

PERIODONTAL LITERATURE REVIEWS

a summary of current knowledge

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PREFACE

This book represents the collective result of many years of continual review of selected journal literature as it relates to all aspects of periodontology--from basic histology through diagnosis, treatment, and supportive therapy. This edition, the first published by The American Academy of Periodontology, is based on the material developed over the past 14 of years by the faculty and staff of the Air Force Residency Program in Periodontics at Wilford Hall USAF Medical Center, San Antonio, Texas. Searching and evaluating the literature constitutes an important part of that training program and, as in many institutions, has resulted in a single document copied for use by the residents, faculty, and Air Force Dental Officers. These "literature reviews" have traditionally been unavailable to a broader readership. With the rapidly escalating number of journals now published and limited amount of time for review, it seems practical to develop an informational resource which summarizes key articles and make it available to the entire periodontal community. When the Air Force Program initially offered its "Periodontal Text of Classic Literature Subject Overviews" as a basis for such a publication, the Academy's Board of Trustees agreed that the AAP could provide a valuable service by assuming responsibility for the publication of this material. This first edition was developed by an ad hoc committee of the Academy, working with the 1993 edition of the Air Force material. Future editions of the text by the Academy will be prepared under the direction of an Editorial Board.

The Committee retained the basic structure and outline of the original material and supplemented it with extensive and critical review of more recent journal articles. Our objective was to select only those articles which were felt to have significantly influenced the practice of periodontics and to provide the reader with an overview of the overwhelming amount of information available. It was not the intent of this committee to produce a textbook or inclusive review of the literature. That would not only be impossible, but would effectively negate the usefulness of the document. Readers who need more in-depth information on a subject can use the extensive reference lists as a basis for further research.

As Chair of the Committee, I would like to extend my sincere personal gratitude and professional acknowledgments to the residents and faculty of the Wilford Hall USAF Medical Center Residency in Periodontics who so generously provided the primary document without which we never could have undertaken this complex task. Special recognition is accorded my committee colleagues, Dr. Fermin Carranza, University of California at Los Angeles; Dr. Connie Drisko, University of Louisville; Dr. John Rapley, University of Missouri-Kansas City; and Dr. Peter Robinson, Northwestern University and their residents and fellow faculty members for the considerable time and energy expended on this project. Without their support, cooperation, and personal commitments to excellence, this review text could not have been completed.

William C. Hallmon
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CHAPTER 1. HISTOLOGY

Section 1. Gingiva and Oral Mucosa

DEFINITIONS

Mucosa: A mucous membrane.

Masticatory Mucosa: The gingiva and the mucosal covering of the hard palate.

Oral Mucosa: The tissue lining the oral cavity.

Alveolar Mucosa: Mucosa covering the basal part of the alveolar process and continuing without demarcation into the vestibular fornix and the floor of the mouth. It is loosely attached to the periosteum and is movable.

Gingiva: The fibrous investing tissue, covered by keratinized epithelium, which immediately surrounds a tooth and is contiguous with its periodontal ligament and with the mucosal tissues of the mouth.

MACROSCOPIC ANATOMY

Anatomic Features of Gingiva

The gingiva consists of free and attached gingiva. The free marginal gingiva surrounds the tooth and is not directly attached to the tooth surface. The attached gingiva is the portion of the gingiva that is firm, dense, stippled, and tightly bound to the underlying periodontium, tooth, and bone. The free gingival margin is defined as the coronal border of the free gingiva.

The free gingival margin generally corresponds to the base of the gingival sulcus. It is present in 30% to 40% of adults and most frequently occurs in the mandibular premolar and incisor regions.

The mucogingival junction (MGJ) represents the junction between the gingiva (keratinized) and alveolar mucosa (non-keratinized) (Lindhe, 1983).

Width and Thickness of Gingiva

Bowers (1963) measured the widths of the facial attached gingiva in the primary and permanent dentitions of 240 subjects. Width of the attached gingiva was determined by subtracting the sulcus probing depth (free gingiva) from the total width of the keratinized tissue (gingival margin to MGJ). Extremes of the width of attached gingiva ranged from 1 to 9 mm. Values were greatest in the incisor regions (especially the lateral incisor) and the least in the canine and first premolar sites. The maxilla usually exhibited a broader zone of attached gingiva than the mandible. Clinically healthy gingiva was noted in subjects with less than 1 mm of attached gingiva, but the tissue was usually inflamed in areas of no attached gingiva. Buccal-lingual tooth position affected the amount of attached gingiva present, and high frenum and muscle attachments were generally associated with narrow zones of attached gingiva.

In a series of studies, Andlin-Sobocki and Bodin (1993) confirmed the pattern of facial keratinized tissue widths in longitudinal observations of children. Increases in widths of facial attached and keratinized tissue were noted in primary and permanent teeth over 2 years. Changes in attached gingiva from a deciduous tooth to permanent successor were inconsistent, but if the deciduous tooth had less than 1 mm of attached gingiva at baseline, the permanent tooth had a wider zone of attached gingiva at the second examination. Facially-positioned teeth had narrower zones of attached and keratinized tissue than well-aligned or lingually-positioned teeth. As teeth moved lingually, an increase in the width of attached and keratinized tissue and a slight decrease in clinical crown height were observed. Teeth moving facially had a decrease in the width of the attached and keratinized tissue.

Measurements of the width of lingual attached gingiva were performed by Voigt et al. (1978) on clinically normal subjects ranging in age from 3 to 36 years. Measurements were determined by subtracting the sulcus depth from the sulcus depth from the width of the keratinized tissue and ranged from 1 to 8 mm. Greatest widths were recorded on the first and second molars (4.7 mm), decreasing at premolar and third molar sites. The smallest widths were observed on the incisors and canines (1.9 mm). With the progression from the primary to the permanent dentition, the width of the lingual attached gingiva decreased.

Thickness of Free and Attached Gingiva

Goaslind et al. (1977) measured the thickness of the free and attached facial gingiva in a population consisting of 10 males (age 25 to 36) with clinically healthy gingiva. Results demonstrated considerable variation of gingival thickness among subjects and among areas within individual subjects. Free gingival thickness averaged 1.56 mm $\pm$ 0.39 mm, increased from anterior to posterior and was directly proportional to sulcus depth. Thickness of the attached gingiva averaged 1.25 mm $\pm$ 0.42 mm, increased from anterior to posterior in the mandibular arch, remained relatively constant in the maxillary anterior, and was inversely proportional to attached gingival width. The overall mean thickness for all areas was 1.41 mm.

Gingival Stippling

Stippling refers to orange peel-like surface characteristics observed in attached gingiva of approximately 40% of the adult population. It was originally assumed that a lack of stippling reflected inflammatory change of the gingiva

but, due to the variability of the presence or absence of stippling in healthy gingiva, it is now believed that one can only correlate the presence of inflammation with the loss of stippling, assuming that stippling was present previously. Owings (1969) performed a histologic study on the relationship between gingival stippling and keratinization of attached gingiva in 45 male subjects.

Findings revealed that 76% of the gingiva exhibited parakeratosis with the remainder exhibiting orthokeratosis, regardless of the pattern of stippling. The glycogen in the attached gingiva was limited exclusively to the spinous layer of the epithelial ridges in tissue with a dense, diffuse pattern of stippling. Owings felt that stippling tended to develop at the intersection of the epithelial ridges due to a decrease in metabolic activity at the central portion. The author suggested that stippling may represent areas in the gingiva which exhibit retarded metabolism.

Karring and Löe (1970) histologically studied the relationship between stippling and subsurface features of the gingiva by means of 3-dimensional wax models reconstructed from serial histologic sections. The morphology of the epithelial-connective tissue interface was variable. Most specimens presented connective tissue papillae projecting into the epithelium; however, more or less continuous ridges also occurred. Depressions in the surface of the epithelium (stippling) coincided with intersections of epithelial ridges.

Retrocuspid Papilla

Levin and Cutright (1977) described the histologic origin of the retrocuspid papilla. The authors suggested that the encircling plexus of an erupting deciduous or permanent tooth causes erosion of the lingual cortical plate and the plexus anastomoses with the lingual mucosal circulation, thereby forming the papilla. Location and blanching lead to the clinical diagnosis and biopsy is unnecessary.

MICROSCOPIC ANATOMY

Periosteal Microvasculature

Nobuto et al. (1989), using scanning electron micrographs from dogs, observed that the vascular plexus distributed in the periosteum of the gingiva formed a coarse structural network which consisted primarily of arterioles and venules. The alveolar mucosa vasculature was comprised of a dense network structure of arterioles, capillaries, and venules which formed a vascular bed.

Layers of the Oral Epithelium

The layers comprising the oral epithelium are the stratum basale, stratum spinosum, stratum granulosum, and the stratum corneum. A brief description of these layers is given below (Lindhe, 1983; Squier et al., 1976) (Table 1).

The stratum basale consists of a basal layer of cuboidal cells along the basement membrane that is the site of cell replication. These cells manifest a full complement of organelles.

TABLE 1. HISTOLOGIC COMPARISON OF ATTACHED GINGIVA AND ALVEOLAR MUCOSA*

Attached Gingiva	Alveolar Mucosa
keratinized	non-keratinized
stippled	unstippled
deep rete pegs	short, wide rete pegs (few)
thick lamina propria	thin lamina propria
few elastic fibers	numerous elastic fibers
indistinct submucosa	distinct submucosa
firmly attached	movable

*Adapted from Squier et al. (1976)

The stratum spinosum is also termed the spinous or prickle cell layer and is named for the appearance of prominent peripheral cytoplasmic processes which resemble spines at the light microscopic level. The cytoplasm is rich in tonofilaments which terminate in the attachment plaques of desmosomes which attach to adjacent cells. Essentially, these are large polyhedral cells with diminished cellular activity when compared to the basal layer; Langerhans cells are usually found in this layer and mitotic figures may be observed in lower layers.

The granular cell layer (stratum granulosum) is characterized by the presence of keratohyalin granules which are believed to play a role in the process of keratinization. These cells are "flattened," and have diminished organelles, pyknotic nuclei, and "degenerative" mitochondria. Desmosomal changes may be observed in the upper regions of this layer and are smaller and more numerous in deeper layers.

The stratum corneum is a keratinized cell layer characterized by flattened, pyknotic cells composed of tightly-packed tonofilaments. Organelles are rare and keratinization may be associated with cell lysis. Continual degradation of desmosomal attachments eventually results in loss of keratinocytes on the epithelial surface.

As cells proceed from the basal to the keratinized layer, they become progressively flatter, organelles diminish, tonofilaments dramatically increase, and the nuclear-cytoplasmic ratio decreases with eventual loss of the nucleus. There is a shift from aerobic to anaerobic metabolism which is accompanied by an increase of glycogen in the upper layers. Keratohyalin granules appear in the granular cell layer, and the number of desmosomes increase while the size of individual desmosomes decreases.

Andersson et al. (1987) determined that histologic specimens prepared with a 300 mOsmol (isotonic) solution, rather than the normally utilized 100 mOsmol (hypo-osmolar) solution, showed much larger intercellular spaces with much less distortion of intercellular organelles. The basal and spinous layers exhibited the most difference between the two methods of processing.

Cell Junctions

Desmosomes constitute the most common cell junction and consist of the outer leaflets of cell membrane of two adjoining cells, the thick inner leaflets of the cell membranes and attachment plaques which represent granular and fibrillar material in the cytoplasm. These attachments are fairly resistant to inflammatory changes but are eventually lost with increasing inflammation.

Hemidesmosomes are half-desmosomes which form the attachment of epithelial cells to basement membrane and to the tooth surface.

Gap junctions represent cell communication channels which are lost in areas of moderate inflammation.

Tight junctions include both zonula occludens (continuous zone) or macula occludens (point contact) and represent fusion of adjacent cell membranes which function in ion exchange, compartmentalization, and cell adhesion. These attachments increase in numbers in the upper epithelial layers, but are lost in areas of moderate inflammation (Thilander, 1968).

Gingival Col

The col is a valley-like depression of the interdental gingiva which connects facial and lingual papillae and conforms to the shape of the interproximal contact area. In a study using Rhesus monkeys, McHugh (1974) confirmed that the interdental area is col-shaped. This area consists of soft tissue residing between papillary peaks on the buccal and lingual. Histological analysis of sections through the actual col showed that it is always lined by squamous epithelium, 5 or more cell-layers thick. Ameloblasts were not found. The previously held theory that the interdental area is more vulnerable due to retention of the primary epithelial attachment was refuted by direct observation and radioautography. Radioautographs indicated cell division in all epithelia lining the col. Histologic features and turnover-rate of the epithelium in the col region are similar to those of the rest of the gingival epithelium. Clinical studies supported the hypothesis that gingivitis starts interdentally because of plaque accumulation and not because of histologic origin.

Specialized Cells Within the Epithelium

Melanocytes secrete melanin and are located in the lower layers of the epithelium.

Langerhans cells (DiFranco et al., 1985) are dendritic-shaped cells believed to be part of the immune surveillance system. Their numbers increase in inflamed tissue (2 to 10x) with cells likely migrating from the underlying connective tissue in response to antigenic challenge. It is postulated that the role of Langerhans cells is in the uptake and presentation of antigens to T-cells, possibly constituting one of the first lines of defense against surface penetration of the host by forcing antigens. Merkel cells (Squier et al., 1976) are non-dendritic clear cells which are present in the basal layers in oral epithelium. These cells have sparse tonofilaments, are often associated with nerve fibers, and are thought to act as touch-sensory cells.

Basement Membrane (Basal Lamina)

The basement membrane represents the junction of the epithelium and underlying connective tissue. It consists of an electron-dense lamina densa (330 to 600 Å) and an electron-lucent lamina lucida (400 to 450 Å) next to the plasma membrane of the epithelial cells with fine fibrils traversing both layers. The epithelial cells are attached to the basal lamina by hemidesmosomes, and type IV collagen found in this region is believed to be secreted by the basal epithelial cells. Laminin is a glycoprotein that mediates attachment of epithelial cells to type IV collagen and is believed to be present in the lamina lucida region. Susi (1969) described anchoring fibrils and reported their presence in various oral tissues; these fibrils were more numerous in the buccal and alveolar mucosa than in the gingiva and may be related to the mobility and stretching forces seen in these tissues. An almost continuous, scalloped network of anchoring fibrils (200 to 400 Å in diameter and 0.2 μm in length) was observed on the connective tissue side of the basement membrane. The fine filaments were more concentrated at the sites of hemidesmosomes. Collagen fibrils extended from the connective tissue to the basement membrane region where they appeared to enter loops formed by projections of the anchoring fibrils into the connective tissue. The collagen fibrils were described as running parallel to the basement membrane for a short distance before returning to the deeper regions of the connective tissue. The author speculated that the anchoring fibrils may be synthesized by the basal epithelial cells and serve to help anchor the epithelium to the underlying connective tissue.

Epithelial (Rete) Ridges

Epithelial (rete) ridges represent areas of epithelial proliferation into the underlying connective tissue. These are believed to promote anchoring of epithelium to the connective tissue by increasing the surface area of attachment. They are more pronounced in the gingiva than in the alveolar mucosa.

Cytokeratins

Mackenzie and Gao (1993) examined gingival cytokeratins (a family of 19 structural proteins found in epithelial cells) and compared the patterns of keratin expression in inflamed gingiva and pocket epithelium. The authors reported that with inflammation, there is a decrease in normal keratin markers of differentiation, and expression of some keratin markers that are normally absent. The pocket epithelium demonstrated a pattern of keratin expression similar to normal junctional epithelium.

Reviews of Gingival Epithelium

Smith (1976) described the histology of the gingival epithelium, including its surface characteristics and its com-

ponents. The author specifically detailed the masticatory gingival epithelium, the crevicular gingival epithelium, and the attached epithelial cuff. Bye and Caffesse (1979) reviewed the process of keratinization of the gingival epithelium. The authors addressed keratinized gingiva location, development, response to mechanical factors, penetrability, and histology. Gargiulo et al. (1961) established the dimensions and relationships of the dentogingival junction in humans. The authors determined that the average mean measurements for sulcus depth was 0.69 mm; the epithelial attachment, 0.97 mm; and the connective tissue attachment, 1.07 mm. The connective tissue attachment length was the most consistent and the epithelial attachment length was the most variable. Squier (1981) discussed sulcular and junctional epithelial permeability characteristics and questioned the value of pursuing keratinization of the sulcular gingival epithelium. He concluded that keratinization of the gingival sulcus may be a pointless task from the aspect of increasing host resistance.

CONNECTIVE TISSUE

The connective tissue (CT) of the gingiva consists of cells, fibers, and ground substance (proteoglycans and glycoproteins). A brief review of these will be presented.

Cells constitute about 5% of the CT and include fibroblasts (65%), mast cells, PMNs, macrophages, lymphocytes, and plasma cells.

Fibers account for approximately 60 to 65% of the CT, with collagen predominating reticulin and elastic fibers. Ground substance comprises 35% of the CT and consists of protein-polysaccharide macro-molecules made up of proteoglycans and glycoproteins. The proteoglycans (PGs) contain glycosaminoglycans (GAGs) as the polysaccharide units which are covalently bonded to one or more protein chains. PGs are usually large molecules in the ground substance which function to regulate diffusion and fluid flow through the matrix, acting as molecular filters.

Glycoproteins (GPs) function in cell-to-cell and cell-to-matrix interactions. Fibronectin (FN) is the principal GP in CT, serving to orient fibroblasts to collagen and provide protein attachment for cell-matrix adhesions. FN may influence the migration of fibroblasts and play a crucial role in maintaining structural integrity of CT. Laminin (LN) is the attachment glycoprotein for epithelial cells, which mediates attachment of these cells to the basement membrane and preferentially binds type IV collagen.

Collagen

The basic structural collagen unit is tropocollagen. It consists of 3 linear polypeptide chains (alpha 1,2,3) of 1,000 amino acid residues that intertwine forming a triple helix 300 nm long and 15 Å wide. At least 10 different types of collagen are recognized today. Type I predominates and is found in all CT, especially skin and bone; type II is found mainly in cartilage. Type III is present primarily in smooth muscle, especially blood vessels. Type IV is found in basement membranes. Glycine accounts for one-third of all amino acids in collagen. Collagen is also rich in proline, hydroxyproline, and hydroxylysine.

The fibroblast is the main cell responsible for biosynthesis but other cells including osteoblasts, odontoblasts, epithelial cells, and chondrocytes also produce collagen. Synthesis begins with the production of alpha-chains on the surface of the rough endoplasmic reticulum. Post-translational changes include hydroxylation of proline and lysine and glycosylation of the alpha-chains allowing triple helix formation. Vitamin C is necessary for hydroxylation, which occurs in the cisternae of the rough endoplasmic reticulum. This intra-cellular collagen is known as procollagen and differs from tropocollagen by the presence of extra-globular peptides (propeptides) attached to ends of the polypeptide chains. Following glycosylation (i.e., carbohydrate addition) in the Golgi apparatus, procollagen is transported and secreted extra-cellularly where endopeptidases cleave the propeptides, resulting in the formation of tropocollagen. Tropocollagen molecules spontaneously aggregate into fibrils in a quarter-staggered array giving the characteristic cross-banding pattern of 640 to 670 Å. Cross links form between the tropocollagen molecules stabilizing the collagen fibrils. Fibrils coalesce forming fibers which can, in turn, aggregate with a resultant bundle formation.

Kleinman et al. (1981) reviewed the types of collagen, their distribution, and biosynthesis. The authors also described the interaction of cells with collagen and other matrix components, emphasizing cell attachment proteins, growth, and differentiation.

Epithelial-Connective Tissue Interactions

Karring et al. (1975) examined the role of connective tissue in determining differentiation of the epithelium by implanting CT from the palates of monkeys (with the epithelium removed) into pouches created in the buccal alveolar mucosa. Control sites received alveolar mucosa CT transplants. No surface changes were noted in the epithelium upon healing. Three to 4 weeks later, the grafts were exposed and allowed to re-epithelialize from surrounding non-keratinized alveolar mucosa. The sites with CT transplants from the palate healed with a keratinized surface displaying the same characteristics as normal gingival epithelium, whereas the control sites were covered with non-keratinized epithelium. The results of this study demonstrated conclusively that the determinant for epithelial differentiation (keratinization or non-keratinization) is the underlying CT and is not the functional stimuli as previously thought.

Gingival Fiber Groups

The gingival collagen fibers are organized into 5 principal and 6 minor groupings. The principal groupings are the dento-gingival, alveolo-gingival, dento-periosteal, circular, and transseptal fibers. The secondary grouping con-

sists of the periostogingival, interpapillary, transgingival, intercircular, intergingival, and semicircular fibers. The dentogingival fibers extend from the cementum into the lamina propria laterally. Alveolo-gingival fibers "fan" coronally into the lamina propria from the periosteum at the alveolar crest. The dento-periosteal fibers extend from the cementum (close to the cemento-enamel junction [CEJ]) into the periosteum at the alveolar crest. Circular fibers circumscribe the tooth and are present in the attached gingival coronal to the alveolar crest and in the free marginal gingiva. The transseptal fibers extend mesially and distally, inserting into the cementum of the adjacent teeth coronal to the alveolar crest (Hassell, 1993).

Gingival Vascular Supply

The vascular supply of the periodontium is reviewed under chapter 9, Surgical Therapy. Nuki and Hock (1974) studied the organization of the gingival vasculature noting that the subepithelial vasculature consisted of a series of interconnecting capillary units made up of at least 2 terminal arterial capillaries, 4 primary venular capillaries, and numerous connecting vessels. The capillary units were among the first vessels affected by inflammation as evidenced by an increase in vessel width (5 to 10 μm) and length (400 to 1,000 μm as well as alteration of vessel morphology (i.e., formation of vessel loops). The authors noted that with continuing inflammation, certain connecting vessels were lost while other vessels became spatially rearranged.

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Section 2. Junctional Epithelium

DEFINITION

Junctional Epithelium: A single or multiple layer of non-keratinizing cells adhering to the tooth surface at the base of the gingival crevice. Formerly called epithelial attachment.

HISTORICAL CONCEPTS

In 1915, G.V. Black suggested that the oral and odontogenic epithelium fuse into a continuous lining as the tooth erupts. According to Black, only the apical end of the sulcular epithelium was attached to the tooth (at the CEJ), creating a subgingival space which extended to the CEJ adjacent to the loosely adapted gingiva. The historical citations to Black, Gottlieb, Becks, Waerhaug, and Orban are drawn from Listgarten's review (1970).

Gottlieb (1921) first coined the term epithelial attachment and proposed that the oral epithelium (OE) fused with the outer enamel epithelium (OEE) of the erupting tooth at the base of the sulcus. It was suggested that the OEE cells were joined to the tooth by a primary cuticle formed by the degenerating ameloblasts. The cells of the OEE immediately adjacent to the primary cuticle became keratinized, forming a secondary cuticle. In time, the epithelial attachment migrated over the cementum with a corresponding apical displacement of the base of the sulcus. The width of the epithelial attachment remained constant even as the attachment migrated apically.

Becks (1929) proposed a modified version of Gottlieb's theory. He felt that the OE migrated over the connective

tissue surface of the OEE. By age 30, the OEE was lost and the OE formed the new attachment.

Waerhaug (1952) suggested that the tooth/epithelial interface which extends to the CEJ was a potential space maintained tightly against the tooth by vascular pressure. This concept became known as the epithelial cuff.

Orban (1960) inserted steel blades into the sulci of dogs and monkeys and demonstrated a firm attachment of epithelial cells to the teeth. The author agreed with Gottlieb's concept of a firm epithelial attachment.

Listgarten (1966) felt that epithelial cells, whether derived from reduced ameloblasts or oral epithelium, attached to the tooth by means of hemidesmosomes and a basement membrane (basal lamina). This attachment was not static since cells move along the tooth surface from the apical portion of the epithelial attachment to the base of the sulcus. Listgarten observed a Type A cuticle covering areas of the tooth where there was no reduced enamel epithelium. This cuticle was similar to cementum but contained no fibrillar collagen. Interposed between the tooth and the basal lamina is a nonmineralized, electron-dense Type B cuticle. The author indicated that the origin of both cuticles was unknown but postulated that cuticle B was similar to the acquired pellicle and was of salivary origin.

CURRENT CONCEPTS

Kobayashi et al. (1976) studied the JE of monkeys, reporting a highly variable relationship between the junctional epithelium and the tooth surface coronal to the CEJ. When present, a homogeneous, electron-dense dental cuticle occurred between the afibrillar cementum and JE. When the dental cuticle was absent, a thin, dense linear border was noticed in the same position. While the origin and function of the dental cuticle are unknown, the linear border was determined to consist of serum proteins from tissue fluids. The authors described a unit of adhesion consisting of the following zones (from tooth to JE): sublamina lucida (95 Å), lamina densa (400 Å), lamina lucida (140 Å), and the hemidesmosome. The special components integrating this unit consisted of pyramidal particles on the inner surface of the peripheral density and fine filaments penetrating the lamina densa, lamina lucida, and the peripheral density. The 140 Å lamina lucida described by the author extended from the peripheral density of the hemidesmosome to the lamina densa.

Stern (1981) reported a width of 400 Å for the lamina lucida. The author's definition of the lamina lucida was the structure between the outer leaflet of the epithelial cell membrane and the lamina densa.

Sabag et al. (1981) described the attachment of epithelium to the cementum root surface to be mediated by 4 to 8 hemidesmosomes per micron at the coronal zone of epithelial attachment and 2 hemidesmosomes per micron in the apical zone. Because of this arrangement, the authors suggested that the coronal zone of the cemental surface may exhibit enhanced adhesion of epithelial attachment when compared to the apical zone.

Conclusive evidence regarding the origin of the dental cuticle is not yet available. Some investigators consider it is a product of the junctional epithelium but the JE may attach to teeth without an intervening cuticle. Friedman et al. (1993) suggested that it represents an accumulation of material from plaque metabolites and inflammatory infiltrates.

ORIGIN OF JUNCTIONAL EPITHELIUM

The following description of the origin of the JE is provided by Lindhe (1989) and is supported by the work of Engler et al. (1965) in Rhesus monkeys. Following enamel formation, the height of the ameloblasts is reduced, combining with the outer enamel epithelium to form a flat cuboidal layer called the reduced enamel epithelium (REE). The cells of the REE produce a basal lamina that is joined to the enamel by hemidesmosomes. As the erupting tooth nears the oral epithelium (OE), the cells of the outer layer of the REE and the basal layer of the OE manifest increased mitotic activity while the former ameloblasts do not divide. When the tooth has penetrated the tissue, the REE and OE fuse at the incisal edge of the tooth. Immediately apical to the incisal edge, the enamel is covered by junctional epithelium; the REE covers the enamel surface apical to the JE. As the tooth becomes fully erupted, all cells of the REE are gradually replaced by cells of the JE. In periodontal disease, the junctional epithelium migrates apically along the root surface. An in vitro study by Terranova and Lyall (1986) suggests that this epithelial migration and subsequent attachment and growth are mediated by laminin and other basement membrane components.

CHARACTERISTICS OF THE JUNCTIONAL EPITHELIUM

Lindhe (1989) described the JE as having a free surface at the base of the gingival sulcus. From this surface, the cells of the JE are desquamated. The JE cells have a basal lamina/hemidesmosomal attachment to both the gingival connective tissue and tooth surface. There are several morphological differences between JE and OE: JE cells are larger than OE cells; the JE intercellular space is wider than that of OE; and, fewer desmosomes interconnect cells of the JE.

Gargiulo et al. (1961) studied the dimensions and relations of the dento-gingival junction in man. The mean average length of the epithelial attachment (phases I through IV) was 0.97 mm with a range of 0.71 to 1.35 mm (mean average).

REPAIR OF THE JUNCTIONAL EPITHELIUM AFTER SURGERY

Listgarten (1972) performed gingivectomies on two young cynomolgus monkeys and examined block sections

of tooth/gingiva at 12 days, and 3, 4, and 7 weeks post-surgery. Electron microscopic examination revealed that the JE was completely re-established at 12 days from an apical extension of the oral epithelium and that the epithelium was 6 to 12 cells thick in the region of the sulcus and 1 cell thick at the apical extent. Hemidesmosomes appeared to form prior to the basal lamina. The basal lamina initially formed in close proximity to the hemidesmosomes at both the tooth and connective tissue interface. At 4 to 7 weeks, the basal lamina appeared complete. If the JE is merely separated and not removed, the reattachment appears to occur rapidly from the apical region coronally with complete reformation of the hemidesmosomal attachment within 5 days (Taylor and Campbell, 1972). A human study by Frank and Fiore-Donno (1972) evaluated healing following an inverse bevel flap in man and demonstrated that a newly differentiated attachment apparatus with normal hemidesmosomal attachment is possible following surgery. This new attachment apparatus was seen on cementum as well as dentin.

TURNOVER OF THE ATTACHMENT EPITHELIUM

Skougaard and Beagrie (1962) investigated renewal time in the gingival epithelium of marmosets using injections of tritiated thymidine and autoradiography. Epithelial cells in the attached gingiva exhibited a renewal rate of 10.4 days, whereas the corresponding rate for the epithelial cuff was 5.8 days. The results suggested that the concept of a permanent epithelial attachment was unlikely given the rapid renewal rate of the epithelial cuff.

Anderson and Stern (1966) used autoradiography to study the rate of migration of attachment epithelium. The authors observed that the rate was comparable to the rate of tooth eruption, suggesting that the location of the attachment is relatively stable. Additionally, it was concluded that the attachment epithelium is not a proliferative tissue since epithelial cells, which give rise to the attachment epithelium, seemingly migrate from the downgrowing oral epithelium. It should be noted that this study was performed on continuously erupting rat incisors. The sulcular epithelium is keratinized, and there is a tendency for hair and bedding to become impacted into the sulcus.

CONNECTIVE TISSUE INFLUENCE ON THE JUNCTIONAL EPITHELIUM

Ten Cate (1989) describes a recombination experiment which demonstrated the nature of connective tissue influence on JE. Epithelial cells overlying lamina propria exhibited development of normally maturing stratified squamous epithelium, whereas epithelial cells combined with deep immature connective tissue did not show this normal pattern. The epithelium which formed appeared very similar to JE. The author suggested that the JE was supported by deep connective tissue (periodontal ligament) and the gingival and sulcular epithelium was supported by a lamina propria. The immature character of the JE was characterized by the presence of hemidesmosomes which were not seen in gingival and sulcular epithelium but which were necessary for epithelial attachment.

EPITHELIAL ATTACHMENT TO IMPLANTS

Listgarten et al. (1991) provided a comparison of periodontal tissues around dental implants and the natural dentition. As in the natural dentition, the junctional epithelium is attached to the implant surface by a basal lamina and hemidesmosomes. Based on these observations, the authors suggested that the epithelial relationship to the dental implants was, therefore, very similar to that of the natural dentition.

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Section 3. Teeth and Supporting Structures

DERIVATION OF SUPPORTING TISSUES

The development of a tooth is divided into bud, cap, and bell stages based on morphology and the shape of the tooth germ's epithelial component. Formative and supporting elements can be identified in the cap stage. The dental papilla (condensed ectomesenchymal cells) form the pulp and dentition. The condensed ectomesenchyme encapsulating the enamel organ and limiting the dental papilla is termed the dental follicle. This is the progenitor of the supporting tissues of the tooth (Sicher, 1962).

ROOT DEVELOPMENT

Root development begins after enamel and dentin formation has reached the future CEJ, just prior to eruption. Mitotic activity within the cervical loop (formed by the fusion of the inner and outer epithelium) results in an apical elongation of the double epithelial cell layer, known as Hertwig's epithelial root sheath. Cells from the root sheath induce the differentiation of pulpal connective tissue cells into odontoblasts. These cells secrete dentin matrix between themselves and the cells of the epithelial root sheath which separates from the dentin. As the root sheath progressively fragments, ectomesenchymal cells from the dental follicle traverse the fenestrated epithelial root sheath and contact the newly formed root dentin. These cells become cementoblasts producing the matrix which mineralizes and anchors the periodontal ligament (PDL). The PDL cells and fiber bundles are also derived from the dental follicle (Sicher, 1962). Fenestrations occur throughout this former sheath, resulting in a network of epithelial rests known as the rests of Malassez.

Histologically, the rests of Malassez appear as clusters of small solid aggregates of epithelial cells which are dispersed at intervals down the cemental side of the PDL like rungs of a ladder. It has been suggested that the epithelial rests form a continuous network around the entire root surface. While the cell rests persist throughout life, decreased prominence has been reported with age. It has also been observed that the rests are more numerous in the gingival region and decrease towards the apex. Potential roles of the rests of Malassez are numerous. Spouge (1980) discussed two main factors concerning the role of rests in periodontitis. These included anatomical continuity with attachment epithelium, and the potential for reactive proliferation of epithelium secondary to inflammation. Tooth transplantation studies (Löe and Waerhaug, 1961) indicated that healing with normal PDL was associated with the presence of prominent epithelial rests, and healing with ankylosis or root resorption demonstrated absence of rests.

CEMENTUM

Structure

Selvig (1965) examined extracted human teeth using light microscopy and microradiographs. He reported that acellular cementum was present primarily at the coronal aspect of the roots. Formation was much slower than cellular cementum. A 10 to 50 μm thick inner zone was noted which appeared less radiodense than the outer layers and contained thin, radially oriented collagen fibrils. The remaining thickness of acellular cementum exhibited numerous radiopaque incremental lines running parallel to the surface. Radiating fibers existed in all layers of acellular cementum. Electron microscopy (EM) revealed a regular appearance consisting of bundles of densely packed fibrils at right angles to the surface. The PDL cells were separated from the calcified surface by a 3 to 5 μm wide zone of precementum consisting of densely packed and completely calcified fibers. Bundles of fibrils in the inner layer were less densely mineralized.

Cellular cementum was generally more irregular and less radiopaque than acellular cementum. It was usually located in the more apical areas of the root and was deposited at a more rapid rate than acellular cementum to compensate for passive eruption. Layers were separated by radiopaque resting lines or layers of acellular cementum. Sharpey's fibers were radially oriented and were generally calcified, but areas containing uncalcified channels of fibers were noted. The calcified Sharpey's fibers (10 μm) presented an uncalcified core (1 to 2 μm) and a highly calcified peripheral portion also seen in alveolar bone. The fibers were separated by calcified matrix containing more randomly arranged fibers.

Biological Changes

Selvig (1969) used microradiography and EM to characterize changes occurring in root surfaces exposed to the oral environment as a result of periodontal disease. Cemental alteration may include no changes (rare), caries (demineralization and remineralization possible), hypermineralization, or a combination of these factors. Since the crystals in hypermineralized areas were thicker and showed a more perfect crystal structure than in unaltered cementum, the author concluded that the increased mineralization was due to growth of pre-existing crystals. When a clean root surface was created by thorough scaling and root planing, a highly mineralized surface zone and a subsurface cuticle reappeared within 3 to 4 weeks. Active root caries was established in one instance within 7 days.

Stahl (1975) reviewed the histologic structure of cementum and the changes associated with periodontal disease and its repair potential. Changes in diseased cementum included decreased number of cementoblasts, decreased crystal size, less mineralization, and cemental resorption. The most common changes reported in exposed cementum in-

cluded increased mineralization and/or demineralization. Changes in unexposed cementum (Selvig, 1966) were observed immediately below the junctional epithelium and consisted of resorption at sites of active inflammation. Accompanying changes included partial destruction of collagen 0.5 to 1 mm below the JE and complete collagen destruction in a narrow zone immediately below the epithelium. The author suggests that inflammation may cause pathologic changes in the acellular cementum by lysis of cementoblasts, changes in precementum, and cemental resorption.

Endotoxin

Endotoxin is a heat-stable, lipopolysaccharide complex found in the cell wall of many Gram-negative microorganisms. It has been shown to be pro-inflammatory, cytotoxic, and pyrogenic, and has been implicated in the causation of periodontal disease.

Surface Absorption

Hatfield and Baumhammers (1971) reported that an inhibitory substance penetrates the surface of cementum associated with periodontitis, preventing the growth of epithelial cells in tissue culture.

In a similar study, Aleo et al. (1974) reported the presence of endotoxin and/or endotoxin-like products in the cementum of periodontally involved teeth. These substances inhibited the proliferation of cultured mouse fibroblasts and were lethal in high concentrations. Phenol extraction resulted in cementum free of endotoxin.

In a follow-up study using cultured human gingival fibroblasts, the same authors (Aleo et al., 1975) demonstrated that these cells do not attach to root surfaces of periodontally involved teeth. Attachment was observed after the cementum was mechanically removed or the endotoxin extracted with phenol-water.

Jones and O'Leary (1978) conducted a clinical study to assess the effectiveness of root planing in removing endotoxin from the roots of periodontally involved teeth. The teeth were assayed for endotoxin using the limulus amoebocyte lysate test (LAL). Results showed that the teeth that were root planed (1.2 ng/ml) were nearly as free of endotoxin as uninvolved teeth (0.25 ng/ml).

Nishimini and O'Leary (1979) compared ultrasonics to hand instrumentation to determine the relative effectiveness of endotoxin removal. The authors observed that root planed teeth had less endotoxin (2.09 ng/ml) than those treated with the ultrasonic instrumentation (16.8 ng/ml). Extracted unerupted molar roots averaged 1.46 ng/ml. The authors concluded that meticulously performed root planing will essentially eliminate all endotoxin from diseased roots.

Daly et al. (1982) investigated the presence and location of lipid and polysaccharide in periodontally diseased cementum. Histological staining revealed polysaccharide and lipid deposits penetrating 3 to 12 μm into the cemental surface. Specimens also demonstrated bacteria at depths of up to 12 μm beneath the cementum and within defects at the CEJ.

These studies provide a rationale for the meticulous root planing of the root surface in order to remove endotoxin, an agent believed to inhibit the healing process.

Surface Phenomenon

Nakib et al. (1982) immersed healthy and diseased teeth in varying concentrations of *E. coli* endotoxin. The teeth were then examined by indirect immunofluorescence and autoradiography for the presence of endotoxin within the cementum. The authors suggested that a weak bond existed between the endotoxin and root surface, since brushing the endotoxin-treated roots for 1 minute removed most of the adherent endotoxin.

Wilson and Moore (1986) developed a method to avoid some of the problems encountered in using the LAL test to detect lipopolysaccharides (LPS) in root surface materials. Analysis is complicated by the low concentrations of LPS presumed to be present on the root and by the fact that the LAL assay is not specific for LPS. Thrombin, thromboplastin, ribonucleic acids, ribonuclease, soluble peptidoglycan fragments, and lipoteichoic acid are also capable of activating coagulation of LAL. The authors reported that most of the LAL activity in root surface materials can be attributed to LPS and that through the sequence of Westphal extraction and ultracentrifugation, the interference associated with LAL assay by LAL-reactive materials (other than LPS) could be reduced to very low levels. This study also confirmed the efficacy of Polymyxin B/Sepharose 4B affinity chromatography in extracting LPS.

Moore and Wilson (1986) used sequential Westphal extraction, ultracentrifugation, and Polymyxin B/Sepharose 4B affinity chromatography to identify and quantitate LPS in periodontally involved root surfaces. In involved roots, 39% of the LPS was loosely adherent and 60% was removed by brushing.

Ito et al. (1985) studied whether or not the material extracted from periodontally involved root surfaces by the hot phenol procedure was endotoxin. The proposed method of confirmation was the local Shwartzman phenomenon in gingival and abdominal sites of rabbits. The material extracted from periodontally involved roots revealed no evidence of LPS, but did demonstrate mild to severe inflammation. Based on these observations, the authors suggested that a heat-stable, phenol-water extractable and highly irritating substance can be obtained from periodontally involved root surfaces. They also noted that this substance may not be endotoxin. It should be noted that these teeth were washed and brushed prior to the extraction of the endotoxin, which, according to other studies (Moore and Wilson, 1986), may remove up to 99% of the endotoxin.

Based on these studies, the excessive removal of cementum during root planing to eliminate endotoxins from the exposed root has been questioned.

Zwarych and Quigley (1965) examined histologic sections through mice molars to determine if an intermediate plexus exists in mammals. The authors concluded that an intermediate plexus did not exist and that the principal fibers were continuous from the alveolar bone to the cementum. Fiber bundles attached to the alveolar bone were large in diameter, but less numerous than those inserting into cementum. Based on these observations, the authors suggested that displacement forces applied to a given area of the cementum are distributed to a greater area of the alveolar septum.

BONE

Anatomy

Osseous tissue is rigid connective tissue that is normally organized into definite structures termed bones. Grossly, two types of bone may be distinguished: spongy or cancellous and cortical or compact. The two varieties have the same types of cells and intercellular substance, but differ in the arrangement of components and ratio of marrow space to bone substance.

Compact bone (cortical) consists of Haversian bone containing Haversian canals which serve as tunnels for small blood vessels. These canals are parallel to each other and the long axis of the bone. The blood supply comes either from the periosteum or the marrow cavity through Volkmann's canals which run perpendicular to the long axis of the bone. The lamellae may be circular, circumferential (periosteal or endosteal), or interstitial. The circular lamellae are approximately 5 μm thick and encircle the Haversian canals. They actually encase the osteocytes in lacunae, which communicate with other osteocytes via canaliculi. The external or internal circumferential lamellae (depending on location, endosteal or periosteal) run parallel to the periosteal or endosteal bone surfaces. Interstitial lamellae occur between circular lamellae and originate from old Haversian systems or peripheral lamellar bone that have been spared by the resorptive and remodeling processes. The Haversian canal and its blood vessels constitute the center of a functional unit termed the osteon. This unit also consists of scattered and concentrically arranged osteocytes and circular lamellae.

Spongy (cancellous) bone consists of a trabeculae or a network of spicules which follow the lines of stress. The spaces between the trabeculae vary in size and shape. This tissue is either of hemopoietic, myeloid, or fat origin. The trabeculae consist of osteocytes embedded in lacunae and irregularly arranged bony lamellae. There is not a distinct Haversian system since blood vessels are never very far away (less than 0.2 mm). The surface of the trabeculae is covered with a thin layer of osteoprogenitor cells and occasional osteoclasts (endosteum).

Bone matrix consists of an intercellular organic matrix with collagenous fibrils in an amorphous ground substance containing inorganic salts. The ground substance consists of protein-polysaccharides and glycoproteins. The inorganic salts are calcium, phosphorus, calcium carbonate, and sodium crystals bound to collagen fibrils. They constitute up to 65% of the bone's weight.

Bone cells include osteoblasts, osteocytes, and osteoclasts. Osteocytes are present in lacunae, surrounded by a 1 to 2 μm mineralized matrix zone. Collagen fibrils without mineralization are present near cell membranes. Cytoplasmic processes extending into canaliculi are relatively inactive cells but may respond to parathormone. Osteoblasts are bone-forming cells which synthesize type I collagen and ground substance (proteoglycans, glycoproteins) and initiate the process of mineralization by means of membrane bound vesicles. Osteoblasts become trapped in the matrix and become osteocytes. Osteoclasts are bone-resorbing cells. They are multinucleated giant cells found in Howship's lacunae on bone surfaces and enveloping the end of bone spicules. They have numerous mitochondria and lysosomes containing acid phosphatase. The plasma membrane at the bone surface is arranged in folds and clefts called the ruffled border. At the periphery is an annular (clear) zone believed to seal off the ruffled border.

The periosteum consists of cambium (inner) and reticular layers (outer) that are up to 10 to 12 cells thick. The reticular layer contains dense connective tissue with fibroblasts. In active bone growth or following injury a prominent osteoblastic (osteoclastic) zone is seen. The endosteum lines the inner surface of marrow cavities and larger Haversian canals. It is the internal equivalent of the cambium layer of the periosteum (Rhodin, 1974).

Clinical Considerations

Boyne (1965) studied the healing extraction site in humans using tetracycline to label newly mineralized bone. The first evidence of calcified osseous matrix was noted at days 7 and 8 outside the alveolus along the entire length of the cribriform plate. Bone formation in the socket was first observed 9 to 10 days postoperatively along the lateral walls.

Elliott and Bowers (1963) studied human skulls and reported: 1) an average of 20% of the sites had some type of defect; 2) fenestrations were more common in the maxilla while dehiscences were more common in the mandible; 3) both types of defects frequently occur bilaterally; and 4) defects were most often seen in areas where a thin alveolus was present.

Manson (1963) used radiographs and microradiography to examine human mandibles (autopsy) with teeth in situ. He reported no difference in the mineral content of the bone in regions of the tooth socket and the adjacent bone. A line similar to the lamina dura could be produced as an artifact. The author concluded that the appearance of the lamina

dura is determined as much by the shape and position of the tooth root in relation to the x-ray beam as by the integrity of the bony plate.

Greenstein et al. (1981) evaluated the association between crestal lamina dura and clinical parameters of bleeding points, pockets, or attachment loss and presence of a crestal lamina dura. The authors suggested that caution be exercised when using the integrity of the crestal lamina dura as an indicator for periodontal treatment.

CUTICLE

Friedman et al. (1993) used light (LM) and transmission electron microscopy (TEM) to examine the dental cuticle of human teeth with root surfaces that had been exposed to the oral cavity. With LM, the cuticle presented as a thin dark staining line next to dentin. With TEM, the dental cuticle was about 1.4 μm in width (variable), lobulated in most areas, and commonly manifested bands of deposition. The cuticle occupied the space from the subgingival plaque to the junctional epithelium. The authors suggested that the cuticle represented an accumulation of inflammatory infiltrates or metabolites of plaque. Speculation was offered that the cuticle may mediate attachment of junctional epithelium as well as bacteria to the tooth surface.

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CHAPTER 2. PERIODONTAL DISEASES

Section 1. Gingivitis and Periodontitis

DEFINITIONS

Inflammation: A localized protective response elicited by injury or destruction of tissue, which serves to destroy, dilute, or wall off both the injurious agent and the injured tissue. A cellular and vascular reaction of tissues to injury.

Gingivitis: Inflammation of the gingiva.

Periodontitis: Inflammation of the supporting tissues of the teeth. Usually a progressively destructive change leading to loss of bone and periodontal ligament. An extension of inflammation from gingiva into the adjacent bone and ligament.

Adult Periodontitis: A form of periodontitis that usually has an onset beyond age 35. Bone resorption usually progresses slowly and predominantly in the horizontal direction. Well-known local environmental factors are prominent and abnormalities in host defense have not been found.

Juvenile Periodontitis: May be generalized or localized; onset during the circumpubertal period; familial distribution; relative paucity of microbial plaque; less acute signs of inflammation than would be expected based upon the severity of destruction; may be associated with abnormalities in leukocyte chemotaxis and bacteriocidal activity.

Prepubertal Periodontitis: May be generalized or localized; onset between eruption of the primary dentition and puberty; may affect the primary and mixed dentition; characterized by severe gingival inflammation, rapid bone loss, tooth mobility, and tooth loss.

Refractory Periodontitis: Includes patients who are unresponsive to any treatment provided—whatever the thoroughness or frequency—as well as patients with recurrent disease at single or multiple sites.

The common forms of gingivitis and periodontitis are inflammatory processes initiated by bacterial plaque. Bacteria in the gingival crevice have been portrayed as an indigenous flora that trigger a self-destructive inflammatory response or as lethal pathogens which invade the host tissues to spark episodic bursts of disease activity. Current evidence suggests that bacterial products penetrate intact sulcular epithelium to initiate inflammation. Substantial evidence exists that bacteria can invade the host tissues as well.

There are multiple defense mechanisms in the gingival sulcus: 1) the primary line of defense by polymorphonuclear leukocytes (PMNs) can form a wall between plaque and the epithelium; 2) the epithelial barrier is permeable and the site of ulceration, an early and important event in the development of gingivitis; 3) saliva, which contains secretory IgA, leukocytes, and lysozymes, aids host defense; 4) gingival fluid may flush substances from the pocket and contains PMNs and plasma factors, such as complement, non-specific opsonins, and immunoglobulins; and, 5) finally, the high rate of tissue turnover in the sulcus is protective (Page, 1986; Miyasaki, 1991).

GINGIVITIS

Etiology (See Page, 1986, for review)

Overwhelming evidence suggests that microbial plaque near the cervical region of the teeth causes gingivitis (Löe et al., 1965; Page, 1976; Moore et al., 1982). Healthy gingiva sulci typically contains a flora of *Streptococcus* and additional species including *Actinomyces, Veillonella,* and *Capnocytophaga. Streptococci* and *Actinomyces* may comprise over 85% of the microbial flora in health (Slots, 1979; Moore et al., 1982). Gingivitis has been characterized by a shift from a *Streptococcus*-dominated plaque to an *Actinomyces*-dominated plaque. Developing gingivitis has been associated with increased numbers of *A. israelii* and *Bacteroides*, especially *Porphyromonas gingivalis*. Gingivitis has also been associated with an increase in motile bacteria and spirochetes (Listgarten et al., 1978). Moore et al. (1982) described a great deal of individual variation in the development of gingivitis flora and reported a progression of species colonizing in a sequential matter in gingivitis. They associated gingivitis with specific *Actinomyces, Streptoccocus, Fusobacterium, Veillonella,* and *Treponema*. Savitt and Socransky (1984) found *Eikenella corrodens, Fusobacterium,* and *C. gingivalis* elevated in gingivitis. *Prevotella intermedia* has been associated with pregnancy gingivitis (Kornman and Loesche, 1980).

Keratinized tissue (attached gingiva) is not an essential prerequisite to the maintenance of periodontal health when dental plaque is controlled (Kennedy et al., 1985). Malposed teeth only weakly correlate with gingivitis. A high sucrose diet may increase gingivitis (Sidi and Ashley, 1984).

Initiation

The initiation of the disease process is multifactorial. Currently, more than 40 components of gingival crevicular fluid (GCF) have been studied to determine their role in the pathogenesis of the disease process. Schwartz et al.

(1972) suggested that endotoxin penetrated healthy, intact crevicular epithelium. Labeled endotoxin was observed primarily in a gradient adjacent to the basal epithelium in the histologic sections examined in this study. Caffesse and Nasjleti (1976) suggested that bacterial hyaluronidase might act as a promoting agent for the penetration of bacterial collagenase through intact gingival epithelium. Hyaluronidase penetrated intact sulcular epithelium and also acted as a "spreading factor," widening the intercellular spaces and facilitating the penetration of labeled collagenase which failed to penetrate intact crevicular epithelium when used alone.

Most collagenase in the sulcus appears to originate from the host (Christner, 1980). Collagenase activity is present in the gingival tissue and in GCF in spontaneously occurring and experimental gingivitis in humans, and the amounts increase almost linearly with increasing severity of inflammation (see Page, 1992, for review). Activities similar to cathepsins B, D, H, and L are present in inflamed gingival tissue and in GCF harvested from inflamed sites (Kunimatsu et al., 1990). Neutral proteases resembling elastase, trypsin, chymotrypsin, and glycylprolyl dipeptidase that can degrade non-collagenous matrix components have been recovered in GCF from inflamed sites with elastase activity being the highest activity observed (Cox and Eley, 1989).

Alkaline phosphatase activity has been associated with bone metabolism and neutrophilic granulocytes (Ishikawa and Cimasoni, 1970). B-glucuronidase and arylsulfatase (Lamster et al., 1985, 1986; Oshrain et al., 1984) levels are associated with inflammation, probing depth, and alveolar bone loss. Cytokines, specifically IL-lα and IL-lβ are present in inflamed gingival tissue (Masada et al., 1990).

Mechanisms

Indirect mechanisms (host response) combine with the direct mechanisms (bacteria and their products) to determine the intensity of the inflammatory reaction (Genco, 1992). Bacterial products penetrate or adhere to tissues and induce a host response by activating complement and causing migration of PMNs (Miyasaki, 1991). Antibodies and cytokines (called acute phase proteins or C-reactive proteins) dramatically increase in inflammation. When bound to bacteria, C-reactive proteins promote binding to complement, which stimulates phagocytes to engulf bacteria (Kinane et al., 1992). Complement is activated in the classical pathway by antigen-antibody and is activated directly via the alternate pathway by bacterial components like LPS (Montgomery et al., 1986; Monefeldt and Tollefsen, 1993). Complement activation results in consequent release of C3A and C5A which trigger mast cells to degranulate and release histamine, in turn, increasing vascular permability. The increased vascular permeability together with leucocyte emigration leads to stasis and inflammatory exudate from the vessels (Genco, 1992). C3A and C5A and bacterial product FMLP are chemoattractants for neutrophils (Schenkein, 1992). In turn, stimulated neutrophils release products which mediate damage to host tissues (Altman, 1992).

Crevicular epithelium may become more permeable with inflammation (Thilander, 1968). Thilander studied gingival biopsies from 12 gingivitis patients using transmission electron microscopy (TEM) and reported: 1) slight changes with dilation of vessels, focal edema, and increased epithelial spaces; 2) moderate changes in which intermediate and tight junctions in the epithelium were lost, areas of basement membrance were missing, and leukocytes appeared to be migrating between the epithelial cells; and 3) marked changes where cytoplasmic changes suggested degeneration.

The Initial Lesion (Experimental Histology)

The pathogenesis of inflammatory periodontal disease remains remarkably consistent with the model of Page and Schroeder (1976). In this model, gingivitis is represented by the initial, early, and established lesion (Page, 1986). The initial lesion is a histological entity which would most likely correlate with a preclinical gingivitis. The initial lesion appears 2 to 4 days after plaque accumulation in previously healthy gingiva and is localized to the gingival sulcus, including the junctional epithelium and the most coronal part of the connective tissue (rarely more than 5% to 10% is involved). Characteristics of the initial lesion include: 1) vasculitis subsequent to the junctional epithelium; 2) extravascular fibrin and serum protein; 3) loss of perivascular collagen; 4) increased migration of leukocytes into the junctional epithelium and sulcus; 5) alteration of the coronal part of the junctional epithelium; and 6) increased crevicular fluid. Exacerbations of acute inflammation may be associated with abscess formation or disease progression (Page, 1976).

The Early Lesion (Experimental Histology)

Clinically, the early lesion correlates with early gingivitis. The early lesion appears 4 to 7 days after plaque accumulation and is characterized by a dense lymphoid infiltrate. Characteristic features include: 1) features of initial lesion are present and progress; i.e., GCF increases with inflammation and crevicular leukocytes peak at 4 to 7 days; 2) collagen loss increases and may reach 60% to 70% in the inflamed tissue; 3) cytoplasmic alterations appear in resident fibroblasts; 4) lymphoid cells accumulate subjacent to the junctional epithelium and make up approximately 75% of the infiltrate (approximately 5% to 15% of the total connective tissue is infiltrated); and 5) basal cells of the junctional epithelium begin to proliferate. Lymphoid cells have a non-specific affinity for inflamed tissue. Cellular hypersensitivity may play a role in the early lesion (Page, 1976).

The Established Lesion (Experimental Histology)

Chronic clinical adult gingivitis correlates with the established lesion which occurs 2 to 3 weeks after the beginning of plaque accumulation. The key feature of the

established lesion is the presence of plasma cells. The lesion is centered around the bottom of the sulcus and relatively confined; features of earlier lesions persist. The predominant immunoglobin is IgG with a small but significant amount of IgA and rarely IgM. Characteristics include: 1) persistence of the manifestations of acute inflammation; 2) continued loss of connective tissue: fibrosis and scarring may also occur; 3) predominance of plasma cells without appreciable bone loss; 4) extravascular immunoglobulins; and 5) the proliferation, apical migration, and lateral extension of the junctional epithelium. Early pocket formation may or may not occur. The established lesion may remain stable for extended periods of time (Page, 1976).

Seymour (1983) conducted an experimental gingivitis study with dental students. The students ceased all forms of oral hygiene for 21 days and had biopsies taken from each first molar at 0, 4, 8, and 21 days. There was an association between the formation of plaque and the development of gingivitis. A lymphocyte-dominated lesion developed between 4 and 7 days and continued up to day 21. A plasma cell lesion did not develop within this time period. The lack of plasma cells in experimental gingivitis is also supported by the work of Brecx et al. (1988).

Quirynen et al. (1991) also conducted an experimental gingivitis study to quantify the influence of gingival inflammation on the rate of plaque formation. Plaque was found to accumulate at a significantly greater rate when gingival tissues were already inflamed; initially inflamed tissues had a 29% greater extension of plaque than initially healthy tissues.

Effects on Cementum

In a TEM study, Selvig (1966) examined 9 extracted teeth from patients and reported early changes which included an increase in small diameter collagen fibrils, presence of microfibrils, and a loss of functional orientation in the periodontal ligaments (PDL) opposite the cementum. Below the epithelial attachment there was a complete destruction of collagen and Sharpey's fibers accompanied by leukocyte infiltration. In the area of fiber dissolution, crystals in the outer layers of cementum were decreased in number and size. Loss of mineralization ranged from 4 to 18 μm. Above this region there was a zone of epithelial attachment to cementum. Degradation of collagen was felt to precede apical migration of epithelium on cementum.

Gingivitis and Gingival Recession

Gingival recession and clefts may form in some sites due to inflammation. Epithelial proliferation in the presence of inflammation may lead to anastomoses between the crevicular and mucosal epithelium in a susceptible site with subsequent recession.

Surgical procedures, trauma, and orthodontics may lead to the resorption of thin alveolar bone (as thin as 0.15 mm) creating conditions favorable for recession. A narrow width (thickness) of attached gingiva and prominent roots may represent susceptible sites for recession due to inflammation (Novaes et al., 1975).

Prepubertal Gingivitis

A 21-day experimental gingivitis study comparing 6 children (age 4 to 5 years) with 6 dental students (age 23 to 29 years) indicated that preschool children developed gingivitis less readily than adults. Children developed significantly less gingival exudate and bleeding units (8.6% versus 43.9%) than adults at day 21 in teeth with the same plaque index. Crevicular leukocytes increased in children, but to a lesser degree than adults. These results were interpreted to suggest a different vascular response between children and adults and a relatively similar cellular response (Mattsson, 1978).

Subsequent studies suggest that in childhood, there is no clear-cut age at which the gingival reaction to bacterial irritation becomes similar to that observed in adults. Instead, gingival activity seems to increase gradually from early childhood to adult age (Mattson and Goldberg, 1985).

Longhurst et al. (1977) reported markedly fewer plasma cells in inflamed gingival tissue from young children than from adults. Morphologically the lesions consisted of approximately 70% lymphocytes, between 11% and 26% macrophages, and fewer numbers of polymorphonuclear leukocytes and plasma cells (Seymour et al., 1981).

High levels of spirochetes (> 2%) were observed only in the prepubertal group (Wojcicki et al., 1987). A study by Moore et al. (1987) also indicated that the young children had fewer cultivable spirochetes than adults. Significant increases in subgingival spirochetes occurred when bleeding upon probing was observed as a sign of inflammation (Armitage et al., 1982). Therefore, a possible correlation exists between fewer spirochetes and a lower incidence of gingivitis found in prepubertal children.

Significance of Gingivitis

(See Page, 1986, for review.) Gingivitis does not necessarily progress to periodontitis. Initially, gingivitis was considered physiologic. In the 1950s and 1960s, the concept that untreated gingivitis progressed to periodontitis emerged. Observations by Marshall-Day et al. (1955) that gingivitis without evidence of bone loss was confined to a younger age group, and bone loss without clinical evidence of gingival disturbance was rarely observed strengthened this view. Suomi et al. (1971) found a loss of epithelial attachment at a rate more than three and one-half times higher in a group with greater gingival inflammation scores. In a longitudinal study of a Sri Lankan population with chronic gingivitis, Löe et al. (1986) reported 89% of the subjects developed periodontitis. However, episodic bursts of attachment loss were observed which did not correlate with clinical gingivitis (Haffajee et al., 1983).

Ranney (1986) suggested that gingivitis, at least as detectable histologically, has not been ruled out as a necessary

TABLE 1. IMMUNOLOGIC FINDINGS OF IMPORTANCE IN THE DIAGNOSIS OF DESQUAMATIVE GINGIVITIS

Underlying Disease	Tissue: Pattern of Immune Deposits	Sera: Types of Antibodies
Pemphigoid, all forms	Epithelial intercellular deposits (100%)	Intercellular antibodies of epithelium (over 95%)
Bullous pemphigoid	Basement membrane zone (100%)	Basement membrane (97%)
Cicatricial pemphigoid	Basement membrane zone (97%)*	Basement membrane (23%)
Lichen planus	Globular deposits (cystoid bodies) in epidermis and dermis (97%) Fibrin deposits along basement membrane	None
Psoriasis	Stratum corneum deposits	None are specific for psoriasis
Other (hormonal, etc.)	Negative or few cystoid bodies	Negative

*Figures in parentheses represent percentage of patients with the respective findings (Nisengard, 1981).

precursor of periodontitis, but it does not invariably progress to periodontitis. He also suggested certain measures of host response (e.g., antibody and PGE_2 in GCF) merit attention as possible predictors of clinical outcome. Waerhaug (1980) observed that inflammation arising from periodontal pockets may affect the deeper supporting structures without being clinically evident in the superficial structures. Is this periodontitis without a pre-existing gingivitis? Only in the immediate sense, since the pocket historically derived from a gingival inflammation. Gingivitis may be considered site specific because averaged measures have little meaning, although Moore et al. (1982) failed to find site specificity in the composition of the flora. When all the data are considered, the most likely interpretation is that gingivitis is a disease; most gingivitis lesions are transient or persistent, but not progressive. However, a small proportion can and does progress to periodontitis.

Desquamative Gingivitis

Desquamative gingivitis is characterized by desquamation or sloughing of the gingival epithelium, leaving an intensely red surface. Sloughing of the epithelium is due to vesiculation and should be considered a sign rather than a disease.

McCarthy et al. (1960) studied 40 cases of desquamative gingivitis over a 12-year period and carefully re-evaluated the existing literature. They concluded that chronic desquamative gingivitis was actually a manifestation of several diseases and therefore had multiple etiologies. Nisengard et al. (1981, 1987) reviewed the manifestations and treatments of these lesions. Approximately 75% of desquamative gingival lesions are dermatologic diseases, with cicatricial pemphigoid and lichen planus comprising over 95% of the dermatoses group. The remaining 25% of desquamative gingival lesions are either of idiopathic origin or can be associated with an endocrine imbalance, aging, chronic infection, or abnormal response to bacterial plaque (Table 1). Most patients are middle-aged and approximately 80% are females. Diagnosis is based on the clinical exam, histology, and immunofluorescence (IF) (Nisengard, 1975). Two types of IF are of prime value for lesions of desquamative gingivitis:

Direct IF. This type detects in vivo bound immunoglobulins and complement in a biopsy specimen by incubating the specimen with fluorescein-labeled antibodies to IgG, IgA, and IgM.

Indirect IF. This type detects serum antibodies and is accomplished by incubation of serum on sections of monkey esophagus with fluorescein-labeled anti-human immunoglobulin.

Pemphigus Vulgaris. Pemphigus vulgaris can involve the skin and mucous membranes. It is seen primarily in older patients of Jewish race and Mediterranean origin. Biopsy and histological examination reveal acantholysis and intraepithelial clefting (Lever et al., 1975). Treatment generally consists of systemic steroids, 40 mg prednisone every other day, with or without immunosuppressant therapy. Referral to a dermatologist is recommended.

Bullous Pemphigoid. Bullous pemphigoid was described as a distinct clinical and histopathologic entity by Lever (1953). The site of the bullae is in the subepidermal region. Lesions are commonly observed on the skin and intraoral lesions are rare.

Cicatricial Pemphigoid. Cicatricial pemphigoid or benign mucous membrane can involve the oral mucosa, the conjunctiva of the eye (symblepharon), and other mucous membranes and skin. The are characterized by subepithelial bullae and basement membrane deposits of IgG and complement. Treatment can consist of nothing for the asymptomatic, topical steroids for mild localized cases, or systemic steroids in more severe cases. Referral to an ophthalmologist is recommended (Nisengard and Neiders, 1981).

Lichen Planus. Lichen planus lesions may manifest as a desquamative lesion in keratotic, erosive, or atrophic forms of the disease (Jandinski et al., 1976). Direct IF shows cystoid bodies of IgG, IgM, and complement in dermis and epidermis. Fibrin deposits can be found along the basement membrane and a degeneration of the basal epithelium with "saw tooth" rete ridges. Treatment is usually with topical steroids.

Psoriasis. Psoriasis presents as skin lesions that are papules covered with silvery scales. Microscopically there is hyperkeratosis, acanthosis, and elongation of the rete ridges and dermal papillae. Several reports suggest that psoriatic

plaques involve an autoimmune mechanism. Direct IF may show antibody and complement bound to stratum corneum antigen. Treatment is with topical steroids.

HIV-RELATED PERIODONTAL DISEASE

Oral Signs and Symptoms

One of the most common features of HIV disease is oral candidiasis. Four major types of candidiasis may be seen in HIV disease: pseudomembranous, hypertrophic, atrophic (erythematous), and angular cheilitis. Silverman et al. (1986) reported that 56% of healthy homosexual males had cultivable oral Candida; prevalence among AIDS-Related Complex (ARC) and AIDS patients was 93% and 70% respectively. The presence of a Candida infection may be predictive of AIDS. Klein et al. (1984) compared 22 adults with unexplained oral candidiasis, reversed T4:T8 ratios, and generalized lymphadenopathy with 20 similar patients who had lymphadenopathy and reversed T4:T8 ratios, but did not have oral candidiasis. Over 50% (13/22) of subjects with candidiasis developed AIDS in a median of 3 months compared to 0/20 patients without candidiasis who were followed for a median of 22 months. Dentists should be aware of the possible implications of oral candidiasis in otherwise clinically healthy individuals. Viral lesions are commonly associated with HIV disease (Greenspan, 1988). Common lesions include oral papillomas, condyloma acuminatum, and focal epithelial hyperplasia, all of which are associated with human papillomaviruses (HPVs). Lesions of recurrent herpes simplex virus are also seen. Herpes zoster (caused by varicella) may also be encountered.

Oral hairy leukoplakia (HL) is a white lesion found primarily on the lateral borders of the tongue. HL was first discovered in 1981 in male homosexuals and has been seen in members of all other risk groups (Greenspan, 1988). Virtually all patients with HL are antibody-positive for HIV. The lesions may also be seen on the buccal/labial mucosa. The surface may be smooth, corrugated, or markedly folded, and does not rub off. HL is probably a virally induced lesion; in fact, Epstein-Barr virus has been detected in biopsies of HL. Silverman et al. (1986) found HL in 28% of 375 homosexual males. Over 50% of patients with HL may go on to develop AIDS.

Oral lesions associated with HIV include recurrent aphthous stomatitis, salivary gland enlargement, and thrombocytopenic purpura (Greenspan, 1988).

The most common AIDS-associated neoplasm is Kaposi's sarcoma (KS), a malignancy arising from lymphatic or vascular endothelium (Greenspan, 1988). According to the Centers for Disease Control and Prevention (CDC), 34% of patients with AIDS have KS. Other studies report an incidence of up to 80%. Intraoral lesions may occur alone or in combination with skin, lymph node, or visceral lesions. They may be blue, red, or purple in color and may be flat or raised, solitary or multiple. The most common oral location for KS is the hard palate, although lesions may occur on the gingiva, buccal mucosa, tongue, and soft palate. On the gingiva, KS may cause diffuse swelling resembling severe gingivitis. Cytomegalovirus (CVM) is commonly associated with KS in HIV-positive subjects. The exact relationship between CMV and KS has not been determined.

Non-Hodgkins lymphoma is another common HIV-associated neoplasm (Greenspan, 1988). It can occur anywhere in the mouth, presenting as a painless swelling which may or may not be ulcerated.

Tuberculosis is another increasingly prevalent infection in those with AIDS.

Over the past several years, an increased incidence and severity of periodontal disease in HIV-infected homosexual males has been observed. These diseases do not respond well to conventional therapy and often progress very rapidly (Winkler et al., 1988). HIV-associated gingivitis (HIV-G) is characterized by generalized gingival swelling and spontaneous bleeding. Necrosis of marginal and papillary gingiva may be seen, and necrosis may extend well into the attached gingiva (Winkler et al., 1988). This lesion has shown a clinical representation described as being "half moon" in appearance at the gingival margin. Frequently, an overlying Candida infection is observed, and successful resolution of the gingivitis may be dependent upon successful treatment of the candidiasis. The most characteristic features of HIV-associated periodontitis (HIV-P) are rapid destruction of the periodontal supporting apparatus and severe soft tissue necrosis. In some HIV-P lesions, more than 90% of the attachment can be destroyed in as little as 3 to 6 months, resulting in early tooth loss (Winkler et al., 1988). Soft tissue necrosis, ulceration, and cratering are often seen immediately adjacent to regions of rapid bone loss. Another distinguishing feature of HIV-P is severe pain, commonly described as a "deep aching" bone pain. Interestingly, the pain often precedes the clinical presentation of HIV-P (Winkler et al., 1988).

Microbiologic assessment of HIV-G and HIV-P shows an increase in *Candida albicans, Prophyromonas gingivalis, Fusobacterium nucleatum, Eikenella corrodens, Actinobacillus actinomycetemcomitans,* and *Wolinella* species compared to controls (Murray et al., 1988). The microbiota associated with HIV-G and HIV-P are similar to those seen in classic periodontitis sites. Interestingly, there is a high prevalence of these organisms in HIV-G. This suggests that HIV-G may be a precursor to HIV-P and that early treatment of the HIV-G lesion may prevent the rapid destruction of periodontal support seen in HIV-P. Winkler and Robinson (1992) note that the organisms in HIV-P are similar to adult periodontitis except that *C. albicans* and *Campylobacteria rectus* are present in higher proportions.

HIV-seropositive homosexual males have significantly higher antibody titers to *A. actinomycetemcomitans, E. cor-*

rodens, F. nucleatum, P. gingivalis, and *P. intermedia* than do HIV-seronegative homosexuals and heterosexuals (Murray et al., 1988).

In a study of 75 HIV-infected individuals (Barr et al., 1992A), only 1% of 218 cultured whole salivas presented cell-free infectious virus. The authors concluded that potential transmission of HIV infection by saliva is unlikely.

Periodontal Management

A widely used treatment for periodontal lesions in HIV+ individuals involves gross scaling to remove visible plaque and calculus deposits and debridement of necrotic tissue when present (Winkler and Robertson, 1992; Grassi et al., 1988). Access for both debridement and for topical antimicrobial therapy is aided by the fact that probing depths are often minimal in the necrotizing ulcerative periodontitis lesion. During debridement, povidine-iodine irrigation has been used for its antiseptic and anesthetic effects (Winkler and Robertson, 1992; Grassi et al., 1988).

Antibiotics should be used with caution due to the increased risk of overgrowth of *Candida albicans* and other microflora associated with HIV infection. To prevent overgrowth of opportunistic microorganisms, the use of a concurrent antifungal agent has been recommended. Narrow spectrum antibiotics, such as metronidazole which leave much of the Gram-positive flora unperturbed, have also been recommended to prevent Candida overgrowth in the management of periodontal lesions in HIV+ individuals (Winkler and Robertson, 1992).

Following initial debridement, follow-up visits are necessary in order to thoroughly remove plaque, calculus, and other deposits and to provide strict plaque control instruction. Home use of an antimicrobial mouthrinse such as chlorhexidine has been shown to be an effective therapeutic aid in reducing the acute symptoms of periodontitis in HIV+ patients and in preventing the recurrence of these lesions (Grassi et al., 1988). Clinical case reports and controlled studies have shown this therapy to be generally effective in reducing the acute symptoms of periodontitis in HIV+ individuals and in halting the progression of the necrotic lesion. The response to therapeutic intervention may, however, depend upon the patient's current HIV stage, intake of systemic medication to treat the HIV infection itself (e.g., zidovudine [AZT], intake of antibiotics, and oral habits (e.g., tobacco smoking).

Scully and McCarthy (1992) emphasize the need for early diagnosis and assessment every 3 months. Local debridement and topical application of antimicrobials such as povidone-iodine or chlorhexidine is beneficial. Yeung et al. (1993) compared the rate of progression of periodontitis between a group of asymptomatic HIV-seropositive men and a healthy control group. HIV-infected patients with preexisting periodontitis tended to experience a greater rate of attachment loss over time compared with controls.

Atypical periodontal lesions observed in HIV+ individuals are often superimposed on conventional periodontitis. In previous studies, the relationship of HIV+ status and severity of periodontitis is unclear. For example, some studies report no direct correlation between the relationship of pocket depth or plaque index with HIV staging (Friedman et al., 1991; Gornitsky et al., 1991). In other studies, attachment loss was greater in later stages of HIV infection (Lucht et al., 1991; Klein et al., 1991) especially when CD4 counts fell below 200 (Barr et al., 1992B). In HIV+ individuals, the prevalence and severity of common forms of periodontal disease such as chronic inflammatory periodontal disease (adult periodontitis) may vary between risk groups due to other factors such as oral hygiene levels, smoking habits, medications, etc.

PERIODONTITIS

Classification: In July 1989, the *Proceedings of the World Workshop in Clinical Periodontics* recommended the following classification of periodontitis:

I. Adult Periodontitis
II. Early-Onset Periodontitis
A. Prepubertal Periodontitis
1. Generalized
2. Localized
B. Juvenile Periodontitis
1. Generalized
2. Localized
C. Rapidly Progressive Periodontitis
III. Periodontitis Associated with Systemic Disease
IV. Necrotizing Ulcerative Periodontitis
V. Refractory Periodontitis

Pathogenesis of Pocket Formation

Changes associated with gingivitis in the periodontal tissues comprise a mixture of resolution and repair that are mostly reversible unless tissue necrosis occurs (Gillet et al., 1990). Pocket development between the tooth surface and gingiva with apical displacement of the junctional epithelium (JE) comprise the first indications of destructive disease (Gillet et al., 1990). Replacement of JE by pocket epithelium results in a thickening of the epithelium and rete ridge development (Muller-Glauser and Schroeder, 1982). The epithelium may become micro-ulcerated and allow penetration of bacteria or bacterial products which could overwhelm the host defenses and lead to a burst of disease activity (Gillet et al., 1990). The initial junctional epithelium defect is within the JE rather than at the JE/tooth interface (Takata and Donath, 1988). The epithelium will grow down the connective tissue/tooth interface to form a long JE. Epithelium along the cementum/soft tissue interface can prevent the establishment of connective tissue reattachment and result in an irreversible change (Gillett et al., 1990).

Lesions of Periodontitis

Page (1976) characterized the histopathological features of periodontitis as:

1. Predominance of plasma cells;
2. Continuing loss of collagen subjacent to the pocket epithelium with fibrosis at more distant sites;
3. Formation of periodontal pockets;
4. Presence of cytopathically altered plasma cells in the absence of altered fibroblasts;
5. Extension of the lesion into alveolar bone and PDL with significant bone loss;
6. Conversion of bone marrow distant from the lesion to fibrous connective tissue; and
7. Widespread manifestation of inflammatory and immunopathologic tissue reaction.

Clinically, periodontitis ranges from periodontal pocket formation, suppuration, fibrosis, destruction of alveolar bone and PDL, mobility, and drifting to eventual tooth exfoliation. Generally, the lesion of periodontitis resembles the established lesion described by Page (1976) with the spread of inflammation into the surrounding tissues with accompanying alveolar bone loss. Bone destruction usually begins along the crest of the interdental septum around communicating blood vessels. Periods of acute exacerbation with pus and abscess formation and periods of quiescence occur.

Distinguishing Periodontitis from Gingivitis

Moskow and Polson (1991) reexamined previous morphologic descriptions of gingival and periodontal inflammation based on a light microscopic study of 105 gingival biopsies and 350 autopsy and surgically-retrieved jaw sections. Clinically normal and marginally inflamed gingiva demonstrated more widespread distribution of inflammatory cells (neutrophils, lymphocytes, and plasma cells) than has usually been reported. A definitive pattern of inflammatory cell type in association with various clinical patterns of soft tissue or osseous destruction could not be established. The character of the cellular infiltrate in both gingivitis and periodontitis was variable and did not seem to be part of a logical consistent progression as Page and Schroeder (1976) described. There did not appear to be a specific cell type characteristic for each "stage" of disease progression. Moskow and Polson (1991) noted that bone changes can occur long before there is any evidence of attachment loss. Internal buttressing of the crestal alveolar bone was a common finding prior to any resorption of the alveolar crest as a result of inflammation. Spread of inflammatory infiltrate into crestal marrow spaces and into the PDL was observed without evidence of gingival fiber destruction. Thus features of the classic advanced lesion were observed prior to the classic early lesion. Since bone changes can occur at a very early stage in the development of the inflammatory lesion, the distinction between gingivitis and periodontitis may not be possible. Intact transeptal fibers were always present over crestal alveolar bone even in the presence of significant osseous resorption (Moskow and Polson, 1991).

Progression to Experimental Periodontitis

In a study by Lindhe et al. (1975), 10 inbred beagle dogs who were brushed twice daily and given a weekly prophylaxis did not subsequently develop periodontitis. Eight of 10 dogs that accumulated plaque on a soft diet developed periodontitis with a loss of attachment of 2.9 mm at 48 months and progressive radiographic bone loss first evident at 24 months. Upper premolars and molars were affected most. These findings suggest that periodontitis might be prevented by removing calculus and providing oral hygiene; gingivitis can proceed to periodontitis; however, 2/10 untreated dogs and some sites did not progress, suggesting variability in host susceptibility.

Spread of Inflammation

Weinmann (1941) studied autopsy histological sections (patients were 14 to 74 years) and observed inflammation following blood vessels into bone marrow spaces while the PDL was generally free of inflammation. Resorption of the alveolar crest generally began on the periosteal side. Akiyoshi and Mori (1967) studied a young autopsy patient using light microscopy and radiographs and concluded that inflammation extends through transeptal fibers along blood vessels directly to interdental canals of the alveolar crest and also indirectly to the alveolar crest via the PDL. In early marginal periodontitis, resorption occurred from the endosteal side of the crest.

Macapanpan et al. (1954) used a rat model (35 rats) with a rubber dam inserted between the upper right first and second molars to determine the effects of trauma on the spread of inflammation up to 72 hours. The authors reported that tension to the PDL reduced resistance to direct infiltration of inflammatory cells from an existing gingivitis, but trauma alone did not cause periodontitis since sites under tension, away from the rubber dam site, were not affected.

Initiation of Active Periodontitis

Allenspach-Petrzilka and Guggenheim (1983) used TEM to examine gingival tissue from 2 female patients with chronic periodontitis and pockets of > 8 mm. The authors reported bacteria in the intercellular spaces of the pocket epithelium and in microabscesses in the underlying connective tissue. Bacteria were observed in the more apical parts of the pocket in the absence of inflammatory tissue. Bacterial invasion leading to focal necrosis or microabscesses might explain the cyclic nature of periodontitis. Listgarten (1986) suggested that increased plaque mass or a reduced host defense might precipitate episodes of periodontal destruction.

Untreated Periodontal Disease

Becker et al. (1979) studied 30 patients (25 to 71 years) initially identified as having periodontal disease who subsequently refused treatment but agreed to a second examination 18 to 115 months after the first (mean 3.72 years). Probing depths, mobility, and radiographs were compared. Untreated patients had a mean tooth loss of 0.36/patient/year, after eliminating 22 teeth initially considered hopeless and 1 patient who lost 25 teeth (the loss rate including the "hopeless" teeth was 0.61 teeth/year). Molars showed the greatest percentage loss. All patients manifested radiographic evidence of progressive bone loss. Increases in probing depth varied from 0.24 mm to 2.46 mm/year, with the greatest increase observed at the lingual interproximal sites.

Goodson et al. (1982) examined 22 patients (at least 20% of pockets 4 mm in depth) monthly for an average period of 13 months (9.3 to 23 months) and monitored attachment loss using regression analysis. Eighty-three percent (83%) of the sites remained unchanged; 5.7% became significantly deeper; and 11.5% became shallower. About one-half of the sites which lost attachment showed spontaneous recovery to the original depth. Deeper sites showed more variability. Attachment loss varied among individuals, with some subjects gaining or losing 4 to 5 mm/year. The findings suggested a dynamic condition of disease exacerbation and remission.

Lindhe et al. (1983) reported on 64 Swedish patients (16 to 64 years, mean 40.5 years) monitored at 3 and 6 years without periodontal therapy who had few sites over 5 mm (0.25%) and a second group of 36 American patients (13 to 63 years, mean 34.3 years) who were followed for 1 year at bimonthly intervals. The Swedish group lost 0.82+0.87 mm the first 3 years and 0.45+0.84 mm the second 3 years. In the American group, 3.2% of the sites lost $>$ 2 mm and 4.3% sites gained $>$ 2 mm attachment over 1 year. These data also suggested rapid loss at some sites rather than progressive bone loss. Also, sites with more advanced attachment loss were not more prone to additional destruction. However, the monitored sites might be affected by bacterial inoculation or redistribution.

Socransky et al. (1984) have suggested a random burst model for periodontitis. The continuous destruction model is challenged by four major lines of evidence: 1) attachment loss rates have been noted which are too fast or too slow to fit a linear progressive model; 2) a large number of sites appear not to be changing; 3) animal studies indicate that disease progression is not continuous in all sites; and 4) severe changes in a specific site are soon brought under control by as yet unknown mechanisms. The authors indicate that sequential (monthly) attachment level measurements have shown that 5.7% sites have probeable attachment gains while 2.8% have losses which ranged from 2 to 5 mm in some sites. Over 6 years, 12% of the sites lost over 2 mm; 40% of these sustained loss in the first 3 years, and half of the sites with no loss in the first 3 years showed loss in the second 3 years.

REFRACTORY PERIODONTITIS

Refractory periodontitis has been variably described. Kornman et al. (1991) referred to treated adult patients who failed to respond to therapy as having refractory periodontitis. Patients who had responded favorably to therapy and then demonstrated signs of disease reactivation were classified as having relapsing (recurrent) periodontitis. Recurrent periodontitis may also apply to disease recurrence in sites without good plaque control or maintenance. Magnusson et al. (1989) described patients who had further disease progression despite good plaque control, treatment by periodontal surgery, tetracycline therapy, and maintenance recalls as refractory. The *Proceedings of the World Workshop in Clinical Periodontics* described refractory periodontitis as disease in multiple sites in patients which continue to demonstrate attachment loss after apparently appropriate therapy. These sites presumably continue to be infected by periodontal pathogens.

Etiology

A patient may be "refractory" to periodontal therapy for several reasons including: 1) inadequate or inappropriate therapy; 2) inadequate maintenance; 3) inadequate plaque control; 4) systemic disease; 5) transient defect in immune response; (6) inadequate or inappropriate immune response; or 7) persistence of periodontal pathogens. Genetic contribution and implications in refractory periodontitis are unknown.

MacFarlane et al. (1992) reported a strong association between a peripheral blood PMN defect and refractory periodontitis. In a retrospective search for associated environmental variables, they found that 90% (28 of 31) of the refractory patients were smokers, demonstrating that local effects of tobacco smoke may compromise the PMN, and should be viewed as an epigenetic factor in periodontitis.

Microbiology

Walker and Gordon (1990) reported *Prevotella intermedia* and *Porphyromonas gingivalis* were the predominant cultivable species in active sites of 24 patients with refractory periodontitis. Magnusson et al. (1991) studied 21 patients who had further disease progression despite treatment by periodontal surgery, tetracycline therapy, and maintenance recalls. This group reported that the microflora found in active sites did not reflect that found in other studies, being predominantly Gram-positive with little of the traditional periodontal pathogens. Large and small motile rods were more frequently associated with active disease. Subjects showed elevated IgG titers to known pathogens, suggestive of prior exposure; however, authors were unable to isolate those species from the pocket. Inability to culture

these specific organisms could be due to the extensive antibiotic, surgical, and non-surgical therapy these individuals received resulting in eradication of the pathogen. The authors postulate that Gram-positive organisms may have become pathogenic and responsible for disease progression in this set of subjects (Magnusson et al., 1991). Conversely, it may be that no specific bacteria are responsible for this disease pattern. Walker et al. (1993) demonstrated that at least 2 patterns or rates of attachment loss may be associated with refractory periodontitis and that each pattern may be indicative of a different flora. The pattern associated with a relatively rapid loss of attachment was characterized by a Gram-negative flora which contained spirochetes, *P. intermedia* and *Fusobacterium* species. A slow, continuous rate was associated with a predominantly Gram-positive flora containing a high proportion of *S. intermedius* and/or a *S. intermedius*-like organism.

Treatment

Treatment for recurrent periodontitis should rely upon conventional periodontal therapy emphasizing good plaque control and maintenance. Treatment for refractory periodontitis may additionally include the following regimens:

Scaling and Root Planing and Amoxicillin/Clavulanate. Magnusson et al. (1989) studied 10 patients who continued to exhibit attachment loss after tetracycline and periodontal surgery. Patients were treated with scaling and root planing and amoxicillin/clavulanate (250 mg TID for 2 weeks). Clinical parameters improved in the first 3 months and were maintained for 12 months post-therapy.

Scaling and Root Planing and Clindamycin. Gordon et al. (1990) evaluated 30 patients with a disease progression (> 3 mm attachment loss) following treatment (scaling and root planing, surgery, and tetracycline). Patients were treated with scaling and root planing and clindamycin (150 mg QID for 7 days). Therapy decreased the annual disease rate (active sites/year) from 8.0% to 0.5%. Clindamycin, in conjunction with periodontal scaling, was effective in suppressing or eliminating the Gram-negative components of the microbiota associated with deep active sites refractory to conventional therapies (Walker and Gordon, 1990).

Several findings suggest that the sequential use of multiple antibiotic agents may offer greater promise as an adjunctive treatment approach for the management of recurrent and/or progressive periodontitis than a single antibiotic regimen.

Scaling and Root Planing with Amoxicillin plus Metronidazole. van Winklehoff et al. (1992) tested 4 deep sites in each of 118 patients positive for *Actinobacillus actinomycetemcomitans* (40 were refractory patients). After therapy consisting of scaling and root planing and metronidazole (250 mg)/amoxicillin (375 mg) TID for 7 days, the organism was eradicated in 114/118. In addition, 34/67 sites were negative for *Prevotella intermedia* and 30/34 were negative for *Porphyromonas gingivalis*.

Amoxicillin/Clavulanate Potassium and Doxycycline. Matisko and Bissada (1993) studied 11 patients with recurrent/progressive periodontitis and demonstrating subgingival infection with *A. actinomycetemcomitans* and/or *P. gingivalis*. Patients receiving doxycycline, 200 mg the first day and 100 mg for 4 days thereafter, and then amoxicillin /clavulanate potassium, 500 mg 3 times daily for 5 days, produced significant improvement. A beneficial and positive effect was evidenced by the gain in probing attachment level and reduction in probing depth for up to 25 weeks.

Combination Therapy. Collins et al. (1993) described the clinical and microbiological features of 30 refractory patients and their response to a combined local and systemic therapy at 6 weeks and 3 years following treatment. The treatment consisted of a 2-week regimen of amoxicillin/clavulanate potassium in conjunction with professional, intramuscular delivery of povidine iodine, and chlorhexidine mouthwash rinse BID. A majority (87%) of the patients had favorable clinical responses to the treatment. It was effective at reducing probing depth, with a 56% decrease in the number of pockets greater than 6 mm at 6 weeks. This was accompanied by an overall gain of ≥ 1 mm of probable attachment gain in 45% of all sites. The clinical effects persisted at 34.4 months with an attachment gain of ≥ 1 mm in 41.2% of sites. These data suggest that this combination therapy is an acceptable, non-invasive alternative for the management of these patients.

By analyzing the results of subgingival microbial samples sent to a diagnostic microbiological laboratory, Listgarten et al. (1993) observed that a substantial number of microorganisms associated with refractory periodontitis are variably resistant to commonly-used antibiotics. Diagnostic microbiology was considered an essential adjunct to the therapist faced with periodontal lesions refractory to conventional treatment.

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Section 2. Early Onset Disease

Children are generally susceptible to the same periodontal maladies that affect adult segments of the population. However, certain disease entities warrant special attention due to their increased incidence and predilection in younger age groups. Knowledge and conceptual understanding of periodontal disease affecting children is ongoing and progressive. Advancing insights into the genesis and progression of these diseases mandate continual update and awareness by the periodontal therapist since this will assuredly impact on diagnosis as well as current and future approaches to management.

DEFINITIONS

Early Onset Periodontitis: Age of onset is usually prior to 35 years; rapid rate of progression of tissue destruction, manifestation of defects in host defense; and composition of the associated flora different from that of adult periodontitis.

According to Page et al. (1983A), prepubertal periodontitis is a genetic-based periodontitis of unknown prevalence

affecting a prepubertal populous during/after eruption of the primary dentition. Two forms have been described:

1. Generalized periodontitis (GPP): Acute fiery-red proliferative gingival inflammation (affecting marginal/attached gingiva) accompanied by rapid destruction of alveolar bone; affects all deciduous teeth and possibly the permanent dentition; peripheral white blood cell counts are markedly elevated with profound functional defects of PMNs and monocytes—no PMNs are found in the gingiva. Oral manifestations are frequently accompanied by otitis media, infections of the upper respiratory tract.

2. Localized periodontitis (LPP): Limited (undetermined) pattern of less rapid periodontal involvement with little/no gingival inflammation; PMNs/"or"/monocytes have functional defects. Recurrent otitis media and other infections are infrequent findings.

The term juvenile periodontitis (JP) was introduced by Butler (1969) and replaces the previously used term periodontosis.

JP, as defined by Baer (1971), "is a disease of the periodontium occurring in an otherwise healthy adolescent which is characterized by a rapid loss of alveolar bone about more than one tooth of the permanent dentition. There are two basic forms in which it occurs. In one form of the disease (localized), the only teeth affected are the first molars and incisors. In the other, more generalized form, it may affect most of the dentition. The amount of destruction is not commensurate with the amount of local irritants present."

Page et al. (1983A) define rapidly progressive periodontitis (RPP) as a distinctive form of periodontitis affecting young individuals ranging in age from puberty to 35 years; characterized by acute and quiescent phases and an inconsistently generalized pattern of distribution. The active phase is characterized by marginal proliferative gingivitis and severe rapid bone loss which may be followed by spontaneous cessation and resolution of gingival inflammation. Systemic findings (e.g., weight loss, mental depression, and malaise) may be present, and the authors have reported functional defects in the PMNs or monocytes in approximately 83% of affected individuals studied.

