simplicity and acceptability of esthetics should characterize the splint.

F. The splint should allow proper oral physiotherapy. Its purpose will be defeated if it fosters accumulation of debris, causing inflammatory changes which impair healing and contribute to mobility.

G. The splint must be in functional harmony with all functional positions and excursions of the mandible; otherwise, the destruction of the periodontium of the splinted teeth will be accelerated and the functional relationships of the rest of the dentition will be disturbed.

H. In the final analysis, clinical judgment will be the determining factor in splint planning. The number and locations of teeth with mobility and the direction of this mobility, the locations and amount of root surface within the periodontium of the firm abutments, the functional and parafunctional demands to be made upon it, and other clinical considerations will determine the extent and type of splint to be used. No set

rules can be made which could apply to all cases.

VI. CLASSIFICATION OF SPLINTS:

Splints may be classified in numerous ways (e.g., type of material, fixed or removable, internal or external), but the most useful classification is probably according to purpose.

A. Temporary splints--used for a short time (usually up to two months) to afford physiologic rest and facilitate healing of mobile teeth following scaling and curettage or surgical procedures. Organization of the clot, maturation of granulation tissue and, in the case of infra-bony pockets, laying down of new bone are disrupted by disturbances of tissue caused by mobile teeth. Temporary splints can be placed prior to starting treatment and the injurious disturbances minimized during healing. After the temporary splint is removed, the teeth are re-evaluated, occlusion re-checked and a permanent splint made if necessary. If teeth which are not being traumatized are going to become firm, they will do so in about two months. If after this time the teeth are still mobile, no useful purpose will be served by re-applying the temporary splint. If they are mobile, but can be retained, a permanent splint should then be made. Examples: External acrylic and wire, removable external metal casting, etc.

B. Permanent splints--serve as instant adjuncts in the maintenance of periodontal helath. They are used when the periodontium has been so altered that ordinarily physiologic forces are potential sources of injury. The benefits afforded the periodontium of the mobile teeth exceed the disadvantage of the burden put on the stable teeth--which would have to be similarly involved if the loose teeth were removed and replaced by a prosthesis. Permanent splints may be either fixed or removable, internal--consisting of the various intracoronal and extracoronal types of gold castings or amalgam and wire or external--removable metal castings. Night guards made in cases of bruxism are considered by some to be permanent splints.

C. Provisional splints--are maintained in position for an indefinite period for the purpose of (1) providing an opportunity for periodontal repair and (2) to determine whether teeth with a questionable prognosis will improve. The type of construction can vary from the orthodontic band type to

internal gold castings. The acrylic and wire intracoronal type could be used as could external metal casting. If a fixed type (except for acrylic and wire) is used, it is cemented to place with zinc oxide and eugenol. Questionable teeth included within these splints can be evaluated every three to four months or more often. A provisional splint is worn until a decision can be reached as to whether or not the questionable teeth can be maintained in periodontal health, at which time the tooth is either removed or included in a permanent splint.

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LECTURE NO. 17

PERIODONTAL SPLINTS

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I. INTRODUCTION:

Once the rationale for using the various types of splints is understood, the numerous technics which have been proposed and are in more or less common usage can be discussed in the light of these basic principles. The purpose of this discussion is not to detail their construction, but to present them in general terms for the purposes of familiarization. For specific information on construction, the reader should consult the appropriate references.

It will be evident that some of the technics will serve for more than one class of splint, but the discussion here will be presented under the type where it will be more generally used.

Splinting falls in the province of occluso-rehabilitation, for surely if a case requires a splint it must be made with the idea of establishing optimal function. This entails the application of various phases of dentistry; i.e., selective grinding, orthodontic movement, restorative dentistry, and in some cases, removable prostheses. All procedures must be coordinated so as to establish a functional environment conducive to the health of the periodontium.

II. TEMPORARY SPLINTS:

Many different types of temporary splints have been reported in the literature and there are many individual variations of each technic.

A. Silk ligature splints are generally limited to use on anterior teeth for very short durations (usually one week),

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but can be used on posteriors if the cervical areas of these teeth are constricted so that the material can be kept away from the gingiva. A double loop of ligature is placed about the distal terminal abutment and tied at the mesial with a surgeon's knot. The procedure is repeated for each tooth to be included until the other terminal tooth has been ligated. The loop is placed incisal to the cingulum and apical to the contact points. In cases of diastemata or missing teeth, additional knots are tied to span the space so that tooth movement will not be inadvertently induced. The ligature is then coated with copal varnish, or better still, self curing acrylic. The disadvantages due to the nature of the material are obvious.

B. Wire ligature splints are limited to lower anteriors and those upper anteriors and posteriors where they can be kept from interfering with occlusion and with gingival health and oral physiotherapy.

A double strand of 0.010" wire is adapted to the cingula and apical to the contact points, looped about the teeth to be included, and the ends twisted together. Then, 0.007" wire is placed interproximally around both facial and lingual wires and twisted. The adjacent interproximal wire should be placed before final tightening. This is preferred to the figure-of-eight wiring which may cause binding and overlapping of loose teeth. If teeth are spaced, the facial and lingual wires can be braided to span the space.

This splint traps food, has poor esthetics, requires frequent adjustment, and irritation from the sharp twisted ends of wire may be a problem. Even though meticulously applied, prevention of apical slippage of the wire with resultant loosening of this splint is almost impossible in normally shaped teeth. In the case of conical teeth, the wire tends to slip incisally. These problems can be reduced and the splint better stabilized if self-curing acrylic is painted over the wire. Care must be taken when applying this splint to avoid occlusal interferences and obstruction of oral physiotherapy.

C. Orthodontic Band Splint. This splint, quite rigid, fairly easily maintained and non-irritating to cheeks and lips, is especially useful in posterior areas.

Use 0.005" band material of a width suitable for the crown length of the case at hand. Join two strips at their

midpoints by spot welding. Adapt the bands to the tooth and spot-weld the seams. Additional bands can be added by first spot-welding the new piece of band material to the contact of an already-formed band and then adapting the band to its tooth and spot-welding the seam. If two pieces of band material will not pass through the contact area, a modification can be adopted where band material is spot-welded to an already-formed band at the buccal and lingual embrasures, then adapted to its tooth and the seam spot-welded.

Sorrin's text contains an excellent description of the technic for orthodontic band splints. Cementation is done with any cement used for orthodontic appliances; i.e., germacidal cryptex or red copper cement.

D. Modification of Silk, Wire Ligature and Orthodontic Band Splints with Self-Curing Acrylic Resin: This modification of the technic gives added rigidity, better esthetics, and is less irritating. A width of approximately 3 mm. of acrylic is applied in a half-round fashion to minimize food retention. Occlusal and incisal wire loops can be added temporarily to prevent slippage during application of the plastic. The acrylic can be trimmed and smoothed when set.

E. Intradental Reinforced Acrylic Splint: Originally developed by Obin and Arvins, this technic is advocated where esthetics, tooth contour, contact and/or occlusion preclude extra coronal splints. It is usually used where a future permanent splint is planned.

An undercut channel is prepared in the occlusal surface. A 3 mm. width and 1 mm. depth were originally recommended, but the width is not too critical since the splint is to be temporary. A piece of twisted stainless steel wire is adapted to the channel and self-curing acrylic resin is painted into it. A round finishing bur is used to trim the acrylic to proper occlusion and contour.

This splint can also be used for anterior teeth by cutting a lingual undercut channel the width of a #559 bur, 1 to 1.5 mm. deep, and following the same procedure as for the posterior teeth.

Shatzkin and Edelman use this splint as a "semi-permanent" splint by preparing the proximals of the anteriors involved similar to Class III cavities in addition to the

lingual channel and dovetailing the lingual channels of the terminal teeth. The wire is bent so as to enter the proximal preparations. Some of these had been in service seven years without replacement according to these authors.

F. Removable Cast Continuous Clasp Appliance: A casting of this type in gold or chrome-cobalt alloy can be used as a temporary or permanent splint. Except for the tips of retentive clasps, it must be kept in the supra-bulge area to prevent moving of teeth during insertion and removal. This may be difficult due to occlusal interference. It can be used to replace missing teeth and act as a partial denture as well as a splint.

G. Acrylic Continuous Splint: Sorrin has described a splint similar to the removable cast splint, but made of 20 gauge stainless steel wire adapted above the cingulum of anteriors and above the height of contour of posterior teeth and covered with heat-cured acrylic. The splint is waxed up on occluded models so that occlusion will not be disturbed, then it is flasked, packed, and cured. The finished splint can be worn as a removable or cemented splint for short periods of time. It does not have the rigidity of the cast removable splint.

H. Friedman Cast Continuous Clasp Splint: This splint is cast with one open end at which ½-mm holes are drilled through both facial and lingual bars to permit wiring at an interproximal area. The splint is made half in the supra bulge area and half in the infra bulge area and is cemented to place and closed with the wire.

III. PROVISIONAL SPLINTS:

Provisional splints can take many forms.

A. Temporarily cemented gold castings can be used with the view that if the questionable tooth has to be extracted, a pontic can be substituted for its crown. If it is determined that the questionable tooth can be retained, the casting can then be used as a permanent splint. In either event, the casting can be utilized as a permanent appliance.

B. Acrylic Crown Splints: These can be used in cases where a permanent splint or bridge will be an eventual necessity, They permit maximum support, orientation of occlusal stresses, and correction of tooth contour. There are numerous modifications of the acrylic crown splint.

Amsterdam and Fox have reported a technic in which they utilize metal bands and acrylic resin. They feel these splints should end just occlusal to the gingival margin; in this way it protects the exposed cementum and still does not encroach upon the gingiva. They emphasize the importance of proper embrasures occluso-gingivally and bucco-lingually. With gingival recession, the facial and lingual bulges must be placed more apically and the contacts broadened occluso-gingivally. A direct technic, it enables the provisional splint to be placed at the same sitting as cutting the crown preparations.