JUVENILE PERIODONTITIS

Clinical Features

Affected individuals are considered healthy adolescents despite reports of abnormal PMN and/or monocyte dysfunction. Localized JP (LJP) patients characteristically do not manifest a predisposition for disseminated infection. Prevalence ranges from 0% to 17% (average, 0.10% to .25%) with differences attributed to varying criteria, geographics, and a diverse data base. Studies suggest increased incidence in blacks when compared to Caucasian populations (Burmeister et al., 1984). Although a higher incidence of JP is reported in circumpubertal females, occurrence of disease among sexes becomes more equally distributed with increasing age, suggesting earlier occurrence among females (Hormand and Frandsen, 1979). Age criteria (12 to 20 years) for JP differ among investigators, but generally entail a circumpubertal onset with an upper age limit of 22 years.

Prevalence of JP has also been reported by Bial and Mellonig (1987), who radiographically evaluated 49,380 male naval recruits for evidence of JP. They reported a prevalence of 0.36% for this condition in their population. Melvin et al. (1991) screened 5,013 naval recruits for JP and reported a prevalence of 0.76% in their population. Prevalences of JP varied considerably between racial groups. Blacks had a much higher JP prevalence (2.1%) than Caucasians (0.09%), and black males had a higher prevalence (3.81%) than black females (1.99%). The overall male to female ratio of JP was found to be 1.1:1.0; however, when race was considered, the female to male ratio of black recruits versus Caucasian recruits was 0.52:1 and 4.3:1, respectively.

Löe and Brown (1991) reported and estimated prevalence of JP in U.S. school children age 14 to 17 years, using the National Institute of Dental Research national survey. Estimated prevalence of localized JP was 0.53% and generalized JP was 0.13%. In LJP, first molars were most commonly affected (40%), followed by second molars (21%) and incisors (10%). Attachment loss was widely distributed in GJP, with 37% of premolars and 33% of molars affected. The percent of adolescents with LJP increased until age 16, while the percent with GJP did not increase after age 15. Males were slightly more likely to have LJP and GJP, with blacks more likely than whites to have either disease. Black males were 2.9 times more likely to have LJP than black females, with the reverse true for whites. Hispanics were 2.4 times more likely to have LJP than non-Hispanics.

Cogen et al. (1992) retrospectively determined the prevalence of destructive periodontal disease in the deciduous dentition of children who later were diagnosed with LJP in their permanent dentition. Examination of the mixed dentition radiographs of 14 black children revealed that 12 had radiographic evidence of LJP in the mixed dentition. Seven of the 14 patients had radiographs of the deciduous dentition, of whom 5 patients had evidence supporting the diagnosis of LJP which involved the deciduous dentition. Sjodin et al. (1993) examined radiographs of the primary and permanent dentition of JP patients for evidence of primary dentition bone loss. Patients of the same age without radiographic evidence of bone loss were used as controls. Forty percent (40%) of all JP patients showed bone loss in the primary dentition as compared to 5% of the controls, and 31% of the JP patients had calculus in the primary dentition compared to 5% of the controls. First molars were most commonly affected followed by central incisors.

JP has historically been clinically associated with a paucity of bacterial plaque, so much so that some authors have stated that "local irritants must not be commensurate with

bone loss'' (Baer, 1971). Burmeister et al. (1984) consistently reported positive correlations between plaque presence and degree of destruction in JP. It must be emphasized that whether bacterial plaque is clinically visible or not, it is always associated with JP. In the majority of LJP patients, subgingival calculus is not present (Lindskog and Blomlof, 1983). LJP progresses 3 to 4 times the rate of adult periodontitis (Baer, 1971); a progression rate of 5 μm/day has been suggested on the basis of retrospective study (Waerhaug, 1977).

Etiology

Areas of etiologic commonality of JP can be identified in the periodontal literature. A full spectrum of correlative significance may be gleaned, but perspectives should be maintained. Factors include developmental (embryogenic) disorders, deciduous patterns of exfoliation, hyperocclusion, systemic abnormalities, qualitative defects of periodontal tissues, genetic predisposition, and microflora.

Developmental (Embryogenic) Disorders. Hiatt and Burrow (1965) proposed that neural crest alterations (virus induced?) could affect ectodermal/mesodermal stem derivatives and thus periodontium and epidermis. Their observations are more consistent with Papillon-Lefevre syndrome.

Deciduous Patterns of Exfoliation. McMillan (1976) suggested that distal root flare/resorption of the deciduous second molar may result in bone resorption at the mesial of the permanent first molars. Could all four first molars be affected simultaneously? What about incisors?

Hyperocclusion. The occlusal relationship in affected patients should be evaluated and adjusted as necessary (Ramfjord, 1952; Evian et al., 1982). In the genesis of JP, excessive occlusion may be a contributing factor as opposed to a primary cause; it should be adjusted if deemed excessive.

Systemic Factors. Factors associated with JP are numerous and range from chemical imbalance (Ross et al., 1958) to psychiatric factors (Gonzales, 1960). Suomalainen et al. (1991) investigated the origin of collagenase in the gingival crevicular fluid (GCF) of 6 JP and 6 adult periodontitis (AP) patients with no history of prior treatment. Results indicate that GCF from AP patients degraded type I and II collagen at equal rates and markedly faster than type III collagen, whereas GCF from JP patients preferentially degraded type I and III over type II collagen. Type III collagen was degraded 4 times faster by JP GCF than by GCF from adult periodontitis patients. The substrate specificity of JP GCF was indicative of collagenases produced by fibroblasts, epithelial cells, and macrophages. It was postulated that *Actinobacillus actinomycetemcomitans* may increase local collagenase production in JP patients by a direct effect on resilient fibroblasts or epithelial cells. It should be recognized that several systemic diseases have oral manifestations consistent with the JP disease pattern; however, these must be distinguished from JP forms described by Baer (1971) in which individuals do not manifest systemic disease. These diseases include Papillon-LeFèvre syndrome (Gorlin et al., 1964), Down's syndrome (Saxen et al., 1977), cyclic neutropenia (Cohen and Morris, 1961), agranulocytosis (Bauer, 1946), Chediak-Higashi disease (Tempel et al., 1972), hypophosphatasia, and diabetes mellitus (Becks, 1941). These entities should be considered in a comprehensive differential diagnosis of JP.

Qualitative Periodontal Defects. These have principally focused on cemental hypoplasia. Originally termed deep cementopathia by Gottlieb (1923, 1928), more recent microscopic observations by Ruben and Shapiro (1978) and Lindskog and Blomlof (1983) support the existence of true cemental hypoplasia in healthy and diseased root surfaces of a limited number of LJP patients.

The Role of Genetics. The genetic role in JP is suggested by reports of familial occurrence (Saxen, 1980A, 1980B). Nishimura et al. (1990) reported the results of a family study of a mother and daughter with increased susceptibility to early-onset periodontitis. They concluded, after assessing microbiological, immunological, host defense, and genetic parameters, that both subjects had an identical condition and that these patients may provide a unique model for improving our understanding of host factors involved in periodontal disease. Transmission of JP has been thought to be through an x-linked mode. Hart et al. (1992) suggested that transmission is via autosomal dominant transmission. The rationale for the previous x-linked mode of inheritance is that: 1) the selection of families for study introduces a bias for females in that females are more likely than males to seek dental care (female ascertainment bias); 2) mothers are more completely studied than fathers because they are more likely to bring children to appointments; and 3) studies that have looked only at affected siblings and have excluded the proband have found an equal ratio of affected females and males.

Evidence supporting autosomal dominant transmission include reports of nearly equal numbers of males and females affected by JP and demonstration of male-to-male transmission of JP (Löe and Brown, 1991).

Microflora. This factor warrants further discussion. Bacteria are always present and may be considered categorically composed of supragingival plaque, attached plaque, unattached or loosely adherent plaque, epithelium-associated bacteria, connective tissue-associated bacteria, and alveolar-associated bacteria. Newman et al. (1976) and Newman and Socransky's (1977) contributions to this area literally provided the impetus for further progressive study. These authors described 5 original groups of bacteria from JP lesions, providing a true conceptual shift from a traditional degenerative based etiology to one based on microflora. While subsequent investigations have identified JP-associated bacteria, two stand out: *Actinobacillus actinomycetemcomitans* (Y-4) and *Capnocytophaga* (3 species

= ochracea, sputigena, and gingivalis). These organisms will briefly be discussed.

Aa is a Gram-negative, non-motile capnophilic coccobacillus whose primary etiologic niche is the oral cavity; it is capable of seeding to distant sites, producing severe infection. Aa produces leukotoxin (PMN chemotaxis inhibiting factor); lymphocyte suppressing factor; lipopolysaccharide endotoxin; bone resorption-inducing toxin; acid/alkaline phosphatase; fibroblast inhibiting factor; collagenase; and epitheliotoxin (Zambon, 1985). This organism possesses tremendous destructive potential.

Aa has been observed in electron microscopic photomicrographs of gingival connective tissue of LJP lesions (Gillett and Johnson, 1982), suggesting tissue invasion. Mandell et al. (1987) identified Aa in all active sites and half of the inactive sites in 8 patients. All blood samples from each of these patients had elevated serum antibody to Aa.

Alaluusua et al. (1991) evaluated the possibility of intrafamilial transmission of Aa. Based on the prevalence and serotype distribution of Aa observed within the families studied, intrafamilial transmittance was suggested. However, since the data were cross-sectional, the direction of transmission could not be determined. The study also demonstrated that Aa does not colonize transiently, but shows persistent oral colonization.

The *Capnocytophaga* are Gram-negative, surface translocating (gliding motility) flexible rods that utilize a CO_2-based metabolism. Destructive potential is mediated by endotoxin, fibroblast proliferation-inhibiting factor and protease. *C. ochraceus* does not promote PMN chemotaxis and vascular exudation (Lindhe and Socransky, 1979), possibly accounting for the "facade" of clinical normalcy generally accompanying LJP. *C. sputigena* and *C. gingivalis* are extremely toxic organisms but thus far have not been specifically related to JP. Both produce a potent leukotoxin affecting PMN competence and have been shown to reduce PMN chemotaxis in vitro as a result of a post-receptor binding event (Shurin et al., 1979; Van Dyke et al., 1982A, 1982B). These, along with other organisms, such as *Eikenella corrodens* and black-pigmented *Bacteroides* species, may act synergistically with Aa to enhance tissue destruction in JP patients (Mandell et al., 1987; Kornman and Robertson, 1985).

Histopathogenesis

As recently as 1976, Waerhaug reported plaque-induced inflammation in LJP with plaque present within 0.2 to 1.1 mm of the epithelial cuff; a severe chronic inflammatory reaction and accompanying collagenolysis was noted in the adjacent connective tissue (CT). The presence of a broad zone of non-infiltrated CT between the inflammatory foci and the external tissue surface was deemed sufficient to mask visible signs of inflammation. No degenerative changes were observed. Numerous studies indicate that LJP lesions are predominantly characterized by a plasma cell inflammatory infiltrate (cells with disrupted plasmalemma) and marked collagen loss (Daly et al., 1980; Seymour and Greaves, 1980; Liljenberg and Lindhe, 1980; Waldrop et al., 1981). This scenario is consistent with an intensive humorally mediated immunologic response (predominantly IgG) and has been documented by immunofluorescence and peroxidase-antiperoxidase methods. Elevated serum antibody (AB) to specific pathogens (e.g., Aa, Capno, Ec, and leukotoxin) associated with LJP have been documented (Ranney et al., 1982; Mandell et al., 1987; Vincent et al., 1987).

Numerous studies suggest that altered PMN function in LJP patients may contribute to early onset and progression (local effect?) of disease. Chemotactic defects in peripheral PMNs have been observed in approximately 73% of JP patients (Cianciola et al., 1977; Clark et al., 1977; Genco and Cianciola, 1977; Van Dyke et al., 1980, 1982A, 1982C, 1982D; and Ranney et al., 1981). Since PMNs are principally defensive cells, impaired PMN function may be expected to favor increased incidence and severity of periodontal disease. If a PMN defect does exist, why are affected individuals otherwise healthy? Clark et al. (1977) suggest that since the defect is generally limited in scope, infections in other body sites can mount a large enough response to overshadow "minor deficiencies" while bacteria implicated in periodontal disease cannot. One must also consider the possibility of a locally induced PMN (bacterial leukotoxin) defect in the case of periodontal disease. Wilton et al. (1977) found that crevicular PMNs differ functionally from peripheral blood PMNs in vivo. Murray and Patters (1980) noted significantly less phagocytic activity in crevicular leukocytes from rapidly progressing disease sites when compared to nondiseased sites of the same patients or cells from chronic periodontitis lesions. This suggests that PMN function may be diminished locally as a result of factors produced by plaque microorganisms.

Treatment

Rationale. Given that bacterial plaque is the primary etiologic factor of JP, therapeutic efforts should be directed toward its control. Since genetic and/or local PMN/monocyte deficits exist, chemotherapy to assist host response appears appropriate.

Prognosis. A prognosis is affected by 1) extent and location of bone loss (furcal invasion decreases success); 2) morphology of bony defect; 3) crown/root ratio; 4) degree of mobility; 5) occlusal factors; 6) oral hygiene efforts; and 7) general health and attitude of the patient.

Modalities. In a review article, Krill and Fry (1987) discussed the various therapeutic options available for treating localized JP. Options discussed encompassed the full range of therapy and included summary comments on early treatment methods, effectiveness of antimicrobials, non-surgical and surgical therapy, tooth transplantation, orthodontics, and adjunctive endodontics.

Non-Surgical Treatment

Plaque Control/Scaling-Root Planing. The therapist must be aware of limited access in cases of increasing probing depths, especially furcated teeth.

Antibiotics. Tetracycline (TCN) is currently the drug of choice in treatment of LJP. Both Aa and *Capnocytophaga* are sensitive to TCN, which can achieve gingival crevicular fluid (GCF) at levels 2 to 10 times greater than serum levels (Walker et al., 1981; Gordon et al., 1981A, 1981B). Slots et al. (1980A and 1980B) showed that 59 strains of Aa were sensitive to TCN at a concentration of 1 mg/ml. The two most frequently used dose-time regimens of TCN as an LJP treatment adjunct are: 250 mg QID x 14 days (start 1 to 2 days pretreatment) Slots et al. (1983), Lindhe and Liljenberg (1984), and Kornman and Robertson (1985) all suggest an extended regimen of 28 days. Novak et al. (1988) suggest non-surgical therapy in early LJP with a 3 to 6 week regimen of TCN and supragingival prophylaxis every 2 weeks for 3 months after TCN therapy.

Studies directed toward local delivery of TCN have been posed (Goodson et al., 1979, 1983, 1985; Goodson, 1985) with hollow fibers giving way to TCN-impregnated ethylene vinyl acetate. This approach delivers high concentrations (80 to 160 X GCF and 322 X blood levels versus systemic levels with a 250 mg dose) without systemic toxicity. Bacteriocidal potential is proportional to increasing dosage.

In summary, the advantages of TCN in LJP are: 1) broad spectrum-bacteriostatic antibiotic; 2) specific for LJP-associated pathogens (Aa, *Capnocytophaga*); 3) concentrated in GCF; 4) affinity for saliva-coated enamel and inflammatory tissue; 5) may suppress bone and fibrinolytic activity; 6) may inhibit collagenase activity, reducing the rate of collagen breakdown (Golub et al., 1985); and 7) is relatively nontoxic to a healthy host.

Doxycycline, a semisynthetic TCN, has been successfully used in conjunction with surgery in the treatment of LJP (Mandell et al., 1986; Mandell and Socransky, 1988). Additional benefits of doxycycline include: 1) lower dosage, taken BID instead of QID; 2) not inhibited with calcium (dairy products); 3) does not induce photosensitivity; 4) decreased gastrointestinal side-effects; and 5) excreted via liver rather than kidney.

The use of metronidazole (MET) in suppressing Aa was compared to TCN in a study by Saxen and Asikainen (1993). All patients received oral hygiene instructions and scaling and root planing. Nine patients were given 200 mg of MET TID for 10 days; 9 patients were given 250 mg of TCN QID for 12 days; and 9 patients received no medication. Clinical parameters improved for all groups, with radiographic evidence of new bone formation.

Metronidazole was thus more effective in the eradication of Aa than TCN. Tetracycline, however, was used for a shorter duration than recommended.

Occlusal Therapy. When occlusion is determined to be a contributing factor in the presence of early onset disease, occlusal therapy should be initiated.

Orthodontic Treatment. Realignment and supereruption of affected teeth have been suggested by some authors (Everett and Baer, 1964; Goldstein and Fritz, 1976) to level or eliminate defects in JP patients.

Surgical Treatment

Surgical approaches to JP management include: 1) modified Widman flap/flap curettage; 2) resective techniques; 3) regenerative procedures; 4) root resection/extraction; and, 5) autotransplantation. No single surgical modality will be applicable to every case of JP; treatment approaches must be selected on the basis of independent case analysis and thus individual patient need.

Modified Widman Flap (MWR)-Flap Curettage (FC). Lindhe and Liljenberg (1984) used MWF with TCN for 14 days to treat 16 LJP patients. When compared with patients treated for adult periodontitis (AP), at 6 months, the gain of clinical attachment was more pronounced in the LJP group (2.1 to 5.1 mm, LJP versus 2.1 to 3.3 mm, AP). At 12 months post-treatment, 6 LJP sites had recurrent lesions (bleeding, probing depths 3 mm) and were retreated; subsequent bone fill and clinical attachment were oberved at 18 and 24 months. At the 5-year follow-up, the authors noted that clinical results remained unchanged from 6 months to 5 years with the exception of the 6 retreated sites. Barnett and Baker (1983) reported reduction of 5 to 8 mm pockets to 3 to 4 mm 1.5 months post-MWF (and TCN 1 gram/day for 14 days) surgery. Van Swol (1981) also reported rapid healing in LJP patients following MWF surgery.

Kornman and Robertson (1985) evaluated JP therapy clinically (bleeding/suppuration; probing depth; gingival retractability) and microbiologically (*Actinobacillus actinomycetemcomitans* (Aa); black pigmented *Bacteroides* [BPB]; surface translocating bacteria [STB]), comparing patient response to scaling and root planing (S/RP) alone, S/RP combined with 1 gram TCN/day for 28 days and MWF supplemented with 1 gram TCN/day for 28 days. Surgical therapy (MWF + TCN) resulted in pocket reduction, resolution of bleeding and suppuration, and dramatic reductions of BPB, Aa, and STB. These findings are consistent with those of Christersson et al. (1985).

Encouraging therapeutic results have been observed in JP lesions using MWF surgery supplemented with systemic TCN. Mandell et al. (1986) compared local TCN delivery via monolithic fibers, surgery (inverse bevel full thickness flaps without osseous recontouring) plus doxycycline (14 days) and doxycycline (14 days) without surgery in the treatment of LJP. Only the surgery-plus-doxycycline group was effective in suppressing or eliminating Aa.

Resective Techniques (Ostectomy-Osteoplasty). These are generally of limited value in management of JP lesions due to the severity of the disease. However, osseous resec-

tion may be useful as second stage procedures (post-grafting) in elimination of minor residual bony defects (Hoffman, 1983).

Regenerative Procedures. These are directed toward reconstitution of the periodontium in the face of severe disease. In theory, it would seem most applicable to JP, considering the exorbitant loss of attachment accompanying this disease. Regenerative response will depend on the magnitude and morphology of the osseous defects and the availability of graft materials, as well as their associated regenerative potential. Mattout and Roche (1984) used an iliac crest autograft in an 18-year-old JP patient, noting complete furcal fill and significant supracrestal repair. Yukna and Sepe (1982) and Mabry et al. (1985) used FDB allografts combined with TCN powder (4:1 ratio) supplemented with systemic TCN in management of LJP lesions. Mean fill of defects ranged from 61% to 80% using this therapeutic approach; "open-curettage" controls (Mabry et al., 1985) demonstrated 50% less defect fill.

Regenerative attempts offer therapeutic promise, but guidelines for treatment success must be realistic (50% bone fill; 75% bone fill). Procedures must be defined to the patient as to expectation and need for subsequent regenerative therapy or second stage surgical procedures.

Root Resection/Extraction. The therapist must weigh the impact of heroic therapy on adjacent teeth which may be potential abutments for a fixed partial denture. The objective is not carte blanc extraction, rather an attempt to provide esthetics, phonetics, and function commensurate with comprehensive dental health. It may be feasible to remove part of the diseased tooth (e.g., root resection, hemisection, etc.), bearing in mind that such therapy requires endodontic and often prosthodontic evaluation and support. Therapy should be kept in perspective during the course of comprehensive management of the JP patient.

Autotransplantation. This entails extraction of JP molars and replacement by autotransplanted third molars (Baer and Gamble, 1966; Borring-Moller and Frandsen, 1978). Donor: third molar, roots incompletely formed, < 90% developed; 2 to 3 mm apical to furca), atraumatically removed and placed in socket or stored in sterile saline. Recipient: mesial-distal width adjusted (polish; sodium fluoride treatment), granulation tissue left in site with alveolar alteration only as needed, intra-occlusion, nonrigid splinting and soft diet for 1 to 2 days. Root formation should continue with no root resorption, ankylosis, or pulpal necrosis.

Supportive Periodontal Treatment (SPT)

SPT, a *sine qua non* for successful periodontal therapy, is the dual responsibility of the therapist and patient. Following definitive treatment and post-treatment evaluation, the patient should be seen at 3-month intervals for SPT. The frequency of appointments may vary in accordance with the patient's needs. The SPT visit should include evaluation for signs of disease activity (bleeding and/or suppuration upon probing, increased probing depths, gingival atypia). Therapeutic attention should be directed to sites reflecting recurrence of disease or periodontal breakdown. Plaque control motivation should be assessed and monitored, with instructions tailored for individual patient needs.

PREPUBERTAL PERIODONTITIS

DEFINITION

Prepubertal Periodontitis: May be generalized or localized; onset between eruption of the primary dentition and puberty; may affect the primary and mixed dentition; characterized by severe gingival inflammation, rapid bone loss, tooth mobility, and tooth loss.

Features according to Page et al. (1983B):

1. Onset during or immediately after eruption of the primary teeth.
2. Prevalence unknown but probably rare.
3. Possibility of a genetic basis for some types.
4. Generalized form:
   - Extremely acute inflammation is present, with proliferation of gingiva;
   - There is very rapid destruction of the alveolar bone and gingiva;
   - Profound functional defects of peripheral blood neutrophils and monocytes are seen; neutrophils are absent from gingival tissue;
   - Peripheral blood white cell count is markedly elevated;
   - Otitis media and skin and upper respiratory infections are frequent findings;
   - Periodontitis may be refractory to antibiotic therapy; and
   - All primary teeth are affected; the permanent dentition may or may not be affected.
5. Localized form:
   - Only some teeth are affected; pattern of involvement not yet determined;
   - Gingival tissues may exhibit little or no inflammation;
   - Destruction is not as rapid as in the generalized form;
   - Functional defects are present in either neutrophils or monocytes, but not both;
   - Recurrent otitis media is not a frequent finding and usually there is no history of frequent infections; and
   - The disease is amenable to treatment by curettage and antibiotic therapy (Page et al., 1983A).

Watanabe (1990) reviewed diagnostic criteria, pathogenesis, and differential diagnosis of prepubertal periodontitis. Children with PP do not have neutropenia, agranulocytosis, aplastic anemia, or other traditional blood dyscrasias, and do not fit the diagnostic criteria for hypophosphatasia or Papillon-Lefèvre syndrome (PLS) (Page's definition did not exclude patients with these diseases). Es-

timates of prevalence range from 0.84% to 26.9%. Variance in reported prevalence may depend on genetic factors, methodological factors, and the selection of non-random sample populations. In otherwise healthy children, PP may be caused by periodontopathic bacteria (*Actinobacillus actinomycetemcomitans, Provotella intermedia, Prophyromonas gingivalis, Capnocytophaga* species, and *Eikenella corrodens* have been proposed). Susceptibility may be related to cementum defects, leukocyte chemotaxis dysfunction, and/or presence of bacteriophage.

Prichard et al. (1984) reported a case of prepubertal periodontitis affecting the deciduous and permanent dentition in a patient with cyclic neutropenia. This report illustrated the importance of obtaining a differential white blood cell count when diagnosing severe oral pathoses of obscure origin.

Several conditions exist that may lead to advanced periodontal deterioration in children. A differential diagnosis would include: hypophosphatasia, Papillon-LeFèvre syndrome, histiocytosis X, neutropenia, cyclic neutropenia, leukemia, diabetes mellitus, scleroderma, fibrous dysplasia, and acrodynia (Goepford, 1981). Page et al. (1983B) listed profound functional defects of peripheral blood neutrophils and monocytes in the features of prepubertal periodontitis. A partial list of neutrophil abnormalities that exhibit periodontal destruction includes: cyclic neutropenia, chronic familial neutropenia, hereditary neutropenia, agranulocytosis, chronic benign neutropenia, chronic idiopathic neutropenia, congenital agranulocytosis, chronic benign granulocytopenia, congenital neutropenia, atypical hereditary neutropenia, familial benign chronic neutropenia, and chronic neutropenia. Range of severity of neutropenia is categorized as mild = 1,000 to 2,000 cells/mm^3; moderate = 500 to 1,000 cells/mm^3; severe = less than 500 cells/mm^3 (Kalkwarf and McLey, 1984).

Diseases Associated with Periodontitis in Prepubescent Children

Hypophosphatasia. An autosomal recessive or dominant disorder caused by low levels of alkaline phosphatase (3 types: infantile, childhood, and adult); mild forms may have no other clinical signs other than early exfoliation of deciduous teeth with minimal signs of inflammation. PMN defect reports are variable.

Papillon-Lefèvre Syndrome. An autosomal recessive trait that may be related to a generalized epithelial dysplasia; clinical features include palmar and plantar hyperkeratosis and periodontal destruction. PMN defects are controversial.

Neutropenia. A number of neutropenias exist; several manifest severe gingivitis with ulcerations, and a history of recurrent infections.

Leukocyte Adhesion Deficiency. An autosomal recessive condition in which the glycoprotein adhesion molecules on leukocytes are reduced. Oral features include severe gingival inflammation and severe alveolar bone loss, which leads to early exfoliation of teeth.

Histiocytosis X. May be the result of the proliferation and dissemination of pathologic Langerhans cells.

Chediak-Higashi Syndrome. A rare autosomal recessive disease in which leukocyte defects are associated with impaired function of cytoplasmic microtubules or microtubule assembly in PMNs.

Leukemias. A group of conditions characterized by progressive uncontrolled proliferation of white blood cells.

Acrodynia. A rare disease thought to be caused by an unusual sensitivity or idiosyncrasy to mercury.

Juvenile Diabetes. A relative or absolute decrease in insulin secretion, availability, or responsiveness, possibly caused by a genetic defect, autoimmunity, or viral infections.

AIDS. An unusual gingivitis resembling an atypical form of acute necrotizing ulcerative gingivitis has been reported, although no reports exist of prepubertal AIDS patients with alveolar bone loss. Any of these diseases may manifest as periodontitis, thus patient evaluation to obtain a differential diagnosis may include complete blood cell count, leukocyte differential and cell morphology, serum alkaline phosphatase, examination for palmar and plantar hyperkeratosis, biopsy, and fasting blood glucose levels (Watanabe, 1990). The diagnosis of PP is made following confirmation that patients are not afflicted with one of the aforementioned systemic diseases.

Watanabe et al. (1991) also analyzed neutrophil chemotaxis and surface CD11b expression by neutrophils (PMNs) in localized prepubertal periodontitis patients and healthy controls. Results of this investigation indicated that there was no statistically significant correlation between neutrophil chemotaxis and surface CD11b expression between localized prepubertal periodontitis and pediatric control subjects. Data from this study, however, indicated that neutrophil chemotaxis was significantly depressed in children relative to that of the healthy adult control group. It is possible that decreased neutrophil chemotaxis in children, in combination with the presence of suspected pathogens, may precipitate periodontitis. The authors suggest that the diagnosis of localized PP cannot be made by clinical appearance and/or the patients' dental history of premature exfoliation of the primary teeth.

A genetic basis for some types of disorders may exist. Waldrop et al. (1987) described a family with deficiencies of MAC-1 (the iC3b receptor of human myeloid cells), lymphocyte function antigen-1 (LFA-1) and p 150, 95. These glycoproteins are normally found on granulocyte, monocyte, and/or lymphocyte surfaces and function in adhesion-dependent immune cell function that contribute to endothelial migration and subsequent infiltration of PMNs and monocytes into extravascular inflammatory sites. Individuals deficient in these glycoproteins manifest necrotic, nonpurulent infections, and delayed wound healing. Perio-

dontal findings include rapid bone loss, fiery-red inflammation, recession, bleeding, and other signs of generalized destruction. This emphasizes the importance of normal leukocyte function in maintaining the periodontium against pathogenic microorganisms. In affected individuals, the degree of host immunocompromise reflects the extent of the so-called "glycoprotein-based" leukocyte adhesion defect (LAD).

Treatment

Treatment for prepubertal periodontitis has ranged from local treatment (curettage) and/or systemic antibiotics to the extraction of involved teeth (Watanabe, 1990). Localized PP may be responsive to treatment by curettage and antibiotics, whereas generalized PP may be refractory to antibiotic therapy. Granulocyte infusion may improve the periodontal condition temporarily (Page et al., 1983A). To date, however, treatment of patients with PP has generally been unsuccessful. Generalized PP patients show early loss of primary and adult dentition (Waldrop et al., 1987).

RAPIDLY PROGRESSIVE PERIODONTITIS

DEFINITION

Rapidly Progressive Periodontitis (RPP): Most of the teeth are affected; the extent of clinical signs of inflammation may be less than expected; the age of onset is usually in the early 20s through the mid 30s.

Features according to Page et al. (1983A) are:

1. Age of onset is between puberty and about age 35.
2. Lesions are generalized, affecting most of the teeth, without any consistent pattern of distribution.
3. Some, but not all, patients may have had juvenile periodontitis previously.
4. There is evidence of severe and rapid bone destruction, after which the destructive process may cease spontaneously or greatly slow down.
5. During the active phase, the gingival tissue is acutely inflamed with marginal proliferation; during the arrested phase, the tissues may appear free of inflammation.
6. The amounts of microbial deposits are highly variable.
7. Approximately 83% of the patients have functional defects in neutrophils or monocytes.
8. The disease sometimes, but not always, has systemic manifestations including weight loss, mental depression, and general malaise.
9. Some individuals are remarkably responsive to treatment by S/RP or open/closed curettage with adjunctive antibiotic therapy.

Chen et al. (1991) investigated immune responses of 36 young adults with rapidly progressive periodontitis to *Porphyromonas gingivalis* (Pg). This study demonstrated that one-third of the RPP patients did produce IgG reactive to Pg but with low avidity (strength of binding). The remaining two-thirds had lower IgG and avidity than control subjects. The change in titer and avidity occurring after treatment may have been due to a decreased bacterial load which has been shown to result in the selection of clones of B lymphocytes that produce antibodies of higher avidity, or the reduced load may allow maturation of the immune system. Results showing that the sero-positive group had more bone loss may be the result of longer presence of the disease, or a faster rate of disease progression which allowed production of higher titers of low avidity antibody.

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Section 3. Acute Periodontal Conditions: Periodontal Abscess

DEFINITION

Periodontal Abscess: Localized purulent inflammation in the periodontal tissues; also called lateral abscess.

CLINICAL FEATURES

The acute periodontal abscess may have symptoms such as throbbing radiating pain, exquisite tenderness of the gingiva to palpation, sensitivity of the tooth to percussion, tooth mobility, and lymphadenitis. It appears as an ovoid elevation of the gingiva. The gingiva is generally edematous and red, with a smooth shiny surface. The shape and consistency of the elevated area may vary from dome-like and relatively firm to pointed and soft. Purulent exudate can usually be expressed from the gingival margin by gentle digital pressure (Glickman, 1979).

RADIOGRAPHIC APPEARANCE

The typical radiographic appearance of the periodontal abscess is that of a discrete radiolucency along the lateral aspect of the root. Many variables may alter this such as: 1) stage of the lesion; 2) extent of bone destruction and morphology of the bone; and 3) the location of the abscess (i.e., abscesses on the facial or lingual surface are obscured by radiopacity of the root). The radiograph alone is not sufficent for the diagnosis (Glickman, 1979).

DIAGNOSIS

Continuity of the lesion with the gingival margin is clinical evidence of the presence of a periodontal abscess. The abscess is not necessarily located on the same surface of the root as the pocket but may follow a tortuous course from the depth of the pocket (Glickman, 1979).

Differential Diagnosis. Table 1 shows the usual characteristics of periapical and periodontal abscesses.

TABLE 1. PERIODONTAL VERSUS PERIAPICAL ABSCESS*

Periapical	Periodontal
non-vital tooth	vital tooth
caries	no caries
no pocket	pocket
apical radiolucency	lateral radiolucency
none or minimal mobility	mobility
sensitive to percussion	*sensitive to percussion* (acute)
draining sinus usually located in apical area	draining sinus usually located at lateral aspect of root

*Adapted from Glickman, 1979.

ETIOLOGY

Newman and Sims (1979) performed microbiological culturing of periodontal abscesses in 9 patients. An exudate sample was taken at the gingival margin after light digital pressure to the external abscess wall. An apical sample was taken from the apical portion of the periodontal pocket. All samples were taken using a barbed broach under flow of anaerobic gas within a cannula. Microbiota recovered from the respective abscess sites were predominantly Gram-negative (66.2%) and anaerobic (65.6%). In the exudate, 49.5% of the predominant cultivable microbiota were Gram-negative anaerobic rods including *Bacteroides melaninogenicus* subspecies, *Fusobacterium* species, and vibrio-corroders. The apical sites contained more Gram-positive organisms (40% versus 27.6%); however, the apical sites were still predominated by Gram-negative (60%) and anaerobic (64.3%) bacteria. *Capnocytophaga* species were isolated in all exudate samples.

Glickman (1979) gives 5 scenarios for abscess formation: 1) extension of the pocket into supporting periodontal tissues along lateral aspect of the root; 2) lateral extension from the inner surface of the pocket into connective tissue of the pocket wall; 3) in the tortuous (complex) pocket, an abscess may form in the deep end (cul-de-sac); 4) incomplete removal of calculus results in shrinkage of the gingival wall and occlusion of the pocket orifice; and, 5) an abscess may form in the absence of periodontitis following trauma to tooth (fractured root) or perforation of the lateral wall of the root during endodontic therapy.

TREATMENT

Grant et al. (1988) describe 3 methods for treating periodontal abscesses: 1) curettage via the sulcus; 2) flap surgery; and, 3) incision and drainage. Treatment may be dictated by availability of time at emergent presentation. Management under these conditions should be reevaluated post-treatment for subsequent therapeutic needs. Antibiotics may be indicated pending patient manifestations and should be supported by culture and sensitivity where practical and indicated.

PROGNOSIS

Nabers et al. (1964) suggest that the acute inflammation present in a periodontal abscess provides excellent potential for regeneration of bone, periodontal ligament, and cementum. They conclude that the prognosis of teeth with extensive osseous pocketing depends not only on the morphology of the lesion but on chronology as well.

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Section 4. Necrotizing Ulcerative Periodontitis

DEFINITIONS

Necrotizing Ulcerative Periodontitis (NUP): Severe and rapidly progressive disease that has a distinctive erythema of the free gingiva, attached gingiva, and alveolar mucosa; extensive soft tissue necrosis; severe loss of periodontal attachment; deep pocket formation is not evident. Previously termed necrotizing ulcerative gingivitis (ANUG).

Acute: 1) Sharp, severe. 2) Denoting the swift onset and course of a disease.

CLINICAL FEATURES

Barnes et al. (1973) state that the diagnosis can be based on clinical findings alone. The authors examined 218 patients and found pain to be the most consistent symptom while bleeding and interdental cratering were the most consistent objective signs. Pseudo-membrane formation and fetor oris were also common. Fever, lymphadenopathy, and malaise are rare and are considered a secondary finding associated with dehydration.

In an army population (active duty and dependents), Barnes et al. (1973) reported an incidence of less than 1% while Pindborg (1951) found an incidence of 6.9%.

Kristoffersen and Lie (1983) suggested a condition which they termed chronic necrotizing gingivitis. However, the concept of "chronic ANUG" is contradictory and recurrent ANUG was considered more appropriate (Johnson and Engel, 1986).

ETIOLOGY

In a classic description of the ANUG lesion, Listgarten (1965) described four distinct zones. The surface bacterial zone consisted of a wide variety of microorganisms including spirochetes and fusiforms. The neutrophil-rich zone was comprised of leukocytes (mainly PMNs) migrating from the underlying connective tissue and adjacent epithelium. Spirochetes were also observed in this layer and were occasionally located within mononuclear leukocytes, suggesting phagocytosis. The necrotic zone was characterized by cellular and connective tissue debris, with the predominant morphotype being spirochetes of varying sizes. The zone of spirochetal infiltration exhibited spirochetes within vital connective tissue, infiltrating to depths of 250 μm beneath the surface of the lesion. The invading spirochetes were predominantly of the medium and large varieties and were

also observed within the intercellular spaces of the adjacent epithelium.

In a more recent light and transmission electron microscopic (TEM) study, Courtois et al. (1983) examined papillae from 8 ANUG patients. They also observed 4 layers as previously reported, but noted blending of the neutrophil-rich and necrotic zones. Bacterial invasion (primarily spirochetes) was present to depths of 155 to 400 μm within the connective tissue. Unlike Listgarten, they noted that plasma cells and lymphocytes were the predominant inflammatory infiltrate.

Loesche et al. (1982) studied 8 ANUG patients using darkfield microscopy and anaerobic culturing. They reported *P. intermedia, Fusobacterium*, and *Treponema* species in each of the plaque samples examined. Of the total cultivable flora, *P. intermedia* represented 24.0%, and *Treponema* species and large spirochetes comprised 30% and 10%, respectively. These results were interpreted to indicate a pathogenic role for *P. intermedia.* Chung et al. (1983) also implicated *P. intermedia* and spirochetes in the etiology of ANUG by reporting high antibody titers to intermediate-sized spirochetes in these patients.

Sabiston (1986) suggests that the correlation between features of ANUG and virus infections (notably cytomegalovirus) point to a viral etiology for ANUG.

Cogen et al. (1983) reported that leukocyte function in ANUG patients showed depressed chemotaxis and phagocytosis. The authors also observed that peripheral blood lymphocytes from ANUG patients exhibited reduced proliferation when stimulated with concanavalin-A mitogen.

MacCarthy and Claffey (1991) evaluated 13 patients (18 to 27 years of age) with a history of ANUG and non-surgical treatment. All 13 patients smoked and 6 had recurring episodes of ANUG. The sites affected by ANUG appeared to have a greater loss of attachment than non-ANUG control sites (2.2 mm and 0.8 mm, respectively). This study associates ANUG with attachment loss, but does not account for the severity of the lesions or the potential for attachment loss as a result of treatment.

PREDISPOSING FACTORS

Stress. This is probably the main factor contributing to the development of ANUG. Pindborg (1951) indicated that psychological stress accompanying transfers of enlisted men in military service may cause a sudden increase in the incidence of ANUG. In a subsequent military study, Goldhaber and Giddon (1964) reported that there was a higher incidence of ANUG in personnel just entering the army or going on leave (i.e., occasions they associated with higher levels of psychological stress). Shannon et al. (1969) observed that patients with ANUG had a higher level of 17 hydroxycorticosteroids (17-OHS) than controls. Maupin and Bell (1975) reported that ANUG patients had significantly higher levels of 17-OHS during the course of the disease than at post-disease evaluations.

Smoking. Pindborg (1951) noted that 98% of the ANUG patients studied were smokers. The effect of smoking appears to be more complex than a mere reflection of patient stress. Clarke et al. (1981) have demonstrated that intra-arterial infusion of epinephrine and nicotine in rabbits resulted in reduced gingival blood flow rates in spite of increased systemic perfusion pressure.

Pre-Existing Inflammation. Schluger (1943) claimed that a low standard of oral hygiene is the most important factor contributing to ANUG, considering it a disease of filth. Pindborg (1951) noted that 90% of ANUG cases begin as simple marginal gingivitis.

HIV-Positive Patients. Smith et al. (1987) reported a high incidence of ANUG in patients at risk of contracting acquired immune deficiency syndrome (AIDS). They also related that these male homosexuals had reduced numbers of the helper/inducer T cell subset (T4+) and an abnormal ratio of helper/inducer T cells to suppressor/cytotoxic T cells (T4:T8) ratio. This association between AIDS and ANUG could be of clinical and scientific interest to the dentist when treating ANUG patients.

TREATMENT

Schluger (1949) describes a simplified treatment of thorough, deep curettage followed by frequent rinses with diluted hydrogen peroxide, primarily as a lavage. Fitch et al. (1963) reported that immediate ultrasonic debridement proved to be highly effective in treating ANUG with rapid relief of symptoms. Goldhaber and Giddon (1964) augmented these local measures with antibiotics (principally penicillin) when treating advanced cases of the disease. Loesche et al. (1982) reported prompt resolution of clinical symptoms following metronidazole treatment. Elimination of the residual soft tissue craters via gingivoplasty is believed to be important to minimize recurrence of the disease (Schluger, 1949; Goldhaber and Giddon, 1964).

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Section 5. Periodontal Cysts

DEFINITIONS

Periodontal Cyst: A small cyst of the periodontal ligament found most often in the mandibular canine and premolar areas; associated with a vital tooth and postulated to originate from the rests of Malassez, the rests of the dental lamina, or a supernumerary tooth bud.

Gingival Cyst: Found within the gingiva, most commonly in the mandibular canine-premolar region. Believed to be derived from epithelial rests of the dental lamina.

HISTOLOGIC AND CLINICAL FEATURES OF THE GINGIVAL CYST

Moskow and Bloom (1983) investigated the embryogenesis of the gingival cyst (GC) in humans. They reported cystic degeneration of the dental lamina as early as 10 weeks in utero and rapid cystic proliferation and growth in 15 to 20 week embryos during the bell stage of tooth development. Distinct GCs were lined with a thin squamous epithelium and filled with keratin. Nxumalo and Shear (1992) described GCs of adults as having a cystic lining consisting of 1 to 3 epithelial cell layers resembling reduced enamel epithelium or a thicker nonkeratinized stratified squamous epithelium with pyknotic nuclei. Clinically, the GC of adults is a painless, well-circumscribed, fluctuant soft tissue swelling < 1 cm in diameter. It commonly occurs between the fifth and sixth decades in the mandibular (73%) canine-premolar area. Superficial bone resorption can occur (Shear, 1983). Nxumalo and Shear (1992) support the junctional epithelium as the tissue of origin because of the frequent occurrence of lining epithelium resembling reduced enamel epithelium and the continuity of the cyst with the junctional epithelium. Similarities between the GC and the lateral periodontal cyst (LPC) suggest that they are both of developmental origin and both are derived from the reduced enamel epithelium.

HISTOLOGIC AND CLINICAL FEATURES OF THE LATERAL PERIODONTAL CYST

Cohen et al. (1984) reported light microscopic observations on 37 lateral periodontal cysts (LPCs). LPCs were more prevalent in the fifth to seventh decade with no sex or race predilection. They are generally asymptomatic, although there is some degree of buccal or lingual plate expansion. The majority occur in the mandibular canine-premolar region and are associated with vital teeth. Detection is by routine radiographic examination. Radiographically, they present as a well-defined, solitary radiolucency associated with the lateral aspect of a tooth root and are round or tear drop in shape. Treatment is by enucleation with no anticipated recurrence. Histologically, the LPC is a cavity supported by fibrous connective tissue with little surrounding inflammation. The epithelium often has areas of focal thickening (plaques) which may be interspersed with glycogen-rich clear-cells.

The LPC has a similar appearance to the GC of the adult and it is believed that the LPC and the GC of the adult develop from the dental lamina (Wysocki et al., 1980). The only difference between the lesions is that the LPC occurs within bone while the GC of the adult occurs within the gingiva. The term LPC should be reserved for those intraosseous cysts with this histologic description and not for all cysts located lateral to the root. Wysocki et al. (1980) reported on 39 examples of the LPC and 10 cases of the GC of the adult and similarly concluded that the dental lamina was the tissue of origin because of the association of both these cysts with the glycogen-rich clear-cell rests of the dental lamina.

LATERAL PERIODONTAL CYST AND PERIODONTAL DEFECTS

Filipowicz and Page (1982) suggested the possibility that proliferation of epithelial elements other than crevicular epithelium could be implicated in isolated periodontal defects. They outlined 4 possible explanations of isolated periodontal defects: 1) an LPC in the marginal periodontium could enlarge until it communicates with the crevicular epithelium; 2) crevicular epithelium could migrate apically until it communicates with an existing LPC; 3) apical migration of the crevicular epithelium could communicate with remnants of Hertwig's root sheath epithelium and initiate proliferation; or 4) pulpal injury causing tissue damage in accessory pulpal canals in the coronal portion of the root could communicate with the periodontium.

Spouge et al. (1986) examined the association between

the reduced enamel epithelium (REE) and the junctional epithelium (JE) and the rests of Malassez (ROM) in the pig. The ROM were present in the deeper portions of the periodontal ligament below the alveolar bony crest. The ROM formed spiral strands of epithelial cells running around the long axis of the tooth. Near the CEJ, the loops diverged away from the tooth surface and the strands became thinner and more scarce. However, the strands remained continuous and eventually joined with either the JE or the REE. The ROM have a proliferative potential and could form an area of entry for apical JE migration through the connective tissue.

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CHAPTER 3. EXAMINATION AND DIAGNOSIS

Section 1. Periodontal Probing

The existence of a pocket implies a history of periodontal disease. While some visual signs of inflammation, such as redness and swelling, are helpful in detecting disease, they are not always present in conjunction with inflammation at the base of the pocket. Van der Velden (1980) noted that 4 to 5 weeks after scaling and root planing, most redness and swelling disappeared, although sites still bled when evaluated by the periodontal pocket bleeding index. Therefore, it is important to evaluate the bottom of the pocket as well as the visual signs of inflammation.

Listgarten (1993) emphasized correct terminology when describing periodontal probing in the literature and encouraged use of the terms proposed in the 1989 *Proceedings of the World Workshop in Clinical Periodontology* (American Academy of Periodontology, 1989). He discounted the term "probing pocket depth," indicating that the true anatomic measurement of the pocket can only be accomplished histologically. "Probing depth" was suggested as a more correct term since it measures the depth of the pocket plus the inconsistent amount of connective tissue penetration. The term "probing attachment level" should be replaced by the more descriptive terms "clinical attachment level" when the measurement is made from a fixed reference on the tooth (CEJ or restoration). "Relative attachment level" applies when the measurement is made from some other reference point (e.g., stent margin or occlusal tooth surface).

A number of variables exist that will affect probing depth measurements, and each must be considered when evaluating results. These variables include: probing reproducibility, probing force, probe angulation, status of gingival health, site location, local anatomy, and type of probe used.

PROBING REPRODUCIBILITY

Isidor et al. (1984) studied the reproducibility of measurements using a flexible splint, gentle pressure, and one examiner. Sixty percent (60%) of the measurements were in complete agreement between the first and second measurements before and after surgery. Ninety-five (95) of the surfaces differed by $\leq$ 1 mm or less and no measurement differed by $\geq$ 3 mm. Depth of the pocket and location of the probing site did not influence the measurements. Van der Velden and de Vries (1980), in a study involving 102 interproximal pockets in 7 periodontal patients, found no differences in reproducibility of measurements between a pressure-sensitive probe set at 0.75N and a Merritt B probe. Aeppli et al. (1985) examined inter- and intraexaminer reproducibility in terms of defining the effects of reproducibility on the sensitivity and specificity of different diagnostic thresholds for true change in probing depth. The mesio-facial surfaces of the Ramfjord teeth were measured in 15 patients by 3 examiners. With a threshold of probing depth increase $>$ 1 mm, it was determined that an increase could be correctly diagnosed 91.3% of the time with a false-positive rate of 0.5%. A threshold of $>$ 2 mm led to higher specificity, but with a great reduction in sensitivity.

Reproducibility and concomitant examiner error has been reduced with the advent of controlled force probes with computer interfaces such as the Florida probe (Magnusson et al., 1988) and Foster-Miller (Alabama) probe (Jeffcoat et al., 1986).

PROBING FORCE

Van der Velden and de Vries (1978) evaluated the effects of probing force on periodontal patients utilizing a pressure-sensitive probe with a tip diameter of 0.63 mm. The patients were free of clinical inflammation at the time of the measurements. Sites were probed at forces of 0.15N, 0.25N, 0.50N, and 0.75N. A positive relation between probing force and probing depth was observed. In a study using condemned teeth, Van der Velden (1979) evaluated the location of the tip of the probe with different probing forces. He reported that a force of 0.75N was in closest agreement with attachment level measurements. Chamberlain et al. (1985) also showed that forces of 0.75N were more reproducible, with the probe extending to the most coronal connective tissue (CT) attachment in health and disease. The authors suggested that one of the difficulties with understanding discrepancies with probing attachment levels in earlier studies may have been due to non-standardized probing forces. Mombelli et al. (1992) used a probe with a steel spring and strain gauges to provide a constant increase in probing force that could be diagrammed. The study indicated that higher probing forces will lead to more reproducible readings, but suggest that lighter forces could detect more subtle changes in attachment levels. Freed et al. (1983) studied different probing forces used by dental care providers based on levels of expertise (periodontists, general dentists, hygienists, and students). Probing forces ranged from 5 to 135 grams with no significant differences between any of the examiner groups, although periodontists and hygienists tended to probe with less force than general dentists and students. Probing force was found to be significantly greater in posterior segments than in anterior segments. In

addition to large interexaminer differences, intraexaminer variability was also large, with a mean range of 44 grams.

PROBE ANGULATION

Persson (1991) compared line-angle measurements to midproximal measurements in untreated sites and found that the mean probing measurement was 1 mm greater with midproximal measurements than with line-angle measurements. This implies that clinical and epidemiological studies using line-angle measurements may underestimate pocket depth and the true level of disease.

The gingival sulcus is histologically or anatomically defined as the distance from the gingival margin to the coronal end of the junctional epithelium to the coronal end of the junctional epithelium (JE) (Listgarten, 1972). However, the ability of the periodontal probe to accurately measure this distance has been questioned by several studies in which the position of the probe tip was evaluated in healthy and diseased tissues. In a study of beagle dogs, Armitage et al. (1977) found that the probe failed to reach the apical termination of the JE in healthy specimens, but extended beyond the most apical cells of the junctional epithelium in periodontal specimens. Human studies such as that by Sivertson and Burgett (1976) indicated that the periodontal probe routinely penetrated to the coronal level of the connective tissue attachment of untreated periodontal pockets. Listgarten et al. (1976) observed that the most common position of the probe tip during routine measurements of periodontal pocket depth was at the coronal portion of the JE. Saglie et al. (1975) noted that probing depths measured in the laboratory were always less deep than those recorded clinically. The authors attributed this to the presence of a zone of completely and partially destructed periodontal fibers which allowed the probe to extend apically to the coronal level of connective tissue attachment. These studies have shown that periodontal probes do not precisely measure and often overestimate the true histologic sulcus, and that inflammation has a significant influence on the degree of probe penetration.

STATUS OF GINGIVAL HEALTH

Glavind and Löe (1967) observed that non-standardized forces in healthy tissue resulted in variations in probing depths of 1 mm compared to variations of over 2 mm in inflamed tissue. Robinson and Vitek (1979) showed a straight line correlation between GI scores and tissue penetration by the probe. Spray et al. (1978) suggested that the state of health of the underlying CT fibers influenced probing measurements, with the healthy fibers acting as a barrier and preventing apical movement of the instrument (the "hammock" effect). Caton et al. (1981) reported that inflamed CT offered less resistance to penetration and that with reduction of inflammation following initial therapy, a more accurate estimate of the sulcus depth resulted. Fowler et al. (1982) showed through histologic examination that in untreated teeth, the probe tip penetrated beyond the apical termination of the JE and into subjacent connective tissue by a mean of 0.45 mm, whereas in the treated specimens the probe tip stopped coronally to this landmark by a mean of 0.74 mm. These findings are in approximate agreement with earlier work (Magnusson and Listgarten, 1980) which reported 1.4 mm in probing attachment gain in treated sites.

Anderson et al. (1991) correlated the degree of clinical and histologic inflammation to probe tip penetration in dogs. A significant correlation was noted between probe tip penetration and amount of tissue inflammation adjacent to the probe. Correlations between gingival index and histologic inflammation and gingival index and, probe penetration were not significant. This suggests that probe penetration is more highly influenced by inflammation at the base of the pocket rather than marginal inflammation.

LOCAL ANATOMY

Crown contours, interproximal versus facial or lingual sites, narrow pockets, tipped or rotated teeth, heavy osseous ledges, and defective restorations and margins can affect probing accuracy. Moriarty et al. (1989) studied the vertical histologic probe position in untreated facial molar furcation sites. When vertical probing was carried out at the midfacial area of Class II and III furcations, the probe tip penetrated into interradicular connective tissue. The probe tip did not approximate tissue at the base of the pocket, but penetrated at various levels along the pocket wall. The authors suggest probing the root surface anterior to and posterior to the furcation entrance to more accurately reflect the true pocket depths at furcations.

TYPE OF PROBE USED

There are numerous types of probes with varying diameters. Some examples are: Michigan, Williams, Marquis (round probes); and Goldman-Fox, Dellich, and Nabers (flat probes). Errors in manufacturing of the probes can significantly affect measurements in clinical research settings. Van der Zee et al. (1991) evaluated the accuracy of probe markings in a variety of probes, noting that few probes coincided with the manufacturer's designated calibration. The tip diameters ranged from 0.28 mm for the Michigan "O" probe to 0.7 mm for Williams' probes. The widths of probe markings were important in that painted bands differed by as much as 0.7 mm. Etched bands had the most accurate width markings while etched grooves were nearly twice as inaccurate. Atassi et al. (1992) compared a parallel-sided probe to a tapered probe. The parallel-sided probe tended to yield deeper probing depths in deeper pockets suggesting that the tapered probe may tend to bind more within the pocket. However, the repeatability was similar for the tapered (81%) and parallel-sided (86%) probes. In addition, when the probe measurements were compared, 89% showed no difference in probing measurement.

STRENGTH OF THE EPITHELIAL ATTACHMENT

It is accepted that the probe cannot penetrate to the CT without damaging the epithelial attachment. However, the strength of the epithelial attachment and its ability to resist the probe is not known (De Waal et al., 1986).

RELATIONSHIP BETWEEN PROBING AND BONE LEVEL MEASUREMENTS

Isidor et al. (1984) evaluated transgingival probing measurements taken just prior to surgery and compared them to measurements taken immediately after flap reflection, utilizing a flexible stent to control probe angulation. Transgingival probing was identical to surgical measurements 60% of the time and within 1 mm of surgical measurements 90% of the time. Disagreement was never greater than 3 mm. Ursell (1989) studied the accuracy of probing with 30g or 60g force and vertical transgingival probing (> 100g force) as an estimate of open bone level measurements. Higher correlations were found with transgingival probing ($r = 0.98$) compared to measurements made at 30g ($r = 0.87$) and 60g ($r = 0.90$). A mean difference between measurements of 0.12 mm was found between transgingival probing and surgical measurements. Agreement between transgingival probing and surgical measurements was unaffected by tooth type, tooth surface, inflammation, or magnitude of bone loss. Correlations were higher for 30g and 60g measurements when inflammation was present. When sites with intrabony defects were considered, the correlation between transgingival probing and surgical measurements was $r = 0.79$ and the mean difference between measurements was 0.92 mm. Akesson et al. (1992) compared estimation of bone levels with bone sounding and periapical, bite-wing, and panoramic radiographs to open measurements. Bone sounding provided the best estimate of open bone level measurements. The percent underestimation was 5% for bone sounding, 13% (maxillary) to 14% (mandibular) for periapicals, 17% (maxillary) to 23% (mandibular) for bite-wings and 18% (maxillary) to 24% (mandibular) for panoramic radiographs. Percent image magnification was generally greater in the maxilla and was 8% for periapical films, 9% for bite-wings, and 25% for panoramic radiographs.

CRITICAL PROBING DEPTH

Lindhe et al. (1982) described the concept of critical probing depth (CPD), above which the result is gain of clinical attachment and below which a loss may occur. This CPD was shown to vary depending on the type of therapy used, with scaling and root planing having a CPD of 2.9 mm and modified Widman flap surgery having a CPD of 4.2 mm. Westfelt et al. (1983) evaluated the significance of frequently repeated recall appointments on CPD in 24 patients following modified Widman surgery. The CPD values after 6 months of maintenance every 2, 4, or 12 weeks were 4.4, 4.9, and 5.4 mm respectively. CPD values for plaque-free sites were lower than for plaque-containing areas and did not differ among the recall groups. Lingual surfaces had lower CPD values than other surfaces, and molar areas had higher CPD values than non-molar teeth. Another factor affecting the CPD may be the surrounding environment of the tooth. Leveling is a theory suggesting that a physiologic response takes place to maintain the same level of anatomical attachment throughout a given area. When healthy areas which are adjacent to pathologically deepened sites are disturbed, one may see leveling, or the loss of attachment in the "healthy" sites and gain of attachment in the deepened sites.

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Section 2. Tooth Mobility

DEFINITIONS

Fremitis: A palpable or visible movement of a tooth when subjected to occlusal forces.

Tooth Mobility: The degree of looseness of a tooth beyond physiologic movement.

MEASUREMENT OF TOOTH MOBILITY

The most commonly used clinical index for mobility is the Miller Index (Miller, 1950). Mobility is detected by using an instrument (e.g., mirror handle) on either side of the tooth and applying force. Using this index, mobility is scored as follows:

1 = first distinguishable sign of movement greater than "normal;"

2 = movement of the crown up to 1 mm in any direction; and

3 = movement of the crown more than 1 mm in any direction and/or vertical depression or rotation of the crown in its socket.

Instruments that have been used to measure and study tooth mobility include the macroperiodontometer, microperiodontometer, and the Periotest. The macroperiodontometer was developed by Muhlemann (1954); however, its application was limited to the anterior teeth and premolars.

The microperiodontometer was developed by O'Leary and Rudd (1963) and proved to be useful in measuring mobility in all of the teeth. Due to the time required to obtain mobility measurements with these instruments, use was essentially limited to research.

More recently, the Periotest has provided an objective means of assessing tooth mobility. The instrument is compact, resembling a dental handpiece, and has an electromagnetically retracting tapping head. The tapping head has a preset constant speed of 0.2 meters per second, and the contact time with the tooth varies from 0.3 to 0.2 milliseconds. Contact time upon impact is less in teeth whose damping by the periodontium is greater (more support), and is therefore less mobile. A strong association between the Periotest value and bone loss has been reported (Shulte et al., 1992). The Periotest has also been suggested as a means of objectively quantifying bone apposition around dental implants (Teerlinck et al., 1991).

DYNAMICS OF TOOTH MOBILITY

The periodontal ligament (PDL) surrounds the roots of the teeth and acts as a shock absorber to some extent. Collagen fibers constitute 50% to 75% of the PDL volume with the oblique fibers predominating (Weatherford, 1977). Tooth mobility seems to occur in two stages (See Weatherford, 1977, for review). First, there is an initial or intravascular stage where movement within the socket is associated with redistribution of the fluids, interstitial contents, and fibers. The second stage occurs gradually and includes elastic deformation of the alveolar bone proper in response to increased forces (Muhlemann, 1967).

TYPES OF MOBILITY

Physiologic mobility is movement that occurs with normal function (100 to 150g). It will vary from tooth-to-tooth and day-to-day and has been defined as movement up to 0.2 mm horizontally and 0.02 mm axially (Weatherford, 1977).

Pathologic mobility may be 10-fold that of "physiologic mobility" and is associated with damage to the PDL initiated by injury to the collagen fibers and associated loss of osseous support. Fremitus is a palpable or visible movement of a tooth when subjected to occlusal forces. Perlitsh (1980) described a "critical mass" of alveolar bone support. He speculates that if < 50% of the total root length remains surrounded by alveolar bone, zones of injury from excessive occlusal forces are irreversible and may involve the entire PDL space. Conversely, alveolar bone support

greater than the "critical mass" provides healthy tissue for repair and changes are reversible. The critical mass for molars is located more coronal due to the complications of the furcations. This is an interesting concept and may be useful in assessing the prognosis of periodontally affected teeth.

CLINICAL IMPLICATIONS OF TOOTH MOBILITY

Ericsson and Lindhe (1984) subjected dogs to excessive jiggling forces (healthy periodontium) and found increased mobility due to loss of bone volume but no loss of CT attachment (physiologic adaptation). When an experimental periodontitis was initiated, no additional loss of attachment was seen compared to control sides. The authors concluded that the permanently increased mobility had no influence on the development of periodontitis.

Perrier and Polson (1982) induced an experimental periodontitis in squirrel monkeys; 10 weeks later, jiggling trauma was imposed for 10 weeks in the presence of good plaque control. Results showed that occlusal trauma in a reduced periodontium caused no additional attachment loss or bone height loss if inflammation was controlled by effective plaque control. However, additional loss of bone volume was seen.

Fleszar et al. (1980) examined the relationship between tooth mobility and clinical responses to periodontal therapy in the Michigan longitudinal studies. The authors reported that shallow sites lost attachment over time but that initially mobile teeth tended to lose more attachment. The 4 to 6 mm sites that were non-mobile initially gained attachment while the 4 to 6 mm sites with 2 and 3 degrees mobility lost some attachment by the second year. All teeth with deep pockets (7 to 12 mm) gained attachment following treatment but mobile teeth (2 and 3 degrees mobility) did not gain attachment.

Kerry et al. (1982) examined changes in mobility over time after 4 modes of periodontal therapy. The authors found that abnormal mobilities tended to decrease following the hygienic phase of therapy. Modified Widman flap therapy, scaling and root planing, and curettage had no influence on further mobility while pocket elimination therapy increased mobility after surgery, decreasing to presurgical levels after 1 year.

Muhlemann and Rateitschak (1957) examined changes in mobility patterns following selective grinding. Teeth in hypofunction were 30% more mobile than those in hyperfunction. Teeth in hypofunction had a decreased width of the PDL; the fibers become less well-arranged and are aligned more parallel to the root (non-functional arrangement). After selective grinding, teeth in both hypofunction and hyperfunction became less mobile; these observations were interpreted by the authors as improvement in periodontal health.

Gillespie and Chasens (1979) showed that supracrestal fibers do not provide support for a healthy premolar; however, as the amount of bone loss increases and the support decreases, the significance of the support offered by the supracrestal fibers increases.

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Section 3. Radiographic Interpretation

Limitations of Radiographs

Radiographs do not: 1) show periodontal pockets; 2) distinguish between successfully treated and untreated cases; 3) record morphology of bony defects; 4) show structures on buccal, lingual, and labial aspects of tooth; 5) show soft-to-hard tissue relationships; or 6) record tooth mobility (Prichard, 1983).

BENEFITS OF RADIOGRAPHS

Radiographs effectively accomplish the following: 1) record (with correct technique) position of septal bone on the tooth in one plane; 2) serve as an adjunct to the clinical exam but cannot offer conclusive evidence alone; 3) record the alveolar bone, alveolar process, and PDL on mesial, distal, and apical aspects of the root in a single plane; 4) document clinical-crown-to-clinical-root ratio; and 5) allow

observation of dense deposits of calculus and metallic restorative margins on proximal tooth surfaces (Prichard, 1983).

INTREPRETATION OF RADIOGRAPHS

Interdental Septa

In the absence of periodontal disease, the configurations of the crests of the interdental septa are determined by relative positions of the cemento-enamel junction (CEJ). When periodontal disease is present, alterations in interdental septa are governed principally by specific pathologic processes. The shape and size of crowns of the teeth, state of eruption, and position of teeth can influence septal contour. If approximating tooth surfaces are relatively flat, septa will be more narrow and pointed. If mesial and distal tooth surfaces are extremely convex, interdental septa will be wide with flat crests. The greater the buccal-lingual dimension of the teeth, the greater the width of the interdental bone. If there is a difference in length of the crowns of adjoining teeth whose occlusal surfaces are in the same plane, the crest will slant upward from the CEJ of the long crown toward the CEJ of the short crown. Any inclination of long axis of the teeth results in a difference in the levels of the mesial and distal CEJs and produces oblique alveolar crests. Assuming mesial and distal contacts between the teeth, there is no correlation between occlusal disharmonies and the radiographic appearance of the crests (Ritchey and Orban, 1953).

Lamina Dura

Using 17 autopsy specimens, Manson (1963) found that the appearance of the lamina dura is determined as much by shape and position of the tooth root in relation to the x-ray beam as by the integrity of this plate of bone. Using microradiographs, he also noted that the bone comprising the socket wall (cribriform plate) has the same mineral content as adjacent bone. The author was able to produce a pseudo-lamina dura as an artifact and concluded that critical interpretation of the integrity of the lamina dura should be avoided. Greenstein et al. (1981) studied the relationship of the crestal lamina dura to clinical parameters in 90 subjects. They reported no correlation between clinical parameters of bleeding pockets or attachment loss and the presence or absence of crestal lamina dura. The authors recommended caution when using the integrity of the crestal lamina dura as an indicator for diagnosis of periodontal disease and predictor of therapeutic needs.

Osseous Defects

Bender and Seltzer (1961), using human mandibles obtained at autopsy, compared artificially-created periodontal and periapical lesions clinically and radiographically. They reported that lesions could not be observed on radiographs as long as they were confined in cancellous structures. However, if lesions encroached on the cancellous-cortical junctions, they were visible. If the inner surface of the cortex was eroded further, the area became even more discernible. Ramadan and Mitchell (1962) reported that: 1) minor destructive changes in the alveolar crest could not be detected by x-ray; 2) destruction of the buccal plate could not be distinguished from destruction of the lingual plate; 3) funnel-shaped defects with intact buccal and lingual plates could not be detected; 4) the long-cone paralleling technique is the most reliable for obtaining acceptable images; 5) removal of the entire buccal and lingual plate did not affect the trabecular pattern; 6) bone destruction caused by abscesses is not seen if it is superimposed by roots; and 7) both junctional and central core of trabecular bone must be removed to affect radiographic architecture.

Rees et al. (1971) examined the radiographic appearance of alveolar osseous defects in dry skulls to determine the predictability of diagnosing the defects on the basis of radiographic appearance. They found that proximal osseous defects and furcation defects on the facial and lingual surfaces of multi-rooted teeth can be identified with a high degree of accuracy based on their radiographic appearances. Conversely, lesions on the facial or lingual root surfaces are extremely difficult to recognize radiographically. These studies indicate that although radiographs are valuable adjuncts, clinical and radiographic findings must be correlated in order to facilitate a correct diagnosis.

Periodontal Ligament (PDL)

Using an artificial model, Van der Linden and Van Aken (1970) reported that the same width of the PDL can be interpreted as being different when the radius of circumference is different, exposure time is changed, or when kilovoltage is changed. The number of PDL projections depends upon the width and depth of root concavity and the thickness of the PDL projections depends upon the width and depth of root concavity and the thickness of the PDL, as well as horizontal angulation of the x-ray beam. The marginal aspect of the PDL in the radiograph varies significantly with the horizontal angulation of the x-ray beam and may lead to a subjective widening or complete loss of the PDL.

Healing

In periodontitis, increased radiolucency and cupped-out appearance of alveolar crests are noted on the radiograph. The cortical layer has been destroyed and underlying marrow spaces have been exposed and enlarged, decreasing the density. After treatment, the marrow spaces become smaller and new cortical bone is laid down, increasing the density. This increased density can lead to the misinterpretation of coronal bone regeneration when, in reality, it is only an increase in quality of bone (Friedman, 1958).

Normal level of crestal alveolar bone: Hausman et al. (1991) evaluated 13- to 14-year-old children to determine the average distance of the CEJ to alveolar bone. They found the average distance was 0.4 to 1.9 mm (mean 1.1

mm) and suggested that this distance increases with age as a result of continuous eruption. Goodson et al. (1984) examined the relationship between changes detected on radiographs and changes in clinical attachment levels using standardized radiographs. They examined 231 sites and observed that clinical attachment loss precedes visual radiographic changes by 6 to 8 months and, in all cases, clinical attachment changes were greater than observed radiographic changes. Radiographic change was not always detected in sites exhibiting clinical attachment change. The authors stated that radiographic changes may have been detected sooner if subtracted images had been used.

XERORADIOGRAPHY

Xeroradiography is a diagnostic x-ray imaging system which uses the xerographic copying process to record x-ray images. Xeroradiographic images (XIs) differ from conventional images, having greater exposure, latitude, and a property termed "edge enhancement" by which fine structures (bone, trabeculae, etc.) and areas of subtle density differences (gingiva, etc.) are visually enhanced. Conventional dental x-ray units can be used to produce high-quality dental XIs at significantly reduced radiation levels. In a human study with 96 patients, similar x-ray projections were made with conventional film and experimental dental xeroradiographs. Resultant images were compared visually and, in all categories (gingival soft tissues, calculus deposits, osseous tissues), information provided by XIs was equal to or greater than conventional radiographs. The authors found dental xeroradiographs to be a highly accurate, low in radiation, rapid, and convenient alternative to conventional intra-oral radiography. There have been, however, numerous technical difficulties with the processing equipment which have limited its use (Gratt et al., 1980).

SUBTRACTION RADIOGRAPHY

Subtraction radiography is a technique which uses computer-assisted imaging to convert different densities recorded on a conventional x-ray film into digitized gray level images. The gray level images of a second film are superimposed over the first and differences subtracted. Two identical films would result in all gray levels being subtracted, leaving a blank image. Differences in bone density over time (gain or loss) would be recorded as different digitized gray levels with subtracted images reflecting gains or losses of density. The rationale for its use is based on the fact with conventional radiographs, more than 30% of the bone mass at the alveolar crest has to be lost (or gained) before it can be recognized. Subtraction radiography can detect changes in bone density as small as 5%. The sensitivity of subtraction to accurately detect changes in bone depends on radiographs with standardized geometry, allowing precise superimposition. If the radiographic images cannot be completely aligned, areas of differing gray levels (structured noise) may appear on the subtracted image, making it difficult to distinguish from gray level variations due to actual bone changes. This has been a major disadvantage of the technique to this point (Hausmann et al., 1985). In a review of radiographic techniques for clinical trials, Reddy (1992) noted that subtraction radiography gives precise information, the technique is time consuming and labor intensive, and advances have been made in subtraction radiography with the digitalization of images.

DIGITAL IMAGING

New computer and video technology has led to the development of digital subtraction radiography (DSR). Light intensity transmitted through a radiograph is measured at each picture element (pixel) by a video camera and converted into gray-level values. The digitized image is stored on a computer and displayed on a TV screen as a positive image. A subsequent radiograph is displayed as a negative image on the screen and aligned to the structures of the baseline image revealing differences in density between baseline and subsequent radiographs. Numerous studies using artificially-created bone defects in dry skulls, cadavers, and animals have determined the diagnostic accuracy of DSR (Braegger, 1988A, 1988B). Few studies have dealt with the naturally-occurring lesion. Hausmann et al. (1986) were able to demonstrate bone changes in 9% of sites in 9 of 15 patients with untreated periodontitis over 6 months using DSR. Braegger (1988A, 1988B) found that bone density changes assessed with computer-assisted densitometric image analysis (CADIA) correlated well with actual calcium loss. Braegger et al. (1987) also detected surgically-induced bone loss (crown lengthening or flap osteoplasty) with a sensitivity of 82% and specificity of 88% by means of CADIA. DSR (i.e., CADIA) enables smaller changes in alveolar bone density, undetectable by conventional radiography, to be detected and quantified. Deas et al. (1991) used CADIA to determine if changes in bone density could be an indicator of progression of periodontitis; 38.3% of the sites investigated lost radiographic density and only 6.1% of sites showed loss of attachment. The authors suggested there was a complex relationship between loss of attachment and changing bone densities and that progression of disease cannot be based solely on loss of bone density.

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Section 4. Mucogingival Considerations

The question of whether or not to treat areas diagnosed as having little or no attached gingiva has been a matter of controversy in the past several years. Prior to the 1980s, preventive soft tissue grafting of areas with minimal attached gingiva was the accepted routine. This was prompted by such studies as that by Lang and Löe in 1972. Using Schiller's stain to identify the mucogingival junction in patients with optimal oral health, these authors reported a correlation between the width of keratinized tissue and attached gingiva and periodontal health. While over 80% of the surfaces with ≥ 2 mm of keratinized tissue and ≥1 mm of attached gingiva were clinically healthy (76% had no gingival exudate), all surfaces with < 2 mm of keratinized gingiva and < 1 mm or more of attached gingiva exhibited clinical inflammation and varying amounts of gingival exudate. These findings suggested that gingival inflammation results at least in part from a movable gingival margin which facilitates the introduction of microorganisms into the sulcus. It was concluded that a width of keratinized tissue of 2 mm (with at least 1 mm being attached) is adequate to maintain gingival health.

Other studies, however, have not supported routine grafting of sites with minimal or no keratinized or attached gingiva. Ten unilateral and 6 contralateral study pairs of premolar teeth, each with one tooth having minimal keratinized gingiva ≤ 1 mm with no attached gingiva and the other having appreciable keratinized tissue (≥ 2 mm) were evaluated for gingival health (Miyasato et al., 1977). The 6 contralateral study pairs were observed over a 25-day period of experimental gingivitis (no oral hygiene). Sites with plaque or high frenum insertions were excluded from the study. No differences in gingival health were observed between sites with or without keratinized tissue. In addition, sites with minimal keratinized gingiva and no attached gingiva were no more prone to develop plaque-induced inflammatory changes than areas with attached gingiva and a greater zone of keratinized tissue.

Using beagle dogs, Wennstrom and Lindhe (1983) studied the effect of plaque at sites with or without attached gingiva and with varying heights of the attachment apparatus, over a 40-day period of experimental gingivitis (no oral hygiene). The results indicated that the inflammatory response to bacterial plaque accumulation is unrelated to the presence or absence of attached gingiva, or to the height of the supporting attachment apparatus. The authors concluded that a free gingival unit which is supported by loosely attached alveolar mucosa is no more susceptible to an inflammation than a free gingival unit which is supported by a wide zone of attached gingiva.

In a 1983 human study, Wennstrom examined the clinical response following the surgical removal of the entire zone of attached gingiva. He reported that gingival recession occurred only during the first 3 post-operative months and remained stable over the next 6 months independent of the presence or absence of attached gingiva or the width of keratinized tissue. Regardless of the presence or absence of attached gingiva, gingival units were without clinical signs of inflammation over the 9-month period.

Kisch et al. (1986) studied canines and premolars with no attached gingiva and mobility of the gingival margin over a 5-year period. The mucogingival margin was identified using Lugol's iodine solution. They failed to demonstrate that unattached and mobile facial gingival surfaces are more susceptible to periodontal breakdown than attached surfaces in subjects with good oral hygiene and clinically healthy gingiva.

In a 5-year longitudinal study, Wennstrom (1987) confirmed the observations from his 1983 studies. In patients maintaining good oral hygiene, the lack of an "adequate" zone of attached gingiva did not result in an increased incidence of soft tissue recession. The author hypothesized that a narrow zone of gingiva apical to a localized recession is a consequence rather than a cause of the recession.

In another longitudinal study, Kennedy et al. (1985) ex-

amined 32 patients over a 6-year maintenance period who had insufficient attached gingiva on one side and a free soft tissue graft on the other. In addition, 10 patients who had not maintained recall appointments were re-examined. Results indicated that both treated and untreated sites of all patients remained stable over the 6-year period. It was concluded that the free gingival graft is a predictable means of enhancing the zone of attached gingiva and, in time, results in creeping attachment. However, if plaque control is adequate, minimal to zero attached gingiva can be maintained in a state of health. It must be noted that control sites with little or no attached gingiva in the unmaintained patients had a 20% frequency of further recession (mean recession = 0.5 mm). This suggests at least some risk of recession in sites with little attached gingiva, whereas no recession was noted on teeth with wide zones of attached gingiva.

A 10-year longitudinal study of sites with minimal keratinized gingiva (< 2 mm of keratinized, but ≥ 1 mm of attached gingiva) in 18 dental students with good oral hygiene, minimal inflammation, and no restorations in the area of observation was completed (Freedman et al., 1992). It was observed that the majority of sites remained unchanged or had a slight increase in keratinized gingiva. It was concluded that in the absence of inflammation, areas with minimal keratinized gingiva remain stable over a long period of time.

Mucogingival considerations in restorative dentistry were addressed in a 1987 study by Stetler and Bissada. They compared the tissue response (GI) around teeth with and without subgingival margins in association with narrow (≤ 2 mm) or wide (≥ 2 mm) zones of keratinized gingiva. Higher GI scores were observed when subgingival margins of restorations were present in areas with a narrow zone of keratinized gingiva. The authors concluded that in the presence of subgingival margins, a greater inflammatory gingival response is associated with a narrow band of keratinized gingiva, although no significant differences were found in attachment levels or bone height. They indicated that if subgingival restorations were to be placed in areas of minimal keratinized gingiva and less than optimal plaque control, augmentation to widen the zone of keratinized tissue may be warranted. It was also noted that in unrestored teeth there was no significant difference in the inflammatory status of sites with or without a wide zone of keratinized tissue.

Mucogingival problems in children were discussed by Maynard and Wilson in a 1980 article. They indicated they had never observed mucogingival problems in the deciduous dentition unless created by a factitial injury. Mucogingival problems tend to originate in the mixed and early permanent dentition resulting from developmental aberrations in eruption and deficiencies in the thickness of the periodontium. The authors indicated the following mucogingival problems may progress with age and should be treated with an autogenous gingival graft: 1) marginal tissue comprised of alveolar mucosa with frenum pull; 2) exposed root surface with minimal keratinized tissue and no attached tissue; 3) labial incisor eruption with minimal keratinized tissue, no attached gingiva, and no lingual movement of the tooth is planned; 4) tooth eruption into a rotated position and minimal keratinized tissue; 5) thin periodontium and labial tooth movement is planned; 6) root exposure during orthodontic movement; 7) maxillary incisor overbite stripping keratinized tissue on facial mandibular incisors. If orthodontic treatment is anticipated, the authors felt autogenous grafts should be placed prior to therapy when mucogingival problems exist.

Tenenbaum and Tenenbaum (1986) studied the width of facial gingiva in subjects aged 3 to 15 years (using a jiggling technique), noting that attached gingiva increases with age in both the primary and permanent dentitions. However, contrary to the findings of Bowers (1963), they reported it does not increase as a result of the transition from the primary to permanent dentition. Since sulcus depth decreased with age, it was concluded that the increase in width of attached gingiva results from decreased sulcus depth. Stated in another way, although sulcus depth decreases with age and results in increased attached gingiva, width of keratinized gingiva does not vary.

Andlin-Sobocki et al. (1991) completed a 3-year longitudinal study of 28 six to 13-year olds who initially presented with labial marginal recession associated with permanent central incisors. Over the 3-year period, an overall reduction in recession occurred with a gradual gain in clinical attachment levels. Since gingival recession on the facial of mandibular incisors often decreases or is totally eliminated over time (in children), the authors suggested that surgical treatment to correct the recession should be postponed until possible spontaneous improvement has been allowed to occur.

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CHAPTER 4. DISEASE ACTIVITY

Section 1. Bleeding Upon Probing

Assessment of bleeding upon probing (BOP) is an important part of the periodontal examination. Bleeding has been demonstrated in clinical and histological studies to be a more sensitive sign of gingival inflammation than visual alterations. Meitner et al. (1979) examined 6,990 gingival surfaces visually, then probed for bleeding 1, 2, and 3 months following prophylaxis. At the first exam, 1,678 sites (24%) demonstrated a combined absence of visual inflammation and BOP. One month later, only 766 of these surfaces were still healthy. Data from surfaces that had changed since the first examination showed that there were a significantly greater number of surfaces which bled upon probing compared to either a color change only or a combination of color change and BOP. The authors concluded that gingival indices based upon BOP are more accurate than those using visual changes.

BLEEDING INDICES

Interdental bleeding following stimulation with wooden interdental cleaners forms the basis of the Eastman Interdental Bleeding Index (EIBI), while bleeding following sweeping a probe in the sulcus from the line angle to the interproximal contact forms the basis of the Papilla Bleeding Index (PBI). Caton et al. (1988) compared visual evaluation, PBI, and EIBI in 82 subjects not currently undergoing periodontal therapy. Visual signs of inflammation were seen at 71% of the sites, EIBI was positive at 65%, and PBI was positive at 42% of the sites. At sites rated visually as non-inflamed, almost twice as many inflammatory lesions were detected by EIBI versus PBI. The authors concluded that the Eastman Interdental Bleeding Index was a more reliable clinical indicator for detecting interdental inflammation than the Papilla Bleeding Index.

HISTOLOGY OF BLEEDING SITES

Histological studies have verified the presence of significantly more inflammation in the bleeding sites compared to non-bleeding sites. Greenstein et al. (1981) evaluated 60 gingival biopsies for visual presence or absence of bleeding after probing using a pressure-controlled probe set at 25g. Sites which bled on probing exhibited a significantly greater percentage (28.7% versus 19.9%) of cell rich-collagen poor connective tissue than non-bleeding sites without an increase in blood vessel lumens. Other studies have characterized the histopathological features of active periodontal lesions using bleeding as one of the criteria of disease activity. Davenport et al. (1982) found that the percentage of infiltrated connective tissue was consistently larger in bleeding lesions with suppuration than in non-bleeding lesions with suppuration. Significant differences were observed between bleeding and non-bleeding sites in terms of the mean percent volume occupied by plasma cells (68% versus 24%) and mononuclear cells (5.5% versus 22%). The pocket epithelium of bleeding lesions demonstrated thinned and ulcerated areas along with proliferation of rete pegs.

Passo et al. (1988) examined the histological features of bleeding sites with and without suppuration. They reported that the percentage of collagen-poor area in bleeding (but not suppurating) sites was similar (27.7%) to the above finding of Greenstein et al. (1981), but was higher in the suppurating area. However, suppuration was not always associated with extensive inflammation, and intense inflammation was also seen in non-suppurating sites. Thus, suppuration did not appear to be a specific indicator of a destructive periodontal lesion. Reinhardt et al. (1988) examined the lymphocyte subpopulation in periodontal active lesions characterized by bleeding and progressive attachment loss.

Polymorphonuclear leukocytes (PMNs) were seen only in 3.7% of the sulcular third of stable sites and in 5.6% to 8.3% of the sulcular third of active sites. Lymphocyte density was greater in the sulcular thirds than in the oral thirds. When compared to stable sites, active sites showed an increased number of plasma cells and a reduced T/B ratio. The T helper/T suppressor ratio did not vary significantly between blood and gingival tissue of any disease group but seemed to follow a trend toward lower numbers of T helper cells. These results indicated that active periodontal lesions characterized by bleeding and attachment loss displayed elevated B cell population and abnormal immune regulation possibly involving the T helper cell subset.

Badersten et al. (1990) studied the longitudinal effects of non-surgical therapy on nonmolar teeth. The diagnostic value of clinical scores of plaque, bleeding, suppuration, and probing depth in predicting probing attachment loss during the maintenance phase was investigated. A combination of linear regression and end-point analysis was used to determine probing attachment loss over the 0 to 60 month period. The authors found that all of the investigated scores were associated with clinical attachment loss and that improved diagnostic predictability was noted with an increased length of time in recording scores. The positive predictive value (Pv+) of accumulated plaque or accumu-

lated bleeding scores reached a maximum of about 30%, corroborating the findings of Lang et al. (1986). A residual probing depth of ≥ 7 mm had about a 50% Pv+ while increased probing depths ≥ 1.0 mm reached 80% Pv+ after 60 months. This led to the conclusion that an increase in probing depth, as opposed to the presence of bleeding on probing, was most valuable in predicting probing attachment loss (our "gold standard" of disease activity?). The use of lower probing pressure (< 0.75 Newton [N]) used in this study) and selection of only those sites with marked bleeding might have improved the predictive value of bleeding upon probing. Suppuration only reached a maximum of 20% positive predictive value, probably because it was a rare occurrence.

Claffey et al. (1990) did a parallel study which included molars. For 42 months following periodontal therapy, plaque, bleeding, suppuration, and probing depth were recorded for 17 subjects. A probing force of 0.50 N was used and 4 of the subjects received no subgingival instrumentation during the 42 months of maintenance. Similar results to Badersten et al. (1990) were obtained: 1) accumulated plaque scores had low Pv+; 2) 41% of sites that bled on probing at 75% or more of the examinations between 3 to 42 months had undergone probing attachment loss; 3) suppuration on probing reached a Pv+ of 40 to 50% but was not a frequent finding; and 4) increase in probing depth of greater than 1 mm reached a Pv+ of 68% at 42 months. The combination of increasing probing depth with bleeding frequency at 75% or more of examinations yielded a predictability score of 87% at 42 months. The best positive predictive value was found using a combination of bleeding upon probing and an increased probing depth of 1 mm or more. A comparison of this group with that of Badersten et al. (1990) revealed higher plaque and bleeding scores in this group of patients. Furcations showed the highest incidence of probing attachment loss. The authors conclude that longer observation periods are needed if the commonly used clinical signs such as BOP and suppuration are to reach meaningful diagnostic values.

In another longitudinal study of maintenance patients, Kaldahl et al. (1990) investigated the relationship of bleeding, suppuration, and presence of supragingival plaque to attachment loss during the second and third years following active therapy which included either coronal scaling, root planing, modified Widman surgery, or flap with osseous resection. A probing force of 25 grams was used to detect bleeding on probing, while an increased pressure of 50 grams was used to record the probing attachment level. They found that as the frequency of bleeding on probing increased, the sensitivity decreased (0.82 to 0.15), the specificity increased (0.20 to 0.88), and positive and negative predictive values remained constant (Pv+ = .15–.18; Pv− = .86). For suppuration, sensitivity was extremely low but specificity was nearly 1.0 while positve predictive value increased (from 0.27 to 0.50) because of the low frequency of suppuration in the population. For supragingival plaque, as the frequency of presence of plaque increased, the sensitivity decreased (0.67 to 0.15), the specificity increased (0.40 to 0.87), and the positive and negative predictive values remained constant (Pv+ = .16–.17; Pv− = .87–.86). The authors conclude that bleeding and plaque are not good prognosticators while suppuration is a weak prognosticator over the 2-year maintenance period. The bleeding symptom associated with a non-aggressive state (gingivitis) is probably much more frequent and therefore may mask bleeding associated with an aggressive inflammatory state (periodontitis).

Greenstein and Caton (1990) published a critical assessment of periodontal disease activity concepts. Several important issues were addressed, including the fact that at any given moment, there is no practical clinical test to determine if disease activity is occurring. Longitudinal monitoring is required. If a 3 mm increase in probing attachment loss is used, as selected by Haffajee et al. (1983), as equivalent to 3 times the standard deviation association with difference between replicate probing measurements, and rounded upward, clinically significant loss may occur before initiating therapy. In this instance, the sensitivity of the test will be low and the specificity high, resulting in a high number of false-negatives and possible undertreatment. Conversely, if a 1 mm increase in probing depth is used as the standard for disease activity, variability in probing accuracy may produce significant numbers of false-positive results, with subsequent overtreatment.

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Section 2. Gingival Crevicular Fluid

DEFINITION

Gingival Crevicular Fluid (GCF): Tissue fluid that seeps through the crevicular epithelium. It is increased in the presence of inflammation.

It has been reported that the flow of GCF can be detected a few days before other clinical signs of inflammation are evident. Crevicular fluid appears during altered states of vascular permeability which may accompany gingival inflammation (Abbott and Caffesse, 1977). On this basis, its measurement has been proposed as an indicator of periodontal disease activity.

METHODS OF COLLECTION AND MEASUREMENT

GCF may be collected by two basic techniques: filter paper strips and capillary micropipettes. Measurements are accomplished by ninhydrin stain assessment or by use of the Periotron (measures filter strip wetness impedance). GCF collection methods using filter paper strips may be divided into the intracrevicular and extracrevicular techniques:

Intracrevicular

The Brill (1962) technique includes placement of a filter strip into the gingival sulcus until resistance is felt. After 3 minutes in place, the strips are removed, dried, and stained with ninhydrin (stains proteins). Löe and Holm-Pedersen (1965) proposed a method involving minimal irritation to the gingival sulcus, in which the paper strip is placed at the entrance of the crevice.

Extracrevicular

Brill and Krasse (1959) and Löe and Holm-Pedersen (1965) also employed an extracrevicular collection technique. The strips were closely adapted to the buccal surfaces of the teeth, across the gingival margin and onto the attached gingiva.

GCF collected with capillary pipettes permits the measurement of fluid volume; however, to assure accurate measurement, relatively large volumes have to be collected. This has the disadvantage of requiring a considerable amount of time in order to obtain a sample, rendering the technique impractical for clinical use. A more precise method is the use of micropipettes which determines the actual volume of GCF (Kaslick, 1970).

The Periotron employs an electronic transducer to measure GCF. This instrument measures electrical capacitance, as the insulating properties of the filter paper strip vary according to the quantity of fluid absorbed within the strip (Suppipat, 1977). The instrument evaluates the flow of current based on the wetness of the strip (impedance). A digital readout registers the area wetted and is indicative of the volume of fluid collected on the paper strip.

QUANTITATIVE ASSESSMENT

Hancock et al. (1979) studied crevicular fluid and gingival inflammation, reporting weak correlations between GCF and clinical or histologic parameters. They concluded that the quantity of fluid may have potential as a clinical indicator of presence but not severity of gingival inflammation. Shapiro et al. (1979) found no statistically significant correlation between the amount of GCF and the histological degree of inflammation. However, there was a positive correlation between the GCF and the clinical assessment of inflammation. Cimasoni (1983), in reviewing this subject, noted that a "positive correlation was always found between the clinical appreciation of gingival inflammation and the amount of gingival fluid." He also reported that the correlation between gingival crevicular fluid flow and histological inflammatory changes was poor.

It is generally agreed that gingival crevicular fluid reflects vascular permeability and thus gingival inflammation. It may indicate the presence of gingival inflammation but there is no evidence showing that it can predict periodontal breakdown or disclose the degree of inflammation. Factors such as circadian periodicity, hormonal alteration, and differences in collection technique may provide sources of error, making interpretation of findings difficult.

QUALITATIVE ASSESSMENT

Hydrogen sulfide is a cytotoxic metabolite of bacterial origin. Solis-Gaffer et al. (1980) proposed that the level of GCF hydrogen sulfide could be an indication of periodontal disease and used a quantitative method to determine the production of hydrogen sulfide from 240 GCF samples collected with paper strips. A strong positive correlation between hydrogen sulfide production and the degree of gingival inflammation was shown. Horowitz and Folke (1973) analyzed hydrogen sulfide production in patients with gingival health (probing depth 2 mm or less), periodontitis (PD $>$ 4 mm), and surgical treatment (pocket elimination). Results indicated that hydrogen sulfide generation increased with sulcular depth and that toothbrushing failed to reduce hydrogen sulfide production in periodontal pockets. These findings appear to favor pocket reduction surgical procedures.

PMN RESPONSE TO CHEMOTACTIC CHALLENGE

Neutrophils (PMNs) are the predominant cell type in the gingival sulcus during the initiation and progression of per-

iodontal disease (Attstrom, 1971; Page and Schroeder, 1976). These cells are responsive to a number of chemotactic factors produced by the gingival microflora and by activated products of the complement system. Most studies have indicated that neutrophils play a protective role in periodontal tissues (Van Dyke et al., 1980). PMN dysfunction (especially defective chemotaxis) has been observed in many cases of severe periodontal disease. It has been shown that PMNs migrate through the gingival connective tissue and junctional epithelium into the gingival sulcus (Van Dyke et al., 1980). Monitoring crevicular PMNs has been advocated as a means of assessing periodontal disease activity.

Golub et al. (1981) described a technique (the sulcular technique) for assessing neutrophil chemotaxis in vivo. Singh et al. (1984) utilized this method to monitor patients with gingivitis, chronic periodontitis, and LJP. In gingivitis and chronic periodontitis, the response to the chemotactic agent (casein) was similar to normal subjects except that the peak cell count was greater. LJP patients showed an abnormal response with two leukocyte peaks compared with a single peak in controls. This in vivo assay of neutrophil response could be useful in determining susceptibility to periodontal breakdown and individual treatment regimens.

Lamster et al. (1985) studied enzymatic profiles in gingival fluid. Arylsulfatase and B-glucuronidase were selected as indicators of the breakdown of connective tissue ground substance. Evidence suggests that ground substance integrity is important for maintenance of the collagen component of the connective tissue. The authors found that the ground substance degrading enzyme activity in crevicular fluid plateaued 2 to 3 weeks after the initiation of experimental gingivitis. The authors observed that this peak in enzyme activity corresponded with the established lesion of gingivitis. Similarly, Oshrain et al. (1984) found greater arylsulfatase volume activity in patients with gingivitis and periodontitis than in normal subjects.

Elevation of serum levels of aspartate aminotransferase is frequently used as a diagnostic indicator of myocardial infarction and as a quantitative measure of the extent of tissue destruction. Chambers et al. (1984) hypothesized that cell death during active periodontitis would release aspartate aminotransferase into the crevicular fluid. They studied the levels of this enzyme in GCF in a ligature-induced periodontitis model in the beagle dog. Results showed significant increases in aspartate aminotransferase levels 2 weeks after ligation. The authors suggested that this peak represented the acute destructive episodes of active periodontal disease.

Lamster et al. (1988) performed a longitudinal evaluation of GCF levels of B-glucuronidase, arylsulfatase, and lactate dehydrogenase in patients with chronic adult periodontitis. They determined that increased GCF levels of B-glucuronidase (a marker for primary granule release from PMNs) was a very strong predictor of impending clinical attachment loss. In contrast, neither arylsulfatase nor lactate dehydrogenase was able to predict attachment loss.

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Section 3. Bacterial Flora

In recent years, traditional clinical assessments of disease progression have been questioned. Haffajee et al. (1983) demonstrated that common clinical assessments of inflammation (gingival erythema, bleeding on probing, and

suppuration) were poor predictors of progression in an untreated population. Similar results were reported by Badersten et al. (1985) and by Lang et al. (1986) who showed that repeated measurements (4/4) of bleeding on probing at maintenance visits was followed by a loss of attachment at only 30% of possible sites. These and other studies suggest that since it is impossible to clinically detect sites which are actually undergoing attachment loss, treatment of all inflamed sites is necessary.

Although bacterial plaque has long been accepted as the primary etiology of periodontal disease, there is some debate over the exact etiologic mechanism of disease activity. The "non-specific plaque" hypothesis proposes that the etiologic mechanism in periodontitis is related to total plaque mass, therefore, reducing the amount of plaque will curtail disease activity. According to the "specific plaque hypothesis," periodontal disease activity is related to specific bacterial species and their products. The latter theory suggests that identification and eradication of putative periodontopathogens should be the goal of treatment. Microbial monitoring is based on the specific plaque hypothesis. In reality, mechanisms mediating periodontal diseases probably relate to both hypotheses or theories. The non-specific theory may be more relevant to chronic adult periodontitis, whereas the specific plaque theory may be more applicable to distinctive periodontal disorders; e.g., localized juvenile periodontitis (Listgarten, 1988).

Since clinical tests are currently unable to provide adequate disease assessment and predictability and since specific microbiologic species seem to be related to disease activity, microbiologic tests were sought as viable alternatives or adjuncts to clinical parameters.

COMPOSITION OF SUBGINGIVAL FLORA TO DISEASE ACTIVITY

Analysis of Bacterial Morphotypes (Darkfield Microscopy [DFM])

With DFM, light enters the microscopic field peripherally so that microorganisms are obliquely illuminated and glow against a dark background.

Listgarten and Hellden (1978) described a technique for classifying bacterial samples on the basis of constituent shape, size, and motility. Two diseased and two healthy sites from 12 patients with severe periodontitis were studied. Clinical parameters (gingival fluid flow, gingival index [GI], periodontal index [PI], and probing depth) were recorded and plaque samples obtained from apical sites of each crevice. Samples were diluted and observed with darkfield microscopy; bacteria were categorized into 9 morphotypes. Results indicated significant differences between flora at healthy and diseased sites. At healthy sites, coccoid cells predominated (74.4% versus 22.3%), while diseased sites presented a greater number of motile rods, curved rods, and spirochetes. The ratio of motile to non-motile cells at healthy sites was 1:49, while at diseased sites the ratio increased to 1:1. The authors suggest that this technique may be used to predict sites with active disease based on percentages of certain bacterial morphotypes.

Listgarten and Levin (1981) studied the correlation between proportions of subgingival spirochetes and motile bacteria and susceptibility of subjects to periodontal deterioration. At baseline and every 2 months for 1 year, clinical and microbiological parameters were recorded for 20 patients treated for moderate-advanced periodontitis. Microbial samples from the deepest pocket in each quadrant were pooled for analysis using DFM. Microbial forms were recorded as coccoid, motile rods, spirochetes, or others. When probing depth at any surface exceeded baseline by $\geq$ 3 mm, the tooth was exited from the study. A significant positive correlation was found between proportions of motile rods and PI and GI scores. Also, a positive correlation between PD and proportions of spirochetes was observed. Spirochetes and/or motile rods predominated in subjects who had 2 or more teeth exited from the study, whereas coccoid forms predominated in sites without exited teeth. The authors concluded that the proportion of spirochetes is a good predictor of periodontal deterioration within the upcoming year.

Armitage et al. (1982) correlated the percentage of motile bacteria with clinical assessments of inflammatory periodontal disease. Two sites were selected from each of 60 subjects and categorized into 6 disease categories, ranging from health to advanced periodontitis. These findings were compared to DFM analysis of subgingival plaque from each site. Bacterial morphotypes were classified as non-motiles, spirochetes, or other motiles. The authors reported that severity of disease correlated significantly with an increase in the percentage of spirochetes, although no such relationship was observed for other motile bacteria. There was also a significant correlation between each clinical parameter (PI, GI, BOP, PD, gingival exudate, attachment loss) and the percentage of spirochetes at individual sites. Bleeding on probing and/or probing depth or loss of attachment $>$ 3 mm were the parameters most closely related to the percentage of spirochetes. In fact, there was an 80% correlation between BOP and high levels (2 to 3 fold increase) of spirochetes. Due to the high correlation of clinical parameters with DFM findings, the need for microbiologic assessment of subgingival flora was questioned.

Rosenberg et al. (1981) examined composite bacterial populations before, during, and after various stages of periodontal therapy. Eighteen (18) patients with moderate periodontitis were scored for PI, GI, and PD. The tooth surface with the deepest pocket in each sextant (per patient) was chosen as the microbial sampling site. Subgingival plaque samples were obtained at baseline, after initial preparation, and at various times after surgical therapy. A pooled sample from each patient was studied by DFM for each time period. There was a significant increase in the mean percentage of coccoid cells and significant decrease in the

proportions of spirochetes and motile rods from baseline to post-initial therapy and from post-initial therapy to post-surgery. In spite of continued improvements in microbiological parameters, little or no change in clinical indices was noted after the surgical phase. Two of 18 patients exhibited increases in motile rods and spirochetes, accompanied by a deterioration of clinical parameters during the treatment period. The authors suggest that despite treatment rendered, microflora of these sites were related to increased disease susceptibility rather than periodontal health.

Evian et al. (1982) correlated proportions of bacterial morphotypes at different diseased sites within the same mouth, determining the extent of correlation with the clinical parameters of periodontitis in 14 patients with untreated moderate to advanced disease. After clinical indices (PD, PI, GI) were recorded, microbial samples were obtained from the deepest pocket in each sextant and examined individually by DFM. Bacterial morphotypes were categorized as cocci, motile rods, spirochetes, or others. Proportions of morphotypes among the 6 sites in any individual mouth varied considerably. In accordance with earlier studies, the mean percentage of spirochetes varied directly with increased PD, PI, and GI scores. However, no significant correlations could be determined when measurements of individual sites were analyzed. The authors suggest that differences in disease activity among sites as well as variability due to the sampling process may account for the results. The authors suggest that by pooling samples from individual patients, it may be possible to minimize sampling errors and still yield useful information about sites at risk.

CULTURAL STUDIES

Tanner et al. (1979) characterized the microbial flora at the apical region of advancing periodontitis lesions. Criteria of disease activity in 8 subjects with advanced periodontitis consisted of at least 2 mm of radiographic bone loss in the previous year. Anaerobic bacterial samples were obtained and cultured. Correlations between bacterial and clinical parameters were statistically evaluated. Microbiota of 2 young adult patients with generalized bone loss and inflammation were dominated by *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans.* Sites of 2 patients with extensive bone loss but minimal inflammation were dominated by *Prevotella intermedia, Eikenella corrodens,* and fusiform-shaped *Bacteroides* (probably *Bacteroides forsythus*). Sites of the remaining 4 patients had moderate inflammation with continued bone loss and demonstrated high levels of *P. gingivalis, F. nucleatum, "fusiform" Bacteroides,* and anaerobic *Vibrios.* Sites with minimal disease revealed higher proportions of Gram-positive organisms. The authors concluded that significant differences do exist in the apical plaque of adult periodontitis patients and that it seems likely that specific groups of organisms are related to certain clinical diseases.

RAPID IDENTIFICATION OF POTENTIAL PATHOGENS

Investigators have attempted to develop methods for rapidly identifying certain bacteria or disease activity based on bacterial antigenic profiles (DNA probes, latex agglutination), enzymatic activity (B-glucuronidase, collagenase, etc.), metabolic end products and/or antibody titers. Review articles by Armitage (1987) and Greenstein (1988) discuss some of the advantages and disadvantages of various rapid identification techniques.

DNA Probes. These are comprised of radio-labeled DNA fragments of specific bacteria such as *P. gingivalis.* Theoretically, when the labeled fragments are added to a digested plaque sample adhering to nitrocellulose, they will combine with any analogous fragments (*P. gingivalis* if present), allowing identification and quantification of the specific organism if present. This method is site specific and simple, but disadvantages include decay of the label, inter-reaction of labeled fragments, and cross-reactivity with DNA of other species.

Latex Agglutination. In this technique, plaque samples are mixed with latex beads that are coated with antibodies specific for certain periopathogens. If the pathogen is present in the plaque sample, cross-bridging of the antibody-pathogen-antibody results in visibly detectable agglutination of the beads. This technique has potential, but is not widely used.

Flow Cytometry. This technique involves reaction of plaque with fluorescein-tagged antibodies specific for selective pathogens. The sample is then dispersed and "tagged" pathogens are counted as they flow past a spectrometer. Disadvantages include equipment expense and an inadequate data base related to plaque microbe determination.

Use of ELISA. In this enzyme-linked immunosorbent assay, plaque samples are diluted and attached to a polystyrene plate to which pathogen specific antibodies (tagged with enzyme substrate) are added. Unattached antibody is then washed off and the plate reacted with an enzyme that is specific for the substrate tag. The remaining antibodies which are attached to the pathogens can be detected by spectrophotometry for color change.

Direct Immunofluorescence (IF). In this technique, fluorescein tagged antibodies are reacted with plaque dilutions and specific staining of organisms is observed using a fluorescent microscope.

Indirect IF. Here untagged antibodies are initially complexed with the antigen. The second step involves addition of fluorescein-tagged antibodies which will react with ("piggybacking") the first antibody (antisera). This gives a much higher fluorescein signal and has been used successfully to detect specific microbes in plaque samples (good for qualitative analysis, time consuming for quantitative analysis). Disadvantages: monoclonal antibodies are so specific they may overlook some of the organisms and polyclonal anti-

bodies may cross-react with other species. Indirect immunofluorescence is probably not clinically feasible.

When complex mixed infections make it difficult to single out individual pathogens, increased antibody titers to particular organisms may suggest pathogenicity. The problem with measuring serum antibody titers is that high levels are not always associated with disease progression, and non-specific cross-reactivity may occur. Titers are sometimes low in the presence of disease progression. This is explained by the fact that the systemic response (blood serum antibodies) may lag behind disease initiation. This lag may continue well beyond resolution of diseased sites and may also influence antibody titers to specific organisms.

RELEVANCE AND CONCERNS

Initially, darkfield studies appeared to offer a means for relating disease activity to proportions of motile bacteria, especially spirochetes. Evian et al. (1982), however, suggested that spirochetes are so ubiquitous in nearly all forms of periodontal disease that their mere presence in bacterial sample is of limited diagnostic value.

The initial study by Listgarten and Levin (1981) was encouraging, because it suggested that spirochetes and motile rods could be used to identify subjects at risk for future breakdown. In a subsequent study, however, Listgarten et al. (1984) were unable to use the percentage of spirochetes in a subgingival sample to identify specific sites at risk for breakdown. In addition, a separate study by the same group (Listgarten et al., 1986) was unable to effectively use microscopic monitoring to prevent recurrence of periodontitis by altering maintenance regimens. When pooled subgingival samples from a patient were examined for spirochete prevalence, levels of spirochetes tended to correlate with disease activity although site of the activity is not identified by this method. However, when levels of spirochetes at similar (> 6 mm) sites within a patient are examined, a correlation between disease activity and number of spirochetes could be shown. Other factors discouraging the use of DFM to monitor disease activity are that the major putative periodontopathogens (e.g., *P. gingivalis, A. actinomycetemcomitans*, and *P. intermedia* are non-motile and would be discounted by a method based solely on bacterial motility and/or number of spirochetes (Slots and Listgarten, 1980).

Technical difficulties associated with cultural studies include: 1) location of plaque sample; 2) tendency of various methods of dispersing bacterial plaque to favor growth of one species over another; and, 3) unavailability of a single culture media/method capable of recovering all bacterial species in subgingival plaque. Selective media may disinfranchise important species and purportedly "non-selective media" may "select" for different segments of microbiota. These 3 factors may be partially responsible for the variety of microbiological findings reported by different laboratories. Furthermore, probing accuracy may present a problem when attempting to associate cultural findings with probing depth. The time and expense associated with culturing presently limit its use to that of a periodontal research diagnostic tool.

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Section 4. Indices Used in Assessment of Periodontal Status

DEFINITION

Index: A relative or arbitrary system of measurement which describes or quantitates a condition. Such indices are appropriate for use in an individual patient or for epidemiological studies.

PURPOSE

Periodontal indices are designed to assess disease prevalence and/or incidence within a population or a given pa-

tient. Prevalence is defined as the number of existing cases in the population at a specific point in time, while incidence is the number of new cases of a disease from one time point to another. The long latent period of periodontal disease makes it difficult to determine a definite time of disease onset, a fact needed to establish incidence rates. Prevalence of disease is usually the main determinant in population health need surveys. For periodontal disease, the degree and severity of inflammation must be determined in order to determine treatment needs. All periodontal indices in current use measure morbidity. However, none are designed to measure tooth mortality related to periodontal disease.

TYPES

Indices may be reversible, irreversible, or composite (See Tables 1 through 8) (Barnes et al., 1986). An irreversible index measures the permanent tissue damage caused by disease. Indices which record radiographic bone loss and attachment loss are considered irreversible. Reversible indices assess active disease and allow for changes in periodontal health status. Examples are: Russell's periodontal index (PI), Ramfjord's periodontal disease index (PDI), and Greene and Vermillion's oral hygiene index (OHI), and OHI-simplified. The PI and PDI indices have reversible and irreversible components and are considered to be composite indices.

Löe (1967) described the gingival index, the plaque index, and the retention index systems. The gingival index (GI) describes qualitative changes in the gingival soft tissue at 4 areas on the tooth (buccal, mesial, distal, and lingual). A score of 0 to 3 is given for each area based on visual characteristics of inflammation after drying of the tissues and the presence or absence of bleeding when a probe is run along the soft tissue wall of the entrance of the gingival crevice. The GI may be used to determine a GI for the tooth (sum of the 4 areas divided by 4), a GI for a group of teeth (grouping scores of incisors, premolars, molars), or a GI for the individual (adding the indices for the teeth and dividing by the total number of teeth examined). Criteria for the GI are: 0 = normal gingiva; 1 = mild inflammation-slight change in color, slight edema, no bleeding on probing; 2 = moderate inflammation-redness, edema, and glazing with bleeding on probing; 3 = severe inflammation-marked redness and edema, ulceration, and spontaneous bleeding.

The plaque index (PI) describes the thickness of the soft debris aggregates in the gingival area of the tooth surfaces, and no attention is paid to the coronal extension of the plaque. A score from 0 to 3 is given for the same areas evaluated in the GI, but is based on the amount of soft matter detected following drying of the tissues and running a probe across the tooth surface at the entrance of the gingival crevice. PI should precede GI when the two indices are used together. Criteria for the PI are: 0 = no plaque in the gingival area; 1 = a film of plaque adhering to the free gingival margin and adjacent area of the tooth, recognized by running a probe across the tooth surface; 2 = moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface which can be seen by the naked eye; 3 = abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface. The retention index describes the quality of the tooth surface as it relates to the presence of retentive factors such as calculus, ill-fitted margins, and carious lesions. Criteria are: 0 = no caries, no calculus, no imperfect margin of dental restoration in gingival location; 1 = supragingival cavity, calculus, or imperfect margin of restoration; 2 = subgingival cavity, calculus, or imperfect margin of restoration; 3 = large cavity, abundance of calculus or grossly insufficient marginal fit of restoration in a supra- and/or subgingival location.

Chaves et al. (1993) evaluated the association between gingival index (GI) bleeding (GI = 2 or 3) and bleeding on probing (BOP) in 125 gingivitis patients (19 to 62 years old). Clinical parameters included GI and PI with a manual probe, probing depth (PD), and BOP with a Florida probe. BOP was considered present if bleeding occurred within 20 seconds of probing. PD ranged from 0.1 to 5.9 mm; 98.6% of depths were 0.1 to 4 mm. Mean GI bleeding was 35.3% and mean BOP bleeding was 40.9%. When sites were evaluated, BOP showed a positive correlation with PD as did GI with PI. There was a good overall correlation between GI and BOP, and agreement varied with PD. Highest agreement between GI and BOP bleeding was for PD > 4 mm (85.4%) and PD > 2 mm (72.3%). In shallow pockets (0.1 mm to 2 mm), the highest percent agreement was for sites with GI = 0 or 1, or negative BOP (77.7%). When the relationship between BOP and visual signs of inflammation was related to PD, the percent BOP increased with increasing PD.

Muhlemann and Son (1971) concluded that bleeding from the sulcus is the earliest clinical symptom of gingivitis and that it precedes discoloration and swelling of gingival units. Sulcus bleeding index (SBI) was scored at time intervals over 17 days in 13 dental students refraining from oral hygiene measures. SBI was determined using a periodontal probe (diameter 0.5 mm) placed in the sulcus parallel to the tooth long axis at facial/lingual sites and directed towards the col at interproximal sites. A score of 1 represented a bleeding point which occurred up to 30 seconds after probing in the absence of gingival swelling or color change. At the start of the test period, 738 gingival units appeared healthy and did not bleed upon sulcus probing (score 0). After 17 days without hygiene, 264 apparently healthy units (score 0) remained. The number of score-1 units (bleeding upon gentle probing in the absence of color change or swelling) increased from 88 to 470. At this time, score-2 units (bleeding with change of color) increased from 6 to 89, while only 9 gingival units were slightly swollen (score 3). Using SBI, 64.2% of gingival units at

TABLE 1. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (PLAQUE AND HYGIENE)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Oral hygiene index (OHI) (Greene and Vermillion)	Debris calculus	Segments	All except 3rd molars	Buccal, lingual	Severity	Epidemiologic surveys, clinical trials, monitoring, individual patient
Oral hygiene index simplified (OHI-S) (Greene and Vermillion)	Debris calculus	Teeth	Max (R) 1st molar Max (R) central incisor Max (L) 1st molar Mnd (L) 1st molar Mnd (L) central incisor Mnd (R) 1st molar	Buccal Facial Buccal Lingual Facial Lingual	Severity	Epidemiologic surveys
OHI-S debris modification (Glass)	Debris	Teeth	Same as OHI-S	Same as OHI-S	Severity	Epidemiologic surveys
Hygiene analysis index (HAI) (Love et al.)	Plaque	Teeth	All	Mesial, distal, facial, lingual	Presence/ absence	Patient motivation, monitoring patient progress
Patient hygiene performance (PHP) (Podshadley and Haley)	Plaque	Teeth	Same as OHI-S	Same as OHI-S	Severity	Epidemiologic surveys
Plaque index (Lennox and Kopczyk)	Plaque bleeding	Teeth	All	Mesial, distal, facial, lingual	Presence/ absence	Patient motivation, monitoring patient progress
Plaque index (Quigley and Hein)	Plaque	Teeth	All except 3rd molars	Buccal, lingual	Severity	Clinical trials, epidemiologic surveys

TABLE 2. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (CALCULUS)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Calculus Surface Index (CSI) (Ennever et al.)	Calculus	Teeth	Mandibular central and lateral incisors	Mesial, distal, facial, lingual	Presence/ absence	Clinical trials
Probe Method Calculus assessment (Volpe et al.)	Calculus	Teeth	All mandibular incisors and cuspids	Lingual (and lingual aspects of inter-proximals)	Severity	Clinical trials, epidemiologic surveys
Marginal Line Calculus Index (MLC) (Muhlemann and Villa; Villa et al.)	Calculus	Teeth	Mandibular central and lateral incisors	Lingual	Severity	Clinical trials, patient progress, patient motivation

TABLE 3. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASE (BLEEDING AND GINGIVAL)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Gingival Bleeding Index (GBI) (Carter and Barnes)	Bleeding	Teeth	All except between 2nd and 3rd molars	Interproximal	Presence/ absence	Clinical trials, patient progress, patient motivation
Sulcus Bleeding Index (SBI) (Muhlemann and Major; Muhlemann and Son)	Bleeding, gingival, color change, edema	Teeth	All maxillary and mandibular central incisors, lateral incisors, cuspids, and 1st bicuspids	Mesial, distal, facial, lingual	Presence/ absence and severity	Clinical trials, epidemiologic surveys, patient progress, patient motivation
PMA Index papillary, marginal, attached gingiva (Massler et al.; Schour and Massler)	Gingival inflammation (edema, bleeding necrosis, recession pocket formation)	Papillary marginal and attached gingival units	Around all teeth except 3rd molars	Facial, lingual	Severity and presence	Epidemiologic surveys
Gingival Index (GI) (Soumi and Barbano)	Gingival inflammation	Segments and teeth (papillary and marginal gingival units)	All	Facial, lingual	Severity	Epidemiologic surveys, clinical trials, patient progress
Gingival Index (GI) (Löe; Löe and Silness)	Inflammation (color changes, edema, bleeding, ulceration)	Teeth	Gingival, plaque and retention indices are intended to be used together as a system. All use the same teeth and same surfaces.			
Plaque Index (used with GI) (Löe)	Debris and plaque	Teeth	Max (R) 1st molar Max (R) lateral incisor	Mesial, distal, facial, lingual	Severity	Epidemiologic surveys, clinical trials, monitoring patient progress
Retention Index (used with GI) (Löe)	Calculus, caries, overhanging restorations, other retentive agents	Teeth	Max (L) 1st bicuspid Mnd (L) 1st molar Mnd (L) lateral incisor Mnd (R) 1st bicuspid			

TABLE 4. INDICES USED TO EVALUATE SIGNS, SYMPTOMS, AND ETIOLOGIC FACTORS ASSOCIATED WITH DENTAL DISEASES (PERIODONTAL)

Index (source)	Measures	Scored by	Soft tissue area or teeth examined	Aspect or surfaces examined	Presence or severity measured	Uses
Periodontal Disease Rate Index (PDR) (Sandler and Stahl)	"Periodontal Disease": inflammation crevice depth, mobility, alveolar bone resorption	Teeth	All	Mesial, distal, facial, lingual, (uses radiographs)	Presence/absence	Crude epidemiologic surveys
Periodontal Index (PI) (Russell)	Inflammation pocket depth tooth mobility	Teeth	All	Facial, lingual, mesial, distal	Severity	Epidemiologic surveys
Periodontal Disease Index (PDI) (Ramfjord)	Pocket depth, crevice depth (from CEJ)	Teeth	Max (R) 1st molar Max (R) lateral incisor Max (L) 1st bicuspid Mnd (L) 1st molar Mnd (L) lateral incisor Mnd (R) 1st bicuspid	Mesial, facial	Severity	Epidemiologic surveys, clinical trials
Gingival Index (a part of PDI) (Ramfjord)	Inflammation, bleeding, edema, ulceration	Teeth	Same as PDI	Mesial, facial	Severity	Epidemiologic surveys, clinical trials
Plaque Index (used with PDI) (Ramfjord [Shick and Ash Modification])	Plaque	Teeth	Same as PDI	Facial, lingual, mesial, distal	Presence and severity	Epidemiologic surveys, clinical trials
Calculus Index (used with PDI) (Ramfjord)	Calculus	Teeth	Same as PDI	Facial, lingual, mesial, distal	Severity	Epidemiologic surveys
Periodontal screening examination (O'Leary)	Gingival Index: inflammation	Segments	All	Gingival index, facial, lingual, interproximals	Severity	Monitor patient progress, epidemiologic surveys
Includes: Gingival Index Periodontal Index Irritant Index	Periodontal Index: pocket depth Irritant Index: plaque, calculus, overhanging restorations, unfilled	Segments Segments Segments	All All	Periodontal index, mesial, line angle Irritant index: all tooth surfaces	Severity Severity	

TABLE 5. COMPARISON OF SCORES IN FREQUENTLY USED PLAQUE AND DEBRIS INDICES

	Scores					
Index	0	1	2	3	4	5
Greene and Vermillion OHI-S Debris Index	No debris or stain	Soft debris covering not more than one third of the tooth surface	Soft debris covering more than one third, but not more than two thirds of the exposed tooth surface	Soft debris covering more than two thirds of the exposed tooth		
Glass modification of OHI-S	No visible debris	Debris visible at gingival margin, but discontinuous. Less than 1 mm in height	Debris continuous at gingival margin. Greater than 1 mm in height	Debris involving entire gingival third of tooth	Debris generally scattered over tooth surface	
Löe Plaque Index	No plaque in the gingival area	A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface	Moderate accumulation of soft deposit is within the gingival margin, which can be seen by the naked eye	Abundance of soft matter within the gingival pocket and/or on the gingival margin		
Ramfjord Plaque Index	No plaque	Plaque present on some but not all interproximal buccal and lingual surfaces of the tooth	Plaque present on all interproximal, buccal and lingual surfaces, but covering less than one half of these surfaces	Plaque extending over all interproximal buccal and lingual surfaces, but covering more than one half of these surfaces		
Quigley and Hein Plaque Index (modified)	No plaque	Separate flecks of plaque at the cervical margin of the tooth	A thin continuous band of plaque (up to 1 mm) at the cervical margin	A band of plaque wider than 1 mm but covering less than one third of crown	Plaque covering at least one third but less than two thirds of the crown	Plaque covering two thirds or more of crown
Podshadley and Haley PHP	No plaque	The tooth surface if mentally divided into 5 areas. Plaque in only one area	Plaque in two areas	Plaque in three areas	Plaque in four areas	Plaque in five areas
Lennox and Kopczyk Plaque Index	Plaque severity is not scored; only presence or absence on four tooth surfaces.					
Love Hygiene Analysis Index	Plaque severity is not scored; only presence or absence on four tooth surfaces					

TABLE 6. COMPARISON OF SCORING SYSTEM IN FREQUENTLY USED CALCULUS INDICES

Scores	Greene and Vermillion OHI-S Calculus Scores	Ramfjord Calculus Index	Ennever et al.		Volpe and Manhold probe method of calculus assessment	Muhlemann and Villa marginal line calculus index	O'Leary Irritant Index
			Calculus Surface Index	Calculus Surface Severity Index			
0	No calculus present	No calculus	Has no severity component. Scores 16 tooth surfaces for presence or absence of calculus	No calculus present	Uses a periodontal probe to measure the height and width of calculus deposits on tooth surface. Results are reported in millimeters	Uses a probe to measure height of calculus deposits on tooth surfaces. Results reported in percentage of surface covered (0, 12.5, 25, 50, 75, and 100%)	No detectable plaque, or calculus, either supragingival or subgingival is found on any tooth in the segment
1	Supragingival calculus covering not more than one third of the exposed tooth surface	Supragingival calculus extending only slightly below the free gingival margin		Calculus observable, but less than 0.5 mm in width and/or thickness			A slight amount of plaque or supragingival calculus not extending more than 2 mm from the gingival margin is found on any tooth in the segment
2	Supragingival calculus covering one third to two thirds of the exposed tooth surface and/or flecks of subgingival calculus	Moderate amount of supra- and subgingival calculus or subgingival calculus alone		Calculus not exceeding 1.0 mm in width and/or thickness			Plaque or supragingival calculus covers up to one half the exposed clinical crown on any tooth in the segment
3	Supragingival calculus covering more than two thirds of the exposed tooth surface and/or a continuous band of subgingival calculus	An abundance of supra- and subgingival calculus		Calculus exceeding 1.0 mm in width and/or thickness			Plaque or supragingival calculus covers more than one half the clinical crown or subgingival calculus deposits or overhanging or deficient restorations are detectable by probing

TABLE 7. COMPARISON OF SCORES IN FREQUENTLY USED GINGIVAL AND BLEEDING INDICES

Index	Scores					
	0	1	2	3	4	5
Schour and Massler PMA Index Papillae	Normal: no inflammation	Mild papillary engorgement; slight increase in size	Obvious increase in size of gingival papilla: hemorrhage on pressure	Ecessive increase in size with spontaneous hemorrhage	Necrotic papilla	Atrophy and loss of papilla (through inflammation)
Marginal	Normal; no inflammation visible	Engorgement; slight increase in size; no bleeding	Obvious engorgement; bleeding upon pressure	Swollen collar; spontaneous hemorrhage; beginning infiltration into attached gingiva	Necrotic gingivitis	Recession of the free marginal gingiva below the CEJ due to inflammatory changes
Attached gingiva	Normal; pale rose; stippled	Slight engorgement with loss of stippling; change in color may or may not be present	Obvious engorgement of attached gingiva with marked increase in redness. Pocket formation present	Advanced periodontitis. Deep pockets evident		
Löe and Silness Gingival Index	Normal gingiva	Mild inflammation, slight change in color, slight edema. No bleeding on probing	Moderate inflammation, redness, edema and glazing. Bleeding on probing	Severe inflammation—marked redness and edema. Ulceration. Spontaneous bleeding		
Ramfjord Gingival Index	Absence of signs of inflammation	Mild to moderate inflammatory gingival changes, not extending around the tooth	Mild to moderately severe gingivitis extending all around the tooth	Severe gingivitis characterized by marked redness, swelling, tendency to bleed and ulceration		
O'Leary Gingival Score	Normal gingiva	Slight to moderate inflammation not surrounding any teeth	Slight to moderate inflammation surrounding one or more teeth	Marked inflammation ulceration, spontaneous bleeding, loss of surface continuity, clefts of gingival tissue		
Muhlemann and Major, Muhlemann and Son Sulcus Bleeding Index	Healthy appearance, no bleeding on sulcus probing	Apparently healthy P and M showing no change in color and no swelling, but bleeding from sulcus on probing	Bleeding on probing and change of color due to inflammation. No swelling, macroscopic edema	Bleeding on probing, change in color and slight edematous swelling	Bleeding on probing, change in color obvious swelling	Bleeding on probing and spontaneous bleeding and change in color, marked swelling with or without ulceration
Soumi and Barbano Gingival Index	Absence of inflammation. Gingiva is pale pink in color and firm in texture. Swelling is not evident and stippling can usually be noted	Presence of severe inflammation. A distinct color change to red or magenta is evident. There may be swelling and loss of stippling. The gingiva may be spongy in texture	Presence of severe inflammation. A distinct color change to red or magenta: swelling, loss of stippling and a spongy consistency. Gingival bleeding upon gentle probing. The inflammation has spread to the attached gingiva			
Carter and Barnes Gingival Bleeding Index	Bleeding severity is not scored. Only the presence or absence of bleeding from a maximum of 28 gingival units following flossing is recorded.					

TABLE 8. COMPARISON OF SCORES IN FREQUENTLY USED PERIODONTAL INDICES

Scores	Russell Periodontal Index	Ramfjord Periodontal Disease Index (Crevice Depth)	O'Leary Periodontal Screening Examination	Scores
0	Negative. There is no overt inflammation		Upon probing of the gingival crevices, a score of 0 is given if the probe does not extend 1 mm apical to the CE Junction of any tooth in the segment and there is no exposure of the CEJ on any surface of any tooth in the segment	0
1	Mild gingivitis. There is an overt area of inflammation in the free gingiva which does not circumscribe the tooth	If the gingival crevice in none of the measured areas extends apically to the CEJ, the gingival score is the PDI score for that tooth (it can range from 0 to 3)		1
2	Gingivitis. Inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment			2
3				3
4		The gingival crevice extends apical to the CEJ, but not more than 3 mm	The probe extends up to 3 mm apical to the CEJ of any tooth in the segment	4
5		The gingival crevice extends apically from 3 to 6 mm in relation to the CEJ	The probe extends from 3 to 6 mm apical to the CEJ of any tooth in the segment	5
6	Gingivitis with pocket formation. The epithelial attachment has been broken and there is a pocket. There is no interference with normal masticatory function, the tooth is firm in its socket	The gingival crevice extends more than 6 mm apically to the CEJ	The probe extends 6 mm or more apical to the CEJ of any tooth in the segment	6
7				7
8	Advanced destruction with loss of masticatory function			8

risk (score 0 at baseline) revealed inflammation. Without using SBI and with visual inspection alone (GI), only 11.9% of units would have been detected as having become inflamed. Bleeding from the gently probed sulcus precedes the appearance of gingival color changes and is the leading and first clinical symptom of marginal gingivitis. The authors conclude that the higher sensitivity of SBI makes it possible to use non-hygiene periods of a reasonable length of time (2 to 3 weeks) in areas with moderate gingivitis activity. Characteristics of the SBI which differentiate it from the GI are: 1) inflammation is diagnosed only if bleeding occurs upon gentle probing of the sulcus; 2) apparently healthy gingival units without color changes or swelling are diagnosed as inflamed (score 1) if the bleeding occurs upon gentle probing; and 3) resulting SB scores are roughly 1 unit higher.

Quirynen et al. (1991) examined 5 plaque indices (Harrap index, Quigley and Hein index, Navy plaque index modified by Clemmer and Barbano, Navy plaque index modified by Hancock and Wirthlin, planimetrical plaque index) to determine which could best discriminate the difference in the rate of plaque formation associated with healthy tissue (phase I) versus with gingival inflammation (phase II) where plaque was allowed to accumulate over a 96-hour period. Significant differences between phase I and phase II plaque accumulations could not be detected using the Harrap index and the Navy index (Hancock and Wirthlin). The Navy index (Clemmer and Barbano) detected a difference at 82 hours while the Quigley and Hein index detected a phase I and phase II difference at 72 hours. The planimetrical index detected a significant difference at 36 hours. The planimetrical plaque index demonstrated the

highest discriminating power; the Harrap and Navy index (Hancock and Wirthlin) was the least discriminating. Although the planimetrical index is highly reproducible, it is time consuming and difficult to use. The Quigley and Hein or Navy index (Clemmer and Barbano) seem to be the best alternative of those indices studied if the planimetrical index is not feasible. Each index has its own advantages and disadvantages and the aim of study should determine the plaque index chosen.

Almas et al. (1991) evaluated the capacity of the community periodontal index of treatment needs (CPITN) to reflect the clinical periodontal status in 52 patients as represented by the standard clinical indices of PI, GI, papilla bleeding index (PBI), and PD. CPITN scores determined for each sextant are as follows: code 4 = pathologic pockets 6 mm or more; code 3 = pathologic pockets 4.0 to 5.5 mm; code 2 = supra-or subgingival calculus; code 1 = gingival bleeding after probing; and code 0 = none of the above signs present. PD was assessed using a pressure-sensitive probe with 0.34 mm tip and 0.25 N force. CPITN was assessed using a (WHO 612) CPITN probe with a ball end of 0.5 mm and 0.25 N force. Six sites per tooth were recorded. CPITN did not correlate with the amount of plaque present, number of sites affected in a given sextant, or GI. There was a tendency for an association between the CPITN and both the PD and the PBI. In spite of the above correlations, extreme ranges for all indices were found within a sextant regardless of the CPITN code; 25% of sites had pockets > 6 mm deep and 70% of sextants had a CPITN score of 4. This study demonstrated that the CPITN functions as an epidemiologic tool for population planning and treatment needs but is not indicated for assessment of individual treatment needs.

Kaldahl et al. (1990) evaluated gingival suppuration and supragingival plaque following 4 modalities of periodontal therapy: coronal scaling (CS) only; coronal and subgingival scaling and root planing (RP); root planing followed by modified Widman surgery (MW); root planing followed by flap reflection with osseous surgery (FO). Clinical assessments were made at baseline (exam 1), 4 weeks following oral hygiene instruction (OHI) and CS or Sc/RP (exam 2), 10 weeks following respective therapies (exam 3), and annually prior to maintenance therapy appointments (exams 4 and 5). This assessment included presence of supragingival plaque, gingival suppuration, probing depth, and clinical attachment level. Patients were seen every 3 months for 2 years for maintenance therapy. The authors found that the prevalence of supragingival plaque between the groups except for FO-treated sites showed more plaque accumulation after surgical therapy. The presence or absence of supragingival plaque at specific sites was dynamic, frequently converting to a new status between 2 examinations. Sites that were not suppurating at 1 exam but were suppurating at the subsequent exam or at both exams had a less favorable response in both probing depth and probing attachment level.

USES

There are 4 ways in which a periodontal index may be used: 1) clinical trials; 2) epidemiologic surveys; 3) evaluation of patient progress; and 4) motivation of the patient to improve hygiene. The ideal index should: 1) be simple to use; 2) require minimum time; 3) require minimum armamentarium; 4) be clear, understandable, and reproducible; 5) be amenable to statistical analysis; 6) be equally sensitive throughout its scale of variable measure; and 7) be acceptable to the patient (Barnes et al., 1986).

CONSIDERATIONS

When using indices or evaluating studies using indices, the following should be considered: 1) studies conducted with different indices should be compared only as to general findings rather than specific details; 2) an index should be selected according to its ability to best evaluate the variable (i.e., presence/absence of plaque rather than quantity of plaque); 3) index methodology should not be modified; 4) those that measure presence or absence of plaque are more applicable to motivation at assessment while those measuring quantity may be more suited for clinical trials and epidemiologic surveys; 5) full-mouth scoring should be reserved for clinical use, a single patient or small population groups, while simplified indices are more useful for epidemiologic surveys and large clinical studies; 6) examiner intra- and inter-reliability should be established prior to use and repeated throughout the study time period.

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CHAPTER 5. ETIOLOGY AND CONTRIBUTING FACTORS

Section 1. Microbiology

DEFINITIONS (Newman and Nisengard, 1988)

Cell Types

Prokaryote: A cell which lacks both a nuclear membrane and a large number of membrane-limited organelles. Prokaryotes are represented by bacteria. Size ranges from 1 to 1.5 μm wide and 2 to 6 μm long.

Eukaryote: A cell which has a nuclear membrane and large numbers of membrane-limited organelles. Algae, fungi, protozoa, plant, and animal cells are in this group.

Bacterial Structures

Capsule: An outermost layer composed of either carbohydrate or protein which provides bacteria with a means of evading certain host defense mechanisms and is involved in the expression of virulence. The capsule also provides for immunologic specificity.

Cell Wall: A rigid limiting layer which is responsible for cell shape and resistance to changes in environmental osmotic pressure. Gram's stain is directed to the cell. Gram-positive cells retain the blue crystal violet and stain blue while the Gram-negative bacteria do not retain the dye and thus stain red with safarin after alcohol treatment. The cell wall of Gram-positive bacteria is thicker (15 to 50 nm) than found in Gram-negative forms (7.5 to 10 nm). About 30% of the outer membrane of the cell wall of Gram-negative bacteria is made up of glycolipid, lipopolysaccharide (LPS).

Flagella: Organelles adapted for motility. A flagellum consists of three regions: a basal body, a hook region, and a distal filament. Unipolarly flagellated cells are termed monotrichous while cells which have flagella distributed over the entire cell surface are termed as peritrichous.

Pili: Cell surface filaments which play a role in bacterial adherence and transfer of genetic material between bacteria (the F or sex pili). Pili are straight and about one-half the length of flagellum.

Fimbriae: Pili which are specific for bacterial adherence. In several Gram-negative bacteria, virulence in a host is regulated by the presence or absence of fimbriae.

Oxygen Requirements

Aerobic: Organisms which grow very well at normal room atmosphere.

Microaerophilic: Organisms which grow best in an atmosphere of reduced oxygen.

Anaerobic: Growth in the absence of oxygen.

Facultative Anaerobic: Growth in either an aerobic or anaerobic environment.

Canophilic: Organisms which require greater concentrations of carbon dioxide.

INTRODUCTION

Robert Koch's postulates for bacterial specificity follow: 1) a bacteria should be able to be isolated from diseased tissues; 2) pure cultures of that bacteria can be obtained; 3) bacteria inoculated in experimental animals should cause the disease; 4) the bacteria should then be isolated in the diseased tissues of the animal.

Efforts to apply Koch's postulates to periodontal disease have been largely unsuccessful. Because of this, Socransky (1977) proposed alternative criteria to identify key bacteria in periodontal infections which included:

- the presence of the putative pathogen in proximity to the periodontal lesions and in high numbers compared to either the absence of the bacteria or presence in much smaller numbers in healthy subjects;
- patients infected with these periodontal pathogens often develop high levels of antibody in serum, saliva, and gingival crevicular fluid and may also develop a cell-mediated immune response to the putative pathogen;
- these bacteria can often demonstrate in vivo production of virulence factors that can be correlated with clinical histopathology;
- experimental implantation of the organism into an animal model should lead to at least some characteristics of naturally occurring periodontal disease; and
- clinical treatment that eliminates these bacteria from periodontal lesions should result in clinical improvement.

Loesche (1975) described the non-specific plaque hypothesis (NSPH) and the specific plaque hypothesis (SPH). According to the NSPH, caries and periodontal disease result from the elaboration of noxious substance by the entire plaque flora, while SPH suggests that only certain plaque cause infections because of the presence of a pathogen(s) and/or a relative increase in the levels of certain indigenous plaque organisms.

Theilade (1986) described destructive periodontitis as the result of subgingival colonization, which is favored by such ecological changes as plaque accumulation, gingivitis, and gingival exudate. These changes increase the numbers of microorganisms and alter their proportions, but no single species appears in active sites which is not also commonly present in inactive sites. The subgingival microorganisms have several virulence factors that promote colonization of

the pockets, destroy host defense mechanisms, and provoke inflammation. It appears that different combinations of indigenous bacteria, rather than just a single species, can produce the pathogenic potential necessary to cause progression from gingivitis to destructive periodontitis.

Slots (1986) studied 196 adults with advanced periodontitis and reported *Actinobacillus actinomycetemcomitans* in 50% of progressing lesions and in only 6% of non-progressing sites, and *Porphyromonas gingivalis* in 42 to 52% of progressing lesions and 14% of non-progressing ones. The median *P. gingivalis* recovery in culture-positive sites was more than 10-fold higher in progressing than in non-progressing sites. *Prevotella intermedia* was recovered from 59 to 89% of progressing lesions and from 36 to 53% of non-progressing sites. *P. intermedia* averaged 5 to 10 higher recovery in infected progressing lesions than in infected non-progressing ones. Only one progressing lesion failed to produce any of the above organisms.

REVIEWS OF PUTATIVE PERIODONTAL PATHOGENS

Spirochetes

Loesche (1981) reviewed the role of spirochetes in periodontal disease and concluded that spirochetes are at least diagnostic of periodontal status if not overly pathogenic. Spirochetes have been categorized based on cellular diameter (small, medium, or large) and by the number of axial filaments. All of the cultivable oral isolates are classified in the genus *Treponema*. Spirochetes comprise 30% of the microscopic count in ANUG with evidence of tissue invasion by the organisms reported.

Actinobacillus actinomycetemcomitans (Aa)

Zambon (1985) reviewed the relationship of Aa to periodontal disease. Aa is an anaerobic, non-motile, coccobacillus for which 3 serotypes have been described. Serotypes a and b are most common, with serotype b being elevated in localized juvenile periodontitis (LJP). Evidence for Aa involvement in the pathogenesis of LJP includes the following: 1) almost all cases of LJP harbor large numbers of Aa; 2) successful treatment of LJP has been correlated with eradication of Aa from the pocket; 3) histopathologic findings have provided evidence for Aa penetration of the crevicular tissues in LJP; 4) immunological studies have demonstrated elevated antibody titers to Aa in LJP; and 5) Aa produces a leukotoxin which may be a major virulence factor.

Bacteroides Species

Van Winkelhoff et al. (1988) reviewed the significance of black-pigmented *Bacteroides* (BPB) in oral infections. BPB are anaerobic, Gram-negative, non-motile, rod-shaped cells which produce brown or black-pigmented colonies when grown on blood-containing media. The BPB have been categorized based on fermentation. *P. gingivalis, B. asaccharolyticus*, and *B. endodontalis* are non-fermentative whereas *P. intermedia* is saccharolytic. The BPB are found in small numbers in the healthy gingival sulcus. Correlations have been found between the degree of clinical inflammation and percentage of *P. gingivalis* recovered. The other BPB are not frequently isolated from periodontal pockets although *P. intermedia* has been found in higher levels in acute necrotizing ulcerative gingivitis. Localized juvenile periodontitis lesion have been shown to harbor *P. gingivalis, P. intermedia*, and *B. endodontalis.*

Known virulence factors of BPB include pili or fimbriae, capsules and vesicles which aid in attachment. The BPB can release non-chemotactic factors which compete for chemotactic receptors and inhibit neutrophil chemotaxis. Among the BPB, *P. gingivalis* is the most virulent species. The lipopolysaccharide of *P. gingivalis* is not inhibited in human serum and can induce production of interleukin-1 by macrophages and monocytes. In short, the BPB possess properties which help explain their pathogenic nature in oral infections.

Wilton et al. (1993) found that patients with a history of destructive periodontitis have a higher level of serum opsonins to *P. gingivalis* than matched controls with no periodontal destruction.

Wolff et al. (1993) determined the distribution and prevalence of 5 bacterial pathogens in subgingival plaque, their relationship with each other, and with probing depth. Plaque was collected from 6,905 sites in 938 subjects. A bacterial concentration fluorescence immunoassay and bacterial specific monoclonal antibodies were used to determine the presence and level of *P. gingivalis* (Pg), *A. actinomycetemcomitans* (Aa), *P. intermedia* (Pi), *E. corrodens* (Ec), and *F. nucleatum* (Fn) in each plaque sample. The prevalence in subjects was lowest for Pg (32%) and highest for Ec (49%). The site-based frequency distribution of these bacterial species ranged from 10.3% for Pg to 18.7% for Ec. Pi and Ec were the bacterial combination most often found together in a subject (27.2%). While 64.0% of the sites were without any of the 5 bacterial species evaluated, 20.2% had only 1 of the 5 bacteria species evaluated. The remaining 15.8% of sites had at least 2 bacteria species present. There was a general linear association of the detection level of the bacterial species and probing depth. The odds ratios were 3.9 (Pg), 3.0 (Aa), 4.0 (Pi), 2.7 (Ec), and 2.8 (Fn) of finding high levels of these bacterial pathogens at >5 mm probing depth ($P = 0.01$) in subjects with a specific bacterium compared to molar sites in subjects without the bacteria. The observation that these 5 bacterial species frequently inhabit the subgingival environment, yet are not associated with advanced disease, suggest that a susceptible host is required, in addition to a "pathogenic bacteria," before disease progression may occur.

VIRULENCE FACTORS OF PUTATIVE PERIODONTAL PATHOGENS

General Considerations

Socransky and Haffajee (1991) critically assessed the microbial mechanisms related to the pathogenesis of periodontitis. Virulence factors were defined as the unique properties which permit a bacterial species to colonize a target organ, defend itself from the host, and cause tissue damage. Virulence factors were divided into those properties which favor bacterial adherence and colonization, and those which mediate host tissue destruction. In regard to adherence, subgingival species have adhesions which include fimbriae and cell-associated proteins. After adherence, colony growth depends on environmental factors such as temperature, pH, oxidation-reduction potential, and available nutrients. Competition among different bacterial species can favor or oppose colonization.

Host defense mechanisms which must be overcome include salivary and gingival crevicular fluid flow, mechanical displacement (e.g., tissue desquamation), specific antibodies, host products (e.g., glycoproteins that block bacterial cell binding), and cells of the immune system. Bacterial mechanisms which may mediate host tissue damage include invasion of the tissue by pathogens or diffusion of bacterial byproducts from the crevice into the gingival tissues. Identification of virulence factors requires studies to detect the appropriate bacterial strains, disclosure of possible virulence factors, confirmation in animal models, and confirmation of virulence in humans.

Endotoxin

Daly et al. (1980) reviewed the role of endotoxin or lipopolysaccharide (LPS) in periodontal disease. LPS is found in the outer membrane of Gram-negative bacteria and can be extracted for study by the hot phenol-water procedure of Westphal. Major portions of LPS include lipid A and heteropolysaccharide. Lipid A has been shown to be the toxic factor in Gram-negative sepsis and can activate the classical complement pathway. The polysaccharide component activates the alternate complement pathway. LPS is a potent B-cell mitogen and can stimulate macrophages to release collagenase and induce bone resorption in vitro. Studies have demonstrated the presence of LPS in the cementum of untreated periodontally involved teeth. The highest concentrations of LPS have been found within the loosely adherent subgingival plaque. A study in dogs demonstrated that tritiated LPS can pass through intact crevicular epithelium.

Bacterial Invasion

Listgarten (1965) described the superficial (250 μm) penetration of spirochetes in the ulcerated region of acute necrotizing ulcerative lesions. Spirochetes were found in the non-necrotic tissue before other bacteria and present in higher concentrations within the intercellular spaces of the epithelium adjacent to the ulcerated lesion, as well as within the connective tissue.

Frank and Voegel (1978) and Frank (1980) have reported the presence of filaments, rods, and coccoid organisms in the intercellular spaces of human pocket epithelium.

Using scanning electron microscopy, Saglie et al. (1982A) found bacteria invading the epithelial wall of deep periodontal pockets in 5 out of 8 cases. In one case the bacteria had traversed the basement lamina and reached the connective tissue. Bacterial morphotypes included cocci, short rods, filaments, and spirochetes.

Gillett and Johnson (1982) observed the bacterial invasion of the connective tissue in cases of juvenile periodontitis, using electron microscopy. The invading flora was described as mixed but composed mainly of Gram-negative bacteria, including cocci, rods, filaments, and spirochetes.

Saglie et al. (1982B) identified the following tissue-invading microorganisms in localized juvenile periodontitis: *A. actinomycetemcomitans, C. sputigena, Mycoplasma*, and spirochetes.

Saglie et al. (1985, 1986, and 1987) have also described the presence of bacteria in the oral epithelium in cases of advanced adult and juvenile periodontitis, and the increased numbers of Langerhans cells in relation to bacterial invasion. Pertuiset et al. (1987) found increased numbers of intragingival bacteria in recurrent sites.

Nisengard and Bascones (1987) published informational overviews from a workshop on bacterial invasion in periodontal disease. Studies on the virulence of *P. gingivalis* (Pg) suggest that invasive strains of Pg from subgingival plaque at periodontal disease sites tend to spread along tissue planes rather than grow in colonies, while non-invasive strains in dental plaque not from diseased sites exhibited a localized abscess formation. Evidence of bacterial invasion by viable *A. actinomycetemcomitans* in LJP has been identified by immunoperoxidase and immunofluorescent studies. The number of Gram-negative bacteria in connective tissue was significantly higher in sites with ongoing attachment loss than at inactive sites.

Sanavi et al. (1985) studied the morphologic features and pattern of bacterial invasion in immunosuppressed rats with ligature-induced gingival inflammation.The authors indicate that bacterial invasion, in which proliferating bacteria penetrate the tissues, should be differentiated from bacterial translocation, in which bacteria are passively transported into the tissues by mechanical means such as biopsy or histological processing.

Sandros et al. (1993) confirmed that *P. gingivalis* (Pg) is capable of adhering and entering into oral epithelial cells in vitro. The presence of coated pits in the epithelial cell surfaces suggested that internalization of Pg was associated with receptor-mediated endocytosis. Formation of outer membrane vesicles (blebs) by intracellular bacteria indicated that

internalized Pg was able to retain its viability. *E. coli* strain HB 101 neither adhered to nor invaded epithelial cells.

CLINICAL STUDIES

Technical Problems

Socransky et al. (1987) reviewed the difficulties encountered in the search for specific bacteria in periodontal disease. Conceptual problems include the complexity of the microbiota, with approximately 300 bacterial species present in plaque. If combinations of species are involved in active disease, the complexity increases dramatically. Another difficulty is the inability to accurately define the disease status at a given site at the time of sample collection. Current methods of disease detection only allow for detection of sites which have recently lost attachment over a period of time. Technical difficulties are present at the time of collection of the bacterial sample, cultivation, and characterization of the bacteria. The small diameter of the sulcus and the lack of a "gold standard" makes it impossible to determine if a representative sample has been collected. The dispersion process following collection of the sample tends to create error by selecting more robust microbes which survive the dispersion process. In the culture process, it is impossible to determine if all bacteria originally sampled grow out. Furthermore, cultured bacteria cannot always be characterized and identified, leading to further error.

In an in vitro study of bacterial sampling by absorbent paper points, Baker et al. (1991) reported that the sample obtained misrepresents the bacterial species actually present. By layering 2 bacterial species in a specific way and then reversing the order, the authors noted that the top layer of bacteria accounted for 90% or more of the colony forming units (CFU). When the bacteria were mixed, equal numbers of CFU were detected. From a practical standpoint, it is likely that few of the bacteria from the apical portion of the pocket are detected, leading to error in the bacteriologic assay.

Associated Studies

Genco et al. (1988) reviewed the source of bacteria in periodontal infections. Indigenous organisms are constant members of the microbiota while exogenous organisms are transient. Opportunistic organisms overgrow as a result of environmental changes or alteration of the host resistance.

Technical advances in anaerobic culturing have allowed the identification of specific bacteria associated with health and periodontal disease. Healthy sites harbored a sparse plaque, mostly Gram-positive cocci like *Actinomyces* and *Streptococci.* Gingivitis harbored increased *Actinomyces* and reduced *Streptococci. A. odontolyticus, A. naeslundii, Fusobacterium nucleatum, Lactobacillus, Veillonella,* and *Treponema* species were the mostly likely etiologic agents in experimental gingivitis. It has been noted that increased *P. intermedia* levels were observed in experimental gingivitis. Severe adult periodontitis was associated with *P. gingivalis.* Other organisms prominent in periodontitis include *F. nucleatum* and *Eubacterium timidium.* In localized juvenile periodontitis, *A. actinomycetemcomitans* is predominant. Acute necrotizing ulcerative gingivitis harbors Pi and intermediate spirochetes. Genco et al. indicate that indigenous organisms play a key role in gingivitis while exogenous organisms seem to be implicated in periodontitis. It must be remembered that if the causative agents are indigenous, they will be difficult to eradicate. In contrast, exogenous pathogens may be more easily eliminated.

In the experimental gingivitis study by Löe et al. (1965), 12 subjects ceased all oral hygiene efforts and were monitored clinically and microbiologically. They found that gingivitis began in 10 to 21 days and resolved within 1 week of renewed oral hygiene efforts. Three phases of plaque morphogenesis were described based on time (days).

Phase 1 (day 1 to 2) was characterized by a sparse flora to a dense mat of Gram-positive cocci and short rods; desquamated epithelial cells; and small accumulations of PMNs along the gingival margin.

During phase 2 (days 2 to 4), filamentous forms and rods increased, although cocci were still present in large numbers. The PMN concentration continued to increase.

Phase 3 (days 6 to 10) was associated with a gradual shift to vibrios and spirochetes. Gram-positive cocci and short rods still constituted 45 to 60% of flora, and PMN concentrations were great.

When the oral hygiene was resumed and healthy gingival conditions re-established, the gingival flora returned to one of predominantly Gram-positive cocci and short rods. No vibrios or spirochetes were observed in health.

Savitt and Socransky (1984) attempted to differentiate the composition of bacterial plaque in health, gingivitis, or adult and juvenile periodontitis. Thirty-six (36) patients were sampled by syringe and curet for culture (with selective media) and darkfield microscopy, respectively. No attempt was made to sample sites with active attachment loss. Generally, Gram-negative and motile forms were less common and coccal forms were elevated at healthy sites. Black-pigmented *Bacteroides* species were detected in 20% of the healthy, 42% of gingivitis, 61% of adult periodontitis, and 73% of juvenile periodontitis sites.

Tanner et al. (1984) studied the microbiota of sites which had lost crestal alveolar bone. In 3 subjects, the percentage of small spirochetes was positively related to sites with recent attachment loss while *P. gingivalis,* "fusiform" *Bacteroides* (*B. forsythus*), medium spirochetes, and curved motile rods were also isolated in higher proportions in sites with recent bone loss. *Streptococcus intermedius* and *Fusobacterium nucleatum* made up a higher proportion of the microbiota in inactive sites. Cluster 1 (Pg, Fn, and "fusiform" *Bacteroides*) corresponded to sites with recent bone loss. Cluster 2 (Pg, Fn, and Gram-positive rod type A) was associated with inactive sites. Gingival swelling and bleed-

TABLE 1. SUMMARY OF PUTATIVE PERIODONTAL PATHOGENS

	Characteristics					Socransky's Alternative Criteria to Identify Pathogens				
Species	Cell Morph	Gram Stain	Motility	Oxygen Requirement	Colony Morph	Periodontal Disease Association	Presence in Disease	Increased Ab Titer	Virulence Factors	Implantation Studies
Actinobacillus actinomycetemcomitans	Coccobacillus	negative	nonmotile	Capnophilic (anaerobic)	star-shaped internal morphology	localized juvenile periodontitis	LJP lesion high in Aa	in LJP, high Ab titer to Aa	leukotoxin tissue invasion	
Prevotella intermedia	Bacillus	negative	nonmotile	anaerobic	brown black	acute necrotizing ulcerative gingivitis	*Pi* cultured ANUG		pili and fimbriae capsule LPS	
Porphyromonas gingivalis	Bacillus	negative	nonmotile	anaerobic	brown black	adult periodontitis	*Pg* from advanced sites		collagenase LPS	
Campylobacter recta	helical to straight rod 2 to 6 μm by .5 to 1 μm	negative	motile (single flagella)	anaerobic	yellow to grey pitting of surface	adult periodontitis				
Spirochetes	sprilla 5 to 20 μm by 0.1 to 0.5 μm	negative	motile	strict anaerobes	no colonies "just haze"	acute necrotizing ulcerative gingivitis			tissue invasion	
Eikenella corrodens	Bacillus	negative	trans locating twitching	facultative anaerobe	pitting, corrosion of agar surface	adult periodontitis				
Fusobacterium nucleatum	Bacillus long, tapered	negative	nonmotile	strict anaerobe		adult periodontitis				
Bacteroides forsythus	Bacillus tapered ends	negative	nonmotile	anaerobe	black brown	adult periodontitis				

Sources: Loesche and Laughon (1981); Zambon (1985 and 1990); Theilade (1986); Genco et al. (1988); Van Winkelhoff et al. (1988); Chen and Wilson (1992).

ing on probing distinguished the sites with recent bone loss from the inactive sites. The clusters formed were independent of the depth of the sample within the pocket. Significantly, no single microorganism was found to predict recent bone loss.

Dzink et al. (1985) compared the cultivable Gram-negative species associated with active sites (as determined by the tolerance method which looked for differences between the means of repeated attachment loss measurements greater than 2 mm) versus inactive (control) sites in the same subject. They determined that higher proportions of Gram-negative rods were present at active sites. *Wolinella recta, P. intermedia,* "fusiform" *Bacteroides,* and *A. actinomycetemcomitans* were the 4 species which were elevated only at the active sites.

Bowden (1990) reviewed the microflora associated with root caries. Early research in the 1970s using animal models emphasized the role of *Actinomyces viscosus* and *Actinomyces naeslundii.* More recently, studies have implicated *Streptococcus mutans* and *Lactobacillus* in the prediction of root caries risk. Table 1 is a summary of the characteristics of the putative periodontal pathogens and selected studies which fulfill Socransky's alternative criteria for bacterial specificity in periodontal disease.

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Section 2. Accretions

DEFINITIONS

Plaque: An organized mass, consisting mainly of microorganisms, that adheres to teeth, prostheses, and oral surfaces and is found in the gingival crevice and periodontal pockets. In addition to microorganisms, plaque consists of an organic, polysaccharide-protein matrix consisting of bacterial by-products such as enzymes, food debris, des-

quamated cells, and inorganic components such as calcium and phosphate.

Pellicle: Tooth- or mucosal-adherent salivary proteins.

Calculus: A hard concretion that forms on teeth or dental prostheses through calcification of bacterial plaque.

Subgingival (Seruminal) Calculus: Calculus formed apical to the gingival margin; often brown or black, hard and tenacious.

Supragingival (Salivary) Calculus: Calculus formed coronal to the gingival margin; usually formed more recently than subgingival calculus.

BASIC CHARACTERISTICS OF PLAQUE

Plaque consists of approximately 80% water and 20% solid material, the latter consisting (dry weight) of 35% cellular (primarily bacteria) and 65% extracellular constituency (e.g., polysaccharides). These polysaccharides include dextrans (95%) that facilitate adhesion, and levans (approximately 5%) which may serve as a hydrolyzable energy source.

An acquired pellicle, derived from salivary glycoproteins, provides a scaffold for progressive and dynamic plaque formation (Genco et al., 1990). Van Houte (1982A, 1982B) notes that Van der Waals forces, glycocalyx, and lectin-like receptors (carbohydrate binding proteins) help mediate bacterial attachments during plaque development. Such adherence also depends on attachment forces, numbers of bacteria, host flora and respective oxidation-reduction potentials, and aforementioned salivary (or gingival fluid) proteins. Lipoteichoic acid from Gram-positive bacteria may aid plaque accumulation by increasing the overall negative charge. The presence of fissures, roughened areas, and gingival areas sheltered from oral forces also influence this process. The author noted that the oral cavity is sterile at birth. The initial sources of floral transmission include the mother and other immediate environmental contacts. Anaerobic bacteria are not established until the teeth erupt, with *S. salivarius* and *A. naeslundii* predominating to this point. Following tooth eruption, *S. sanguis, S. mutans, A. viscosus*, and lactobacilli are present. Black-pigmented *Bacteroides* (BPB) and spirochetes are not consistently isolated until late adolescence or adulthood. Selected BPB are purportedly facilitated by selected nutritional factors (hemin, vitamin K, and estrogen), altered redoxpotential and attachment to certain Gram-positive bacteria (e.g., *Actinomyces*). As subgingival niches develop and bacterial successions are influenced by plaque thickness, a more anaerobic flora develops. Subgingival growth primarily represents an extension from the supragingival plaque. Its formation is facilitated by the presence of gingival crevicular fluid.

PLAQUE MORPHOLOGY

Listgarten (1976) examined the structure of the microbial flora on natural tooth surfaces in periodontal health and disease (53 teeth and suggested 5 categories of disease based on gingival inflammation, probing depth, and amount/pattern of radiographic bone loss). He reported that:

1. Normal samples presented a thin layer of adherent bacterial cells—predominantly Gram-positive cocci (GP), lower numbers of Gram-negative organisms (GN), and no spirochetes (S) or flagellated bacteria (FB)—and ranged from a few cells to 60 μm thick.

2. Gingivitis samples consisted of densely packed cells 0.4 mm thicker than normal. "Corncob" formations (central filament, *Bacterionema matruchotii*, covered by streptococci) were present in supragingival plaque. Flagellated bacteria and spirochetes were found in apical plaque sites, covering underlying bacteria in some areas.

3. In periodontitis, dense supragingival plaque mimicked that of gingivitis. Increasing numbers of flagellated bacteria and fewer large filaments comprised a transitional zone between the supragingival and largely motile subgingival plaque. Distinct features of the subgingival plaque included thin layers of smaller cells adhering to the root which contained "bristle-brush" and "test-tube brush" formations (Gram-negative filament, *Leptotrichia buccalis*, surrounded by flagellated rods and short filaments aligned perpendicular to axial surface). Tissue associated flora included spirochetes, FB, cocci, and GN rods.

4. The flora associated with juvenile periodontitis was sparse and simple. Small clumps of GN cocci and thin layers of GN filamentous bacteria were present.

5. Post-juvenile periodontitis was characterized by a flora similar to periodontitis.

Vrahopoulos et al. (1992) examined the ultrastructural morphology of subgingival plaque from patients with chronic adult periodontitis. In the plaque just coronal to the apical plaque border, they described 3 to 4 distinct morphologic layers. The plaque nearest the cementum was densely-packed Gram-positive coccoid cells aligned perpendicular to the root surfaces. The superficial layer (nearest the tissue) was mainly Gram-negative rods and cocci. The two layers between the cemental and superficial layers consisted of a mixture of Gram-positive and Gram-negative bacteria with spirochetes randomly distributed among the other forms. In the superficial layer, "corncob," "rosettes," and "test-tube brush" configurations were identified. They also noted that the most apical organisms were almost always lysed, and bacterial cell-ghosts extended apically for a variable distance from the actual apical plaque border into the so-called plaque-free zone.

Corbet and Davies (1993) reported that levels of supragingival plaque and calculus have been related to progressive periodontal disease. They also noted that control of supragingival plaque in conjunction with professional subgingival tooth cleaning forms the basis for the management of periodontal disease. However, the contribution of supragingival plaque control alone in managing progressive periodontal disease is not clear. There are studies which address, directly or indirectly, the contribution of supragin-

gival plaque control alone in the management of progressive periodontal disease. The effects of supragingival plaque control alone have been evaluated clinically, histologically, and microbiologically. Collectively, these effects may not be as marked as when professional subgingival tooth cleaning is also performed. However, given the patterns of periodontal disease found in adults in many communities, these studies can form the basis for advocating high individual levels of supragingival plaque control as a community measure in the management of periodontal disease. Further long-term investigations into this approach appear warranted.

CALCULUS

Composition

The inorganic constituents of calculus include calcium, phosphorus, carbonate, sodium, magnesium, potassium, and trace elements (fluoride, zinc) (Genco et al., 1990). The major crystalline form in mature calculus is hydroxyapatite; lesser amounts of octacalcium phosphate ($Ca_8[HPO_4]_4$), whitlockite (a magnesium containing tricalcium phosphate), and brushite are present. Subgingival calculus contains greater concentrations of calcium, magnesium, and fluoride compared to supragingival calculus (relates to greater concentrations of these ions in gingival crevicular fluid [GCF] versus saliva). In maturing calculus (< 3 months), brushite may account for 50% of the crystalline forms. Developmentally, crystalline forms appear in the following order: brushite, octacalcium phosphate, whitlockite, and hydroxyapatite.

Organic constituents account for approximately 15 to 20% of the dry weight of mature supragingival plaque (protein, 50 to 60%; carbohydrate, 12 to 20%; lipids, 10 to 15%).

Formation

Calculus formation may begin in as little as 4 to 8 hours and calcifying plaques may become 50% mineralized in 48 hours (Carranza, 1979). Unmineralized plaque is always present on the mineralized surface. The genesis of calculus formation parallels that of plaque, as previously described. The principal mineral source for supragingival and subgingival calculus respectively is saliva and GCF (Genco et al., 1990).

Genco et al. (1990) describes the formation of calculus and the presentation of incremental lines relating to calculus formation. The lines are oriented horizontally in supragingival calculus and vertically in subgingival calculus. The stratifications suggest that calculus deposits increase by apposition of new layers of calcifying plaque. Anerud et al. (1991) longitudinally (15 years) examined calculus formation in Sri Lankan tea laborers who had no professional dental care. They reported that subgingival calculus formation began 6 to 8 years after eruption, continuing to approximately 30 years of age, at which time it leveled off. Teeth with calculus showed a significantly higher rate of attachment loss than teeth without calculus. Subgingival calculus was found to form first on the mandibular incisors and maxillary molars, suggesting that the initial deposits in a supragingival location might have created conditions facilitating subgingival calculus formation.

Mineralization

Genco et al. (1990) reviews 4 theories of mineralization of calculus: 1) The Booster mechanism in which high pH, calcium, and phosphorus concentrations allow precipitation of calcium phosphate. Loss of CO_2, NH_3 production, acid/alkaline phosphatase activity, and calcium liberation are influencing factors. 2) The epitaxic concept suggests that calcium and phosphorus levels are inadequate for spontaneous precipitation but great enough to support growth of hydroxyapatite crystals about "nuclei/seed" sites. Crystal growth proceeds in the presence of metastable ionic solutions. "Nucleators" may include Ca-phospholipid-PO_4 complexes, collagen molecules, and proteoglycans. This theory is widely held. 3) The inhibition theory is based on alteration of the inhibition mechanism which maintains certain sites as noncalcifying. Calcification occurs when this mechanism is disrupted by a number of possible agents (e.g., pyrophosphate degradation by alkaline phosphatase, yielding PO_4). 4) The transformation theory suggests that amorphous, noncrystalline deposits (and brushite) may be transformed to octacalcium PO_4 and hydroxyapatite. Pyrophosphate may play a role in this process.

Attachment

Zander (1953) described 4 modes of calculus attachment in 50 teeth using light microscopy (LM): 1) attachment by secondary cuticle; 2) microscopic irregularities in the cemental surface; 3) microbial penetration of cementum; and 4) cemental resorption bays. Subsequently, calculus attachment in areas of cemental separation (Moskow, 1969) and by direct contact of calcified matrix to tooth structure (Selvig, 1970) was reported.

Using light (LM), scanning electron (SEM), and transmission electron (TEM) microscopy, Canis et al. (1979) found no evidence of direct extension of microorganisms into cementum, and attributed Zander's observation by LM to artifact. Attachment in mechanical undercuts (e.g., resorption bays, cemental tears, and areas of root gouging/caries) was fairly common. Intimate adaptation of calculus to cementum mediated by an indistinguishable interface ("calculocementum") was frequently observed.

Morphology

Based on LM and TEM observations, Friskopp (1983) described morphological characteristics of supragingival and subgingival calculus. Supragingival calculus was heterogenous, presenting filamentous microorganisms, small needle-shaped crystals, and large ribbon-like crystals (islets of intermicrobial calcification). Distribution of the small

needle-like crystals (100 nm long) occurring near the inner bacterial membrane appeared influenced by microorganisms. Bundles/rosettes of large crystals (1 nm to 50 nm long) were associated primarily with the small crystals (versus microorganisms). Subgingival calculus was homogeneous at the LM level, containing microorganisms (cocci, filaments, and rods), but no calcified material. Only small crystals (< 50 nm) were present in the calculus itself, initially occurring within the microorganisms; a few noncalcified microorganisms were observed. The bacterial cell wall was the last structure calcified in supragingival and subgingival calculus.

CALCULUS AND PERIODONTAL DISEASE

Calculus and Inflammation

Mandel (1986) provided a detailed review of calculus and periodontal disease. The author discussed epidemiologic, clinical morphological, and experimental aspects. Salient points of this review are presented below, noting the original authors and publication dates.

Schroeder (1969) considered plaque the cause of inflammation and calculus the result of inflammation which in turn promotes chronic inflammation. While studying 200 dental students and 200 dental clinic patients, Alexander (1971) reported a closer match between the distributions of gingival indices and plaque than between gingival indices and calculus. Buckley (1980) examined 300 teenagers and reported a higher correlation between gingival indices and plaque than between gingival indices and calculus. In a national health examination survey, Douglas (1983) found that 51.4% of subjects were free of gingival disease in 1971-1974 compared to 26.1% in 1960-1962; the debris scores had improved significantly but not the calculus scores.

Calculus Toxicity

Supragingival calculus is completely permeated by dyes in 24 hours (Baumhammers and Rohrbaugh, 1970). It has a spongy appearance and contains empty spaces (Lustman, 1976). Calculus and cementum from periodontally diseased teeth have induced bone resorption in vitro (Patters, 1982).

Calculus and Attachment Loss

Lennon and Clerehugh (1984) determined that calculus was the best predictor of attachment loss epidemiologically. Tagge (1975) noted that removal of calculus resulted in statistically greater improvement in probing depths and soft tissue response than oral hygiene alone (22 patients). Chawla (1975) observed that scaling and oral hygiene correlated directly to improvement in clinical health; plaque control alone failed to result in such improvement (1,500 patients). Hughes and Caffesse (1978) found that plaque control had minimal influence on attachment levels and that root instrumentation was the primary contributor to positive gingival changes (15 patients). Cercek (1983) compared brushing and flossing in 7 patients to removal of only subgingival plaque or removal of subgingival plaque and calculus. He observed that removal of subgingival plaque only resulted in little reduction in bleeding scores or improvement in probing attachment levels (neither did supragingival plaque removal by brushing/flossing). Only the removal of both subgingival plaque and calculus resulted in clinical improvement. Ramfjord (1982) found that with professional tooth cleaning every 3 months, the level of oral hygiene was not critical for maintenance. (End of Mandel review.)

Calculus and Defect Depth

Richardson et al. (1990) evaluated the relationship between apical calculus position and defect depth and morphology in 260 intrabony defects in 39 patients. Using loops and fiberoptic lighting, the most apical level of calculus was grooved with a bur. Histologic evaluation of en bloc tissue specimens failed to reveal calculus apical to the groove in any specimen. Mean distance of apical calculus to defect base was significantly greater for 3-wall defects than for 1- and 2-wall defects and increased as intrabony defect depth increased. In most cases, the apical extent of calculus was found at mid-depth of intrabony defects. Reasons for absence of calculus at the base of the defect include: 1) the apical aspect of the defect is the area of most recent tissue destruction and organisms had not yet calcified; 2) the most apical portion of defect is not pathologically exposed but is the zone of cementum and Sharpey's fiber attachment; 3) defects may have been produced by traumatic occlusion, resulting in loss of bone height and volume but not attachment loss.

Calculus and Healing

In order to achieve faster healing post-mucoperiosteal flap reflection, instrumentation of the root surface is required. Fujikawa et al. (1988) showed that it took dogs 120 days in non-instrumented areas (where calculus was not disturbed) to achieve the same healing that occurred sooner (in about 30 days) on instrumented root surfaces. Calculus retained after instrumentation is associated with increased inflammatory infiltrate in the connective tissue.

Interruption of Calculus Formation

Suomi (1974) observed that dentifrices containing calculus-reducing agents had no significant effect on gingivitis.

Zacherl et al. (1985) and Lobene (1986), each using slightly different formulations of a dentifrice containing 3.3% pyrophosphate and 0.24% sodium fluoride, were able to show reductions of 37% and 44% in supragingival calculus in 6 months and 3 months respectively. The pyrophosphate is believed to inhibit calcification by preventing the initial calcification nucleus from growing, possibly by "poisoning" the growth centers of the crystal.

Rosling and Lindhe (1987) compared the relative efficacy of calculus inhibition of 2 commercial tartar control

toothpastes, (CR) and (CO), to a placebo dentifrice in 161 adults with history of supragingival calculus buildup. At baseline and after 3 months (brushing twice a day), there was no significant difference in calculus formation among the 3 groups. At 6 months, calculus in CR and CO relative to placebo was reduced by 9% and 42.2%, respectively. At both 3- and 6-month exams, the CO group demonstrated significantly more calculus-free surfaces than the placebo group. CO provided a statistically significant reduction in supragingival calculus after 6 months when compared to CR or placebo. CO and CR both contain 0.243% sodium fluoride and 3.3% soluble pyrophosphate in a silica abrasive system. The soluble pyrophosphate in CO is from a mixture of 1.5% tetrasodium and 4.5% tetrapotassium pyrophosphates whereas that in CR is from tetrasodium and disodium dihydrogen pyrophosphates.

Radiographic Detection

Buchanan et al. (1987) quantified the sensitivity and specificity associated with radiographic calculus detection on proximal surfaces of teeth with severe periodontitis. Periapical radiographs were taken on 18 patients using a paralleling device, with standardized kVp, mA, exposure time, and processing. Extracted teeth were stained with methylene blue and the percentage of proximal root surface area occupied by calculus was calculated. Calculus detection on radiographic surfaces as compared with visual assessment of the same tooth surface demonstrated a sensitivity of 43.8% (only 43.8% of surfaces with visual calculus were detected radiographically) and specificity of 92.5% (92.5% of surfaces visually free of calculus showed no calculus radiographically). Sensitivity was unaffected by tooth type, but specificity decreased from anterior teeth to posterior teeth. In the majority of surfaces with thin or moderate calculus deposits, radiographic evaluation was not effective as a diagnostic method. Conventional oral radiography predicted calculus on less than half of the proximal surfaces where calculus was present visually.

Subgingival calculus appears to contribute significantly to the chronicity and progression of periodontal disease. Calculus may be analogous to a ligature (Friskopp, 1984); i.e., it may extend the sphere of influence of microbial plaque's bone resorptive activity, limit self-cleansing mechanisms, and promote new plaque formation.

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Section 3. Immunology

DEFINITIONS

Antibody: A class of serum proteins that are induced following interaction with an antigen. They bind specifically to the antigen that induced their formation.

Antigen: Any foreign material that is specifically bound by antibody.

Cell-mediated immunity: An immune reaction mediated by T-cells (activated lymphocytes release biologic response modifiers [lymphokines] on exposure to antigen).

Chemotaxis: The migration of cells along a concentration gradient of an attractant.

Complement: A group of serum proteins involved in the control of inflammation, the activation of phagocytes, and the lytic attack on cell membranes. The system can be activated by interaction with antigen-antibody complexes or by bacterial substances.

Immunoglobulin: A glycoprotein composed of "heavy" and "light" peptide chains; functions as antibody

in serum and secretions. There are five major classes abbreviated as IgG, IgA, IgM, IgD, and IgE, each with specialized functions. The classes are described below:

IgG: Most abundant immunoglobulin (Ig) of internal body fluids, particularly extravascular, where it combats microorganisms and their toxins. Fixes complement through the classical pathway. Crosses the placental barrier to provide defense against infection during babies' first weeks of life. Binds to macrophages and polymorphonuclear cells.

IgA: Major Ig in sero-mucous secretions where it defends external body surfaces. Aggregated IgA binds to polymorphonuclear leukocytes and can also activate the alternative complement pathway.

IgM: Very effective agglutinator; produced early in the immune response. Largely confined to the bloodstream where it plays an important role against bacteremia. Fixes complement through the classical pathway.

IgD: Present primarily on the on surface of B lymphocytes.

IgE: Contact with antigen leads to degranulation of mast cells with release of vasoactive amines. Responsible for symptoms of atopic allergy. The main physiologic role of IgE is protection of external mucosal surfaces of the body where an acute inflammatory reaction is triggered, thereby recruiting plasma factors and effector cells.

Lymphocyte: A spherical cell of the lymphoid series (7 to 20 μm in diameter) with a large, round nucleus and scant cytoplasm. It is the principal cell involved in the immune response. There are two major populations, T- (or thymus-dependent) lymphocytes and B- (or bursa-equivalent) lymphocytes. B-lymphocytes differentiate and become antibody-producing plasma cells, while T-lymphocytes are involved in a variety of cell-mediated immune reactions.

Lymphokine: Soluble factors released from lymphocytes that transmit signals for growth and differentiations of various cell types.

Macrophage: A large phagocytic cell of the monocyte series. Important as an antigen-presenting cell and as a producer of certain cytokines such as interleukin-1 and gamma interferon.

Mitogen: A substance that causes DNA synthesis, blast transformation, and mitosis in lymphocytes.

Neutrophil: The predominant polymorphonuclear leukocyte comprising up to 70% of the peripheral white blood cells that is important in infection and injury repair. May have impaired function in some forms of early onset periodontitis.

Opsonin: A substance (e.g., antibody, complement) capable of enhancing phagocytosis.

INTRODUCTION

Schonfeld and Checchi (1985) further defined complement as a multicomponent system which has several important functions. It can be activated by the complex or "classical" pathway (initiated by antigen bound to IgG or IgM which in turn is bound to the first component of complement) and the properdin or "alternate" pathway (activated by bacterial endotoxins and certain other substances). An activated complement complex may lyse cells on which the antigen-antibody complexes are found, stimulate the release of histamine and thromboxanes from mast cells, increase vascular permeability and smooth muscle contraction, effect chemotaxis for PMNs and macrophages, and account for the symptoms of immediate hypersensitivity. The anaphylatoxic and chemotactic factors of activated complement (C3a and C5a) are produced in both pathways.

Immunological mechanisms which can protect the host against infectious organisms may also cause tissue destruction. These include: 1) type I anaphylaxis is mediated by IgE bound to mast cells or basophils, which may degranulate, resulting in possible anaphylactic reaction; 2) type II antibody-dependent cytotoxic (IgG or IgM mediated) autoimmunity; 3) type III immune complex mediated responses which activate complement; and 4) type IV cell mediated (delayed-type) responses which are slower than type I, II, or III reactions and involve activation of special T cells which release lymphokines or perform effector functions.

SELECTIVE IMMUNE SYSTEM RESPONSE

Specific lymphocytes capable of reacting to specific antigens undergo division to produce amplification of the cell clones which are reactive to respective antigens. An exception to this is polyclonal B cell activation in which certain substances activate B cells without regard to their antigenic specificity. Activated cells either differentiate into effector (or plasma) cells or become "memory" cells which can mount a much stronger second response to an antigen.

Host tissue damage can result from immune response when oral bacteria and their products gain access to the gingival connective tissue and react with specific T and B cells which release lymphokines. These can cause death of gingival fibroblasts, enhance osteoclastic bone resorption, and activate and attract PMNs and macrophages. The PMNs and macrophages, when activated, can contribute to tissue destruction through release of enzymes. Additionally, immunoglobulins produced by plasma cells can activate the complement system with potential destructive effects. When components of the immune system are activated, the ensuing inflammatory response is accompanied by some degree of tissue destruction.

Genco and Slots (1984) reviewed the host immune response in periodontal disease. The immune systems responding to bacterial infections include the mucosal or secretory immune system, neutrophil-antibody-complement system, lymphocyte-macrophage system, and immunoregulatory systems. While antibodies have the potential to inhibit mucosal bacterial adherence, their role in preventing coloni-

zation of periodontopathic organisms is unknown. This is also the case of the immune response's role relative to temperature, pH, oxidation-reduction potential, nutrition, bacterial antagonisms, and synergisms. Microbial killing may result from complement-dependent cytolysis/complement-antibody mediated cytolysis and phagocytes functioning independently or in combination with opsonic factors such as antibody and complement. Phagocytes can increase bactericidal activity through oxygen reduction, excitation mechanisms, or oxgyen independent mechanisms such as lysosome, lactoferrin, and azurophilic granules.

Serum antibody studies have shown correlations between predominant organisms in several forms of periodontal disease and antibody titers. These include *Porphymonas gingivalis* (Pg) and severe adult periodontitis, *Actinobacillus actinomycetemcomitans* (Aa), and localized juvenile periodontitis (LJP), and intermediate-sized spirochetes, and *Prevotella intermedia* (Pi) with acute necrotizing ulcerative gingivitis.

Complement plays an extensive role in periodontal disease through its effects on phagocytosis, chemotaxis, alteration of vascular permeability, killing of cells, lymphokine production, antibody synthesis, lysosomal enzyme release, and bone resorption. Complement proteins (or cleavage products) bind to receptors on neutrophils, platelets, mast cells, macrophages, erythrocytes, and specific target cells. Its activation in periodontal disease originates locally via the alternative pathway through C3 and Factor B cleavage in adult periodontitis and LJP and via the classical pathway in LJP as evidenced by the marked depression of C4 levels.

Depressed neutrophil chemotaxis and phagocytosis have been demonstrated in LJP. Approximately 75% of classic LJP patients suffer from a peripheral blood neutrophil chemotactic abnormality due to a reduced chemotactic gradient response. This is due to a reduced number of cell surface receptors for the synthetic peptide N-formyl-1-leucyl-1-phenylalanine (FMLP) and may be hereditary. LJP patients demonstrate antibodies to Aa and Aa antigen in cells. The containment of LJP to localized areas may be due to host antibodies that opsonize Aa leading to effective ingestion and killing by phagocytes.

Hypersensitivity to periodontopathic microbial antigens has been demonstrated using lymphokine production or the blastogenesis assay as in vitro indicators of cellular immunity. Three patterns of reactivity of peripheral blood monocytes following stimulation by oral organisms have been suggested: 1) specific or non-specific stimulation of lymphocyte blastogenesis by mitogenic or polyclonal activation of lymphocytes, regardless of disease activity level; 2) peripheral blood lymphoproliferative response, in a few individuals with non-categorized disease status; and 3) stimulation of more positive responses by Gram-negative anaerobic organisms (e.g., *P. gingivalis* and *Treponema denticola*) in patients with destructive disease as compared to subjects with gingivitis or healthy subjects.

The severity of periodontal disease may be a consequence of B-cell hyperactivity. The lymphoproliferative response results in a production of lymphokines, such as alpha-lymphotoxin and osteoclast activating factor, which may in turn produce tissue destruction. Lymphocytes can induce macrophage activation to produce tissue destructive factors such as collagenase and oxidizing agents. It has been observed that patients with deficient lymphocyte functions have less gingival disease than immunocompetent patients, but patients with reduced neutrophil numbers or function are very susceptible to disease.

Lymphocytes and macrophages also produce factors which recruit fibroblasts to areas of inflammation and lead to their proliferation. The latter results in increased collagen production for repair.

POLYMORPHONUCLEAR LEUKOCYTES (PMNS) AND PERIODONTAL DISEASE

Miller and Lamster (1984) reviewed the role of PMNs in periodontal diseases, comparing this relationship to a double-edged sword. On one edge, the primary role of PMNs is defensive, playing an essential role in containing gingival bacteria and their products. On the opposite edge, PMNs release extracellular lysosomal enzymes which may contribute to localized tissue destruction. PMN impairment or absence accompanying diseases such as Chédiak-Higashi syndrome, agranulocytosis, cyclic neutropenia, and diabetes mellitus have been associated with severe periodontal destruction. The presence of PMNs in the gingival crevicular fluid may have a future role as a clinical diagnostic test in the determination of disease activity pending development of an effective and predictive assay.

Miyasaki (1991) reviewed the role of the neutrophil in controlling periodontal bacteria. Neutrophils kill bacteria or influence bacterial growth by oxidative (cytosol, membrane granules) or non-oxidative mechanisms (azurophil granules, defensins). Delivery of antimicrobial substances by the neutrophil can occur by 4 mechanisms: 1) delivery of oxygen metabolites or so-called "respiratory burst" (this occurs as phagocytes consume and transfer dioxygen resulting in superoxide and hydrogen peroxide production); 2) secretion which involves release of cytoplasmic granule contents as a result of fusion of granules with the plasma membrane; 3) phagocytosis by engulfment of particles within a membrane bound structure called a phagosome; phagosomes fuse with lysosomes to form phagolysosomes, which effectively deliver high concentrations of granule contents; and 4) death as a result of injury or failed phagocytosis (cytolysis) or as a result of programmed cell death (apoptosis). Miyasaki also described the "order" of components of the host defenses in response to a bacterial challenge. Initially protection is afforded by serum complement; activation of complement produces an influx of neutrophils. As the reaction becomes more chronic, monocytes/ macrophages arrive. If the antigen is not destroyed at this point through

phagocytosis, the antigen is presented to T-lymphocytes which in turn activate B-lymphocytes resulting in maturation of immunoglobulin-producing plasma cells. All of these responses are an attempt of the body to contain the bacterial antigen.

Newman and Addison (1982) compared the functional activity of gingival crevicular PMNs in LJP patients with healthy controls. They observed that although 80% of the gingival crevicular PMNs were viable in both patient groups, 95% of those in LJP patients exhibited altered morphology and none phagocytized the test organism, *Candida guillermondiae*. Respective percentages for healthy controls were 10% and 25%. Seventy-five percent (75%) of LJP PMNs (versus 50% of controls) were able to phagocytize Gram-negative and Gram-positive organisms in vivo, but did not effectively lyse the cells once engulfed. The authors concluded that LJP-affected gingival crevicular PMNs showed reduced phagocytic function compared to normal or periodontitis-affected PMNs. The decreased functional behavior of these cells may relate to their life stage (i.e., functional end-stage) and not accurately reflect their original capabilities.

Clark et al. (1977) also assessed chemotactic activity levels of PMNs from patients with LJP, adult periodontitis, advanced generalized periodontitis and healthy controls. They reported that PMNs of 7 out of 9 LJP patients exhibited an impaired chemotactic level, approaching 62.3% of that noted in the normal controls. The serum from 5 out of 9 LJP patients inhibited chemotaxis by 68.0 to 80.2% versus 31.6% for normal controls. Three of 4 advanced generalized patients also demonstrated reduced cellular chemotactic responses and serum chemotaxis inhibition. None of the adult periodontitis patients had reduced PMN chemotactic levels and only 1 out of 5 had serum chemotaxis inhibition. The authors concluded that in LJP, PMNs may contribute to host destruction by failing to protect the host adequately and by releasing their toxic lysosomal or metabolic products into the adjacent tissues.