Briefly, an elastic impression is made and filled with tooth-colored acrylic. When the acrylic is hard, it is removed from the impression and each crown is hollowed out to a thin shell. The preparations are cut and gold or copper bands are adapted to each tooth. These are cut down so that 1 mm. of metal is left on the facial surface and 4 mm. on the other surfaces. The occlusal edge of the band is notched and if copper bands are used, they are flash-plated with gold. Dry foil is adapted to protect the gingiva and the bands are seated. Doughy self-curing acrylic is then placed into the crown shells and the whole is inserted with the patient then occluding his teeth. The new acrylic is allowed to set and the appliance is removed, and acrylic added to the margin of the festooned band. The appliance is then trimmed, contoured, polished and checked for seat, contour, and occlusion prior to cementation. Missing teeth can be supplied if necessary.

IV. PERMANENT SPLINTS:

Permanent splints can be made of numerous materials by many technics. Since splinting is in the realm of occlusal rehabilitation, the conventional types of retainers used in occlusal rehabilitation are logically the first ones to consider. The veneered gold crown and conventional three-quarter crown are popular retainers for splints. Less conventional pinlays, pinledges, and modifications of these have been proposed in various technics. Recently, reinforced amalgam splinting technics have been reported.

Attention should be directed to specific factors involved in avoiding gingival injury and assuring gingival health when splints are to be used.

Tooth position. Slight modification of original arch relationships due to migration can usually be corrected by occlusal adjustment; however, severe pathologic migration is

usually not amenable to equilibration and attempts to prevent further migration by splinting or rehabilitation without first returning the teeth to their original positions by orthodontic measures usually results in failure. The gingiva near malposed teeth is especially vulnerable to recurrent disease because of retention of debris. Sometimes reshaping the crown of a malposed tooth by prosthetic means without changing the root position (i.e. "warping") can create gingival abnormalities.

Tooth preparation in relation to the gingival margin. It is axiomatic that teeth should be prepared in an environment of gingival health. Inflammation and pockets should be eliminated before tooth preparation. Waerhaug says the margin of a restoration in a caries-prone individual must terminate in the gingival sulcus while in a noncaries-prone, periodontal disease prone individual it should terminate at, or slightly coronal to, the gingival margin with modification of the latter rule necessary where esthetics is a prime consideration. Glickman and Stein state the margin should be at the coronal most level of the epithelial attachment if it is to be carried into the gingival third of the tooth.

Temporary coverage. The same need for meticulous protection of the gingiva when using permanent coverage applies to temporary coverage of abutments prepared with margins at the bottom of the sulcus.

Occlusal form. Occlusal surfaces should direct functional forces in the long axis of each tooth. A shallow modified cuspal carving that permits freedom in all functional mandibular movements usually suffices.

Buccal and lingual contours. Three-quarter crowns, inlays, pinlays, and pinledge preparations usually leave sufficient natural tooth structure to provide a guide to wax up. Full coverage requires exacting establishment of crown form which is dictated by gingival relationship. Over-contouring or under-contouring causes a disruption in normal food deflection and the result is accumulation and impaction respectively.

Interproximal contours. Adjacent teeth should be contoured and contacted to prevent food impaction and/or retention.

Pontic supplies. Ridge contact should be kept to a minimum. Either sanitary or bullet-shaped pontics in the posterior and modified ridge lap design in the anterior where esthetics is a factor are recommended.

A. FULL COVERAGE GOLD CASTINGS:

The full crown is a very valuable restoration and, when indicated, satisfies requirements which are met by no other restoration; however, even when ideally constructed, it introduces a greater risk of gingival disease than any other type of restoration. It substitutes foreign material for the natural tooth wall of the gingival sulcus which fosters accumulation of debris and inflammation. Waerhaug and Stein have shown the bacterial flora of the gingival sulcus is altered in the presence of crowns regardless of the material used. The technics for utilizing full crowns for splints are amply covered in texts and journals.

A modified splinting technic using full coverage has recently been reported by Prichard and Fedder. It utilizes telescopic crowns in which the copings are constructed with parallelism and the telescopic super-structure is seated on them. This method preserves tooth structure which would otherwise be sacrificed to obtain parallel abutments. The copings are permanently cemented and the superstructure is temporarily cemented.

In this way, the coping margins can be very accurately adapted, the hermetic seal of the abutment and coping is never lost, as it can be when one piece crowns are temporarily cemented. The tissue under pontics can be observed, modifications made in the superstructure if needed, new teeth can be included in the splint at any time, and teeth with a doubtful prognosis can be watched and if extraction is later indicated, the crown can be changed to a pontic without sacrifice of the entire splint.

Another feature of this splint is that by soldering the copings in segments and overlapping short segments of superstructure from one segment of copings to another, the entire arch can be splinted without establishing parallelism of all teeth. The abutments for each segment of copings must be parallel and the copings for each segment of superstructure are parallel. This permits splinting in cases where total parallelism is impossible or impractical. Care must be taken, however, not to incorporate the ill effects of "warping" into the splint.

B. PARTIAL COVERAGE GOLD CASTINGS:

Shooshan and others have deplored the wanton cutting

of teeth for full coverage with subsequent poor restorations, and advocate the use of MOD onlays, 3/4 crowns, pinledges and pinlays, and the use of full coverage when other preparations will not suffice. Stein and Glickman state that ideally, preparations should terminate well coronal to gingival margins, avoiding the entire gingival third of the crown where possible.

1. Shoosban has described a technic for pinledge and MOD onlay castings in splinting in which preparation is accomplished free hand with the conventional armamentarium. Special paralleling devices can be used but are not essential. One piece castings are produced--pinledges and pinlay retainers in the anterior and MOD onlays in the posterior--which he claims are very satisfactory in periodontal splinting.

2. Curtis and Baum have reported a pinledge technic for splinting mandibular anterior teeth in which the pontistructor is used for paralleling pin holes and a jeweler's drill in a back action handpiece is used to prepare the pin holes. The pin holes bisect the angle formed by the lingual surfaces and the long axis of the teeth. They place as many pin holes as possible in each abutment (seven in some cuspids) at a depth of 1 to 2½ mm. The margins of the lingual preparations are clearly defined and the incisal edges may or may not be involved. The application of this technic is limited to the well-aligned abutments.

Sarrell and Friedman offer a gold casting in splinting lower anterior teeth which uses one horizontal pin per tooth. This pin perforates the labial surface incisal to the pulp. No lingual preparation is required.

The type C pontostructor is used to make the parallel pinholes from the labial 1/3rd the distance from the pulp chamber to the incisal edge as determined from radiographs.

The casting is made in hard gold with irridio-platinum pins. The originators claim that by altering the path of insertion, lapped teeth are often amenable to this technic. Maxillary anteriors can be splinted by this method if sufficient clearance can be attained without excessive removal of tooth structure (i.e. edge-to-edge bites, cases where bite can be

opened 1 mm., open anterior bites). In upper splints the incisal edges are involved to preclude dislogment during function.

The technic can be modified and used in cases where nonparallel pins might be necessary, by casting a lock to a pin which fits into a key on the lingual casting. This same type of device can be used to connect an anterior splint to a posterior splint to provide "around-the arch" splinting.

Esthetics can be improved by removing a small amount of the pin on the labial and placing a tooth-colored restoration.

Contraindications include:

1. Insufficient dentin to securely anchor pin.

2. Insufficient dentin between pulp and incisal enamel.

3. Insufficient labial to lingual width of tooth (3 mm. pin length is minimum.).

C. QUASI-FIXED PERMANENT BAR SPLINTS: Overby has devised a cast bar splint which is fastened directly to the natural teeth by the use of screws and sleeves and requires a minimum of tooth preparation.

An investment cast is made from a hydrocolloid impression. A #1 round bur is used to prepare holes in the cast in the cingula of anterior teeth (parallel to the long axis) and the mesiolingual of posterior teeth (horizontal perpendicular to the long axis). Special screws are placed in these holes and a bar is waxed up occlusal to the height of contour. (If occlusion is a problem, the height of contour can be lowered by narrowing the occlusal tables of the natural teeth). The screws are removed and the bar is cast in gold or chrome-cobalt.

The casting is seated in the mouth and holes are drilled with a #557 bur 2½ mm. deep through the holes in the casting. Special screws and sleeves are then cemented into these holes so that the bar is held tightly against the tooth. When the cement around the sleeves is hard, the screws are removed, the excess cement cleaned off, and the screws replaced.

Minor tooth movement can be accomplished by repositioning the tooth on the cast, waxing the bar to the new position and after the bar is inserted, drawing the tooth to it with an elastic.

The sleeves can be put on the occlusal surface of posteriors by preparing a 1 mm-deep occlusal seat prior to the impression.

This splint can be used to supply missing teeth and the bar can be removed by the dentist for cleaning; however, the patient still must be scrupulous in his oral hygiene.

Overby has recently suggested a modification of this splint in which the abutments are restored with gold castings of varying types and the bar is fastened to these castings with screws.

D. REINFORCED AMALGAM SPLINT:

Lloyd and Baer reported a permanent, fixed, intracoronal splint in 1959 and Alloy reported a variation of their technic in 1962. The amalgam splint is still being evaluated, and despite reinforcement, fracture of the amalgam at the axio-pulpal line angle does occur. This disadvantage was expressed by the first reporter of an amalgam splinting technic, W. G. Cross in 1954.

Amalgam splints are usually employed in cases of slight mobility wherein proximal contacts can be opened with resultant food impaction. They are contraindicated (1) where they would have to form long interproximal bridges, (2) where the splint would oppose very firm antagonists, (3) in patients with clenching or bruxing habits.

This type of splint cannot be used for anterior teeth because sufficient material to prevent interproximal fracture could not be used. It is basically a unilateral splint, but can be used in conjunction with a removable cast extracoronal metal splint to provide around the arch stabilization.

The reported technics of amalgam splinting differ but slightly. The one sitting technic developed at Walter Reed Hospital by Major Parks Paul will be described. The other technics differ mainly in the reinforcing material and in the use of an indirect method for fabricating the matrix as opposed to the direct technic described here.

1. Prior to administration of anesthesia, adapt self-curing acrylic (doughy consistency) to labial and lingual of the teeth to be splinted, making sure that acrylic fills the interproximal area.