DeNardin and DeLuca (1990) utilized monoclonal antibodies (MOABs) specific for FMLP chemotactic peptide receptors, noting that 5 out of 7 reaction sites demonstrated reduced binding against PMNs from chemotaxis defective LJP donors. The decrease was possibly due to qualitative differences in the epitope, epitope masking, or reduced expression of FMLP receptors on the PMN surface.

Using beagle dogs, Wennstrom and Heijl (1980) found that extracts of *Actinomyces viscosus* derived from bacterial plaque (associated with gingivitis) exerted a greater chemotactic affect on leukocytes than extracts of *Capnocytophaga ochracea* (associated with periodontitis). Both were greater than saline control units. Complement activation appeared to be the dominant source of chemotaxis in this study.

CELL MEDIATED IMMUNITY

Seymour (1991) and Seymour and Powell (1979) reviewed the immune response as it relates to the pathogenesis of periodontal disease and hypothesized that periodontal disease in adults occurs in 2 forms, a stable lesion and a progressive lesion. Advanced (progressive) forms of periodontal disease are dominated by a B-lymphocyte/plasma cell lesion, while early and stable forms are dominated by T-lymphocytes. Based on these observations, the goal of therapy should be to convert B cell to T cell lesions. Depressed T-helper:T-suppressor ratios have been demonstrated in gingivitis and ligature-induced periodontitis suggesting that a local immunoregulatory imbalance is associated with disease. Disease susceptibility has been evaluated using the autologous mixed lymphocyte response (AMLR) as an in vitro measure of immunoregulation. A depressed AMLR has been demonstrated in some, but not all patients with severe periodontitis. A reduced AMLR has been demonstrated in generalized juvenile periodontitis versus LJP. This depressed AMLR may be either the result or cause of disease. PMNs have also had a protective role.

SYSTEMIC DISEASES

Systemic diseases associated with severe periodontitis (such as agranulocytosis, cyclic neutropenia, Chédiak-Higashi syndrome, and lazy leukocyte syndrome) have been shown to have qualitative and/or quantitative PMN deficiencies. In modeling genetic factors, immuno-responsive genes may divide the population into two groups consisting of susceptible and non-susceptible individuals. Susceptible individuals are in balance (or stable) with their oral flora until untoward shifts result in progressive lesions. Non-susceptible individuals may also experience environmental disruptions that precipitate shifts to progressive lesions.

NEUTROPENIA AND AGRANULOCYTOSIS

Neutropenia is the reduction in the number of PMNs in the peripheral blood to below 1,500 per mm^3. This condition may be transient and insignificant. When it is below 500 per mm^3 and predisposes to infection, it is called agranulocytosis. One of the known causes of agranulocytosis is drug idiosyncrasies (aminopyrine, chloramphenicol, sulfonamides, chlorpromazine, etc.). Agranulocytosis reduces the defense mechanisms and leads to infections, including ulcerative necrotizing lesions of the gingiva and other areas of the oral cavity. Less frequently, ulcers and infections occur in anal-genital areas, GI tract, skin, urinary tract.

Leukocyte Adhesion Deficiency

These patients have an inherited chemotactic defect of the adhesion glcoproteins. Infections occur early in life and include generalized prepubertal and postpubertal severe periodontal diseases (Waldrop et al., 1987).

Job's Syndrome (hyper-Immunoglobulinemia E)

This is a rare condition in which a complex autosomal recessive disorder induces a defect in neutrophil motility that leads to deficient chemotaxis and infection and "cold" abscesses in skin and respiratory tract. Severe periodontal disease has been described but studies are incomplete.

Chédiak-Higashi Syndrome

Neutrophils have decreased chemotaxis and bactericidal activity resulting in neutropenia, depressed inflammation, and severe periodontal destruction, and ulcerations. Lavine et al. (1976) described severe periodontal disease in mink and mice with this syndrome.

Papillon-Lefèvre Syndrome (PLS)

This syndrome represents rapid destruction of the periodontium and palmo-plantar hyperkeratotic lesions at an early age (prepubertal). PLS has been found to be associated with diminished neutrophil activity (Van Dyke et al., 1984) and an increase in circulating NK cells (Genco, 1992).

Chronic Granulomatous Diseases

This is another rare inherited disorder associated with severe, life-threatening, suppurative infections of skin, liver, lymph nodes, and other organs. Neutrophils and monocytes from these patients have a defective oxygen metabolism and are unable to kill many species of bacteria and fungi. Patients with CDG have oral ulcerations and gingivitis, but there is no correlation with severe periodontal destruction (Cohen et al., 1985).

Periodontal Diseases

Altman et al. (1985) studied PMN and monocyte (MN) chemotaxis in 7 patients with prepubertal, 37 with juvenile, 35 with rapidly progressive, and 8 patients with adult type periodontitis. In the prepubertal groups, 5 patients had abnormal PMN chemotaxis, 5 had depressed MN chemotaxis, and 1 had reduced serum chemotactic activity. All patients in this group had at least 1 form of reduced leukocyte chemotactic activity. In the juvenile periodontitis group, 17 patients had abnormal PMN responses: 16 were depressed and 1 was enhanced. MN chemotaxis was depressed in 4 of these patients and elevated in 2. Five patients had reduced serum chemotactic activity and 1 manifested a serum chemotactic inhibitor. In total, 65% of juvenile periodontal patients had some form of abnormality. In the rapidly progressive group, 15 had abnormal PMN chemotaxis, 7 had aberrant MN chemotaxis, 4 had reduced serum chemotactic activity, and 8 had a serum inhibitor of chemotaxis. No abnormalities were found in the PMNs or sera of the adult periodontitis patients. Overall, 66% of the early-onset patients manifested some form of cell or serum-related leukocyte chemotactic abnormality.

Reinhardt et al. (1988) evaluated lymphocyte subset densities and distributions in gingival biopsies from active, stable, or healthy sites. Sections were labeled with monoclonal antibodies for 1) pan T cells, 2) T-suppressor (Ts) cells, 3) T-helper (Th) cells, and 4) pan B cells. Lymphocyte populations were identified from the sulcular, middle, and oral one-third of each section. Relative proportions of lymphocyte subsets were also analyzed in peripheral blood samples using direct immunofluorescence. Pan B cells were significantly more prevalent in infiltrates from active sites than stable or healthy sites. The T/B cell ratio was significantly lower in active versus stable sites or blood. The Th/Ts cell ratio did not vary significantly between groups, but a trend toward lower relative numbers of Th cells in sulcular infiltrates of active sites was noted. These results support the premise that active periodontal sites display elevated B cell populations and abnormal immune regulation possibly involving the Th cell subset.

Donaldson and Ranney (1982) compared the blastogenic response of periodontally healthy subjects with matched groups of subjects having either LJP, severe periodontitis, or moderate periodontitis. Bacterial stimulants of known pathogens to these disease entities were utilized. Peripheral blood lymphocytes (PBL) were harvested after a 4-hour pulse with tritiated thymidine on days 4 and 6 of culture. The healthy subjects responded as frequently as those in all the diseased groups. The dose-response distributions of these groups were indistinguishable and the magnitude of the responses was not substantially different between groups. These results suggest a nonspecific activation of blastogenic response to antigenic stimulation rather than specific sensitization occurring during initiation or progression of periodontitis.

Using mitogens and hemogenates of periodontopathogens, Osterberg and Page (1983) evaluated the blastogenic responsiveness of peripheral blood monocytes (PBM) obtained from LJP, rapidly progressive periodontitis (RPP), adult periodontitis (AP), and healthy control subjects. Blastogenic responsiveness to unstimulated cell cultures, putative periodontal pathogens *Bacteroides melaninogenicus*, *Capnocytophaga*, *Fusobacterium nucleatum*, *Actinomyces viscosus*, and to mitogens phytohemaglutinin and pokeweed mitogen was assessed by tritiated thymidine uptake after 3 days (mitogens) and 5 days (bacterial). They found that since PBMs from periodontally diseased subjects respond to plaque and bacteria by undergoing blastogenesis, measurement of lymphoid cell responsiveness could help establish diagnosis, prognosis, and recall intervals. Results indicated that the AP and healthy groups differed with regard to spontaneous blastogenic activity in unstimulated cultures. This reflects different proliferation rates of T-lymphocyte subsets which respond to the presence of autologous non-T cells and ultimately to a different immune response. Patients with chronic periodontitis may have basic abnormalities in mechanisms of immune regulation. Results also demonstrated marked increases in unstimulated and bacterially stimulated PBM responsiveness during ther-

apy, with decreased responsiveness to the mitogens and autologous plaque. This enhanced immune responsiveness may be a consequence of a developing immune response accompanying inoculation of bacterial-substances into the blood and lymph during periodontal treatment. Protective immunity could be a beneficial effect of treatment.

Tew and Miller (1981) compared the cellular response of young adults with severe periodontitis (SP) to those with a healthy periodontium (HP). They evaluated T-cell and B-cell levels, blast transformation, production of leukocyte inhibitory factor (LIF), and phagocytosis and killing by peripheral PMNs. They found that PMNs and B-cell levels were virtually identical. While blastogenesis was not statistically significantly different, SP subjects tended to respond to bacterial extracts more frequently. Responses to bacterial extracts were higher in female SP and lower in male SP patients when compared to the HP group. No differences existed in group responses to phytohemaglutinin (PHA). Thymidine uptake in unstimulated control cultures of SP subjects was significantly lower than the average background in cultures from HP subjects. Production of LIF was not different in the SP and HP groups, nor was the phagocytosis or killing of *Streptococcus sanguis* by PMNs.

O'Neill and Woodson (1982) evaluated lymphoid cells from human gingival tissues classified as normal, periodontal inflammation without pocket formation (group 1) or periodontal inflammation with pocket formation (group 2). These cells were assessed for their ability to kill gingival fibroblasts in vitro and to produce lymphotoxin without in vitro stimulation. No cytotoxic activity was exhibited by normal cells while activity increased from group 1 to group 2. The cells from the latter group were very active. Lymphotoxin production followed a similar pattern. The authors concluded that chronically inflamed gingiva exhibited a localized hyperimmune response in which gingival lymphocytes were activated, with potential tissue destruction accompanying lymphotoxin production.

Celenligil and Kansu (1990) evaluated the phenotypic properties of gingival lymphocytes in adult periodontitis using immunohistological analysis. Gingival tissue lymphocytes were identified using monoclonal and polyclonal antibodies. All specimens revealed a significant degree of CD3+ (mature) cell infiltration beneath the pocket epithelium compared to the oral epithelial side. CD4+ (T-helper) cells and CD8+ (T-suppressor) cells were evenly distributed. Numerous HLA-DR+ cells were also noted. There was a predominance of IgG-bearing plasma cells identified in the lamina propria, followed by IgA-positive cells and a few IgM-positive cells. These findings suggest that T-cell mediated regulatory mechanisms play an important role in the pathogenesis of adult periodontitis.

Using a rat model, Yamashita and Ohfuji (1991) transferred a single *Actinobacillus actinomycetemcomitans* (Aa) T-helper (Th) cell specific clone to a group of heterozygous rats (Aa+Th+). A second (Aa+Th-) and third group (Aa-Th-) received no T cells. Beginning 1 day after transfer, the first and second groups were infected orally with Aa for 5 consecutive days. A significantly higher number of lymphocytes were recovered from the gingival tissues of the Aa+Th+ group than either of the other groups. The Aa-Th- group exhibited significantly elevated serum IgG and IgM to Aa compared to the other groups. Bone loss was significantly reduced in the Aa+Th+ group compared to the Aa-Th- group and was approximately equal to the third uninfected group. This experiment supports the hypothesis that T-cell regulation can affect periodontal disease with Th cells apparently interfering with periodontal bone loss.

Okata and Ito (1987) evaluated the effect of T-cell influence on IgG synthesis in T-cell independent polyclonal B cell activation. Results supported the hypothesis that T-helper cells become activated and introduce signals to B cells. T-helper cells could not respond to antigens from *Actinomyces viscosus* or bacterial lipopolysaccharide from *E. coli* but could recognize the "self" major histocompatibility complex class II antigen expressed on the surface of B cells. These cells subsequently activated and participated in T independent B cell activation. The results suggest that T cells may regulate polyclonal B cell activation by oral bacteria in periodontal inflammation and thereby participate in the development of IgG-rich periodontal lesions.

Ito and Harada (1988) continued the investigation related to the previous study by in vitro examination of the effect of autoreactive T cells on T-independent polyclonal B cell activation (PBA) IgG synthesis in a mouse model. Th cells were activated by interaction with Ia antigens expressed on B cells. Activated T cells enhanced IgG synthesis in the PBA reactions. Ia antigen expression on B cells was increased when stimulated by polyclonal B-cell activators. The supernatant from an autoreactive T cell line also enhanced IgG synthesis in PBA. The results suggest that autoreactive T cells may play a role in the establishment of the IgG plasma cell-rich periodontal lesion.

Ranney and Zander (1970) demonstrated that hypersensitivity reactions of antibody to bacterially-produced antigens may be important in periodontal disease. Reactions ranged from inflammation upon initial challenge with antigen to an acute destructive lesion characterized by many of the features of human periodontal disease. These changes were observed after 3 months of repeated challenges.

HUMORAL IMMUNITY

Tew and Engel (1989) reviewed polyclonal B cell activation (PBA) in periodontitis. Both antigen-specific and polyclonal activation of lymphocytes may occur in periodontally diseased tissues. The B-cell life cycle consists of 4 stages: resting, activation, proliferation, and differentiation. Differentiation is influenced by certain substances or events resulting in either immunoglobulin secreting cells (plasma cells) or memory (memory B cells). Activation occurs pri-

marily through antigen interaction with cell surface immunoglobulin receptors. Other factors such as lipopolysaccharide (LPS) and pokeweed mitogen may also induce B cell activation. These PBA factors may stimulate 30% of B cells, with different factors apparently affecting selected subpopulations or clones of B cells. B-cell growth is influenced by substances which provide competence signals (e.g., interleukin-4) that enhance major histocompatibility class II molecules (HLA-DR) and prompt phase entry of resting B cells upon LPS stimulation. Progression signals such as B cell growth factor also promote DNA synthesis. Activated B cells produce effector molecules which may also play a role in the progression of periodontal disease. These effectors include immunoglobulin, interleukin 1, interleukin 2, interferon, and tumor necrosis factor. Antibodies produced by PBA factors are not highly avid or specific; however, the high numbers of clones activated make it likely that reaction with a given microbial invader may occur. Resulting antibodies may participate in blocking adhesion, thereby increasing opsonization and enhancing complement-lysis. B cells and plasma cells are the predominant inflammatory cells in the established and advanced periodontal lesions.

Studies suggest that patients with B cells which are more responsive to B cell mitogens may be most susceptible to periodontal destruction. Inherited hyper-responsiveness to B cell mitogens may explain the familial tendency observed in early-onset periodontitis. The amount of specific antibody stimulated by antigen alone or PBA factors alone is small compared with the level of specific antibody obtained by the combination of specific antigen plus non-specific activator. Human serum immunoglobulin levels are only modestly elevated in patients with periodontal disease because of the localization of the lesion and the concentration of locally-produced immunoglobulin in GCF (versus the blood vascular system). Bacterial-derived PBA factors are linked with many periodontitis-associated Gram-negative bacteria and several Gram-positive species. Bacterial components which may elicit PBA include LPS, peptidoglycan from the cell envelope, and extracts of bacterial products. Activated B cells and plasma cells may form antibodies which react with bacteria or host tissues to form immune complexes, leading to complement activation and complement mediated toxicity reactions. Antibody-dependent, cell-mediated cytotoxicity may also occur. Serum levels of autoantibody (produced by PBA factors) to type 1 collagen may be higher in periodontitis patients than normal controls. Polyclonally activated B cells also produce IL-1 which demonstrates bone-resorbing activity as well as numerous other effects on connective tissue and the immune and inflammatory systems.

Klausen and Houghen (1989) evaluated the role of T-lymphocytes and B-lymphocytes in the development of marginal periodontitis using a rat model consisting of nude (congenitally T-lymphocyte deficient), thymus-grafted nude (lymphocyte reconstituted), anti-u treated (temporarily T-lymphocyte deficient) and normal rats. A group of rats were inoculated with Aa, Pg, and a strain of oral spirochetes. Ninety-five percent (95%) of the inoculated rats had increased serum levels of IgG or IgM against one or more of the test microorganisms, with nude mice having the lowest. Inoculated rats had significantly less periodontal bone support than the controls. A temporary deficiency led to significantly less periodontal bone support than the congenital T-lymphocyte deficiency or normal rats. No difference was found between normal, congenital, or temporary deficiencies. The authors concluded that congenital T-lymphocyte deficiency did not interfere with the development of periodontal diseases in this model, whereas a temporary and moderate reduction in B-lymphocyte numbers seemed to predispose periodontal bone loss.

Ebersole and Frey (1987) investigated the relationship between local and systemic host antibody responses, colonization of subgingival plaque by periodontal disease-associated microorganisms and the progression of periodontal disease in 61 patients. They reported 54 to 78% agreement between the distribution of elevated static crevicular fluid antibody and presence of corresponding microorganisms for most periodontopathogens. Data showed that antibody was produced locally since local levels were greater than serum levels.

Turner and Dai (1989) evaluated serum and gingival tissue antibody levels to 8 oral microbial antigens in adult periodontitis and healthy patients. Using an enzyme-linked immunosorbent assay (ELISA), they found that antibody levels of diseased serum samples were significantly higher than those of healthy serum samples. Serum antibodies against Pg antigens were significantly higher than all others except Pi and *B. asaccharolyticus*, while gingival tissue antibody levels against Pg antigens were significantly higher than all other antigens. The results suggest selective, specific, and localized antibody production during or following the establishment of chronic adult periodontitis.

Using serum samples, Vincent and Falkler (1987) evaluated the effect of clinically successful periodontal therapy in LJP, rapidly progressive (RPP), and periodontally healthy subjects. They evaluated antibody levels of Pg, *B. ochraceus*, *Fusobacterium nucleatum*, and Aa using an ELISA. LJP patients showed an initial rise in antibody levels immediately following therapy and a significant decrease in antibody levels 3 to 4 years later. The RPP patients demonstrated only a significant decrease at 3 to 4 years. The antibody levels of both LJP and RPP patients remained significantly higher than healthy subjects at all time points.

Using direct immunofluorescence, histologic sections, and gingival eluates, Okada and Kida (1982) studied the cell types involved with advanced periodontitis in humans.

In lamina propria, 65% of the mononuclear cells were plasma cells that produced predominantly IgG. Cells producing IgA represented 11.2% and those producing IgM, 1.3%. In the sulcular areas, many of the Ig negative cells were T-lymphocytes, accounting for about 30% of the infiltrated cells. Only a few T cells were found in the lamina propria. Macrophages and monocytes were detected in and subjacent to the pocket epithelium. When all cells with Fc receptors were examined, 30% consisted of macrophages and monocytes. Two types of T cells with Fc receptors were delineated. One type had Fc receptors for IgG (T-gamma) and the other, Fc receptors for IgM (T-mu). When comparing these "T cell families" it was noted that the T-mu cell quantities were equivalent in peripheral blood and gingival tissues. T-gamma cells, however, were less numerous in local gingival tissues than in the peripheral blood. This may represent an imbalance in the T-gamma cells and may be part of a local immunological imbalance accounting for progression of periodontal disease.

Daly and Cripps (1983) investigated immunoglobulin production in vitro for lymphocytes recovered from gingival tissue of patients with chronic marginal gingivitis. The authors assessed IgG production over 7 days, reporting that 56% of the IgG was present by 24 hours, 80% by day 3, and 86% by day 5. IgG production by gingival lymphocytes was higher than that of optimally stimulated peripheral blood lymphocytes, while IgA levels were similar. The results indicated that lymphocytes recovered from chronically-inflamed gingival tissue secreted immunoglobulin progressively throughout a 7-day culture period. Gingival B-lymphocytes appeared to have been highly stimulated in vivo and were actively secreting immunoglobulin at the time of recovery from tissue.

Yamashita and Ohfuji (1988) evaluated the blastogenic response and immunoglobulin production (IgG, IgM) by lymphocytes (GL) obtained from the inflamed gingiva of dogs. The responses of GLs were compared to those of peripheral blood lymphocytes (PBL) and submandibular lymph node cells (SNL) following stimulation with 3 mitogens. The responses of PBLs and SNLs were substantial while GL response levels remained at the level of unstimulated lymphocytes. GLs did produce and secrete IgG and IgM, with IgG production elevated above that of PBLs and SNLs. IgM production was less than PBLs and SNLs. The local response of GLs is apparently different from the systemic response of PBLs and SNLs.

Reinhardt and McDonald (1989) investigated IgG subclasses in the gingival crevicular fluid (GCF) of periodontally active and clinically similar, but stable or healthy sites. Using an ELISA (monoclonal antibodies), IgG subclass and albumin concentrations in serum and interproximal GCF samples were quantified. Despite variability, mean IgG1 and IgG4 concentrations were higher in GCF from active periodontitis areas than from stable sites. Mean adjusted concentrations in GCF were generally greater than in serum, especially for IgG4. This increased level of IgG4 may be a useful indicator of the immunological changes which take place in active periodontitis.

Hall and Falkler (1990) evaluated the local production of immunoglobulins in diseased tissue from LJP patients. IgG was the major immunoglobulin present in the first-day tissue culture medium in 92% of the LJP tissue explant cultures. This figure decreased to 43% in day 4 supernatants. IgA was present in 15% of the cultures, with no evidence of IgM in any tissue culture specimen. This study further supports the protective or destructive involvement of local immune processes in the pathogenesis of disease.

Murray and Burstein (1989) examined the immune response to Pg in serum and GCF of patients with gingivitis, untreated adult periodontitis, and treated adult periodontitis. Using ELISA, untreated patients demonstrated a humoral immune response to Pg, producing significantly higher serum and local GCF levels of IgG than did treated patients. The ratio of GCF to serum antibody levels was not significantly different among any of the groups. The authors concluded that the GCF levels of antibody may be due to leakage of serum into the GCF rather than a site-specific response to infection.

Mackler and Waldrop (1978) investigated subclasses of IgG-bearing cells in gingival biopsies in order to further define inflammatory cell infiltrates and correlate findings with different stages of human periodontal disease. Mild gingivitis was characterized by lymphocytes which lacked surface IgG and Fc receptors suggestive of thymus dependent cells. In severe gingivitis the localized cellular infiltrate changed, with increasing numbers of IgG1 (22%) and IgG3 (17%) labeled lymphocytes, and with lower numbers of IgG4 (7%) and IgG2 (1%). Destructive periodontitis was characterized by high numbers of plasma cells (57%) distributed throughout the gingiva. The subclass distribution was IgG1 (25%), IgG4 (19%), IgG3 (18%), and IgG2 (1%). This distribution did not reflect normal serum concentrations. These findings support the concept that a shift from a T cell to a B cell dominated lesion occurs in advancing periodontal disease.

Ogawa and Tarkowski (1989) also analyzed the distribution of IgM, IgG, and IgA secreting cells isolated from gingiva at different stages of periodontal disease. The total number of plasma cells increased with the severity of disease. The majority were IgG isotypes with significant numbers of IgA+ cells also present. Few IgM+ cells were observed. Monoclonal antibodies were utilized to analyze IgG and IgA subclasses. Analysis of slight, moderate, and advanced stages of periodontal disease showed a progressive increase in spot-forming cell numbers. The major isotype was IgG followed by IgA. IgG1 was the major IgG subclass followed by IgG2. IgG3 and IgG4 subclass levels were found to be similar to each other. IgG4 levels in-

creased in more advanced disease, IgA1 predominated in moderate stages, and selective increases in IgA occurred in more advanced stages of disease. These gingival responses are similar to those found in synovia of rheumatoid arthritis subjects and in mitogen triggered spleen and peripheral blood mononuclear cells.

Hara and Maeda (1987) compared gingival tissue specimens from patients with chronic periodontitis with clinically healthy or gingivitis subjects to determine relative numbers of immunoglobulin-bearing cells. The authors reported a predominance of IgG-bearing cells, followed by IgA-bearing cells. IgM and IgE-bearing cells were present in small numbers. The latter cell types were elevated in moderately and severely infiltrated lesions relative to the total inflammatory cells present. Their findings support the concept that a hypersensitivity reaction mediated by IgE may play a role in periodontitis.

Nisengard (1977) has written an excellent review on the role of immunology in periodontal disease which discusses basic immunology, including information on immunoglobulins, complement, and hypersensitivity reactions.

Ebersole (1990) authored a comprehensive review detailing the systemic humoral immune responses in periodontal disease. Information includes: 1) general aspects of humoral immune responses to bacteria; 2) bacterial specificity of antibody responses in periodontal disease; 3) antigen specificity of antibody responses in periodontal disease; 4) longitudinal considerations in humoral immune responses in periodontitis; 5) function of humoral antibody responses in periodontal disease; 6) diagnostic potential of antibody responses in periodontal disease; and 7) future considerations of humoral responses in periodontal disease. This review is invaluable as a reference for immunology relative to periodontitis.

Lally and McArthur (1982) investigated the biosynthesis of complement components in chronically-inflamed gingiva. C3 and C5 synthesis was detected in 8 out of 10 individuals with periodontal disease, compared to none in normal controls. Results indicated that greater consumption of complement occurs in periodontal disease than previously predicted on the basis of serum studies. The local production of complement could play a role in modulating the inflammatory response in the gingiva. The macrophage is involved with extrahepatic synthesis of complement. C5 synthesis has been observed in a variety of sites but usually by monocytes. Both C3 and C5 synthesis can occur at mucosal sites as well. Complement may have a modulating effect on host defense and plaque organisms in the gingival crevice.

Anusaksathien and Dolby (1991) reviewed evidence for an autoimmune component in the host immune response of periodontal disease. They reported that the majority of studies address detection of antibodies (AB) to host components, especially collagen. ABs to DNA and aggregated IgG have also been reported, as have in vitro reports of killing of cells of the periodontium by mononuclear cells isolated from patients with periodontal disease. The presence of autoantibodies in periodontal disease may be explained by the following: 1) enhanced presentation of self antigens (Ag) through increased expression of the molecule associated with Ag presentation; 2) altered T-helper or T-suppressor cell function; 3) polyclonal activation of cells which have the ability to produce autoantibodies; 4) idiosyncrasies of the Ag-idiotype network; 5) bacterial or viral cross reactivity with self-antigen leading to the production of cross reactive ABs; and 6) genetic predisposing factors.

Autoantibodies detected in periodontal disease appear to be derived from pre-existing natural ABs and play a physiologic role in the elimination of dead cells and damaged tissue constitutents resulting from the tissue degradation associated with periodontal disease. The possibility remains that this system, established to deal with the consequences of tissue damage, may in certain circumstances become excessive and contribute to the progress of the disease.

PERIODONTAL STATUS IN IMMUNOSUPPRESSED PATIENTS

Schuller et al. (1973) investigated the severity of periodontal disease in 33 renal transplant patients receiving prednisone and azathioprine to suppress the immune responses and prevent rejection. The findings indicate that persons on immunosuppressive therapy show no correlation between age, plaque, and calculus. This implies inhibition of inflammation and/or the immune response.

Tollefsen et al. (1978) studied chronic gingival lesions in 4 categories of patients. One group of healthy subjects were kept plaque-free (CO), while in the second group (CP) moderate accumulations of plaque were permitted. The third group (UH) was comprised of patients with uremia and in hemodialysis. A fourth group (IS) had received renal allografts and were on an immunosuppressive regimen. Differential blood counts and serum immunoglobulin quantitation from the UH and the IS groups gave mean values within normal ranges. Gingival biopsies were obtained for each subject. The connective tissue inflammation (CTI) scores were compared between the groups. Despite abundant local plaque accumulations, the UH group displayed essentially the same CTI scores as plaque-free controls (CO), while the IS group showed a significantly lower CTI score than the 2 in question. The CTI scores of the CP group (controls with plaque) were significantly higher than those of the UH, IS, and CO groups.

Tollefsen et al. (1982) took gingival biopsies from 3 main categories of patients. One group (IS) consisted of 19 patients of whom 16 had received renal allografts. All were treated with immunosuppressive agents. A second group (UH) was comprised of 19 patients who suffered from chronic renal failure. Control specimens were obtained from 30 systemically healthy patients with plaque-free teeth

and healthy gingiva. Samples were also taken from 30 other systemically healthy persons with less efficient oral hygiene. All specimens were examined by light microscopy. In addition, 11 selected biopsies were processed for light microscopy. Beneath the dento-gingival epithelium, the control group with plaque had a significantly higher number of cells than the other groups. Residual cell infiltrates were always present in samples from the plaque-free healthy subjects and the uremic patients, whereas scaling and an adequate plaque control virtually eliminated inflammatory cells from the IS specimens. Lymphocytes predominated in the lesions of the UH and IS patients with clinical loss of attachment and persistent inadequate oral hygiene. The authors concluded that immunosuppression does not abolish the host reaction to dental plaque, but the inflammatory and/or immune responses are different from those in otherwise healthy subjects.

Been and Engel (1982) reported that the administration of immunosuppressive drugs significantly reduced the level of gingival inflammation in the presence of high levels of plaque.

Tollefsen and Johansen (1985) compared the periodontal condition of 33 prospective and 26 renal transplant recipients with systemically healthy patients, matched for age, teeth present, social status, and sex. Progressive uremia and immunosuppression by drug therapy resulted in less clinical gingivitis.

Novak and Polson (1989) studied the effects of levamisole on experimental periodontitis. The immunomodulating agent, levamisole hydrochloride, enhances PMN chemotaxis. Levamisole was administered by oro-gastric intubation to 4 squirrel monkeys (experimental) every 2 days for 18 days. After 2 doses of levamisole, marginal periodontitis was induced around several teeth. Similar periodontitis was induced in 4 control monkeys not receiving levamisole. All animals were killed 2 weeks after induction of periodontitis. Clinically, gingival inflammation was more pronounced in experimental animals at both 7 and 14 days after initiation of periodontitis. The enhancement of the inflammatory response by levamisole resulted in a denser band of inflammatory cells between plaque and gingival tissues, but this did not afford any additional protection against the initiation, progression, and extent of periodontal destruction.

Tolo (1991) reviewed periodontal diseases in immunocompromised patients and defined immune deficiency as either primary or secondary. Primary type deficiencies involve a total deficit of one portion of the immune system, with males affected most frequently. Affected individuals usually live for only a few years. Secondary type deficiencies represent substandard responses of the immune system to challenge. The prevalence of immune deficiency among patients with periodontal disease is unknown. Factors important to resisting infections include: circulating granulocytes above 500/mm^2, immediate granulocyte response to infection and leukocyte adhesion and locomotion. Expression of specific glycoproteins (Mac-1, LFA-1, and p150,95) stored intracellularly in secondary granules mediate adhesion and locomotion. In adhesion, Mac-1 or CR3 are the surface receptors for C3b. LFA-1 is important for adhesion, phagocytosis, and production of hydrogen peroxide. Granulocytes can move at 40 to 50 μm/hour and macrophages at 10 to 20 μm/hour. Total hereditary deficiencies of these glycoproteins lead to death by age 3. Moderate deficiencies result in gingivitis or periodontitis characterized by an absence of pus.

In LJP patients, defective neutrophil chemotaxis is present 70 to 80% of the time. Colonizing bacteria may interfere with defense mechanisms. Neutrophils and macrophages express Fc-receptors that moderate attachment to opsonins such as IgG, IgM, and C3b which stimulate the cells to phagocytize the antigen. Some organisms can produce Fc-binding proteins which interfere with complement activation.

Increased levels of autoantibody production by gingival plasma cells have been observed in patients with periodontal disease. Rheumatoid factor, another autoantibody, may also play a role in periodontal disease. The inflammatory infiltrate in the gingival sulcus may cause accumulation of lymphocytes and initiate the production of anti-IgG and anti-type I collagen antibodies by polyclonal B stimulation. In periodontal disease there is an increase in both IgG and IgA. Since monomeric IgA is present, it may block the opsonic effect of IgG in complement activation. IgG1 and IgG3 are the most effective C3 activating subclasses.

Tolo (1991) indicates that a compromised immune system may be an important factor in the progression of periodontal disease.

Dahlen et al. (1993) investigated the presence of caries and periodontal disease in 22 females and 3 males with primary hypogammaglobulinemia or IgG subclass deficiencies with or without comcomitant IgA deficiency. Only 1 patient showed more tooth loss than that found in the normal Swedish population. One patient demonstrated advanced periodontal disease. No patient exhibited more severe dental caries than that of comparable normal Swedes. Microbiological samples from periodontal pockets and saliva showed recovery of potential periodontopathic and cariogenic bacteria within normal ranges. This study could not support the notion that immunodeficient subjects exhibit an increased risk of developing periodontal disease or caries.

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Section 4. Furcation Anatomy and Furcation Invasion

DEFINITIONS

Furcation: The anatomic area of a multirooted tooth where the roots diverge.

Furcation Invasion: Pathologic resorption of bone within a furcation.

INTRODUCTION

Furcations may be divided into three parts on the basis of anatomy. 1) roof; 2) surface immediately coronal to root separation (flute); and 3) the area of root separation where the roots are separated by alveolar bone (root separation) (Grant et al., 1988) (Figure 1).

The etiology of furcation invasion has been attributed to the extension of periodontal inflammatory disease (Waerhaug, 1980), trauma from occlusion in the presence of inflammation (Glickman, 1963; Lindhe and Svanberg, 1974), pulpal disease (Bender and Seltzer, 1972), defective plaque-retentive restorations (Gilmore and Sheiham, 1971) or because of anatomic variations. Proximal furcae of maxillary molars and birooted first premolars tend to be invaded early in the progression of marginal periodontitis, with invasion of mandibular furcae occurring later due to inherent buccal and lingual positions (Schluger et al., 1990).

CLASSIFICATION

Many systems have been suggested for classifying furcation invasions. A brief review of 3 systems is presented below

Glickman (1958) divided furcation invasions into 4 grades:

Grade I: Pocket formation into the flute but intact interradicular bone;

Grade II: Loss of interradicular bone and pocket formation of varying depths into the furca but not completely through to the opposite side of the tooth;

Grade III: Through-and-through lesion;

Grade IV: Same as Grade III with gingival recession, rendering the furca clearly visible to clinical examination.

In 1975, Hamp et al. proposed 3 levels:

Degree I: Horizontal loss of periodontal tissue support < 3 mm;

Degree II: Horizontal loss of support > 3 mm but not encompassing the total width of the furcation;

Degree III: Horizontal through-and-through destruction of the periodontal tissue in the furcation.

Tarnow and Fletcher (1984) proposed the following classification based on the vertical component of bone loss in furcations:

Subclass A: 0 to 3 mm probeable depth;

Subclass B: 4 to 6 mm probeable depth;

Subclass C: > 7 mm probeable depth.

ANATOMICAL CONSIDERATIONS

Mardam-Bey et al. (1991) reviewed anatomical factors which may predispose the furcation to attachment loss. Studies included in their review and other investigations by others have identified furcation and root morphology, enamel pearls, certain enamel projections, and the existence of accessory pulp canals as factors of concern in furcation invasion:

Furcation Morphology

Bower (1979A) reported that 81% of all furcation entrance diameters measure < 1 mm, with 58% < 0.75 mm. Since commonly used curets have blade face widths ranging from 0.75 to 1.10 mm, it is unlikely that proper instrumentation of furcations can be achieved with curets alone. Similar findings were reported by Chiu et al. (1991) in maxillary and mandibular permanent first molars from Hong Kong Chinese. This study revealed 49% of furcation entrances to be < 0.75 mm in width, and the authors suggested that sharpening curets to narrow blade width or using an ultrasonic tip which has a 0.5 mm diameter at the terminal end may improve instrumentation of furcations. In a study of furcation root surface anatomy, Bower (1979B) also found that the furcal aspects of maxillary roots were concave in 94% of mesiobuccal roots, 31% of distobuccal roots, and 17% of palatal roots. Mean depths of these concavities were 0.3 mm, 0.1 mm, and 0.1 mm respectively. Mandibular molars presented concavities in 100% of the mesial roots and 99% of the distal roots, with mean depths of 0.7 mm and 0.5 mm, respectively.

Root Morphology

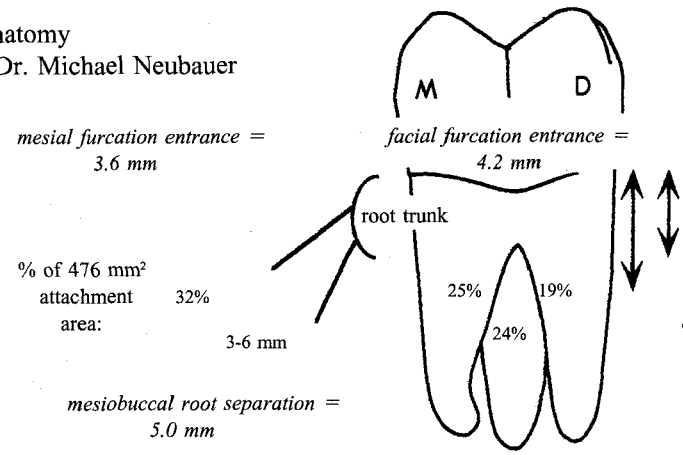
Booker and Loughlin (1985) found mesial concavities in 100% of 50 maxillary first premolars evaluated. The average CEJ to furcation distance was 7.9 mm. They reported that 100% of the two rooted maxillary first premolars examined had "developmental depressions" in the furcal aspect of the buccal root at the 9.4 mm level. Furcation entrances for maxillary molars are located 3.6 mm, 4.2 mm, and 4.8 mm apical to the CEJ on the mesial, facial and distal surfaces, respectively (Gher and Dunlap, 1985).

Gher and Vernino (1980) reported that 78% of maxillary first premolars have a developmental depression on the furcation surface of the buccal root (buccal furcation groove). The mesiobuccal root of the maxillary first molar presented a developmental depression on the distal surface, with other roots having concavities on furcal aspects. The surface area of the mesiobuccal root may be greater or equal to that of the palatal root. The distal and mesial roots of mandibular first molars had mesial and distal concavities.

Hermann et al. (1983) investigated the potential attachment area of the maxillary first molar noting that the surface area of the root trunk was significantly greater than any of the individual roots. The root trunk averaged 32% of total root surface area, the mesiobuccal 25%, the palatal 24%, and the distobuccal 19%. Horizontal attachment loss which extends to the level of the furcation involves the root trunk and results in a loss of one third of the total support of the tooth (Grant et al., 1988). Using the surface measurements reported by Dunlap and Gher (1985), the respective percentage of total root surface for mandibular first molars

Figure 1. Furcation Anatomy
Original drawings by Dr. Michael Neubauer

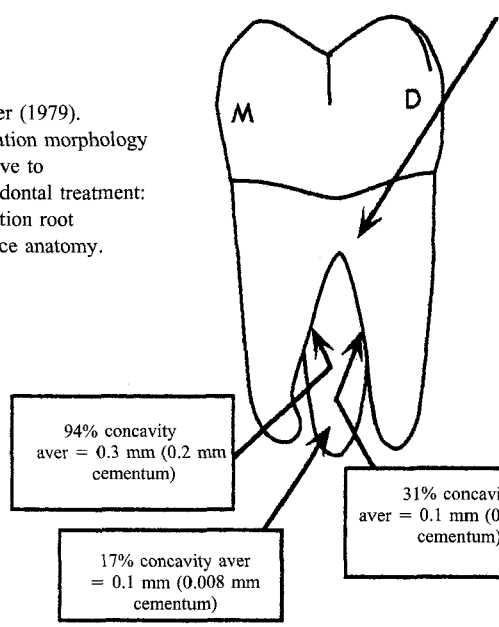
Hermann and Gher (1983). The potential attachment area of the maxillary first molar

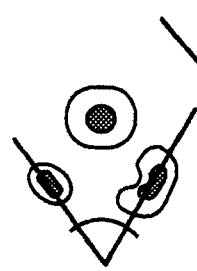


Gher and Dunlap (1985). Linear variation of the root surface area of the maxillary first molar

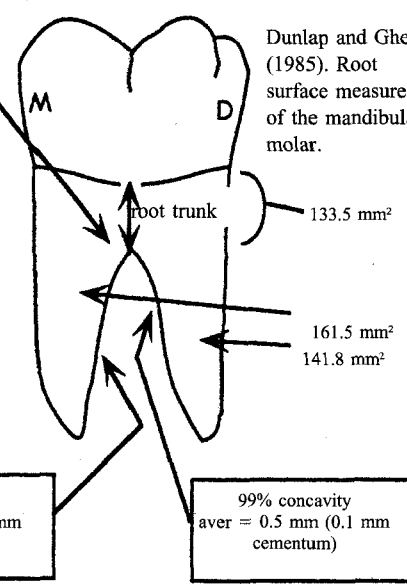
Bower (1979). Furcation morphology relative to periodontal treatment—furcation entrance architecture
81% < 1.0 mm; 58% < 0.75 mm
(aver curet = 0.75–1.10 mm)

Bower (1979). Furcation morphology relative to periodontal treatment: furcation root surface anatomy.



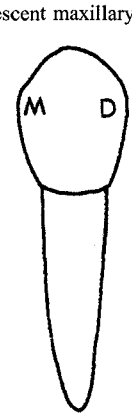


Dunlap and Gher (1985). Root surface measurements of the mandibular first molar.

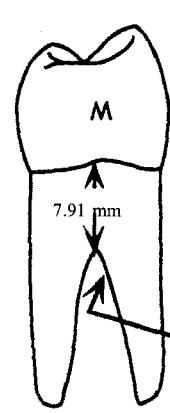


Booker and Loughlin (1985). A morphologic study of the mesial root surface of the adolescent maxillary first premolar.

100% concavity
single rooted teeth: 0.35 mm deep increasing to 0.59 @ 4.7 mm
double rooted teeth:
0.44 mm deep increasing to 1.08 @ 4.7 mm



Most had concavities at or below 2.35 mm from CEJ > in furcated teeth and less deep than M



Increasing cemental thickness as proceed apically

"Buccal Furcation Groove" found at 9.4 mm level (in 100% of teeth)

37% for the mesial root; 32% for the distal root; and 31% for the root trunk.

Anderson et al. (1983) used stereophotogrammetry to confirm that the mesial root of the mandibular first molar had the greatest root surface area (average 251.9 mm^2). The distal root often appears larger radiographically and clinically.

Intermediate Bifurcation Ridges

Intermediate bifurcation ridges were first described by Everett et al. (1958) and were reported in 73% of the mandibular molars studied. These ridges were primarily cementum, originating on the mesial surface of the distal root, crossing the bifurcation and ending high on the mesial root. The type of surface in the bifurcation from which the intermediate bifurcation ridge originated was also studied. The ridge projected above a buccolingual concavity in 15% of the teeth examined, projected from a convex bifurcational surface in 39%, and in the remainder of the teeth the interradicular surface was flat. If the interradicular area was either concave or flat, it was delineated on its buccal and lingual borders by prominent line angles between the respective sides of the tooth and the bifurcational surface. These prominent borders were designated the buccal or lingual bifurcation ridges and consisted essentially of dentin formations covered with only a small amount of cementum. The intermediate bifurcation ridge was also studied by Burch and Hulen (1974) who reported an incidence of 76.8%. These studies underscore the deterrents that anatomical factors may pose during the performance of effective plaque control by the patient. These aberrations may also present complications for the therapist in root preparation during initial therapy.

Enamel Pearls and Projections

Enamel on the furcal area of the root surface may manifest as cervical enamel projections (CEPs) or enamel pearls. Connective tissue attachment is prevented by the presence of enamel, potentially predisposing the area to attachment loss.

In a review article, Moskow and Canut (1990) cited an incidence rate for enamel pearls ranging from 1.1 to 9.7% (mean 2.69%) with a predilection for maxillary third and second molars. According to Masters and Hoskins (1964), CEPs occur primarily on buccal surfaces of molars (28.6% mandibular, 17% maxillary) and may be classifed as: Grade I: distinct change in CEJ contour with enamel projecting toward the bifurcation; Grade II: CEP approaching the furcation, but not actually making contact with it; and Grade III: CEP extending into the furcation proper. Masters and Hoskins (1964) noted CEPs in $>$ 90% of isolated mandibular bifurcation involvement.

Bissada and Abdelmalek (1973) observed a 50% association between CEPs and furcation invasion. Swan and Hurt (1976) reported a statistically significant relationship between tooth surfaces with Grade II or III CEPs and furcation invasion and concluded that when sufficiently pronounced, CEPs may be an etiologic factor in tissue breakdown. However, Leib et al. (1967) found no relationship between CEPs and incidence of furcation invasion. Most authors agree that Grade I CEPs are most common and that buccal surfaces are most often affected. In decreasing order of incidence, CEPs occur in mandibular second molars, maxillary second molars, mandibular first molars, and maxillary first molars.

Accessory Pulp Canals (APCs)

Controversy exists regarding the role of pulpal disease in the etiology of furcation invasion. The prevalence of accessory canals in the furcal region have been reported by several authors. Bender and Seltzer (1972) noted APCs "in great numbers" in human, dog, and monkey teeth, concluding that the periodontal lesion can develop as a result of pulpal disease. Lowman et al. (1973) studied extracted teeth, reporting the incidence of accessory canals in the coronal and middle thirds of the root surface as 55% in maxillary molars and 63% in mandibular molars. They proposed that pulpal disease may influence the periodontium. Vertucci and Williams (1974) observed accessory canals in the furcation region in 46% of human lower first molars observed. They believed that the isolated periodontal lesion in the furcation area may be of pulpal origin. Burch and Hulen (1974) demonstrated openings in the furcation area in 76% of maxillary and mandibular molars. The authors felt that lesions within the furcation area may be associated with pulpal pathoses. Kirkham (1975) found no accessory canals in the furcation areas of 45 extracted maxillary and mandibular molars and premolars. According to Gutmann (1978), the incidence of accessory canals was 29.4% in mandibular molars and 27.4% in maxillary molars.

DIAGNOSIS

The diagnosis of furcation invasion is best determined by using a combination of radiographs, periodontal probing with a curved explorer or Nabers probe, and bone sounding (Kalkwarf and Reinhardt, 1988). Ross and Thompson (1980) evaluated 387 molars from 100 patients and reported a 90% incidence of furcation invasion in maxillary teeth and 35% incidence in mandibular teeth. Proximal surfaces of maxillary molars were twice as likely to be involved as buccal surfaces. The authors found that radiographs detected furcation invasion in 22% of maxillary and 8% of mandibular molars. Waerhaug (1980) reported greater attachment loss on furcal surfaces (62.8%) than outer surfaces (47.3%). The author suggested that attachment loss was related to the downgrowth of subgingival plaque, especially in furcations. Hardekopf et al. (1987) determined that the association of a radiographic furcation arrow with Degree II or III proximal furcation invasion was significant when compared with uninvolved furcations. The existence of a buccal furcation did not influence its appearance. While cautioning that radiographic and clinical evidence must be

correlated for proper diagnosis, the authors believe that the presence of the furcation arrow is a reliable diagnostic tool. The image at the mesial furcation was 19% for Degree 1; 44% for Degree 2; and 55% for Degree 3 furcation invasions. For the distal furcations, the "furcation arrow" incidence was 12% for Degree 1; 30% for Degree 2; and 52% for Degree 3.

Zappa et al. (1993) assessed the association between clinical depth of involved furcations and their bony defects. Six dentists evaluated furca lesions in 12 patients using the Ramfjord index and the Hamp index and their findings were compared with measurements during surgery. Findings indicated that clinical assessment overestimates the true defect depth.

THERAPY

Therapeutic options that may enhance adequate root preparation include finishing burs, modified curet tips, and ultrasonic instrumentation. Leon and Vogel (1987) reported that ultrasonic scalers were more effective than hand instruments in closed debridement of furcally invaded teeth. The prognosis of multirooted teeth with furcation invasion was once so poor that extraction was the treatment of choice. Treatment options may include: scaling and root planing; open flap debridement; gingivectomy/apically-positioned flaps; odontoplasty, ostectomy/osteoplasty-tunnel procedures; root amputation; tooth resection; and regeneration, including guided tissue regeneration.

Ross and Thompson (1978) reported that relatively conservative treatment of teeth with maxillary furcation involvement resulted in a long-term functional survival rate of 88% for a period of 5 to 24 years after treatment. Their approach to treatment involved combinations of the following procedures: scaling, curettage, comprehensive occlusal correction by coronal reshaping, periodontal surgery of soft tissue, and oral hygiene instruction. It is significant to note that no osseous surgery, root resections, hemisections, or tunneling procedures were performed.

Kalkwarf et al. (1988B) compared results following different therapies for teeth with maxillary and mandibular furcation involvement. Therapies included flap with osseous resectional surgery (FO), coronal scaling (CS), root planing (RP), and modified Widman surgery (MW). FO resulted in the most dramatic reductions in probing depth but also was responsible for a loss in clinical attachment level (CAL). All other groups showed a gain in CAL. However, FO treated teeth demonstrated a lower percentage of sites (2.6%) with significant CAL loss during 2 years of maintenance than CS (6.7%), RP (8.4%), and MW (5.9%).

Carranza and Jolkovsky (1991) noted that techniques developed in the last decade have greatly improved the diagnosis of Grade II furcation involvement. The recommended technique combines the principles of guided tissue regeneration using polytetrafluoroethylene membranes with grafting with porous hydroxyapatite or decalcified freeze-dried bone. Grade III and Grade IV furcation involvements still have a poor long-term prognosis because predictable reconstructive techniques for their treatment have not been demonstrated. When possible, a root resection approach may be advisable.

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Section 5. Additional Local and Anatomic Factors

INTRODUCTION

Numerous local anatomic factors have been allegedly associated with isolated periodontal destruction. Such factors include but are not limited to marginal ridge discrepancies, open contacts, food impaction, palato-gingival grooves, and cervical enamel projections. The rationale for implicating these factors with site-related periodontal destruction relates to plaque accumulation and an anatomic susceptibility to breakdown.

DEFINITION

Food Impaction: The forceful wedging of food into the interproximal space by chewing pressure (vertical impaction) or the forcing of food interproximally by tongue or cheek pressure (horizontal impaction).

FOOD IMPACTION—OPEN CONTACTS

Hirschfeld (1930) classified food impaction according to etiological factors as follows: Class I (occlusal wear); Class II (loss of support proximally); Class III (extrusion beyond occlusal plane); Class IV (congenital tooth abnormalities); and Class V (improper restorative design).

Food impaction may contribute to plaque-induced inflammation and root caries if oral hygiene is ineffective. Uneven marginal ridges, plunger cusps, overbite, open contacts, and defective restorations may also promote food impaction (Carranza, 1990).

Hancock et al. (1980) studied the influence of interdental contacts on periodontal status, assessing food impaction, gingival inflammation, plaque, caries, calculus, restorations, and overhangs. No significant relationship was noted between contact type and gingival index or pocket depth. Four percent (4%) of all interdental contacts exhibited food impaction and probing depths were greatest at open contacts. However, while open contacts were correlated with food impaction and food impaction was correlated with increased probing depth, there was no direct correlation between open/loose contacts and probing depth. The authors concluded that food impaction contributes to periodontal pathosis.

Koral et al. (1981) reviewed 90 patient records demonstrating radiographic evidence of open contacts on bitewing films, with no evidence of open contacts on the contralateral side. Periodontal disease severity was classified as Type I through IV based on record entries and radiographic bone loss. When compared by site, only the Type II group (incipient periodontitis) showed bone loss around the open contact site exceeding the bone loss around the control. With the possible exception of an association in incipient disease, results demonstrated that open contacts were not associated with a greater degree of bone loss than closed contacts.

Jernberg et al. (1983) compared periodontal status adjacent to unilateral open contacts and contralateral closed contacts in 104 patients. Seventy-five percent (75%) of these open contacts studied were in anterior teeth. Slight, but significantly greater, clinical attachment level (CAL) and probing depth (PD) were observed at open contact sites: 60.6% of patients had greater CAL at open contacts compared to 17.3% at closed contacts. Similarly, 49% of patients had deeper PD at open contacts compared to 22.1% at closed contacts. Mean CAL and PD were 2.80 mm and 3.04 mm for open contacts versus 2.32 mm and 2.77 mm for closed contacts, respectively. Significantly greater prevalence of food impaction and occlusal interference was found at open contacts. The significant trend toward increased PD and CAL suggests that closure of the open contact may be beneficial.

MARGINAL RIDGE RELATIONSHIPS

Kepic and O'Leary (1978) examined marginal ridge discrepancies (MRDs) in posterior teeth of 100 patients, cor-

relating PD, plaque (PLI), calculus (CI), gingival status (GI), and attachment loss (AL). Correlations between MRDs and PD, PLI, CI, GI, and AL were low. It was the authors' opinion that effective oral hygiene in the presence of gross MRDs could maintain papillary gingival health with little, if any, attachment loss. Pihlstrom et al. (1986) reported a correlation between attachment loss and uneven marginal ridges.

PALATO-GINGIVAL GROOVES

Palato-gingival grooves (PGG) are developmental anomalies of the maxillary incisor teeth which usually begin in the central fossa, cross the cingulum, and extend varying distances/directions apically. They are also termed palato-radicular, radicular-lingual, and disto-lingual grooves (Withers et al., 1981; Kogon, 1986).

Withers et al. (1981) examined 2,099 maxillary incisors in 531 patients noting the presence of PGGs and correlating them with health status of adjacent periodontal tissues. The authors reported PGGs in 8.5% of individuals examined and 2.33% of maxillary incisor teeth (4.4% in lateral incisors; 0.28% in central incisors). PGGs were associated with poorer periodontal health (GI, PDI) and greater plaque accumulation.

Kogon (1986) examined 3,168 extracted maxillary lateral and central incisors with a dissecting microscope, reporting palato-radicular grooves (PRGs) in 4.6% (5.6% in lateral incisors; 3.4% in central incisors); 54% of the PRGs terminated on the root and 58% of these extended $\geq$ 5 mm apical to the CEJ. Root involvement was more frequent and severe in central incisors; enamel rarely extended onto the root surface (4%).

In a similar study, Everett and Kramer (1972) examined 672 extracted lateral incisors, reporting an incidence of 1.9% PRGs. Extension to the affected root apex was observed in 0.5% of the teeth.

Hou and Tsai (1993) examined a total of 404 maxillary central and lateral incisors in 101 individuals for the presence of PRGs and correlated their presence with plaque index, gingival index, and probing depth in the area. They concluded that: 1) the proportional test showed statistical non-significance among the sexes; 2) PRGs were greatest in lateral compared to central incisors; 3) PRGs were most often located in the midpalatal part of the affected tooth as opposed to the mesial or distal area; however, more distal and mesial PRGs were associated with probing depths when compared to centrally located PRGs; and 4) a direct statistical relationship was established between the depth, location of PRGs, and the gingival and plaque indices and development of probing depths in affected teeth.

OTHER FACTORS

Tal (1984) investigated the correlations between interproximal distance and frequency of intrabony pockets (IBP). He reported a positive and statistically significant correlation between interproximal distances and the presence of IBP. The frequency of IBPs increased with increasing interdental distance and were frequently associated with interproximal distances > 2.6 mm, up to 4.6 mm. Two IBPs in the same interdental area were present only when these areas were > 3.1 mm.

Heins and Wieder (1986) investigated interproximal widths in 29 human (post mortem) specimens: 116 second premolar-first molar and first molar-second molar interproximal spaces. Inter-root distances ranged from 0.2 to 4.5 mm with the narrowest distance observed between the roots of the maxillary first and second molars. Bone did not exist between the roots of adjacent teeth when the inter-root distance was < 0.3 mm; rather, a continuous PDL space from root to root was present. Root proximity of 0.3 to 0.5 mm demonstrated cortical bone and PDL without cancellous bone. Cancellous bone was only observed between the laminae durae separating adjacent teeth when the inter-root distance was > 0.5 mm.

Kugelberg (1990) compared the periodontal status of second molars 2 and 4 years after surgical removal of impacted mandibular third molars in patients < 25 years and < 26 years of age. Two years after third molar removal, 16.7% of cases < 25 years had vertical defects > 4 mm compared to 40.7% in the > 26 year age group. At the 4-year examination, 4.2% of those < 25 years old and 44.4% of those > 26 years old had vertical defects > 4 mm. Vertical defects associated with the distal of second molars improved or were maintained in all subjects < 25 years old. Nearly 30% of the defects in individuals > 26 years old deteriorated further. The author concluded that age is an important factor in healing, and suggested that if the need for extraction can be anticipated, early removal of impacted third molars might have a beneficial effect on the periodontal health of the adjacent second molar.

Kugelberg et al. (1991) prospectively studied periodontal healing after removal of 176 mandibular third molars in patients < 20 or > 30 years of age. In the < 20 year group, initial probing depths were > 7 mm in 21.5% versus 45.8% of the > 30 year old group. At 1 year, 4.8% of the > 30 year old cases and no cases in the < 20 year old group had probing depths > 7 mm. Bone loss > 50% of the root surface was found initially in 18.3% of the < 20 year old group and 41% of the > 30 year old group. At 1 year, only 2.2% of patients < 20 years old had bone loss of > 50% compared to 37.3% of the > 30 year old patients. Intrabony defects > 4 mm were reduced from 32.3% to 14.0% in those < 20 years of age and from 59% to 47% in those > 30 years of age. Widened follicles did not appear to affect healing in the younger age group, whereas 78.3% of widened follicles in patients > 30 years old were associated with deep intrabony defects. A higher prevalence of intrabony defects was found in smokers > 30 years old. The authors concluded that periodontal healing was impaired after third molar removal in patients over 30 years old,

particularly when associated with an intrabony defect or widened follicle.

SUMMARY

All factors reviewed above should be considered as contributors or predisposing elements associated with adjacent periodontal destruction. As such, their role is absolutely secondary to the etiologic impact of bacterial plaque.

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Section 6. The Role of Occlusion in Periodontal Diseases

DEFINITIONS

Occlusal Trauma: An injury to the attachment apparatus as a result of excessive occlusal force.

Primary Occlusal Trauma: Injury resulting from excessive occlusal forces applied to a tooth or teeth with normal support.

Secondary Occlusal Trauma: Injury resulting from normal occlusal forces applied to a tooth or teeth with inadequate support.

Occlusion, Physiologic: Occlusion in harmony with the functions of the masticatory system.

Occlusal trauma as it relates to periodontal disease and therapy has been and remains a controversial issue. Research findings are based on human and various animal model systems.

HUMAN STUDIES

Glickman (1963) proposed the concept of co-destruction in an effort to clarify the role of occlusion in periodontal disease. Glickman stated that, while occlusion does not cause gingival inflammation or pocket formation, inflammation of the supporting tissues in the presence of occlusal trauma alters alignment of transseptal fibers allowing inflammation to spread into the periodontal ligament space with resultant intrabony pocket formation. Glickman defined a zone of irritation bound by the marginal gingiva and transseptal fibers (apically) and suggested a zone of co-destruction bound coronally by the transseptal and labially/lingually by alveolar crest fibers. He hypothesized that occlusal trauma does not affect the zone of irritation; however, inflammation and occlusal trauma become co-destructive in periodontitis.

In a study on human autopsy material, Glickman and Smulow (1965) corroborated the relationship between excessive occlusal forces and the pathway of gingival inflammation shown experimentally in animals. They opine that gingival inflammation and trauma from occlusion are different types of pathologic processes which participate in a single disease, periodontitis. Together they exert a combined co-destructive effect which produces angular bone defects and intrabony pockets.

Stahl (1968) studied 4 surgical human specimens and concluded that there was great variability in periodontal tissue responses to the combination of trauma from occlusion and marked gingival inflammation.

Waerhaug (1979) postulated that bacterial plaque in conjunction with variation in local anatomy was the primary cause of intrabony defect formation and not occlusal trauma. He examined 31 complete dentitions (106 interdental sites) from autopsy specimens, radiographs, occlusal analyses, and mobility assessment prior to jaw removal and preparation of specimens for histologic exam and found no evidence implicating traumatic occlusal forces as a co-factor in the formation of bony defects. In general, the shape of the interdental septum was dependent on the location of the cemento-enamel junctions of adjacent teeth. Loss of attachment was always associated with the apical growth of subgingival plaque located 0.2 to 1.8 mm (mean 0.96 mm) from areas of connective tissue lysis. The reduction in alveolar crestal height was also related to the presence of subgingival plaque, ranging from 0.5 to 2.7 mm (mean 1.63 mm) from the alveolar crest. (Note that a subsequent Waerhaug article [1979] using extracted teeth indicated that the range of bacterial plaque influence was 2.5 mm). The author indicated that angular defects resulted when subgingival plaque advanced to different levels on adjacent teeth

and circumferential defects formed when the alveolus was thicker than the range of bacterial influence.

Yuodelis and Mann (1965) reported a direct relationship between periodontal disease and molar non-working contacts in a retrospective study of charts, non-standardized radiographs and study models of 54 patients (413 molars) with periodontal disease. Fifty-three percent (53%) of molar teeth had non-working contacts and mobility. Bone loss (mean 0.4 mm) and probing depth associated with the latter teeth were significantly greater. No healthy controls were included, nor was there a clinical examination of these subjects to confirm non-standardized charted findings.

A subsequent study by Shefter and McFall (1984) reported no relationship between occlusal disharmonies (centric, balancing, or protrusive contacts) and pathoses associated with inflammatory periodontal disease in 66 patients 15 to 62 years of age (mean 30 years) with mild to moderate periodontitis. Seventy-eight percent (78%) had a deviation from centric relation (CR) to centric occlusion (CO). CR contacts occurred most commonly on first premolars. Bilateral group function was the most common excursive pattern (46%). Fifty-six percent (56%) had non-working contacts in lateral movements (33% in protrusive movements). Second molars exhibited the most non-functional contacts, followed by first molars, total of 75%.

Pihlstrom et al. (1986) examined maxillary first molars clinically and radiographically in 300 individuals 20 to 40 years of age for signs of trauma from occlusion, pattern of occlusal contracts, and severity of periodontitis; only the mesiofacial aspect was examined for probing depth and clinical attachment levels. Teeth with contacts in CR, working, non-working or protrusive positions did not exhibit any greater severity of periodontitis than teeth without these contacts. Teeth with wear facets or thickened lamina dura had less attachment loss and more bone support than teeth without these findings. Only 14 teeth demonstrated signs of traumatic occlusion (bidigital mobility, functional mobility, widened PDL space). These teeth also had greater probing depths, loss of attachment, and less bone support and higher gingival and calculus indices. Contrary to Kepic and O'Leary's (1978) findings, Pihlstrom et al. found that teeth with uneven marginal ridges had more attachment loss. Unfortunately, Pihlstrom et al. did not examine second molar teeth, the site where Shefter and McFall reported the highest prevalence of non-working contacts.

In an 8-year longitudinal study of 82 patients and 1,974 teeth, Fleszar et al. (1980) examined the relationships between tooth mobility and periodontal therapy. All patients received initial preparation, occlusal adjustment, one of three modes of surgical treatment and 3-month supportive periodontal treatment. There was a mean difference of 1.57 mm in attachment gain between Types 0 and 3 mobility for teeth with 7 to 12 mm pockets, 2 years postoperatively. Pockets of clinically mobile teeth did not respond as well to treatment as firm teeth with comparable initial disease severity; however, mobile teeth were successfully treated and maintained.

ANIMAL STUDIES

Rat Model

Stahl et al. (1957) studied the effects of vertical occlusal trauma in normal and protein-deprived rats. Trauma was induced by placing amalgam to the level of the cusps in a channel carved in first and second maxillary molars. Animals with the occlusal irritant and normal diet for 6 weeks showed changes, particularly in furcation areas, consisting of disorganization of fiber bundles, edema, and degeneration of fibers. Protein-deprived animals had similar but more severe changes.

Itoiz et al. (1963) used 3 different methods to create occlusal trauma in rats: amalgam placed in a channel carved in the occlusal surface of molars, cementation of a pinhead in the molar, and cementation of a stainless steel arch wire between the 2 upper first molars. Traumatic changes were analyzed in the opposite molars, using histologic and histometric methods. Trauma from occlusion resulted in areas of necrosis in the periodontal ligament, increase in bone resorption, and areas of cementum resorption in the furcation areas.

Glickman et al. (1966) studied in rats the effect of alloxan diabetes on the tissue response to increased occlusal forces produced by overfilling a continuous trench on the occlusal surfaces of upper right first and second molars. They concluded that alloxan diabetes aggravated and prolonged the effects of trauma from occlusion by inhibiting the reparative phase.

Dotto et al. (1967) studied the vascular changes resulting from occlusal trauma in rats. They used quantitative and histometric methods and described an increased vascularization which reached statistically significant values after 21 days of trauma and which persisted for the duration of the experiment (4 months). Blood vessels that normally occupy a position closer to the bone showed a displacement toward the cemental surface.

Primate Model

Zander and Mühlemann (1956) used 9 monkeys and placed a hollow screw-type device that applied a horizontal force on teeth. Six of the animals were subjected also to various systemic stresses. No differences were found between systemically stressed and non-stressed animals. All animals subjected to trauma showed necrosis and hyalinization of the periodontal ligament, osteoclastic activity in pressure zones, and new bone formation in tension areas.

Glickman et al. (1961) studied the effect of splinting teeth with and without occlusal interferences on the supporting structures in 5 adult monkeys. Animals were sacrificed after 10 to 132 days; the remaining animal was a control. The findings indicated that furcations were the areas most susceptible to occlusal trauma, while only slight

changes occurred in interdental areas. When an excessive force was applied to 1 tooth in a splint, the periodontal tissues of all splinted teeth suffered comparable injury.

Glickman and Smulow (1962) created excessive occlusal forces upon the periodontal tissues of 6 adult monkeys by constructing gold crowns in abnormal occlusal relationships. The animals were sacrificed after 10 to 132 days. The authors reported that excessive occlusal forces alter the pathway of gingival inflammation into the underlying periodontal tissues and affect the pattern of bone destruction. Excessive occlusal pressure was more significant than tension in determining the pathway of gingival inflammation. Injury to the periodontium induced by artificial alterations in the occlusion is reversible. Periodontal injury induced by attrition tended to persist.

Glickman and Smulow (1968) also analyzed the effect of chronic trauma in monkeys by combining a high gold crown to cause hyperocclusion with an orthodontic appliance and a spring to bring the tooth back to its original position when the teeth were apart. The experiment lasted 6 months. The authors mention that chronic trauma from occlusions occurs in 3 stages: injury, repair, and adaptive alterations.

Comar et al. (1969) placed cast gold crowns with occlusal interferences and gross marginal overhangs in 4 Rhesus monkeys, sacrificed at 5, 14, 21, and 98 days. This study failed to show a change in the pathway of inflammation under the influence of trauma from occlusion.

Kenney (1971) placed gold inlays which were "high" in centric and protrusive in upper incisors of 4 Rhesus monkeys. The animals had moderate gingivitis and were scaled 2 weeks prior to crown cementation. Histologic evidence of tramatic occlusion or orthodontic movement was seen in all animals; the inlays did not produce any change in the intensity or distribution of the inflammatory cells.

Pihlstrom and Ramfjord (1972) compared the periodontal status of non-functional teeth to functional teeth clinically, radiographically, and histologically in Rhesus monkeys. Non-functional teeth exhibited more plaque and gingival inflammation, increased loss of bony support, a narrowing of the PDL (within 3 months), and increased deposition of cementum.

The Eastman Dental Center (Polson et al., 1976A, 1976B, 1979; Polson and Zander, 1983; Polson, 1986) studies utilized squirrel monkeys to examine the role of occlusion in periodontal disease. Marginal periodontitis was initiated using silk ligatures to enhance plaque retention. Primarily horizontal, non-excessive jiggling forces were applied by placing orthodontic ligatures in alternating interproximal sites on a daily basis. The following questions were addressed in these studies: Does occlusal trauma cause periodontal disease? Does occlusal trauma influence the progression of periodontal disease? Is bone loss reversible when trauma is removed in a normal periodontium (adaptive changes) versus an inflamed reduced periodontium? Is bone loss reversible when inflammation is removed and trauma remains in an inflamed reduced periodontium? Is bone loss reversible when both inflammation and trauma are removed? Does trauma affect intrabony pockets any differently than suprabony pockets?

Polson (1974) produced a single episode of trauma subjacent to an established periodontitis. There was no difference in loss of connective tissue attachment and loss of alveolar bone between experimental and control teeth. Meitner (1975) examined the effect of jiggling trauma on marginal periodontitis. There was no difference in three of four pairs of surfaces examined, indicating that it was unlikely jiggling trauma had accelerated the loss of connective tissue attachment. Polson et al. (1976A), in a similar study of the normal periodontium, reported no loss of connective tissue attachment. There was some loss in alveolar crest height and considerable reduction in volume (40%) of interproximal bone. Subsequently, Polson et al. (1976B) showed these adaptive changes to be largely reversible when jiggling forces were withdrawn and areas allowed to heal. When jiggling trauma was removed from teeth with an inflamed but reduced periodontium, there was no decrease in tooth hypermobility and no bone regeneration, suggesting that bone regeneration may be inhibited in the presence of inflammation.

Kantor et al. (1976) removed both the jiggling forces and inflammation. No alteration in connective tissue attachment levels occurred but new bone formation did occur without an increase in alveolar bone height. When marginal inflammation is resolved where tooth mobility is due only to marginal periodontitis (no superimposed trauma), tooth mobility is significantly reduced (Polson et al., 1979). In the latter study, no coronal gain of connective tissue attachment or crestal alveolar bone level occurred, although bone density increased and PDL widths decreased.

Polson and Zander (1983) investigated the effect of trauma on surgically-created intrabony defects versus similar non-traumatized defects. Although a greater loss of bone volume occurred in the traumatized sites, no differences were observed in loss of connective tissue attachment. These findings concur with the earlier findings of Polson et al.

Dog Model

Glickman and Weiss (1955) induced trauma from occlusion in 6 dogs by means of cast onlays cemented to anterior teeth. Animals were sacrificed after 3 to 110 days. In spite of forces of sufficient severity to produce notable changes in the periodontal ligament and extensive resorption in the alveolar bone, they produced no deepening of the gingival sulcus and no change in the position of the junctional epithelium on the root. This investigation clarified a point debated at the time that was based on two papers (Box, 1935; Stones, 1938), now only of historical interest.

Goldman (1956) placed high crowns in a dog for 5 days.

After sacrifice and perfusion with india ink, he observed that forces strong enough to obliterate blood supply in the periodontal ligament did not affect gingival blood supply.

Utilizing a beagle dog model, the Gothenburg group addressed many of the same questions as the Eastman Dental Center group. Lindhe and Svanberg (1974) performed experiments in 6 beagle dogs that were fed a soft diet which facilitated dental plaque formation. During a pre-experimental period of 7 weeks, periodontitis was induced by 1) surgically creating a bony pocket and 2) adapting a copper band to the exposed root surface. Two dogs were sacrificed at the end of this period, and tissues prepared for histological examination. In the remaining 4 dogs, trauma from occlusion was produced on the mandibular left fourth premolar by the installation of a cap splint and a bar device. The contralateral premolar served as control. At the start of, and at regular intervals during an experimental period of 180 days, tooth mobility, gingival inflammation, and plaque accumulation were assessed. After sacrifice, radiographs were taken and the tissues prepared for histology. Only the test teeth showed a gradually increasing horizontal mobility, but gingival inflammation and plaque index were similar on both sides. Radiographs revealed 1) horizontal bone loss in both test and control areas and 2) angular bone destruction only in the test areas. Histological sections showed that the degree of apical proliferation of the pocket epithelium was more pronounced in test than in control regions.

In a subsequent experiment, Svanberg and Lindhe (1974) created trauma from occlusion in beagle dogs with and without previously-created experimental periodontitis. The dogs were sacrificed after 7, 14, 30, and 180 days. The findings indicated that dogs with a healthy periodontium differed in their reaction to a jiggling type of occlusal trauma when compared with dogs with an established periodontitis. Whereas the periodontal ligament in the former group had become adapted to the altered occlusion, that of the latter showed increased vascular leakage, leukocyte migration, and osteoclastic activity.

In 1976, Lindhe and Ericsson reported on experiments performed in 5 dogs fed a soft diet which allowed dental plaque accumulation. Experimental periodontal breakdown was introduced on day 0. After 180 days, experimental periodontitis was introduced in the mandibular fourth premolars. On day 280, the pockets around the fourth premolars were surgically eradicated, a notch placed at the bottom of the pocket, and trauma from occlusion removed from 1 of the 2 mandibular fourth premolars. From day 280 to 370, the teeth of the animals were brushed twice daily. The animals were then sacrificed. Results indicated that jiggling-type occlusal trauma and tooth hypermobility do not adversely affect healing following periodontal surgery.

Ericsson et al. (1977) demonstrated that it is possible to shift a supragingival plaque subgingivally by orthodontic tipping or apical movement, resulting in intrabony pocket formation.

Ericsson and Lindhe (1982) studied the effect of a prolonged period of jiggling force application on the rate of progression of ligature-induced, plaque-associated marginal periodontitis in the beagle dog. This investigation demonstrated that jiggling forces enhance the rate of periodontal destruction induced by the ligature and plaque, and, therefore, act as a co-destructive factor.

Ericsson and Lindhe (1984) showed that the degree of periodontal breakdown, initiated and maintained by ligature placement and plaque accumulation, was similar around teeth with a wide periodontal ligament space and teeth with a normal width. In other words, progression of plaque-associated lesions appeared to be unrelated to the width of the periodontal ligament space and to the degree of horizontal tooth mobility.

Lindhe and Ericsson (1982) removed jiggling forces superimposed on an experimental periodontitis and observed a reduction in tooth mobility and PDL width but no change in the periodontal lesion; i.e., no improvement in connective tissue attachment level.

Significant differences in experimental design between the Eastman and Gothenburg groups include the animal model, means of inducing periodontitis and occlusal trauma, and degree and direction of occlusal force.

Conclusions From Human and Animal Studies

1. Trauma from occlusion does not initiate gingivitis.
2. Trauma from occlusion does not initiate connective tissue attachment loss.
3. Occlusion may play a secondary role in the progression of periodontal disease.
4. Inflammation should be removed initially and potential occlusal factors subsequently reevaluated.
5. Healing following surgical treatment of periodontal disease may be more advantageous in non-mobile than in mobile teeth.
6. Tooth mobility is not necessarily synonymous with trauma from occlusion.

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Section 7. Habits (Factitial/Smoking)

DEFINITIONS

Habit: An act that has become a repeated performance, almost automatic, such as bruxism or tongue thrusting.

Factitious: Pertaining to a state or situation produced by other than natural means; self-inflicted.

FLOSSING CLEFTS

Fourteen (14) flossing clefts were observed in 10 patients ranging in age from 22 to 42 years. Diagnostic suspicions were confirmed after observing patients' flossing techniques. Of the 14 clefts, 2 were classified as acute and 12 chronic. Clefts were consistently associated with effective plaque control and probing depths ranging from 1 to 3 mm. All "injury sites" were asymptomatic with the exception of the 2 ulcerated lesions. Histologically, the depressions were lined with stratified squamous epithelium exhibiting a thin layer of parakeratin and chronic inflammatory cells dispersed throughout the underlying connective tissue. The morphology of the flossing clefts was not found to constitute an impediment to plaque control (Hallmon et al., 1986).

FACTITIAL INJURIES

Forty-nine (49) cases of self-inflicted gingival injuries have been reported in the literature. These are usually seen in children younger than 12 and more frequently involve females. Most reported injuries resulted from "scratching" or "picking" the gingiva with a fingernail. Features common to self-inflicted injuries are: 1) failure to correspond

to any known disease; 2) bizarre configuration with sharp outlines; 3) lesions within reach of patient's hands; and 4) may occur singly, but often multiple injuries may be seen (Stewart and Kernohan, 1972). Motivations reported include work avoidance, attention gathering, emotional or psychotic disorders and narcotics acquisition. A case was reported in which a 25-year-old man presented with gingival recession and mobility of the mandibular anterior teeth. After oral hygiene (OH) instruction and debridement, the condition of the mandibular incisors improved while similar lesions became evident in the maxillary premolar regions. After several appointments which included biopsy and consultations, it was ascertained that the patient was vigorously brushing and flossing for three 30-minute sessions daily. Once proper oral hygiene was applied, the lesions resolved (Pattison, 1983).

SMOKING

Arno et al. (1958) examined 1,016 individuals for gingivitis as it related to various factors including tobacco consumption. They found a significant correlation between tobacco consumption and gingivitis when hygiene and age were kept constant.

Arno et al. (1959) analyzed the relationship betwen tobacco consumption and radiographic bone loss in a group of 728 men aged 21 to 45. They reported that alveolar bone loss increased with increasing tobacco consumption.

Summers and Oberman (1968) analyzed the presence and prevalence of periodontal disease in 408 subjects 20 years of age and older and its relation to 12 pertinent social and physiological variables, including smoking. They found that periodontal disease was more severe in smokers for all age groups. The authors speculate that smoking may affect periodontal disease directly as a source of gingival irritation, and/or indirectly by affecting the gingival tissue response through vascular alterations. They also noted that smokers had poorer oral hygiene than non-smokers.

Bergström and Eliasson (1987) examined 235 subjects 21 to 60 years of age, with above average oral hygiene status and dental care habits; 72 were smokers. Alveolar bone height was significantly reduced in smokers, even after correcting for age and oral hygiene. The authors concluded that smoking is a risk factor for periodontal disease.

Sheiham (1971) studied two random samples of industrial workers in England and Northern Ireland. Examination incorporated the oral hygiene index and Russell's periodontal index. In both populations, smokers had more debris, calculus, and periodontal disease than non-smokers. Those who smoked 1 to 10 cigarettes per day had cleaner mouths and less severe disease than those who smoked more. Comparable levels of periodontal disease were observed in smokers and non-smokers with similar oral hygiene.

Ismail et al. (1983) reviewed data from the National Health and Nutrition Examination Survey (1974) to investigate the association between periodontal health and reported use of different types of tobacco products. Approximately 3,000 individuals responded to a smoking questionnaire and received a dental examination. Smokers had higher periodontal (PI), debris, calculus, and oral hygiene index scores than non-smokers. After controlling for age, OH, and other factors, the smokers had significantly higher PI scores than the non-smokers. No significant differences were found between the duration, type of tobacco, or the number of cigarettes smoked and the PI.

Baab and Oberg (1987) studied 12 periodontally healthy habitual smokers (19 to 25 years) to determine gingival and skin blood flow, heart rate, and blood pressure during active smoking. Laser Doppler fiberoptic probes were used to determine blood flow. Intraoral laser probes were placed 1 mm into the sulcus of a single molar or canine. Gingival blood flow increased, skin flow decreased, and blood pressure and heart rate increased. The study failed to confirm the findings of Clarke et al. (1981) which demonstrated decreased gingival blood flow after interarterial nicotine/epinephrine administration. It should be noted that the laser Doppler readings are sensitive to movement and may have been affected by lip/tongue movements during smoking.

Bergström and Preber (1986) induced experimental gingivitis in 20 dental students, 10 of whom were smokers. Plaque rate formation was similar in both groups, but smokers displayed a less pronounced inflammatory response. Danielsen et al. (1990) reported the same results in a similar study. These findings may indicate that smokers have a reduced capacity to mount and maintain an effective defense against the plaque challenge.

Preber and Bergström (1986) analyzed the effect of smoking on non-surgical periodontal treatment. In both smokers and in non-smokers, treatment reduced probing depth; however, the reduction was consistently greater in non-smokers.

Miller (1987) analyzed the factors associated with unsatisfactory results in cases of root coverage. The author reported a 100% correlation between failure to obtain root coverage and heavy smoking (more than 10 cigarettes/day). Light or occasional smokers (5 cigarettes/day or less) responded as favorably as non-smokers. Heavy smokers who refrained from smoking during the first 2 weeks of healing had results comparable to non-smokers.

Preber and Bergström (1990) studied the influence of cigarette smoking on the outcome of surgical therapy (Widman flap) in 54 patients with moderate to advanced periodontitis, of whom 24 were smokers. The authors found that smoking impaired the results of surgical therapy including a statistically significant difference in probing depth reduction at the 12-month follow-up.

Raulin et al. (1988) studied the in vitro effects of different concentrations of nicotine on human fibroblasts. Fibroblast attachment to glass and healthy extracted human teeth was evaluated after exposure to nicotine in concentrations of 25, 50, 100, 200, and 400 ng/ml. The lowest

concentration of nicotine (25 ng/ml) disrupted the attachment process, producing a haphazard orientation of the fibroblasts and surface characteristics of poorly-attached fibroblasts. Fibroblasts changed from well-attached cells with smooth surfaces with few microvilli and thin cytoplasmic extensions to cells with rough surfaces containing many blebs, microvilli, and broad cytoplasmic extensions that did not appear well-attached. Plasma levels of nicotine in tobacco users varied from 22 to 73 ng/ml.

Kraal and Kenney (1979) compared the ability of polymorphonuclear leukocytes (PMNs) from smokers and non-smokers to respond to chemoattractants. Twenty (20) healthy male subjects (10 smokers and 10 non-smokers) with no periodontitis were selected. Blood and saliva samples were collected at baseline and again 1 to 10 days later. The second sampling took place after both groups smoked one cigarette. PMNs and sera were isolated from blood samples of each subject. PMNs from each subject were tested for chemotactic activity against autologous untreated serum, treated serum and saliva in Boyden chambers. No difference was demonstrated in the ability of PMNs from smokers and non-smokers to react to the chemo-attractants and no difference was found between the ability of chemotactic agents from smokers and non-smokers to attract PMNs.

Goultschin et al. (1990) analyzed periodontal needs and the smoking habits of 344 hospital personnel. They reported that smoking and the number of cigarettes smoked had a clearly deleterious effect on the periodontal status and that younger women were more susceptible to this effect.

Haber et al. (1993) analyzed the role of smoking as a risk for periodontitis in diabetic and non-diabetic patients. Among non-diabetics, the prevalence of periodontitis was markedly higher in current smokers as compared to never smokers. The effect of smoking among IDDM subjects was similar to that observed in the non-diabetic group.

Stoltenberg et al. (1993) studied a group of 615 adults and concluded: 1) after matching for age, sex, plaque, and calculus, the odds of having a mean posterior proximal probing depth equal to or greater than 3.5 mm were 5.3 times greater for smokers than for non-smokers; 2) the prevalence of *P. gingivalis*, *A. actinomycetemcomitans*, *P. intermedia*, *E. corrodens*, and *F. nucleatum* does not differ between smokers and non-smokers; and 3) *A. actinomycetemcomitans*, *P. intermedia*, *E. corrodens*, and smoking were each associated with increased mean posterior proximal probing depth. However, cigarette smoking was a stronger risk indicator for the presence of a mean probing depth equal to or greater than 3.5 mm than any of the 5 bacteria commonly associated with periodontal disease.

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Section 8. Systemic Factors

DIABETES MELLITUS

Gingivitis

Gusberti et al. (1983) studied 77 insulin-dependent diabetes mellitus (IDDM) children, aged 6 to 15, and measured blood glucose levels (via glycosylated hemoglobin and fasting blood sugar) and the incidence of gingivitis. Prior to puberty, poorly-controlled diabetic patients with increased blood glucose had a higher incidence and severity of gingivitis than controlled diabetics. During puberty, there was a general increase in gingivitis independent of the blood

glucose levels. This suggested that before puberty, altered glucose metabolism can enhance the severity of gingivitis.

Cianciola et al. (1982) studied the prevalence of periodontal disease in 263 IDDM patients who were compared to 108 age-matched controls. The population ranged in age from 4 to 33 with most of the patients aged 4 to 18. These authors found that there was a significant increase in gingivitis after age 11 in IDDM patients which was greater than the non-diabetic controls. They also noted that granulation tissue may exude from the gingival crevice in IDDM patients with severe periodontitis.

Ervasti et al. (1985) examined 50 IDDM adult patients who were matched by age, sex, and social class with a control group. They reported no significant difference between the diabetic group as a whole and the control group. However, when diabetic patients were grouped into well-controlled, moderately well-controlled, and poorly-controlled patients, the authors observed several trends. Well-controlled diabetics had significantly less gingival bleeding than the control population while poorly-controlled diabetics had significantly more gingival bleeding than well- or moderately-controlled diabetics or the control population. In general, the number of non-bleeding surfaces diminished as the control worsened, with control of diabetes one of the most prognostic independent variables. These studies indicate that the level of diabetic control is the important factor in the level of gingivitis, with well-controlled diabetics apparently responding in a manner similar to the normal population.

Periodontitis

Glavind et al. (1968) studied controlled diabetics, aged 20 to 40, and compared them to a normal population. The investigators found no difference in the populations up to age 30; however, patients older than 30 or patients who had been diagnosed with diabetes longer than 10 years had significantly more periodontal attachment loss. Patients with vascular changes in the retina also had more attachment loss.

Cohen et al. (1970) compared diabetic and non-diabetic patients over a 2-year period and consistently observed more gingival inflammation and attachment loss in the diabetic patients at each examination. Although both groups had soft deposits on the teeth, the amount present in the diabetic group was less.

Sznajder et al. (1978) examined 83 diabetics and 65 non-diabetics 9 to 50 years of age, and reported increased attachment loss in diabetics over 30 years old, with no differences in plaque and calculus between diabetics and non-diabetics.

Cianciola et al. (1982) demonstrated increased bone and attachment loss (periodontitis) in IDDM patients, compared to controls (siblings of the diabetics and non-related, non-diabetic patients), despite comparable plaque indices. In the IDDM patients, there was a sharp increase in periodontitis observed at age 13. From age 4 to 12, no periodontitis occurred in the IDDM patients. From age 13 to 18, 11.3 to 16% of the IDDM patients demonstrated periodontitis. This incidence rose to 39% for patients > 19. Overall, among subjects between 11 to 18 years old, periodontitis was found in 9.8% of the IDDM patients compared to 1.7% of the control patients. The authors found that the pattern of bone loss was initially similar to that seen in LJP and was related to the chronological age of the patient rather than the duration of the diabetes.

Tervonen and Knuutila (1986) reported no difference in the level of periodontitis when comparing well-controlled diabetics to non-diabetic controls. However, poorly-controlled diabetics demonstrated increased loss of attachment and alveolar bone.

Novaes et al. (1991) compared the periodontal status of 30 Brazilian IDDM patients ages 5 to 18 with non-diabetic controls. They concluded that: 1) a statistically higher accumulation of plaque occurs among the diabetic patients (1.23 versus 0.81), among female diabetics (1.34 versus 1.10), and among older patients (difference significant at the 5% level); 2) the gingival index was higher among diabetics than controls (0.52 versus 0.15), with no significant difference with respect to age and sex; 3) probing depth did not differ significantly between diabetics and controls in relation to increasing age, but in relation to sex, diabetic females showed deeper pockets in the palatal region; and 4) alveolar bone loss was significantly greater in diabetics than in non-diabetics in the upper and lower anterior region.

Emrich et al. (1991) studied the relationship between diabetes mellitus and oral health in 1,342 Pima Indians from the Gila River Indian Community in Arizona, which has the world's highest reported incidence and prevalence of NIDDM (Type 2) diabetes mellitus. The authors found that diabetes increases the risk of developing destructive periodontal disease about threefold. This increased risk cannot be explained by age, sex, hygiene, or other dental measures.

Seppälä et al. (1993) evaluated the progression of periodontal disease in subjects aged 35 to 55 years with long-standing, insulin-dependent diabetes mellitus. Regarding age, sex, type, and duration of diabetes, environment, and nutrition, the participants of this study constituted a homogenous group. They report that under similar plaque conditions, poorly-controlled diabetics have more gingivitis, more bleeding on probing, greater loss of attachment, and more bone loss than well-controlled subjects.

In contrast to the above studies, Barnett et al. (1984) examined forty-five 10- to-18-year-old diabetic patients and found no correlation between the degree of diabetic control or the duration of diabetes compared to either the gingival index or the periodontal index. While this study conflicted with previous studies, it should be pointed out that none of the patients in this study exhibited any loss of interproximal bone, thus making comparison with previous studies difficult. In addition, these subjects were a rather homogeneous

group from a private endocrinology practice and were probably very compliant.

Possible Etiologic Factors

Vascular changes. Degenerative vascular changes seen in other tissues are also seen in the gingiva; i.e., increased thickness of the basement membrane and vessel walls. It is postulated, though not proven, that these changes interfere with the delivery of nutrients to the tissues with the resulting decreased oxygen diffusion and decreased elimination of metabolic wastes contributing to an increase in the severity of periodontitis and a decrease in wound healing (Frantzis et al., 1971).

Collagen breakdown. Increased collagen breakdown (through the stimulation of collagenase activity) and altered collagen metabolism (decreased collagen synthesis; altered collagen maturation) are felt to be present in diabetic patients. These defects may be endogenous since diabetics have been found to produce an increase in gingival collagenase activity under germ-free conditions. The above defects may contribute to impaired wound healing and an increased severity of periodontitis in the diabetic patient (Golub et al., 1983; Ramamurthy and Golub, 1983). Golub et al. (1983) found that the administration of minocycline reduced collagenolytic activity 62% in a conventional rat population and by 70% in a germ-free diabetic rat population. In humans, minocycline caused significant reductions in the gingival index, gingival crevicular fluid, active and total collagenase activity, and Gram-negative organisms. It was felt that the minocycline reduced the breakdown of intact collagen and inhibited the production of collagen digestion fragments.

Altered oral microbial flora. Studies have shown that the microbial flora is different in the diabetic. In IDDM patients with periodontal disease, *Capnocytophaga* sp. is thought to be the predominant organism (Mashimo et al., 1983). This alteration in the microbial flora may lead to the increase in periodontal disease.

Altered defense mechanism. Polymorphonuclear leukocytes (PMNs) functions, such as chemotaxis and phagocytosis, have been shown to be decreased in diabetic patients with periodontal disease (Manouchehr-Pour et al., 1981; Manouchehr-Pour and Bissada, 1983; Leeper et al., 1985). These defects may, in fact, be genetic. Leeper et al. (1985) demonstrated that normal siblings of diabetic patients demonstrate decreased PMN chemotaxis. In addition, these authors noted that decreased PMN chemotaxis was more pronounced in poorly-controlled diabetics. This defect in the body's immune system may predispose the diabetic to periodontal disease.

Increased glucose has been found in the gingival crevicular fluid (GCF). This alters the environment and may allow the growth of different subgingival bacteria and/or alter PMN function (Kjellman et al., 1970).

Effects of Periodontitis on Diabetes

It is known that the presence of acute infection makes diabetic control more difficult. Similarly, it is postulated that the presence of periodontal disease may exacerbate the clinical symptoms of diabetes and make diabetic control more difficult.

Williams and Mahan (1960) reported a significant reduction in insulin requirements in 7 of 9 patients with diabetes and periodontal disease who underwent periodontal therapy. As such, diabetes may be adversely influenced by periodontal disease and conversely, periodontal disease may be more severe in the diabetic patient. This is important in that periodontal therapy may alter the patient's insulin requirement, thus requiring an adjustment in the insulin dosage. Therefore, it is important to inform the patient's physician prior to initiating periodontal therapy.

In contrast, Parrish (1985) found that treatment of periodontal disease did not alter the patient's insulin requirements. It is possible that during periodontal therapy, the poorly or minimally-compliant diabetic patients may take a greater interest in their overall health and become more compliant with their diabetic treatment.

LEUKEMIA

DEFINITION

Leukemias: Malignant neoplasms of the hematopoietic stem cells, characterized by diffuse displacement of the bone marrow by neoplastic cells. In most cases, the leukemic cells spill over into the blood, where they may be seen in large numbers. These cells may also infiltrate the liver, spleen, lymph nodes, and other tissues throughout the body (Robbins and Kumor, 1987).

Classifications

Leukemias are classified as acute or chronic and according to the cell type involved.

Acute Leukemias: Immature cells, with a rapidly fatal course.

Chronic Leukemias: Relatively well-differentiated leukocytes and prolonged course.

Lymphocytic and Myelocytic Leukemias: These refer to the cell types involved and can be acute or chronic.

Monocytic Leukemia: This is an extremely rare form of the disease.

There are 8 to 10 new cases of all leukemias per 100,000 population each year, divided as follows: acute mylogenous leukemia (AML), 46%; chronic lymphocytic leukemia (CLL), 29%; chronic myelocytic leukemia (CML), 14%; and acute lymphocytic leukemia (ALL), 11%. Leukemias represent 3% of all cancers in the United States.

Oral Manifestations: Oral manifestations are very rare in chronic leukemia, and the following descriptions refer almost exclusively to acute leukemia.

Barrett (1984) classified the gingival lesions in acute leukemia as follows:

1. Direct infiltration
2. Direct drug toxicity
   A. Erosion/ulceration
   B. Epithelial retention
   C. Connective tissue hyperplasia
3. Graft versus host disease
4. Secondary to marrow/lymphoid tissue depression
   A. Hemorrhage
   B. Neutropenic ulceration
   C. Infections
      a. Viral
      b. Fungal
      c. Bacterial

Leukemic Infiltration of the Periodontium: Dreizen et al. (1983) studied 1,076 leukemic patients and found that 66 (6.1%) had leukemic infiltrates in the skin and/or gingiva. In 33 of these 66 patients (50%), lesions were confined to the gingiva, 28 (42.4%) had only skin lesions, and 5 (7.6%) had both. Gingival infiltrates were found in 38 (3.6%) of the 1,049 dentulous patients and in none of the 27 edentulous patients. Patients with acute monocytic leukemia (AMoL) had the highest incidence of gingival infiltrate (66.7%), followed by those with acute myelomonocytic leukemia (AMML) (18.5%), and those with acute myelocytic leukemia (AML) (3.7%).

Microscopically, the gingiva exhibits a dense, diffuse infiltration of predominantly immature leukocytes in the attached and marginal gingiva. The periodontal ligament may also be infiltrated with mature and immature leukocytes (Carranza, 1990).

Carranza et al. (1965) and Brown et al. (1969) investigated the periodontal changes in AKR mice, which develop leukemia spontaneously. They reported the presence of infiltrate in marrow spaces and in the periodontal ligament, resulting in osteoporosis of the alveolar bone with destruction of the supporting bone, disappearance of the periodontal fibers, and tooth exfoliation.

Bleeding. Gingival bleeding is a common finding in leukemic patients. Bleeding occurs secondary to thrombocytopenia as a result of the replacement of the bone marrow by leukemic cells. Lynch and Ship (1967) reported oral bleeding aas a presenting sign in 17.7% of patients with acute leukemia and in 4.4% of patients with chronic leukemia.

Oral ulcerations and infections. Viral, fungal, and bacterial infections can occur in the oral mucosa owing to the lowered tissue resistance caused by the granulocytopenia that results from the leukemic replacement of bone marrow cells. These can include exacerbation of existing gingivitis or periodontitis as well as acute necrotizing ulcerative gingivitis.

Periodontal management of leukemic patients. Periodontal treatment in leukemic patients should be modified because of the enhanced susceptibility to infection, increased bleeding tendency, and effects of drugs that the patient may be receiving.

Consultation with the hematologist is needed in order to determine the treatment plan. Acute leukemia patients should receive only emergency treatment, while attempting to avoid tissue injury. Antibiotics; other microbials, such as chlorhexidine rinses; and antifungal agents should be used if needed. Patients with chronic leukemia and those in remission can receive scaling and root planing but periodontal surgery should be avoided. If treatment is absolutely necessary, bleeding time should be taken on the day of the intervention and the procedure postponed if results are low (Otomo-Corgel, 1990).

PREGNANCY/ORAL CONTRACEPTIVES

Management of the Pregnant Patient

While pregnancy is not a disease state, special considerations in the dental management of these patients are required. Physiologic changes include increases in heart rate, cardiac output, red cell mass, respiratory vital capacity, oxygen consumption and respiratory rate. Increased energy demands by the fetus and increased snacking may elevate the mother's insulin requirements, unmasking a prediabetic state. The safety of the developing baby is also of concern and treatment should be planned for times when the fetus is least affected. Because organogenesis occurs mainly in the first trimester, most developmental defects take place during this time. Most medications appear to cross the placental barrier and ingestion of materials (drugs) by the mother is the second most common cause of teratogenesis (Gier and Janes, 1983). CNS depression with narcotic use and spontaneous abortions following nitrous oxide administration have been reported. Non-steroidal anti-inflammatory drugs may interfere with closure of the ductus arteriosus if taken during the third trimester.

Additionally, tetracycline, vancomycin, and streptomycin should be avoided because of staining of teeth (fourth to ninth month), and ototoxic/nephrotoxic effects. Erythromycin, penicillin, and cephalosporins are considered safe, but consultation with the patient's obstetrician is recommended before prescribing any drug. As the fetus continues to grow, the mother's bladder and abdominal vessels are impinged upon and the diaphragm displaced upward causing decreased respiratory volume. While emergency treatment can be accomplished any time during the pregnancy, the second trimester is considered the best time to render treatment since organogenesis is complete and the mother is not as uncomfortable as during the first and third trimesters (Chiodo and Rosenstein, 1985). Radiographic exposure to the fetus is zero if proper technique and equipment is used (Alcox, 1978).

Effect of Pregnancy on the Periodontium

Estrogens, progestins, and gonadotropins interrelate to maintain the menstrual cycle. The main function of estro-

gens during the reproductive cycle is to faciliate cellular proliferation of the stromal cells, glands, and blood vessels of the endometrium (Zachariasen, 1989).

Progesterone has the opposite effect, increasing vascular permeability, PMNs in the gingival sulcus, and prostaglandin E-2 (Kalkwarf, 1978). The pattern of pregnancy gingivitis seems to follow the hormonal cycle. It initially increases with rising gonadotropin levels, is maintained from the fourth to eighth month (with rising estrogen and progesterone levels) and falls off in the last month with the abrupt decrease in hormone secretion (Löe and Silness, 1963). O'Neal (1979) compared gingival and plaque index scores to plasma levels of estradiol and progesterone for 26 subjects at weeks 14 and 30 of gestation. He reported that plaque scores decreased, gingivitis scores increased, and that other hormone levels rose. However, a direct association between the hormone levels and gingival changes could not be demonstrated.

Kornman and Loesche (1980) also studied the effect of pregnancy using 20 subjects and 11 controls. Monthly examinations were performed to assess the plaque index, gingival index, and bleeding score. Subgingival plaque samples were taken with a curet and sonicated prior to culturing. They found that during the second trimester there was an increase in gingivitis and gingival bleeding without an increase in plaque levels. The ratio of bacterial anaerobes to aerobes and the proportions of *B. melaninogenicus, P. intermedia* (2.2% to 10.1%), and *Porphyromonas gingivalis* (Pg) increased. Plaque uptake of steroids and *B. melaninogenicus* increased during the third trimester. The authors suggest that estradiol or progesterone can substitute for menadione (vitamin K) as an essential growth factor for *P. intermedia*, but not *P. gingivalis* or *B. coherences.*

In 1981, Jensen et al. studied the effect of hormone levels on the gingival status of a larger group of females. Participants included 54 pregnant, 23 non-pregnant on oral contraceptives, and 27 non-pregnant subjects. Results revealed that the pregnant group had a 2 to 3 times higher mean gingival crevicular fluid flow than the non-pregnant groups. No difference was observed between the two non-pregnant groups. The pregnant group also had higher GI scores than either non-pregnant group. The pregnant group had a 55 times greater recovery rate for *Bacteroides* species compared to the non-pregnant group. The non-pregnant group on oral contraceptives had a 16 times greater increase compared to the non-pregnant group.

Oral Contraceptive Therapy

Kalkwarf (1978) conducted a cross-sectional study of 168 females on various birth control pill (BCP) formulations. He observed that those taking oral contraceptives had significantly higher gingival inflammation levels and significantly less oral debris. Responses varied among different brands, with some producing more exaggerated effects. No correlation was noted between gingival inflammation, debris levels, and duration of time that the subject had been taking BCPs. He suggested that the periodontal effects might have been due to 1) alteration of the microvasculature; 2) gingival permeability; or 3) increasing synthesis of prostaglandins.

Some medications can interfere with the desired effect of BCPs and allow pregnancy. Rifampin induces hepatic enzymes responsible for the metabolism of steroid contraceptives and also causes an increase in plasma sex-hormone binding-globulin capacity, making less free steroid available. Ampicillin, other penicillins, and tetracycline interfere with BCPs via a different mechanism. Steroid contraceptives are conjugated in the liver and excreted back into the intestines in bile. Bacterial enzymes hydrolyze the conjugates and free the steroids allowing reabsorption. The newer lower level estrogen and progesterone pills rely on this reabsorption of the respective steroids from the intestinal tract to maintain adequate blood levels. The antibiotics listed above suppress the intestinal flora that produce the hydrolytic enzymes. This interferes with reabsorption resulting in lower inadequate blood levels of steroids. It is imperative that the patient be advised to use alternative means of birth control during periods of therapeutic or prophylactic exposure to the noted antibiotics (Barnett, 1985).

DIET AND NUTRITION

DEFINITION

Malnutrition: Any disorder of nutrition; it may be due to unbalanced or insufficient diet or to defective assimilation or utilization of foods (Dorland's, 1988).

The Recommended Dietary Allowance (RDA) is determined by the Food and Nutrition Board of the National Academy of Sciences (National Research Council) and is designed for the maintenance of good nutrition of practically all healthy people in the United States. The RDA exceeds the minimum daily requirement.

EFFECT OF NUTRITION ON THE PERIODONTIUM

Vogel et al. (1984) described 4 ways that the pathogenesis of periodontal disease could be influenced through nutrition:

Immune and inflammatory process. Subclinical vitamin C or iron deficiencies can cause defects in PMN function. Iron deficiencies can also cause macrophage dysfunction. Zinc regulates the inflammatory process by stabilizing membranes and decreasing lysosomal and histamine release.

Bone metabolism. The $Ca:PO_4$ ratio is 1:2.8 in a large segment of the population (ideal 1:1). The association between a decreased $Ca:PO_4$ ratio and increased alveolar bone loss in the presence of inflammation is contradictory.

Collagen metabolism. Vitamin C, iron, and zinc play a role in collagen metabolism. Deficiencies can result in decreased resistance of gingival tissue to plaque.

Epithelial barrier function. Subclinical deficiencies of

protein, zinc, folic acid, vitamin C, or iron may cause a reversible increase in permeability.

DePaola (1984) noted that end-organ deficiencies could impair the repair process. However, supplementation with vitamin C or folic acid did not prevent or arrest periodontal disease.

Fermentable carbohydrates do not appear to play a significant role in periodontal disease; however, they should be of concern to the periodontist, due to their role in root caries (Robinson, 1984).

Vogel and Wechsler (1979) compared 4-day nutrition surveys of 35 periodontitis patients and 1,222 general population subjects. They found that the means for both groups were above the recommended daily allowances (RDA). The calcium-phosphate ratio for the periodontal group was 0.62 (ideal = 1) and 13 of the 35 had deficient calcium intake. The daily dietary intake of periodontitis patients was not significantly different from the general population.

VITAMIN C

Cotran et al. (1989) indicated that most mammals and some amphibians and reptiles can synthesize ascorbic acid from glucose via glucuronic acid since they possess gluconolactone oxidase. Humans, as well as other primates, lack this oxidase and cannot synthesize ascorbic acid. Ascorbic acid is present in milk, some animal products (e.g., liver and fish), and is abundant in many fruits and vegetables.

A deficiency in vitamin C produces scurvy. With the abundance of ascorbic acid in so many foods, scurvy is not a world problem. However, it may be found in affluent populations as a conditioned deficiency, particularly among the elderly, those who live alone, alcoholics, and others who have erratic and irregular eating patterns. It may appear in infants maintained on formulas of processed milk without adequate supplementation.

Scurvy results in the following symptoms, which are more marked in children than in adults: purpura and ecchymosis of skin, most prominently along the back of lower legs, and in the gingival mucosa. Loose attachment of the periosteum and the hemorrhagic diathesis leads to extensive subperiosteal hematomas and bleeding into the joint space. Skeletal changes occur mainly in children and consist of a primary disturbance in the formation of the osteoid matrix (not mineralization, as in rickets).

Gingival swelling, hemorrhages, and secondary bacterial infection are commonly seen in advanced scurvy, but the deficiency only predisposes to the bacterial infection. Wound healing and localization of focal infections are both impaired in scurvy. Anemia is also a common finding.

Glickman (1948A) placed 16 guinea pigs on a vitamin C-free diet; 9 were used as controls. Animals were sacrificed after 35 days. Histological findings showed that gingival inflammation was not a prominent finding and was not increased in the controls or experimentals. Pockets, when present, were deeper in experimentals. Edema, hemorrhage, and collagen were seen in the periodontal ligaments of vitamin C-deficient animals.

In a subsequent paper, Glickman (1948B) induced gingival inflammation in vitamin C-deficient and control animals and reported an exaggerated periodontal destruction in the deficient animals, due to inability to form a peripheral limiting connective tissue barrier, reduction in inflammatory cells, diminished vascular response, and inhibition of fibroblast formation and differentiation to form osteoblasts.

Waerhaug (1958) placed 4 monkeys on a vitamin C-deficient diet for 42 to 257 days. Clinical signs of scurvy appeared after 90 days. Histologically, after 257 days, the monkeys had nearly complete destruction of the fibers of the periodontal ligament, particularly those inserted into the bone, but not as much as those inserted into cementum, increased osteoclastic resorption, and hemorrhages. Fibers located above the alveolar crest and below the epithelial attachment were the least affected. Tooth loss occurred only at late stages.

Woolfe et al. (1980) reviewed the relationship between ascorbic acid and periodontal disease. The following possible etiological relationships were noted:

1. The periodontium contains a significant amount of collagen, which is constantly being broken down and resynthesized. Insufficient levels of ascorbic acid influence collagen metabolism and the ability of the tissue to withstand insult and repair itself. However, no experimental evidence to support this concept has been reported.

2. Bone changes in ascorbic acid deficiency occur very late in the deficiency state. Furthermore, osteoporosis in the scorbutic monkey is not found in association with periodontal pockets.

3. Ascorbic acid deficiency increases the permability of the oral mucosa.

4. Ascorbic acid in vitro enhances PMN chemotaxis but does not influence its phagocytic activity. The importance of this is not clear.

5. Ascorbic acid is necessary to maintain the integrity of the intercellular cement substance of capillary walls.

6. Vitamin C deficiency may alter the ecologic equilibrium of plaque bacteria, although this has not been proven.

7. Human studies have not shown a correlation of vitamin C levels and disease incidence or severity.

A subsequent study by Woolfe et al. (1984) analyzed the effect of megadoses (1 gm) of ascorbic acid daily on gingival clinical parameters and the vitamin content of blood and gingival tissues. Ten non-deficient individuals, carefully matched according to age, periodontal status, and oral hygiene level, received either the vitamin or a placebo. After 1 week, all patients were scaled and root planed and given oral hygiene instructions. After 2, 6, and 7 weeks, blood samples and clinical parameters were obtained. A gingival biopsy was taken at week 6.

Correlations between the clinical parameters and ascor-

bic acid levels at the different time periods revealed no significant differences between the vitamin and placebo groups. This suggested that the use of megadoses of vitamin C in normal human subjects does not have a predictable or strong effect on the gingival response to periodontal therapy.

AGING FACTORS INFLUENCING THERAPY

The treatment decision relative to the patient's periodontal needs should not be based on age alone. The relative state of health is more important than the chronological age. Unless there is a contraindication, the health care provider is obligated to provide the elderly patient with the same state of the art treatment offered to younger patients. Only the patient has the right to make treatment decisions based on their longevity (Greenwell and Bissada, 1989).

EFFECT OF AGE ON THE PERIODONTIUM

Using 4 cadaver jaws of increasing age, Grant and Bernick (1972) studied the effect of age upon the periodontium. They found that cellularity decreased and collagen fiber coarseness increased. Tensile strength increased while thermal contraction, the ratio between ground substance and collagen, collagen turnover, and water content all decreased.

Van der Velden (1984) reviewed the literature and reported the following changes in the periodontium may accompany aging: There is a gradual breakdown of the periodontium with age which may relate to either age or the cumulative effect of longer exposure to periodontitis. The epithelium becomes thinner, less keratinized, and shows increased cell density. The connective tissue becomes denser, coarsely textured, and exhibits fewer cellular elements. The PDL shows less fiber and cellular content and becomes irregular. With age, there is an increase in the width of the cementum, with cementum formation being essentially cellular. The periodontal surface of the bone becomes more jagged, collagen fibers insert less regularly, there are more interstitial lamella and fewer cells in the osteogenic layer. The width of the PDL appears to decrease with age and unchanged function.

Van der Velden et al. (1985) investigated the rate of development of experimentally induced gingival inflammation in relation to the susceptibility to periodontal disease. By selection according to age, a younger (25 to 39 years) and an older (45 to 54 years) age group, with a comparable reduced but healthy periodontium was studied. This equal amount of periodontal breakdown may suggest that the younger age group represented individuals with a relatively higher degree of susceptibility to periodontal disease. At the start of the experiment, each patient was instructed to abstain from oral hygiene procedures in one quadrant of the mouth for a period of 18 days. Results showed that all subjects developed signs of gingival inflammation. Regarding the development of redness and swelling, no differences could be assessed between the two age groups. However, analysis of the bleeding scores revealed that bleeding on probing developed more rapidly in the younger age group. It was concluded that those patients who have suffered from a more rapid form of periodontal disease also develop inflammation (bleeding on probing) more rapidly.

TREATMENT RESPONSE

Lindhe et al. (1985) studied the effect of age on healing following periodontal surgery and found no significant difference between 3 age groups (< 40, 40 to 49, > 49) regarding dimensional alteration of the dentogingival tissues at buccal or interproximal sites. Similar changes in attachment levels, probing depths, and degree of gingivitis were observed for all age groups.

Robinson (1979) recommends more frequent recall appointments for elderly patients due to increased recession and greater amounts of exposed cementum. He suggests oral prophylaxis twice a month for the first 3 months followed by a 3-month recall program.

Picozzi and Neidle (1984) indicated that aging alters the patient's response to pharmacologic agents. Geriatric pharmacology is complicated by multiple disease states, multiple medications, non-compliance, and altered pharmacodynamics and pharmacokinetics. While absorption is not altered, distribution is affected by a decrease in total body water, increased fat:lean ratio and decline in plasma proteins. Metabolism may be altered by liver disease. Renal excretion might be impaired and kidney function begins to decline at about age 40.

DRUG-INDUCED GINGIVAL HYPERPLASIA

DEFINITION

Gingival Hyperplasia: An enlargement of the gingiva due to an increase in the number of cells.

Carranza (1990) classified gingival enlargements as inflammatory, non-inflammatory or fibrotic, combined, conditioned (hormonal, nutritional, blood diseases, idiopathic) neoplastic, and developmental. Drug-induced gingival enlargements are either non-inflammatory or combined. The three major drugs or classes of drugs implicated in this process are phenytoin, cyclosporin, and the calcium channel blocking agents such as nifedipine.

Phenytoin. Phenytoin was introduced in 1938 for the control of epileptic seizures. Kimball (1939) was among the first to report gingival overgrowth associated with phenytoin, reporting that 57% of patients taking the drug had gingival overgrowth. It has since been estimated that of the 2 million individuals taking phenytoin, approximately 40 to 50% will develop gingival overgrowth to some extent (Butler et al., 1987). Overgrowths appear to be more common in children and young

adults, with no predilection for gender or race. Rates of occurrence appear unaffected by the duration of treatment.

Angelopoulos (1975) reviewed the literature and described the clinical and histologic appearance of phenytoin-induced gingival overgrowth. Clinically, in the vast majority of cases the first sign is an enlargement of the interdental papillae. Less commonly, the marginal gingiva may begin increasing in size. Gradually, gingival changes become more prominent, and enlargement takes the form of coalescent lobulations representing the hyperplastic papillae and extending labially and, less often, lingually. Lobulations are usually separated by a small cleft and commonly exhibit partial coverage of the anatomical crown to varying degrees. Overgrowths are localized to the anterior regions in a majority of cases, and the degree of overgrowth is most marked in anterior areas. Vestibular gingiva is more commonly affected than lingual gingiva and it is generally agreed that there are no clinical signs of overgrowth in edentulous regions. In uncomplicated phenytoin-induced overgrowth, the tissue has a normal pink color and is hard, firm, resilient, and rubbery. It may also be stippled and have a granular or smooth appearance and may not bleed easily.

Secondary inflammation resulting from plaque retention and other local irritants may cause these areas to become dark red, edematous, spongy, and friable. Histologically, in the majority of cases there are chronic inflammatory cells, mainly lymphocytes and plasma cells. The overlying epithelium is characterized by thin, elongated rete ridges and acanthosis. Basic changes in the connective tissue are a proliferation of fibroblasts and increased formation of collagen fibers. The amount of ground substance has also been reported to increase, and is associated with fibroblastic activity.

Dahllof et al. (1984) studied the volume density of various connective tissue components in phenytoin-induced overgrowths and reported a significant increase in the non-collagenous matrix and a corresponding decrease in the collagenous matrix of overgrowth tissue as compared to control specimens. The density of cells in both groups was the same (the specimens examined did not contain a significant inflammatory infiltrate due to rigorous plaque control prior to biopsy). Because the non-collagenous matrix is composed of glycosaminoglycans (GAGs), the authors proposed that increased synthesis of GAGs which bind large amounts of water and thereby occupy large volumes in the tissue might explain observed increases in volume of the non-collagenous matrix compartment.

In summary, when compared to normal gingiva, phenytoin-induced gingival overgrowths demonstrated a relative decrease in epithelium, increases in connective tissue and inflammation, and no change in vascularity. This indicates the main changes involve the connective tissue and inflammatory components rather than epithelium; however, one must remember the histopathological findings in this condition are by no means specific or pathognomonic.

While the exact mechanism of this process remains unknown, various theories have been proposed. The risk factor most associated with gingival overgrowth is poor oral hygiene. Seymour et al. (1987) found significant correlation between plaque score and gingival overgrowth in epileptic patients on phenytoin therapy. Pihlstrom et al. (1980) confirmed that a preventive program of frequent prophylaxis and oral hygiene reinforcement was effective in minimizing gingival enlargements. Although they further concluded that plasma and salivary levels of phenytoin were not correlated with the minimal degree of overgrowth observed in their study, there are conflicting reports regarding the association of dose and serum levels of phenytoin and the severity of gingival overgrowth.

Others have proposed the existence of fibroblast subpopulations which preferentially proliferate in response to phenytoin ingestion. Hassell (1982) and Hassell et al. (1986) demonstrated that fibroblasts from overgrowth lesions exhibited enhanced protein synthesis and produced inactive collagenase, thereby creating a net connective tissue overgrowth in affected individuals. Sooriyamoorthy and Gower (1989) evaluated the effect of phenytoin on the metabolism of testosterone by human gingival fibroblasts and reported an increased number of receptors for 5-a-dihydrotestosterone on fibroblasts removed from hyperplastic tissue. Since phenytoin stimulates the conversion of testosterone to 5-a-dihydrotestosterone, it may provide a metabolic pathway to enhance gingival growth.

Mallek and Nakamoto (1981) reviewed the role of folic acid and its relation to phenytoin-induced overgrowth. Among the side effects of anticonvulsant drugs in general, and dilantin specifically, is a decreased serum level of folic acid. The authors suggest that because folic acid metabolites play a complex role in regulating the cell division of human skin fibroblasts, it is possible that a deficiency of folic acid could alter this regulation and thereby cause uncontrolled proliferation of fibroblasts and their products.

Backman et al. (1989) and Drew et al. (1987) reported beneficial effects following the administration of folic acid. Finally, the role of genetics in this condition has recently received considerable attention.

Dill et al. (1993) note that phenytoin increases macrophage production of platelet-derived growth factor (PDGF), an important cytokine in connective tissue growth and repair, and that excessive production of PDGF could lead to redundant growth.

Cyclosporin. Cyclosporin is a powerful immunosuppressant agent primarily used to prolong the survival of allogeneic transplants in humans. The proposed mechanism of action is specific and reversible inhibition of immunocompetent lymphocytes (particularly T-cells). The incidence of gingival overgrowth varies from study to study, with a range of between 25 to 81% (Seymour and Jacobs, 1992).

Clinically, cyclosporin overgrowth closely resembles that of phenytoin. Rateitschick-Pluss et al. (1983) histologically

examined cyclosporin-associated gingival enlargements, identifying primarily connective tissue covered by irregular, multilayered, parakeratinized epithelium of varying thickness. In some areas, epithelial ridges penetrated deep into the connective tissue, with the latter being highly vascularized and exhibiting irregularly arranged collagen fiber bundles. Focal accumulations of infiltrating inflammatory cells were also observed. While a degree of fibroplasia characterized by the presence of increased numbers of fibroblasts has been noted within the gingival connective tissue (Wysocki et al., 1983), other studies have failed to demonstrate an increase in the numerical density of fibroblasts (McGaw and Porter, 1988). Such contrasting findings may be due to differences in the evolutionary stage of the overgrowth with changes in fibroblast density occurring as the lesion progresses. Accordingly, cyclosporin-induced gingival enlargement may not be a true hyperplasia and hence the term gingival overgrowth or enlargement is more appropriate.

The ultrastructural features of cyclosporine A-induced gingival hyperplasia were studied by Mariani et al. (1993). The authors reported that, although many fibroplasts are present, there is a particular abundance of amorphous material compared to fibrous substance, as well as marked plasma cell infiltration. The ultrastructural features presented by these cells (marked development of rough endoplasmic reticulum, Golgi apparatus, and plasmatic granules) indicate a distinct synthetc immunoglobulin activity. The authors concluded that cyclosporine enlargement is a local manifestation of a general phenomenon.

Although the etiology of cyclosporin-induced gingival overgrowth is unknown, it has been hypothesized that the drug acts only as an important cofactor in the pathogenesis of gingival enlargement. There is little doubt the fibroblast plays an important role in the pathogenesis of this condition; however, individual patient sensitivity to the drug or its metabolites, plasma concentrations of the drug, dental plaque scores, and gingivitis have all been proposed as important contributing factors (Wysocki et al., 1983; Seymour et al., 1987).

Although Seymour and Smith (1991) reported that plaque control measures alone did not prevent gingival overgrowth in cyclosporin-treated adult renal transplant patients, Hancock and Swan (1992) provided clinical evidence in a case report that plaque control without drug withdrawal or surgical excision can be successful in significantly reducing established nifedipine-induced gingival overgrowth.

Renal transplant patients medicated with a combination of cyclosporin and nifedipine have a significantly higher gingival overgrowth score ($P = 0.046$) when compared with the group receiving cyclosporin alone (Thomason et al., 1993).

Nifedipine. Nifedipine is a calcium channel blocking agent used in the treatment of vasospastic angina, chronic stable angina, and ventricular arrhythmias. Its principal action is to inhibit the influx of extracellular calcium ions across cardiac and vascular smooth muscle cell membranes, without changing serum calcium concentration. This prevents the contractile processes of cardiac and vascular smooth muscle from occurring, resulting in a dilatation of the main coronary and systemic arteries. The incidence of gingival overgrowth in response to nifedipine, and the role of drug dosage and duration are presently speculative (Butler et al., 1987).

Clinically, nifedipine-induced gingival overgrowth closely resembles phenytoin enlargement. Histologically, Lucas et al. (1985) observed that tissues from nifedipine- and phenytoin-induced overgrowths were remarkably similar. Both exhibited increased extracellular ground substance as well as increased numbers of fibroblasts. In addition, fibroblasts with numerous, large cytoplasmic structures resembling secretory granules were the striking electron microscopic finding in specimens examined. Because the authors believed that these granules represented newly synthesized ground substance prior to extrusion from the fibroblast, they concluded that an increase in ground substance is the primary cause of overgrowth in these conditions.

Steele et al. (1994) evaluated 120 dentate patients taking calcium antagonists for more than 3 months and a control group. The authors reported that 38% of the patients taking nifedipine, 21% of the patients taking diltiazem, and 19% of the patients receiving verapamil had gingival enlargement, compared with only 4% among controls.

Romanos et al. (1993) demonstrated the localization of collagen types I, III, IV, V, VI, and VII, as well as the glycoprotein fibronectin in nifedipine-induced gingival enlargement. Following indirect immunofluorescence (incubation with antibodies against these extracellular components), the tissue sections showed a diffuse distribution, with anti-types I and III in the stroma and fluorescent staining of the basement membranes of the epithelium, blood vessels, and nerves with collagen type IV antibodies. The increased number of vessels were localized near the surface of the lesion. Collagen type V (filamentous) and collagen type VI, (microfibrillar) components were also localized in the tissue, demonstrating completely different patterns of distribution. Collagen type V appeared "crater-like" and type VI displayed a "honeycomb-shaped" structural model. The blood vessels were not stained but the adjacent areas demonstrated intense fluorescence with these antibodies. Collagen type VII showed a characteristic linear staining near the epithelial basement membrance. In contrast to these, fibronectin localized with a varied intensity in the different areas of the tissue and presented a "cloud-like" structure. This study suggests differences between the matrix components in nifedipine-induced hyperplasia and confirms the heterogeneity of the matrix in health and in gingival alterations.

Morisaki et al. (1993) studied the relationship between gingival inflammation and nifedipine-induced gingival en-

largement in rats. They noted marked gingival overgrowth in the molar regions, regardless of gingival infection although the latter increased the degree of enlargement.

Treatment of Drug-Induced Gingival Hyperplasia

The best treatment of drug-induced gingival enlargement is discontinuing use of the associated drug. The enlargement will slowly become smaller and disappear in a matter of weeks to months. However, this is not practical with most patients because of the drug's important role in medical treatment. Therefore, prevention of secondary inflammation and surgical treatment of the enlargement become the only realistic choices (Carranza, 1990).

Treatment of drug-induced gingival overgrowth is based upon the severity of overgrowth and the ultimate goals of therapy. Both non-surgical and surgical approaches to treatment have been used successfully. Non-surgical therapy consisting of thorough scaling and root planing plus improvement in oral hygiene is the primary method of treating the inflammatory component of gingival overgrowth. It is of little benefit for reducing the fibrous component. Adjunctive therapy such as the daily use of chlorhexidine gluconate mouthrinses may also be considered. Although dramatic results can be obtained using non-surgical treatment alone, some lesions will not respond adequately and require surgical intervention. Surgical approaches to gingival overgrowth are gingivectomy and the flap technique. Gingivectomy is simple and facilitates maintenance care, but it creates an open wound and potential for post-operative complications (Carranza, 1990).

The flap technique thins gingival tissues internally and repositions them apically without significantly removing or altering the external surface of the gingiva (Carranza, 1990). This technique facilitates wound closure and maintenance care, and reduces potential for post-operative complications. It is, however, more technically demanding when compared to the gingivectomy technique (Carranza, 1990). Thus far, no definitive statement can be made regarding the long-term success of surgical therapy (Rateitschak-Pluss et al., 1983).

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CHAPTER 6. ORAL PHYSIOTHERAPY

The objective of oral physiotherapy (oral hygiene) is the complete daily removal of dental plaque with a minimum of effort, time, and devices, using the simplest methods possible. The patient's plaque control procedures must be modified as changes occur in the soft tissue anatomy following periodontal surgery in order to be effective.

TOOTHBRUSHING

Manual Brushes

Parfitt (1963) reported that the toothbrush chosen by patients, often on the recommendation of their dentist, is usually too hard or stiff. Results often show that the bristles of the hard brush only reach the most accessible surfaces of the teeth, areas normally cleaned by the mastication of food and by the friction of the soft tissues. The hard brush does not clean interproximally and debris collects between the teeth, in relatively exposed positions, and at the gingival margin. An old brush with splayed bristles causes gingival damage. In attempting to brush the teeth at the gingival margin, the splayed bristles tend to pierce the soft tissues, causing pain and bleeding. The areas most often brushed by the patient are the buccal and labial surfaces of the upper teeth, and to a slightly lesser degree the same surfaces of the lower teeth. The palatal surfaces of the upper teeth and the lingual surfaces of the lower teeth are rarely cleaned during usual routine care. The terminal teeth in the dental arches, particularly the distal surfaces of these teeth, are difficult to reach with a toothbrush and invariably harbor debris.

O'Leary et al. (1970A) studied the deposition of particulate matter in the crevicular tissue by toothbrushing using two widely taught and accepted techniques. Thirty-eight (38) patients requiring surgical elimination of periodontal pockets brushed for 10 seconds with the roll or Bass technique at designated sites. For each toothbrushing technique, brushes had were pre-soaked in a solution containing carbon particles. On completion of the brushing procedure, the test sites were immediately rinsed and biopsied. No carbon particles were observed in the crevicular epithelium or underlying connective tissue of any test section for either technique. However, the results of this study do not eliminate the possibility that bacteria are sometimes introduced into the crevicular tissue and the circulating blood during toothbrushing, since common bacteria in crevicular tissue are considerably smaller than the carbon particles (mean size 2.5 μm) used in this study.

Lang et al. (1973) studied the frequency of effective oral hygiene procedures necessary to maintain gingival health. Thirty-two (32) dental students were assigned to four groups removing plaque at 12, 48, 72, and 96 hour intervals. The oral hygiene procedures were performed using Charters' brushing technique supplemented by dental floss and interdental woodsticks. Plaque was assessed using the plaque index and gingival health by the gingival index. Results demonstrated that effective oral hygiene procedures at intervals of 48 hours are compatible with gingival health. However, if the intervals between complete removal of bacterial plaque exceeded 48 hours, gingivitis developed.

Waerhaug (1981) reported on the effect of toothbrushing on subgingival plaque formation. Thirty-two (32) upper and lower molars in 4 healthy adult monkeys were used in a split-mouth design in which all teeth were cleaned initially, and then only one side was brushed using the Bass method 3 times a week for one year. During brushing, it could be noted that the bristles penetrated as far as 0.9 mm (0.5 to 1.0 mm) below the gingival margin.

Rotary Brushes

Long and Killoy (1985) evaluated the effectiveness of the Interplak versus manual toothbrushing (modified Bass) in removing plaque in 14 orthodontic patients. Following disclosant and modified O'Leary scoring, mean post-brushing scores were 34% (range 12.5 to 52.9%) for the manual toothbrush and 16.5% (range 5.5 to 31.9%) for the Interplak instrument. The improvement in postbrushing scores after use of the Interplak instrument was significantly better than that of the manual toothbrush.

Youngblood et al. (1985) designed a study to examine the effectiveness of the Interplak instrument versus manual toothbrushing (modified Bass) in removing subgingival and interproximal plaque. Patients requiring extraction of teeth for periodontal or prosthetic reasons were chosen for study. Based on staining with 0.125% toluidine blue, the average depth of cleansing was 0.70 mm for the manual brush and 1.40 mm for the Interplak instrument, representing a statistically significant difference.

Killoy et al. (1989) evaluated the effectiveness of a counter rotary toothbrush and conventional toothbrushes in removing plaque and reducing gingival bleeding. Both brushes reduced plaque indices and bleeding index, but the counter-rotary brush was superior in reducing bleeding index and plaque indices as measured by modified O'Leary and Turesky indices at all time intervals. There were no significant differences when the Surface Area Plaque Index was used.

Brushing Techniques

In the Charters' technique, the brush is placed at right angles to the long axis of the teeth, gently forcing the bris-

tles between the teeth, being careful not to pierce the gums; i.e., bristles do not rest on the gums. With the bristles between the teeth, gentle pressure is exerted during slight rotary movements, causing the sides of the bristles to come into contact with the gum margin, producing an ideal massage. After 3 to 4 movements, the brush is removed and replaced in the same area, making another 3 to 4 movements. The brush is then moved the distance of one embrasure, and the process repeated, holding the sides of the bristles firmly on the gum margin. When teeth are missing, patients must depend on the sense of touch to keep the proper pressure, with the sides of the bristles on the gum margin. The Charters method is especially suitable for gingival massage. When used in conjunction with a soft-to-medium brush, this technique can be recommended for temporary cleaning in areas of healing wounds following periodontal surgery (Charters, 1948).

With the Bass method of toothbrushing, the head of a soft-to-medium toothbrush is placed parallel with the occlusal plane with the "tip" of the brush distal to the last molar. The bristles are placed at the gingival margin with an apical angle of 45° to the long axis of the teeth. A gentle vibratory motion is then exerted in the long axis of the bristles and the bristles are forced into the gingival sulcus as well as into the interproximal embrasures. This action should produce perceptible blanching of the gingiva. The brush is activated with a short back-and-forth motion without dislodging the tips of the bristles, completing 20 such strokes in the same position. Lift the brush, move it anteriorly, and repeat the process. For the palatal aspect of the maxillary anteriors and the lingual of the mandibular anteriors, the toothbrush is held in a vertical position. This technique may be used on the buccal, facial, palatal, and lingual surfaces of all teeth (Bass, 1948). With the modified Bass technique, an additional step is included. Following the vibratory motion, the bristles are swept towards the occlusal surface of the tooth, cleaning the remaining facial or lingual surface.

In the modified Stillman method of brushing, the bristle ends rest partly on the cervical position of the teeth and partly on the adjacent gingiva, directly apically at an oblique angle to the long axis of the teeth. Pressure is applied laterally against the gingival margin, producing a perceptible blanching. The brush is activated with 20 short back-and-forth strokes and is simultaneously moved in a coronal direction along the attached gingiva, the gingival margin, and the tooth surface. This process is repeated on all tooth surfaces, proceeding systematically around the mouth. With this technique, the sides rather than the ends of the bristles are used and penetration of the bristles into the gingival sulci is avoided. The modified Stillman method is recommended for cleaning in areas with progressing gingival recession and root exposure in order to prevent abrasive tissue destruction (Hirschfeld, 1953).

According to the Stillman method, the bristles rest partly on the gingiva and partly on the cervical portion of the teeth. The bristles are placed obliquely to the long axis of the tooth, or at an angle to the plane of the gingival surface and directed apically. Pressure on the gingiva is desired with the least amount of friction or injury. Sufficient pressure is applied by bending the bristles slightly, creating a perceptible gingival blanching. The brushing action is repeated several times, incorporating a slight rotary motion, but not enough to cause displacement of the bristles. The bristles may be bent in any of 3 directions, but the ends of the bristles should always remain as placed. This process is repeated on all tooth surfaces, proceeding systematically around the mouth. Only the occlusal surfaces of premolars and molars should be "scrubbed" (Stillman, 1932).

In the Fones' technique, the teeth are in occlusion and the brush is pressed rather vigorously against the teeth and gums and revolved in circles with as large a diameter as possible. This technique is fairly effective for young children who cannot or do not need a more complex toothbrushing procedure. Its value is limited in the treatment of periodontitis because it does not adequately engage the interproximal areas. The roll technique is easily performed and is most appropriate when the patient has normal oral health. In this technique, the bristles are placed well up on the gingiva at a 45° angle. The sides of the bristles are pressed against the tissue and simultaneously rolled incisally or occlusally against the gingiva and teeth, similar to the turning of a latchkey (Hine, 1950).

DENTAL FLOSS

Lamberts et al. (1982) compared waxed and unwaxed floss to determine ability to remove plaque and effect on gingival health during a home oral hygiene program. Four groups of 20 patients on periodontal maintenance recall were given one of 4 floss products (2 waxed, 2 unwaxed). Seven to 10 days after a thorough prophylaxis, patients were given oral hygiene instructions by videotape. At 0, 28, and 56 days the patients were evaluated for plaque, gingivitis, gingival bleeding, and crevicular fluid. It was determined there was no statistical difference between the 4 types of floss in regards to their ability to remove plaque or prevent gingivitis.

In an identical study design, Wunderlich et al. (1982) demonstrated no difference between waxed and unwaxed floss in maintaining gingival health.

Wong and Wade (1985) compared the effectiveness of Super Floss and waxed dental floss as proximal surface cleansing agents in 34 subjects. Each subject used one agent twice daily for 2 weeks and then switched over to the other agent with the same frequency and for the same period. Plaque was stained with erythrosin and the plaque index of Wolffe used for evaluation. Super Floss was found to be superior (50.1%) to waxed dental floss (45.3%) in removing proximal plaque, but neither was 100% effective. No differences were found between maxillary and mandibular teeth.

A 2-week clinical trial was designed by Graves et al. (1989) to test the comparative effectiveness of waxed and unwaxed dental floss, dental tape, and toothbrushing in reducing interproximal bleeding. One hundred-nineteen (119) subjects with gingival inflammation were randomly assisted to 1 of 4 groups at the beginning of the study. The dental tape and the 2 dental flosses were equally effective in reducing interproximal bleeding and doubly effective as toothbrushing alone.

INTERPROXIMAL CLEANING DEVICES

Three experimental studies were conducted by Gjermo and Flotra (1970) comparing the effect of different interdental cleaning devices. The results demonstrated that in young adults, dental floss removed more plaque at lingual interproximal surfaces than toothpicks. Toothpicks combined with a multi-tufted brush used on the oral surfaces were as effective in removing interproximal plaque as dental floss. The use of dental floss or toothpicks combined with a single-tufted brush may reduce the amount of plaque adhering to interproximal surfaces by an average of 50%. Wide interdental spaces are most efficiently cleaned with an interdental brush.

In 1976 Waerhaug evaluated the effects of the interdental brush. Sixty-seven (67) teeth scheduled for extraction were used for study. Before extraction, the teeth were cleaned with an interdental brush and a mark was made on the tooth surface to indicate the level of the gingival margin. After extraction, the teeth were stained and examined using a stereoscope. Observations indicates that plaque can be removed from 2 to 2.5 mm subgingivally using the interdental brush.

DISCLOSING AGENTS

According to a review article by Tan (1981), disclosing agents have a demonstrated use in research, but there is no convincing evidence that they are of value either for home care or in the dental office. First described in 1914, they stain foreign matter including food debris, plaque, calculus, and pellicle. Raybin (1945) suggested six uses: by operator to aid in prophylaxis, monitoring, diagnosing, and education; by patient for home care; and by researcher to compare effectiveness of various products. Arnim (1963) introduced erythrosin. Fluorescent disclosing is more specific for plaque and not visible without UV light. Block et al. (1972) introduced an agent that stains mature plaque blue and immature plaque red. Disclosant wafers have been shown to improve plaque scores initially, but show a decline in use after 2 weeks. (End of Tan review).

ORAL IRRIGATION DEVICES

O'Leary et al. (1970B) evaluated the ability of water pressure from oral irrigating devices to drive particulate matter into the crevicular tissue. The study was carried out in 3 parts with the same 3 oral irrigating devices in each part. India ink was used as a marker in the irrigating fluid. One device delivered a pulsating stream of water whose pressure was controlled by a pressure valve; the second device delivered a continuous stream of water whose pressure was controlled by a faucet valve; and the third device released a continuous stream of water whose pressure was generated by an effervescent tablet. Biopsy specimens from humans and beagle dogs were then evaluated on the basis of the number of inflammatory cells and the presence of carbon particles. The results demonstrated that carbon particles frequently penetrated the crevicular epithelium and underlying connective tissue of beagle and human gingival tissue when the areas were irrigated with water containing oxygen. The incidence of penetration did not appear to vary with the differing water pressures employed.

The comparative effectiveness of mouthrinsing and direct irrigation in the subgingival area was assessed by Pitcher et al. (1980). Eighty-two (82) untreated, periodontally-diseased teeth had gingival margins marked with a bur prior to probing. A disclosing solution was applied as a 10 ml rinse or irrigated toward the apex at the gingival margin (2.5 ml using 1.2 mm needle). After staining, the teeth were extracted and measured for dye penetration. The apical plaque front was stained with crystal violet. Direct irrigation at the crevice produced significantly greater penetration (mean 1.8 mm, PD = 5.4 mm) than mouthrinsing (mean, 0.2 mm; PD = 4.7 mm), but neither technique reached the apical plaque front. It was concluded that penetration with irrigation tended to increase with increasing pocket depth and that subgingival plaque ended 1.5 mm coronal to probing depth.

Hardy et al. (1982) studied a technique of direct irrigation that would reproducibly gain access to the apical plaque border. A total of 98 proximal surfaces on 68 teeth scheduled for extraction were studied. After making reference marks at the level of the gingival margin, disclosing solution was delivered from the gingival margin (superficial irrigation) and 3 mm apical to the gingival margin with a 23-gauge needle (deep irrigation) and the teeth extracted. The direct irrigation group penetrated to the apical plaque border 81% of the time and bottom of the pocket 56%, compared to the superficial irrigation (10% and 6%, respectively). Superficial irrigation penetrated an average of about 35% of pocket depth and deep irrigation penetrated an average of 95% of pocket depth. The results indicate that deep irrigation (3 mm) within periodontal pockets provides an efficient and predictable means of reaching the subgingival plaque apical border.

The effectiveness of an oral irrigator as a vehicle for delivering an aqueous solution into periodontal pockets was evaluated by Eakle et al. (1986). Nine patients requiring extraction due to advanced periodontitis were divided into 2 groups, each irrigated to the gingival margin using an irrigator containing erythrosin dye; one group at a 45° angle and the other at a 90° angle. The teeth were extracted and

the apical penetration of dye measured. Penetration at 90° was 71% for shallow pockets (0 to 3 mm); 44% for moderate pockets (4 to 7 mm); and 68% for deep pockets (≥ 7 mm). The 45° application showed 54%, 46%, and 58% respectively. No soft tissue injuries resulted. The results suggest that the oral irrigator will deliver an aqueous solution into periodontal pockets and will penetrate on average to approximately half the depth of the periodontal pockets.

Sanders et al. (1986) evaluated the effects of daily supragingival pulsated jet irrigation with 0.02% chlorhexidine and 0.05% metronidazole on the subgingival plaque as monitored using darkfield microscopy. Twenty-two (22) patients with periodontal pockets ≥ 4 mm and radiographic bone loss were monitored for 84 days. The results indicated only marginal benefits from supragingival irrigation regarding favorable shifts in the composition of subgingival plaque, as monitored by darkfield microscopy.

Greenstein (1987) reviewed the role of subgingival irrigation with chemotherapeutic agents. Many authors report oral rinses do not permeate subgingivally, and irrigation at the gingival margin does not reach greater than 3 mm. If subgingival irrigation is accomplished by inserting the tip 3 mm subgingivally, then agents could reach the apical border of plaque. Evaluating alterations in microflora is best accomplished on patients who have not been root planed prior to study. Studies with no prior scaling indicated that chlorhexidine reduced spirochetes but not always motile forms. *Bacteroides* was briefly reduced by SNF_2 and hydrogen peroxide, and *A. actinomycetemcomitans* was affected by hydrogen peroxide. *Treponemes* seemed to be unaffected by irrigation. Chlorhexidine reduced supragingival plaque at treated sites and decreased probing depth by about 1 mm after a single treatment. If daily subgingival irrigation was preceded by root planing, pockets were reduced about 2 mm. The gingival index was also reduced. After root planing, jet irrigation with or without antimicrobial agents reduced the bleeding index and probing depths. Improvement in clinical parameters without root planing lasted up to 10 weeks and 2 to 6 months with root planing. Chlorhexidine remains the agent of choice, but in high spirochete infections metronidazole may be more effective.

Using scanning electron microscopy (SEM) Cobb et al. (1988) evaluated human gingival biopsies of the pocket wall following supragingival irrigation with a Water Pik device set at 60 psi. Results showed the irrigation force induced no epithelial micro-ulceration or alteration of cell morphology, confirming the safety of using pulsating irrigation.

Ciancio et al. (1989) evaluated the efficacy of an antimicrobial mouthrinse delivered by an oral irrigation device twice daily. The gingival index, gingival crevicular fluid volume, plaque index, modified papillary bleeding index, probing depth, and attachment level were recorded in 66 patients. Subgingival plaque was sampled by means of sterile paper points and assessed by phase contrast and immunofluorescence microscopy. Parameters were repeated at 3 and 6 weeks. Results indicated that irrigation with or without an antimicrobial agent was effective in reducing plaque, suggesting that oral irrigation may have beneficial effects on oral health and that use of a chemotherapeutic agent will lead to greater reductions in plaque and gingival bleeding and to moderate decreases in the total bacteria counts detected by phase contract microscopy.

Scaling and root planing (SRP) was compared to SRP plus multiple irrigations with 2% chlorhexidine (CHX) or 2% CHX multiple irrigations alone (Southard et al., 1989). The effects of treatment on clinical and microbiological indices were evaluated overa 25-week period. All parameters were significantly reduced (versus baseline) in the SRP, SRP + CHX, and CHX alone. SRP + CHX resulted in significantly greater reduction in *P. gingivalis* counts which lasted up to 15 weeks. This combination therapy also resulted in significantly ($P \leq 0.05$) greater attachment gain at 5 and 7 weeks, compared to SRP alone. This study shows an adjunctive effect of a professionally applied irrigation with an antimicrobial to the beneficial effect of SRP.

In a 1990 study Flemmig et al. reported that the antimicrobial irrigation with 0.06% CHX reduced plaque (53.3%) and gingivitis (42.5%) significantly more than the 0.12% CHX (43.3%; 24.1%) rinse or water irrigation.

Braun and Ciancio (1992) evaluated the depth of subgingival delivery in mild to moderate periodontal disease using a newly designed irrigation tip in a powered oral irrigator. A total of 145 sites (teeth planned for extraction) were evaluated in 14 patients using the modified plaque index and plaque index. In the subgingival irrigation group (70 sites), the tip was placed 1 to 2 mm into the selected pocket, and sites irrigated with erythrosin solution 5 seconds per site each. The control group rinsed for 30 seconds with the erythrosin solution. Following examination, it was determined that in the rinse (control) group, mean pocket penetration in all sites ≤ 6 mm was 21%. In the irrigated group, mean pocket penetration was 90% in sites ≤ 6 mm and 64% in sites ≥ 7 mm. Lack of patient discomfort following irrigation suggests that tissue injury is not significant using this delivery system.

TRAUMA AND ORAL HYGIENE

Gillette and Van House (1980) characterized abrasion as notch-shaped lesions usually at the CEJ and extending apically. Erosiona is chemically induced, saucer-shaped lesion. Toothbrush irritation is mechanical abrasion of the gingiva by the toothbrush. Lesions often appear fresh and ulcerated, and usually repair within 3 days following modified brushing. Recession also may be caused by calculus, plaque, habits, iatrogenic dentistry, and periodontal surgery. Water irrigating devices may cause periodontal abscesses, cellulitis, penetration of the maxillary sinus, gingival hemorrhage, and transient bacteremias. Dentifrices, mouthwashes, chewing gums, and disclosing agents may cause

allergic responses. Toothbrush bristles may cause acute abscesses. Bacteremias occur with oral hygiene procedures and increase with more advanced disease. Elimination of periodontal disease in at-risk patients is important; e.g., those requiring subacute bacterial endocarditis prophylaxis.

Hirschfeld (1953) discussed injuries of the marginal gingiva that may accompany toothbrushing. The author noted the effects of technique and repeated trauma, describing lacerations, stabbing, puncture, and perforation injuries.

Radentz et al. (1976) evaluated for brushing technique and type, and type and amount of dentifrice in 80 individuals, who were scored for cervical abrasion, gingival recession, pH, plaque, and gingival bleeding. The 40 individuals (50%) with cervical abrasion applied 17% more dentifrice than the non-abrasion group, demonstrating a significant correlation between quantity and prevalence. The highest percentage of abrasion occurred in the maxillary right quadrant, and the first three areas brushed. It was most prevalent on maxillary first molars and maxillary and mandibular premolars. Gingival recession also correlated well with cervical abrasion lesions and tended to occur in the older participants. The authors indicate that abrasion appears related to amount of dentifrice, gingival recession, and initial location of brushing.

In 1976 Sangnes reported that abrasion of tooth structure is mostly associated with the dentifrice while gingival lesions are more related to the toothbrush. Abrasion is the pathologic wearing away of substance by the friction of a foreign body. Tooth surfaces are polished and lesions appear on the side opposite the dominant hand, more often on maxilla. Abrasion tends to increase with hard bristles, horizontal brushing and brushing frequency. Gingival lesions develop more rapidly in the premolar areas (prominent areas) and gingival bleeding is more prevalent with a brush having thicker, unrounded bristles.

Reissten et al. (1978) measured the abrasion effect of enamel- and cementum-brushing with and without toothpaste using SEM on 12 unerupted third molar teeth. Horizontal brushing (soft nylon bristles) of the outer enamel and cementum surfaces was performed with a dentifrice slurry ($CACO_3$ abrasive) for 5 and 10 minutes. After brushing with toothpaste, all enamel surfaces were free of scratches, while the cementum surfaces exhibited scratches, which increased with the brushing period. Brushed control teeth with saline revealed much less abrasiveness when compared with toothpaste. This study suggests that the toothpaste is principally responsible for abrasiveness when used on a soft toothbrush.

Niemi et al. (1984) evaluated the stiffness of toothbrush bristles and the abrasiveness of dentifrices for influence on the degree of gingival erosion in 24 dental hygiene students. Two toothbrushes with different bristle thicknesses (soft 0.15 mm; hard, 0.23 mm) and 2 dentifrices, moderately and highly abrasive, were used. The use of the hard brush resulted in lower plaque scores and more gingival erosion than the soft brush. With both brushes a significantly greater number of lesions were recorded after use of an abrasive powder than when no dentifrice was used. The results indicate that the modest decrease in plaque scores with increasing stiffness of the toothbrush bristles and with increasing abrasiveness of the dentifrice is accompanied by increased damage to the soft gingival tissues.

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CHAPTER 7. ROOT TREATMENT, REATTACHMENT, AND REPAIR

Section 1. Root Healing

DEFINITIONS

Repair: Healing of a wound by tissue that does not fully restore the architecture or the function of the part.

Reattachment: To attach again. The reunion of epithelial and connective tissues with root surfaces and bone such as occurs after an incision or injury. Not to be confused with new attachment.

New Attachment: The union of connective tissue or epithelium with a root surface that has been deprived of its original attachment apparatus. This new attachment may be epithelial adhesion and/or connective tissue adaptation or attachment and may include new cementum.

Regeneration: Reproduction or reconstitution of a lost or injured part.

HEALING BY A LONG JUNCTIONAL EPITHELIUM

Following surgery, the curetted root surface may be repopulated by 4 different types of cells: epithelial; gingival connective tissue; bone; and periodontal ligament cells. The cells which repopulate the root surface determine the nature of the attachment that will form. Periodontal wound healing following traditional surgical procedures results in the formation of a long junctional epithelium along the root surfaces, with no new connective tissue attachment. The epithelial downgrowth prevents the formation of a new connective tissue attachment by preventing repopulation of the root surface by cells derived from the periodontal ligament. However, the coverage of the root surface by an epithelial layer has a beneficial effect; i.e., the prevention of root resorption and ankylosis, which otherwise could be induced by gingival connective tissue and bone.

Waerhaug (1955) studied the healing following scaling and root planing in one dog with subgingival calculus on 4 cuspids. Two of the teeth were scaled and polished and 2 served as untreated controls. After the removal of calculus, the bleeding subsided and normal conditions were observed. It was concluded that a complete removal of subgingival calculus will, under favorable conditions, lead to a re-formation of a normal epithelial cuff in areas earlier covered with calculus, and it may result in a more or less complete disappearance of the inflammation caused by the calculus.

Caton and Zander (1976) studied the healing after surgical treatment. They created a periodontal pocket on 1 molar in a monkey. The pocket was treated by flap curettage, followed by plaque removal every other day for 1 year. Radiographs taken after 1 year showed increased radiodensity of the crestal bone. The histological sections showed a long junctional epithelium extending to the most apical point of root instrumentation. The connective tissue fibers between the junctional epithelium and the bone were oriented parallel to the long axis of the tooth. They concluded that repair of an osseous defect can occur opposite junctional epithelium on the root surface without new attachment of connective tissue.

Caton and Zander (1979) created 22 pairs of periodontal pocket in 2 monkeys. All teeth were scaled, then a plaque control program consisting of toothbrushing, flossing, and topical application of 2% chlorhexidine 3 times a week was initiated. On one side of the jaw, root planing and soft tissue curettage were performed and were repeated at 3, 6, and 9 months after initial therapy. The treatment resulted in the formation of a long junctional epithelium with no new connective tissue attachment. In 8 of the 22 pockets, the procedure produced discontinuities or "windows" of connective tissue attachment in the junctional epithelium. The resistance to probing following root planing and soft tissue curettage appears to result from the formation of a long junctional epithelium rather than new connective tissue attachment.

Caton et al. (1980) compared the healing after 4 different surgical procedures. Periodontal pockets were induced in 8 monkeys then treated by 1) modified Widman flap (MWF) without osseous surgery; 2) MWF without osseous surgery but with autogenous red marrow and cancellous bone; 3) MWF without osseous surgery but with beta tricalcium phosphate; and 4) periodic root planing and soft tissue curettage. Histometric measurements after 12 months of healing demonstrated that all treatment procedures resulted in the reformation of an epithelial lining (long junctional epithelium) with no difference between treatments. The most apical cells of the junctional epithelium were consistently located at or close to the level of the root surface which had been planed. Adjacent to the epithelial lining were fibers oriented parallel to the root surface. In a few specimens, principal fibers were inserted into new cementum and adjacent alveolar bone. This area could represent healing of the root surface injured during instrumentation.

Proye and Polson (1982) studied the effect of root surface alterations on periodontal healing. Three teeth in each of 4 monkeys were extracted and the coronal third of the root surfaces was planed to remove the attached periodontal fibers and cementum. The teeth were reimplanted into their sockets

within 15 minutes. Histological examination showed a zone of fibrin containing erythrocytes and PMNs adjacent to the denuded root surface 1 day after reimplantation. Epithelium migrated rapidly along the denuded root, reached the alveolar crest at 3 days, and was within the ligament space at 7 days. At 21 days, the epithelium was at the apical limit of root instrumentation. There was no evidence of connective tissue attachment to any portion of a denuded root surface. It was concluded that the absence of fibers on the root surface results in apical migration of the epithelium, and precludes formation of new connective tissue attachment.

In a followup study, Polson and Caton (1983) evaluated the factors influencing periodontal repair and regeneration. In 2 monkeys, central incisors with reduced periodontium were transplanted into sockets of normal height, and central incisors with normal periodontium were transplanted into sockets of reduced height. After 40 days of healing, the normal roots transplanted into the reduced periodontium had connective tissue reattachment in the periodontal ligament and supracrestal regions. The exposed roots placed into the normal periodontium were lined with epithelium interposed between the root surface and the alveolar bone. The results indicated that root surface alterations, rather than the presence of a reduced periodontium, inhibit new connective tissue attachment.

Lindhe et al. (1984) studied the contribution of alveolar bone to connective tissue re-attachment following treatment. The maxillary and mandibular incisors in 3 monkeys were extracted and the buccal root surfaces of the incisors from the left side of the jaws were planed. In 2 of the monkeys, the buccal alveolar bone plate was removed. All teeth were reimplanted into their original sockets within 4 minutes. Histologic examination after 6 months showed that irrespective of the presence or absence of alveolar bone, connective tissue reattachment failed to form on that part of the tooth that had been root planed; instead a long junctional epithelium had formed. However, in non-root planed teeth a connective tissue reattachment had occurred. Alveolar bone located adjacent to a root surface may have limited influence on the biological conditions which determine whether periodontal healing results in connective tissue reattachment or new attachment.

Magnusson et al. (1983) evaluated the resistance of the long junctional epithelium to plaque infection in 4 monkeys. Eight test teeth with induced periodontitis were treated surgically. After 4 months of plaque control, plaque was allowed to accumulate for 6 months on 4 of the treated teeth and 3 control teeth. Ligatures were placed on the remaining 4 test teeth and on 3 control teeth to enhance subgingival plaque formation. The infiltrated connective tissue of the test teeth covered about 60% of the junctional epithelium while for the controls it was 90%. The inflammatory lesion in the connective tissue did not extend deeper into the periodontal tissues in sites with a long junctional epithelium than in gingival units of normal height. It was concluded that the barrier function of a long junctional epithelium against plaque infection is not inferior to that provided by a dentogingival epithelium of normal height.

The resistance of the long junctional epithelium was also studied by Beaumont et al. (1984). Inflammation by ligatures was induced in 6 dogs. Three of the dogs had induced periodontitis and were treated surgically which resulted in long junctional epithelium; the remaining 3 dogs had healthy periodontium. Healing was evaluated over periods ranging from 4 to 20 days. There were no instances of sulcular ulceration in the group with established long junctional epithelial attachment, but ulcerated sulcular epithelium was seen often in the earlier time periods of the previously healthy group. It was concluded that there was no appreciable difference in resistance to disease between a long junctional epithelium and a true connective tissue attachment.

The coverage of the root surface by an epithelial layer has a beneficial effect; i.e., the prevention of root resorption and ankylosis, which otherwise could be induced by gingival connective tissue and bone. Karring et al. (1984) studied the potential for root resorption during periodontal wound healing. In 2 monkeys, teeth with induced periodontitis were extracted, and the roots were planed. After crown resection, the roots were partially embedded into sockets prepared in the buccal surfaces of the jawbone. The coronal periodontitis affected the roots located in contact with the connective tissue of the mucosal flap after suturing. Healing was evaluated between 1 and 24 weeks. The parts of the coronal root surfaces which were covered with epithelium as a result of exposure exhibited no resorption or ankylosis. The root portions in contact with bone or gingival connective tissue regularly displayed root resorption. The results indicate that root resorption is a progressive process in roots exposed to bone and/or gingival connective tissue and that epithelial downgrowth exhibits a protective function to this process.

HEALING BY REATTACHMENT

During surgery, if healthy root surfaces are left undisturbed, healing will result in the reunion of the gingival connective tissues with the root surfaces and bone. This healing will be characterized by the reformation of the functionally oriented attachment apparatus that was present before surgery.

Karring et al. (1980) studied the healing following implantation of periodontitis-affected roots into bone tissue in 3 beagle dogs. Following crown resection of 12 teeth, the periodontitis-affected portion of the roots was scaled and root planed. The roots were extracted and implanted into bone cavities prepared in edentulous areas of the jaws so that epithelial migration into the wound and bacterial infection were prevented during healing. The results after 1, 2, and 3 months of healing demonstrated that new connective tissue attachment did not occur to periodontitis-affected

root surfaces placed adjacent to bone tissue, but healing was characterized by repair phenomena; i.e., root resorption and ankylosis. In areas where periodontal ligament tissue was preserved, a functionally oriented attachment apparatus was reformed.

Nyman et al. (1980) in a similar experiment studied the healing following implantation of periodontitis-affected roots into gingival connective tissue. The study was performed on 28 teeth in 1 dog and 2 monkeys. Following root resection and scaling and root planing of the periodontitis-affected portion of the teeth, the extracted roots were implanted into grooves prepared in edentulous areas of the jaws so that the roots were embedded to half their circumference in bone, leaving the remaining part to be covered by the gingival connective tissue of the repositioned flap of the recipient site. Histologic examination after 2 and 3 months of healing disclosed that a new connective tissue attachment failed to form on the previously exposed root surface located in contact with gingival connective tissue. In addition, root resorption was seen on this portion of the roots, which indicated that gingival connective tissue does not possess the ability to form new connective attachment, and may induce resorption of the root. In areas where the periodontal ligament was preserved prior to transplantation, a fibrous reattachment occurred between the root and the adjacent gingival tissue. Resorption and ankylosis were seen in areas adjacent to bone.

HEALING BY NEW ATTACHMENT

Healing after treatment can be in the form of new attachment. This new attachment is characterized by the union of connective tissue or epithelium with the root surface that has been deprived of its original attachment apparatus. Several clinical and histological studies have confirmed that healing by new attachment is possible, and several techniques have been employed to achieve this type of healing.

Animal Studies

The healing of surgical wounds by new connective tissue attachment was studied by Listgarten et al. (1982). A surgical wound was created on the mesial surface of the left maxillary first molar of rats and the root surface curetted free of soft tissue and cementum. The rats were sacrificed between 10 days and 12 months after surgery. The junctional epithelium became re-established by migration of epithelium from the wound edge along the cut gingival surface facing the tooth, until contact was established near the apical border of the instrumented root surface. The entire epithelial attachment was displaced coronally, primarily at the expense of sulcus depth which decreased with time, and by replacement of the apical portion of the junctional epithelium by a connective tissue junction of increasing dimension.

New connective tissue attachment was also reported by Polson and Proye (1983) after citric acid root conditioning. Twenty-four (24) teeth in 4 monkeys were extracted, then reimplanted after either root planing the coronal one third or root planing the coronal one third followed by topical application of citric acid. Histological examinations were performed at 1, 3, 7, and 21 days after implantation. Epithelium migrated rapidly along the denuded, non-acid treated root surfaces reaching the level of root denudation at 21 days. Epithelium did not migrate apically along denuded root surfaces treated with citric acid. At 1 and 3 days, inflammatory cells were enmeshed in a fibrin network which appeared to be attached to the root surface by arcade-like structures. At 7 and 21 days, the region had repopulated with connective tissue cells, and collagen fibers had replaced the fibrin. It was concluded that collagen fiber attachment to the root surface was preceded by fibrin linkage, and that the linkage process occurred as an initial event in the wound healing response.

Karring et al. (1985) studied the formation of new connective tissue attachment in a submerged environment. Periodontitis was induced in 4 monkeys. Three months later, the teeth were root planed, the crowns resected, and the roots covered by a laterally displaced flap. The roots that remained covered had newly formed cementum with inserting collagen fibers on the instrumented root portions. New fibrous attachment was 1.0 ± 0.7 mm. The part of the roots coronal to the newly formed cementum exhibited resorption as the predominant feature. In sites with angular bony defects, regrowth of supporting bone had occurred in the bottom of the defect. The authors concluded that new connective tissue attachment forms on previously periodontitis-involved roots by coronal migration of cells originating from the periodontal ligament.

Blomlof et al. (1987) compared 5 different methods for new attachment formation. Four monkeys with induced periodontitis were treated by 1 of 5 methods: plaque control only; surgery with ultrasonics or hand instrumentation; or chemical treatment by cetylpyridinium chloride and sodium-n-lauroyl sarcosine with or without citric acid. Results of surgery with ultrasonic or hand instrumentation were very similar. Epithelium covered the denuded dentin surface and bone formation was minimal. Both chemically-treated groups resulted in a significant new attachment formation, with the citric acid group showing a slight tendency for more new attachment. The supracrestal fiber bundle was 2 to 3 times thicker in the chemically-treated groups than the mechanically-scaled roots.

Selvig et al. (1988) studied new connective tissue formation in fenestration wounds. Full thickness flaps were reflected over the maxillary incisors in 8 dogs. A fenestration was made labially over each root 3 to 5 mm from the alveolar crest. The flap was repositioned and sutured. After 7 days of healing, fibroblasts, macrophages, and a few leukocytes were present near the treated root surface. At 14 days, interdigitation of the newly-synthesized fibers and the fibrils of the demineralized dental matrix was pronounced.

At 21 days, collagen fibers attached to the cementum or dentin surface now contained fibrils of mature width. Initial reattachment to an instrumented, demineralized root surface included deposition of newly formed collagen fibrils in close approximation to, but not in direct continuity with exposed matrix fibrils. In areas of resorption, new fibrils may adhere to the surface of hard tissue without any fibrillar interdigitation.

Human Clinical Studies

Proye et al. (1982) monitored 128 pockets in 10 patients immediately before and 1, 2, 3, and 4 weeks after a single episode of subgingival root planing. Significant probing depth reduction (initial) occurred at 1 week and was associated with gingival recession, was reduced further (secondary) at 3 weeks, and was associated with gain in clinical attachment. It was concluded that substantial reduction in probing depth occurs within 3 weeks after a single episode of root planing owing to initial gingival recession and secondary gain in clinical attachment.

Nyman et al. (1988) evaluated the role of diseased cementum on new attachment formation. Eleven (11) patients were treated surgically using a split mouth design. In 2 quadrants (control), the teeth were scaled and root planed to remove all cementum. In the remaining quadrants (test), calculus was removed without removal of cementum and the teeth were polished. The patients were followed for 24 months. The results showed that the same degree of improvement was achieved following both types of treatment: there was some gain of probing attachment for both treatment modalities.

Human Histologic Studies

Nyman et al. (1982) reported on a case of a mandibular lateral incisor with attachment loss of 11 mm that was treated with a barrier membrane. A Millipore filter was placed between the flap and the tooth to prevent the epithelium and the gingival connective tissue from reaching contact with the curetted root surface. The tooth was removed en bloc after 3 months of healing. New cementum with inserting fibers was observed extending to a level 5 mm coronal to the alveolar bone crest. New bone had been formed within the angular bony defect. It was concluded that regeneration of cementum including fibrous attachment may be achieved by cells originating from the periodontal ligament, provided that epithelial cells and gingival connective tissue cells are prevented from occupying the wound area adjacent to the root during the initial phase of healing.

Lopez and Belvederessi (1983) implanted 26 root fragments without periodontal ligament and 18 root fragments with periodontal ligament in pouches created in the connective tissue under the mucosa of 44 patients. The implants together with the adjacent tissues were removed between the third and twenty-sixth week after implantation. The implants without periodontal ligament failed to form cementum or bone-like tissue, and in the twenty-sixth week they still showed resorption. Ten of the implants with periodontal ligament showed deposition of cementum with collagen fibers attached to it after the twelfth week. The formation of new attachment could be ascribed to the influence of cells of the remaining periodontal ligament on the implanted root fragments.

Bowers et al. (1989 A, B, and C) in a 3-part study evaluated the regeneration of periodontal tissues in a submerged and non-submerged environment with and without grafting material. In Part I, the formation of new attachment (new bone, new cementum, and an intervening periodontal ligament) was studied in 9 patients with 25 submerged and 22 non-submerged defects. Histologic evaluation after 6 months showed that a new attachment did form on pathologically exposed root surfaces in a submerged environment (0.75 mm). Complete regeneration was limited by the amount of bone and cementum formation. Periodontal ligament fibers were embedded in cementum and bone and were most frequently oriented parallel to the root. In Part II, new attachment was evaluated in grafted and non-grafted submerged defects in 10 patients. The results showed that after 6 months of healing, grafting with demineralized freeze-dried bone allograft (DFDBA) enhanced the amount and frequency of new attachment apparatus (1.76 mm versus 0.76 mm for non-grafted sites), new cementum (1.88 mm versus 1.48 mm for non-grafted sites), and new bone (1.96 mm versus 0.80 mm for non-grafted sites) in a submerged environment. In Part III, new attachment was evaluated in a non-submerged environment with and without bone grafts. Twelve patients had 32 defects treated with DFDBA and 25 defects treated with open debridement. Histometric evaluation after 6 months of healing demonstrated that grafted defects had a mean new attachment apparatus of 1.21 mm. There was a mean of 1.24 mm of new cementum formation, 0.13 mm of connective tissue attachment, and 1.75 mm of new bone formation. The junctional epithelium was located 1.36 mm coronal to the calculus reference notch. In non-grafted sites, a long junctional epithelium formed along the entire length of exposed root surfaces.

THE EFFECTS OF TREATMENT ON GINGIVAL FIBROBLASTS

In Vitro Studies

The cells in the healing site can only attach to a biologically acceptable root surface. Periodontal treatment should produce a root surface that will promote cell growth and attachment.

Aleo et al. (1975) studied in vitro the attachment of human fibroblasts to root surfaces. Untreated periodontally involved teeth were extracted and cut longitudinally. Three groups of 20 or more teeth were employed: 1) received no treatment; 2) endotoxin extracted with 45% phenol in water; 3) cementum was mechanically removed. Teeth were

incubated with human gingival fibroblasts for 24 to 48 hours. Microscopic examination demonstrated uniform attachment to the uninvolved portion of the root surface whereas the involved portion of the root surface allowed only a few cells to attach. When the endotoxin was removed from the root surface by phenol extraction or by mechanical removal of the diseased cementum, the fibroblasts attached normally to the root surface.

Gilman and Maxey (1986) compared ultrasonics to ultrasonics plus air powder abrasive for their ability to remove endotoxin. Six teeth were extracted and sectioned into 12 specimens. Test specimens were instrumented with the ultrasonics or ultrasonics plus air powder abrasive. Four calculus-covered control specimens were not instrumented. Eight root specimens were placed in fibroblast tissue culture and were stained for determination of fibroblast viability after 48 hours. No fibroblast growth took place on calculus control specimens. Ultrasonics specimens showed light fibroblast growth and viability. Ultrasonics plus air powder abrasive specimens showed superior growth and vitality of fibroblasts.

THE EFFECT OF PLAQUE CONTROL ON HEALING FOLLOWING TREATMENT

Bacterial plaque is the main etiologic factor in periodontal disease. Studies have established that periodontal disease will not initiate or progress in the absence of plaque. Also, when healing is considered, numerous reports have demonstrated that the results of treatment will be compromised if bacterial plaque is not removed during the healing period, and that optimal healing can only be achieved in a plaque-free environment.

Human Clinical Studies

Rosling et al. (1976) treated 24 patients with modified Widman flap surgery. The test group was recalled once every 2 weeks and given professional tooth cleaning. Control patients were recalled once every 12 months for prophylaxis. All patients were re-examined 6, 12, and 24 months after surgery. There was a gain of attachment in the test group (3.0, 3.2, and 3.5 mm, at 6, 12, and 24 months, respectively), whereas in the control group there was a continuous loss of attachment following surgery. In the control patients, 58 of 62 2-walled and all 3-walled defects were present after 2 years. In the test group, both 2-walled and 3-walled defects had a "bone fill" of approximately 80%.

Polson and Heijl (1978) treated 15 defects in 9 patients by reverse bevel flaps. Patients were recalled once a week for 3 to 6 weeks. Re-entry procedure performed 6 to 8 months after initial surgery showed a bony morphology with the surface texture of alveolar bone adjacent to the tooth in the area of the defect. The results showed that after surgical debridement and establishment of optimal plaque control, the amount of coronal bone regeneration averaged 2.5 mm (77%) and the average amount of crestal resorption was 0.7 mm (18%). Assessment of tooth mobility showed a tendency of a given tooth to decrease by 1 degree of mobility. It was concluded that intrabony defects may predictably remodel after surgical debridement and establishment of optimal plaque control.

Human Histologic Studies

Waerhaug (1978A) treated 21 patients with a total of 39 teeth scheduled for extraction with root planing, some with flap access. Patients were instructed to carry out supragingival plaque control, and were observed for periods ranging from 15 days to 7 months. Teeth were then extracted and microscopic observations reported. It was found that reformation of a normal dento-epithelial junction invariably occurs when calculus, including plaque, is completely removed. If good supragingival plaque control is maintained, no further subgingival plaque will form and health can be maintained. Residual plaque may give rise to reformation of plaque within the pocket; however with excellent plaque control, the tissues may appear clinically healthy. Residual plaque progresses apically, with a loss of attachment occurring at the same speed (2 μm/day).

Waerhaug (1978B) treated 84 condemned teeth with probing depths of ≥ 3 mm by scaling and root planing, some with flap access. Thirty-one (31) teeth were extracted immediately, and 53 had healing times of up to 1 year before extraction. The results of this light microscopic study demonstrated that the distance from the plaque front to intact periodontal fibers is 0.5 mm to > 1.0 mm. It was concluded that the chances of removing all subgingival plaque are fairly good if probing depth is ≤ 3 mm; in the 3 to 5 mm range, chances of failure are greater than the chances of success, and if probing depth exceeds 5 mm the chance of failure dominates. If all subgingival plaque is removed, the junctional epithelium will be readapted to the plaque-free tooth surface. If new supragingival plaque is allowed to form or subgingival plaque is not removed, they will give rise to the reformation of subgingival plaque within the pocket. Surgical elimination of pathological pockets ≥ 3 mm is the most predictable method for attaining good subgingival plaque control.

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Section 2. Scaling and Root Planing

ROOT SMOOTHNESS (HAND VERSUS ULTRASONICS)

Controversy still exists over the superiority of ultrasonics versus hand instruments in calculus removal, cementum removal, endotoxin removal, and root surface smoothness.

The smoothness of the root surface after instrumentation was studied by Kerry (1967). One hundred and eighty (180) anterior teeth from 43 patients were scaled and root planed using 5 different methods: curets; one of two ultrasonic units; curets followed by ultrasonics; and ultrasonics followed by curets. The teeth were extracted and the relative roughness was determined with a Profilometer. The smoothest roots were obtained by first using the ultrasonics and finishing with curets. Almost equally as smooth were the curetted only roots. In the middle range was the group of curets followed by ultrasonics. The roughest roots were produced by the ultrasonic tips. Hand curets produced smoother root surfaces than the ultrasonic instruments.

In a scanning electron microscopic (SEM) study Wilkinson and Maybury (1973) found that teeth root planed by curets were smooth and flat and no longer harbored small particles of calculus. The root surfaces treated by ultrasonics looked chipped and fractured with the appearance of irregular ridges. They concluded that both methods of instrumentation were equally effective in removing foreign matter from the tooth, but curets produced smoother surfaces than ultrasonics.

Jones et al. (1972) treated 54 teeth using tungsten carbide curets, scalers, or ultrasonics before extraction and examination under SEM. Twenty-six (26) teeth were completely free of calculus after treatment. No difference in the efficiency of calculus removal was found between the various instruments used; the ultrasonics caused least damage to the root surface, while scalers and curets caused slight damage.

Pameijer et al. (1972) using SEM found no difference in root topography when teeth were instrumented by ultrasonics or hand instruments. Hand instruments removed substantially more tooth structure than ultrasonics. Instrumentation of a polished dentinal surface by hand instruments, however, removed tooth structure and left a rough surface when compared to ultrasonic instruments.

Lie and Meyer (1977) using SEM showed that calculus removal was considerably more complete with the diamond point than with curets, ultrasonics, or Roto-Pro instrument. The ultrasonic instrument gave the least satisfactory cleaning of the tooth surface. When the loss of tooth substance was scored, only minor differences were found between the Roto-Pro, curets, and the ultrasonic instrument, while the

diamond scored considerably higher than any of the other instruments.

Breininger et al. (1987) instrumented 30 molar and 30 non-molar teeth with either hand curets or ultrasonics. These treated teeth plus 20 untreated controls were extracted, stained with 0.5% toluidine blue, and examined under SEM for residual stainable material and calculus. The results showed that a large percentage of treated proximal root surfaces had stainable deposits, but these surfaces were often "unexpectedly" free of microbes. The majority of stained deposits was composed of adherent fibrin and instrumentation debris. When plaque was found, it was in small "mini-colonies" (< 0.55 mm diameter). Both instrumentation methods appeared to be effective in bacterial debridement but only partially effective in removing subgingival calculus.

The effect of root roughness on plaque accumulation and inflammation of the adjacent gingival tissues was studied by Rosenberg and Ash (1974). Fifty-eight (58) teeth were extracted 28 to 232 days after instrumentation. Using a Profilometer to measure root roughness, they found a statistically significant difference in mean roughness between curetted teeth (mean 9.51) and either teeth treated with ultrasonics (mean 17.21) or control teeth (18.30). No significant differences in mean plaque scores or mean inflammatory indices were observed between the 3 groups. It was concluded that root roughness was not significantly related to the mean inflammatory index of the adjacent gingival tissues or to supragingival plaque accumulation.

Khatiblou and Ghodssi (1983) studied the effects of root roughness on healing following surgical treatment. Eighteen (18) single rooted teeth in 12 patients with advanced periodontitis were divided into 2 groups. Modified Widman flaps were performed for both groups. In one group, shallow horizontal grooves were made on root surfaces to roughen them after root planing. Healing was evaluated 4 months after surgery. Results indicated that there were no significant differences between the two groups in terms of probing depth reduction and gain of attachment. Both groups showed a gain of attachment and reduced probing depth as a result of the surgical treatment. It was concluded that clinical healing is not affected by varying degrees of root surface roughness.

CALCULUS REMOVAL

Several studies evaluated the effectiveness of calculus removal using ultrasonics, hand curets, or a combination of ultrasonics and hand instruments.

Rabbani et al. (1981) studied the influence of probing depth on the efficiency of calculus removal. Sixty-two (62) teeth were scaled and root planed with hand instruments, and 57 were left untreated and served as controls. The gingival margin was marked on the teeth. The teeth were then extracted, stained with 1% methylene blue, and viewed under a stereomicroscope. The results indicated a high correlation between probing depth and the remaining calculus after scaling. Sites with probing depths less than 3 mm were the easiest to scale and those deeper than 5 mm were the most difficult. Tooth type did not influence the results.

Stambaugh et al. (1981) scaled 42 sites on 7 teeth with an ultrasonic instrument followed one week later by hand curets. Teeth were extracted immediately after hand instrumentation. Measurements were taken before treatment, 1 week after ultrasonic instrumentation, and after extraction of the teeth. The average depth of pocket instrumented to a plaque and calculus free surface "curet efficiency" was 3.73 mm, and was not deeper than 4 mm (range 2.7 to 4.1 mm). The maximum mean probing depth at which evidence could be seen of instrumentation on the root surface was termed "instrument limit" and 6.21 mm, (range 2 to 10 mm). Instrumentation was more efficient on the distal and mesial than on the buccal and lingual surfaces. The results of the study support the surgical debridement and the reduction of pockets in areas of deep probing depth.

Gellin et al. (1986) compared the effectiveness of calculus removal using either a sonic scaler, hand curets, or a sonic scaler plus hand curets. Six-hundred-ninety (690) root surfaces in 11 patients with moderate to advanced periodontitis were studied. The results showed that the percentage of surfaces with residual calculus was: sonic scaler only (31.9%); curets only (26.8%); and sonic scaler plus curets (16.9%). The combination of sonic scaler and curets was more effective in the removal of subgingival calculus than either method used alone. As probing depth increased, the percentage of surfaces with residual calculus increased for all 3 methods.

Kepic et al. (1990) treated 31 teeth by closed scaling and root planing with either ultrasonic (14) or hand instruments (17). After a healing period of 4 to 8 weeks, the teeth were root planed again using the same instruments after flap reflection. The teeth were then extracted and prepared for light microscopic evaluation. Twelve of the 14 teeth treated by ultrasonics and 12 of the 17 teeth treated by hand instruments retained calculus. Hand instrumentation appeared to be more effective than ultrasonics in removing cementum from proximal surfaces. Five blocks were studied under a scanning electron microscope. All 5 specimens displayed residual calculus at either the light microscope, the SEM level, or both. The results indicate that complete removal of calculus from a periodontally diseased root surface is rare.

Sherman et al. (1990) instrumented 476 surfaces on 101 extracted teeth using ultrasonics and hand instruments. The teeth were then evaluated stereomicroscopically for the presence of calculus. The percent surface area with calculus was determined by computerized imaging analysis. Fifty-seven percent (57%) of all surfaces had residual microscopic calculus and the mean percent calculus per surface area was 3.1% (0 to 31.9%). The inter-examiner and intra-examiner clinical agreement in detecting calculus was low. There was a high false-negative response (77.4% of the

surfaces with microscopic calculus were clinically scored as being free of calculus) and a low false-positive response (11.8% of the surfaces microscopically free of calculus were clinically determined to have calculus). This study indicates the difficulties in clinically determining the thoroughness of subgingival instrumentation.

Rateitschak et al. (1992) non-surgically scaled and root planed 10 single-rooted teeth in 4 patients with advanced periodontitis. The teeth were then extracted and examined under SEM. Twenty-nine (29) of the 40 curetted root surfaces were free of residues, if they were reached by the curet. On the remaining 11 surfaces, only small amounts of plaque and minute islands of calculus were detected, primarily at the line angles and also in grooves and depressions in the root surfaces. Instrumentation to the base of the pocket was not achieved completely on 75% of the treated root surfaces. Surfaces that can be reached by curets are usually free of plaque and calculus; however, in many cases the base of the pocket will not be reached. It is for this reason that deep periodontal pockets should be treated surgically.

OPEN VERSUS CLOSED APPROACH

Root instrumentation could be performed using either a closed (non-surgical) or an open (surgical) approach. It is generally agreed that open scaling and root planing gives a better access to the root surfaces and improves calculus removal using either ultrasonics or hand instruments. This is especially true in sites with greater probing depth.

The effectiveness of instrumentation with or without flap reflection was compared by Eaton et al. (1985). Periodontally-involved buccal root surfaces on the anterior teeth of 33 patients were instrumented either before or after the reflection of the flaps. The remaining deposits were stained, then photographed. The findings revealed that root planing under direct vision at the time of surgery was more effective than blind instrumentation. However, in no instance was any root surface found to be completely free of stainable deposits.

Caffesse et al. (1986) found that for 1 to 3 mm pockets S/RP alone and flap plus S/RP were equally effective in obtaining calculus-free surfaces (86%). For 4 to 6 mm pockets 43% of the surfaces were calculus-free when S/RP alone and 76% when flap plus S/RP was performed. In sites greater than 6 mm, S/RP alone obtained only 32% calculus-free surfaces while flap plus S/RP obtained 50% calculus-free surfaces. The extent of residual calculus was directly related to probing depth, was greater following S/RP alone, and was greatest at the CEJ or in association with grooves, fossae, or furcations.

Brayer et al. (1989) distributed 114 periodontally involved, single-rooted teeth among 4 operators of 2 experience levels for either an open or closed session of scaling and root planing. The results showed that there was no difference in scaling and root planing effectiveness for experience level or type of procedure in shallow (1 to 3 mm) pockets. However, in moderate (4 to 6 mm) and deep (≥ 6 mm) periodontal pockets, scaling and root planing combined with an open flap procedure was more effective than S/RP alone for both experience levels. Also, the more experienced operators produced a significantly greater number of calculus-free root surfaces than the less experienced operators in periodontal pockets with moderate and deep probing depths. Best calculus removal was accomplished by experienced operators employing an open procedure.

Parashis et al. (1993) treated 30 mandibular molars with furcation involvement using either a closed or an open approach, or with an open approach using rotary diamond. After extraction, the teeth were assessed under a stereomicroscope and the percentage of residual calculus was calculated on external and furcation surfaces. The percentage of residual calculus on the external surfaces was significantly higher after closed than open root planing. Probing depth influenced the effectiveness of scaling and root planing, with more residual calculus observed for depths equal to or greater than 7 mm for both groups. The most effective method was the combination of open root planing and rotary diamond.

Closed and open scaling and root planing were also compared by Wylam et al. (1993). Sixty (60) multi-rooted teeth were assigned to one of 3 groups: untreated controls, closed scaling and root planing, and open flap scaling and root planing. Following extraction, the mean percent stained surface area was 54.3% in the closed group compared to 33.0% in the open flap group. No difference was found between shallow sites (< 3 mm) and deeper sites (> 3 mm). Examination of furcation regions demonstrated heavy residual stainable deposits for both treatment methods, with no significant differences between techniques.

FURCATION AND ROOT MORPHOLOGY

Root morphology plays a major role when root instrumentation is considered. Multi-rooted teeth with furcation invasion are harder to instrument than single-root teeth. Other anatomical variations such as root grooves, narrow furcation openings, or furcation ridges make complete calculus removal harder if not impossible, even when an open approach is used.

The effectiveness of instrumenting furcation areas was studied by Matia et al. (1986). Forty-eight (48) patients with 50 mandibular molars with severe periodontitis scheduled for extraction were selected. Twenty (20) teeth were instrumented with curets, 10 after surgical exposure (open) of the furcation, and 10 without surgical exposure (closed). Twenty (20) teeth were instrumented with an ultrasonic scaler, 10 teeth open and 10 teeth closed. The remaining 10 teeth were not instrumented and served as untreated controls. The teeth were extracted after instrumentation and the furcations were assessed under a stereomicroscope for residual calculus. The results indicated that calculus removal

in the furcation area is more effective when a surgical flap is utilized, and that the ultrasonic scaler is more effective than the curet in removing calculus in the furcation area utilizing a surgical flap.

Fleischer et al. (1989) compared open and closed scaling and root planing on 50 molars designated for extraction. They found that calculus-free root surfaces were obtained significantly more often with flap access than with a non-surgical approach. Their results suggest that, although both surgical access and a more experienced operator significantly enhance calculus removal in molars with furcation invasion, total calculus removal in furcations utilizing conventional instrumentation may be limited.

The influence of root morphology on the effectiveness of calculus removal was studied by Fox and Bosworth (1987). The mesial and distal surfaces of 168 extracted teeth, representing all tooth types except third molars, were examined to document the presence or absence of proximal concavities. Results showed that teeth from nearly every tooth position, both maxillary and mandibular had concavities at or within 5 mm apical to their cemento-enamel junction (CEJ). It was concluded that proximal concavities are extremely common, the existence of which may complicate restorative and periodontal therapy as well as the patient's ability to maintain effective plaque control.