2. Remove labial and lingual sections just after initial set; cool with water if necessary.

3. Matrices are trimmed, checked for adaptation and put aside.

4. The cavity preparations should conform to all the requirements of good Class II amalgam restorations. Make the occlusal floor wide, deep and even.

5. The proximal step should be extended bucco-lingually to self-cleansing areas and should have a well-formed gingival floor. This will give a wide occluso-apical and bucco-lingual cavity preparation and insure good bulk of amalgam in these areas.

6. Replace the acrylic matrices and maintain them in place securely with a #1 ivory matrix retainer or a rubber dam clamp or both if the teeth are very mobile; the matrix can serve to stabilize the teeth during placement of amalgam and prevent fracture during condensation.

7. With a cylindrical stone, trim imterproximal areas of matrix to conform to the tooth preparations.

8. Stainless steel wire .01 gauge is doubled and twisted and cut to proper length to fit occlusal portion of preparations.

9. Mix the amalgam. Pack interproximal areas first to height of occlusal step. Place steel wire into preparation. (It can be held down with a serrated plugger). Finish packing amalgam. Use amalgam condenser if available.

10. In a large amalgam splint, use a plastic mix of amalgam and work rapidly to prevent initial set

of amalgam before the condensation is completed.

11. After initial set, remove matrices carefully and carve the restorations. Remove any occlusal prematurities at this time to prevent fractures. Check for overhang and excess along margins.

12. Patient is cautioned against chewing on side of splint for 24 hours and he is then dismissed.

13. At next sitting, splint is polished and checked for occlusal prematurities again. Contra-angle #8 round burs and round and tapered amalgam finishing burs will complete most of the polishing.

E. SPECIAL PURPOSE REMOVABLE SPLINTS:

1. The Hawley retainer can be used in cases of moderate periodontal involvement in which teeth have been repositioned. More of a retainer than a splint even in periodontal cases, it is usually worn at night on a more-or-less permenant basis.

2. Night (bite) guards are used in cases of bruxing or clenching and grinding habits. They spread the forces transmitted to the teeth in parafunction over the whole arch regardless of mandibular position and eliminate the tipping forces on inclined planes. Sorrin advises covering the arch showing the greater amount of mobility. Both arches may be covered if desired. This appliance will not cause eradication of the parafunctional habit which is the more desirable approach in these cases.

V. SUMMARY:

Each of the numerous methods of splinting has its own inherent strengths and weaknesses. There are also advantages and disadvantages applicable to splinting in general. There are precautions to be observed in the construction of splints lest they ultimately defeat the purpose for which they were made. Splinting is a valuable adjunct in periodontal therapy when used intelligently, but splints will be no better than the clinical judgment governing the selection of technic and teeth to be included.

The strengths and weaknesses of the various technics have been included in their discussions. Some of the disadvantages of splinting in general include: (1) the mechanical shortcomings of operative dentistry, (2) interference with home care and the fostering of debris retention, (3) interference with continuous eruption and normal mesial migration, and (4) possible overloading of opposing teeth. One must be very cautious to plan and construct splints so that these disadvantages can be minimized.

Splinting also permits the temporary retention of hopelessly involved teeth by permitting them to remain relatively symptom free despite deep pockets and even frank suppuration. The possible hazard of acute periodontal abscesses and probable undue loss of bone from adjacent teeth (which may be most strategic) should be obvious. This practice, which can easily jeopardize the patient's entire dentition and possibly his general health as well, should be guarded against in all cases employing splints.

If we keep in mind that the purpose of periodontics is (1) to maintain and (2) to restore, if necessary, the health of the supporting structures of entire dentitions and not the heroic salvation of individual teeth, then splinting will fall into its proper perspective.

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LECTURE NO. 18

CALCULUS

Samuel Kobrinsky*

I. INTRODUCTION:

It is generally agreed that gingival deposits and calculus in particular constitute the most important local etiologic factor in the causation of periodontal disease. This finding is substantiated by both clinical and experimental evidence. However, the precise mechanism by which calculus produces periodontal disease is poorly understood and, in fact, little is understood about the mechanism of calculus formation.

II. COMPOSITION:

Calculus is classified according to its position in relation to the gingival margin as supra- and subgingival, or salivary and serumal.

A. SUPRA- AND SUBGINGIVAL:

Supragingival calculus is generally light in color, is not firmly attached and may be easily removed from the teeth; is located on the teeth in close proximity to the major salivary gland ducts. The two areas in which supragingival calculus tends to occur with greatest frequency are: (1) On the buccal of the maxillary molars opposite Stensen's duct of the parotid gland, and (2) on the lingual of the mandibular incisors opposite the orifices of Wharton's and Bartholin's ducts of the sublingual and submaxillary glands.

Subgingival calculus varies in color from dark brown or green to black. It is firmly attached to the teeth beneath the gingival margin and is removed only with great difficulty. It may be found on the dentition in any area of the mouth.

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B. SALIVARY AND SERUMAL CALCULUS:

Calculus is often differentiated into salivary and serumal (subgingival) with the assumption that the supragingival calculus is a salivary deposit and subgingival calculus is a deposit from the blood stream. A more recent feeling is that the saliva is the source of both calculus types. Hodge and Leung show that with the exception of water and organic material, the composition of both is the same.

Jens Waerhaug has recently revived the controversy with the suggestion that mineral salts in the subgingival calculus may be derived from the blood serum rather than from the saliva.

C. CHEMICAL COMPOSITION:

The chemical analyses of calculus vary widely, depending on the age of the calculus and the amount of food and bacteria incorporated in it. Generally, calculus deposits are composed of 67% calcium phosphate, 8% calcium carbonate, 1.5% calcium fluoride, 1% magnesium phosphate, and about 22% water and organic matter. Other elements found in trace amounts are Na, K, Cl, Fe, SO4, and SiO2.

Chemical and X-ray diffraction studies have shown that calculus consists primarily of calcium phosphate arranged in a hydroxy-apatite crystal lattice structure similar to bone, enamel, and dentine. Small amounts of brushite and whitlockite crystals have been found in calculus. In some instances calculus is found to be stratified with non-mineralized material alternating with mineralized material.

In addition to its inorganic chemical components calculus contains an organic ground substance which consists of mucus, food debris, bacteria, fungi, leukocytes, and desquamated epithelial cells.

The bacterial content of calculus is important both in relation to the mechanism for calculus formation and as an etiologic factor in periodontal disease. In assaying bacterial content, Yardeni divided calculus into three zones:

1. Calculus mass - here he found great numbers of gram-positive filaments of the actinomyces type.

There were also some gram-positive threads which could be identified as leptotrichia. There were numerous borrelia. There were few gram-negative cocci.

2. The internal surface of the calculus was relatively sterile.

3. A zone encircling and adjacent to the calculus contained primarily gram-negative cocci and rods.

III. FORMATION OF CALCULUS:

A. ATTACHMENT OF CALCULUS:

Zander describes four methods by which calculus may attach to the root surface.

1. A relatively loose attachment by means of the secondary cuticle.

2. Attachment to microscopic irregularities in the cemental surface.

3. Attachment by penetration of bacteria to various depths of the cementum.

4. Attachment to area of cemental resorption.

It is probably a combination of two or more of the above methods which is the means by which calculus attaches to the root surface.

B. MECHANISM OF CALCULUS FORMATION:

There are several main theories for the mechanism of calculus formation.

1. Bacterial Theory.

Microorganisms in the course of their metabolism produce environmental changes leading to a deposition of calcium salts. The actinomyces and leptothrix organisms were found to increase the alkalinity of saliva which reduces the solubility of the dissolved calcium salts. The organisms also break

down the colloidal protein necessary to keep the calcium salts suspended in solution. The production of the enzyme phosphatase by these organisms is believed to cause this precipitation of calcium salts.

Mandel proposes a supplement to the bacterial enzyme theory. He points out that the bacterial cell walls are composed of 25% mucopolysaccharide and may act in the formation of calculus by virtue of chemical composition rather than enzyme production. Calculus formation may be initiated by carbo-proteins which chelate calcium which, in turn, acts as an epitaxy nucleus.

Evidence of the role of bacteria in calculus formation is based on the following:

a. When living actinomyces organisms were introduced into sterile saliva, calcareous masses which resembled calculus were precipitated.

b. When the experiment was repeated with nonviable organisms, there was no precipitation.

c. Filamentous organisms are always associated with in vivo salivary calculus.

d. The actinomyces have been shown to contain the enzyme phosphatase which is capable of liberating phosphate ions from organic phosphate.

Mandel describes the following sequence of calculus formation in humans:

Day 3-5. Formation of a plaque consisting of an amorphous or finely granular organic matrix containing masses of a variety of gram-positive and negative coccoid bacteria and occasional filamentous forms. The matrix is a mucopolysaccharide derived from saliva or bacteria.

Day 7. Coccoid bacteria are present but the surface and central portions contain many filamentous organisms.

Day 12. At this stage, the plaque now contains almost solely gram-positive and negative filamentous organisms.

Day 14. Evidence of calcification has occurred in all cases in numerous separate foci.

Stanley describes calculus formation as part of a "cyclic phenomenon of periodontitis." Microorganisms, food debris, desquamated cells, and saliva collect as a soft mass in the interproximal areas of the teeth. This mass causes a gingival inflammation and pocket ulceration, and exudate is released which combines with the mass. This mixture then forms the matrix in which calcification and the formation of subgingival calculus occurs. The pressure of the adjoining soft tissue forms the calculus into a scale. After calcification, the process is temporarily arrested but the entire cycle is soon repeated with an apical shift of the pocket and what was subgingival calculus becomes supragingival.

2. Physico-chemical Theories:

a. The carbon dioxide loss theory.

Hodge and Leung explain calculus formation on the basis of a loss of CO2 from the saliva and a subsequent rise in the pH. When saliva is secreted, it contains CO_2 at a tension of 54-65 mm. of mercury. The CO_2 tension in the atmosphere is only 0.3 mm. of mercury so CO_2 escapes from the saliva. The pH of saliva depends on the ratio between free and combined carbonic acid and is elevated when the CO_2 is lost. With increased alkalinity a dissociation of phosphoric acid occurs which liberates phosphate ions. When the phosphate ion concentration exceeds the solubility product of hydroxyapatite, the salts precipitate from solution.