REMOVAL OF TOOTH STRUCTURE

An excessive amount of tooth structure can be removed during root planing. Special attention should be paid not to overinstrument the roots. Riffle (1953) found that it was impossible to distinguish between curetting cementum and curetting dentin. When dentin was removed a V-shaped ditch was created near the CEJ.

Borghetti et al. (1987) root planed 4 periodontally involved teeth with a curet from 1 to 4 repeated "firm" strokes per surface. Teeth were subsequently extracted, sectioned, and measured for cementum thickness. The results showed that the amount of cementum removed increases with the number of strokes with the curet. Except for coronal areas, cementum was never completely removed; at best was reduced by two-thirds. Root planing seems to be more effective in the coronal areas where the cementum is thinner than in the apical areas. It was concluded that total removal of cementum cannot be accomplished under routine clinical conditions with a curet.

The removal of tooth structure was also studied by Ritz et al. (1991). Three-hundred-sixty (360) sites on 90 extracted mandibular incisors were instrumented with 4 different instruments: hand curet, ultrasonic scaler, air-scaler, and fine grit diamond. Twelve strokes were used with clinically appropriate forces of application. The loss of tooth substance was measured with a device especially constructed for this investigation. Only a thin layer of root substance (11.6 μm) was removed by the ultrasonic scaler, compared to the much greater losses sustained with the air-scaler (93.5 μm), the curet (108.9 μm) and the diamond bur (118.7 μm). The ultrasonic scaler caused the least amount of substance loss while the diamond bur caused the most amount of loss.

Zappa et al. (1991) scaled and root planed 40 extracted teeth. Low forces (mean 3.04 N) were used in 30 teeth and high forces (mean 8.84 N) in 10 teeth. Root substance loss was measured after 5, 10, 20, and 40 working strokes. The results showed that the mean cumulative loss of root substance across 40 strokes was 148.7 μm at low forces, and 343.3 μm at high forces. The results suggest that high forces remove more root substance, and loss per stroke becomes less with increasing numbers of strokes.

ENDOTOXIN REMOVAL

One of the aims of root instrumentation is the removal of endotoxin from the periodontally involved root surface to make it biologically acceptable. Jones and O'Leary (1978) compared 296 root surfaces from 5 treatment groups for the presence of endotoxin. The groups were: subgingival root planing, supragingival root planing, untreated roots with disease, gross scaled roots in vitro, and healthy non-diseased root surfaces. Pooled samples had endotoxin extracted by water/phenol method and assayed for quantity of endotoxin by the limulus lysate test. It was found that the root planed groups (both supra- and subgingival) had far less endotoxin recovered than the gross scaled or untreated groups; the amounts were close to non-diseased tooth levels. It was concluded that root planing was able to render previously diseased root surfaces nearly free of endotoxin, to levels comparable to healthy root surfaces of unerupted teeth.

Nishimine and O'Leary (1979) compared endotoxin removal by hand curets and ultrasonics. Two groups of 46 teeth each were treated, one by curets and the other by ultrasonics, and were compared to 2 control groups, one of 46 untreated periodontally diseased teeth and the other of 31 unerupted healthy teeth. The results showed that thorough root planing with curets produces root surfaces nearly as endotoxin free (2.09 ng/ml) as the surfaces of unerupted healthy teeth (1.46 ng/ml), and that curets are more effective than ultrasonics in removing endotoxin from the periodontally involved root surfaces. Ultrasonics treated root surfaces had 16.8 ng/ml and untreated periodontally diseased surfaces had 169.5 ng/ml.

Gilman and Maxey (1986) compared ultrasonics to ultrasonics plus air powder abrasive for their ability to remove endotoxin. Six teeth were extracted and sectioned into 12 specimens. Test specimens were instrumented with the ultrasonics or ultrasonics plus air powder abrasive. Four calculus-covered control specimens were not instrumented. Eight root specimens were placed in fibroblast tissue culture and were stained for determination of fibroblast viability after 48 hours. No fibroblast growth took place on calculus control specimens. Ultrasonic specimens showed light fi-

broblast growth and viability. Ultrasonics plus air powder abrasive specimens showed superior growth and vitality of fibroblasts.

Assad et al. (1987) studied the chemical removal of endotoxin from the root surface. Twenty (20) extracted periodontally involved teeth were cut into halves bucco-lingually and sterilized. The control half of each tooth was rubbed with saline and the experimental half was rubbed with 2% sodium desoxycholate followed by human plasma. Both groups were then placed in separate petri dishes, with fibroblast cell suspension. The control tooth surfaces showed a mean of 307 ± 63 attached cells. The experimental surfaces exhibited a mean of 650 ± 130 attached cells. The findings suggest that the desoxycholate/plasma combination enhanced in vitro fibroblast attachment to diseased root surfaces.

Nyman et al. (1988) evaluated the effect of removing diseased cementum on healing following surgery. Eleven patients were treated surgically using a split mouth design. In 2 quadrants (control), the teeth were scaled and root planed to remove all cementum. In the remaining quadrants (test), calculus was removed without removal of cementum and the teeth were polished. The patients were followed for 24 months. The results showed that the same degree of improvement was achieved following both types of treatment. There was some gain of probing attachment for both treatment modalities.

HEALING RESPONSE AND THE EFFECT OF THERAPY

The primary goal of periodontal treatment is to arrest the progression of disease, which could be done using hand or ultrasonic instruments and employing a closed or an open approach. The best way to determine which technique is superior in achieving that goal is by evaluating the healing response following treatment.

Tagge et al. (1975) evaluated 3 matched sites in each of 22 patients for the effects of scaling and oral hygiene versus oral hygiene alone. One site served as control, the second received oral hygiene alone, and the third was treated by root planing and oral hygiene. Eight to 9 weeks after treatment, measurements were taken and biopsies were obtained. Microscopically and clinically, scaling and root planing with oral hygiene was shown to be more effective in reducing gingivitis scores, probing depths, and gain in attachment levels than oral hygiene alone.

Hughes and Caffesse (1978) treated 61 teeth in 15 patients by scaling and root planing. Clinical measurements and scores were taken at initial exam, 1 week, and 1 month after treatment. The findings indicated that thorough scaling and root planing of teeth with severe inflammation of the gingiva is commonly followed within 1 week to 1 month after scaling by a decrease in probing depth, gain in attachment, gingival recession, and a decrease in the width of the keratinized tissue. No change in the location of the mucogingival junction occurred after treatment.

Torfason et al. (1979) treated 51 pairs of single rooted teeth with 4 to 6 mm probing depth in 18 patients with either hand or ultrasonic instruments using a split-mouth design. Instrumentation was repeated after 4 weeks. Measurements taken after 8 weeks showed a gradual reduction of probing depth and the number of bleeding sites. There were no significant differences between the two groups except ultrasonic treatment required less time to treat. They concluded that for treatment of 4 to 6 mm probing depth, there is no significant difference between hand instrumentation and ultrasonic in terms of clinical improvement.

Badersten et al. (1981) also found no difference in the healing response following treatment using hand or ultrasonic instruments; 528 tooth surfaces of single-rooted teeth in 15 patients with moderate periodontitis were treated by hand and ultrasonic non-surgical therapy. Improvements in plaque scores, bleeding on probing, decreased probing and attachment levels were similar for both treatment methods. It was shown that shallower sites had a slight loss of attachment while deeper sites showed some improvement.

Badersten et al. (1984A) evaluated the response of deep sites in 16 patients with advanced periodontal disease using hand or ultrasonic non-surgical therapy. Comparable results were obtained by both methods. It was shown that the deep probing depths could be successfully treated non-surgically. It was shown that shallower sites were at risk of losing attachment, while the deep sites were more likely to gain attachment. Deeper residual probing sites were more likely to bleed on probing.

Cercek et al. (1983) monitored 7 periodontitis patients during 3 phases of treatment: 1) toothbrushing and flossing; 2) Perio-Aid used sub-gingivally; and 3) sub-gingival debridement. The mean probing depth of 4.4 mm was reduced to 4.0 mm in phase I; no improvement in phase II; and reduced to 3.2 mm after instrumentation. Clinical attachment level showed a slight loss through phase II, but improved attachment levels were found after instrumentation. Minimal effect was derived from patient performed plaque control, whether supra- or subgingival. The bulk of the effect was derived from professional subgingival instrumentation (scaling and root planing). This is one of the few studies that examines the separate effects of plaque control and that of scaling and root planing on periodontal healing.

Badersten et al. (1985B) studied the incisors, canines, and premolars in 33 patients with generalized periodontal destruction for patterns of clinical attachment loss. Patients received supra- and subgingival debridement after oral hygiene instructions, and were followed for 24 months. Measurements were made every third month and 7 patterns of probing attachment were identified. Seventy-three percent (73%) of the sites showed a gradual loss of probing attachment. Seventeen percent (17%) showed an early loss followed by a stabilization in attachment level. Shallower sites

showed a pattern of early loss followed by stabilization while deeper sites showed a gradual loss.

Claffey et al. (1988) treated 1,248 sites in 9 patients by a single episode of root debridement with ultrasonics. Probing depth and attachment level were measured by 3 different examiners before instrumentation and at 3, 6, and 12 months after treatment. Results showed an initial mean loss of probing attachment of 0.5 to 0.6 mm as a result of instrumentation. Only 5% of all sites lost ≥ 1 mm of attachment from pre-instrumentation to 12 months. Only 2% of all sites lost attachment from post-instrumentation to 12 months. The results suggest that the observed attachment loss was either directly attributable to instrumentation or to a remodeling process as a result of therapy rather than to progressive disease.

OPERATOR VARIABILITY

The effect of operator variability on healing following non-surgical therapy was evaluated by Badersten et al. (1985A). The incisors, canines, and premolars were studied in 20 patients with generalized severe periodontitis. The periodontal pockets were debrided using either hand and/or ultrasonic instruments under local anesthesia by a periodontist or by 1 of a group of 5 dental hygienists. A split-mouth design was used with measurements recorded at the initial examination and every third month. The results indicated that deep periodontal pockets in single-rooted teeth may be successfully treated by plaque control and 1 episode of instrumentation and that operator variability may be limited.

Brayer et al. (1989) found no difference in scaling and root planing effectiveness for experience level in shallow (1 to 3 mm) pockets. However, the more experienced operators produced a significantly greater number of calculus-free root surfaces than the less experienced operators in periodontal sites with moderate and deep probing depths. Fleischer et al. (1989) also found that operators with more experience achieved calculus-free root surfaces significantly more often than operators of lesser experience with both an open and closed procedure.

SINGLE VERSUS REPEATED INSTRUMENTATION

Badersten et al. (1984B) evaluated the effect of single versus repeated instrumentation on healing following non-surgical treatment. Incisors, canines, and premolars were studied in 13 patients with severe periodontitis. Teeth were instrumented using ultrasonic instruments. Instrumentation was repeated in one side of the jaw after 3 and 6 months. A gradual and marked improvement took place during the first 9 months. No differences in results could be observed when comparing the effects of a single versus repeated instrumentation. It was concluded that deep periodontal pockets in incisors, canines, and premolars may be treated by plaque control and one episode of instrumentation.

THE EFFECT OF SCALING AND ROOT PLANING ON THE DENTIN AND THE PULP

Fischer et al. (1991) evaluated the effect of instrumentation on the pulp in 11 patients with periodontally diseased mandibular incisors. The subjects were divided into 2 groups according to marginal bone loss. The pulp sensitivity was evaluated by an electric pulp test. Dentin sensitivity was evaluated with 2 forms of controlled stimulations (probe and air-jet) and with a questionnaire. No changes in pulp sensitivity were found after scaling, but a clinically significant increase in dentin sensitivity to probe and/or air stimuli was observed in 6 patients. A natural mechanism of desensitization seemed to have occurred 2 weeks after subgingival debridement.

Fogel and Pashley (1993) used unerupted third molars in their in vitro study. The crowns were removed and longitudinal slices cut. The hydraulic conductance of the root dentin was measured before and after root planing, acid etching, and potassium oxalate application using a fluid filtration method. The results showed that root planing creates a smear layer that reduces the permeability of the underlying dentin. However, this smear layer is acid labile. Thus, root planing may ultimately cause increased dentin permeability and the associated sequelae of sensitive dentin, bacterial invasion of tubules, reduced periodontal reattachment, and pulpal irritation.

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Section 3. Ultrasonics and Air Abrasives

DEFINITION

Ultrasonic Scaler: An instrument vibrating in the ultrasonic range (approximately 30,000 cps) which, accompanied by a stream of water, can be used to remove adherent deposits from teeth.

PRINCIPLES OF ULTRASONICS

Magnetostrictive units contain a generator that converts 60 HZ, 120-volt current into high-frequency current that continually alters the shape of the magnetostrictive bimetallic stack. As the stack vibrates, the scaler tip vibrates. Ferromagnetic metals (nickel-cobalt alloys) in the stack change length in accordance with alterations in polarity. The resulting 25,000 contractions and expansions per second produce the ultrasonic wave, moving the ultrasonic tip an amplitude of approximately 0.0015 cm. The greater the power setting on the unit, the greater the distance traveled by the tip. Water flow through the tip dissipates heat and produces a cavitational effect. Cavitation is almost an instantaneous release of energy resulting from alternating pressures of the water which is accompanied by rapidly expanding and contracting the air bubbles that collapse in the water. As the bubbles change size at the root surface, they dislodge and wash away debris.

Piezoelectric units produce ultrasonic energy with a crystal system which expands and contracts when an electric current is applied, creating a reciprocal rather than an elliptical motion. The low electromagnetic interference (EMI) level emitted by piezoelectric scalers is not hazardous to cardiac pacemakers; therefore, it is a safe alternative to magnetostrictive scalers (Brown et al., 1987).

Checchi et al. (1991) studied the effect of sharpening on the ultrasonic scaler tip movement. Physical behavior of the scaler was not significantly modified by changes in tip di-

ameter, although the resonant frequency of the tip was changed.

ULTRASONICS VERSUS HAND INSTRUMENTS

Plaque

Thornton and Garnick (1982) compared removal of subgingival plaque by ultrasonic and hand instrumentation. Twenty-four (24) periodontally hopeless teeth were treated by: 1) scaling with hand instruments; 2) scaling with an ultrasonic unit; or as 3) uninstrumented controls. Following extraction and staining, plaque removal was assessed with a compensating polar planimeter. Residual plaque was present on 33% of the surfaces of hand scaled teeth and 34% of ultrasonically scaled teeth. Uninstrumented teeth exhibited 87% total root surface coverage with plaque. Walmsley (1990) showed increased plaque removal due to the cavitation during ultrasonic scaling as compared to ultrasonic scaling without water spray and water spray alone. Baehini et al. (1992) reported no difference in microscopic or cultural data between ultrasonic and sonic instrumentation.

Calculus

Jones et al. (1972) treated 54 teeth scheduled for extraction using curets, scalers, or ultrasonics and then exmined them under a scanning electron microscope (SEM). Twenty-six (26) of the teeth were completely free of calculus after treatment and no difference in the efficiency of calculus removal was observed between the various instruments. The ultrasonic unit caused least damage to the root surface, although scalers and curets caused little damage.

Nishimine and O'Leary (1979) compared the effectiveness of hand instruments and ultrasonic scalers in removing calculus and endotoxin from proximal root surfaces treated before extraction. Visual inspection revealed that 30.4% of ultrasonically scaled teeth had residual calculus compared to 21.7% of teeth root planed with hand instruments. Hunter et al. (1984) compared hand and ultrasonic instrumentation during open flap root planing. Overall, hand-scaled root surfaces demonstrated less residual calculus (5.78%) than ultrasonically-treated surfaces (6.17%). Hand-scaled anterior teeth had less residual calculus (3.55%) on the available surface area than ultrasonically-scaled anterior teeth (5.49%). Conversely, posterior teeth had less residual calculus with ultrasonic scaling (6.87%) than hand-scaling (7.42%). Gellin (1986) evaluated the effect of hand versus sonic instrumentation on the removal of calculus by visually examining the root surfaces during periodontal flap surgery. The percentage of surfaces with residual calculus for each method of instrumentation was: 1) sonic scaler only (31.9%); 2) curets only (26.8%); and 3) sonic scaler and curets (16.9%). The authors concluded that: 1) there was no consistent difference between curets and the sonic scaler; 2) the combination of the sonic scaler and curet instrumentation was more effective than either method alone; 3) as the probing depth increased, the percentage of surfaces with residual calculus increased; and 4) subgingival calculus removal was more difficult in multi-rooted teeth and for proximal surfaces.

Kepic et al. (1990) treated 31 teeth by closed scaling and root planing with either an ultrasonic or hand instruments repeated the instrumentation 4 to 8 weeks later following flap reflection, and extracted the teeth. Light microscope (LM) evaluation indicated that 12 of the 14 teeth treated by ultrasound and 12 of the 17 treated by hand instruments retained calculus. In addition to LM, 5 blocks were evaluated by scanning electron microscope (SEM). All 5 specimens displayed residual calculus at either the light microscope, the SEM level, or both. The results indicate that complete removal of calculus from a periodontally diseased root surface is rare.

Microflora

Leon and Vogel (1987) compared the effectiveness of hand scaling and ultrasonic debridement in furcations. Before treatment, Class I furcations had more coccoid cells and fewer motile bacteria than Class III furcations. Class II furcations had percentages of bacteria between those of Class I and Class III furcations. In Class I furcations, hand scaling and ultrasonic debridement had equivalent effects on the flora with no significant differences between the 2 treatment modalities. When compared to baseline at 2 weeks, both treatments altered the microbiota and gingival crevicular fluid levels to one more consistent with health. In Class II and III furcations, both hand instrumentation and ultrasonics resulted in a bacterial form consistent with health at 2 weeks post-debridement. By 4 weeks, the microbial profile was returning to one consistent with disease. At all times, ultrasonic instrumentation provided greater improvement in microbial parameters than hand instrumentation in both Class II and Class III furcations. The authors suggested that this may be due to better access. Oosterwaal et al. (1987) treated single-rooted teeth and showed that hand scaling and ultrasonic treatment were equally effective in reducing probing depths; bleeding scores; and microscopic counts of rods, spirochetes, and motile forms. In addition, there was a reduction in total colony-forming units and numbers of *Bacteroides* and *Capnocytophaga*, resulting in a subgingival microbiota consistent with periodontal health.

Breininger et al. (1987) compared the effectiveness of ultrasonic and hand scaling in the removal of subgingival plaque and calculus. Both methods were only partially effective in removing subgingival calculus; however, both methods were "remarkably effective" at supragingival plaque removal. When plaque was present after instrumentation, it was usually found in "mini colonies" less than 0.5 mm in diameter. Cuticle-like substances were frequently found on ultrasonic but not hand-instrumented surfaces. Thilo and Baehni (1987) reported vibrations generated by an ultrasonic scaler have the potential to alter the composition of dental plaque and to kill spirochetes in vitro.

Wound Healing

Rosenberg and Ash (1974) studied 58 teeth from 20 prospective denture patients which were assigned to curets, ultrasound, or control groups. Twenty-eight (28) to 232 days after instrumentation and before extraction, plaque scores and labial biopsies were performed. After extraction, root surface roughness was determined with a Profilometer. A statistically significant difference in mean roughness was present between curetted teeth (mean 9.51) and either Cavitron (mean 17.21) and control teeth (18.30). No significant differences in mean plaque scores or mean inflammatory indices were observed between the 3 groups. Root roughness was not significantly related to the mean inflammatory index of the adjacent gingival tissues or to supragingival plaque accumulation.

In a study by Khatiblou and Ghodssi (1983), 18 single-rooted teeth in 12 patients with advanced periodontitis were divided into two groups and modified Widman flaps performed on both groups. In 1 group, shallow horizontal grooves created roughened root surfaces after root planing. The other group served as an unroughened control. Healing was evaluated 4 months after surgery, indicating no significant differences between the groups. Both groups showed attachment gain and reduced probing depth as a result of the surgical treatment. The authors concluded that clinical healing is not affected by varying degrees of root surface roughness.

Torfason et al. (1979) studied 51 pairs of single-rooted teeth with 4 to 6 mm pockets in 18 patients who were treated with either hand or ultrasonic instruments using a split-mouth design. Instrumentation was repeated after 4 weeks. Measurements taken after 8 weeks showed a gradual reduction of probing depth and the number of bleeding sites. For treatment of 4 to 6 mm pockets, there was no significant difference between hand instrumentation and ultrasonics in terms of clinical improvement, although ultrasonic instrumentation required less time.

Badersten et al. (1981) treated 528 tooth surfaces of incisors, canines, and premolars in 15 patients with severely advanced periodontal disease by hand and ultrasonic non-surgical therapy. Improvements in plaque scores, bleeding on probing, decreased probing depths, and attachment levels were similar for both treatment methods. Shallower sites had a slight loss of attachment while deeper sites showed some improvement.

Badersten et al. (1984A) treated 16 patients with severely advanced periodontal disease by hand or ultrasonic non-surgical therapy. Comparable results were obtained by both methods, indicating that the deep probing depths could be successfully treated non-surgically, based on probing depth, probing attachment levels, bleeding on probing, plaque, and gingival recession. Shallower sites were at a risk of losing attachment, while the deep sites were more likely to gain attachment. Deeper residual probing sites were more likely to bleed on probing.

Incisors, canines, and premolars in 33 patients with generalized periodontal destruction were studied by Badersten et al. (1985) for patterns of probing attachment loss. Patients received supra- and subgingival debridement after oral hygiene instruction and were followed for 24 months. Measurements were made after every third month and 7 patterns of probing attachment identified. Seventy-three percent (73%) of sites showed a gradual loss of probing attachment; 17% showed an early loss followed by a stabilization in attachment level. Shallower sites showed a pattern of early attachment loss followed by stabilization while deeper sites showed gradual loss.

Root Surface

Roughness. In a study by Kerry (1967), 180 anterior teeth from 43 patients were divided into 5 groups and the roots were scaled and root planed by curets; Cavitron EW.PP; Cavitron EW.P10; curets followed by ultrasonics; and ultrasonics followed by curets. Following extraction, the relative root roughness was determined with a Profilometer. The smoothest roots were obtained by ultrasonics followed by curets. The roughest roots were produced by the ultrasonic tips. Hand curets produced smoother root surfaces than the ultrasonic instruments.

Pameijer et al. (1972) studied 25 teeth scheduled for extraction and 10 freshly extracted teeth which were treated with either hand or ultrasonic instruments or were left untreated. The 10 extracted teeth were ground flat; polished and then treated with both previous methods. Replicas were made to duplicate the original morphology and topography of the specimens which were then studied utilizing scanning electron microscopy (SEM). No differences were observed in root topography, whether instrumented by ultrasonics or hand instruments. Hand instruments removed substantially more tooth structure than ultrasonics. Instrumentation of a polished dentinal surface by hand instruments, however, removed tooth structure and left a rough surface when compared to ultrasonic instruments.

Hunter et al. (1984) found that 81.2% of ultrasonically-treated teeth were rough (gouges or ripples 50 μm in depth), while only 43.4% of hand-scaled surfaces were graded as rough. Dragoo (1992) compared hand instruments to modified and unmodified Cavitron tips. He reported that the modified tips (reduced in size) produced smoother roots with less damage, better access to the bottom of the pocket, and better plaque and calculus removal than either hand scalers or ultrasonic scalers with unmodified inserts. Less operator time was required and less operator fatigue occurred with modified tips.

Endotoxin. Garrett (1977) suggested ultrasonics followed by hand instrumentation for superior endotoxin removal and production of a smoother root surface. Nishimine et al. (1979) compared effectiveness of hand instruments and ultrasonic scalers in removing endotoxin from root surfaces in vivo using Westphal (phenol-water)

extraction and the limulus amebocyte lysate test. Endotoxin levels reported were: 1) healthy controls (unerupted third molars), 1.46 ng/ml; 2) teeth roots planed with hand instruments, 2.09 ng/ml; 3) ultrasonic-treated teeth, 16.8 ng/ml; and 4) untreated perio-diseased controls, 169.5 ng/ml. Checchi et al. (1988) showed no significant difference in in vitro fibroblast growth between periodontally involved root surfaces treated with curets or ultrasonic scalers. The authors concluded that both treatments resulted in the removal of endotoxin from diseased root surfaces. Chiew et al. (1991) confirmed the superficial location of bacterial toxic products associated with periodontally involved root surfaces. Smart et al. (1990) achieved root surface cleanliness (removal of endotoxin) with a Cavitron and light pressure (50 grams/force for 0.8 seconds/mm^2) on extracted roots with no clinically detectable calculus.

Cementum Removal. Hunter et al. (1984) reported approximately equal amounts of cementum removal by ultrasonics and hand instruments with neither method removing all cementum. Pameijer (1972) stated that ultrasonic instruments will not plane root surfaces while Wilkinson and Maybury (1988) indicated that ultrasonics could remove cementum, but only by producing root damage.

Nyman et al. (1988) treated 11 patients surgically using a split-mouth design. In 2 quadrants (control), the teeth were scaled and root planed to remove all cementum. In the remaining quadrants (test), calculus was removed without removal of cementum and the teeth polished. The patients were followed for 24 months. Results indicated that the same degree of improvement was achieved regardless of treatment and that some gain of probing attachment accompanied both treatment modalities.

Bone. Horton et al. (1975A) studied the effect of ultrasonic instrumentation on bone removal during periodontal surgery. Healing was uneventful with no post-operative complications and minimal patient discomfort. Histologically, no alterations in osteocytes, vascular channels, or underlying periodontal tissues were noted. In another report (Horton et al., 1975B), the authors showed faster healing of surgical defects in alveolar bone with ultrasonics than with rotary burs. Glick and Freeman (1980) found no significant difference in post-surgical bone loss in cats after full mucoperiosteal flap reflection and debridement with either hand instruments or ultrasonics. Three month re-entry surgery revealed 0.333 $\pm$ 0.077 mm mean bone loss with ultrasonic debridement versus 0.329 $\pm$ 0.075 mm mean bone loss with hand instrumentation.

Walmsley et al. (1990) evaluated the effect of cavitational action of the ultrasonic scaler on root surfaces . Using a gold ingot and extracted teeth, the ultrasonic tip was held against the surface and also away from the surface. Photomicrographs and scanning electron microscopy studies revealed that cavitational activity within the cooling water supply of the ultrasonic scaler results in superficial removal of root surface constituents.

Single Versus Repeated Instrumentation. Badersten et al. (1984B) studied incisors, canines, and premolars in 13 patients with severe periodontitis. Teeth were instrumented using ultrasonic instruments, and repeated instrumentation in one side of the jaw was performed after 3 and 6 months. A gradual and marked improvement took place during the first 9 months. No differences in results could be observed when comparing the effects of a single versus repeated instrumentation, suggesting that deep periodontal pockets in incisors, canines, and premolars may be treated by plaque control and 1 episode of instrumentation.

Antimicrobial Lavage. The penetration depth of the water from an ultrasonic instrument into the periodontal pocket was evaluated histologically by Nosal et al. (1991). Patients having teeth planned for extraction and exhibiting probing depths at least 3 mm in depth were used for study. Erythrosin dye was added to the coolant which was delivered to the apical extent of the pocket by vertical movement of the ultrasonic probe tip. After extraction of the tooth, the dye-stained root surface was observed along the full extent of the probe tip's penetration path. The findings indicate that the ultrasonic instrument may be an effective system for both removal of plaque and calculus while simultaneously delivering a chemotherapeutic agent. Limited dispersion of the erythrosin dye in a lateral direction indicates that thorough debridement of the root surface is necessary to adequately deliver chemical agents.

Single-rooted Versus Multi-rooted Teeth. Hunter et al. (1984) compared open flap root planing techniques and reported that hand instruments removed calculus better in anterior teeth, while the Cavitron was more effective in posterior teeth. Leon and Vogel (1987) showed that hand instruments and ultrasonics were equally effective in Grade I furcation; however, ultrasonics were more effective in Grade II and III furcations, based on differential darkfield microscopy and gingival crevicular fluid evaluation parameters. Loos et al. (1987) compared the clinical effectiveness of a single treatment with ultrasonic and sonic scalers using a split-mouth design in 10 patients. Similar changes in clinical parameters were observed for ultrasonic (3.3 minutes/tooth) and sonic scalers (4.0 minutes/tooth).

PREPROCEDURAL RINSING

Fine et al. (1992) reported that preprocedural rinsing with an antiseptic mouthwash (Listerine) can significantly reduce the microbial content of aerosols generated during ultrasonic scaling. Gross et al. (1992) showed no significant difference in mean combined total colony-forming units (CFU) per cubic foot (CF) for magnetostrictive, piezoelectric or air turbine sonic scalers. The magnetostrictive scaler generated the lowest CFU/CF at the deepest level of penetration, but there was no significant difference in level of a simulated lung penetration of the aerosol produced by any of the 3 instruments.

AIR ABRASIVES

Mechanism of Action. Abrasive particles propelled by high-speed air emerge from a point source at the tip of the handpiece. The abrasive powder is composed of sodium bicarbonate treated with 0.5 to 0.8% tricalcium phosphate to improve flow characteristics. The powder is converted into a slurry aerosol at the point source by turbulent mixing with 95 F water spray.

Advantages. Weaks (1984) evaluated an air abrasive unit (AAU) for effectiveness in removal of stain and plaque and its effect on the marginal gingiva, reporting complete removal of extensive stain and plaque in significantly less time (5.5 ± 3.6 minutes) than a rubber cup and pumice (13.4 ± 6.0 minutes).

Disadvantages. Weaks (1984) reported increased soft tissue trauma immediately following use of an air abrasive unit; however, this effect was not detected after 6 days. Finlayson and Stevens (1988) reported oral emphysema following use of an AAU during maintenance of deep periodontal pockets associated with teeth numbers 13 to 15. The complication resolved after 7 days (pen VK 500 mg QID).

Effects of AAU on Root Surfaces. Atkinson et al. (1984) noted that an AAU removed an average of 636.6 set μm (range, 470 to 856 μm) of root structure in 30 seconds of exposure. They also observed "partially obliterated" dental tubules, but found it impossible to distinguish occluded and open tubules. Horning et al. (1987) reported a mean of 80 μm of cementum removed after 40 seconds of exposure of extracted teeth to the air powder spray. The decreased cementum removal in this study may have been due to distribution of the spray over a surface area of 30 mm^2 versus 3/14 mm^2 in Atkinson's study.

Horning et al. (1987) studied 32 teeth scheduled for extraction using flap reflection, ultrasonic scaling, and either an AAU or manual root planing. The ultrasonic plus AAU and ultrasonic plus root planing techniques were similar in plaque, calculus, and cementum removal (both methods left some calculus but no plaque). The study showed no time advantage in using the AAU; however, it was less fatiguing than manual root planing. Clinically, the AAU showed more favorable (clean, white, and smooth) root surfaces including proximal flutings and furcal areas. Compared to hand instrumentation at 200× and 3000× magnification, the AAU-treated surfaces were smoother and had less debris. The AAU treated surfaces often exhibited tiny globular, crystalline particles, assumed to be tricalcium phosphate particles.

Effects of AAU on Wound Healing After Surgery. Pippin (1988) investigated the effects of an AAU on wound healing after periodontal flap surgery in dogs, including abrasive spray on tissue and roots and abrasive powder on bone to study the effect of abrasive spray on tissue. No effort was made to shield the connective tissue side of the flap from overspray and no rinsing was performed, leaving residual material on tooth and tissue surfaces. The flaps in 3 dogs were intentionally sprayed for 5 seconds at a distance of 6 mm. After 2 and 4 days, a fibrinopurulent exudate was associated with the sulcus and bone. Moderate inflammation of bone resolved by 7 and 14 days postoperatively. The effect of the abrasive spray on the root surfaces was evaluated following a 20-second sweeping spray of the buccal surfaces with the handpiece tip held 4 to 6 mm from the root surface. The AAU treated and control sides healed equally well, and no significant difference was observed in inflammatory response. Inflammation was greatest at days 2 and 4, lessened by day 7, with little inflammation present at 14 days post-operatively. To study the effect of AAU powder on bone, each dog also had a separate flap reflected and a 40 mg bolus of dry powder placed directly on the bone. The flap was replaced and sutured. At 2 and 4 days, there was clinical ulceration and partial necrosis of the flap immediately overlying the powder. Histologically, there was acute inflammation and active bone resorption. At 7 and 14 days in general, the inflammatory response to the powder had subsided and few osteoclastic lacunae were present.

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Section 4. Root Conditioning

A primary goal of periodontal therapy is to treat the diseased root surface making it biologically compatible with a healthy periodontium. This includes removing the endotoxins, bacteria, and other antigens found in the cementum of the root surface. A prerequisite for this root preparation is scaling and root planing which was shown by Jones and O'Leary (1978) to remove nearly all detectable levels of bacterial endotoxins. Another form of root conditioning used to help achieve this goal and facilitate new attachment is root surface demineralization. In a review article, Holden and Smith (1983) state that root conditioning was performed as early as 1883 when Marshall placed aromatic sulfuric acid on root surfaces, Younger used lactic acid in 1897, and in 1899 when Stewart decalcified the root surface with pure hydrochloric or sulfuric acid.

CITRIC ACID: HISTOLOGIC RESULTS

Register and Burdick (1975) studied several demineralizing agents for optimum concentration and time of application in gaining reattachment with cementogenesis. Dogs and cats were used as the experimental model and agents tested included hydrochloric, lactic, citric, phosphoric, trichloroacetic, and formic acids and a proprietary demineralizer RDO. Citric and lactic acids and RDO produced slightly wider bands of cementum deposition while trichloroacetic and formic acids stimulated more dentin resorption before cementum deposition. It was determined that citric acid at pH 1 for 2 to 3 minutes would be the best agent. They later showed the formation of cementum pins (perpendicularly extending fiber bundles seen in the tubules at 3 weeks, which appear continuous with and inseparable from the induced cementum at 6 weeks) extending into dentin tubules widened by demineralization when denuded root surfaces in dogs were treated by citric acid pH 1 for 2 minutes.

Garrett et al. (1978) used scanning and transmission electron microscopes to examine the morphological effects of citric acid on periodontally diseased root surfaces. Scanning microscopy showed acid decreased the surface characteristics of non-root planed teeth. Non-etched root planed surfaces were smooth and flat. Acid-etched root planed surfaces were flat with frequent depressions and numerous fiber-like structures. Transmission microscopy revealed root planed and acid-etched surfaces produced a zone of demineralization of 4 μm wide. This zone was dominated by exposed collagen fibrils. Lasho et al. (1983) also showed numerous collagen fibers exposed by the application of sat-

urated citric acid, EDTA, or NaOCl followed by rinsing with 5% citric acid.

Polson et al. (1984) showed by SEM evaluation that root planing leaves an amorphous layer 2 to 15 μm thick which consists of organic and inorganic material. When these surfaces were treated by citric acid (pH 1 for 3 minutes) this smear layer was removed. The result was a fibrous matlike structure with a fibrillar texture having numerous funnel-shaped depressions corresponding to open dentinal tubules. Similarly, Sterrett and Murphy (1989) used SEM photographs to evaluate extracted periodontally diseased-root surfaces that had been scaled and root planed, stored in formalin, and then treated with a 5-minute cotton pellet application of either passively placed or burnished citric acid. They examined the dentinal surfaces for root roughness and maximal exposure of the collagen surface. The smear layer was removed by both treatments. The burnished specimens were found to have patent dentinal tubules and an intertubular area with a very distinct "shag carpet" appearance of deeply tufted collagen fibrils. The passively placed citric acid specimen exhibited open dentinal tubules with a matted collagen surface. They proposed that the burnishing application removed more inorganic material through a combined mechanical/chemical process while fluffing and separating the entangled fixed dentin collagen.

Hanes et al. (1988) evaluated the initial wound healing response to demineralization in the same model as the previous study. They showed that acid-treated teeth had a fibrillar zone 3 to 8 μm thick consisting of collagenous fibrils of the dentin exposed during acid treatment. There appeared to be a layer of cells in dynamic activity and distinct attachment to dentin with cells migrating over the root surface. In the controls, there were large areas devoid of cells and other connective tissue components. This suggests that citric acid treatment may result in fibrin clot stabilization and initiate wound healing that results in new connective tissue attachment.

Fardal and Lowenberg (1990) evaluated in vitro citric acid conditioning compared to EDTA conditioning on fibroblasts cultured on sections of human periodontally involved teeth on migration, attachment and orientation. They found that: 1) root planing improves diseased roots and that root planing followed by citric acid demineralization improves diseased roots to a level comparable to non-diseased roots; 2) citric acid demineralization alone improves diseased roots to a level comparable to root planed diseased roots; and 3) acid demineralization results in both collagen fiber exposure and a more hospitable environment.

Different methods of citric acid application and time have been proposed. Codelli et al. (1991) evaluated citric acid effects upon extracted previously diseased human teeth relative to the duration and method of application. They found that passive applications for 5 minutes and burnished applications for 3 minutes both produced seemingly optimal surface characteristics consisting of a fine, fibrillar network of exposed collagen and a reduced or eliminated smear layer.

Wen et al. (1992) compared different application techniques for citric acid demineralization using scanning electron microscopy. Citric acid pH 1 was applied to dentin surfaces prepared from extracted teeth by 1) immersion; 2) placement of saturated cotton pellets; 3) burnishing with cotton pellets; or 4) camel hair brush. Immersion demonstrated tufting of intertubular dentin fibrils and wide open dentinal orifices. Pellet placement revealed a more matted surface and some debris inside the orifices. Burnishing resulted in a variation of characteristics. Two of 8 slabs showed tufting with widened tubular openings, while 6 of 8 showed surface smearing with complete obturation of the tubules. The camel hair brush resulted in surface characteristics close to those treated by immersion (tufting with widened tubules). Immersion resulted in the greatest number of openings followed by cotton pellet placement and camel hair brush.

Sterrett et al. (1993) examined the effects of citric acid concentration and application time on dentin demineralization. The measurements of calcium parts per million released for citric acid concentrations of 0, 10, 20, 25, 30, 35, 40, and 65% were determined at 1, 2, and 3 minutes. The peak demineralization for 1 minute was 30% (pH 1.55), for 2 minutes was 25% (pH 1.62), and for 3 minutes was 25%. For all concentrations, demineralization was time dependent.

WOUND HEALING AND ATTACHMENT EFFECTS

Animal Histology: Positive Effects

Register and Burdick (1976) examined reattachment with cementogenesis in dogs. Citric acid pH 1 was applied with cotton tip applicators for 2 minutes. Denuded root surfaces healed with cementogenesis with a secure fiber attachment at 6 weeks. However, circumferential and bifurcation defects only healed with approximately 10% reattachment.

Crigger et al. (1978) also studied the effect of citric acid in the dog model. Through and through furcation defects were created and allowed to accumulate plaque for 42 days. The denuded roots were treated with citric acid pH 1 for 3 minutes. These were compared histologically to non-acid treated roots. The controls healed by long junctional epithelium leaving a patent furcation. Thirteen of 23 acid-treated furcations demonstrated complete new attachment; 8 were incomplete and 2 remained patent.

Polson and Proye (1982) also studied the effects of citric acid conditioning in the monkey. Twelve teeth in 4 monkeys were extracted and the coronal third was planed to remove the fibers and cementum. The root surfaces were then treated with citric acid for 3 minutes and then re-implanted into their sockets. They were histologically examined at 1, 3, 7, and 21 days. At days 1 and 3, a fibrin linkage was shown between the periodontal ligament and the root surface. A new connective tissue attachment was present at 21 days with no cementum formation. Extensive root resorption had occurred with some new bone formation.

This led Polson and Proye (1983) to determine the healing sequence related to the fibrin clot and its interaction with collagen. Twenty-four (24) teeth in 4 monkeys were extracted and root planed and 12 teeth treated with citric acid pH 1. They were reimplanted and then biopsied at 1, 3, 7, and 21 days. At 1 and 3 days there was a fibrin network which appeared to be attached to the root surface. Teeth not treated with citric acid had epithelium migrating apically, reaching the crest by day 3, and by day 21 had reached the apical extent of root planing. Those teeth treated with citric acid had collagen fibers replacing the fibrin network by days 7 and 21. The epithelium was located at the CEJ. They concluded that the fibrin network was the initial stage in healing and precedes the collagen attachment.

The importance of the fibrin linkage was also shown by Woodyard et al. (1984). They studied the effects of citric acid on root coverage with pedicle flap procedures in the monkey model. Healing was studied histologically at 0, 3, 7, 14, 21, 28, and 42 days after treatment. Test teeth were treated by citric acid application. They showed the citric acid-treated teeth had a fibrin network while the controls did not. Controls displayed proliferation of the epithelium apical to the notch. Although citric acid treatment did not show enhanced root coverage, it did result in greater amounts of new connective tissue attachment.

Polson et al. (1986) evaluated the cellular, connective tissue, and epithelial response of demineralization on periodontitis affected dentin surfaces. Dentin specimens were obtained from root surfaces covered by calculus. Experimental specimens were immersed in citric acid pH 1 for 3 minutes. All specimens were then implanted into the necks of rats with 1 mm protruding through the skin. Biopsies were prepared at 1, 3, 5, and 10 days for histological examination. Healing of those specimens treated with citric acid occurred by inflammatory cells and fibroblasts in a fibrin network and attached fibers oriented obliquely and perpendicular to the root surface. The non-acid treated specimens showed fewer attached cells with epithelial migration to the apical portion resulting in extrusion. In a similar follow-up study, Polson and Hanes (1987) compared non-periodontitis affected specimens to periodontitis-affected root. Specimens were treated with citric acid pH 1 for 3 minutes and then implanted transcutaneously in the neck of rats. Healing was initiated by a fibrin network which prevented the apical migration of epithelium, allowing fiber attachment in the periodontitis affected specimens. In non-periodontitis specimens healing resulted in a similar attachment. In a follow-up study Hanes et al. (1988) evaluated the initial wound healing response to demineralization in the same model as the previous study. They showed acid treated teeth had a fibrillar zone 3 to 8 μm thick consisting of collagenous fibrils of the dentin exposed during acid treatment. There appeared to be a layer of cells in dynamic activity and distinct attachment to dentin with cells migrating over the root surface. In the controls there were large areas devoid of cells and other connective tissue components. They suggest that citric acid treatment may result in fibrin clot stabilization and initiate wound healing that results in new connective tissue attachment.

Steinberg et al. (1986) studied the effect of various root surface alterations on thrombogenicity and the morphological appearance of initial clot formation. Periodontally-involved human teeth were extracted, sectioned, and reimplanted. One section was immediately removed while the other was removed 1 minute later and examined by scanning electron microscopy. Platelet attachment conditions were examined: 1) intact fibers; 2) periodontitis, no treatment; 3) root planed; 4) root planed plus citric acid; and 5) root planed, citric acid, and collagenase incubation. Platelet attachment was greatest when the intact fiber was present. Citric acid enhanced platelet attachment in the diseased surfaces.

Selvig et al. (1988) also studied the development of attachment on citric acid treated teeth. Eight beagle dogs had fenestration defects created which were treated with citric acid pH 1 for 3 minutes. Biopsies were obtained at 7, 14, and 21 days. They concluded that initial reattachment to an instrumented, demineralized root surface generally takes place by interdigitation between newly synthesized collagen fibrils of the cementum or dentin matrix. In areas of resorption, new fibrils may adhere to the surface of hard tissue without any fibrillar interdigitation.

Wikesjö et al. (1991) studied the effect of citric acid treatment on root resorption. Surgically-created defects were treated in 6 beagle dogs with citric acid or stannous fluoride and the flaps replaced to cover the tooth to the level of the cusp tips. After 12 weeks, histology showed 45% of the defect in the saline treated controls healed by long junctional epithelium; 78% of the defects in stannous fluoride healed by long junctional epithelium, while only 17% of the defects healed by long junctional epithelium in citric acid treated specimens. Control and acid-treated teeth showed similar amounts of root resorption, suggesting citric acid does not enhance or prevent resorption.

Animal Histology: No Effect

Nyman et al. (1981) studied the potential for new attachment in the monkey model using citric acid. Experimental periodontitis was treated by flap and citric acid pH 1 for 3 minutes. The monkeys were sacrificed 6 months after surgery. Root planed alone (controls) and acid-treated teeth resulted in healing by long junctional epithelium. It was determined that citric acid application did not promote formation of new cementum and connective tissue.

Bogle et al. (1981) also provided evidence that citric acid conditioning might not be significant. Citric acid-root conditioning was used in naturally occurring furcation defects in dogs. They found epithelialization of the furcation

fornix in 17/26 defects. Complete new attachment occurred in 2 and incomplete new attachment in 7 defects.

Isidor et al. (1985) failed to demonstrate a difference for citric acid conditioning in the monkey model with orthodontic elastic-induced periodontitis. Histologic sections showed 1.0 mm of newly formed connective tissue for the non-acid treated controls and 1.1 mm for the acid-treated test teeth.

Nyman et al. (1985) also studied the effects of citric acid on root planed teeth that were re-implanted. Five adult monkeys were used, forming 3 groups. Group 1 had teeth extracted and immediately re-implanted; group 2 was root planed and then re-implanted; and group 3 was root planed, treated with citric acid pH 1, and re-implanted. Six months later animals were examined histologically. Immediately re-implanted teeth showed connective tissue reattachment to a level 1 mm apical to the CEJ. Root planed teeth demonstrated apical migration of the epithelium to areas of resorption were ankylosis was present. Teeth root planed and treated with citric acid were similar to the root planed only group. They concluded citric acid had no effect on the healing of reimplanted teeth.

Aukhil and Pettersson (1987) studied the effect of citric acid on cell density. Maxillary canines in 6 dogs were used. Experimental roots were conditioned with citric acid pH 1 for 3 minutes and the dogs were sacrificed after 10 days. They found fibroblast cell density to be less on the acid treated surfaces when compared to controls. It was suggested that citric acid conditioning may result in low cell density during the early stages of healing.

Dyer et al. (1993) used the beagle dog to study the effects of demineralization during guided tissue regeneration. Teeth in 12 quadrants were treated, 4 by citric acid, 4 by tetracycline, and 4 by membrane alone. Histometric analysis demonstrated that root conditioning by either agent did not enhance the amount of connective tissue and bone gained by membrane alone. These results are substantiated by Parashis and Mitsis (1993).

Human Histology: Positive Effects

Cole et al. (1980) examined specimens histologically to determine if new attachment to periodontally-diseased root surfaces could be achieved by topical application of citric acid. Teeth treated by flap procedures had citric acid applied for 5 minutes. Four months later block sections were recovered. In all 10 specimens, connective tissue forming a periodontal ligament extended 1.2 to 2.6 mm coronal from the reference notch.

Albair et al. (1982) also histologically examined the effects of citric acid on formation of new connective tissue attachment. Eight patients requiring extractions for prosthetic reasons were treated by flaps with vigorous root planing. Experimental teeth were treated with citric acid for 5 minutes while contralateral teeth served as non-acid treated controls. Six to 15 weeks later the teeth were extracted and examined by scanning and light microscopy. Six of 9 acid-treated teeth displayed connective tissue coronal to the notch with fibers generally exhibiting a functional orientation. The control displayed a junctional epithelium.

Common and McFall (1983) compared treatment of experimentally-induced human recession using laterally positioned pedicle flap surgery with and without citric acid conditioning. Block sections were obtained at 1, 2, 4, 12, and 20 weeks to observe healing. Citric acid (pH 1) was rubbed onto the prepared root surface for 2 minutes. Control teeth exhibited a long junctional epithelium with no cementogenesis. The citric acid-treated pedicles had a connective tissue attachment to new cementum and, at 1 month postsurgery, did not separate from the teeth as easily as the controls.

Frank et al. (1983) made observations with electron microscopy on teeth treated with citric acid. After treatment by flap procedures, roots were conditioned with citric acid pH 1 for 3 minutes. Sixty-seven (67) days after the surgery the teeth were removed. They determined that two types of connective tissue reattachment occurred. One was splicing of the newly secreted collagen fibrils by mineralization of the decalcified dentin band, while the second involved cementum formation on top of the dentin surface.

Lopez (1984) studied connective tissue healing of periodontally-involved teeth treated by citric acid pH 1 for 5 minutes. Experimental teeth were extracted, cementum removed, treated by citric acid, and then placed in a pouch under the mucosa. They were recovered at 2, 6, 12, 18, 20, and 24 weeks for histological evaluation. At various time intervals they showed resorption, connective tissue attachment to old cementum, and dentin and fibers attached perpendicular to the root surface. They concluded that a new connective tissue attachment could form, even in the absence of periodontal ligament cells.

Human Histology: No Effect

Stahl and Froum (1977) evaluated the effects of citric acid on pocket closure both clinically and histologically. Seven extracted teeth from 2 patients were examined. Root surfaces were treated with citric acid and measurements were repeated at 4, 8, 12, and 16 weeks. Block sections were performed at the 16-week visit. In 5 of 6 citric acid-treated teeth, no evidence was observed of accelerated cementogenesis or functional connective tissue attachment.

Kashani et al. (1984) obtained human histology on citric acid-treated teeth extracted 3 months after surgery. Maxillary anterior teeth planned for extraction were treated with citric acid pH for 1 to 5 minutes. There was no difference on pocket closure between citric acid treated and non-acid treated teeth, which was by long junctional epithelium.

Cogen et al. (1984) compared root planing alone, citric acid alone, and a combination of root planing plus citric acid on fibroblast attachment to diseased roots. Human gingival fibroblasts adhered and grew on root planed surfaces

but not on surfaces treated by citric acid alone. Addition of citric acid treatment after root planing offered no additional fibroblastic attachment compared to root planing alone.

CITRIC ACID: CLINICAL RESULTS

Human Studies: Positive Effects

Cole et al. (1981) examined the effects of citric acid in a pilot study after replaced flap surgery. A split mouth design was used in 12 patients with advanced periodontitis who were treated with citric acid pH 1 for 3 to 5 minutes on the experimental side. A probing attachment level gain of 2.1 mm for the acid-treated teeth resulted, compared to 1.5 mm for controls (60% of the acid-treated areas gained 2 mm of attachment while about 40% of the controls gained 2 mm). The clinical results cannot reveal if improvement is from gain in connective tissue attachment or improved adaptation of the junctional epithelium.

This was followed by a similar study by Renvert and Egelberg (1981) where 13 periodontally involved patients had intraosseous defects treated with citric acid pH 1 for 3 minutes. Six months after surgery, final measurements of probing depth, attachment level, and bone level were carried out. For acid treated teeth there was a gain in probing attachment level of 2.0 mm while the non-acid treated controls showed a gain of 1.2 to 1.3 mm. In 19 of 26 acid-treated teeth gain in probing attachment was 2 mm or more.

Caffesse et al. (1988) treated two sextants in each of 29 subjects with modified Widman flap surgery while another two sextants received the same treatment supplemented with citric acid and fibronectin application. While citric acid/fibronectin application improved probing depth and probing attachment levels to a statistically significant degree, the difference was clinically insignificant (a matter of 0.2 to 0.3 mm).

Human Studies: No Effect

Parodi and Esper (1984) tested the ability of citric acid to promote new attachment and induce bone formation in alveolar defects in humans. Twenty (20) lower molars with Class II and III furcation defects were used. The experimental group was treated with citric acid pH 1 for 3 minutes. At 6 months a re-entry was done to repeat measurements. Results showed a reduction in probing depth (2 to 3 mm) gain in attachment (1 to 1.5 mm), and a gain in bone level (1 mm) for both groups. The results show no difference between acid and non-acid treated teeth.

Renvert et al. (1985) also evaluated the relationship between citric acid conditioning and osseous grafts. They treated 19 patients by mucoperiosteal flaps, debridement, root planing, and citric acid with or without autogenous osseous grafts. They found that osseous grafting did not enhance the results achieved by citric acid conditioning alone and provided results similar to that expected with surgical debridement alone.

Marks and Mehta (1986) evaluated citric acid conditioning (pH 1 for 3 minutes) on 3 patients involving 72 teeth with moderate periodontitis. Results at 12 months showed citric acid did not enhance new connective tissue attachment as measured clinically.

Smith et al. (1986) used a split mouth design to study the effects of citric acid on new attachment during surgery. Experimental sites were treated with citric acid pH 1 for 3 minutes. Clinical attachment levels were evaluated at 3 and 6 months after surgery. There was no difference between acid treated and non-acid treated teeth.

Moore et al. (1987) clinically evaluated the results of citric acid treatment during replaced flap surgery. In a split mouth design, 12 patients had the experimental teeth treated with citric acid pH 0.6 for 3 minutes. Measurements were made from a fixed stent at 3 and 9 months after surgery. They showed that both controls and acid-treated teeth demonstrated gain in attachment levels, but there was no difference between them.

CITRIC ACID EFFECTS ON OTHER TISSUES

Nilveus and Selvig (1983) studied the effects of citric acid on the dental pulp after topical application using 6 beagle dogs. After removal of the alveolar plate, the surfaces were root planed and treated with citric acid or without. Biopsies were obtained after 1 and 15 weeks. It was determined that reparative dentin formed but did not cause inflammatory reactions in the pulp.

Crigger et al. (1983) evaluated the effects of citric acid on exposed connective tissue after flap procedures. Buccal and lingual flaps were raised in 4 dogs. On the test side, citric acid was applied to the inner flap for 3 minutes while the control side was treated with saline. Histology was performed at 3, 7, 14, and 21 days. They demonstrated no irreversible effects resulted on the exposed soft tissues or underlying alveolar bone at any time point.

Ryan et al. (1984) showed a different pulpal response to citric acid treatment while studying cats. Nine cats each provided 1 negative and 1 positive control and 2 experimental canine teeth. Positive controls were treated by surgery only while the experimental teeth received surgery with citric acid conditioning. Positive controls showed mild to moderate short-term and mild to no pulpal reactions long-term. Five experimental teeth became abscessed or necrotic, although 4 teeth were relatively non-inflamed.

Valenza et al. (1987) examined histologically the effects of citric acid on the gingival epithelium. Nine patients had citric acid pH 1 applied locally to the gingiva for 5 to 10 minutes. Gingival biopsies were taken before and after application. Citric acid resulted in edema of the prickle cell layer with disarrangement of the tonofilaments and karyolysis of the nucleus. It was suggested that the alterations may contribute to the prevention of the formation of a long junctional epithelium.

CITRIC ACID: ANTIBACTERIAL EFFECTS

Daly (1982) reported on the antibacterial effects of citric acid. Twenty (20) human teeth affected by periodontal disease were extracted. Ten (10) teeth were immersed in citric acid pH 1 for 3 minutes. Samples from the surfaces were plated on a culture dish. Citric acid treated teeth reduced both aerobic and anaerobic numbers, while there was no difference in numbers before and after saline treatment in the control teeth.

Sarbinoff et al. (1983) also studied the effect chemical treatments had upon endotoxin levels. They found that antiformin alone or in combination with citric acid neutralization resulted in endotoxin levels of less than 1 ng/gm, approaching levels found in undiseased roots. Citric acid alone did not remove endotoxin. Besides the effects upon the root, citric acid may also affect the flora.

Forgas and Gound (1987) compared the effects on dark-field microscopic parameters of root planing alone versus root planing plus antiformin-citric acid application. Both treatments resulted in decreased proportions of spirochetes and motile rods, with no differences between treatments. Microscopic parameters returned to baseline at 12 weeks in both groups.

Tanaka et al. (1989) studied the effects of citric acid on retained plaque and calculus after instrumentation. Five extracted teeth were sectioned longitudinally, and 1 segment was treated with citric acid pH 1 for 3 minutes. Controls showed surface debris and large amounts of bacteria on the retained calculus. Acid treated teeth showed little debris with virtually no bacteria. The surface morphology varied from layered-like to honeycombed.

Corley and Killoy (1982) studied the stability of citric acid solutions used for root conditioning. A solution of citric acid pH 1 achieved by 61 grams of citric acid crystals in 100 ml of distilled water was tested for the effects of light, time, and air exposure. A stable pH was maintained for a 5 month period. They showed that the solution was not affected by time, light, or air exposure.

TETRACYCLINE

Wikesjö et al. (1986) evaluated the effects of tetracycline conditioning on dentin surfaces. Dentin slabs were prepared from extracted bovine teeth. They were immersed in various concentrations of tetracycline solutions for 5 minutes. Morphological effects were compared to slabs treated with saline and inhibition of bacterial growth was tested by inoculating pretreated slabs. Immersion of the slabs removed the smear layer and exposed a regular pattern of open dentin tubules. Maximum binding of tetracycline was greatest with concentrations greater than 50 mg/ml. Maximal bacterial inhibition was achieved at 11 and 33 μm/ml tetracycline.

Terranova et al. (1986) studied the effects of tetracycline root conditioning on cell adhesion, migration, and proliferation. Assays using human gingival epithelial and connective tissue cells were done on dentin blocks prepared from bovine teeth. Tetracycline (TTC) and non-TTC treated slabs were incubated with fibronectin. Maximal binding of fibronectin occurred when slabs were immersed in 100 mg/ml and above of TTC, which varied in a dose dependent manner. TTC also reversed the greater binding of laminin in control specimens. When slabs were treated with TTC and fibronectin there was a 4-fold increase in the attachment of fibroblastic cells. TTC bound 3 times more cells than citric acid and 7 times more than controls.

Alger et al. (1990) used 22 human non-molar teeth with moderate to advanced periodontitis to compare root surface treatments of root planing versus a 3 minute burnished application of tetracycline-hydrochloride (TCN). They also added a 5-minute application of fibronectin (10 mg/ml) to the TCN treatment in a third group. The teeth were removed in block sections at 90 days and examined histologically. New attachment was not found in any of the specimens. TCN was found to result in small amounts of reattachment, which the addition of fibronectin generally inhibited.

Demirel et al. (1991) evaluated the substantivity of doxycycline on disease-affected cementum and dentin by treating prepared root surfaces with 3-minute applications of aqueous solutions of doxycycline HCl in concentrations of 1, 10, 50, and 100 mg/ml. The specimens were then rinsed and incubated for either 10 minutes, or 7 or 14 days in seeded agar containing either *A. viscosus, Actinobacillus actinomycetemcomitans* (Aa) or *Porphyromonas gingivalis* (Pg), with substantivity determined by agar diffusion inhibition assay. Doxycycline substantivity was found to be similar on both dentin and cementum at all concentrations and time intervals. Only the 100 mg/ml concentration of doxycycline produced zones of inhibition in all test organisms at all time intervals, while the 50 mg/ml concentration was effective at all times, except on day 14 with Aa. Aa was found to be most resistant to doxycycline, while Pg was found to be most sensitive. They concluded that cementum and dentin may be capable of acting as reservoirs for doxycycline with its slow release taking place for several days.

Stabholz et al. (1993) assessed in vitro the substantivity of tetracycline. Fifty-one extracted teeth were root planed and then immersed in 10 or 50 mg/ml solutions for 1, 3, and 5 minutes. The 10 mg/ml concentration of TCN showed antimicrobial activity for 4 days while the 50 mg/ml concentration demonstrated antimicrobial activity up to 14 days. Chlorhexidine was also tested (0.12 and 0.2%) and showed activity for only 24 hours.

Parashis and Mitsis (1993) studied the effect of tetracycline (TCN) root conditioning in conjunction with guided tissue regeneration. Controls were treated by expanded polytetrafluoroethylene membranes alone while test teeth

were treated with TCN plus membrane. The change in vertical attachment was 1.7 mm for test teeth and 1.6 mm for controls. The horizontal changes were 4.7 mm for test teeth and 4.8 mm for controls, indicating there was no advantage to TCN when compared to membrane alone.

Lafferty et al. (1993) compared the surface effects of tetracycline and citric acid on periodontally diseased teeth which were extracted and root planed. SEM evaluation demonstrated both agents to be equally effective in removing the smear layer resulting in a similar surface morphology. All specimens demonstrated opened dentinal tubules and a fibrillar matrix with a matted appearance.

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CHAPTER 8. NON-SURGICAL THERAPY

Section 1. Studies

Periodontal therapy can be broadly classified as surgical and non-surgical therapy. Non-surgical therapy includes plaque control, supra- and subgingival scaling, root planing, and the adjunctive use of chemical agents. The purpose of this section is to review longitudinal studies of non-surgical therapy.

The first comparison of surgical and non-surgical therapy was reported by Pihlstrom and coworkers (Minnesota studies). Subsequently there were reports by Ramfjord and coworkers (Michigan studies), Lindhe and coworkers (Gothenburg studies), Isidor and coworkers (Aarhus studies), Becker and coworkers (Tucson-Michigan-Houston studies), and Kaldahl and coworkers (Nebraska studies). Egelberg and coworkers (Loma Linda studies) examined the effect of non-surgical therapy on attachment levels. For all but the Loma Linda studies, a control group or control side was employed.

Some studies used single-rooted teeth only while others included multi-rooted teeth. All studies were done in a university setting except for the Tucson-Michigan-Houston studies which were conducted in a private practice setting.

An inconsistency in the various reports exists regarding the effect of personal oral hygiene. While it is clear to anyone involved in periodontal therapy that the better the personal plaque control, the better the result, it is not clear that perfect plaque control must exist to have a generally successful result (Ramfjord et al., 1982). The Minnesota, Michigan, and Aarhus studies reported that patients with imperfect plaque control fared as well, in terms of attachment level results, as patients with high plaque control scores. The Gothenburg studies reported that plaque-free sites did not lose attachment while plaque-associated sites tended to lose attachment. The Aarhus studies reported that the Gothenburg studies performed only supragingival tooth-cleaning at maintenance visits while the Minnesota, Michigan, and Aarhus groups performed subgingival cleaning during maintenance. The subgingival cleaning apparently helps disrupt the subgingival ecosystem and reduce the pathogenicity of the flora, thereby minimizing attachment loss even in the face of imperfect patient performed oral hygiene efforts. This means that subgingival instrumentation is absolutely essential at maintenance visits.

LONGITUDINAL STUDIES

Minnesota Studies. Pihlstrom et al. (1981), in a 4-year study utilizing multi-rooted teeth, compared scaling and root planing (S/RP) to modified Widman surgery. Seventeen (17) patients received thorough S/RP (RP group) as well as OHI. A modified Widman flap (RP+Sx) was then performed on one half of each subject's dentition. Patients were recalled 3 to 4 times a year for 4 years. The data were separated into 3 groups by initial pocket depth; 1 to 3 mm, 4 to 6 mm, and ≥ 7 mm. Both methods resulted in increased probing depth and loss of attachment in the 1 to 3 mm group. in the 4 to 6 mm group both procedures resulted in reduction in probing depth and maintenance of attachment levels with the RP resulting in slightly more gain in attachment. The ≥ 7 mm group showed the greatest reduction in probing depth and gain in attachment with better results in the RP+Sx procedures. The results indicate that both procedures were effective in treating moderate to advanced periodontitis. The additional flap procedure tended to result in greater probing reduction and attachment gain for deeper pockets. The present study also indicated that it may be possible to arrest the progress of periodontal disease even in the presence of relatively poor plaque control by the patient.

Pihlstrom et al. (1983) in a second report analyzed the 6.5 year results of the previous study. This report concludes that scaling and root planing alone or in combination with modified Widman flap surgery resulted in sustained decreases in gingivitis, plaque, and calculus and neither procedure appears to be superior with respect to these parameters. Seventeen (17) patients diagnosed with moderate-advanced periodontitis were utilized in a split-mouth design study to compare the effects of scaling and root planing alone and combined with modified Widman flap surgery. Data were collected at baseline, 6 months following active therapy and every year up to 4 years, then at 5 1/2 and 6 1/2 years. Probing depth did not change for 1 to 3 mm pockets treated by either scaling and root planing alone or in combination with modified Widman flap surgery. For pockets 4 to 6 mm, both treatment procedures resulted in equally effective sustained pocket reduction. Deep pockets (≥ 7 mm) were initially reduced more by the flap procedure. After 2 years, no consistent difference between treatment methods was found in degree of pocket reduction. For pockets initially 4 to 6 mm in depth, attachment level was maintained by both procedures. Pockets ≥ 7 mm in depth treated by either procedure resulted in a sustained gain in attachment.

Pihlstrom et al. (1984) in a third report examined the response of molar and non-molar teeth to scaling and root planing alone or S/RP plus a flap procedure. At 6 1/2 years, non-molar teeth had an average of about 1.0 mm less probing depth than molar teeth irrespective of type of procedure performed. There was greater probing depth and more apical attachment level on molar than on non-molar teeth treated by either method for 4 to 6 mm pockets. In ≥ 7 mm pockets, the flap resulted in less pocket depth on non-mo-

lars than molars, but no difference in the attachment level for either method. Nineteen (19) of the 453 teeth included in the study were extracted throughout the study; 11 of these were extracted after therapy; 5 maxillary second molars, 2 maxillary first molars, 2 mandibular second molars, 1 deciduous canine, and 1 mandibular lateral incisor.

Michigan Studies. Hill et al. (1981) published a 2-year study of scaling and root planing compared to modified Widman surgery. This 90-patient study included multi-rooted teeth. Following a hygienic phase which included S/RP and OHI, each quadrant was treated by 1 of 4 treatments (pocket elimination, modified Widman flap (MWF), subgingival curettage, and scaling and root planing). Measurements which included pocket depth and attachment levels were taken at the initial exam, after the hygienic phase and 1 and 2 years after treatment. In the 1 to 3 mm crevices there was a slight loss of attachment after all types of treatments. In the 4 to 6 mm pockets there was a significant reduction in probing depth after all modalities with the greatest reductions after pocket elimination and MWF, and a loss of attachment for pocket elimination and a gain for curettage and scaling. In the ≥ 7 mm pockets there was a significant reduction after all modalities with the greatest reduction after pocket elimination, and no significant differences in attachment results among the 4 methods. None of the surgical modalities had any better effect than scaling and root planing alone in maintenance of periodontal support which was not directly related to reduction in pocket depth.

The following 2 studies were part of the Michigan surgery studies but point out the effects of patient-performed oral hygiene measures. Ramfjord et al. (1982) studied 78 patients treated with occlusal adjustment, followed by surgical therapy, and recall prophylaxis every 3 months for 8 years. They were grouped into 3 classes: 1 to 3 mm, 4 to 6 mm, and 7 to 12 mm. Plaque was scored according to the periodontal disease index (PDI). The scores were used to test the hypothesis of equal effect of plaque scores above and below the median for the 3 severity groups of the initial disease based on probing depth. The 25% of patients with the highest plaque scores in 1 group and the 25% with the lowest scores in another group were also compared. These scores were then related to variations in probing depth and attachment levels. The results showed no more return of probing depth with poorer than average oral hygiene than with better than average. A comparison of the 25% of patients having the lowest plaque scores with the 25% having the highest score showed no significant differences in pocket depth responses over the 8 years. After 1 year, there was no indication that poorer oral hygiene leads to a greater loss of attachment than better oral hygiene. Similar results were seen in the 4 to 6 and the ≥ 7 mm group after 4 years of study.

Morrison et al. (1982) in another examination of data from the previously mentioned 8-year longitudinal study analyzed the effect of gingivitis scores on probing depth and attachment levels. For pockets 1 to 3 mm and 4 to 6 mm there was no difference in pocket reduction maintenance. For attachment there was no difference in 1 to 3 mm probing depths and in 4 to 6 mm pockets, lower gingivitis scores had better gain the first 2 years but thereafter no difference was recorded. For 7 to 12 mm pockets, the lower gingivitis scores seemed to result in better probing levels and attachment gain for the first 3 years but this was not maintained throughout the experiment. The severity of gingivitis did not affect the maintenance of pocket depth reduction or clinical attachment levels.

Gothenburg Studies. Lindhe et al. (1982A) reported results of a 2-year study of 15 patients comparing S/RP to modified Widman surgery. Mutilated teeth were included in this study. Patients with advanced periodontal disease were entered into a split mouth design to compare the results of subgingival debridement performed in conjunction with a modified Widman flap or scaling and root planing alone. Scaling and root planing took 6 to 8 hours over a 4-week period. Oral hygiene and the gingival condition in both groups improved significantly. Both treatments resulted in a decrease in probing depth. Initial values were 4.2 and 4.1 mm and decreased to 2.4 and 2.5 (surgery) and 2.9 and 2.8 mm (no surgery). Attachment levels improved following non-surgical therapy at 6 and 12 months, but at 24 months returned to baseline values. Surgical treatment resulted in a slight loss of attachment of 0.3 mm. When comparing single-rooted to multi-rooted teeth, there was a trend for slightly better results for single-rooted teeth. These similar results can be maintained over time in patients with proper oral hygiene levels.

Lindhe et al. (1982B) in another report from the previous study determined the critical probing depth for S/RP and modified Widman surgery. Probing depths shallower than the critical probing depth tend to lose attachment following the procedure. The results showed that the critical probing depth for the S/RP group was 2.9 mm ± 0.4 and for the MWF group was 4.2 mm ± 0.2 which indicates that in patients with a large number of shallow probing depths, a non-surgical approach is preferable, while in patients with a large number of pockets > 4.2 mm, surgical treatment may result in more gain of attachment. The results also showed that the level of oral hygiene established during healing and maintenance is more critical for the resulting probing depths and attachment levels than the mode of treatment used.

Lindhe et al. (1984) reported 5-year results of a continuation of the previous study. The results showed that patients who maintained good oral hygiene had more reduction in probing and a greater gain in attachment than patients who failed to perform good plaque control, indicating that the patients' self-performed plaque control had a decisive influence on the long-term effect of treatment. Sites with initial probing depth exceeding 3 mm responded equally well to non-surgical and surgical treatment.

Aarhus Studies. Isidor et al. (1984), in a 6-month study on single-rooted teeth, compared 3 treatments utilizing a split-mouth design: S/RP versus modified Widman surgery versus reverse bevel flap. Seventeen (17) patients were treated for advanced periodontitis. One side of both the maxilla and the mandible were treated with modified Widman flap. On the other side, one quadrant was treated with reverse bevel flap surgery without osseous recontouring, and the last quadrant was treated with S/RP alone. Patients were recalled every 2 weeks, and examination was performed at 3 and 6 months after the completion of treatment. At 6 months S/RP resulted in considerable reduction in pocket depth, but more shallow pockets were obtained following surgical treatment. Clinical gain of attachment was obtained following all 3 modalities but S/RP resulted in slightly more gain of attachment than the 2 surgical procedures.

Isidor et al. (1985) reported 1-year results of the previous study. Lateral incisors, canines, and premolars in the maxilla and mandible in 16 patients diagnosed with advanced periodontitis were used for study. Each patient was then treated with reverse bevel flap surgery in 1 quadrant, modified Widman flap surgery in 2 quadrants, with the fourth quadrant treated with S/RP. They were then recalled every second week for professional toothcleaning. The plaque index and bleeding on probing were assessed prior to and 3, 6, and 12 months after treatment. Probing depths and clinical attachment levels were assessed prior to and 1 year after treatment. Radiographs were taken using the bisecting angle technique before and 1 year after treatment, and the bone level was expressed as a percentage of the distance from the apex of the tooth to the normal bone level. Angular bony defects corresponding to 15% or more of the distance between the normal level of the bone and the apex of the involved tooth were located. The results of this study indicate that when comparing modified Widman flap surgery, reverse bevel flap surgery, and S/RP for regeneration of alveolar bone, only the modified Widman flap surgery resulted in significant coronal regrowth of bone in angular bony defects.

Isidor and Karring (1986) reported 5-year results of the previous studies. Sixteen (16) patients with advanced periodontitis were subjected to supra- and subgingival scaling and oral hygiene instructions. This was followed by modified Widman flap, reverse bevel flap, or S/RP. Patients were then recalled regularly for the next 5 years Surgical and non-surgical treatment resulted in pocket reduction which was maintained over the 5 years. All methods were effective in halting the progression of periodontitis. No correlation was found between oral hygiene and recurrence of periodontitis, suggesting subgingival scaling at frequent recalls is an important factor in halting the progression of disease.

Tucson-Michigan-Houston Studies. These studies were conducted in a private practice setting in Tucson, AZ. The results reported are essentially the same as those reported by the university studies, thus confirming the validity of university research and its applicability to the private practice setting.

Becker et al. (1988) reported 1-year results of a study comparing S/RP, modified Widman surgery, and osseous surgery utilizing a split mouth design. The study population consisted of 16 patients with 2 or more sites with $\geq$ 6 mm of clinical attachment loss in the posterior dentition. All patients had a baseline examination including the plaque index, gingival index, probing depth, clinical attachment levels, mobility, and furcation status. The probing depths were classified as 1 to 3 mm; 4 to 6 mm; or $\geq$ 7 mm. The clinical attachment level measurements were classified as 0 to 2 mm; 3 to 5 mm; and $\geq$ 6 mm. Quadrants were randomly assigned to 1 of 3 treatment groups: scaling and root planing, modified Widman flap surgery, or osseous surgery. At 1 year post-treatment, osseous and modified Widman surgery had significantly greater probing reduction when compared to scaling and root planing. For pockets $\geq$ 7 mm, osseous and modified Widman surgery had significantly greater reduction when compared to scaling and root planing. For pockets 1 to 3 mm, osseous surgery had significantly greater clinical attachment loss when compared with scaling and root planing. The results indicate that at 1 year, scaling and root planing, osseous surgery, and the modified Widman procedure were equally effective in treating moderate to advanced periodontitis.

Kerry et al. (1990) reported 5-year probing depth results of the previous study. Sixteen (16) patients with moderate periodontitis were treated in private practice by periodontists highly competent in performing scaling and root planing, modified Widman flap, and osseous surgery. Patients were evaluated for 5 years. At the 5-year evaluation, plaque and gingival indices were reduced and maintained throughout the study with no difference between treatment methods; 1 to 3 mm probing depths increased insignificantly but were stable at 3 years; 4 to 6 mm pockets were reduced significantly, but diminished over time. There was a difference between scaling and root planing compared to osseous surgery at 3 and 4 years, but not at 5. Similar trends were found for $\geq$ 7 mm pockets. All 3 procedures reduced pocket depth significantly, with no difference between procedures at 5 years.

Becker et al. (1990) reported 5-year attachment level and gingival recession results of the previous studies. Sixteen (16) patients were treated for moderate periodontitis with either scaling and root planing, modified Widman flap, or osseous surgery. Evaluation were made after the hygienic phase, postsurgery, 6 weeks, 6 months, and at yearly intervals for 5 years. Pockets 1 to 3 mm showed significant loss of attachment; 4 to 6 mm pockets, as well as $>$ 7 mm pockets, showed an insignificant gain of clinical attachment with no difference among procedures. All procedures pro-

duced significant recession postsurgery. It was concluded that all techniques behave similarly regarding clinical attachment levels and gingival recession.

Nebraska Studies. Kaldahl et al. (1988) reported 2-year results of a split mouth design study of multi-rooted teeth that compared supragingival scaling to subgingival scaling to modified Widman surgery to osseous surgery. Eighty-two (82) patients with moderate to advanced periodontitis had each of 4 quadrants randomly assigned to receive coronal scaling (CS), subgingival scaling and root planing (RP), root planing plus modified Widman flap (MW), flap with osseous resection (FO). Approximately 20% of the CS teeth were retreated. The FO group showed the greatest reduction in probing depth followed by MW, RP, and CS. In deep sites MW, RP, and FO demonstrated the largest gain in attachment while CS was the least.

Kaldahl et al. (1990) reported 2-year results of the previous study that compared the site response. Eighty-two (82) patients with moderate to advanced periodontitis were treated in a split mouth design with coronal scaling (CS), root planing (RP), modified Widman flap (MW), and flap with osseous resection (FO), followed by maintenance treatment for 2 years. Four tooth/site groupings were evaluated: 1) interproximal sites of single- rooted teeth (T1); 2) facial and lingual sites of single-rooted teeth (T2); 3) non-furcation sites of molar teeth (T3); and 4) furcation sites of molar teeth (T4). The sites were further subdivided by their initial probing depth severity (1 to 4 mm, 5 to 6 mm, and ≥ 7 mm). The results showed that single-rooted sites ≥ 5 mm had a greater mean probing depth reduction and greater probing attachment gain than did the molar sites. Furcation sites showed a greater increase in probing depth and loss of attachment during the 2 years of maintenance. No therapy had a distinct advantage over another in enhancing the relative response of a particular tooth/site group to the other groupings.

Kalkwarf et al. (1992) reported 2-year results of the 2 previous studies that analyzed patient preference of treatment method. Seventy-five (75) patients were evaluated using an interview after 3 years of maintenance care. Each quadrant in each subject was randomly assigned to 1 of 4 types of periodontal therapy: 1) coronal scaling (CS); 2) CS plus subgingival scaling and root planing (RP); 3) CS/RP followed by modified Widman surgery (MW); or 4) CS/RP followed by flap with osseous resectional surgery. During the hygienic phase of therapy, patients were instructed in plaque control and teeth were instrumented with scalers and curets. Maintenance therapy was performed at 3-month intervals by a dental hygienist. At the conclusion of 3 years of maintenance care, a 7 question interview was conducted with each patient to obtain perceptions regarding the results of therapy in each region of their mouth. The results of this study indicate that the ability of the patient to cope with post-therapy sequelae following either coronal scaling, root planing, modified Widman surgery, or flap with osseous resectional surgery is not significantly different.

Loma Linda Studies. The following may be the only study in the periodontal literature that evaluates the separate effects of oral hygiene and S/RP. Most studies evaluate the combined effect. Cercek and coworkers evaluated the separate effect of: 1) supragingival plaque control; 2) subgingival plaque control; 3) S/RP.

Cercek et al. (1983) reported results of a 2-year study that compared supragingival plaque control to subgingival plaque control to scaling and root planing. Seven patients with chronic periodontitis were monitored during 3 phases of treatment: 1) tooth brushing and flossing; 2) Perio-Aid used subgingivally; and 3) subgingival debridement. Plaque scores ranged from 38 to 99% with a mean of 74% at the initial exam. These scores were reduced to 5 to 15% and were maintained throughout the study. The mean bleeding score of 71.7% was reduced to 40.9% in Phase I, no change in Phase II, and reduced to 23% in Phase III. Deeper sites showed more bleeding than shallower sites throughout the study. The mean probing depth of 4.4 mm was reduced to 4.0 mm in Phase I, no improvement in Phase II, and reduced to 3.2 mm after instrumentation. Probing attachment level showed a slight loss through Phase II, but improved attachment levels were found after instrumentation. An increasing gingival recession was noted during the study. Minimal effect was derived from patient-performed plaque control, whether supra- or subgingival. The bulk of the effect was derived from professional subgingival instrumentation (S/RP).

Badersten et al. (1981) in a 13-month study of patients with moderate periodontitis compared the effect of hand versus ultrasonic instrumentation on attachment levels of single-rooted teeth. Incisors, canines, and premolars in 15 patients with moderately advanced periodontitis were treated by hand and ultrasonic non-surgical therapy. Improvements in plaque scores, bleeding on probing, decreased probing and attachment levels were similar for both treatment methods. It was shown that shallower sites had a slight loss of attachment while deeper sites showed some improvement.

Badersten et al. (1984A) reported 24-month results of a study comparing hand to ultrasonic instrumentation in patients with severe periodontitis. Sixteen (16) patients with severely advanced periodontal disease were treated by hand or ultrasonic non-surgical therapy. Comparable results were obtained by both methods. It was shown that the deep probing depths could be successfully treated non-surgically based on probing depth, probing attachment levels, bleeding on probing, plaque, and gingival recession. It was shown that shallower sites were at risk of losing attachment, while the deep sites were more likely to gain attachment. Deeper residual probing sites were more likely to bleed on probing.

Badersten et al. (1984B) compared the effect of a single

session of S/RP to repeated sessions of S/RP. Incisors, canines, and premolars were studied in 13 patients with severe periodontitis. Teeth were instrumented using ultrasonic instruments, and repeated instrumentation in one side of the jaw was performed after 3 and 6 months. A gradual and marked improvement took place during the first 9 months. No differences in results could be observed when comparing the effects of a single versus repeated instrumentation. Deep periodontal pockets in incisors, canines, and premolars may be treated by plaque control and one episode of instrumentation.

Badersten et al. (1985A) reported a study of the effect of operator variability on the results of the scaling and root planing procedure. Twenty (20) patients whose dentition displayed generalized severe periodontal destruction were selected for the study. The incisors, canines, and premolars in either the maxilla or the mandible were studied. The periodontal pockets were debrided using either hand and/or ultrasonic instruments under local anesthesia by a periodontist or by 1 of 5 dental hygienists. A split mouth design was used with measurements of dental plaque, bleeding on probing, probing depth, and probing AL recorded at the initial exam and at every third month by an examiner not involved with treatment. The results indicate that deep periodontal pockets in incisors, canines, and premolars may be successfully treated by plaque control and one episode of instrumentation and that operator variability between highly skilled clinicians is minimal.

Badersten et al. (1985B) examined patterns of probing attachment loss following scaling and root planing. Incisors, canines, and premolars in 33 patients with generalized periodontal destruction were studied for patterns of probing attachment loss. Patients received supra- and subgingival debridement after oral hygiene instructions, and were followed for 24 months. Measurements were made every third month and 7 patterns of probing attachment were identified. Seventy-three percent (73%) of sites showed a gradual change. Seventeen percent (17) showed an early loss followed by a stabilization of attachment levels. Shallower sites showed a pattern of early loss followed by stabilization while deeper sites showed a gradual loss.

SUMMARY

Non-surgical therapy is an effective method of periodontal therapy. When proper results are not achieved, surgical treatment should follow. Some guidelines are furnished by Lindhe et al. (1982B) in the critical probing depth study.

All research groups found non-surgical therapy to be effective in molar and nonmolar teeth, in shallow and deep sites and whether the study was conducted in a university or private practice setting.

Repeated instrumentation was of little benefit because calculus was often missed on the second attempt for the same reason it was missed the first time.

The primary caveat with non-surgical therapy is that there are sites and even patients where it may not be effective. This must be recognized at the reevaluation appointment and appropriate therapy, probably surgery, should be instituted.

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Section 2. Microbiologically Monitored and Modulated Periodontal Therapeutics

INTRODUCTION

In the late 1970s and early 1980s, a non-surgical approach to periodontal therapy was developed and introduced by Dr. Paul Keyes (Keyes et al. 1978A, 1978B, 1982 and 1985). Microbiologically monitored and modulated periodontal therapeutics (MMPT) were described as a method of identifying therapeutic objectives and targets. The treatment was to be predictable, based on an antimicrobial approach. Whenever therapeutic endpoints were not attained, adjustments in therapy (additional S/RP, irrigation, systemic antibiotics, surgery) were to be used. The procedural steps of MMPT include: 1) clinical examination and microbial assessment consisting of phase contrast microscopic examination of subgingival plaque obtained from 2 or 3 of the most severely diseased sites using a sterile curet. Health and disease were distinguished by the presence of motile bacteria and > 125 white blood cells (WBC) per field; 2) patient education involving phase contrast visualization of the plaque samples on a TV monitor; 3) professional treatment including instrumentation of the teeth (without anesthetics) and irrigation with an antimicrobial solution (chloramine T, a topical sulfonamide agent with hypochlorite action); 4) patient training in oral hygiene measures including use of antimicrobial substances mixed into a thick paste (hydrogen peroxide, sodium bicarbonate, and salt water delivered by an irrigation device); 5) reevaluation including microbiological monitoring and clinical examination; and 6) recall for microbiologic monitoring and for areas of unresolved disease modulation of therapy in the form of additional S/RP, irrigation, systemic antibiotics (usually tetracycline or metronidazole), and surgery if antimicrobial therapy fails.

The rationale for the technique is based on the concept that certain bacterial complexes (motile forms and spirochetes) are not compatible with health. These bacterial populations can be prevented or suppressed by appropriate therapy and when controlled, progressive destruction abates. Motile bacteria, spirochetes, and WBCs can readily be detected by phase contrast (PCM) or darkfield microscopy (DFM). Keyes used non-dispersed samples to preserve colony characteristics and architecture, which were examined by PCM and assigned a value (+, ++, +++, ++++).

In an attempt to validate the use of PCM, Keyes and Rams (1983) studied several groups of subjects without controls. Healthy sites had no spirochetes, large motile rods, brush forms, or protozoa. Non-motile complexes of cocci and filamentous forms or corn-cob formations were seen. WBCs were present in low numbers (< 5/field). Gingivitis cases had large numbers of smaller rods and cocci, no protozoan species, and few WBCs. Severe periodontitis patients harbored large numbers of spirochetes and motile rods that were organized into brush forms or rosettes. Also present were *Entamoeba and Trichomonas* species and large numbers of WBCs (> 125/field). According to MMPT, floras associated with health and disease may be distinguished on the basis of bacterial type, number, and organization, coordinated behavior between organisms, and the inflammatory potential as indicated by the number of leukocytes present. The authors believe that MMPT may help identify persons at risk of developing destructive periodontal disease.

Rams et al. (1985) studied 47 adults with advanced periodontal disease without controls. Long-term patient followup after 3 and 6.5 years of treatment via MMPT showed tooth mortality of 0.6%, a decrease of bleeding on probing from 41% to 1.3%, and slight pocket depth reduction for pockets > 3 mm. It is significant that 46/47 subjects proved "refractory" to the Keyes theory, requiring adjunctive systemic antibiotics, often on multiple occasions.

CHEMICAL AGENTS OF MMPT

Sodium bicarbonate was evaluated in a study by Newbrun et al. (1984) which also determined the in vitro effectiveness of sodium fluoride and magnesium sulfate against 11 oral microorganisms. They found that subgingival microbes (except *F. nucleatum* and *P. gingivalis*) were inhibited at lower salt concentrations than supragingival bacteria. The bicarbonate ion seemed to be responsible for inhibiting bacterial growth. Bactericidal concentrations of sodium bicarbonate required 30 to 120 minutes contact to kill 99% of the bacteria. Although sodium bicarbonate is not as potent as sodium fluoride, sodium lauryl sulfate, or chloramine T, it is readily available, inexpensive, and safe. The ability to retain bactericidal concentrations in the subgingival environment for 30 to 120 minutes is questionable.

The use of sodium bicarbonate combined with hydrogen peroxide was evaluated by Fletcher et al. (1984), who reported that several *Streptococci* and *Mycoplasma* were susceptible to 0.5% hydrogen peroxide alone. The combination of sodium bicarbonate with the peroxide diminished the antibacterial activity from 2 to 16X, with 5% sodium bicarbonate failing to inhibit these organisms.

Weitzman et al. (1984) questioned the chronic use of hydrogen peroxide due to potential carcinogenicity, co-carcinogenicity, and tumor promotion potential that has been reported in experimental animals.

Keyes and Rams (1984) studied the potential antibacterial effects of periodate salts on microbes obtained from one periodontitis patient. These salts are oxidizers which release

iodine. In vitro exposure to periodate salts for 1 minute eliminated all spirochetes and rods.

REVIEW ARTICLES

Greenwell and Bissada reviewed the controlled evaluations of Keyes' method and concluded there was no scientific evidence for any effect beyond that obtained with conventional non-surgical therapy. Microscopic evaluation of bacterial morphotypes had not been shown to provide clinically useful information in terms of periodontal diagnosis. The frequent use of antibiotics was considered generally unnecessary and was not recommended on the broad scale advocated by Keyes.

To review the role of microscopic monitoring in detecting periodontal disease, Greenstein and Polson (1985) discussed several articles. Generally, microscopic monitoring can demonstrate differences in the subgingival microflora, but it is not clear whether the organisms monitored are involved in the pathogenesis of periodontitis or are merely co-habitants of deeper pockets. It is impossible to determine threshold values of bacteria that initiate disease, and the ability to predict attachment loss based on spirochetes is not consistent. Host susceptibility may be a more important factor than the determination of bacterial morphotype. Until the relationship between organisms and pathogenesis has been clarified, chairside microscopic monitoring of bacterial populations has not been shown to provide clinically useful information and must be interpreted cautiously.

Valentine (1985) reviewed literature related to each step of the Keyes' technique and concluded that the benefits of the technique are almost exclusively derived from the detailed oral hygiene procedures and root planing. Little or no sound evidence was found to support the use of the antimicrobials during any step of the treatment in adult periodontitis.

Omar and Newman (1986) discussed several inherent problems associated with the various stages of darkfield microscopy. Problems in sampling include: 1) variability between and within individuals of morphotype counts from sites with comparable pocket depths; 2) contamination of the sample; 3) reduction of sample volume after scaling; and 4) difficult interproximal access. Problems in dispersion include: 1) contamination; 2) uneven distribution of different morphotypes; and 3) destruction of delicate organisms. Problems in slide preparation include contamination, logistical difficulties, and time of cell viability. Problems in identification and counting include: 1) Brownian movement versus true motility; 2) fragmented bacteria; 3) confusion of *Campylobacter* with spirochetes; and, 4) confusion of flagella with flagella-like structures. Problems in morphotype grouping include lack of motility in many periodontopathogens or inability to consistently demonstrate motility in all motile species.

The current position of the Academy regarding MMPT is outlined in a 1994 position paper (American Academy of Periodontology). The Keyes' technique consists of current controversial periodontal therapy to which microscopic monitoring, increased use of systemic antibodies, and use of local chemical agents have been added. These additional approaches do not appear to offer increased benefits over conventional periodontal therapy for cases of adult periodontitis and may, in certain situations, offer substantial negative effects. New technologies for specific bacterial identification, new diagnostics, and new chemotherapeutic agents offer great possibilities for enhanced management of periodontal diseases. Such technologies must be assessed in controlled, long-term studies to determine added advantages over existing conventional tecnhiques prior to routine use.

SHORT-TERM STUDIES (6 MONTHS OR LESS)

Cerra and Killoy (1982) reported on a 21-day study using a split-mouth design and no S/RP. Four patients with 4 to 7 mm pockets were examined for microbial flora on days 1 and 21 of this experiment. They were taught the Bass method of brushing and instructed to apply sodium bicarbonate and 3% hydrogen peroxide to one side of the mouth and fluoridated toothpaste to the other daily for 1 minute. There were no statistically significant differences between test and control sides. Pocket depth for the test on day 1 was 5.19 and at day 21 was 4.19; for the controls, it was 4.75 on day 1 and 3.88 on day 21. Bacterial counts were not reduced for any morphotypes for either test or control sides.

Greenwell et al. (1983) compared the effect of Keyes' method of oral hygiene with conventional oral hygiene during an 8-week study, using a split-mouth design. Eighteen (18) patients were divided into 2 equal groups, 9 with moderate-severe periodontitis and 9 who had previously received pocket elimination surgery. Based on PI, GI, gingival fluid flow, darkfield microscopic counts, probing depth, and bleeding on probing, no significant differences were seen between the 2 oral hygiene methods. The authors stated that patients treated surgically had significantly better periodontal health than those treated non-surgically.

Greenwell et al. (1985) compared the Keyes' oral hygiene technique to conventional oral hygiene in non-treated subjects, those receiving scaling and root planing only and patients treated surgically. Based on clinical and microbiological indicators, results revealed no statistically significant differences between Keyes' technique and conventional oral hygiene in areas treated with a single session of S/RP. In untreated areas (no S/RP) Keyes' method was more effective than conventional oral hygiene. Surgical status was the most significant factor in reducing clinical and and bacteriological indicators. The results indicated that the Keyes' technique should not be relied on to control the pathogenic subgingival microflora.

West and King (1983) compared toothbrushing with sodium bicarbonate-hydrogen peroxide (Keyes) to toothpowder and water to determine reduction of pocket suppuration

and darkfield bacteria counts. After S/RP in each group, suppuration was reduced about 70%. The sequential or concurrent mode of therapy had no impact on treatment results.

Wolff et al. (1982) reported on a 2-group, 62-patient, 16-week study which evaluated the effectiveness of the antimicrobial home treatment advocated by Keyes using phase contrast microscopy. No differences were detected between conventional oral hygiene measures and those associated with the Keyes' technique at 8 and 16 weeks.

In a study by Rosling et al. (1982), 20 patients with moderate to severe periodontitis were given oral hygiene instructions and a supragingival cleaning, and were then randomly assigned to test or control groups. Half the mouth was subgingivally scaled under local anesthesia. The test group received a bicarbonate-peroxide mixture professionally applied subgingivally and irrigation with Betadine. Controls were irrigated with saline. For 3 months, each group received a professional cleaning every 2 weeks. The PI scores were similarly reduced for both groups. The GI scores were lower in the scaled than the unscaled quadrants at 3 and 6 months. Probing depths in 4 to 6 mm pockets were reduced in scaled areas of tests and controls. Gains in clinical attachment level (CAL) were greater in the scaled test sites than in scaled controls. The reduction in bacterial counts was greater than controls in both scaled and unscaled quadrants. The professional application of the baking soda-peroxide mixture differed from the typical patient application and may have accounted for the significant differences found in this study.

LONG-TERM STUDIES (1 TO 4 YEARS)

Pihlstrom et al. (1987) studied 231 patients who received scaling and root planing and were then assigned to 4 groups: 1) conventional oral hygiene; 2) conventional oral hygiene plus microscopic viewing of subgingival microbial flora; 3) the use of sodium bicarbonate, hydrogen peroxide, and sodium chloride (regimen S/P); and 4) S/P plus microscopic viewing of the subgingival microbial flora. After baseline clinical data collection and plaque sampling were completed, subjects in all groups received a professional scaling, root planing, and tooth polishing. Microbial and clinical data were collected at baseline, and at 8, 16, and 24 months from selected teeth. Clinical results showed that both procedures effectively reduced clinical signs of disease when combined with professional care, with no difference between the two regimens.

In a report of the microbiological results, Wolff et al. (1987) noted that there was no effect on spirochetes observed between subjects who did or did not view their plaque microscopically, when sites were stratified into molars and non-molars. There was no significant difference between frequency of reinstrumentation between sites monitored clinically or microscopically.

A study of patient compliance results by Bakdash et al. (1987) indicated that 23% of patients using conventional oral hygiene and 43% on the Keyes' regimen cited inconvenience as the reason for non-compliance; 74% of conventional and 58% of Keyes' groups used the asssigned regimen 4 to 7 days per week. There was no evidence that microscopic viewing of plaque was a significant motivating factor.

In a followup study, Wolff et al. (1989) evaluated 171 subjects. Higher levels of compliance were observed in the groups using conventional oral hygiene procedures. There were no significant differences in clinical health betwen the salt and peroxide as compared to the conventional oral hygiene groups.

Rosling et al. (1983) reported on 20 patients who received supragingival scaling in all 4 quadrants, then subgingival scaling and root planing in 2 quadrants only. The test group received professional application of a mixture of H_2O_2-NaCl-$NaHCO_3$ which was irrigated by a water solution of Betadine (test group); the control group was irrigated only with saline. Home care instruction included the use of the mixture by the test group and the use of a regular toothpaste by the control group. Parameters studied included: plaque index, gingival index, probing depth, attachment levels, subtraction radiography for changes in bone density, and microbiological examination. The 2-month results indicated that professional and personal use of the mixture will significantly enhance the microbiological and clinical effects of periodontal scaling and root planing. The professional application of the baking soda-peroxide mixture differs from the typical patient application and may account for the significant differences found in this study.

Rosling et al. (1986) studied the use of topical antimicrobial therapy and diagnosis of subgingival bacteria in the management of inflammatory periodontal disease. Sixty (60) patients, diagnosed with moderate to severe periodontitis, were included in 3 treatment studies. All patients (10 per test and control) were examined at baseline and at 3, 6, and 12 months after completion of treatment. Clinical parameters assessed were the PI, GI, PD, and clinical attachment level (CAL). Standardized radiographs were taken to detect changes in alveolar bone mass and microbiological samples were collected from diseased areas and assessed for the occurrence of *A. actinomycetemcomitans* and *P. gingivalis*. Study A compared subgingival scaling plus a topical antimicrobial to subgingival scaling alone. Study B assessed the effects of scaling alone, scaling plus surgery, and scaling, surgery, and topical administration of a 0.5% Betadine solution. Areas with gain of CAL demonstrated BOP in 24% of sites while the frequency of no bleeding units in areas with loss of CAL was 81%. *A. actinomycetemcomitans* and *P. gingivalis* were identified in 5 of the 8 progressing periodontitis lesions. In contrast, these organisms were identified in only 8 of the 28 lesions with no apparent change in the CAL. Neither of the organisms was present in the sites showing a gain in CAL. The results indicate a role for topical microbial agents as an adjunct to

mechanical subgingival debridement in the treatment of adult periodontitis. The professional application of baking soda-peroxide mixture differs from the typical patient application and may account for the significant differences found in this study.

SUMMARY

In summary, MMPT has no benefit beyond that obtained from S/RP alone. The only controlled studies that have shown a significant difference between conventional and Keyes' regimens employed professional application of the baking soda mixture. Results were similar between short-term and long-term studies and between 2-group and split-mouth designs. The literature is in agreement that Keyes' oral hygiene agents have no benefit beyond that of conventional methods. Microscopic diagnostic methods provided no advantages over conventional methods.

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CHAPTER 9. SURGICAL THERAPY

Section 1. General Principles

DEFINITIONS

Surgery: That branch of medical science concerned with the treatment of diseases or injuries by means of manual or operative methods.

Periodontal Surgery: Any surgical procedure used to treat periodontal disease or to modify the morphology of the periodontium.

GOAL OF PERIODONTAL SURGICAL THERAPY

To restore health and function to the periodontium and to preserve teeth for a lifetime (Kakehashi and Parakkal, 1982).

INDICATIONS FOR PERIODONTAL SURGICAL THERAPY

Indications for periodontal surgical therapy may include the following (Barrington, 1981):

1. Access for root debridement;
2. Elimination of pockets by removal and/or recontouring of soft or osseous tissues;
3. Removal of diseased periodontal tissues creating a favorable environment for new attachment and/or readaptation of soft and/or osseous tissues;
4. Correction of mucogingival deficiencies or defects (e.g., root coverage, increase zone of keratinized tissue, ridge augmentation);
5. Establishment of tissue contours that facilitate oral hygiene maintenance;
6. Establishment of esthetics by reducing soft tissue sites of enlargement-overgrowth or by augmenting sites with soft and/or hard tissue deficiencies;
7. Creation of a favorable restorative environment;
8. Establishment of drainage or emergent periodontal problems (e.g., gingival or periodontal abscess);
9. Determining or improving treatment prognosis (including exploratory procedures;
10. Biopsy and diagnosis; and
11. Regenerative procedures.

ANATOMIC CONSIDERATIONS IN PERIODONTAL THERAPY

Osseous Structures

The maxilla may be described as a "hollow bony box" consisting of the following four processes: 1) frontal; 2) zygomatic; 3) palatal; and 4) alveolar. The maxillary sinus occupies the entire body of the maxilla and may extend into the zygomatic and alveolar processes. This may be clinically significant since no medullary component may exist between the cortical bone investing the teeth and the sinus proper. The sinus may also extend into edentulous areas (pneumatization). The bony crest on the lateral surface of the maxilla is termed the zygomaticoalveolar crest. This bony ridge determines vestibular height in the maxillary molar region. Palatal tori may be present at the midline of the hard palate while smaller exostoses are frequently observed over the palatal roots of the molars (Clarke and Bueltman, 1971).

The mandible is a horseshoe-shaped bone which is grossly characterized by the mental protuberance, body, and ramus. It contains paired foramina (F) per side (inferior alveolar F; mental F) which transmit neural/vascular structures, bearing the same names. Other important landmarks include the mylohyoid ridge, genial tubercles, temporal crests, alveolar processes, and external oblique ridges (Clarke and Bueltman, 1971).

Vascular Supply

The vascular supply of the periodontium originates from branches of the external carotid artery. The main branches which supply structures in the oral cavity are the lingual, facial, and maxillary arteries. The inferior alveolar and greater palatine arteries are branches of the maxillary artery (Clarke and Bueltman, 1971).

The blood supply of the gingiva is derived primarily from supraperiosteal vessels which represent terminal afferent branches of the following arteries: 1) sublingual; 2) mental; 3) buccal; 4) facial; 5) greater palatine; 6) infraorbital; and 7) the posterior superior dental. These vessels anastomose with those supplying the alveolar bone and periodontal ligament. Prior to entering the apical foramina respective dental arteries (branches of superior or inferior alveolar dental artery) are the originating sites of the intraseptal arteries. Coursing coronally, these alveolar vessels provide numerous lateral/terminal branches (rami perforantes) which traverse the lamina dura at all levels, anastomosing with vessels in the periodontal ligament (PDL) space which also originate apically. The PDL vessels derive apically from the dental artery (previously described) and rami perforantes coursing into the PDL space forming a circumferential net. A plexus of vessels with numerous venules (dento-gingival plexus) is located beneath the junctional epithelium; in health, capillary loops are not found in this plexus. In contrast, the subepithelial plexus of the free and attached gingiva manifest capillary loops (7 μm) which supply individual connective tissue papilla. While a basic understanding of the periodontal vasculature is facilitated by reviewing individual anatomic sources, this unit

actually represents a functional vascular syncytium which significantly impacts the technical provision of periodontal therapy (Klaus et al., 1989; Schluger et al., 1990).

Innervation

The oral cavity is innervated primarily by branches of the trigeminal nerve (5th cranial nerve [CN]). The sensory portion supplies the skin of the face, oral mucous membranes, and the teeth. The motor portion supplies the 4 paired muscles of mastication, and the mylohyoid and digastric muscles. The maxillary (second) division of the trigeminal nerve sends anterior, middle, and posterior superior branches to the maxillary teeth. The infraorbital, nasopalatine and the greater palatine nerves supply sensation to areas of skin and mucous membrane. The mandibular (third) division branches are the buccal, lingual, inferior alveolar, and mylohyoid nerves. The terminal branches of the inferior alveolar nerve are the mental and incisive nerves (Clarke and Bueltman, 1971).

Musculature

The four primary muscles of mastication are innervated by the mandibular division of the trigeminal nerve. The primary function of the temporalis, medial pterygoid, and masseter muscles is elevation of the mandible, while the lateral pterygoids are mainly responsible for protrusion. These muscles work in concert with the accessory muscles of mastication allowing coordinated, functional mandibular movements. The buccinator (also considered a muscle of facial expression) is innervated by the facial nerve (7th CN). The anterior digastric muscles help depress the mandible and are innervated by the mandibular division of the 5th CN. The mylohyoid functions to depress and retract the mandible. The geniohyoid has a similar function as the mylohyoid and is innervated by the cervical plexus (DuBrul, 1980).

Anatomic Spaces

Potential anatomic spaces of the oral cavity are found within subcutaneous or submucosal connective tissues and sites delineated by fascial membranes which may allow communication with the orbit, the neck, and the mediastinum. These spaces are the canine, buccal, masticator (pterygomandibular), mental, submandibular (made up of the submental, sublingual, and submaxillary spaces), lateral pharyngeal, and retropharyngeal (parapharyngeal) spaces (Clarke and Bueltman, 1971).

Surgical Anatomy

A detailed understanding of surgical anatomy is essential if complications during periodontal surgery are to be avoided. The depth of the vestibule in the mandibular anterior region may be limited by the attachment of the mentalis muscle and prominence of the mental tuberosity. An unusually high or large genial tubercle may impede osseous recontouring in the area. The extent of the external oblique ridge may also limit the surgical treatment of intrabony defects, or make apical positioning of flaps difficult. The vertical bony prominence of the mandibular ramus may limit treatment possibilities for the distal aspect of terminal mandibular molars. On the lingual aspect of the mandible, incision of the lingual nerve and/or lingual artery must be avoided. Surgical manipulation of the tissues in this area can generally be safely accomplished by careful reflection of a full thickness flap. Perforation of the periosteum and damage to structures within the flap can be avoided by following the lateral flare of the mandible in this region maintaining bony contact during tissue retraction and elevation (Clarke and Bueltman, 1971).

The maxillary sinus closely approximates the roots of the maxillary molar teeth and should be noted radiographically when considering extensive osseous recontouring, regenerative procedures or placement of implants. The greater palatine artery must be avoided during flap reflection or graft (hard or soft tissue) harvesting in this region. Vertical incisions in the posterior palate should be avoided. Prominent palatal exostosis or a flat palate may render osseous interproximal ramping difficult (Clarke and Bueltman, 1971).

CLINICAL CONSIDERATIONS

General risk factors accompanying periodontal surgical therapy include hemorrhage, transient bacteremia, stress, and infection.

Flap design and incisions of the envelope type are adequate for most situations. Vertical incisions have limited use, but when used judiciously may be helpful; they are best avoided on the posterior palate and mandibular lingual areas (Clarke and Bueltman,1971; Hunt, 1976).

Excessive hemorrhage may be controlled by direct pressure, vasoconstriction from the local anesthetic solution, suture ties, and burnishing the offending vessel against bone. Synthetic hemostatic agents may also be used. Longer procedures tend to produce more blood loss. Baab et al. (1977) studied blood loss (BL) during periodontal flap surgery, reporting mean loss of 134 ml (16 to 592 range) per site. Duration of surgery and amount of local anesthetic used were significantly correlated with BL; however, there was no correlation between number of teeth in the surgical field or length of incisions. For procedures less than 2 hours, no more than 125 ml BL occurred. Mandibular surgery was associated with greater BL (151 ml) when compared to maxillary surgery (110 ml). IV fluid replacement was recommended when BL exceeds 500 ml or if orthostatic hypotension occurs (i.e., drop in systolic BP of 20 mm or diastolic of 10 mm).

Nerve trauma may occur in several ways. Incision during flap reflection may result in paresthesia of the lip or tongue. Damage to the inferior alveolar nerve may occur during preparation for implant placement or during placement of the implant itself. Nerve damage may also occur

as a result of post-surgical infection or progressive pathosis. General safety factors include a thorough understanding by the surgeon of the bony and soft tissue anatomy in the surgical area and periphery. Soft tissues should be protected with metal retractors when using rotary instruments. Stable fingers are also fundamental to good surgical technique (Clarke and Bueltman, 1971; Hunt, 1976).

The postoperative infection rate following periodontal surgery is about 1% (Pack and Haber, 1983). Infections should be treated aggressively pursuant to diagnosis. It is imperative that affected sites be adequately debrided and proper drainage established. A decision to prescribe antibiotics should be based on the systemic health of the patient and presence of objective clinical indicators. Infections in the area of the maxillary anterior teeth may involve the canine space and can spread to the orbit and/or the buccal space. Infections of the buccal space may spread to the masticator space with potential communication with the parotid and the lateral pharyngeal spaces. Infections in the area of mandibular anterior teeth can involve the mental space and may spread to the buccal space. Infections on the lingual aspect of the mandible may affect the submandibular space which is composed of the sublingual, submaxillary and submental spaces. Route of extension is via the submaxillary space through the lateral pharyngeal space into the retropharyngeal space. Infections in the retropharyngeal space may drain into the mediastinum if the alar fascia ruptures. Ludwig's angina is a cellulitis of the submandibular space. The patient may be febrile with a protruding tongue, "board-like" swelling of the floor of the mouth and dyspnea. This is a life-threatening situation due to the possibility of asphyxiation and requires immediate hospitalization and aggressive therapy (Hunt, 1976).

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Section 2. Electrosurgery

DEFINITION

Electrosurgery: Division of tissue by high-frequency electrical current applied locally with a metal instrument or needle.

GENERAL INFORMATION

Electrosurgery utilizes controlled high-frequency currents ranging from 1.5 to 7.5 million cycles per second. While it has been used for nearly a century, it became more popular in the late 1960s when improved technology afforded better control of the electrical current. The filtered, fully rectified, high-frequency current was developed by Dr. Irving Ellman in the early 1970s. In contrast to this electrosective current, partially rectified current (damped) provides good electrocoagulative properties. Oral electrosurgery utilizes a biterminal technique. The active electrode consists of a small wire which is used at the operative site while the passive electrode is a conductive plate placed at a distant site. The 3 classes of active electrodes include: 1) single wire electrodes for incising tissue; 2) loop electrodes for planing tissue; and 3) heavy, bulkier electrodes for coagulation procedures. The 4 electrosurgical techniques are electrosection, electrocoagulation, electrofulguration, and electrodesiccation.

Electrosection and electrocoagulation are biterminal techniques and are the electrosurgical procedures most commonly used in dentistry. Electrosection requires an undamped or continuous wave train. Three types of electrosection applications have been described and include incisions, excisions, and planing. Incisions and excisions are performed with a single-wire active electrode that can be bent or adapted to the type of cutting procedure. Tissue planing may be accomplished by selection of an appropriate loop electrode. Electrocoagulation employs a damped or interrupted wave train and may prevent or assist in local control of hemorrhage. There are three types of coagulation electrodes: ball, bar, and cone electrodes. Monoterminal techniques are seldomly used and include electrofulguration and electrodesiccation (Flocken, 1980).

MECHANISM OF ACTION

In electrosurgery, radio-frequency energy is concentrated, splitting tissue cells and creating a micro-thin layer of coagulated tissue. Lateral heat due to tissue impedance or resistance accounts for the thin coagulated layer. The thickness of the zone of coagulum and amount of color change are directly related to lateral heat production and may be controlled by the operator. In general, the smaller the color change and thinner the layer of coagulated tissue, the better the healing response. Lateral heat is a product of five factors: duration of contact; dose of current; electrode size and volume; current selection; and tissue impedance. An excess of any one of these factors should be offset by

adjusting and reducing the other factors. Tissue impedance is highest in enamel, followed by dentin and bone, cartilage, dense fibrous tissue, skin, muscle and connective tissue, mucous membrane, and diseased and inflamed tissues. Because of the low impedance of diseased tissue, control of bleeding is more difficult than in healthy tissue. Since the electrosurgery unit is similar to a radio transmitter, tuning of the unit is required for best results. Six factors influence the tuning process: 1) manufacturer variation in the unit; 2) patient variation of impedance; 3) body tissue variation of impedance; 4) grounding potential of operatory environments; 5) current output variation related to local environmental changes; and 6) active and passive electrode proximity. Correct tuning may be defined as adjustment of the above factors to cause the least tissue color change without drag which is the adherence of soft tissue to the electrode which impedes smooth incision. It occurs when insufficient current is being used. Sparking within the tissues during electrosurgical procedures should be avoided. Sparking is caused by: 1) use of current that is too high; 2) excessive tissue dryness at the operative site; 3) failure to use the passive electrode; 4) contact with metal; 5) operating in diseased tissue; or 6) a defective electrosurgical unit (Flocken, 1980).

ADVANTAGES OF ELECTROSURGERY

Flocken (1980) outlined several possible advantages of electrosurgery over traditional surgical techniques. This modality: 1) permits any degree of hemorrhage control desired; 2) prevents bacterial seeding into the incision site; 3) has active flexible electrodes, which can be shaped to conform to any requirement; they never need sharpening; they are self-sterilizing and require no digital pressure to function; 4) permits planing of soft tissue; 5) provides a better view of the operative site; 6) eliminates scar formation; 7) increases operative efficiency; 8) reduces chair time for each operation; 9) improves the quality of restorations; 10) reduces operator fatigue and frustration; and 11) minimizes postoperative discomfort and treatments. Only two drawbacks to the use of electrosurgery were mentioned: it is contraindicated in patients with cardiac pacemakers and produces an unpleasant odor and taste.

TISSUE RESPONSE TO ELECTROSURGERY CONTACT

An appropriate preface to this section is found in an article by Krejci et al. (1987)which reviews controlled clinical studies of oral tissue response to electrosurgery. The authors noted that most studies evaluating electrosurgery have been poorly documented or poorly controlled. With this in mind, the following synopsis of oral tissue response to electrosurgery should be considered.

Epithelium

The electrosurgery incision in epithelium results from volatilization of cells in the line of delivered high frequency energy. This may lead to loss of cellular detail secondary to the lateral heat produced, but subsequent wound healing stages do not appear adversely affected. Use of the instrument in the gingival crevice may result in varying degrees of gingival recession. Although this may not be clinically significant, misuse may result in increased recession (Krejci et al. 1987).

Connective Tissue

Controlled human studies evaluating the histologic changes in connective tissue accompanying electrosurgery reported a small denatured zone (averaging 100 microns) resulting from lateral heat adjacent to the path of incision (Kalkwarf et al., 1981, 1983). This zone does not appear to interfere with wound healing and gradually disappears within 14 days. Misuse may cause adverse alterations in the connective tissue and delay the healing response (Krejci et al., 1987).

Bone

Studies reviewed by Krejci et al. (1987) indicate that carefully controlled use of the electrosurgery unit within accepted clinical guidelines (i.e., time of exposure and energy production) may elicit minor, clinically insignificant changes at the alveolar crest. Misuse, however (e.g., longer exposure to the activated electrode or direct contact with denuded bone), may result in bone necrosis and delayed healing. These findings differ from those of Azzi et al. (1983) who compared the effects of electrosection and full thickness flap reflection on alveolar bone in mongrel dogs, reporting destruction extending to the middle one-third of the periodontal ligament in electrosurgical sites. The initial response was acute inflammation which was followed by osteoclastic and osteoblastic bone remodeling. The destructive effects were similar regardless of electrode application time, leading the authors to conclude that any contact of the electrode with bone should be avoided.

Cementum and Periodontal Attachment

Electrode contact with the root surface may create root resorption and cemental shrinkage, inhibiting connective tissue reattachment (Krejci et al., 1987).

Pulpal Tissue

Pulpal studies indicate that intermittent contact of a metallic restoration with an active electrosurgery electrode (less than 0.4 seconds) results in the delivery of well-controlled current which results in minor pulpal stimulation capable of spontaneous recovery. Exposures exceeding 0.4 seconds or with uncontrolled intensity are capable of eliciting pulpal necrosis. Electrosurgery use for pulpotomy procedures appears to be biologically acceptable (Krejci et al., 1987).

GUIDELINES FOR CLINICAL USE OF ELECTROSURGERY

Krejci et al. (1987) have provided the following clinical guidelines for use of electrosurgery: 1) use a higher fre-

quency unit tuned to optimal power output and a fully rectified, filtered waveform; 2) use the smallest possible electrode; 3) make incisions at a minimum rate of 7 mm per second; 4) allow an 8-second cooling period between successive incisions using a needle electrode at the same surgical site; this period should be increased to 15 seconds if a loop electrode is being used; 5) allow for gingival recession when an electrosurgical incision is used for troughing or excising the gingival crevice; 6) avoid contact of the active electrode with the cemental surface when connective tissue reattachment is desired; 7) anticipate slight osseous remodeling of a clinically insignificant nature with proper electrosurgery use; however, improper use may produce irreversible changes capable of resulting in diminished periodontal support; 8) limit contact with metallic restorations to less than 0.4 seconds; 9) electrosurgery may be used for pulpotomy procedures; 10) use the electrosurgery unit to provide electrofulguration and subsequent hemorrhage control only when all other clinical methods have failed and expect delayed healing response after fulguration; and 11) use electrosurgery to safely excise inflammatory papillary hyperplasia.

INDICATIONS FOR ELECTROSURGERY

Indications for the use of electrosurgery as described by Flocken (1980) may include crown lengthening, contouring edentulous ridges, removing hyperplastic tissue, desensitization of hypersensitive dentin, gingivectomy or gingivoplasty, frenectomy and operculectomy, biopsy, incision and drainage of abscesses, and periodontal surgery. Cautious application is recommended.

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Section 3. Gingivectomy/ Gingivoplasty

DEFINITIONS

Gingivectomy: The excision of a portion of the gingiva; usually performed to reduce the soft tissue wall of a periodontal pocket.

Gingivoplasty: A surgical reshaping of the gingiva.

OBJECTIVES AND INDICATIONS

Objectives include: 1) eradication of pockets; and 2) creation of a physiologic gingival sulcus and contours (Waite, 1975). Indications for gingivectomy include gingival overgrowth or enlargement, pseudo-pockets, idiopathic gingival abromatosis, and minor corrective procedures commensurate with patient needs (Rateitschak et al., 1985).

INDICATIONS FOR GINGIVOPLASTY

Gingivoplasty may be used to correct soft tissue deformities (e.g., post-orthodontic treatment, post-periodontal surgery, ANUG), and to enhance esthetics (e.g., altered passive eruption) (Pollack, 1964).

CONTRAINDICATIONS FOR GINGIVECTOMY

Contraindications for gingivectomy include: intrabony defects, thickening of marginal alveolar bone, and absence (or a narrow zone) of attached gingiva (Rateitschak et al., 1985).

TECHNIQUE

Waite (1975) reviewed the gingivectomy technique, noting that preoperative scaling facilitates resolution of inflammation and allows assessment of the patient's oral hygiene. The surgical procedure includes measurement and transgingival marking of the pseudo-pockets with a probe or marking forceps and excision at the pocket wall apically to assure elimination. The remaining soft tissues are contoured to restore physiologic gingival form. Clinically, removal of granulation tissue may necessitate curettage to the periosteum or alveolar bone.

Epithelization: Tritiated Thymidine

In a radioautographic study of healing in monkeys following gingivectomy, Engler et al. (1966) concluded that epithelial cells begin wound coverage 12 to 24 hours following surgery. demonstrating maximum cell division during the second day. While complete healing required 4 to 5 weeks, surface healing was obtained after 2 weeks. The authors noted that epithelium migrated at a rate of 0.5 mm per day and that increased thymidine uptake was limited to a zone of 2 mm from the wound margin.

In an electron microscopy study, Listgarten (1972) demonstrated complete re-establishment of the junctional epithelium as early as 12 days post-operatively, following gingivectomy in monkeys. In animal and human studies, Stahl et al. (1972) reported epithelialization at 7 to 14 days and connective tissue maturation 10 to 30 days following gingivectomy.

Connective Tissue

In a radioautographic study of connective tissue (CT) healing following gingivectomy in Rhesus monkeys, Ramjford et al. (1966) concluded that healing begins 0.3 to 0.5 mm beneath the protective "poly band" surface. Following surface epithelization, CT proliferation of all supracrestal

tissues occurred up to the basement membrane of the new epithelium. The authors noted that CT begins 1 to 2 days after the gingivectomy and peaks at 3 to 4 days. Formation of a physiologic gingival crevice, functional regeneration, and maturation of the gingival CT required 3 to 5 weeks.

Alveolar Bone Response

Slight loss of continuity of the osteoblast layer on the outer aspect of the alveolar crest occurs during the initial 12 hours. This was followed by new bone formation as early as the fourth day. New cementoid formation appeared at 10 to 15 days (Glickman, 1972).

CLINICAL STUDIES

Wennstrom (1983), in a human study, compared the regeneration potential of the zone of keratinized and attached gingiva following the surgical removal of the entire zone of existing gingiva via a gingivectomy versus a flap-excision (FLEX) procedure. Results revealed that a new zone of keratinized gingiva consistently regenerated, following surgical excision of the entire portion of the gingiva. This zone of keratinized gingiva was wider in the gingivectomy units than in the FLEX units. The granulation tissue which developed adjacent to the teeth following each procedure was seen as having the capacity to induce keratinization of the covering epithelium, particularly that which formed following the gingivectomy procedure.

Donnenfeld and Glickman (1966) examined the biometric effects of gingivectomy and reported that it eliminates pockets without significant clinical or statistical change in the location of the healed sulcus or width of the attached gingiva. The 0.3 mm reduction in width of attached gingiva was attributed to coronal migration of the mucogingival junction and slight apical shift shift of the healed sulcus base. Rosling et al. (1976) conducted a 2-year clinical study which compared the apically positioned flap (APF), APF and osseous surgery (OS), Widman flap (WF), WF and osseous surgery, and gingivectomy. Although all of these proved effective to varying degrees, the gingivectomy was accompanied by reduced pocket depths, greatest reduction of alveolar bone height, and the least regeneration of intrabony defects.

INSTRUMENTATION

Instruments that have proven useful in the gingivectomy procedure include surgical knives (e.g., Kirkland, Buck); gingivectomy clippers (including surgical scissors); coarse rotating abrasive stones; ultra-speed diamond stones; electrosurgery; and cryotherapy (Waite, 1975). Pick and Colvard (1993) described the use of the dental laser to perform gingivectomy.

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Section 4. Repositioned Flaps

DEFINITIONS

Flap: A loosened section of tissue separated from the surrounding tissues except at its base.

Modified Widman Flap: A scalloped, replaced, mucoperiosteal flap, accomplished with an internal bevel incision, that provides access for root planing.

Repositioned Flap: A flap that is moved laterally, coronally, or apically to a new position.

ANTERIOR CURTAIN PROCEDURE

Frisch et al. (1967) described a modified surgical approach for periodontal defects in this area. In the presence of healthy mid-labial sulci, a curtain of tissue which includes the labial one-third of the labial interproximal papilla is preserved facially. Lingual interproximal defects are managed by gingivectomy or a palatal flap, depending on the presence of osseous involvement. Advantages of this procedure include its simplicity, conservative nature, esthetic preservation, minimization and avoidance of speech defects, and minimization of labial alveolar bone loss. Disadvantages include less than ideal labial contour, greater oral hygiene demands, and application limited to the maxillary anterior sextant.

OPEN DEBRIDEMENT

Becker et al. (1986) studied the repair of narrow, medium, and wide 3-wall intrabony defects following open flap debridement procedures in humans. After calculus removal and root planing, hydrocolloid impressions were made of the surgical defect and greatest width recorded clinically. Surgical re-entry and clinical measurement were accomplished at 9 to 16 months. Mean defect fill was 2.56

mm (61%) on models and 3.26 mm based on direct measurements. The authors concluded that open debridement of intrabony defects has potential for repair with significant, but varying, amounts of bone.

MODIFIED WIDMAN FLAP

Ramfjord and Nissle (1974) described the modified Widman flap (MWF). The procedure emphasizes conservative surgical flap access using sharp incisions to avoid excessive tissue trauma and close interproximal flap adaptation of healthy collagenous tissues to root planed tooth surfaces.

Ramfjord (1977) reviewed the present status of the modified Widman procedure. Following a detailed description of the MWF procedure, the author noted that creation and maintenance of a biologically acceptable root surface is the key to success. The procedure is indicated for deep pockets, intrabony pockets, and when minimal recession is desired. The advantages of the procedure include ability to coapt the tissues to the root surfaces, access to the root surfaces, esthetic results, less likelihood of root sensitivity and caries, and a favorable environment for oral hygiene maintenance. Disadvantages include flat or concave interproximal soft tissue contours often present following dressing removal. Meticulous oral hygiene is emphasized for such areas. Smith et al. (1987) and Svoboda et al. (1984) evaluated the effect of retention of gingival sulcular epithelium following MWF and intrasulcular incision techniques. Comparable clinical results were observed in patients receiving both techniques, leading the authors of both studies to conclude that removal of sulcular epithelium during periodontal surgery provided no therapeutic advantage.

EXCISIONAL NEW ATTACHMENT PROCEDURE [ENAP]

Yukna and Lawrence (1980) described the ENAP as a means of treating suprabony pockets consisting essentially of subgingival curettage with a surgical knife. Internally bevelled incisions extend from the gingival margin to the base of the pocket, allowing debridement, root preparation, and primary closure. The modified ENAP includes an initial incision directed toward the alveolar crest (rather than the root surface), affording better access and maximizing healing capabilities of the periodontal ligament. A disadvantage is the potential for attachment loss that may accompany removal of intact supracrestal connective tissue fibers. Healing of the ENAP (and modified ENAP) consists of a long junctional epithelium to the depth of the surgical wound with occasional presence of connective tissue adhesion.

In a 5-year evaluation of the ENAP, Yukna and Williams (1980) reported a net gain in clinical attachment of 1.8 mm and an overall mean decrease of 1.8 mm in probing depth (3.0 mm). Probing depths increased slightly and new attachment gain decreased slightly at the 1, 3, and 5-year post-operative evaluation periods. This report supports the clinical success of the ENAP at 5 years.

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Section 5. Mucogingival Surgery

DEFINITION

Mucogingival Surgery: Periodontal surgical procedures used to correct defects in the morphology, position, and/or amount of gingiva.

INTRODUCTION

While over the past decade there has been less emphasis on mucogingival procedures to increase the amount of attached gingiva, they continue to play an important role in the comprehensive management of the periodontal patient. This discussion will examine the indications and rationale for mucogingival surgical procedures, including free autogenous soft tissue grafts and pedicle grafts.

INDICATIONS FOR MUCOGINGIVAL PROCEDURES

Historically, mucogingival surgery was used to increase the amount of attached gingiva. A certain amount of attached gingiva was considered necessary to maintain gingival health and prevent gingival recession. Factors influencing this relationship included prominence of the tooth in the arch, amount of attached gingiva present, and ability of the patient to adequately control the accumulation of bacterial plaque. Although establishing an adequate width of keratinized tissue has been emphasized, the thickness of this tissue is at least equally important in preventing soft tissue recession in the presence of bacterial plaque. Other indications for mucogingival procedures include elimination of frenum and muscle attachments, increasing vestibular depth, coverage of gingival clefts, modification of edentulous ridges prior to prosthetics, establishing a zone of attached gingiva prior to coronally positioning a graft, and for restorative considerations, particularly if subgingival

margins are contemplated in areas of thin tissue (Nery and Davies, 1977).

GENERAL PRINCIPLES

Morman and Ciancio (1977) used fluorescein angiography in human biopsy specimens to examine alterations in gingival circulation following various modifications of mucogingival flap design. Blood supply to a flap was primarily directed caudo-cranially from the vestibule to the gingival margin. Internal beveled incisions severing the anastomosis between the gingival and periodontal/interdental vasculature had no effect on circulation, verifying circulatory independence. The following concepts were suggested when designing periodontal flaps: 1) flaps should have a broad base which includes major gingival vessels; 2) a flap's length to width ratio should not exceed 2:1; 3) minimal tension should be produced by suturing techniques and the tissue should be managed gently during the surgical procedure; 4) partial thickness flaps covering avascular areas should not be too thin, so that more blood vessels are included in them; and 5) the apical portion of periodontal flaps should be full thickness when possible.

Cattermole and Wade (1978) compared linear and scalloped incisions in the reverse bevel elevation of full thickness buccal and palatal flap reflections in humans. Although both flaps healed after 4 weeks, the linear incision showed interdental tissue that was not fully mature and more inflamed. At 12 weeks, it was difficult to distinguish which type of incision had been used and no significant differences in healing, pain, GI, PI, and GCF were observed.

Lindhe and Nyman (1980) examined alterations in gingival margin position on the buccal surfaces of human teeth professionally maintained for 10 to 11 years following periodontal surgery. Regardless of the presence or absence of keratinized soft tissue, changes observed in gingival margin position were similar. Gingival recession was not produced by daily tooth brushing combined with professional maintenance care. Conversely, approximately 1 mm of coronal regrowth of the gingival margin occurred. Their results supported those of Dorfman et al. (1980), showing that sites with or without adequate attached gingiva maintain attachment levels over a long period of time. Hangorsky and Bissada (1980) also demonstrated that the absence of keratinized gingiva does not jeopardize gingival health. Hall (1981) reviewed the mucogingival therapy literature and concluded that a minimum width of attached soft tissue necessary for health had not been established. It is difficult to predict if recession will occur in areas of narrow or absent attached gingiva; however, recent successes in covering exposed root surfaces through various grafting procedures have made prophylactic grafting less of a concern. Creeping attachment was found to enhance root coverage in many cases of soft tissue grafting by a mean of 0.89 mm over the first postoperative year. Noting the high degree of success when placing grafts on either denuded bone or periosteum, the author concluded that perforation of the periosteum during receptor bed preparation is probably of little concern.

Allen (1988) discussed the use of mucogingival procedures with particular emphasis on maxillary esthetics. Indications for the possible need for mucogingival corrective procedures include: 1) inadequate keratinized gingiva; 2) gingival recession; 3) excessive gingival display; 4) insufficient clinical crown length; 5) asymmetric gingival margins; 6) flat marginal contour; 7) improper gingival margin relationships; 8) lack of harmony with the lip line and gingival margins; and 9) alveolar ridge deficiencies. The ideal relationships of the gingival margins of the maxillary anterior teeth were described. The gingival margins of the central incisors are symmetric and are either even with or 1 mm apical to the margins of the lateral incisors. The gingival margins of the canines are 1 mm apical to the level of the lateral incisors. A line drawn horizontally at the level of the canine gingival margins should parallel the interpupillary line. The incisor gingival margins should peak slightly to the distal giving the appearance of distal inclination. The smile should expose minimal gingiva apical to the centrals and canines and should be in harmony with the smile line. The crowns of central incisors and canines can usually be exposed to an overall length of 11 to 12 mm to attain maximal gingival reduction. The lateral incisors should be exposed 1.5 mm less than the length of the centrals.

Partial Thickness Flaps

Wood et al. (1972) used human re-entry to compare crestal radicular bone responses to full and partial thickness flaps (PTF). Regardless of the flap procedure, loss of crestal bone depended to a great extent on the thickness of preexisting bone. Teeth with the thinnest radicular bone demonstrated greater bone loss postoperatively. Mean bone loss for full and partial thickness flaps was 0.62 mm and 0.98 mm, respectively. Use of the PTF in areas of thin gingiva resulted in a very thin non-protective layer of connective tissue which provided significant osteoclastic activity. It was also speculated that the compromised vasculature of a thin PTF could produce necrosis of the flap margin, resulting in exposure of the poorly protected underlying bone and increased susceptibility to resorption. The authors concluded that partial thickness flaps are not indicated in areas of thin connective tissue.

Staffileno et al. (1966) studied histologic repair of the periodontium in dogs following resection of a split thickness flap. Results demonstrated that split thickness flaps with periosteal retention produced minimal tissue destruction, rapid repair, slight alteration of the dentogingival junction, and maximum preservation of periodontal supporting structures.

Karring et al. (1975) histologically examined the development of granulation tissue after periosteal retention and

denudation procedures in monkeys. Following both procedures, granulation tissue originated from the residual periosteal connective tissue, PDL, bone marrow spaces, and the adjacent gingiva and alveolar mucosa. Bone resorption was generally more severe with the denudation procedure; however, greater amounts of loss were occasionally seen following periosteal retention. The point of transition between keratinized and non-keratinized epithelium was found to correspond to the junction between connective tissue with and without regenerated elastic fibers, demonstrating the inductive influence of connective tissue on the overlying epithelium.

Wilderman et al. (1960) studied histologic wound healing of exposed alveolar bone in dogs. Differences in the anatomy of interdental and radicular bone appeared responsible for varying degrees of osteoclastic resorption. Where adequate marrow spaces remained (interdentally), there was complete restoration of bone. In contrast, radicular areas showed 50% bony restoration, demonstrating functional repair with double the fibrous attachment of new gingiva as compared to the original condition and an epithelial attachment located more apically compared to interdental sites.

Hiatt et al. (1968) examined healing and reattachment of mucoperiosteal flaps in dogs. At 2 to 3 days, flap adhesion was mediated by fibrin which prevented downgrowth of epithelium if the flap was well-adapted. Accelerated repair observed in tightly-adapted flaps was attributed to the decreased time required for fibrin resorption and replacement by connective tissue. Retained vital cementum appeared to accelerate connective tissue attachment. Dentin surfaces which had been denuded of cementum by root planing underwent resorption prior to new cementum formation.

Frank et al. (1972) demonstrated differentiation of new attachment apparatus at the ultrastructural level in humans. These results supported the electron microscopic observations of Listgarten (1967, 1972), showing re-establishment of new epithelial attachment in monkeys after gingivectomy and mucoperiosteal surgery. Thilander and Hugoson (1970) also demonstrated re-establishment of an attachment apparatus in cats after deep scaling.

Pedicle Grafts

Pedicle grafts differ from free autogenous soft tissue grafts in that the base of the flap contains its own blood supply which nourishes the graft and facilitates the re-establishment of vascular union with the recipient site. Pedicle grafts may be split or full-thickness. Some early studies (Pfeifer and Heller, 1971; Sugarman, 1969) reported that the use of full-thickness lateral sliding grafts resulted in a connective tissue attachment to the root surface (one half CT, one half JE) while a partial thickness flap yielded a long junctional epithelial attachment. While it has been felt that a split-thickness flap with preservation of a periosteum over the donor site would protect underlying bone from resorption, Wood et al. (1972) observed increased crestal bone resorption with split-thickness flaps as compared to full-thickness counterparts (0.98 mm versus 0.62 mm).

Grupe and Warren (1956) were the first to report on the use of a lateral sliding flap to repair isolated gingival defects. The procedure consisted of removing the epithelial lining surrounding the defect and freshening the wound margins. A full-thickness flap was elevated one tooth away from the defect and rotated to cover the defect. Corn (1964) reported the use of pedicle grafts to correct mucogingival defects, utilizing an edentulous area as the donor site.

In 1967, Hattler described a procedure to correct conditions where the attached gingiva on the facial surfaces of 2 or 3 consecutive teeth was deemed inadequate. This technique involves the development of partial thickness flaps around the involved teeth and sliding the entire flap 1/2 tooth width, placing the interdental papillary tissues over the buccal surfaces of the affected teeth.

Cohen and Ross (1968) described the double-papilla repositioned flap to cover defects where a sufficient amount of gingiva was not present or where there was insufficient gingiva on an adjacent area for a lateral sliding flap. The papillae from each side of the tooth were reflected and rotated over the midfacial aspect of the recipient tooth and sutured. This technique offers the advantages of dual blood supply and denudation of interdental bone only, which is less susceptible to permanent damage after surgical exposure.

Coronally-Positioned, Free, Autogenous, Soft Tissue Grafts

Bernimoulin et al. (1975) first reported the coronally-positioned (previously-placed) free, autogenous, soft tissue graft as a two-stage procedure. First, a free autogenous soft tissue graft is placed apical to an area of denuded root surface. After an adequate healing period, the graft is coronally positioned over the denuded root surface. In 1977, Maynard presented 6 requirements for success of coronally positioned grafts: 1) presence of shallow crevicular depths on proximal surfaces; 2) approximately normal interproximal bone heights; 3) tissue height should be within 1 mm of the CEJ on adjacent teeth; 4) adequate healing of the free graft prior to coronal positioning (6 weeks); 5) reduction of any root prominence within the plane of the adjacent alveolar bone; and 6) adequate release of the flap at the second-stage procedure to prevent retraction during healing. The second-stage procedure utilizes a split-thickness dissection with mesial and distal vertical releasing incisions until adequate flap mobility is obtained. The flap is sutured 0.5 to 1 mm coronal to the CEJ and covered with a periodontal dressing. This procedure is indicated when root sensitivity or cosmetic concerns relative to recession become therapeutic considerations.

Guinard et al. (1978) and Caffesse and Guinard (1980) compared lateral sliding flaps and coronally positioned flaps in the treatment of localized gingival recessions. In the 6-

month report (Guinard et al, 1978), they found that both techniques rendered satisfactory results and no differences were reported regarding gain of tissue coverage, sulcus depth or gain of attached gingiva. An average of 2.71 mm of soft tissue coverage was obtained, with an average coverage of 67% of the recession. The only difference between the 2 techniques was an increase in root exposure of approximately 1 mm at the lateral sliding flap donor site while no additional recession was observed with the coronally positioned flap. Results were stable 1-month post-therapy and remained so after 3 years. The 1 mm of gingival recession created on the donor tooth when a lateral sliding flap was used did not repair over the 3 years of observation (Caffesse and Guinard, 1980).

Allen and Miller (1989) reported the use of a single-stage coronally positioned flap in the treatment of shallow marginal recession. The defects were Miller Class I and had a minimum keratinized tissue width of 3 mm. Recession ranged between 2.5 to 4 mm. The technique consisted of citric acid root treatment, a split-thickness flap extending into the vestibule, and surface gingivoplasty of the papillae to produce a bleeding bed. Flaps were sutured into position and dressed. Complete root coverage was attained in 84% of the sites, with a mean gain of 3.18 mm root coverage.

Tarnow (1986) described the semilunar coronally positioned flap. A semilunar incision is made that follows the curvature of the free marginal gingiva and extends into the papillae, staying at least 2 mm from the papilla tip on either side. The incision is made far enough apically to ensure that the apical portion of the flap rests on bone after repositioning. A split-thickness dissection of the flap is made and the flap is repositioned and held in place with light pressure and dressed. Advantages of the technique according to the author include: 1) no tension on the flap after repositioning; 2) no shortening of the vestibule; 3) no reflection of the papillae, thereby avoiding esthetic compromise; and 4) no suturing.

Free, Autogenous, Soft Tissue Grafts (FASTG)

Sullivan and Atkins (1968) explored the feasibility and healing patterns of the FASTG and correlated plastic surgical principles to the practice of periodontics. This procedure involves the preparation of a recipient site which is accomplished by supraperiosteal dissection to remove epithelium, connective tissue, and muscle down to the periosteum. Placement of a FASTG directly on denuded bone was reported by Dordick et al. (1976), James and McFall (1978), and Caffesse et al. (1979) who demonstrated comparable success rates compared to grafts placed on the periosteum. James and McFall (1978) reported less shrinkage of FASTG placed on bone (25% versus 50% on periosteum). Dordick et al. (1976) reported a firmer, less mobile grafting results when placed on denuded bone. Caffesse et al. (1979) reported delayed healing during the first 28 days postoperatively when FASTG were placed on denuded bone.

James et al. (1978) performed a histologic comparison of wound healing between FASTG placed directly on denuded bone and periosteum. More marrow space-to-soft tissue communication occurred at "graft to bone" sites. Epithelial thickness was greater over the free grafts placed on bone until the twelfth week, at which time thickness was comparable. Free grafts on bone showed less postoperative swelling, but there was no difference in the degree of inflammation. Resorption of bone occurred at graft-to-bone sites, which allowed an adequate blood supply. However, placement of grafts on thin denuded bone may be contraindicated.

Wilderman and Wentz (1965) reported wound healing events of pedicle flaps in dogs. Four stages of healing were found to occur: 1) adaptation stage (0 to 4 days) when a fibrin clot containing PMNs was present between the flap and the crestal bone; 2) proliferation stage (4 to 21 days) when granulation tissue invaded the fibrin clot, fibroblasts were present on the root surface (6 to 10 days), epithelium migrated apically (10 to 14 days), osteoclastic activity occurred (4 to 14 days) and an average of 1 mm of crestal bone was resorbed; 3) attachment stage (21 to 28 days) when collagen formation was visible, cementum formation occurred and osteoblastic activity reached its peak; and 4) maturation stage (28 to 180 days) showed new PDL fibers orienting perpendicularly to the root surface. Repair consisted of a combination of connective tissue attachment (2.1 mm) and long junctional epithelium (2.0 mm).

Sugarman (1969) confirmed attachment of free soft tissue grafts and pedicle flaps by a combination of connective tissue and long junctional epithelium in humans.

Appropriate graft donor material should consist of keratinized tissue with a dense lamina propria. Studies by Karring et al. (1975) found that the phenotypic expression of epithelial surface was determined by the underlying connective tissue. Common areas for donor material include edentulous ridges, attached gingiva, and palatal mucosa. Donor tissue should be approximately 33% larger than the anticipated healed graft due to shrinkage during healing (Egli et al., 1975). According to Sullivan and Atkins (1968), a thick graft will have greater primary contraction (immediately after removal) due to the increased amount of elastic fibers but less secondary contraction during healing (due to cicatrization) and will have greater resistance to functional stresses. A thin graft will have less primary contraction and more secondary contraction. Split-thickness grafts are further categorized as thin, intermediate, and thick based on the thickness of their lamina propria.

Sullivan and Atkins (1968) recommended using of intermediate split-thickness grafts and full thickness grafts. Soehren et al. (1973) reported that the thickness of the palatal epithelium ranged from 0.1 to 0.6 mm with a mean thickness of 0.34 mm. These authors recommend the use of grafts no less than 0.75 to 1.25 mm in thickness to assure

that there is an adequate connective tissue component. The graft should be sutured to the periosteal bed for optimum immobilization between the graft and the recipient bed. A periodontal dressing may assist in maintaining positive pressure and aid graft immobilization.

Sullivan and Atkins (1968) reported the use of the FASTG to cover root recession. Root recession was classified into one of four types: 1) deep-wide: extends into alveolar mucosa; most difficult to treat; can expect 1 to 2 mm of new tissue over the apical portion; 2) shallow-wide: also expect 1 to 2 mm of new tissue over the apical portion; may get coverage of a large part of the defect; 3) deep-narrow: extends into alveolar mucosa; rarely seen; may be completely covered; and 4) shallow-narrow: maintained by conservative therapy, graft gives predictable results.

Creeping attachment following grafting has been reported by Matter (1980). This is a phenomenon of additional root coverage during healing which may be observed between 1 month and 1 year post-grafting. The author also reported an average of 1.2 mm of coronal creep at 1 year with no additional change.

In a 2-year study comparing graft versus no graft, Dorfman et al. (1980) concluded that plaque control was more important than the width of the attached gingiva in determining eventual breakdown and recession. They also found that the use of the FASTG was a predictable means of increasing the width of the attached gingiva. In a follow-up study 2 years later, these authors reported basically the same results except that 10% of the non-grafted cases showed additional soft tissue recession with equivalent plaque scores compared to grafted sites (Dorfman et al., 1982).

Edel (1974) reported the use of free connective tissue grafts as an alternative to epithelialized donor tissue. In his report on 14 successful grafts, Edel found that the resultant increase in attached gingiva was stable at 6 months with a mean contraction of 28%. Complete epithelialization of the connective tissue surface was seen at 2 weeks with keratinization evident at 4 weeks. The graft was blended into the surrounding tissues at 6 weeks with formation of well-developed rete ridges. The results of this study confirmed that the connective tissue determines the character of the overlying epithelium.

Holbrook and Ochsenbein (1983) used FASTG in a single procedure to cover denuded root surfaces and were the first to suggest butt joint margins at the junction of the recipient bed and donor tissue. The recipient bed is extended one-tooth width lateral to the denuded roots and 5 mm apical to the gingival margin of the denuded root. Root prominence is reduced by root planing. Donor tissue should cover the gingival bed extending at least 3 mm apical to the margin of the denuded root(s). A graft of approximately 1.5 mm uniform thickness is utilized with butt margins. A precise suturing technique (''Holbrook'') is described utilizing a horizontal continuous suture to stretch the graft 2 to 3 mm. The authors feel this counteracts primary contraction, making the graft more receptive to revascularization. Circumferential sutures compress the graft at the borders of the denuded root and are inserted into the periosteal bed slightly apical to the inferior margin of the graft. Two separate interdental concavity sutures adapt the graft mesially and distally. These sutures are inserted into the periosteum at the depth of the interdental concavity diagonally traversing the graft mesially and distally. In 50 randomly-selected cases, recessions < 3 mm had 95.5% root coverage, recessions 3 to 5 mm had 80.6% coverage and recessions ≥ 5 mm had coverage of 76.6%. The most difficult tooth root to cover was the maxillary canine. Visible recession is the clinically observable root measured from the CEJ to the gingival margin. Hidden recession is defined by the authors as the ''depth of the sulcus or pocket as measured from the soft tissue margin to the junctional epithelium.''

Miller (1985) described a technique for root coverage using a free soft tissue autograft and citric acid treatment. Predictable root coverage depended upon the type of gingival recession and Miller presented an expanded classification of marginal recession. Class I defects present as marginal recession coronal to the mucogingival junction with no periodontal loss in the interdental areas. Class II defects also show no interdental periodontal loss but have recession extending beyond the mucogingival junction. This includes both visible and hidden recession. According to Miller, 100% root coverage can be predictably achieved in both Class I and II defects. Class III defects have recession extending to or beyond the mucogingival junction, but with some soft tissue or bone loss in the interdental areas (only partial root coverage can be expected). Class IV defects are similar to Class III defects except there is severe bone or soft tissue loss interdentally (root coverage cannot be anticipated). Miller's technique includes root planing to reduce root convexity and minimize the mesiodistal dimension of the root before the recipient bed is prepared. Root planing is pronounced along the CEJ to create a butt-type joint for the graft. After root planing, saturated citric acid is vigorously burnished into the root surface for 5 minutes (pellets changed 2 to 3 times/minute). Horizontal incisions are made at the level of the CEJ preserving the interdental papillae (height permitting); vertical incisions at proximal line angles of adjacent teeth facilitate completion of bed preparation. All incisions should result in ''butt joints'' between the recipient and donor tissues. Periosteal fenestration (which may compromise blood supply of bed) is not used. A thick palatal graft with a thin layer of submucosa is placed on a moderately bleeding bed and stabilized with sutures at the papillae and apical corners of the graft extending into periosteum. Results of 100 consecutive grafts showed 100% root coverage in 90% of Class I and

II defects (Class I = 100%; Class II = 88%). In Class III sites, a high percentage of the attainable coverage was obtained. The average root coverage for all sites was 3.79 mm with a mean gain of 4.54 mm in clinical attachment.

Miller (1987) discussed factors associated with incomplete root coverage. This article stressed adherence to several features of the technique: 1) flattening of the root in the CEJ region creating a butt joint; 2) use of citric acid burnished into the root surface for 5 minutes prior to preparation of the recipient site; 3) use of horizontal incisions at the level of the CEJ to preserve interdental papillae for enhanced circulation; 4) utilizing thick graft tissue with right-angle margins, retaining a thin layer of submucosa that is placed as soon as possible on a moderately-bleeding bed; 5) stabilizing the graft with sutures to allow intimate adaptation to the periosteal bed; 6) avoiding pressure over the graft in an attempt to minimize hematoma formation, as pressure may compromise necessary blood flow to the graft; 7) avoiding trauma to the graft during initial healing; and 8) avoiding excessive smoking during the postoperative period, since patients smoking > 10 cigarettes/day are associated with a greater failure rate for 100% root coverage. Miller defines complete root coverage as the soft tissue margin at the CEJ with clinical attachment to the root, sulcus depth < 2 mm and no bleeding on probing. Miller reports a 90% success rate in achieving 100% root coverage compared to Holbrook and Ochsenbein (1983) who report a 44% success rate using a 1-stage approach (Miller's use of figures). Other authors utilizing a 2-stage coronally positioned graft report success rates of 44% (Bernimoulin, 1975) and 36% (Guinard and Caffesse, 1978). Miller emphasizes that no single factor can be credited as the most important factor in successful grafting for root coverage but that inattention to any single factor may result in incomplete coverage.

Raetzke (1985) described a technique for obtaining root coverage using free connective tissue grafts. In this technique, the collar of marginal tissue around a localized area of recession is excised, the root is debrided and planed, and a split thickness envelope created around the denuded root surface. In the premolar/molar region of the palate, two horizontal incisions are made 1 to 2 mm apart to the depth of the palatal mucosa and a wedge of connective tissue is removed with its small band of epithelium. The connective tissue graft is placed in the previously created envelope covering the exposed root surface. The palatal site is closed with sutures. Overall, 80% of the exposed root surfaces were covered, with 5 of 12 cases reporting complete root coverage. Advantages include minimal trauma to both donor and recipient sites with rapid healing, favorable healing over deep and wide areas of recession, and excellent esthetic results. Potential difficulties include difficulty in obtaining sufficient graft material in "thin" palates where necrosis of palatal tissue at the donor site is also a hazard. Palpation for palatal exostoses is recommended prior to selecting donor sites.

Reconstruction of Deformed Edentulous Ridges

Seibert (1983) described the principles and surgical procedures involved in reconstructing deformed partially edentulous ridges utilizing full thickness onlay grafts. Ridge defects were classified according to tissue loss. Class I defects are bucco-lingual tissue deformities with a normal apico-coronal ridge height. Class II defects present with loss of tissue in an apico-coronal dimension with normal buccolingual dimension. Class III defects are combination defects with loss in both bucco-lingual and apico-coronal dimensions. In this technique, recipient sites are minimally prepared by removing surface epithelium and CT to a depth of 1 mm. Margins may be butt-joint or beveled depending upon tissue contours of recipient sites. Full thickness grafts containing fatty and/or glandular submucosa are obtained from palatal tissue medial to premolar/molar areas matching the 3-dimensional shape of the defect in the ridge. After the graft is harvested, a series of deep parallel "stab" incisions are made in the exposed CT of the recipient site. These striations are believed to stimulate capillary growth into the graft from larger vessels leading to a "hematoma-like" reaction (and swelling of the graft) and resultant increase in total volume. The onlay graft is trimmed, if necessary, to obtain a snug fit against the exposed CT and sutured in place. Palatal hemostasis is obtained with a hemostatic dressing and a palatal stent. A provisional prosthesis may be placed with light contact against the graft surface and adjusted as necessary at future visits for patient comfort. If sufficient ridge reconstruction is not obtained after the initial attempt, a secondary procedure may be accomplished 6 weeks after the initial surgery. Very little primary or secondary shrinkage has been observed with full thickness onlay grafts and they appear to be dimensionally stable after 3 months of healing.

Frenectomy

Insertion points of the frena may become troublesome when the gingival margin is involved (Corn, 1964). This may result from an unusually high insertion of the frenum or because of recession. Frenal insertions can distend and retract the marginal gingiva or papilla when the lip is stretched. The author emphasized that the importance of the frenum attachment in the etiology of recession must be addressed directly.

West (1968) observed that frenectomy may result in scar formation which could prevent the mesial movement of the central incisors. Edwards (1977) indicated that orthodontic closure of diastemata without excision of the associated frena has been clinically associated with relapse separation of the teeth. It is noted that a frenectomy may be needed after orthodontic therapy.

The histologic morphology of frena has been another

area of controversy in the literature. Henry et al. (1976) studied 11 fresh biopsy specimens and concluded that excision of the superior labial frenum could not be based on removing muscle tissue (allegedly responsible for the "muscle pull" or tension) because no trace of muscle was found in the biopsies. The "destructive capacity" of frena was attributed to the elastic and connective tissue components rather than muscular elements.

In contrast, Ross et al. (1990) retrospectively examined biopsies of 40 frenal specimens from various intraoral sites (including 21 maxillary labial) and found that approximately 37.5% contained skeletal or striated muscle.

Regardless of the presence of muscle tissue in the frenum, it can be concluded that a frenectomy may be indicated if it is associated with a receded gingival margin, inability to adequately cleanse or debride the area or if any other indication exists for a mucogingival procedure. Frenectomy may be accomplished in conjunction with flap reflection and placement of a free gingival graft.

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Section 6. Osseous Resective Surgery

DEFINITIONS

Osseous Surgery: Periodontal surgery involving modification of the bony support of the teeth.

Osteoplasty: Reshaping of the alveolar process to achieve a more physiologic form without removal of supporting bone.

Ostectomy: The excision of a bone or portion of bone. In periodontics, ostectomy is done to correct or reduce deformities caused by periodontitis in the marginal and interalveolar bone and includes the removal of supporting bone (other terms for supporting bone are: alveolar bone proper, cribriform plate, and bundle bone).

Historical Perspective

Prior to 1935, bone associated with periodontal disease was considered infected or necrotic, and the most accepted treatment was surgical removal. Kronfeld (1935) demonstrated that bone was neither necrotic nor infected, and advocated the control of inflammation as the proper way to treat periodontitis. As a result of Kronfeld's work, and the introduction of the gingivectomy by Orban (1939), interest in osseous surgery declined; however, the recurrence of pockets following gingivectomy soon became a concern. Interest in osseous surgery was renewed by Schluger in 1949, when he advocated osseous resection and recontouring as the best way to maintain minimal pocket depth.

Theoretical and Diagnostic Approaches to Osseous Resective Surgery

Basis and Indications for Osseous Resective Surgery. According to Schluger (1949), frequent regrowth of soft tissue following gingivectomy is due to its inability to follow sharp and irregular contours of bony deformities. The author notes that since bone remodels more slowly than soft tissue, it should be contoured to facilitate gingival conformity. This may be achieved by making a long sweeping incision apical to the deepest pockets in the area, which usually results in exposed bone and post-operative pain; or, by gingivectomy, which precedes reflection of remaining soft tissue and osseous resection.

Indications for osseous resection include: 1) failure of the gingivectomy technique; 2) mesial of tipped molars; 3) deep isolated pockets; 4) some deep buccal and lingual pockets; and 5) saucer-shaped interproximal pockets. Soft tissue can tolerate variations in alveolar bone height up to 30° (60° in cases of prominent roots). Schluger (1949) recommends thinning thick bony ledges around teeth to a knife-edge margin. If pocket elimination will result in furcation exposure or sacrifice of too much bone on adjacent teeth, osseous resection may be contraindicated.

Osteoplasty/Ostectomy. Friedman (1955) further elaborated on the concept of osseous surgery and re-emphasized that irregular bony contours are not followed by the gingiva since the soft tissues tended to restore a regular scalloped contour. He classified osseous surgery into osteoplasty and ostectomy, advocating reshaping of underlying alveolar bone and pocket elimination in order to achieve proper gingival architecture. Indications for osteoplasty included deep interproximal pockets in posterior teeth involving buccal interdental bone; pockets in areas with thick bony ledges; and tilted lower second molars. Ostectomy was indicated for correction of shallow interproximal bony defects.

Advantages/Disadvantages of Osseous Resective Surgery. Siebert (1976) listed the advantages of osseous surgery as 1) visualization of osseous defects (dependability); 2) minimal treatment time; 3) simplicity; and 4) elimination of additional surgical sites. The main disadvantage is loss of attachment. Factors that should be considered prior to surgery include: 1) length and shape of roots; 2) location and dimensions of the defect; 3) width of investing bone; 4) root prominence; and 5) relationship of the intrabony defects to adjacent teeth and other anatomic structures. Gingivoplasty following osseous surgery should be done at 14 days as needed.

Palatal Exostoses. Nery et al. (1977) noted that palatal gingivectomies in molar areas often heal slower than other sites due to the presence of palatal exostoses. Of 681 skulls examined, 40% had palatal exostoses. The 40 to 55 year age group had the highest prevalence (50%), followed by the 56 year or older group (30%), and the 17 to 39 year group (19%). Nery et al. classified palatal exostoses into 5 groups: small nodule (most common), large nodule, sharp ridge, spike projection, and combination. The presence of palatal exostoses must be considered when surgically managing in the palatal molar area.

Bone Sounding. Easley (1967) described "bone sounding" as a technique for determining bony contours, bony

ledges, exostoses, and interdental defect morphology. In addition to vertical probing, horizontal probing is used to determine facial and lingual alveolar crestal bone heights.

Techniques Involved in Osseous Resective Surgery

Palatal Approach. Ochsenbein and Bohannan (1963) described the palatal approach to osseous crater removal. The advantages of the palatal approach include: 1) existence of keratinized tissue on the palate; 2) greater surgical access to larger palatal embrasures; 3) cleansing effect of the tongue on the palatal; and 4) less post-surgical bone resorption on the palatal due to the presence of increased cancellous bone. Thin facial bone and inadequate embrasure space make crater removal from the facial approach more difficult. Ochsenbein and Bohannan (1964) classified craters as Class I, 2 to 3 mm deep with thick facial and lingual walls; Class II (most common), 4 to 5 mm deep with thinner facial and lingual walls; Class III, 6 to 7 mm deep with a sharp drop from the walls to a flat base; and Class IV (least common), a crater of varying depth but with very thin facial and lingual walls. Class I craters can be managed by palatal ramping, while Class II and III craters require both facial and palatal ramping. Class IV craters usually require removal of both the facial and lingual walls. The treatment of the maxillary first molar is complicated by a facial root trunk length that averages only 4 mm and prominent facial roots due to arch position, which result in less radicular bone covering the facial roots. The authors emphasize that exposure of furcations during osseous surgery must be avoided.

Lingual Approach. Tibbetts et al. (1976) described the lingual approach to osseous surgery for the mandibular posterior area. Factors (advantages) supporting a lingual approach to osseous surgery in the mandibular posterior area include 1) avoidance of thick shelves of bone (e.g., external oblique ridge) which limit elimination of osseous defects facially; 2) presence of shorter root trunk lengths on the facial of mandibular molars; 3) lingual inclinations of the mandibular posterior teeth (9° for second premolar; 20° for molars), which places the lingual furcation more apical; 4) more lingual location of interdental craters (directly below the contact area); and 5) the creation of better access for surgery and hygiene, since the lingual embrasure is usually wider than the facial. Additional advantages to the lingual approach include: thicker bone; more attached gingiva; and greater vestibular depth on the lingual. Precautions include avoidance of the lingual nerve and artery.

Effects of Osseous Resective Surgery. Selipsky (1976) reported that properly performed osseous resective surgery results in an average loss of 0.6 mm of supporting bone height around the circumference of the tooth (maximum circumferential loss 1.5 mm). The maximum supporting bone lost around a single root surface was 3.0 mm. More bone was removed when treating isolated defects. Removal of buccal or lingual bone has a smaller impact on tooth support than removal of interproximal bone, due to the smaller surface area of buccal and lingual roots. Teeth treated with osseous resection had increased mobility that returned to the presurgical levels after 1 year. The technique sequence described for osseous resection includes: vertical grooving between teeth and roots; buccal and lingual osteoplasty; removal of lips of remaining craters; and buccal and lingual ostectomy. The author felt that vertical grooving prevented overzealous ostectomy by making the roots more prominent, allowing the gingiva to assume a more scalloped architecture.

Intrabony Defect Depth Versus Root Trunk Length. Ochsenbein (1986) noted that the extent of osseous resective surgery depends on the relationship between depth of the interproximal defects and molar root trunk length. Maxillary molars have short root trunks (3 to 5 mm, average 4 mm) based on measurements from the CEJ to the furcation entrance. Respective measurements for mandibular molars are 2 to 4 mm (average 3 mm). Shallow craters (1 to 2 mm) can usually be eliminated by a palatal approach even in the presence of short root trunk length. If complete elimination of medium (3 to 4 mm) or deep (> 5 mm) defects would result in furcation exposure or reverse architecture, only partial crater elimination should be attempted. Craters in the maxillary premolar regions are usually palatally located and can be treated from a palatal approach. The author suggested that osseous craters in the maxillary anteriors should be treated without osseous resection for esthetic reasons and that mandibular molars should be managed with the lingual approach, while avoiding unnecessary removal of bone adjacent to teeth. In the case of deep craters, compromised treatment may be necessary, including acceptance of reverse architecture.

Healing after Osseous Resective Surgery

Resorption of Exposed Bone. Pfeifer (1963) histologically evaluated the healing response of normal human bone following apical positioning of flaps 2 mm below crestal bone. Leaving bone exposed resulted in minimal bone resorption. Bone and soft tissue repair was rapid with connective tissue proliferating from the PDL at days 2 through 10, resulting in a 2 mm or greater band of connective tissue attachment to the root. Osteoclastic activity peaked at 4 to 10 days.

Osteoplasty Versus Flap Curettage. Donnenfeld et al. (1970) compared osteoplasty to flap curettage by examining pocket elimination, clinical attachment level, and alveolar bone height in four patients. Control segments were treated surgically with defect and root debridement, while experimental segments were treated similarly but included osteoplasty. Results indicated that pockets were eliminated in all control and experimental areas. Osteoplasty sites lost more

bone and attachment than the control sites; however, differences were not statistically significant. Re-entry at 6 months suggested that flap reflection alone resulted in comparable osseous reshaping to that obtained by osteoplasty, but that the combined effect of "bone grinding" and remodeling can contribute to greater reduction of bone support than the flap procedure alone. The authors concluded that the need for grinding to establish an ideal bony architecture is not necessary for gingival health.

Temporary Versus Permanently Exposed Bone. Wilderman (1964) studied healing of alveolar bone in dogs following temporary exposure (replaced flap) and permanent exposure (denudation or apically positioned flap). Permanent bone exposure resulted in undermining resorption of alveolar bone at 2 to 10 days. Interdental and furcation areas consist of a broad base of cancellous bone which will repair completely. Radicular bone generally consisted of a thin zone of cancellous bone between cortical plates and regenerated to 50% of its original height. From 6 to 14 days, soft tissue repair occurred by migration of granulation tissue from wound edges and exposed PDL. By 21 days, epithelial proliferation from the wound edges covers newly formed granulation tissue, with complete epithelial and connective tissue maturation complete by 6 months. Temporary bone exposure (replaced flap) resulted in resorption (days 4 to 8) followed by repair at 21 to 28 days. Unlike permanent bone exposure (undermining resorption), bone healed by primary intention with almost total repair. The author demonstrated gain in attached gingiva when alveolar bone was denuded, but at the expense of alveolar crest height (2 to 4 mm loss) and/or vestibular depth. Osseous denudation resulted in the least amount of bone loss in areas of broad crestal bone with adequate marrow spaces. Disadvantages of denudation included postoperative pain, slow healing, and potential bone loss. Replaced flap procedures minimize these adverse effects. With the exception of the lateral sliding flap, the author discouraged the use of split thickness flaps (restricted visibility and access to bone), deferring to full thickness flaps.

Long-Term Healing Following Flap and Osseous Surgery. Wilderman et al. (1970) studied long-term histologic repair of human tissue following mucogingival flap and osseous surgery and reported that osteoblastic activity was still present 1 year post-osseous surgery. Initial crestal bone loss of 1.2 mm followed by 0.4 mm of new bone apposition resulted in an average reduction in the alveolar crest height of 0.8 mm. Bone thickness was an important determining factor of the amount of post-operative bone loss. Thick bone with marrow spaces exhibited less resorption and greater repair than thin bone. Similar values were reported by Moghaddas and Stahl (1980) in humans 6 months following osseous surgery. The authors used a facial approach to eliminate osseous craters and reported occurrence of remodeling during healing. At 6 months, loss of crestal bone at interdental, radicular, and furcation sites was 0.23 mm, 0.55 mm, and 0.88 mm, respectively. The authors concluded from re-entry measurements and stone models that regeneration of crestal bone does not occur and that healing occurs by repair. Following treatment of ligature-induced angular bony defects in monkeys, Caton and Nyman (1981) reported a loss of connective tissue attachment of 0.45 mm 1 year after ostectomy and a reduction of 0.6 mm in interdental bone height.

Long-term healing results following flap and osseous surgery are summarized in Table 1.

The effect of rotary diamond instrumentation on alveolar bone in dogs was studied by Lobene and Glickman (1963). At 28 days following thinning of facial radicular bone with slow speed, water cooled, rotary diamonds, bone levels ranged from 0.1 mm gain to 1.7 mm loss (mean 0.88 mm loss). In control sites (flap reflection only), facial bone levels ranged from 0.1 mm gain to 0.5 mm loss (mean 0.13 mm loss). The authors reported that final post-surgical bone contours could not be predicted at surgery because of the extensive bone remodeling occurring after grinding. It was also concluded that grinding on alveolar bone results in bone necrosis, reduction in bone height and delayed healing.

Long-Term Effectiveness of Osseous Resective Surgery

Lindhe and Nyman (1975) published 5-year results of an evaluation of 1,620 teeth in 75 patients who had advanced periodontal disease and were treated with surgical pocket elimination. Prior to surgery, 113 of 247 teeth with furcation invasion (45%) were extracted. The remaining 134 teeth with furcation invasion were treated by scaling/root planing or furcation odontoplasty (41%), root resections (51%), and tunneling procedures (7%). All patients had excellent oral hygiene and were recalled every 3 to 6 months for 5 years. At 5 years, plaque and gingival index scores were decreased; probing depths decreased from a mean of 5.7 mm to less than 3 mm; radiographic bone scores indicated no further bone loss; mobile teeth decreased from 57% to 26%; and no teeth were lost. Of the original 75 patients in the aforementioned study, 61 were evaluated at 14 years after osseous surgery (Lindhe and Nyman, 1984). The results reported at 5 years were maintained at 14 years in most patients. Only 0.8% of the sites developed $>$ 2 mm loss of attachment. The authors concluded that pocket elimination surgery combined with good oral hygiene and periodic scaling and root planing resulted in periodontal health.

Comparative Studies

The effectiveness of osseous surgery and open flap curettage was studied by Smith et al. (1980). Twelve patients with moderate periodontitis were included in the study. Two to 3 months after presurgical therapy, contralateral posterior sextants were treated with either apically positioned flaps with osseous recontouring, as described by

TABLE 1. EFFECTS OF OSSEOUS SURGERY ON BONE HEALING

Study	Model	Healing Time	Results
Pfeifer (1963)	Human	21 days	Leaving bone exposed resulted in minimal bone resorption.
Lobene and Glickman (1963)	Dog	28 days	Osteoplasty with diamond rotary instruments resulted in more bone loss (0.88 mm average) than flap reflection alone (0.13 mm average).
Wilderman et al. (1970)	Human	545 days	Osseous surgery resulted in 0.8 mm net reduction of alveolar crest (initial loss of 1.2 mm followed by 0.4 mm new bone apposition).
Donnenfeld et al. (1970)	Human	6 months	Grinding on bone with subsequent remodeling resulted in greater attachment loss than flap elevation alone (serious) weaknesses in this study.
Moghaddas and Stahl (1980)	Human	6 months	Osseous surgery resulted in 0.23 mm interdental bone loss; 0.55 mm loss in alveolar crestal bone height; and 0.88 mm furcation bone loss.
Caton and Nyman (1981)	Monkey	12 months	Osseous surgery resulted in 0.45 mm attachment loss and 0.6 mm interdental bone loss.

Ochsenbein and Bohannan (1963, 1964) and Tibbetts et al. (1976) or open flap curettage. Both procedures were equally effective in reducing plaque, inflammation and probing depth; however, pocket reduction achieved by osseous recontouring was maintained over 6 months while pockets tended to recur after open flap curettage. Osseous surgery resulted in a net loss of attachment of 1.4 mm and open flap curettage resulted in an attachment gain of 0.9 mm. The authors concluded that either procedure could achieve periodontal health.

Olsen et al. (1985) published a 5-year follow-up on 8 of the 12 patients from the aforementioned study. Patients were placed on 6-month recall for the first 2 years and 3-month recall for the last 3 years. At 5 years, plaque and gingival indices for both procedures were similar, but osseous surgery resulted in greater pocket reduction and attachment loss.

Becker et al. (1988) published results comparing scaling, osseous resection, and modified Widman flap procedures on posterior teeth in 16 patients. Patients were seen every 3 months for maintenance. The results after 1 year showed that both modified Widman and osseous surgery are effective in reducing pockets with each resulting in a slight gain of clinical attachment at 1 year. Scaling was effective at maintaining attachment levels but was not as effective in reducing pocket depth.

Kaldahl et al. (1988) reported the 2-year results of a longitudinal study comparing coronal scaling, root planing, modified Widman flap and flap with osseous resective surgery in 82 patients. Results indicated that 1) all therapies reduced probing depth; 2) osseous resection was the most effective in reducing probing depth; 3) probing depths increased in direct proportion to the depth of the pocket; 4) osseous resection produced loss of clinical attachment in the 1 to 4 mm pocket; 5) modified Widman flap and root planing produced the greatest gain of clinical attachment in 5 to 6 mm pockets; and 6) osseous resection resulted in the most recession.

Justification for Osseous Surgery

Disease Activity Studies. In a 1990 study, Badersten et al. monitored non-molar teeth in 39 subjects for 5 years after S/RP seeking a clinical value that may be predictive of loss of probing attachment level > 1.5 mm. The positive predictive value of residual probing depths > 7 mm was 52%, while an increase in PD > 1.0 mm was 78%.

Claffey et al. (1990): 17 patients were monitored for 3.5 years after S/RP to determine a clinical value that may be predictive of loss of probing attachment level > 1.5 mm. They reported a positive predictive value of residual probing depth > 7 mm of 50%. A combination of residual probing depth > 7 mm and bleeding frequency $> 75\%$ had a positive predictive value of 67%.

Microflora/Plaque. The following observations were made by Waerhaug (1978A): If complete subgingival plaque removal has occurred, and adequate supragingival plaque control is instituted, no further subgingival plaque will be formed, and periodontal health can be maintained. In 8 out of the 39 experimental teeth (teeth with at least 2 mm of probeable calculus subjected to S/RP), some subgingival plaque remained following scaling, and these remnants gave rise to a rapid reformation of plaque within the pocket. Small or large remnants of subgingival plaque do not cause clinically symptomatic inflammatory reactions if a high standard of supragingival plaque control is maintained. This is the origin of the term "submarginal gingivitis."

In a subsequent study, Waerhaug (1978B) made the following observations. The deeper the pocket depths, the poorer the plaque removal. The numbers for incomplete subgingival plaque removal were: < 3 mm (17%); 3 to 5 mm (61%); > 5 mm (89%). If it is completely removed and supragingival plaque is adequate, then subgingival plaque will not regenerate. The author's definition of a sulcus was a plaque-free crevice. His definition of a pathologic pocket was one with subgingival plaque attached to the root surface. Since plaque reformation is a slow process, it may take months or a year to completely reform. Waerhaug felt that the most predictable means for obtaining adequate sub-

gingival plaque control was to eliminate pathologic pockets ≥ 3.0 mm. Armitage et al. (1982) found a positive correlation between the percentage of subgingival spirochetes and probing depth.

SUMMARY

The principal goal of osseous surgery is the creation of a bony architecture which is compatible with the maintenance of a physiologic gingival architecture (Schluger, 1949).

The amount of bone removed during properly performed osseous resective surgery is minimal and should be considered clinically insignificant; judicious osteoplasty facilitates improved flap adaptation (Selipsky, 1976). Osseous resective surgery effectively reduces probing depth, but is accomplished by clinical attachment loss. Root planing and modified Widman flap procedures result in the greatest gains in clinical attachment, but do not reduce probing depths as effectively as osseous reflective surgery (Kaldahl et al., 1988).

Indications for osseous surgery include thick ridges of bone; tori; exostoses; incipient furcation invasions; furcation invasion defects; furcation invasions requiring root amputation or hemisection; shallow craters; and minor angular defects (Barrington, 1981).

Contraindications for osseous resective surgery include anatomic limitations, esthetic limitations, inadequate or potentially compromised periodontal attachment, and instances where alternative therapy would be more effective (Wilson et al., 1992).

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Section 7. Root Resection and Odontoplasty

DEFINITIONS

Root Resection: Surgical removal of all or a portion of a tooth root.

Root Amputation: The removal of a root from a multi-rooted tooth.

Hemisection: The surgical separation of a multi-rooted tooth, especially a mandibular molar, through the furcation in such a way that a root and the associated portion of the crown may be removed.

Indications for Root Resection

Indications include: 1) severe bone loss affecting one or more roots; 2) Class II or III furcation invasions/involvements; 3) unfavorable root proximity with adjacent teeth; 4) root fracture, perforation, root caries, or root resorption involving 1 or more roots; and 5) when required endodontic treatment of a particular root cannot be effectively performed (Basaraba, 1969).

Contraindications to Root Resection

Contraindications include: 1) insufficient bone supporting the remaining root(s); 2) unfavorable anatomical situations (i.e., long root trunk, convergent or fused roots); 3) significant discrepancies in adjacent proximal bone heights; 4) impossible to perform required endodontic treatment for the remaining root(s); 5) lack of usefulness of remaining root(s); and 6) non-restorability of the remaining root(s) (Basaraba, 1969).

Endodontic Therapy and Root Resection

Haskell et al. (1980) reported 12 cases of vital root amputation of maxillary molars. A pulp cap, consisting of calcium hydroxide powder, zinc-oxide eugenol cement, and an amalgam alloy seal was placed. Within 5 months, 3 of the 12 teeth lost vitality, 1 tooth was removed for periodontal reasons after 7 months, and 8 of the 12 continued to test clinically vital at 1 year. Histological evaluation of the tooth extracted for periodontal reasons demonstrated an almost complete dentin bridge adjacent to the mummified zone where CaOH was attached to the amputation site.

Filipowicz et al. (1984) followed 86 maxillary molar vital root amputated teeth for 9 years. Dycal and amalgam restorations were placed over the pulp stumps. At 6 months, 41% were non-vital; 62% at 1 year, and 87% at 5 years. Gerstein (1977) concluded that vital root resection with anticipated long-term vitality is a high-risk procedure.

Teeth known to require root resection should receive endodontic treatment prior to resection, while teeth with questionable need for amputation should be assessed at the time of periodontal surgery. If resection is accomplished prior to endodontic therapy, pulpal follow-up should be accomplished as soon as possible. Smukler and Tagger (1976) determined that endodontic therapy can be delayed for 2 weeks after vital root amputation without severe adverse clinical or histological effects. Although pulp stumps were left exposed to the oral cavity, no spontaneous pulpal pain occurred 2 weeks post-amputation. Pulp polyps occurred in 11 of the 26 teeth (21 maxillary molars, 5 mandibular molars) and thermal sensitivity increased in 12 teeth. While endodontic therapy was not greatly hindered by the procedure, difficulty in achieving anesthesia was encountered in most teeth. Periodontal healing was unaffected by delaying endodontic therapy.

Longitudinal Studies of Molar Teeth—Resection Versus Non-Resection

Erpenstein (1983) reported 1 to 7 year (mean 2.9 years) re-evaluations of 34 root resected molars (7 maxillary molars, 27 mandibular molars); only 9 of the resections were accomplished for periodontal reasons. Only 4 of the 34 teeth were evaluated for longer than 5 years. Seven teeth failed, 6 for endodontic and 1 for periodontal reasons. Resections were accomplished with and without surgical access and no osseous surgery was done.

Hamp et al. (1975) treated 310 periodontally involved multirooted teeth. Forty-four percent (44%) were extracted during initial treatment and 50% (87) of the remaining teeth received root resection. Resections were accomplished during pocket elimination surgery. Only 1 root was preserved in 25 of 39 (64%) root-resected maxillary molars. None of the resected teeth were lost during 5 years of maintenance therapy, although 5 developed caries. The authors contributed their success to total elimination of plaque retention areas in the furcations and meticulous patient oral hygiene in conjunction with regular maintenance therapy. In a 10-year follow-up study, Langer et al. (1981) reported on 100 teeth (50 maxillary molars, 50 mandibular molars) which had received root resection primarily for periodontal reasons. In the first 5 years, only 16% of the teeth failed; however, by 10 years, 38% had failed (84% of all failures occurred between 5 and 10 years). Root fracture accounted for 47.4% of the failures, progressive periodontal breakdown for 26.3%, endodontic failure for 18.4%, and cement washout for 7.9%. Nearly twice as many mandibular molars failed as maxillary molars. Mandibular teeth primarily failed due to root fracture (15/25), while periodontal breakdown accounted for most maxillary failures (7/13). The authors attributed the high incidence of fractures to parafunctional nocturnal habits, small size of roots, and weakening of the tooth due to endodontic and prosthetic (post and cores; long spans) treatment.

Green (1986) observed 122 molar teeth (1 to 20 years) that had received root resection following furcal invasion. Sixty-two (62) of these teeth were lost with 24% (15) of the failures occurring during the first 5 years; 58% during the first 8 years; and 73% during the first 10 years. Loss was primarily due to continued breakdown of the periodontium despite 3 to 6 month maintenance.

Buhler (1988) evaluated 28 root resected teeth over a 10-year period. No failures occurred during the first 4 years, 10.7% (3) failed during 5 to 7 years, and a total of 32.1% (9) had failed by 10 years. Failure included endodontic reasons (3), periodontal reasons (2), combination endo-periodontal reasons (2), root fracture (1), and prosthetic reason (1).

Carnevale et al. (1991) studied 488 resected molars, reporting the results for the 3 to 6 year group (62.4%) and a 7 to 11 year group (37.6%). The major reason for resection was periodontal, including Class II or III furcation invasion (81.4%), and deep marginal osseous defects (16.2%). Resection due to endodontic reasons accounted for only 2.2%. All teeth received cast restorations and only 12.8% were restored with single crowns. There were 28 failures (5.7%), 18 of which required extraction. The highest cause of failure was root fracture, followed by caries. Of the failing teeth, there was a higher percentage of teeth requiring extraction in the group that failed due to caries. Only 3 teeth (0.6%) had a recurrence of periodontal breakdown (PD > 5 mm). A higher rate of failure and tooth loss was observed in the 3 to 6 year group when compared to the 7 to 11 year group. A low rate of endodontic failure was found. Only 4 teeth were extracted for endodontic problems, 3 teeth received retrograde fillings, and 2 teeth were endodontically retreated. Undoubtedly, furcation sites respond differently than non-furcation sites.

Kalkwarf et al. (1988) reported additional attachment loss in furcations 2 years post-treatment despite various treatment modalities/quadrant consisting of coronal scaling only, scaling and root planing, scaling and root planing plus modified Widman surgery, and scaling and root planing plus osseous resection. Retrospective longitudinal studies have consistently recorded a high mortality of teeth with furcation invasion regardless of whether the patient's response was categorized as well-maintained, downhill, or extreme downhill (Table 1).

Ross and Thompson (1978) maintained 88% (341/387) of maxillary molars with furcation invasion over a period of 5 to 24 years without root resection or osseous surgery. Treatment consisted of scaling, curettage, occlusal adjustment, gingivectomy/gingivoplasty, and apically positioned flaps in areas of minimal attached gingiva. Eighty-four percent (84%) of these molars had an initial loss of > 50% radiographic bone loss. Forty-six (46) molars were extracted, with 33% (15) functioning for 11 to 18 years and 22% (10) for 6 to 10 years following initial treatment.

Prosthetic Treatment of Root Resected Teeth

Gerstein (1977) advocated full coronal coverage due to the high risk of fracture in endodontically treated root resected teeth. Loss of integrity of the marginal ridge, transverse ridge, and encroachment on the buccal-lingual cusp thickness predisposes the tooth to fracture. A thorough knowledge of the anatomy of the tooth after root resection is essential because the final crown preparation is dictated by the unique contours of the remaining portions of the resected tooth. Abrams and Trachtenberg (1974) advocated: the use of a provisional restoration to resolve any problems in restorative contour, plaque control, and gingival health; smooth continuous contours adjacent to the missing root; adequate embrasures for hygiene access; and flat or concave transitional line angles. Basaraba (1969) recommended: occlusal narrowing; establishment of centric contacts that direct forces along the long axis of the tooth; and elimination of all lateral contacts in root resected teeth.

Keough (1982) noted that root resection in maxillary molars creates an L-shaped configuration when viewed from an occlusal aspect. When the angle between the two legs of this "L" is acute, a cul-de-sac is present which hinders oral hygiene access. The restoration should be designed to compensate for this angle and allow access to this area. Root concavities dictate the outline of the final preparation, and the periodontist should perform odontoplasty (barrel-in) coronal to these concavities to ensure a restoration that will conform to the contour of the remaining tooth.

Majzoub and Kon (1992) performed disto-facial root amputations on 50 extracted maxillary molars and measured the radicular areas. The mean concavity depth on the distal aspect of the teeth was 2.47 mm. The mean minimal mesio-distal dimension of remaining tooth structure was 3.67 mm. The mean distance from the floor of the pulp chamber to the most coronal area of root separation was only 2.70 mm. The results suggest that the depth of concavity will significantly impact maintenance and hygiene; the minimal width of tooth structure can favor fracture; the narrow dimension from chamber floor to furcation opening will often violate biologic width (2.04 mm).

Although splinting of root resected teeth has been advocated in the past, Klavan (1975) observed that removal of 1 root of a maxillary molar does not increase the mobility of the tooth in normal function and that splinting does not seem to be indicated. Only 3 of 33 resected maxillary molars examined had measurable mobility during the 11 to 84 month post-resection evaluation. Two of these 3 teeth were removable partial denture abutments. The author concluded that the use of resected teeth for removable partial denture abutments seems questionable at best.

Tunnel Preparations

Hamp et al. (1975) described the use of a tunnel to provide a complete opening of the furcation, enabling post-surgical access with an interdental toothbrush, and aiding elimination of pathologically deepened soft and hard tissue pockets. They reported 5-year post-tunnel treatment of 7 teeth (6 mandibular first molars and 1 maxillary first premolar) with initial degree III furcation invasion. Three of the teeth had probing depths exceeding 3 mm while 4 of 7 (57%) had caries within the tunnels (3 were extracted).

Hellden et al. (1989) evaluated 149 teeth which had received tunnel preparation 10 to 107 months earlier (mean

37.5 months). The majority of probing sites were less than 3 mm, 11.5 to 36.6% ranged from 4 to 6 mm, and less than 3.8% were greater than 6 mm. Overall, 23.5% of the teeth developed caries. Carious lesions were equally distributed between "inside the tunnel," "outside the tunnel," or a combination of both locations. When root caries incidence was expressed as percent of available root surfaces, 11% of the surfaces developed caries. When compared to Ravald and Hamp's (1981) findings of less than 5% incidence of new caries on exposed root surfaces 2 and 4 years following surgical treatment, tunneled teeth appear to be at higher risk for the development of caries (Table 2).

TABLE 1. PERCENT OF TEETH LOST INITIALLY DIAGNOSED WITH FURCATION INVASION

	Well-Maintained	Downhill	Extreme Downhill	Total
Hirschfeld and Wasserman, 1978	19.3%	69.9%	84.4%	31.4%
McFall, 1982	27.3%	68.9%	92.3%	56.9%
Goldman et al. 1986	16.9%	66.0%	93.0%	43.5%

SUMMARY

Root resection is accepted as a valid treatment modality, when the extent of periodontal involvement dictates (Basaraba, 1969). Endodontic therapy should be completed prior to resection if root resection is a certainty; however, when resection is uncertain, vital root resection can be accomplished during periodontal surgery with minimal sequelae. Endodontic therapy should follow vital root resection in a timely manner. Resection should be accomplished via surgical access. Improved root access allows for proper debridement, pocket elimination, improved flap adaptation, and proper contouring of the resected surface. The distofacial root is the most commonly resected root of maxillary molars, while the mesial root is the most commonly resected root of mandibular molars. The restorative treatment plan should be tailored to the individual tooth. Restorative treatment should allow for adequate access for hygiene and elimination of lateral occlusal forces. Resected teeth may not perform well as removable partial denture abutments. Teeth with tunnel preparations should be monitored closely as they are at increased risk for caries development.