Another possibility is that desquamated epithelial cells which produce the enzyme phosphatase may induce deposition of calcium phosphate from the saliva.

b. Glimsher has proposed an epitaxy or seeding principal. There is formed on the tooth a template of organic material whose structure is similar to the apatite structure which acts as a seeding mechanism and this draws down the inorganic apatite on to the template. Epitaxy refers to the process whereby a solid

phase can be precipitated from a supersaturated solution by some pre-existing solid substrate. It requires that the atomic configuration on the surface of the substrate be similar in some respects to the basic atomic configuration or crystal lattice in the material to be precipitated. One of the best examples of the phenomenon of epitaxy, outside of the biological field, can be demonstrated in the so-called cloud seeding effects, using potassium iodide. The crystal lattice of potassium iodide resembles ice; that is, the potassium and iodine atoms are arranged in space in a manner similar to the hydrogen and oxygen atoms in the solid phase of water. When potassium iodide crystals are introduced into an atmosphere supersaturated with respect to water, a seeding effect takes place; that is, the water in the gas phase is precipitated out on these tiny potassium iodide "seeds," forming small crystals of snow or ice.

In the body, it has been proposed that collagen and other proteins, like mucopolysaccharides, act as a seeding material for the supersaturated fluids. Mucopolysaccharide is composed of long chains of polypeptides connected in segmented units. These units are called macromolecules. Apparently, the configuration of certain chemical groups on these polypeptide chains are similar to those found in hydroxyapatite. As a result, mucopolysaccharide acts as a seeding material which induces the formation of the solid hydroxyapatite phase. It is significant that formation of hydroxyapatite crystals is never seen in the body in the absence of this protein or some other protein material.

c. Another theory is that calcification may occur as an absorption phenomenon in which calcium salts are precipitated in the mouth about colloid precipitates or desquamated epithelial cells which carry electric charges.

d. Rapp suggests that a slight rise in the pH would cause precipitation of insoluble phosphate

salts from the saliva of calculus--susceptible individuals whose saliva is supersaturated with calcium and phosphate.

Several authors have incriminated free ammonia in the saliva changing soluble calcium salts into less soluble salts. They feel that ammonia combines with CO_2 and sets free insoluble calcium phosphates.

C. SYSTEMIC AND DIETARY FACTORS:

Various systemic and dietary factors have been related to the formation of calculus.

1. A soft diet increases calculus production.

2. Diets containing fibrous foods seem to inhibit calculus formation, probably due to their detergent action.

3. Vitamin A and D deficiencies caused severe calculus formation in experimental animals.

4. Emotional stress has been implicated in calculus formation by causing disturbances in the phosphorus calcium balance.

5. Vitamin C deficiency causes the tissues to become soft and spongy and to bleed easily. Desquamation of the buccal mucosa occurs. This material, augmented by food remnants, forms a necrotic mass which, when deposited in the interdental spaces, provides an excellent nidus for ubiquitous microorganisms, pyogenic and mycelial. This conglomerate supplies the matrix or the nucleus for the deposition of calcareous material.

6. It was observed by one investigator that tobacco users are more prone to calculus formation in direct ratio to the amounts of tobacco used.

IV. OTHER DENTAL DEPOSITS:

In addition to calculus, other materials occur on the tooth surface. These may be pigmented or nonpigmented.

A. MUCIN:

An organic film composed of mucinous material from the saliva. The film may contain bacteria, fungi, food debris, desquamated epithelial cells. The film may become pigmented by food pigments or the action of chromogenic bacteria.

B. MATERIA ALBA:

A white or greyish deposit usually found at the gingival margins and interproximally. It consists primarily of bacteria and mycotic organisms. Marginal erythema is commonly found associated with materia alba.

Beckwith and Williams destroyed the bacterial component of materia alba by heat and then injected the material in experimental animals. Redness and swelling in the injection area suggests the possibility that toxic products of materia alba may be absorbed thrcugh the gingival epithelium in humans and result in localized inflammation.

D. BROWN STAIN:

Found predominantly on the lingual of the mandibular incisors and on the buccal of the maxillary molars suggests a relationship to an organic component of the saliva. It shows staining properties similar to keratin.

V. THE RELATION OF CALCULUS TO PERIODONTAL DISEASE:

Calculus by virtue of its physical hardness is thought by some to act primarily as a mechanical traumatic agent. Other men feel that tissue irritation caused by microorganisms and their toxins overshadow the mechanical irritation of calculus.

A. PHYSICAL THEORY:

1. King and Martin maintain that the influence of the oral microflora is secondary to irritation and traumatic injury to the gum surfaces. They state that "in the absence of nutritional deficiencies, retention of food debris about the teeth and dental deposits of salivary calculus comprise the main causative agents of the common nonulcerative forms of gingival disease."

2. Again, the finding of Buer and Newton that germ-free mice are subject to a form of periodontal disease associated with calculus-like deposits suggests that mechanical rather than chemical irritation is the prime factor.

B. CHEMICAL THEORY:

There are those, however, who feel that mechanical irritation plays but a minor role in the etiology of periodontal disease.

1. The chemical concept of calculus formation is supported by animal studies of Mitchell and Johnson, and Koenig and Michlmann, who point out that uncalcified gingival plaques may lead to gingival inflammation, gingival recession, and pocket formation. However, this does not necessarily mean that these plaques are not causing mechanical irritation. In fact, a common finding was the presence of impacted food, hair, and other debris within the plaques.

2. Waerhaug states that tissue irritation is caused by bacteria and their toxins and that whether the deposits are calcified, like calculus, or uncalcified, like plaque, makes little difference since microorganisms are present in both types of deposits.

3. Schultz-Handt, Dewar, and Bibby found that Mechanical irritation need not necessarily precede bacterial invasion.

   They report that hyaluronidase found in gingival debris and in gingival bacteria, is able to change or even remove the intercellular substance of crevicular epithelium.

VI. CONCLUSIONS:

There is some contention as to which state comes first: The formation of the pocket, or the formation of calculus. Glickman feels that pocket formation and calculus deposition are associated conditions. However, he does state that the initial deposition of calculus stimulates inflammatory changes which lead to pocket formation. The subsequent deposition of subgingival calculus then is a passive process which is fostered by the environment created by the pocket.

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LECTURE NO. 19

MICROBIOLOGY OF PERIODONTAL DISEASE

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I. INTRODUCTION:

The oral microbial flora is one of the most varied to be found in or on the human body. It is also one of the most dense of the body floras. According to MacDonald, Gibbons, and Socransky, the bacterial populations in close approximation to mucosal surfaces of the periodontium reach numbers at least as high as 10^{10} per gram of materia alba.

The mouth is the first mucous membrane area to develop a microbiota after birth. In utero, the oral cavity of the developing child is ordinarily germ free. The first microorganisms that the oral cavity comes in contact with are those of the mother's genital tract; such as some species of lactobacilli, corynebacterium, staphlococci, streptococci, yeasts, protozoa, and viruses. Shortly after birth, the oral cavity comes in contact with microorganisms of the external environment and within a short time has acquired a characteristic flora of the type that will inhabit it, with slight changes during the individual's lifetime.

The microorganisms involved in periodontal disease are generally those which are normal inhabitants of the oral cavity. An understanding of the microorganisms of the oral cavity and their mechanisms of action is essential to understanding the microbiology of periodontal disease.

II. MICROBIOTA INDIGENOUS TO THE ORAL CAVITY:

A. Types of organisms:

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1. Oral Streptococci:

The streptococci are the most common and abundant group of microorganisms present in the oral cavity. The aerobic viridans streptococci are the most abundant. Whether or not anaerobic streptococci are normal inhabitants is a matter of controversy. There are many more anaerobic microorganisms present in the dentate state than in the edentulous condition; this is true of all microorganisms not just the streptococci. The anaerobic streptococci which inhabit the oral cavity are potential pathogens as they are often isolated from such infections as Ludwigs' angina, sinusitis, pulmonary abscesses, and others.

2. Oral Lactobacilli:

The lactobacilli have often been implicated in the process of dental caries. Present day knowledge does not ascertain that they are the causative organisms of dental caries; however, they are characteristic of the oral region and seem to be more prominent in a caries prone mouth than in a caries free mouth.

3. Oral Filamentous Microorganisms:

Generally the filamentous organisms are divided into two groups:

a. Actinomyces --in large numbers may produce actinomycosis.

b. Leptotrichia--these organisms have been related to plaque and calculus formation.

4. Oral Spirochetes:

These most commonly inhabit the gingival crevices and interproximal areas and are greatly reduced in numbers in the infant and edentulous adult. Their pathogenicity seems to be questionable as pure cultures do not cause a characteristic or transferable infection although they may be a part of a mixed infection.

5. Bacteroides:

These are difficult to culture and are often associated with the spirochetes in the gingival sulcus. The fusobacterium plauti-vincenti and fusobacterium nucleatum are two species often associated with fusospirochetal disease.

6. Oral Vibrios:

Species of the genus vibrio have a wide range of activity, decomposing cellulose and causing several diseases. It has been stated that the oral vibrios increase in number during periodontal disease. (Burnett)

7. Oral Yeasts:

Candida albicans is the only pathogenic yeast common to the oral cavity. It causes candidiasis (thrush) under debilitating conditions. Although other species of yeasts present in the oral cavity have not been found to be pathogenic, they are an abundant source of vitamins and growth factors and they may stimulate the growth and activity of other oral microorganisms.

8. Oral Corynebacterium:

These are diptheroid type organisms and while none of them has been proven pathogenic, there is some recent evidence that they may be active in periodontal diseases by virtue of their enzymatic activity. (MacDonald and Gibbons)

9. Viruses:

Herpes virus homonis is consistently found as an inhabitant of the cells of the oral mucous membranes of most adults. Other viruses have been isolated from the oral cavity, however, due to the difficulty of culturing viruses, information is still lacking as to the true role of viruses in oral infections and periodontal disease.

10. Oral Protozoa:

Endamoeba gingivalis is fairly prevalent in mouths with periodontal lesions and gingival pockets. It is found in only 10 per cent of healthy mouths. For this reason, it has been associated with suppurative gingival lesions, although it appears to be an opportunist rather than a causative organism. (Burnett)

11. Miscellaneous Organisms:

Staphlococci, veilonella, PPLO organisms, "L" microorganisms and others are commonly found in the oral cavity. It has recently been postulated that the "L" organisms may be responsible for recurrent types of conditions such as recurrent apthous stomatitis.

B. Factors Regulating the Oral Microbiota:

1. Topography of the environment:

In infancy and after the teeth are lost, the aerobic types of microorganisms predominate. When the teeth are present, numerous areas are available where a degree of anaerobiosis can be maintained. These areas are the interproximal surfaces, the gingival sulci, and enamel pits and fissures.

2. Oral Hygiene:

With good oral hygiene, the total number of microorganisms decreases and is predominately aerobic. Neglect of oral hygiene results in an increase of the total flora and an anaerobic and putrefactive character probably owing to the accumulation of food and tissue debris in the gingival sulcus and to increased plaque formation.

3. Saliva:

The most important regulating mechanism of the oral flora is the saliva. Saliva is the culture medium in which the oral microorganisms live, grow,

reproduce, and carry out their various functions. Important factors to consider in the regulation of the oral microbiota regarding saliva are:

a. Nutritional elements present in the saliva.

The exact content of amino acids, carbohydrates, minerals and other elements vary from the saliva of one individual to another. Certain microorganisms are very fastideous in their nutritional requirements and the lack of one element may make the environment unsuitable for their maintenance.

b. Rate of flow.

The rate of flow and total amount of saliva excreted vary considerably and directly influence the composition of the saliva. Xerostomia is typically accompanied by a general increase of the oral microflora due to the decrease in solvent and cleansing action of the secretion.

c. pH.

The pH of saliva greatly influences the oral microflora qualitatively as most microorganisms are quite exacting in their requirements of acid or alkaline environments.

d. Growth inhibiting and accelerating substances present in saliva.

Substances such as ammonia, urea, enzymes such as amylase, catalase, and lysozyme, and other substances have been found to accelerate or inhibit the growth of microorganisms by either their direct action on the microorganisms or by their action on nutrient products making themmore available to the microorganisms.

III. MECHANISMS OF DISEASE:

A. "Bacteria do not produce disease simply by their physical presence, although their adequate multiplication is essential to

infection. To produce disease, some constituent or product of the bacteria must react with the tissue or cells, to destroy them or to interfere with their normal functioning." (Burnett)

B. Substances produced by microorganisms or are an integral part of the microorganism that are detrimental to cells or tissues.

1. Exotoxins.

Produced mainly by gram-positive pathogenic bacteria. These are very lethal in nature and diffuse away from the organisms that produce them. Exotoxins act by interfering with some metabolic process essential to cellular life.

2. Endotoxins.

These are intimately associated with the cell wall of most gram-negative bacteria and are liberated upon autolysis. Endotoxins operate through their action on the vascular bed or by tissue destruction.

3. Extracellular enzymes.

Substances produced by bacteria which are not directly toxic but play a role in the infectious process.

a. Collagenase.

Proteolytic enzyme capable of disintegrating collagen.

b. Coagulase.

An enzyme produced by many pathogenic staphlococci capable of coagulating plasma. This contributes to the formation of fibrin walls and the localization of the infectious process. This walling off also prevents the body defenses from combating the infection.

c. Hyaluronidases.

Enzymes which hydrolyze hyaluronic acid (a constituent of the ground substance of connective tissue) and aid in the spread of infection.

d. Streptokinase.

Produced by many hemolytic streptococci and is able to dissolve coagulated plasma aiding in the spread of the streptococci through tissues.

e. Other enzymes such as hemolysins (dissolve R.B.C's.), leukocidins (lyse W.B.C's.), and lecithinases (splits lecithin, an essential component of cells).

4. Surface components of bacteria.

Some bacterial strains possess substances within their cell walls or upon their surfaces which allow them to withstand phagocytosis. Other strains produce an effect upon the endothelium of blood vessels giving rise to increased permeability and allow wider dissemination of the invading microorganisms.

5. Allergic reactions.

Cells and tissues can become allergic to compounds of bacterial cells, usually proteins. The tissues then react unfavorably to these substances by elliciting the inflammatory response.

C. Mechanisms of host resistance to disease.

1. Physiologic barriers.

The skin and, in the oral cavity, the intact mucous membrane acts as a physical barrier. Also a film of mucous and antibacterial substances in saliva on the surface of the mucous membrane act to prevent the invasion of microorganisms.

2. The inflammatory response.

   Including the liberation of antibacterial substances and phagocytosis.

3. Antibiodies.

   Many of the antibodies are essential to phagocytosis, for if they are not present, phagocytosis cannot occur. Antitoxins are important in neutralizing the toxins produced by microorganisms.

4. Tissue immunity.

   Certain tissues are resistant to specific microorganisms and resist their destructive efforts. This mechanism, while recognized, is not well understood.

IV. <u>THE ROLE OF MICROORGANISMS IN PERIODONTAL DISEASE</u>:

A. The microorganisms involved in periodontal disease are, almost without exception, those which are normal inhabitants of the oral cavity or its mucous membrane. Although the presence of indigenous parasites upon mucous membrane is not incompatible with continued good health, and may even contribute to it, these bacteria can also play a significant part in infection and diseases of the periodontium. (Burnett)

B. The role of microorganisms in ANUG, gingivitis, and periodontitis.

1. Rosebury states "The bacteriological picture of gingivitis, ANUG, and periodontitis is similar and bacteria obtained from all these conditions demonstrate a comparable capacity to produce infection when injected into experimental animals. The difference in the role of bacteria in the three conditions is mainly quantitative and qualitative, but the same basic organisms are involved."

2. The work of many investigators shows an increase in the number of spirochetes, vibrios, protozoans, and fusiforms in periodontal diseases. However, experimental treatment of periodontal diseases with antimicrobial substances has shown only temporary cessation of the disease.

C. Microorganisms as a primary factor in the etiology of periodontal disease.

1. The role of bacteria as a primary factor has largely been based on animal research and while some of the results are interesting there is no conclusive evidence that microorganisms alone produce periodontal disease. Scherp states: "An essential role in periodontal disease is generally attributed to the gingival crevicular microbiota, but the pathogenicity of the individual microorganisms and their mechanisms of action are incompletely determined."

2. Experimental lesions have been produced in animals by the subcutaneous injections of purulent material obtained from periodontal exudates. This work has been reproduced by many workers, such as Rosebury, MacDonald, Hemmens, etc.

3. Recent work by MacDonald and Gibbons is worthy of mention at this point. They have produced infections in guinea pigs similar to periodontal disease by the introduction of a mixed culture of four organisms. The four organisms are: two bacteroides (one being bacteroides melaninogenicus), a facultative diptheroid, and a gram-negative motile anerobe. Bacteroides melaninogenicus produces significant amounts of ammonia which have the ability to lyse epithelium and could facilitate access to the underlying connective tissue. The same organism also produces a minute quantity of collagenase, which, although not potent in destroying collagen, does destroy it to some extent, and may account for the insideous chronic nature of periodontal disease. All four organisms have the ability to hydrolyze DNA and RNA which have the ability to liquify exudates and may facilitate spread of infection in the periodontal pocket.

D. Microorganisms as a secondary factor in the etiology of periodontal disease.

1. "The gross similarity of the microbial population of the gingival crevice in health and in disease has given rise to the view that the increase of

the crevicular bacteria that accompanies the development of gingivitis, periodontitis, or periodontosis is merely a result of a favorable environment created by unrelated pathological changes." -(Burnett). The residents of the gingival sulcus are not ordinarily pathogenic in their natural habitat; however, factors that decrease the resistance of the tissues creating an opportunity for increased bacterial activity can cause the indigenous parasites of the periodontium to become pathogenic. The resistance of the host to bacterial activity varies greatly and in the oral cavity, local conditions exert important influences upon resistance to microorganisms.

2. Local conditions affecting host resistance.

   a. Local irritants such as calculus, trauma, occlusal disharmonies, etc., cause inflammation in the periodontal tissues. This creates an increase in bulk which in turn creates deeper crevices where anaerobic bacteria can flourish and multiply.

   b. The inflammatory response to local irritants causes vascular stasis altering the nutritional status of the tissues and thus lowers tissue resistance to bacterial invasion.

   c. Traumatic incidents induce localized degenerative changes which increase the tissues susceptibility to injury by bacterial products.

3. Systemic conditions affecting host resistance.

   a. Age.

      In old age, the resistive mechanisms are lowered by a slowing of the metabolic process essential for the production of phagocytic cells and immune substances, specifically antibodies.

   b. Any condition causing malnutrition lowers the host's resistance by an inability to

adequately produce immune substances and phagocytic cells.

c. Any state causing a leukopenia lowers resistance of the host by lessening the availability of phagocytic cells to cope with bacterial proliferation.

d. Altered metabolic states, such as diabetes mellitus, lowers resistance.

4. The crevicular epithelium is thin and not keratinized and, therefore, does not present a formidable barrier against penetration by bacterial products or invasion by bacteria. However, in the healthy gingival sulcus, the positive flow of tissue fluid described by Harvey, Waerhaug, Brill, and others, may help compensate for the lack of barrier presented by the crevicular epithelium. This same tissue fluid contains antibodies and along with polymorphonuclear leukocytes, regularly found in the gingival sulcus, it is possible that phagocytosis could occur in the sulcus. According to Krasse, the gingival pocket fluid contains at least seven different serum proteins and should thus be an excellent medium for more fastidious organisms. Due to the difficulties associated with culturing and identifying the anaerobic flora in the gingival sulcus and the difficulties associated with defining the defense mechanisms present in the sulcus, the intimate host-parasite relationship in this area remains rather obscure and confused.

V. THE ROLE OF MICROORGANISMS IN CALCULUS FORMATION:

A. The first major contribution to the role of bacteria in calculus formation was the work of Naeslund in 1925. Specifically, Naeslund claimed that the actinomyces and leptothrix organisms anchored the calculus to the tooth, provided a stroma for the precipitated calcium salts, and stimulated calcium salt precipitation. This theory, with minor variations, has been relatively well accepted and similar work by Citron, Bulleid, Bibby and others has substantiated the fact that filamentous microorganisms grow well in the environment of the gingival crevice and provide an organic framework, fixing the calcium salts to the tooth.

B. Gnotobiotic contribution to calculus formation.

Fitzgerald and his workers were rather surprised

when, in some of their early germ-free rat experiments, they noted supragingival calculus-like deposits on the maxillary molars. Baer and Newton also reported a similar phenomenon in germ-free mice. Upn X-ray diffraction studies, these deposits demonstrated the typical apatite patterns found in calculus. The extent of the calculus deposits in the germ free animal seldom approaches that seen in the conventional animal. Fitzgerald regards this as an indication that, in the normal non-germ-free state, microorganisms contribute to the severity of the deposits.

C. The role of calculus vs. bacteria in periodontal disease.

1. There are two diverse opinions as to which comes first, the calculus or the bacteria.

2. Glickman states: "Calculus is an irritant which is responsible for the initiation of gingival inflammation. The irritating effect of calculus results from a combination of mechanical, chemical, and bacterial factors. Pocket formation results from the increase in the bulk of the gingival margin. The formed pocket is an ideal area for growth of bacteria and stagnation of saliva and further formation of calculus."

3. Mandel states: "It seems highly possible that the mineralized subgingival deposit is the result, not the cause, of periodontal disease. The mechanical effect of the calculus deposit is secondary to the bacterial effect." Mandel postulates that the bacteria appear first and give rise to inflammatory changes in the soft tissues. He further describes how salts from the tissue fluid and hemorrhagic exudate then mineralize the bacterial-mucoprotein matrix and calculus forms.

VI. CONCLUSION:

The role of microorganisms in periodontal disease is greatly clouded and has not been definitely established. The role of viruses has not been explored to any great degree and this may be an important avenue for future research. Our present day knowledge assumes that periodontal disease is the end result of a complex interplay of multiple etiological factors vs. the resistance of the host.

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LECTURE NO. 20

NUTRITION AND PERIODONTAL DISEASE

Samuel Kobrinsky*

I. INTRODUCTION:

A variety of nutritional deficiencies have been shown to influence the integrity of the various tissues of the periodontium. The maintenance of each component of the periodontium is influenced by one or more acute or chronic states of nutritional deficiency.

II. PERIODONTAL DISEASE AS RELATED TO NUTRITIONAL DISORDERS:

A. CARBOHYDRATES:

Faulty carbohydrate metabolism may result in uncontrolled diabetes mellitus. The acidosis and negative nitrogen balance lower tissue resistance and periodontal disease is a common finding in the diabetic. Animal experiments in which diabetes is induced by the drug alloxan have shown the prime effect to be nonspecific osteoporotic changes in the alveolar bone.

The role of bacteria as an etiologic factor in periodontal disease is well established. There is a direct proportion between carbohydrate intake and bacterial counts in the oral cavity. Carbohydrates are essential nutrients of the oral bacteria because they supply both carbon for cell synthesis and energy for the bacterial cells.

A recent study of the effect of carbohydrates on the soft tissues of the rice rat has led to dramatic conclusions. An experimental group which was fed a carbohydrate-free diet, had 81 per cent less gingival disturbances than the group which was fed a normal diet containing carbohydrate.

B. FATS:

Fat is an important nutrient because of the energy it

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supplies and because it is a source of, and a carrier of, the fat-soluble Vitamins A, D. E, and K, and the essential fatty acids.

Fat has a "protein-sparing" action. If there is enough fat in the diet to supply the body with fuel for energy, it will spare protein (including collagen) from being diverted from tissue production to energy production.

Disturbances in fat metabolism may result in systemic diseases which have periodontal manifestations. These are xanthomatosis (Hand-Schuller-Christian, Letter, Sieve, eosinophilic granuloma) and the non-xanthomatosis (Gancher's disease, Niemann-Pick disease).

C. PROTEIN:

Generally, a protein deficiency may lead to poor growth and reproduction, poor wound healing, increased susceptibility to infection, anemia and edema. Osteoporosis may result from interference with osteoplastic activity. Retardation of fibroplastic activity results in poor wound healing.

Acute and chronic deficiencies of protein have been related to periodontal disease in animals by a number of investigators.

Chawla and Glickman found that protein deprivation in the albino rat resulted in degeneration of the connective tissue components of the gingiva and the periodontal ligament, osteoporosis of the bone, and retardation of the formation of cementum. They found that the osteoporotic changes in the periodontium were comparable to changes occurring in the vertebrae and the femur. The osteoporosis resulted from reduced deposition of ostecid, reduced numbers of osteoblasts, and a retardation in the histo differentiation of connective tissue cells into osteoblasts. Stahl found that when there was a food impaction situation superimposed over a protein deficiency, there was increased resorption of the alveolar crest with an apical shift of the epithelial attachment and an increased inflammatory infiltration.

D. MINERALS:

There are seven principal minerals in the body (calcium, phosphorus, magnesium, sodium potassium and sulfur),

and although they contribute only 2 to 3 percent of the total body weight, they are indispensable for maintaining health. A primary function of these minerals is to import rigidity and strength to the teeth and bones.

1. Calcium and Phosphorus

Deficiencies of calcium and phosphorus as reported by Becks and Weber result in minor inflammatory changes in the epithelium, thickening of the PDM, resorption of the cementum, and osteoporosis of the alveolar bone with wide demineralized osteoid borders. In some cases, obliteration of the lamina dura and the PDM was noted.

An equilibrium exists between calcium and phosphorus in the blood stream, tissue fluid, and the teeth and bones. The metabolism of calcium and phosphorus is inter-related with Vitamin D which regulates the absorption of calcium and phosphorus from the intestinal tract. Deficiencies in the young are referred to as rickets; in the adults, the condition is termed osteomalacia.

Hypervitaminosis D produces hypercalcification of the bone and cementum with resulting ankylosis between tooth and bone. Excess of Vitamin D may also produce pathologic calcification of the gingiva.

2. Magnesium

Experimentally, magnesium deficiency in rats has produced gingival hypertrophy, loosening of the teeth, and evidence of periodontal disease.

E. VITAMINS:

1. Vitamin A

Vitamin A deficiency has been related to alteration of the periodontium by several investigators. Generally, a gingival hyperplasia and hyperkeratosis has been reported.

The effects of a deficiency on the teeth as reported include retarded tooth eruption, imperfect root formation, malposition, and thickening of the cementum.

Among the bone findings are: Increased density with fewer marrow spaces and hypercalcification.

Glickman and Stoller noted that no evidence of pathology was observed in the periodontal membrane or in the interproximal alveolar bone unless there was simultaneous food impaction and other local irritating factors. When these factors were present, the Vitamin A deficient experimental animals had a higher tendency toward margin gingivitis, apical proliferation of the epithelial attachment, and gingival and periodontal abscess formation.

Hypervitaminosis A has been shown to produce excess bone fragility in experimental animals. Generalized bone resorptive activity and osteoporosis produced multiple spontaneous fractures in experimental animals.

2. Vitamin B Complex

a. Thiamine (B_1)

Manifestations of B_1 deficiency in humans termed beriberi, are cardiovascular symptoms, paralysis, edema, and loss of appetite. Oral changes are herpetic-like lesions and some dentinal hypersensitivity.

b. Riboflavin (B_2)

Oral manifestations of riboflavin deficiency are angular cheilosis, engorgement of the fungiform papillae giving the tongue a purplish red color, and fissuring of the tongue. Symptoms are soreness of lips and tongue, burning, and paresthesia. Experimental riboflavin deficiency in monkey has been reported to cause necrosis of the gingiva, the periodontal membrane, and some areas of the alveolar process.

c. Nicotinic Acid (Niacin)

Nicotinic acid deficiency or aniacinosis results in pellagra which is characterized by dermatitis, diarrhea, and dementia--the 3 D's of the disease.

King found a definite relationship between pellagra and the incidence of ANUG. He found that when niacin was administered in addition to local therapy, the recovery rate of the disease was significantly increased.

d. Pantothenic Acid

Although periodontal disease has been reported in animals with pantothenic acid deficiency, occurrence of comparable changes in humans has not been described.

3. Vitamin C

Deficiencies of Vitamin C results in a failure of the formation and maintenance of intercellular substances in tissues of mesenchymal origin. The effect on bone is retarded osteoid formation, impaired osteoplastic function, and a generalized osteoporosis. In vitamin C deficiency, there is an increased capillary permeability with subsequent susceptibility to traumatic hemorrhages.

The main feature of scurvy or vitamin C deficiency is the inability of the fibroblasts to form intercellular substances. This may be directly related to the failure of healing in depleted animals. An interesting observation is that wound repair which entails the rapid formation of large amounts of collagen containing tissue, increased the nutritional Vitamin C requirements in experimental animals.

Hemorrhage of the gingiva and loosening of the teeth have long been recognized as clinical signs of scurvy. Histopathologic studies of Vitamin C deficiency in man have shown epithelial destruction, degeneration of blood vessels, capillary stasis, and deepening of the periodontal pockets.

Increased mobility of the teeth was considered to be caused mainly by the scorbutic destruction of the supporting structures. Westin found that when Vitamin C was added to the diet, a dramatic healing of the periodontal structures followed, and he maintained that rarely did permanent damage result.

Waerhaug, experimenting with scorbutic monkeys, found bone formation to be arrested and increased osteoclasia to be a common finding. He found striking changes in the connective tissue; collagen was nearly completely broken down. Sharpey's fibers were seen to extend from the cementum but were absent at the alveolar wall. The epithelium seemed to be little affected. He concluded, because of this, that the histopathologic picture of the periodontal structures in C avitaminosis does not resemble that of periodontal disease in man.

Glickman concurs with Waerhaugs findings and states that the deficiency itself is not responsible for the initiation of, or an increase in the incidence of marginal gingivitis and the initiation of gingival inflammation in acute Vitamin C deficiency requires the presence of irritation from food remnants or debris in the gingival sulcus. However, when pocket formation does occur, it is modified by the deficiency in that the pockets are of greater depth than under normal circumstances.

This exaggerated destruction in the presence of inflammation in acute Vitamin C deficiency is partially due to the inability of the body to limit the inflammatory reaction. Ordinarily, a barrier of collagen fibrils, fibrin, and inflammatory cells separate areas of injury from the underlying bone. C-deficient animals have lost the ability to form such a barrier.

V. SUMMARY:

The relationship between many of the nutritional disorders and periodontal disease is beyond question. However, specific

cause and effect relationships have been largely on annual experimentation which is always subject to qualifying limitations.

When there is a distorted response of the periodontium to local factors, a nutritional disorder must be considered in the differential diagnosis and treatment, although it may be empirical.

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LECTURE NO. 21

SYSTEMIC DISTURBANCES AS RELATED TO PERIODONTAL DISEASES

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I. THE ROLE OF SYSTEMIC FACTORS:

Little is known regarding the role of systemic factors in most of the human periodontal diseases, and an assessment of the relative importance of local and systemic factors can be no more than speculation.

It is generally believed that systemic factors play a part in the etiology of periodontal diseases by in some way reducing local tissue resistance. Nevertheless, there are relatively few cases in which it is possible to point with any certainty to the systemic factor involved.

The following systemic factors appear to be of importance in some periodontal diseases.

A. RACIAL OR HEREDITARY FACTORS:

It has been generally accepted that certain races are more prone than others to periodontal disease. An "inherited inferiority of the dental organ" has been suggested to account for this. The results of some recent surveys have thrown doubt on the long-held conviction that racial factors are of great importance in susceptibility to periodontal diseases, but they have not excluded such a possibility.

B. NUTRITIONAL DEFICIENCIES:

That gingivitis may be associated with scurvy is well recognized, but surveys of gingivitis in areas where nutritional deficiencies are evident in the population have not thrown any consistent association between the deficiencies and gingivitis. This, however, does not prove that nutrition and periodontal diseases are unrelated; before any progress can be made toward answering this question, thorough dietary and nutritional

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surveys are needed, both in groups with a very low prevalence of periodontal disease and in groups with a very high prevalence. (See Lecture No. 20)

C. SYSTEMIC DISEASES:

Severe diseases such as diabetes mellitus or blood dyscrasias lower tissue resistance, and acute febrile disease may adversely affect conditions in the mouth. In parts of the world where severe chronic diseases are common, they may play an important etiological role in periodontal disease.

D. HORMONAL DISTURBANCES:

Certain types of gingivitis appear to be associated with endocrine imbalance. During pregnancy, any pre-existing gingivitis tends to become more severe and to change in character. The rather rare chronic desquamative gingivitis occurs mainly in women at the time of the menopause, or during puberty or early adolescence. The most common form of gingivitis in children and young adults is the hyperplastic type, and it has been postulated that the temporary endocrine imbalance associated with puberty is responsible for this particular type of inflammatory reaction. A variation in the severity of gingivitis has also been noted in relation to the menstrual periods.

With the exception of the cases occuring during pregnancy, it has seldom been possible to demonstrate other signs of endocrine imbalance, and treatment with hormone preparations has proved unsatisfactory.

E. PSYCHOSOMATIC FACTORS:

Very little is known about the effect of psychosomatic factors on periodontal disease. There is some evidence that they play a definite part in the etiology of acute necrotizing gingivitis. They may also be important in conditions such as bruxism which can aggravate periodontal disease.

F. DRUGS AND HEAVY METALS:

Regular use of the anticonvolsant drug diphenyl-hydantoin (Dilantin) is often associated with gross gingival enlargement.

In edentulous mouths, no change occurs in the mucosa covering the alveolar process and it therefore appears probable that the effect of the drug is to modify a pre-existing gingivitis. Control of periodontal disease in patients taking this drug is nevertheless a difficult problem.

Ingestion of salts of some metals (Bismuth, mercury or lead) causes dark deposits in the gingiva around teeth with bacterial plaque or calculus. Again, this would appear to be a modifying influence on the inflammatory reaction salts of the metal being deposited around capillaries in the connective tissue; presumably, absorbed products of bacteria react with the salts in the tissue fluids to form insoluble products (sulfides) which are precipitated. There is no evidence that drugs or metals are important in the etiology of other periodontal diseases.

II. EFFECT OF SYSTEMIC DISTURBANCES:

A. HEMATOLOGIC DISTURBANCES:

Oral changes very often are the earliest indications of the presence of hemotologic disturbances, but cannot be relied upon for the diagnosis of the exact nature of the patient's blood dyscrasia.

1. Leukemia

Oral changes occur most frequently in acute and subacute monocytic leukemia, less frequently in acute and subacute lymphatic and myelogenous leukemia and rarely in chronic leukemia. The clinical changes which may occur are diffuse, cyanotic, bluish-red, discoloration of the gingival mucosa; a diffuse edematous enlargement, a roundness and tenseness of the gingival margins, blunting of the interdental papillae, and varying degrees of inflammation with ulceration, necrosis and pseudomembrane formation. Microscopically, the gingival presents a dense, diffuse infiltration of predominatly immature leukocytes in the marginal and attached gingiva. The normal connective tissue components are displaced by leukemic cells. The nature of the

cells depends on the type of leukemia. The epithelium may be thinned or hyperplastic. Degeneration associated with intra-cellular edema and leukocytic infiltration with diminished keratinization are common findings.

Local irritation is an important factor in the causation of changes in the periodontium in leukemia. In the absence of calculus, food debris, food impaction, overhanging restorations or trauma, leukemic patients may present no evidence of periodontal disease.

In leukemia, the response to irritation is altered. There is a pronounced infiltration of immature leukemic cells in addition to the usual inflammatory cells. With pronounced cellular infiltration there is degeneration of the gingival tissue. The gingiva assumes a peculiar bluish-red color; it is sponge-like and friable and bleeds upon the slightest provocation or even spontaneously and persistently. This altered tissue is markedly susceptible to bacterial infection. Because of the degenerated anoxemic condition of the gingiva, the bacterial infection is so severe that acute gingival necrosis and pseudomembrane formation are common findings. Associated disturbances are loss of appetite, nausea, blood loss from persistent gingival bleeding and constant gnawing pain.

Of particular clinical importance is the fact that the degree of local irritation markedly influences the severity of oral changes in the leukemic patient. By eliminating local irritants it is often possible to alleviate severe oral changes.

2. <u>Oral Changes in Anemia</u>

   a. Pernicious anemia is most frequently found in people over age 40. Both sexes are

affected equally. The disease is characterized by nervous, cardiovascular and gastro-intestinal symptoms. Macrocytic hyperchromic anemia is characterized by a severe decrease in the number of red blood cells (1,000,000 per cubic millimeter) decreased platelet count (40,000), decrease in white blood cells, poikilocytosis (shape), and polychromatophilia (affinity for stains), and red blood cells containing nuclear fragments.

Oral changes are seen in mucosa, lips, and tongue in 75% of the cases. The mucosa and lips are pale and yellowish. The tongue is smooth, red and shiny (atrophy of the papillae). Marked pallor of the gingiva is a striking finding in pernicious anemia, with a wide variety of inflammatory changes depending on the nature of the local irritation.

b. Microcytic hypochromic anemia:

This type of anemia is caused by a deficiency in iron and other substances concerned with hemoglobin production, occurs in chronic blood loss, and is associated with inadequate iron ingestion or absorption. It is characterized by a moderate decrease in the number of red blood cells (3,000,000), a lowered color index (.5), an increased platelet count (500,000), and decreased hemoglobin.

The oral changes include a change in color of the mucosa. The attached and marginal gingiva are ashen-grey. The interdental papillae and the marginal gingiva which are involved by inflammation present a purplish-red appearance which stands out in contrast to the adjacent gingival pallor. Simple iron deficiency anemia is often responsible for atrophic tongue changes.

c. Erythroblastic Anemia (Cooley's Anemia):

An inherited disorder characterized by a

hemolytic anemia, splenomegaly, nucleated red blood cells in the peripheral blood and generalized skeletal lesions.

Oral changes include pallor and cyanosis of the mucosa and marked malocclusion due to overgrowth of the alveolar ridge of the maxilla. There is an associated spreading of the teeth with creation of large interdental spaces. Radiographic examination reveals generalized rarefaction of the jaws with an alteration of the trabecular pattern, and obliteration of the lamina dura in some areas.

d. Infectious Mononucleosis:

This is a benign communicable disease which usually occurs in children or young adults. It is characterized by sudden onset, headache, fever, muscular ache, sore throat, nausea, vomiting, swelling and tenderness of lymph nodes.

The oral findings include diffuse erythema of of the entire mucosa with petechia in some cases. The marginal and interdental papillae are swollen and red and bleed on slightest trauma or even spontaneously.

e. Agranulocytosis (malignant neutropenia):

Agranulocytosis is an acute disease characterized by extreme leukopenia and neutropenia and accompanied by necrotic changes in the periodontium and ulceration of the oral mucous membrane, skin and digestive tract.

Drug idiosyncrasy is the most common cause of agranulocytosis (amidopyrine, barbiturates, sulfanamides, etc.).

The absence of a notable inflammatory reaction because of lack of granulocytes is a striking feature. Gingival hemorrhage, increased salivation and fetid odor are accompanying clinical features.

f. Polycythemia:

It is characterized by an increased red cell production in the bone marrow, splenomegaly and an increased red cell count ranging from seven to ten million per cubic millimeter.

There is a reddish-blue cyanotic discoloration of the skin, with comparable involvement of the oral and pharyngeal mucous membrane. Bright red, diffuse discoloration of the gingiva and tongue are sometimes seen.

B. METALLIC INTOXICATION:

Ingestion of metals such as mercury, lead and bismuth in medicinal compounds and through industrial contact may result in oral manifestations. These chemicals act as irritants which accentuate the pre-existent inflammation and often lead to ulceration of the gingiva and adjacent mucosa and destruction of the underlying bone.

C. DEBILITATING DISEASES AND THE PERIODONTIUM:

Debilitating diseases such as syphilis, chronic nephritis, and tuberculosis may predispose to periodontal disease by impairing tissue resistance to local irritants and creating a tendency toward alveolar bone resorption. A type of membranous stomatitis has been described associated with debilitation in uremia, and a dry sore mouth with edema, purulent inflammation, and bleeding of the gingiva has been noted with primary renal disease. The absence of periodontal disease in chronically-ill patients has been presented as evidence that in individual cases, systemic disease may exert no deleterious effect upon the periodontium.

D. PSYCOSOMATIC DISORDERS AND THE PERIODONTIUM:

There are two ways in which psycosomatic disorders may be induced in the oral cavity: (a) through the development of habits which are injurious to the tissues, and (b) by the direct effect of the autonomic nervous system upon the physiologic tissue balance.

Under conditions of mental and emotional stress, relief may be obtained from muscular activity which leads to neurotic habits, such as grinding or clenching of the teeth, tongue and lip habits, movement of jaws into a functional position, etc. Because of their injurious effect on the periodontal tissues, such habits are common etiologic factors in periodontal disease.

Disorders of psychosomatic origin may be produced by the action of the autonomic nervous system. Alterations in the vascular supply caused by autonomic stimulation may adversely effect the health of the periodontium by impairing tissue nutrition. Diminution in the secretion of saliva in emotional disorders may lead to xerostomia.

III. HORMONAL INFLUENCES ON THE PERIODONTIUM:

A. Hormones are organic substances of varying complexity produced by endocrine glands. They are excreted directly into the blood stream.

1. Hypothyroidism:

The effects of hypothyroidism vary with the age at which it occurs. The basal metabolic rate is depressed and growth is retarded. Cretinism, juvenile myxedema and adult myxedema are the three clinical syndromes which result.

Notable changes in the periodontal tissue have been attributed to cretinism. Chronic periodontal disease with severe bone loss has been described in patients with myxedema with the suggestion that the latter condition contributed to the periodontal destruction.

2. Hyperthyrodism (Graves Disease):

Hyperfunction of the thyroid gland produces cardiovascular effects, nervousness, emotional instability, loss of weight, exophthalmia, etc. Infants show increased growth and development with early eruption of teeth; the teeth and jaws are well formed. Alveolar bone appears somewhat

rarefied and partly decalcified. Increased salivary flow due to sympathetic stimulation, but no other oral changes in the adult.

3. Hypopituitarism:

This is a deficiency in the secretion of the anterior lobe of the pituitary, and is marked by a retardation in the growth of all tissues The earlier in life the condition occurs, the more severe will be the clinical change. Hypopituitarism in children results in dwarfism. In dwarfism, the cranium and face develop very slowly. There is underdevelopment of the sinuses, retardation in the development of the teeth and jaws, a delayed resorption of the deciduous teeth, and marked retardation in formation and eruption of the permanent teeth. Retardation of growth of the mandible results in reduced intermaxillary space crowding of the teeth and a tendency toward a distal relationship of the mandible.

4. Hyperpituitarism:

The increased secretion of the anterior lobe results in gigantism or acromegaly depending on the age at which it occurs. Acromegaly is characterized by a disproportinate overgrowth of the facial bones. A marked overgrowth of the alveolar processes causes an increase in the size of the dental arch and consequent spacing of the teeth. Hypercementosis is another feature of the increased rate of growth.

5. Hypoparathyroidism:

Results from accidental removal of gland during thyroidectomy or from deficiencies occuring early in life. There is a resulting hypocalcemia and a resultant increased excitability of the nervous system. The develcping enamel and dentine show alternate irregular zones of undercalcification and overcalcification.

6. Hyperparathyroidism:

Hypersecretion leads to generalized osteitis fibrosa, demineralization of the skeleton, formation of bone cysts and giant cell tumors.

Oral findings have been described as malocclusion and tooth mobility. Cystic-like cavities of the jaws, osteoporosis and absence of lamina dura. It has been reported that 25 per cent of patients with hyperparathyroidism had signs and symptoms of alveolar bone destruction, looseness of teeth and widening of the periodontal membrane space.

7. Diabetes:

Dryness of the mouth, diffuse erythema of the oral mucosa, coated tongue, redness of the tongue, a tendency toward periodontal abscess formation, "diabetic periodontoclasia" and "diabetic stomatitis," enlarged gingivae, gingival polyps, bleeding, swollen, tender gingival papilla and loose teeth have all been described as oral changes which are either caused by or intimately associated with diabetes. Vertical and horizontal destruction of alveolar bone have also been attributed to diabetes. Diabetes is also identified with a greater tendency toward increased severity of inflammation and susceptibility to infection. Other investigators recognize no particular relationship between diabetes and oral disease and maintain that when the two exist together it is a matter of coincidence rather than a specific cause and effect relationship.

On the basis of studies of human biopsy material, it has been suggested that the gingiva and periodontal granulation tissue in diabetes mellitus presents a fatty infiltration which may account for lowered resistance in the periodontal area. Hyperplasia of the gingiva and hyperkeratosis have also been described as microscopic features

in diabetes. Microscopic changes in the gingiva in human diabetes include a change in surface character from stippled to smooth, lack of hornification, intranuclear vacuolization in the epithelium, increased intensity of the inflammation, change in the structural appearance of the connective tissue, and presence of calcified foreign bodies. However, none of these changes were considered unique or characteristic for diabetes.

Periodontal disease in diabetics presents no unique microscopic features. It has not been satisfactorily demonstrated that diabetes is responsible for the onset of gingival disease or for the production of specific gingival changes. Some diabetic patients do present a tendency toward loss of alveolar bone which may be manifested even in the absence of gingival disease. The progress of bone loss associated with gingival disease in such patients would be more rapid than that which would occur in the absence of diabetes.

Diabetes is a systemic disturbance which of itself or through associated metabolic changes can effect the progress of alveolar bone loss in periodontal disease. Therefore, the control of diabetes is an essential remedial measure in the management of diabetic patients with periodontal disease. However, it is important not to depend on the control of diabetes alone for the cessation of the alveolar bone loss or the alleviation of other phases of periodontal disease such as gingivitis or pocket formation. The elimination of all contributory factors is necessary if periodontal health is to be established and maintained in patients with controlled diabetes.

IV. GINGIVAL CHANGES IN PUBERTY, MENSTRUATION, PREGNANCY, AND MENOPAUSE:

A. PUBERTY:

Puberty is often accompanied by an exaggerated response

of the gingiva to local irritation. Marked clinical changes are produced by slight local factors. Pronounced inflammation, bluish-red discoloration, and enlargement of the gingiva occur.

B. MENSTRUAL CYCLE:

In general, the menstrual cycle is accompanied by no notable gingival changes. Minor gingival disorders such as the periodic appearance of bleeding gums in the days preceeding the menstrual flow are occasionally encountered. Aphthous and herpetic lesions are sometimes encountered in association with the menstrual cycle.

C. PREGNANCY:

In pregnancy, there is a tendency for the response of the gingiva to local irritation to be exaggerated. In the absence of local irritation, the gingiva in pregnancy presents no notable clinical changes.

The so-called "pregnancy tumor" is an inflammatory lesion of the gingiva which usually appears after the third month of pregnancy with a reported incidence of from two per cent to five per cent. It appears as a painless, mushroom-like mass protruding from the gingival margin in the interproximal labial or lingual areas to which it is attached by a sessile of pedunculated base.

D. MENOPAUSAL GINGIVOSTOMATITIS:

This condition occurs during the menopause or in the post-menopausal period. It is not a common condition. The gingival picture includes fading of the mucosal pinkness, dryness and gingival bleeding. Comparable changes are seen in the remainder of the oral mucosa. The patient complains of a dry, burning sensation with extreme sensitivity to thermal changes. Microscopically, the gingiva presents atrophy of the germinal and prickle cell layers of the epithelium.

The signs and symptoms of menopausal gingivostomatitis are to a degree comparable to those found in desquamative gingivitis. The prevailing opinion is that both of these conditions arise from an atrophy and diminished keratinization

of the oral epithelium associated with a dimminution in estrogen or a disturbance in its utilization.

V. MISCELLANEOUS SYSTEMIC FACTORS:

A. DILANTIN HYPERPLASIA:

Marginal gingival hyperplasia is the type of gingival enlargement generally associated with dilantin sodium; diffuse hyperplasia also occurs but is seen less frequently.

The primary or basic lesion is a painless hyperplasia of the gingiva which occurs interproximally, buccally, lingually, and circumscribing the necks of the individual teeth. The hyperplasia has not been observed in edentulous mouths, or in spaces created by missing teeth. Histologic examination reveals a non-specific hyperplasia of the epithelium and connective tissue. There is an especially pronounced formation of collagen fibrils condensed in definite bundle formation.

Changes in size, color, shape, and consistency, associated with various local conditions are secondary to the primary hyperplastic tendency. Complication by secondary inflammatory changes result in a diffuse increase in size and bulging away from the tooth surface.

The course of the basic condition is a chronic one, with fluctuation in severity apparently unrelated to local influences or drug dosage. Spontaneous disappearance however has not been observed unless dilantin is discontinued.

B. SCLERODERMA:

A disease characterized by a primary induration and edema of the skin and later with atrophy and pigmentation. There is frequent involvement of the oral cavity with involvement of the tongue most frequently followed by the buccal mucosa and the gingiva. Radiographically, a widening of the periodontal space is observed occasionally with an obliteration of the lamina dura. Clinically, the teeth remain firm. There is histologic evidence of degeneration of the periodontal tissues. The effects of these changes are minimal in the absence of local destructive factors.

VI. SUMMARY:

Other forms of systemic disease could be discussed, but the above should suffice to point out that in most instances systemic disease is a contributory factor rather than an etiologic factor in periodontal disease. In those instances where it initiates periodontal disease, the course of the disease is determined by the superimposed local factors.

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