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TABLE 2. ROOT RESECTION STUDIES

Study	Length	Number and Type of Teeth	Root(s) Resected	Reason for Resection	Number Lost and Cause	Additional Findings
Hamp et al. 1975	5 years	87 teeth (39 maxillary molars; 44 mandibular molars; 4 premolars)	DB = 31 MB = 18 P = 15 M = 25 D = 19	Furca involvement	None lost at 5 years	25 maxillary molars had 2 roots removed; 135 of initial 310 multirooted teeth extracted during initial tx; 5 resected teeth developed caries
		7 tunnels (6 mandibular molars)			3 extracted due to caries	4/7 developed caries; 3/7 had pocket depths > 3 mm
Klavan 1975	11-84 months (mean 38.4 months)	34 maxillary molars	DB = 32 MB = 2 P = 1	Periodontal involvement	Abscess and furca invasion, 1	24 not splinted 7 FPD abutments 2 RPD abutments (both became mobile)
Smukler and Tagger 1976	2 weeks	26 molars (21 maxillary 5 mandibular)	DB = 13 MB = 5 P = 3 M = 2 D = 3	Became necessary in course of routine periodontal practice	NA	Pulp polyps formed in 11 Thermal sensitivity in 12 (*no coverage of exposed pulps*)
Haskell et al. 1980	1-3 years	12 maxillary molars (1st molar = 11)	DB = 8 MB = 4	Intrabony lesions around at least one root	Periodontal conditions, 1	Vital root amps, 8/12 vital at one year

Table 2. Continued

Study	Length	Number and Type of Teeth	Root(s) Resected	Reason for Resection	Number Lost and Cause	Additional Findings
Langer et al. 1981	10 years	100 molars (50 maxillary 50 mandibular)	NA	Most due to periodontal condition (no number given)	Root fx = 18 Periodontal condition = 10 Endodontic = 7 Cement washout = 3	Maxillary failures 13 (perio = 7) Mandibular failures 25 (fx = 15) 4 years = 6% failure 7 years = 27% failure 10 years = 38% failure (84% of failures occurred at 5-10 yrs)
Erpenstein 1983	1-7 years (mean 2.9)	34 molars (7 maxillary, 27 mandibular)	DB = 1 MB = 4 P = 1 M = 24 D = 3	Periodontal condition = 9 Endodontic = 20 Caries = 4 Other = 1	Periodontal = 1 Endodontic = 6	Only 16 patients had periodontal examination recorded; of these, 9 had advanced periodontitis
Filipowicz et al. 1984	5 years	86 maxillary molars	NA	NA	6 months (41% non-vital) 12 months (62% non-vital) 5 years (87% non-vital)	Vital root amps Dycal and amalgam Pulp caps
Green 1986	1-20 years	122 molars	NA	All had furca invasion	62 failures (41 maxillary, 21 mandibular), most due to periodontal breakdown	
Buhler 1988	10 years	28 teeth (16 maxillary molars, 1 premolar 14 mandibular molars)	DB = 15 MB = 5 P = 4 M = 13 D = 1	Periodontal treatment	fx = 1, Periodontal condition = 2 Endodontic = 3 Periodontal condition/ endodontic = 2 Other = 1	4 years = 0 failures 7 years = 10.7% failure 10 years = 32.1% failure
Hellden et al. 1989	10-107 months (mean 37.5)	149 teeth (6 maxillary premolars; 91 maxillary molars; 52 mandibular molars)			6 extracted due to caries 6 hemisected due to caries 3 others extracted 23 additional molars developed caries	23.5% developed caries; 11% of surfaces

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Section 8. Osseous Grafting

DEFINITIONS

Regeneration: Reproduction or reconstitution of a lost or injured part.

Repair: Healing of a wound by tissue that does not fully restore the architecture or the function of the part. Historical note: Although in the past, fiber orientation was used to determine new attachment, it is now believed that parallel fibers can be a result of early stages of periodontal ligament (PDL) development or a parallel orientation may be the result of low power microscopic evaluation (at higher magnifications, fibers appear attached in a perpendicular manner).

Reattachment: To attach again. The reunion of epithelial and connective tissues with root surfaces and bone such as occurs after an incision or injury. Not to be confused with new attachment.

New Attachment: The union of connective tissue or epithelium with a root surface that has been deprived of its original attachment apparatus. This new attachment may be epithelial adhesion and/or connective tissue adaptation or attachment and may include new cementum.

Osteogenesis: Development of bone; formation of bone.

Autograft: Tissue transferred from one position to another within the same individual.

Allograft: A graft between genetically dissimilar members of the same species.

Goal of Osseous Grafting

Schallhorn (1972) reported that the objectives of osseous grafting were pocket elimination, restoration of the lost alveolar process, and regeneration of a functional attachment apparatus.

Factors Influencing Graft Success

Factors include: oral hygiene, defect morphology (number of walls, narrow versus wide), furcation involvement, operator technique, and graft material used. Another characteristic that may influence grafting success is the particle size of the graft material. Zaner and Yukna (1984) examined particle size of autogenous bone obtained by high and low speed burs and hand chisels, freeze-dried bone allograft obtained from a bone bank, and several alloplastic implant materials. They determined that bone-blend had the smallest and most uniform particle size (210 x 105 μm) and hand chiseled bone chips, the largest and least uniform particle size (1559 x 789 μm). Grafts obtained with high and low speed burs had a particle size of roughly 300 to 500 μm. Since a minimum pore size of 100 μm is needed between particles to allow vascularization and bone formation, it was concluded that a particle size of about 380 μm would be most appropriate.

GRAFT SOURCES

Autografts

Autogenous **iliac crest marrow** grafts are considered the most predictable method of osseous regeneration. This may be due to osteogenic, osteoinductive, and osteoconductive properties associated with undifferentiated cells or osteoblasts surviving within the graft material that may form new bone; necrosis of the graft material and release of substances that may stimulate new bone formation; and nonviable cellular elements within the graft that may act as a scaffold for new host bone formation. In a study of 182 transplant sites, Schallhorn et al. (1970) utilized fresh and frozen cancellous bone and marrow from the iliac crest. The mean increases in crestal bone height in "no-wall" defects was 2.57 mm. While 1-walled defects demonstrated complete fill in 11 of 21 sites and a mean increase in bone height of 3.75 mm, 2-walled defects all demonstrated complete fill to the coronal margin of the existing bony walls with a mean increase in bone height of 4.18 mm. Overall, the mean bone fill for all defects was 3.33 mm. Teeth with furcation invasion (mostly Class II) demonstrated complete furcation fill in 7 of 8 sites, while the remaining furcation showed partial fill of two-thirds of the original defect. The mean increase in height of the furcation grafts was 4.5 mm. The frozen specimens demonstrated the greatest mean bone apposition, which was believed to be due to cellular breakdown and release of an inductive substance. New attachment was present at 3 months.

Dragoo and Sullivan (1973A) evaluated fresh autogenous iliac bone grafts clinically and histologically over a 2 to 8 month period. Clinically, there was a 2.1 mm increase in bone level as measured by bone sounding and a 1.56 mm radiographic increase, with the difference attributed to measurement error. Histologically, the authors found 1.4 mm of gingival recession, 1.02 mm sulcus depth, 1.34 mm of epithelial attachment, a connective tissue attachment of 1.02 mm, and supracrestal bone regeneration of 0.7 mm. New cementum was present as early as 2 months, with a functionally oriented PDL noted at 3 months. Osteoblastic activity was greatest at 2 months, but persisted at 8 months. Also at 8 months, the PDL was completely matured. Autogenous iliac crest marrow may be used fresh or may be stored prior to use. In a study in dogs, Bierly et al. (1975) found that iliac bone could be stored in minimum essential medium (MEM) at refrigeration temperature (4°) for a maximum of 7 days before sustaining a dramatic decrease in graft viability. Sottosanti and Bierly (1975) reviewed storage techniques for iliac marrow. They suggested that mar-

row to be used within 7 days of harvest could be stored in MEM in a standard refrigerator and that marrow to be stored longer than 7 days should be placed in MEM containing 12 to 15% glycerol as a cryopreservative and then slowly cooled prior to placement in freezer. Material should not be stored frozen for longer than 2 months. When ready for use, the authors state that the marrow should be quickly warmed in a 37°C water bath prior to placement. They felt that there was no need to remove the glycerol at this low concentration. Marx et al. (1979) studied cellular survival of iliac marrow-cancellous bone specimens over a 4-hour period by placing cell suspensions in 6 different media and testing each specimen every half-hour using vital dye exclusion and tritiated thymidine uptake to measure the percent viability. Five percent dextrose in water (D5W), normal saline, and tissue culture medium number 199 demonstrated 92 to 100% cell viability over the 4-hour period. Distilled water retained 0% viable cells. This study showed that a high survival rate can be obtained with D5W, normal saline, or tissue culture media with long delays (4 hours) between harvest and placement of the graft.

Schallhorn (1972) described 5 categories of postoperative complications associated with autogenous iliac grafts including infection; sequestration (the most common complication); variable healing time; rapid defect recurrence related to poor oral hygiene; erratic maintenance schedules or poor nutritional status; and root resorption. Viability of the marrow elements is thought to play a role in root resorption, with previously frozen marrow not demonstrating a resorptive association. Excessive root preparation with dentin exposure, hypermobility, and hypomobility have also been implicated in root resorption. Dragoo and Sullivan (1973B) reported that 2.8% of 250 sites which received fresh iliac grafts showed root resorption. Furthermore, root resorption was always associated with chronic inflammation of the adjacent gingiva, while resolution of the inflammation was accompanied by arrest and repair of the resorptive lesion. All cases of root resorption originated at or coronal to the bony crest. Possible etiologic factors include undifferentiated cells from the marrow, proteolytic enzyme activity of PMNs, macrophages, and other host cells and spread of the resorptive process from sequestered bone.

Autogenous intraoral bone has also been used as donor material in osseous grafting. High or slow speed handpieces, chisels, trephines, or rongeurs may be used to remove bone from donor sites. In addition, a technique has been described where bone from an area adjacent to the defect is forcibly pushed into direct contact with the root surface without separating the bone from its base. This procedure was called bone swaging by Ewen (1965) or a contiguous autogenous transplant by Ross et al. (1966).

Diem et al. (1972) introduced a **bone blend** and its preparation in which intraoral cortical and/or cancellous bone from an extraction site, edentulous ridge, exostosis, or the region of the defect was placed in a sterile amalgam capsule with a pestle and triturated for 60 seconds.

Hiatt and Schallhorn (1973) compared grafts with **cortical-cancellous** material obtained from the maxillary tuberosity, edentulous ridges, and extraction sites to those using iliac crest bone. A mean increase in bone height of 3.44 mm accompanied intraoral bone grafts. Measurements taken from 9 months to 7 years, consisting of radiographic evaluation and measurement of bone height, re-entry, or bone sounding showed no significant difference between the 3 types of donor tissue. With the exception of furcations and crestal defects, the results of grafts using intraoral bone were comparable to those obtained with iliac grafts. Histologic evaluation of a single block section demonstrated formation of new cementum, bone, and PDL. The importance of site morphology was discussed with an increased number of osseous walls resulting in an increased predictability and degree of success. The authors noted that marrow from the maxilla had the greatest probability of containing foci of red marrow with associated pleuripotential cells. Kucaba and Simpson (1978) investigated the maxillary tuberosity for presence of hematopoetic marrow. They found that 53% of autopsy specimens contained red marrow mainly located in the most superior, posterior aspect of the tuberosity. However, none of the 11 specimens taken from these sites in live patients during periodontal surgery contained hematopoetic marrow.

Other sources of intraoral graft material include edentulous ridges, exostoses/tori, healing extraction sites, and surgically-created osseous defects. Evian et al. (1982) investigated the optimal time period for obtaining graft material from extraction sites. They found that by 8 to 12 weeks there was a substantial quantity of mature bone present which contained osteoblasts and osteoid. Soehren and Van Swol (1979) noted that it took longer in the mandible (12 weeks) than in the maxilla (8 weeks) to obtain similar grafting material. The difference in maturation time between the maxilla and mandible may relate to the more vascular nature of the maxilla. The impacted third molar extraction site yielded the best material. Halliday (1969) used a trephine to surgically create defects in the mandible which were re-entered in 6 to 8 weeks for retrieval of graft material.

Langer and Geib (1977) evaluated early re-entry procedures after the placement of intraoral cancellous bone grafts from the tuberosity and extraction sites into combination 1-, 2-, and 3-walled defects. No pre-surgical initial preparation was completed at graft sites and surgical flaps did not completely cover the bone grafts. Re-entry with connective tissue fiber retention was performed 3 months following surgery. Upon re-entry, reattachment was judged by clinical connective tissue attachment. Results indicated that re-entry at 3 months did not adversely affect the maturation of a graft.

Robinson (1970) discussed the use of graft material derived from grindings of cortical intraoral bone which were mixed with blood to form an **osseous coagulum**. The author believed that this technique would offer an adequate source of bone as well as provide small particles. While success has been noted with this technique (Mellonig et al. 1981), no quantitation of results in large populations has been noted.

Allografts

Schallhorn and Hiatt (1972) treated 194 sites of varying morphology with **iliac crest allografts** which were matched to the patient by human lymphocyte antigen (HLA) typing. Success was determined by pre- and post-osseous charting 26 months after surgery. Bony apposition averaged 3.62 mm in 1-, 2-, and 3-walled defects; 3.30 mm in furcations; and 2.0 mm in no-walled defects for a total mean apposition of 3.07 mm. Limited histology indicated replacement of the allograft with viable bone and evidence of root resorption. While no adverse periodontal reactions were noted with grafts, 3 of 20 patients developed cytotoxic antibody activity to a panel of human lymphocytes.

Sepe et al. (1978) reported the use of **freeze-dried bone allografts** (FDBA) placed by 53 periodontists in over 800 defects with varying morphology. Re-entry on 189 sites was performed at 1 year and demonstrated that 60% of the sites had more than 50% bone fill. Osseous regeneration was least effective in the furcations. Again, no adverse effects were noted. In an attempt to compare osseous grafting with open flap curettage in a controlled manner, Altiere et al. (1979) treated 10 pairs of intraosseous defects. Irradiated FDBA was compared to control sites which received a flap curettage only. At 40 to 50 weeks, there was no significant difference between FDBA-treated defects and control defects with respect to pocket reduction, osseous regeneration, or amount of new attachment. While this study reports the worst performance of osseous grafts, it differs in that irradiated bone was used. This may remove or compromise the inductive potential present in the graft material.

Shapoff et al. (1980) found that small particles (100 to 300 μm) of FDBA enhance osteogenesis when combined with autogenous marrow to a much greater degree than do large particles (1000 to 2000 μm) of FDBA. They suggest that smaller particles may increase surface area around which new bone may form; increase the surface area for resorption, leading to exposure of greater amounts of bone morphogentic protein in the bone matrix and thus increase osteoinduction; increase the number of pores, physically enhancing osteogenesis; and/or enhance necrosis of marrow with release of osteogenic substances and facilitation of osteoblast differentiation. The reconstitution of FDBA with a tetracycline solution resulted in enhanced bone formation from 3 to 5 weeks in nylon mesh chambers placed in bony defects in maxilla and mandibles of baboons (Drury and Yukna, 1991).

Quattlebaum et al. (1988) investigated the antigenicity of FDBA in human periodontal osseous defects and could not detect anti-HLA antibodies in any of the 20 patients at any time. However, the authors also noted that it is possible that the amount of FDBA used during routine periodontal grafting procedures provides an insufficient antigenic challenge to the patient. The safety of FDBA was shown by Mellonig et al. (1992) who evaluated HIV-spiked human cortical bone and bone obtained from an AIDS patient. The bone was tested for the presence of HIV both before and after processing. The acid decalcification process and use of virucidal agents destroyed the HIV, thus showing the safety of DFDBA. Only 2 cases of HIV transmission from grafting have been known to occur and both were with unprocessed fresh or frozen bone allografts.

The osteogenic potential of **demineralized freeze-dried bone allografts** (DFDBA) has been investigated by Quintero et al. (1982). In a clinical study of DFDBAs in 1-, 2-, and wide 3- walled defects using a re-entry procedure at 4 to 6 months, these authors reported an average bone fill of 65% (2.4 mm). This was accompanied by a mean increase in probing attachment level of 1.9 mm. Histology was not performed, so the character of the new soft tissue attachment could not be determined. Mellonig et al. (1981) surgically created defects in guinea pig calvaria to evaluate the osteogenic potential of 4 different grafting materials. Samples of FDBA, DFDBA, autogenous osseous coagulum, and autogenous bone blend were placed in the defects and the areas examined histologically at varying time intervals. There was a significantly greater amount of new bone formation with the DFDBA than with any of the other materials. Osseous coagulum and bone blend were equally osteogenic/inductive, while the FDBA showed the least amount of new bone formation. Data from this animal model showed that the amount of new bone 28 to 42 days after grafting was virtually equal among the various grafting techniques.

Rummelhart et al. (1989) compared DFDBA to FDBA in 22 intra-patient paired defects in 9 patients. Grafted sites were re-entered 6 to 12 months post-operatively. No significant differences were found between treatment groups in any parameter measured. Mean osseous repair of 1.7 mm occurred with DFDBA and 2.4 mm with FDBA. The clinical response of both treatment groups may have been affected by a predominance (55%) of 1-walled defects treated.

Sanders et al. (1983) compared the clinical effects of FDBA alone and **composite FDBA/autogenous bone grafts** (FDBA/ABG) in a large number of 1-, 2-, and wide 3-walled defects as well as furcation defects. The ABGs were either bone blend, osseous coagulum, or iliac marrow. In results similar to Sepe et al. (1978), 63% of FDBA grafts showed more than 50% bone regeneration, while 80% of composite FDBA/ABG grafts resulted in more than 50% bone fill, with complete fill in 33%. Successful grafts (50%

bone fill) were related to good primary wound closure and vital teeth. The difference was most pronounced in furcations and 1- and 2-walled defects. The apparent success of the composite grafts may have been due to the osteoinductive effect of either the FDBA or the autogenous material. A greater success rate (more than 50% fill) was noted when antibiotics were used (85%) than when they were not used (38%). There was a lower success rate noted for grafts adjacent to endodontically treated teeth; however, this may have been related to difficulty in diagnosing residual endodontic pathology and to possible inadequate initial endodontic treatment.

Controlled Studies

One of the criticisms of the osseous grafting literature is the lack of comparison to ungrafted controls in many of the studies (Bowers et al., 1982). In the last of a series of articles, Froum et al. (1976) compared osseous coagulum-bone blend to open curettage with respect to osseous fill of 1-, 2-, and 3-walled defects as measured at a 7 to 13 week surgical re-entry. The average osseous fill was 70.6% (2.98 mm) in osseous coagulum grafted sites, 60.7% (4.36 mm) in iliac crest sites (1975 study), and 21.8% (0.66 mm) fill in the open debridement sites. In a subset of the patients who received both procedures, grafted sites demonstrated 2.18 mm of fill (3.22 mm initial defect depth) compared to 0.75 mm of fill (2.55 mm initial defect depth) for the open debridement sites. The original defect depth was greater in grafted than non-grafted sites. The deeper defects tended to show greater fill regardless of the treatment. Seven to 13 weeks may have been too early to adequately evaluate healing, since Dragoo and Sullivan (1973A) had previously shown that osteoblastic activity was still present at 8 months.

Histologic Studies

Radiographs, bone sounding, and re-entry procedures have often been used as methods of evaluating regeneration. However, none of these procedures is capable of demonstrating the formation of a new attachment apparatus. In an attempt to demonstrate that autogenous grafts were capable of forming a functional PDL, Moskow et al. (1979) presented the histologic assessment of a single 1-, 2-, and 3-walled combination osseous defect treated with cancellous alveolar bone taken from an edentulous ridge. A block section obtained 28 weeks after grafting revealed a long junctional epithelium between the root surface and the newly formed bone. The only areas of newly formed cementum and functionally oriented and inserted PDL were at the very base of the defect, a finding expected with any procedure due to the proximity of the PDL to the base of the defect. The clinical evidence of newly formed bone did not necessarily connote the presence of new attachment; i.e., new cementum and functional PDL. Listgarten and Rosenberg (1979) treated 1-, 2-, and 3-walled and combination defects with autogenous intraoral bone, iliac crest allografts, or no grafts. Grafts were placed with or without root planing, and patients were not placed on regular maintenance intervals. Oral hygiene was ineffective. Fifteen (15) block sections were obtained after 6 to 12 months for histologic preparation. Sections demonstrated the presence of a long junctional epithelium below the alveolar crest at 52 to 85% of the total defect depth in all grafted and non-grafted sites. A long junctional epithelium was frequently found apical to the grafted bone, between the bone and root surface. Conversely, Dragoo and Sullivan (1973A) found histologic new attachments as early as 3 months after grafting with fresh autogenous iliac marrow, with complete maturation of the attachment apparatus at 8 months. In a single case treated with autogenous intraoral bone, Hiatt and Schallhorn (1973) showed formation of new attachment in the grafted site. Bowers et al. (1982) compiled the results of 26 studies which histologically evaluated new attachment in intraosseous lesions treated with grafting and non-grafting techniques. New bone formation was seen in 87% of grafted specimens and new cementum in 85%. A functional PDL was reported in most of the cases, although some reported a parallel orientation. The junctional epithelium was found apical to the alveolar crest in 19% of grafted cases. In general, non-grafted sites showed less bone fill, less new cementum, and a greater chance of repair by a long junctional epithelium rather than regeneration of a functional attachment.

New Attachment Studies

As mentioned previously, presence of bone fill subsequent to grafting procedures does not prove new attachment. Bowers et al. (1985, 1989) compared the potential for regeneration in osseous defects associated with submerged and non-submerged roots, with and without DFDBA, in a 6-month human histologic study. Non-submerged, non-grafted sites were found to heal by long junctional epithelium, while non-grafted submerged defects healed by connective tissue attachment (< 1 mm new attachment). Ninety-six percent (96%) submerged and 68% of non-submerged defects grafted with DFDBA formed a new attachment apparatus with bone and cementum formation. This attachment usually extended from the base of the defect to < 1 mm coronal to the sub-calculus notch. The authors pointed out that while epithelial exclusion may be necessary for regeneration to occur, it is only one of several factors, as only limited bone and cementum formation were observed even under these circumstances. Epithelial exclusion in this study was associated with a greater degree of regeneration and new connective tissue attachment in the absence of root resorption or ankylosis.

Karring et al. (1984) reported the effect of epithelial downgrowth on periodontal wound healing using the monkey model. Periodontally involved teeth were extracted, root planed, and reimplanted with 1 surface in contact with the bone and the other with the connective tissue surface

of the repositioned flap. Epithelial migration was allowed to occur by surgical exposure of the coronal surface at periods up to 24 weeks. The authors reported root resorption at the connective tissue interface and ankylosis at the bone interface, the degree of which was dependent on the duration of implantation. The portions of the roots covered with epithelium showed no resorption. The authors concluded that epithelium forms a protective barrier which prevents root resorption and ankylosis. The root resorbing properties of gingival connective tissue and the ability of granulation tissue derived from alveolar bone to induce root resorption and ankylosis in animal models were discussed. The infrequent occurrence of such events following osseous grafting in humans may be explained by coronal migration of PDL cells and/or apical migration of the junctional epithelium.

SUMMARY

There are numerous reports showing the success of osseous grafting; however, the majority have not used controls. Additionally, the method of assessing success was generally through radiographs or osseous measurements. Long-term studies utilizing adequate controls and histologic evaluation are needed to make firm conclusions on the efficiency of osseous grafting as a means of providing regeneration of the periodontal attachment apparatus (Gara and Adams, 1981).

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Section 9. Alloplastic Materials for Treatment of Intrabony Defects

DEFINITIONS

Graft: 1. Any tissue or organ used for implantation or transplantation. 2. A piece of living tissue placed in contact with injured tissue to repair a defect or supply a deficiency. 3. To induce union between normally separate tissues.

Implant: 1. An alloplastic material or device that is surgically placed into the oral tissue beneath the mucosal or periosteal layer or within bone for functional, therapeutic, or esthetic purposes. 2. To insert a graft or alloplastic device into the oral hard or soft tissue for replacement of missing or damaged anatomical parts, or for stabilization of a periodontally compromised tooth or group of teeth.

INTRODUCTION

If the present goal of periodontal therapy is to provide a healthy dentition that will function for life, then the ideal goal for the future is the reconstitution of bone and connective tissue attachment which has been destroyed by the disease process (Zander et al., 1976). At present, one of the most widely used periodontal regenerative modalities is bone graft therapy. Unfortunately, bone graft materials derived from the host or other living tissues may be complicated by inherent limitations. Consequently, dental research and industry have been increasingly concerned over the past 2 decades with biologically inert, synthetic materials for implantation into intrabony periodontal defects. The first major breakthrough for modern alloplastic bone implant materials came during the past 2 decades with the development of advanced processing techniques for the calcium phosphate ceramics. This led to significant improvement in the bioproperties of these materials, making them a feasible alternative for use as alloplastic implant materials (Han and Carranza, 1984; Krejci et al., 1987). Subsequent extensive clinical investigation of calcium phosphate ceramics as implant materials, along with documented clinical success, has contributed to the current demand for alloplastic implant materials.

OSSEOUS GRAFT AND IMPLANT MATERIALS–ADVANTAGES AND DISADVANTAGES

Since each graft or implant material has unique strengths and weaknesses, the practitioner must become familiar with these prior to selection and use. Autogenous materials, such as iliac crest bone marrow, bone blend, and osseous coagulum, have the advantage of safety and no potential for immunogenicity or cross contamination. Autogenous bone has a well-documented osteogenic potential and is logically the material of choice. However, in cases where insufficient autogenous material is available or an additional surgical site is contraindicated, other materials must be considered. Allograft materials such as bone marrow and decalcified freeze-dried bone have virtually unlimited availability and a purported osteogenic potential comparable to autogenous bone. However, a slight risk of immunogenicity and pathogenicity exists (MMWR, 1988; Buck, 1989). Mellonig et al. (1992) demonstrated that processing of demineralized freeze-dried bone allografts results in inactivation of HIV. After HIV-spiked and AIDS bone donor samples were processed, the HIV was destroyed. The authors attribute the HIV inactivation to the demineralization and the virucidal processing agent (ethanol and non-ionic detergent). The inherent shortcomings of bone autografts and allograft materials have focused considerable attention on synthetic materials for the treatment of periodontal defects. These materials are available in unlimited quantity, require no donor surgical site, and are non-antigenic and sterile (Bissada and Hangorsky, 1980; Ferraro, 1979; Han and Carranza, 1984).

Ideal Properties of Alloplastic Implant Materials

Although some of the current alloplastic implant materials have a great deal of potential, no ideal material exists today. Any successful laboratory-synthesized alternative to autografts and allografts must possess properties that clinically appeal to the practitioner. They should also afford the highest potential for restoring lost form and function to the periodontium. The following qualities are necessary if an alloplastic implant material is expected to meet these therapeutic goals: biocompatibility with host tissues–nontoxic, non-allergenic, non-carcinogenic, and non-inflammatory; sufficient porosity to allow bone conduction–growth of bone into and around the implant; ability to stimulate bone induction; resorbability with replacement by bone; radiopacity which permits radiographic visualization; capability to withstand sterilization procedures without compromising desired characteristics; easy to obtain and inexpensive; similar physical properties to the tissue it replaces; and stable to variations in temperature and humidity (Bissada and Hangorsky, 1980; Frame et al., 1980; Levin et al, 1974A; Alderman, 1969; Ganeles et al., 1986).

NON-CERAMIC ALLOPLASTIC IMPLANT MATERIALS

Although most synthetic materials currently used to treat periodontal defects are calcium phosphate ceramic compounds (tricalcium phosphate, porous hydroxyapatite, nonporous hydroxyapatite or combinations of hydroxyapatite and tricalcium phosphate [biphasic calcium phosphate]), a number of non-ceramic synthetic materials have also been used in recent years. These include materials such as calcium sulfate (plaster of paris) which is readily available, inexpensive, easy to handle, and sterilizable. Although it has been shown to be biocompatible and readily absorbed, there is no evidence that it has any osteoinductive properties (Shaffer and App, 1971).

Hard Tissue Replacement (HTR)

HTR is a calcium-layered composite of a polymethylmethacrylate core (PMMA) with polyhydroxylethyl meth-

acrylate (PHEMA or polyhema). It is microporous, nonresorbable, sterile, and ready to use in granular or molded forms. Treated with barium to promote radiopacity, it is also touted as being strong, easy to use, inexpensive, hydrophilic for easier handling, and electrically charged in order to promote osteogenesis (Ashman and Bruin, 1985; Ashman, 1988). Its most important dental application to date has been for bone maintenance, i.e., placement in extraction sockets to prevent bone loss and for ridge augmentation (Leake, 1988). There is some evidence that fibroblasts attach to this material, suggesting that it may augment connective tissue repair and promote wound healing (Kamen, 1988). HTR has been shown to be well tolerated and easy to handle (Shamiri et al., 1988) and has been used with apparent clinical success for filling periodontal defects (Murray, 1988). Histologically, it resulted in gain in clinical closure varying from epithelial adhesion to degrees of functionally oriented new connective tissue attachment to cementum. Limited bone formation was also noted at the periphery of some particles (Stahl et al., 1990). Compared to open flap debridement alone, use of HTR has been shown at 6 months post-implantation to result in greater decrease in probing depth and gain in clinical attachment (Yukna, 1990). Although most reports indicate that this material is biologically well accepted, Kwan et al. (1990) reported 2 cases in which an inflammatory reaction to the material occurred. Since there have been few controlled clinical studies verifying the efficacy of HTR, no conclusions regarding the predictability of the histologic healing responses can be made.

CERAMICS AS ALLOPLASTIC IMPLANT MATERIALS

Ceramics are enticing because of well-documented biocompatibility and chemical and physical resemblance to bone mineral (Han and Carranza, 1984). Another desirable feature includes an ability to develop strong bonding to living bone by natural cementing mechanisms, reportedly stronger than either the constituent bone or ceramic material (Jarcho, 1986). In addition, most calcium phosphate materials are radiopaque, sterilizable, easy to obtain, and stable (Table 1).

The basic manufacturing process of calcium phosphate ceramic implant materials involves preparation of ceramic powders in aqueous solutions. The powders are then subjected to high pressures and high heat, a process known as sintering. Differing manufacturing processes of these ceramics can control particle and pore size, shape, distribution, and density of the material. There are many ceramic periodontal implant materials available on the market with various physical and chemical characteristics, and tissue reaction properties. Most of the ceramics currently used as implant materials are composed of either hydroxyapatite (also referred to as HA, hydroxylapatite, durapatite, or tribasic calcium phosphate) or tricalcium phosphate (TCP or β-TCP) in various forms. Both materials have a calcium to phosphate ratio similar to bone and have been shown to be biologically compatible (Froum et al., 1982; Moskow and Lubarr, 1982; Rabalais et al., 1981). These materials are further grouped as either porous or non-porous, and resorbable or non-resorbable. Those materials with moderate sized particles (0.3 to 0.5 mm, 40 to 60 mesh) are generally used for filling periodontal defects, while those with larger particles (0.5 to 1.0 mm, 20 to 40 mesh) are more appropriate for ridge augmentation procedures.

Tricalcium Phosphate (TCP)

This processed ceramic material, also referred to as β-tricalcium phosphate, should not be confused with tribasic calcium phosphate, a generic name for hydroxyapatite (Metsger et al., 1982). Tricalcium phosphate is a highly purified, multicrystalline, porous form of calcium phosphate. The calcium/phosphate ratio of TCP is 1.5 and is similar to that found in bone mineral (1.7). However, it is not a natural component of bone mineral (Han and Carranza, 1984). TCP is partially resorbable and is often considered desirable for repair of non-pathologic sites where resorption of the implant material and concurrent bone replacement might be expected (Jarcho, 1986). When used to repair marginal periodontal defects, TCP may provide degrees of repair equal to or exceeding autogenous bone (Metsger et al., 1982).

Animal Studies. Numerous animal studies have shown that TCP is compatible with host tissue and elicits no adverse reactions (Bhaskar et al., 1971; Levin et al., 1974A and B, 1975; Nery et al., 1975; Cameron et al., 1977; McDavid et al., 1979). Ingrowth of bone into the pores of CP has been observed in dogs with repair of the periodontium (Nery et al., 1975; Cameron et al., 1977). Additionally, TCP seems to undergo progressive degradation and replacement by calcified bone as shown in dogs (Bhaskar et al., 1971; Cameron et al., 1977; Ferraro, 1979) and may even stimulate bone formation (Levin et al., 1974B). Histologically, a long junctional epithelial attachment, rather than a connective tissue attachment, occurs in the healing defect (Caton et al., 1980A and B).

Human Studies. The relative success of TCP that was demonstrated in animal studies precipitated a number of human clinical investigations. These have provided further evidence of TCP's biocompatibility (Nery, 1978; Strub and Gaberthal, 1979; Hoexter, 1983; Judy, 1983; Snyder et al., 1984; Baldock et al., 1985; Bowers et al., 1986; Stahl, 1986; Froum and Stahl, 1987). They also indicated that use of TCP results in partial defect fill; i.e., bone and/or ceramic (Nery, 1978; Strub and Gaberthal, 1979; Snyder et al., 1984; Baldock et al., 1985; Stahl and Froum, 1987; Froum and Stahl, 1987; Saffar et al., 1990). At 6 months, TCP has been shown to be well retained, provide flap support, reduce mobility, and preserve function (Hoexter, 1983).

Nery (1978) clinically evaluated TCP-treated periodontal osseous defects in 6 patients over 2 to 16 months. A mean

TABLE 1. HUMAN STUDIES USING CERAMIC IMPLANT MATERIALS TO TREAT OSSEOUS PERIODONTAL DEFECTS

Study	Design	Material(s)	Re-entry Time	Defect Fill*	Probing Depth Reduction	Clinical Attachment Gain/Gain in Closure
Strub and Gaberthal 1979	8 pts 29 defects TCP compared to 18 defects allogenic frozen bone—same mouth	TCP and frozen bone	12 months	TCP-1.2 Bone-1.5	TCP-1.8 Bone-2.0	†
Snyder et al. 1984	10 pts 15 defects No controls	TCP	18 months	2.8	3.6	2.7
Baldock et al. 1985	2 pts 13 defects No controls	TCP	Block biopsy at 3, 6, 9 months; no reentry	1.8 (X-ray only)	4.5	2.0
Stahl and Froum 1986	4 pts 8 defects No controls	TCP	Block section 3-8 months; no reentry	†	5.6	2.6
Froum and Stahl 1987	1 pt 5 defects No controls	TCP	Block section 13-18 months; no reentry	†	5.1	2.3
Rabalais et al. 1981	8 pts 37 defects HA compared to 29 defects debrided controls—same mouth	Non-porous HA (250–425 μm particles)	6 months	HA-1.7 Cont-0.5	HA-2.6 Cont-2.3	HA-1.0 Cont-0.9
Meffert et al. 1985	12 pts 16 HA defects compared to 12 defects debrided controls—same mouth	Non-porous HA (40–60 mesh particles)	9 months	HA-3.4 Cont-0.5	†	†
Yukna et al. 1986	14 pts 50 HA defects compared to 31 defects debrided controls—same mouth	Non-porous HA (40–60 mesh particles)	6 months	HA-1.6 Cont-0.4	HA-2.5 Cont-2.3	HA-1.0 Cont-0.8
Yukna et al. 1989	6 pts 62 HA defects compared to 32 defects debrided controls—same mouth	Non-porous HA (40-60 mesh)	5 years soft tissue measurements only; no reentry	†	HA-2.8 Cont-1.4	HA-1.1 Cont-0.5
Kenney et al. 1985	25 pts 25 HA defects compared to 25 defects debrided controls—same mouth	Porous HA	6 months (15 exp and 15 control defects)	HA-3.5 Cont-0.7	HA-4.3 Cont-2.5	HA-3.6 Cont-1.2
Stahl and Froum 1987	3 pts 12 HA defects No controls	Porous HA	Block sections; no reentry	†	4.4	2.8
Kenney et al. 1988	10 pts 10 solid porous HA defects compared to 10 granular porous HA defects—same mouth	Porous HA	No reentry	†	Solid-5.2 Gran-4.2	Solid-3.0 Gran-2.6
Krejci et al. 1987	12 pts Comparison of 12 non-porous, 12 porous, and 12 debrided control defects—same mouth	Non-porous HA vs. porous HA	6 months	Por-1.0 Nonpor-1.4 Cont-0.5	Por-3.0 Nonpor-2.3 Cont-2.4	†
Bowen et al. 1989	6 pts 17 HA defects compared to 17 DFDBA defects—same mouth	DFDBA vs. porous HA	6 months	HA-2.1 DFDBA-2.2	HA-2.9 DFDBA-2.9	HA-1.6 DFDBA-2.1

TABLE 1. Continued

Study	Design	Material(s)	Re-entry Time	Defect Fill*	Probing Depth Reduction	Clinical Attachment Gain/Gain in Closure
Oreamuno et al. 1990	24 pts 24 porous HA defects compared to 24 DFDBA paired defects—same mouth	DFDBA vs. porous HA	6 months	HA-3.3 DFDBA-2.4	HA-4.3 DFDBA-3.8	HA-2.9 DFDBA-2.1
Barnett et al. 1989	7 pts 19 HA defects compared to 19 FDBA defects—same mouth	FDBA vs. porous HA	6-12 months	HA-1.3 FDBA-2.1	HA-1.4 FDBA-3.0	HA-1.3 FDBA-2.2

*Mean values in millimeters
†Information not available

gain in hard tissue height (bone and/or ceramic) of 5.2 mm was found. No histology or re-entry was accomplished, controls were absent, and results were based largely on standardized radiographs.

Baldock et al. (1985) evaluated the use of TCP ceramic implant material in 13 osseous defects in 2 patients. Histological (at 3, 6, and 9 months), and clinical and radiographic evaluations (no controls) were used. Clinically, a mean gain of probing attachment level of 2.0 mm and a radiographic "fill" of 1.8 mm occurred. Histologically, TCP particles were encapsulated by fibrous connective tissue and failed to stimulate bone growth. New cementum was observed, but there was limited evidence of new attachment. Radiographic fill appeared to have occurred by mechanical obstruction rather than new bone growth. Minimal ingrowth of bone occurred and TCP was not totally resorbed at 9 months. In the absence of histologically substantiated bone formation or new connective tissue attachment, it was concluded that TCP had little beneficial effect on defect repair.

Bowers et al. (1986) histologically evaluated 4 TCP-treated sites from 1 patient. At 1 year post-implantation, bone and osteoid formation around TCP particles was observed. It was concluded that TCP might serve as a nidus for new bone formation in the intrabony defect. While resorption of the material may continue to occur over a period of years, active bone formation can occur supracrestally and in the soft tissue coronal to the defect after 1 year.

Stahl and Froum (1986) and Froum and Stahl (1987) also studied intraosseous healing histologically at 3 to 8 months and then 13 to 18 months after placement of TCP in 8 intrabony lesions in 4 patients. An average gain in clinical closure (clinical attachment gain) of 2.6 mm and 2.3 mm was observed respectively. Histologic evidence failed to indicate any osteogenesis, cementogenesis, or new connective tissue attachment. Instead, slowly resorbing TCP particles, acting as an inert fill material, became well encapsulated by gingival connective tissue. Active root resorption was seen immediately apical to the junctional epithelium at 1 site and wound closure was by a long junctional epithelium.

Saffar et al. (1990) evaluated 5 biopsies of defects implanted with TCP at 16 to 40 months after implantation. The findings suggested that TCP does promote bone formation as it slowly resorbs. At 1 year, the mean linear bone gain, as quantified on standardized radiographs, was 4 mm (80% fill). Both defect fill and resorption of TCP varied between individuals, taking as long as 40 months to occur in some instances. This may explain why previous short-term studies could not demonstrate bone formation and integration of the material after grafting.

Tricalcium Phosphate/Collagen Complexes as Implant Materials. Bell and Beirne (1988) have hypothesized that a composite TCP and collagen graft complex might serve as a biocompatible resorbable binder in periodontal defects. Sugaya et al. (1990) found that defects in dogs treated with a TCP-atelocollagen complex had a significantly higher suppression of epithelial downgrowth and a higher rate of new cementum and bone formation than controls.

Biphasic Calcium Phosphate (BCP). This material is a 2-phased calcium phosphate material composed of varying amounts of hydroxyapatite and TCP. Because of the TCP content, it is considered to be partially resorbable. Nery (1990A) found that BCP mixed with fibrillar collagen was superior to surgery alone in periodontal defects in dogs. Nery (1990B) further showed that in humans, use of this material without collagen was no more effective than autogenous bone or open flap curettage at 3 years. Nery et al. (1992) used varying concentrations of HA and β-TCP as implants in experimentally created periodontal defects in dogs and concluded that ratios of HA/β-TCP of 65/35 and 85/15 resulted in the greatest gains in attachment level. All BCP combinations improved attachment levels when compared to non-implanted controls.

Hydroxyapatite (HA)

HA is a non-resorbable ceramic material used as an implant material for the treatment of osseous periodontal de-

fects. Like TCP, this material has been the subject of extensive study, largely due to its close crystal and chemical resemblance to vertebrate tooth and bone mineral (calcium/phosphate ratio of 1.7, the same as that of bone mineral). HA is non-inflammatory, non-antigenic, and highly biocompatible to human tissues; it is commercially available in two basic forms, non-porous and porous.

Non-Porous Hydroxyapatite. Often generically referred to as durapatite, this material is an extremely dense, pure, non-resorbable ceramic material possessing great strength. Because of its physical qualities, and its similarities to human hard tissues, it has long been considered for use as a bone replacement material.

Animal Studies. Early studies showed HA was biocompatible in animals (Jarcho et al., 1977; Frame, 1981). HA particles become encapsulated by fibrous connective tissue and there is no evidence of new bone associated with the implant material.

Human Studies. In the first human study utilizing this material in periodontal defects (Rabalais et al., 1981), HA was implanted in 38 osseous defects and compared to 29 debrided controls in 8 patients. At the 6 month post-surgical re-entry, the HA was enmeshed in a soft connective tissue matrix. The implantation sites were hard, resisted probe penetration, and had significantly more defect fill (1.7 mm) versus controls (0.5 mm). Regardless of original probing depth, no difference between experimental and control sites was found with regard to clinical attachment gain, decrease in probing depth, or soft tissue recession. Only 16% of the HA implanted sites failed, compared to almost 50% of the controls. It was also noted that graft effectiveness appeared to increase with increased probing depth.

In a histological assessment (Froum et al., 1982), 4 sites in 4 different patients with osseous defects exceeding 4 mm in depth were examined at 2 to 8 months after placement of HA implants. The material served adequately as a foreign body fill, was well tolerated, and afforded no new attachment. Moskow and Lubarr (1983) histologically evaluated a single site implanted with both HA and autogenous bone chips. At 9 weeks, fibrous encapsulation of the HA particles was observed without any evidence of inflammation or extrusion. Although new bone was associated with the bone fragments, no indication of osseous changes adjacent to the HA particles was observed. Meffert et al. (1985) noted less crestal bone resorption with HA than surgical curettage after 9 months. The resistance of the implant material to probe penetration and its acceptance by hard and soft tissues suggested an ability to stabilize the remaining osseous structure. Shepard et al. (1986) observed new bone associated with the fibrous interstitium surrounding the HA crystals but could find no evidence of new attachment. Healing was by long junctional epithelium to the depth of the original defect. Ganeles et al. (1986) examined HA implant biopsy material and reported osseous regeneration in association with the HA granules in only 2 of 19 cases. Healing was generally by a long junctional epithelium.

In recent years the most extensive work using HA as an alloplastic implant material for treatment of periodontal defects has been by Yukna et al. (1984, 1985, 1986, 1989A, 1989B). These studies compared both short- and long-term (up to 5 years) response of HA-implanted periodontal osseous defects to defects in the same mouth treated with debridement alone. It is also important to consider the limitations of this work when evaluating the significance of the results. Most of the data were obtained from soft tissue measurements only; i.e., gingival recession, clinical attachment level, and probing depth. Direct assessment of long-term hard tissue changes (re-entry) was not accomplished in most cases and no histological evaluations were made. A summary of Yukna's findings indicates that the use of HA ceramic as a bone implant material in periodontal osseous defects yields at least as good and often better results than those following surgical defect debridement alone. The HA-treated sites were stable for a 5-year post-surgical period and showed clinical improvement, while flap debridement sites were not stable and regressed at a rate 3 to 5 times that of the implanted sites. Yukna (Yukna, 1989A) concluded that use of HA is clinically beneficial in most cases, provided it is used judiciously, only when indicated, and with realistic expectations; at present, however, these materials are little more than "clinically useful, biocompatible fillers that allow certain therapeutic goals to be reached and maintained."

Non-Porous HA Summary. Non-porous HA characteristics include: 1) non-porous HA is a hard, inert, highly biocompatible filler for osseous defects; 2) although it may help stabilize the remaining structure, it may stimulate formation of some new connective tissue attachment apparatus; healing generally is by long junctional epithelium rather than connective tissue attachment; 3) its crystals become surrounded with a fibrous encapsulation and may be associated with osseous regeneration; however, such new bone formation is not a predictable event, and 4) since its use produces similar and perhaps even better long-term clinical results than surgical debridement alone, it may be clinically beneficial.

Porous Hydroxyapatite. Porous HA is a ceramic implant material formed by hydrothermal conversion of the calcium carbonate exoskeleton of the Porites coral into hydroxyapatite. This conversion, known as the replamineform process, yields a material similar to the microstructure of natural bone. The interconnecting channels of this material are reportedly of sufficient size (190 to 230 μm) to support fibrovascular ingrowth and subsequent bone formation (White et al., 1972, 1975, 1986). Porous hydroxyapatite is currently being marketed and used as an implant material for intrabony periodontal defects . Available in both solid

and granular forms, both appear equally effective (Kenney, 1986A).

Animal Studies. The biocompatibility of porous HA has been adequately verified in animals (Piecuch, 1982; West and Brustein, 1985; Minegishi et al., 1988). Although bony ingrowth has been a consistent finding with porous HA, El Deeb et al. (1987) showed that this material is osteoconductive rather than osteoinductive; i.e., a scaffold for bone growth. When porous HA was implanted into surgically created periodontal defects in dogs, West and Brustein (1985) found evidence of fibrovascular tissue, bone, and periodontal ligament formation around the implant. In surgically created osseous defects in monkeys (Minegishi et al., 1988), the implanted porous HA granules were rapidly surrounded by fibrous connective tissue or new bone. At 1 year, the surgically created bone defects were almost completely repaired. The granules were integrated with new bone and the periodontal ligament reformed between the thin cementum and the new bone.

Human Studies. Kenney et al. (1985, 1986, 1987, 1988A, 1988B) have extensively investigated the use of porous HA graft material in periodontal defects. Porous HA was placed in angular intrabony periodontal defects in 25 patients (Kenney et al., 1985, 1987), while non-grafted matched defects in the same patients served as controls. Surgical re-entry at 6 and 12 months revealed greater reduction in probing depth and increase in attachment levels, as well as more bone fill in the implanted sites as compared to controls. No mobility of the grafts was noted and the surrounding bone appeared to be incorporated into the implant. The graft material and surrounding bone were similar in appearance. Similarly, superior clinical results have been obtained at 3 years after implantation (Frentzen et al., 1989). Defect fill consisting of a mixture of HA granules and regenerated bone was noted as early as 10 weeks.

When porous HA was placed in Class II furcation defects in mandibular molars and compared to grafted controls (Kenney et al., 1988B), bone fill of grafted defects was accompanied by improvement in probing attachment levels and probing depths. Lekovic et al. (1990) used porous HA in conjunction with polytetrafluoroethylene membranes (PTFE) to treat Class II furcation defects in lower molars. In each subject, 1 of 2 paired defects was implanted with porous HA and then covered with PTFE membrane, while the other defect was treated with debridement and PTFE only. At 6 months, both procedures resulted in similar reduction in probing depth, but the porous HA/PTFE sites resulted in less gingival recession and more horizontal and vertical defect fill.

Kenney et al. (1986) histologically examined samples of porous HA at 6 months following implantation into osseous periodontal defects. For the first time, evidence of this material's ability to stimulate osteogenesis within the porous structure of the implant was obtained. Since block sections were not obtained, it was not possible to determine if new connective tissue or cementum was formed. No evidence of resorption of the material was observed, indicating that biodegradation of this material occurs slowly, if at all.

Stahl and Froum (1987) evaluated porous HA clinically and histologically using block sections. At 1-year post-implantation in 12 human periodontal defects, acceptable tissue response with attachment gain and reduced probing depths was observed. Histological evaluation revealed bone formation in the implant pores, as well as peripherally; however, there was no evidence of new attachment and closure was by long junctional epithelium. Similar histological findings were obtained by Carranza et al. (1987) on 2 block sections at 5 and 6 months after implantation. Although 1 of the sections exhibited some new cementum formation opposite the implanted material, the conclusions were the same. Porous HA has the potential for bony ingrowth into the pores and ultimately within the lesion itself, but no new connective tissue attachment can be expected.

Porous HA Versus Bone Allografts. It, therefore, appears that use of porous HA may produce better clinical results in treating osseous defects than surgical debridement alone. But it is equally important for the clinician to know how this alloplastic material compares with biologically derived grafting materials, such as decalcified and non-decalcified freeze-dried bone allograft. Bowen et al. (1989) compared porous HA to decalcified freeze-dried bone allograft (DFDBA) in 6 patients having at least 2 comparable periodontal defects (total of 17 pairs of defects) respectively grafted with DFDBA and porous HA. Standardized radiographs and clinical measurements were taken after initial preparation and again at 6 months post-implantation. The 6-month re-entry revealed that although both treatment modalities reduced probing depth and demonstrated a gain in clinical attachment levels, there were no significant differences in any of the soft or hard tissue measurements (2.2 mm bone repair with DFDBA and 2.1 mm with HA). Although no histological evaluation substantiated the presence or absence of new attachment, the authors concluded that if regeneration of the periodontium is the desired goal, then DFDBA may be the proper choice. If defect "fill" is the objective, then, based on this study, DFDBA or porous HA might be equally effective.

In a study of similar design by Oreamuno et al. (1990), no apparent difference in healing patterns was noted between porous HA and DFDBA, but greater probing reduction and gain in attachment level were observed with the porous HA. Re-entry at 6 months revealed crestal bone loss with DFDBA and significantly greater defect fill with porous HA. Although the apparent advantage of the porous HA may be clinically insignificant, the relative efficacy of both materials was demonstrated.

Barnett et al. (1989) compared porous HA to non-decalcified freeze-dried bone allograft (FDBA). Nineteen (19)

matched pairs of intrabony defects in 7 patients were either implanted with porous HA or grafted with FDBA and re-entered 7 to 11 months later. Mean bone fill for FDBA was 2.1 mm, compared to 1.3 mm for porous HA. The mean decrease in probing depth was 3.0 mm and 1.3 mm, respectively, although recession accounted for more decreased probing depth in the FDBA than porous HA. Attachment gain for the FDBA was 2.2 mm, compared to 1.3 mm for the porous HA. Osseous repair of at least 50% was observed in 74% of the FDBA grafted defects and 42% of the porous HA implanted ones. The FDBA treated sites could be debrided to a solid surface, but the porous HA sites often retained fibrotic tissue at the base of the defect. Histologic analysis of curetted graft material revealed little osteogenic activity associated with either material. The authors concluded that little difference exists in repair potential between porous HA and FDBA.

Porous HA in Combination with Other Biologically Derived Materials. Minabe et al. (1988) compared porous HA alone to porous HA combined with collagen in the treatment of periodontal defects in dogs. Findings indicated that the addition of the collagen resulted in an increased amount of new cementum formation. While no such porous HA/collagen implant systems are currently available for human use, further research may be warranted.

Porous HA Summary. Porous HA characteristics include: 1) porous HA is biocompatible with human tissues and can generally be expected to result in greater improvement in probing depth, attachment level, and depth of the osseous lesion, than non-implanted surgically treated defects; 2) although porous HA provides for bony ingrowth into its pores and ultimately within the lesion itself, there is no evidence that it stimulates new attachment formation; closure is by long junctional epithelium; 3) similar clinical results might be expected with the use of porous HA, as compared to bone allografts, in the fill and subsequent healing of osseous periodontal defects; however, true regeneration is more likely with bone allograft materials (Table 2).

TABLE 2. CURRENTLY AVAILABLE CERAMIC ALLOPLASTIC IMPLANT MATERIALS

Property/Trade Name	Material	Comment
Resorbable		
Synthograft	Tricalcium phosphate	Identical to Synthograft
Peri-Oss	Tricalcium phosphate	Ridge augmentation material
Augmen	Tricalcium phosphate	May not be truly resorbable
Osteogen	Hydroxyapatite	
Partially resorbable		
Triosit	Biphasic calcium Phosphate (BCP)	A combination of HA and TCP
Porous non-resorbable		
Interpore 200	Replamineform hydroxyapatite	Available in block or granules
Permagraft	Replamineform hydroxyapatite	Similar to Interpore 200
Periograf	Hydroxyapatite	
Calcitite 2040	Hydroxyapatite	Ridge augmentation material
Calcitite 4060	Hydroxyapatite	
Non-porous non-resorbable		
Alveograf	Hydroxyapatite	Ridge augmentation material
Orthomatrix HA-500	Hydroxyapatite	
Orthomatrix HA-1000	Hydroxyapatite	Ridge augmentation material
Bioglass	Glass	
Cervital	Glass	European version of Bioglass

SUMMARY

It is extremely difficult to form definitive conclusions as to the relative efficacy of alloplastic implant materials in the management of periodontal defects. Considerable variation exists in the limited number of clinical human trials which have been conducted using these materials with respect to use of controls, evaluation techniques, re-entry procedures, and histological data. Consequently, direct comparison of results can easily lead to invalid conclusions, unless variations in study design are considered. Bearing this in mind, the following overall observations may be made:

1. Although all currently available alloplastic implant materials appear to be biologically compatible, non-antigenic, non-inflammatory, and otherwise safe, use of the patient's own bone remains the best choice for treating periodontal defects. This assumes that harvesting procedures are feasible and an adequate donor source is present.

2. Use of alloplastic implant materials can be expected to produce at least equal long-term (5 year) clinical results, compared to surgical debridement alone.

3. Currently available alloplastic implant materials might be considered a reasonable alternative to bone allograft material, especially if moral/religious reservations, fear of cross contamination, or immunologic responses to allograft material exist. Clinical closure and defect fill is generally similar in both biological and synthetic materials but regeneration is far more likely with bone allograft.

4. Although new bone formation may be seen in association with some alloplastic implant materials, there is no evidence that any are predictably osteoinductive or able to stimulate the formation of a new connective tissue attachment apparatus.

5. No currently available alloplastic implant material is clearly superior to any other in treating periodontal defects. All are inert osteoconductive fill materials which serve as a nidus or scaffold for new bone formation. Their use generally results in defect fill, stabilization of the remaining

osseous structure, clinical attachment gain, and decreased probing depths. Healing is characterized by one or a combination of the following: fibrous encapsulation of the implant granules, bony ingrowth into the material, slow resorption of the implant, and/or formation of a long junctional epithelium and/or connective tissue adhesion without regeneration of the periodontium.

6. Most documented success and biocompatibility have been obtained with the calcium phosphate materials, i.e., tricalcium phosphate and hydroxyapatite. These materials chemically resemble natural human hard tissues, are radiopaque, sterilizable, easy to obtain, and stable.

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Section 10. Guided Tissue Regeneration

DEFINITIONS

Repair: Healing of a wound by tissue that does not fully restore the architecture or function of the part.

Regeneration: Reproduction or reconstitution of a lost or injured part.

New Attachment: The union of connective tissue or epithelium with root surface that has been deprived of its original attachment apparatus. This new attachment may be epithelial adhesion and/or connective adaptation or attachment and may include new cementum.

Reattachment: To attach again. The reunion of epithelial and connective tissues with root surfaces and bone such as occurs after an incision or injury. Not to be confused with new attachment.

Guided Tissue Regeneration (GTR): Procedures attempting to regenerate lost periodontal structures through differential tissue responses. Barrier techniques, using materials such as expanded-polytetrafluoroethylene, polyglactin, polylactic acid, and collagen, are employed in the hope of excluding epithelium and the gingival corium from the

root surface in the belief that they interfere with regeneration.

Periodontal Regeneration: Restoration of lost periodontium.

Potential Healing Responses of the Periodontal Tissues

Melcher (1976) first presented the basic concepts which have led to the development of the clinical techniques collectively known as GTR. He suggested that there were 4 separate compartments of connective tissue (CT) in the periodontium: the gingival corium, periodontal ligament (PDL), cementum, and bone. Melcher felt that the CT cells in each of these compartments represented different cellular phenotypes capable of repopulation and determining the regenerative response obtained. Based on this concept, he hypothesized that PDL regeneration can only come from the PDL itself. This idea of distinctive progenitor cells in the periodontal ligament has been supported by Aukhil et al. (1986A), who described in vivo differentiation of progenitor cells from the PDL. In this investigation, fenestration wounds were made in buccal cortical plates of the mandibular anterior teeth in 6 beagle dogs. Exposed root surfaces were curetted to remove all cementum and either demineralized with citric acid or washed with saline (contralateral wound). Nucleopore membranes were attached with cyanoacrylate resin to prevent progenitor cell contact with root dentin leaving 0.5 mm denuded root exposed around the membrane border. Histologic analysis of the specimens after 3 months of healing revealed no new cementum over the membrane area attached to root dentin, whereas, at wound borders and in areas where the membrane had detached, new cementum with CT attachment was seen. No root resorption was seen in any of the specimens. These findings suggested that progenitor cells from the PDL may differentiate into cementoblasts upon contacting root dentin, thereby substantiating the potential inductive effects of dentin on cementum.

Additional investigations by Isidor et al. (1986) examined the effects on new attachment formation on preventing coronal growth of PDL tissues during healing. This work provided a model system for exclusion techniques whereby elastic ligatures were utilized to both prevent and allow coronal growth of granulation tissue from the PDL depending upon the tautness of ligature placement. Four primates were utilized and histologic examination of 3-month healing results analyzed. Results indicated that new attachment failed to occur when the coronal growth of PDL tissue was prevented, thereby reinforcing the need for PDL cell repopulation of the root surface for new attachment formation. Apparent prevention of root resorption by epithelial migration during healing was also noted.

A more recent investigation by Iglhaut et al. (1988) studied PDL and bone cellular kinetics following surgical wounding. A total of 4 primates received fenestration wounds, and PDL and cementum removal followed by Millipore filter placement. Both histologic and autoradiographic examination of specimens from 1 hour to 21 days revealed cellular migration into wounded sites at 3 days from both bone and PDL. Results suggested that both bone and PDL contribute cells for wound repopulation when the epithelium and flap CT are excluded from contact with the root during healing. In lieu of these investigations, Melcher (1987) has amended his original hypothesis of isolated compartments of CT in the periodontium to include the influence of bone, thereby disavowing the PDL as a closed compartment.

Investigations of Guided Tissue Regeneration

The healing of periodontal wounds has been confounded by the propensity for rapid epithelial proliferation and resultant mediation of the tooth-soft tissue interface to the base of the defect. Furthermore, changes in the structural morphology of the periodontium, secondary to the destructive effects of chronic periodontitis, have resulted in changes in the spatial relationships of selective CT compartments available for wound healing. Efforts to compensate and/or correct these problems form the conceptual basis for guided tissue regeneration.

Epithelial Exclusion. Ellegaard et al. (1974) attempted to retard the apical migration of the epithelium by using free gingival grafts (FGG) over intrabony defects. This technique included surgical preparation of the intrabony defect, FGG recipient bed preparation, and placement of an autogenous bone graft into the defect. An FGG was then placed over the bone grafted defect to delay apical migration of the epithelium and facilitate maturation of the granulation tissue adjacent to the root surface. Although the term "regeneration" was used to describe the results of this technique, no histologic evidence was provided to validate this claim. Only wide interproximal spaces and areas neighboring edentulous spaces qualify for this technique.

Prichard (1977) was undoubtedly a pioneer of the epithelial exclusion concept with his introduction of the interdental denudation procedure. This technique included excision of all soft tissue in the interproximal region, leaving bare interalveolar bone. Prichard (1983) alluded to the use of epithelial exclusion in the management of vertical bony defects. He discussed a 7-step plan for managing 3-walled intrabony defects. The plan included the following (the first 4 steps being deemed essential for success, with the last 3 of varying importance):

1) Removal of the gingiva to the margins of the bony walls of the defect, thereby leaving the defect open to prevent epithelial migration. (Failure to leave the defect open was thought to be the most common cause of failure.)

2) Removal of the transseptal and alveolar crest fibers of the PDL, as well as granulomatous tissue from the defect.

3) Removal of all calculus but as little cementum as possible.

4) Surgical dressing must not be allowed to enter the defect.

5) An antibiotic (preferably tetracycline) should be prescribed.

6) An occlusal adjustment should be completed if necessary.

7) No presurgical scaling should be done on the root in the intrabony defect.

Becker et al. (1986A) studied changes in intrabony defects following open flap debridement using the Prichard technique. Thirteen (13) patients were treated as described by Prichard (1983) and placed on 3 to 4 month maintenance intervals. Surgical re-entry was completed at 9 to 16 months, during which time repeat measurements and direct defect impressions were obtained. Volumetric analysis of pre- and post-treatment defect size was made on casts by filling defects with gunpowder and comparing the weight differences between residual defects. Volumetric analysis revealed that 50% (7/14) of the defects had a $\geq$ 50% decrease in defect volume. The mean percentage fill was 47.5%. Additionally, Becker et al. (1986B) reported findings from the treatment of 36 intrabony defects in 35 patients using open debridement procedures alone. Scaling and root planing was not completed since the inflamed lesion was thought to have the necessary cell population for repair. Flaps were apically positioned in an attempt to leave the defects open. Patients were maintained on 3 to 4 month recall and surgical re-entry was completed at 14 months postsurgery. Results indicated a mean gain in clinical attachment level of 2.44 mm and a reduction in defect depth by a combination of crestal resorption (mean 0.48 mm) and defect repair (mean 2.55 mm). Results of the Becker et al. (1986A, B) studies are very similar to those reported in other osseous grafting studies regarding percent of defect fill.

Animal Studies. Magnusson et al. (1985) studied the effects of Millipore filters on exclusion of gingival CT and epithelium during wound healing in a primate model. Pore size was varied (0.2 and 5.0 μm) at test sites, while control sites received no filters. Specimens were assessed histologically at 6 months postoperatively. A new fibrous attachment on 50% of the denuded root surface was achieved. This compared favorably to root coverage achieved by Gottlow et al. (1984) utilizing a submerged wound model. Results of these studies imply that the formation of new attachment may be related to isolation and guiding of specific cell populations.

Magnusson et al. (1988) evaluated the use of biodegradable membranes in surgically created defects in 2 mongrel dogs. Bone removal to approximately 25% of its original height on selected teeth in each dog. Eight defects were covered with Millipore filters, 8 with biodegradable membrane (polylactic acid), and 8 controls with no membranes. Results indicated new CT attachment to 46% of the planed cementum within the surgical defects treated with the polylactic acid/biodegradable membranes, with an average of 2.1 mm of coronal bone regrowth. Millipore filter sites displayed new attachment to only 25% of the planed surface with an average 1.7 mm of new bone growth, while control teeth showed new attachment to 12% of the root surface with only 0.8 mm of new bone growth.

Pfeifer et al. (1989) evaluated the effectiveness of resorbable collagen membranes (from bovine dermis) in producing new attachment in surgically created defects in 4 beagle dogs. Both cross-linked and non-cross-linked collagen membranes were placed. Healing was evaluated histologically at 1, 3, 4, and 8 weeks postsurgery. Results indicated that the cross-linked membrane could persist for 6 to 8 weeks and was preferred over the non-cross-linked membrane (which was totally resorbed in approximately 3 weeks). The cross-linked membrane was effective in inhibiting epithelial downgrowth and in producing tissue regeneration. A second surgical procedure was not required for membrane removal.

Seibert and Nyman (1990) conducted a pilot study evaluating GTR on bucco-lingual ridge defects (13 $\times$ 7 mm, created 3 months earlier) as a means to provide ridge augmentation in 2 beagle dogs. On upper quadrants, porous hydroxyapatite (HA) or tissue growth matrix (TGM) implant materials were placed with and without expanded polytetrafluorothylene (ePTFE) membrane. On the lower arch, 3 of the quadrants received ePTFE membrane with no implant, while the other quadrant served as a sham-operated control. Histologic analysis was completed 55 to 90 days following ridge augmentation. Results indicated complete bone fill 90 days following ridge augmentation, in the membrane only and membrane with HA sites. The quadrant with TGM also displayed new bone within the pores adjacent to the existing bone surfaces; however, the pores adjacent to the soft tissue surface were filled with non-mineralized CT. Flap separation or dehiscence proved to be a major cause of surgical failure. This study failed to provide data regarding volumetric fill of existing defects, thus making comparisons of results to other studies difficult.

Pontoriero et al. (1992) attempted to evaluate the critical factors in grafting Class III furcation defects using ePTFE membranes. In the beagle model, they created Class III furcation defects of different sizes in an apico-coronal direction and treated these defects with debridement and ePTFE membrane placement. After 4 months of healing, the sites were evaluated histologically. In the small defects, the bone regenerated to its original height. In the larger defects not only was there little defect fill, but significant soft tissue recession also occurred, exposing the furcation area. Overall, if the defect was $>$ 3 mm in an apico-coronal dimension, gingival recession was more apt to occur with subsequent failure to develop complete new attachment.

Human Studies. Much of the data regarding GTR procedures in humans is based on case reports. In a series of case reports, Gottlow et al. (1986) described new attachment formation in the human periodontium by GTR. A total of 12 teeth in 10 patients were treated with teflon membranes and replaced flaps. Histologic data were evaluated 3 months post-membrane removal revealing 2.8 to 4.5 mm of new attachment in the membrane treated sites versus no new attachment in control sites. Defect morphology (horizontal versus vertical bone loss [best prognosis]) was viewed as a key factor in predictability of regenerative procedures, reinforcing the concept of space dependent cell migration for regeneration.

Becker et al. (1987) described a surgical and suturing method for the subgingival placement of ePTFE as well as the results of 3 treated cases, one of which was biopsied at 3 months. Evidence of clinical and histologic new attachment secondary to ePTFE use was shown. The new tissue observed at re-entry was termed "open probing new attachment" if it did not have the consistency of bone. A simultaneous suturing technique of the ePTFE and the flap was described as a means to maintain the subgingival position of the material. The authors suggested that ePTFE be removed at 4 to 6 weeks post-insertion.

Stahl et al. (1990) presented human clinical and histologic data from GTR in intrabony lesions. The authors used different ePTFE membranes. New attachment was seen with both types of teflon membranes as early as 5 weeks postsurgery. The topography of the bony lesion was considered as a key controlling factor determining regeneration.

Numerous authors have suggested the use of GTR procedures in the treatment of furcation defects. In 2 controlled human clinical trials, similar results with membrane exclusion techniques were obtained. Pontoriero et al. (1987) reported closure in 19 of 21 Class II furcation defects using ePTFE membrane over the furcation entrance (note that 5 of 19 had 1 mm less than complete fill). This was in contrast to surgically debrided control sites in which only 2 of 21 (< 20%) closures occurred. In Class III furcation defects, 4 of 16 exhibited complete closure; 9 of 16 partial closure; and 3 remained as through and through lesions. None of the 16 control sites exhibited complete closure. In a similar study, Pontoriero et al. (1988) reported that in 90% of mandibular molar sites with Class II furcation involvement treated with ePTFE membranes, clinical evidence of complete closure was present at 6 months. However, less than 20% of the control defects demonstrated closure following treatment. Histologic evidence of regeneration was not included in the 2 preceding controlled clinical trials.

In non-controlled human clinical studies, Becker et al. (1988) reported results in 27 patients following placement of ePTFE membranes. The authors noted that the consistency of the tissue in the defects at the time of re-entry was firm, rubbery, and resistant to the forces of probing. They felt that this material was not bone and noted no radiographic changes in affected areas. This tissue which resisted the forces of probing was termed "open probing clinical attachment." There was a mean gain in clinical attachment of 1.3 mm for Class III furcations, 2.3 mm for Class II furcations, and 4.5 mm for 3-walled intrabony defects. Caffesse et al. (1990) reported results in which GTR was used to treat Class II furcation defects in mandibular molars. Clinically there was a mean gain in clinical attachment level of 1.8 mm for GTR (ePTFE) treated sites versus 0.6 mm for control sites (sham-operated). No furcations demonstrated complete closure. Comparisons of results from this case report to the previous work published by Becker et al. (1988) and Pontoriero et al. (1987, 1988) may be difficult due to differences in probing techniques and maintenance regimens. Becker et al. (1988) used only 1 measurement in the middle of the furcation (away from the root surfaces) while Caffesse et al. (1990) recorded probing depth and clinical attachment levels at the furcal aspects of the mesial and distal roots. Pontoriero et al. (1987, 1988) appointed subjects for professional tooth cleaning every 2 weeks, while Caffesse et al. (1990) placed patients on a 3-month recall. Many of these patients had received long-term maintenance therapy, suggesting the "chronic condition" of the treated furcation sites (Caffesse et al., 1990).

Gantes et al. (1988) treated 30 mandibular, buccal Class II furcation defects with a regenerative technique utilizing citric acid root conditioning and coronally positioned flaps secured by crown-attached sutures. In addition, decalcified freeze-dried allogeneic bone grafts were placed in 16 of the 30 defects. Twelve months following therapy, an average of 67% of the defects volume was filled with bone, while 43% of treated defects were completely closed by bone fill. No significant differences were observed between defects treated with and without bone grafts.

Another treatment approach for furcal defects was reported by Schallhorn and McClain (1988). The authors utilized a combined approach of ePTFE membrane, citric acid root conditioning, and composite osseous grafting (autogenous material mixed with either tricalcium phosphate or demineralized freeze-dried bone allograft material) in 95 defects in 39 patients. They reported complete fill in 33 of 46 (72%) furcation defects using the combined approach versus 5 of 16 (31%) furcation defects when membranes alone were used. (Success was measured at the membrane removal appointment.) In a companion study, McClain and Schallhorn (1993) assessed the long-term results of these cases, reporting after 5 years that GTR with composite grafting and root conditioning enhanced complete furcation fill in Class II and III defects. It also led to a 5 year stability of gains in clinical probing attachment levels, and in furcation fill.

Lekovic et al. (1990) reported the use of porous hydroxyapatite (HA) in conjunction with ePTFE membranes

in the treatment of Class II furcation defects. Mean clinical attachment level gains were 2.40 + 0.48 mm for ePTFE only sites and 2.93 + 0.64 mm for ePTFE + HA site. While their results were encouraging, they were less than the probing depth reduction and clinical attachment gain reported by Pontoriero et al. (1987, 1988). The 2-week professional prophylaxis regimen utilized by Pontoriero and coworkers may account for these differences.

Blumenthal and Steinberg (1990) offered another GTR approach, combining demineralized bone-collagen gel (autolysed antigen extracted allogeneic bone and microfibrillar collagen/Zyderm) implants with collagen membrane barriers. At 1 year re-entry, 93% of all intrabony defects treated had 50% or greater fill. The use of non-standardized measurements and patient selection criteria raised questions regarding the accuracy of these results. Blumenthal (1993) also compared the use of collagen membranes with e-PTFE membranes in paired Class II mandibular buccal furcations over 1 year in 12 patients. Both modalities resulted in similar clinical improvements, and both were effective in gaining vertical open probing new attachment and both were effective for horizontal defect fill. Paul (1992) and Van Swol et al. (1993) also demonstrated regeneration in Class II furcations using collagen membranes.

Chung et al. (1990) evaluated a highly cross-linked, bioresorbable, type I collagen membrane in GTR in 10 patients. Mean gains in probing attachment level of 0.56 + 0.57 mm (range: −0.3 to 1.6 mm) and bone defect fill of 1.16 + 0.95 mm (range: −0.2 to 2.4 mm) were observed. These results were likely minimized by the reporting mean values and inclusion of healthy sites in the data.

Lekovic et al. (1991) reported the use of connective tissue grafts including periosteum to cover the furcation opening in 15 Class II furcations. The controls were debrided only with the tissue being replaced back to its original height. Six month re-entry measurements revealed a probing depth of 4.67 for controls and 2.33 for the connective tissue sites. The control group had no gain in mean attachment level, whereas the experimental group responded with 2.40 mm. The horizontal measurements did not change for the control group, but improved by a mean of 1.60 mm for the experimental group. The authors conclude that their results are very similar to ePTFE membrane results.

Handelsman et al. (1991) evaluated the effect of root conditioning with citric acid (3 minute application, pH = 1) prior to using ePTFE membrane material in intrabony defects. Based on clinical measurements taken during a surgical re-entry procedure, no significant differences were found between the control and the citric acid group. Overall, 72% of the sites showed 50% defect fill.

Anderegg et al. (1991) evaluated decalcified freeze-dried bone allograft (DFDBA) in combination with ePTFE membranes in Class II and Class III mandibular molar furcations compared to the membrane alone. Based on re-entry surgery at 6 months to examine hard tissue changes, DFDBA sites showed statistically greater improvement in horizontal and vertical bone repair compared to controls. Horizontal improvement was noted in all 27 Class II furcations, with 4 completely filled and 13 (10 experimental and 3 control) having at least 2 mm of horizontal bone fill. Overall, the DFDBA sites with membranes had an 85% decrease in osseous defect depth compared to a 50% decrease with the membrane alone. Generally the deeper the vertical component of the defect, the greater the osseous fill.

Metzler et al. (1991) compared open flap debridement with open flap debridement and an ePTFE membrane in maxillary Class II furcation defects. The authors judged success by surgical re-entry at 6 months and measured the hard tissue changes in a horizontal and vertical dimension. These measurements were referred to as HOPA (horizontal open probing attachment) and VOPA (vertical open probing attachment). These terms should be distinguished from "open probing clinical attachment" (Becker et al., 1987) which describes not hard, but "rubbery" tissue at membrane removal, usually 4 to 6 weeks.

At 6 months a surgical re-entry procedure was performed to assess the hard tissue changes. No significant differences were observed between the 2 sites for recession, probing depth reduction, attachment level changes, or alveolar crest resorption. There was a significant gain in VOPA (1.5 mm versus 0.6 mm) and HOPA (0.9 mm versus 0.3 mm) for the membrane group over the open flap debridement alone group.

Gantes et al. (1991) reported the treatment of Class III mandibular molar furcation sites using citric acid and coronally positioned flaps in the control group, and DFDBA, citric acid, and coronally positioned flaps in the experimental group. Comparison of the 2 techniques was evaluated by closed clinical measurements. From a total of 27 Class III furcation sites (14 control, 13 DFDBA), complete soft tissue closure was noted in 1 non-grafted site and 3 grafted furcations. The addition of DFDBA did not enhance defect closure compared to coronally positioning the flaps alone. Restoration of Class III furcations was not a predictable procedure. In an observational study of maxillary and mandibular molar teeth extracted for periodontal reasons in a Chinese population, Lu (1992) evaluated the contours present in the area (2 mm below the CEJ) where an ePTFE membrane would be placed during surgery. His data indicated that membrane placement in this area was likely to have a significant gap between the stretched membrane and the tooth surface, and may contribute to failure. The author suggested that supragingival placement of the membrane's coronal margin may be the solution to maintaining epithelial exclusion.

Yukna (1992) compared the use of freeze-dried dura mater allografts to ePTFE membranes in Class II mandibular molar furcations. He reported equal results from the 2 techniques, based on surgical re-entry. However, ePTFE did not give results comparable to other studies using this

material. In no case was there complete closure of the Class II defect with either material. He also noted that the improvement in open probing attachment levels seen at the time of membrane removal was lost over the intervening months, more so for the vertical than the horizontal component.

Gottlow et al. (1992) evaluated the long-term stability of initial clinical gains (bleeding on probing, probing depth, clinical attachment level) observed 6 months post-surgically compared to measurements obtained over 1 to 5 years. Eighty (80) of 88 sites had gained ≥ 2 mm CAL, and 60 of the 80 sites had gained ≥ 3 mm at 6 months. Over 5 years the number of sites followed decreased from 80 to 9. The stability of the sites showed that the clinical gains at 6 months had been maintained with 93% present at 1 year, 92% at 2 years, 90% at year 3, and 100% at years 4 and 5. Contrary to Yukna (1992), the authors concluded that the improvement in clinical parameters can be maintained over 5 years in a majority of cases.

Other applications for the use of GTR have been shown by Pini Prato et al. (1992) who used ePTFE membranes for coverage of previously restored root surfaces. Also, Andregg and Metzler (1993) used ePTFE membranes to treat palato-gingival grooves in 10 patients with good clinical results.

MATERIALS

Free Gingival Grafts (FGG) Overlying Autogenous Bone Grafts

Using FGG over 88 intraoral (sites/tuberosity) bone grafts, Ellegaard et al. (1974) showed complete regeneration (based on clinical and radiographic measures) in about 60% of sites with 10% exhibiting > 3 mm residual pockets compared with 40% to 60% in controls.

Nucleopore/Millipore Filters

These are non-resorbable and require a second procedure for removal (Magnusson et al., 1985; Aukhil et al., 1986A; Iglhaut et al., 1988).

Teflon/ePTFE Membranes

These are also non-resorbable and require a second procedure for removal. These membranes are made of expanded polytetrafluoroethylene (ePTFE) and are biocompatible. They consist of 2 parts: 1) an open microstructure collar to inhibit epithelial migration and 2) an occlusive apron that isolates the root surface from the surrounding tissues (Gottlow et al., 1986; Becker et al., 1987, 1988; Pontoriero et al., 1987, 1988; Schallhorn and McClain, 1988; Stahl et al., 1990; Caffesse et al., 1990; Lekovic et al., 1990; Seibert and Nyman, 1990).

Biobrane Membrane

This is a non-resorbable membrane which requires a second procedure for removal. It is a biocomposite consisting of an ultrathin, semi-permeable silicone membrane mechanically bonded to a flexible knitted nylon fabric and coated with hydrophilic collagen peptides. It is primarily a biological dressing that is used in the treatment of skin burns (Aukhil et al., 1986B).

Polylactic Acid Membranes

These are biodegradable and degradation is primarily by hydrolysis so a second procedure for removal is not required (Magnusson et al., 1988). The commercially available membrane consists of a polymer of polyactic acid which has been softened by a citric acid ester to facilitate handling. The matrix barrier consists of an external and internal layer separated by an interspace designed to allow gingival comnnective integration while excluding epithlium

TABLE 1. SUMMARY OF GUIDED TISSUE REGENERATION STUDIES (modified from Minabe, 1991)

Study	Animal Model/ Number	Experimental Sites	Experimental Model/Defect	Membrane Type/ Pore Size	Observation Period	Treatment Effects		
I. Animal Studies/GTR/Nondegradable Barrier (Closed Type Model)								
Gottlow (1984)	Monkey/3	Premolar/ molar	Dehiscence/ experimental periodontitis	Teflon/ Gore-Tex	3 months	New attachment	Exp.	Control
						0-59%	3	5
						60-99%	2	1
						100%	4	0
						mean:	77% (0.9-6.8 mm)	33% (0-2.5 mm)
II. Animal Studies/GTR/Nonabsorbable Barrier								
Magnusson (1985)	Monkey/6	Premolar/ molar	Wide dehiscence	Millipore Filter/0.25 μm	6 months		Exp.	Control
						New attachment	54% (2.9 mm)	2% (0.1 mm)
						New bone	20% (1.1 mm)	0% (0 mm)
Aukhil (1986)	Beagle dog/6	Premolar	Horizontal/ natural pedoni	Biobrane	4 months	CT attachment	(0.72 mm)	
						New attachment	(0.51 mm)	

TABLE 1. Continued

Study	Animal Model/ Number	Experimental Sites	Experimental Model/Defect	Membrane Type/ Pore Size	Observation Period	Treatment Effects
III. Animal Studies/GTR/Absorbable Barrier						
Magnusson (1988)	Mongrel dog/2	Premolar	Dehiscence	Millipore filter polylactic acid/ (70 μm thick)	2 months	Poly / Milli / Control New attachment 46% / 25% / 12% 2.5 / 1.4 / 0.7 mm New bone 2.1 / 1.7 / 0.8 mm
Pfeifer (1980)	Beagle dog/4	Molar	Furcal defects/ experimentally created	Collagen (bovine dermal) crosslinked/ non-crosslink	1,3,4,8 weeks	Cross-linked membrane persisted 6-8 weeks; non-cross-linked resorbed in approximately 3 weeks.
Siebert (1990) Pilot Study	Beagle dog/2	Max/man arches	B-L ridge defects (13 × 7 mm) surgically created	Interpore 200 (PHA)/tissue growth matrix (TGM w/& w/o Gore-Tex)	55-90 days	No data for volumetric fill; complete bone fill in GTM only and GTM + PHA sites; some bone in TGM
IV. Clinical Studies/GTR/Nonabsorbable Barrier						
Gottlow (1986) Case reports	Human/10	Cuspid/molar	Class II & III furcations/flat surface + angular bony defect	Teflon/Gore-Tex	3 months/ 6 months	New attachment 40% Probing attachment Gain 5.6 mm
Pontoriero (1987)	Human/37	Molar	Class II & III furcations	Teflon/Gore-Tex	6 months	Completely Closed Sites Class II 67%/exp 10%/con Class III 25%/exp 0%/con
Becker (1987) Case reports	Human/3	Cuspid/molar	Case 1: Class III Case 2: 1 wall Case 3: 2 wall	Teflon/Gore-Tex	3 months/ 6 months	Probing Attachment Gain Case 1: 4 mm Case 2: 2-4 mm Case 3: 4 mm
Pontoriero (1988)	Human/21	Molar	Class II furcations	Teflon/Gore-Tex	6 months	Probing Attachment Gain Vert: 4.1 mm/exp 1.5 mm/con Horz: 4.1 mm/exp 1.9 mm/con
Becker (1988)	Human/27	Premolar/ molar	Class II & III furcations; 3 wall defects	Teflon/Gore-Tex	6 months (membrane removed after 6 weeks)	Probing Attachment Gain Class II: 2.3 mm Class III: 1.3 mm 3 wall: 4.5 mm
Schallhorn (1988)	Human/39	Molar	Class II & III furcations; dehiscence/ horizontal/ wide intrabony defects	Teflon/Gore-Tex; citric acid composite grafts	Variable/up to 6 months	Complete fill: combined approach, 72% of furcas; membranes only, 31% of furcas
Caffesse (1990) Case report	Human/9	Molar	Class II furcations	Teflon/ Gore-Tex	6 months	Clinical Attachment Level Gain 1.8 mm/exp GTM 0.6 mm/con no GTM
Lekovic (1990)	Human/15	Molar	Class II furcations	Teflon/ Gore-Tex; PHA grafts	6 months	Clinical Attachment Level Gain (mean values) 2.40 ± 0.48 mm for GTM only 2.93 ± 0.64 mm for GTM + PHA

TABLE 1. Continued

Study	Animal Model/ Number	Experimental Sites	Experimental Model/Defect	Membrane Type/ Pore Size	Observation Period	Treatment Effects
Stahl (1990)	Human/5	Molar/ nonmolar	Intrabony defects	Teflon/Gore-Tex; Emflon	5-8, 14, 22, 30, weeks	Clinical and histological assessment. New attachment seen as early as 5 weeks with both teflon membranes.
Handelsman (1991)	Human/ 16 pts, 18 defects	All teeth	Intrabony defects, est. 4 mm deep	Citric acid 3 min vs. no citric acid, Gore-Tex mem. on all	Clin meas. 6 months, re-entry at 9 months	No differences between citric acid and no citric acid. 60% of original defect fill. 16 sites had > 2 mm bone fill, 10 sites > 3.3 mm fill. 72% of sites showed > 50% defect fill
Anderegg (1991)	Human/15 pts, 30 defects	Max/man molars	Class II or III paired furcation defects	DFDBA vs. no DFDBA, Gore-Tex on all	Clin and surg re-entry at 6 months	No difference for attachment levels, Exper. sites had greater reduction in probing depth 1.7 mm vs. 1.4 mm for control. 4 sites completely closed, HOPA exp: 2.4 mm increase, control: 1.0 mm increase w/o DFDBA Conclusion: combined therapy better.
Metzler (1991)	Human/17	Max molars, 12 pair facial 5 pr. interprox.	Class II furcation	Open flap debride vs. debride and Gore-Tex	6 month re-entry	No difference for recession, probing depth, clinical attachment gain; results unpredictable. Conclusion: Gore-Tex offers little advantage in max Class II furcations.
V. Clinical Studies/GTR/Absorbable Barrier						
Blumenthal (1990)	Human/10	Molar/ nonmolar	Intrabony defects 1,2,3 wall & combination	Collagen gel/ Collagen membrane autolysed/ antigen-extracted allogeneic bone grafts	12 months	93% of all defects had 50% or greater fill
Chung (1990)	Human/10	Molar/nonmolar	Intrabony defects	Perio-Barrier/ cross-linked Type 1 collagen membrane	12 months	Probing Attachment Level Gain (mean values) 0.56 mm/exp −0.71 mm/con bone defect fill (mean values) 1./16 mm/exp 0.00 mm/con
Yukna (1991)	Human/11	11 pairs Man molars	Class II furcas bilateral defects	Freeze-dried dura mater allografts vs. Gore-Tex membrane	12 month re-entry surgery	Equal results for FDDMA and Gore-Tex membrane. More loss of keratinized tissue with Gore-Tex. Defect fill 1.0 mm, Decreased probing depth, Gore-Tex = 0 mm, FDDMA = 1 mm. % Horiz. fill: Gore-Tex = 20%, FDDMA = 40%.
VI. Miscellaneous						
Ellegaard (1974)	Human	N/A	Intrabony defects	Free gingival grafts/ Autogenous bone grafts	N/A	Complete regeneration in 60% of defects; 10% with residual pockets > 3 mm
Gantes (1988)	Human/22	Molars	Class II furcations	Citric acid/ DFD allogenic bone grafts/ coronally positioned flaps—crown attached sutures	12 months	67% defect fill (average) 43% of defects with complete closure with bone fill. No difference with and without bone grafts

with eventual CT integration with the PDL tissue (Gottow et al., 1994).

Collagen

These membranes are biodegradable and do not require a second procedure for removal. Effectiveness depends on the specific collagen type. Cross-linking (chemically with glutaraldehyde) prolongs existence. The material is chemotactic for PDL fibroblasts; a barrier for migrating epithelial cells; hemostatic; fibrillar scaffold for early vascular and tissue ingrowth, and exhibits varying degrees of immunogenicity. Forms of collagen include: membranes, gels, atelocollagen (telopeptides pepsinized making it less antigenic), and avitene (microfibrillar collagen hemostat from purified bovine corium collagen) (Pfeifer et al., 1989; Blumenthal and Steinberg, 1990; Chung et al., 1990).

Freeze-Dried Dura Mater Allografts

This is a resorbable human allograft material that is composed mainly of collagen and is devoid of immunogenicity (Yukna, 1992). While some risk of disease transmission exists, it can be reduced by the lyophilization and sterilization processes used. One documented case of Creutzfeldt-Jakob disease (a fatal CNS degenerative disease of viral etiology) has been transferred from 1 patient to another with a fresh dura mater allograft (non-dental graft) (Thorn et al., 1991).

Salonen and Persson (1990) analyzed the growth and migration of gingival epithelial cells on materials with different surface properties. A total of 125 explants were prepared from specimens of attached gingiva, placed on Millipore-HA filter, Biopore, or ePTFE material, and then histologically examined after culture periods of 4, 6, and 8 days. Results indicated that greater cell migration occurred on the Millipore-HA filter than on the Biopore or ePTFE material. Differences in epithelial cell attachment and migration to varying substrata may be explained by their ability to bind glycoproteins to varying degrees. Millipore-HA consists of mixed esters of cellulose and therefore has a higher protein-binding capacity than either Biopore or ePTFE material. Scanning electron microscopic examination of these materials disclosed surface differences. The Millipore-HA had a non-homologous surface while the others appeared smooth. Overall, these data suggested that the substrata not only provided contact guidance which influenced cell migration by the shape of the substratum surface, but also induced mitosis and migration because of their protein-binding capacity and wettability characteristics (Table 1).

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Section 11. Growth Factors

DEFINITIONS

Growth Factors: A diverse group of polypeptides which have important roles in regulation of growth and development of a variety of organs.

Fibroblast Growth Factor: A family of growth factors with mitogenic properties for fibroblasts and mesoderm-derived cell types.

Platelet-Derived Growth Factor (PDGF): A glycoprotein carried in the α-granules of platelets and released during blood clotting; a potent growth factor for cells of mesenchymal origin, including fibroblasts and smooth muscle cells.

Transforming Growth Factor (TGF): A family of growth factors involved in the regulation of cell growth and differentiation.

Cytokines: A broad family of humoral factors that mediate considerable roles in growth, differentiation, and tissue damage by cellular receptors.

Lymphokine: Soluble factors released from lymphocytes that transmit signals for growth and differentiations of various cell types.

FACTOR OVERVIEW: Wirthlin (1989)

Bone Morphogenetic Protein (BMP) and Osteogenin

BMP and osteogenin are an acid-resistant group of glycoproteins with osteoinductive properties. At least 7 BMPs have been isolated from bovine and human sources including BMP-1, BMP-2 (BMP-2A, OP-2), BMP-3 (osteogenin), BMP-4 (BMP-2B), BMP-5, BMP-6 (Vgr-1), and BMP-7 (OP-1). BMP-2 through BMP-7 have been shown by sequence similarity to be members of the TGF-β superfamily of molecules, while the amino acid sequence of osteogenin matches that of BMP-3. BMP-6 has been shown to be the human homologue of the murine protein Vgr-1, and the proteins OP-1 and OP-2 are identical to BMP-7 and BMP-2, respectively. Purification isolation has yielded 2 μg of BMP per 10 kg of bovine bone.

The implantation of BMP-incorporated bone matrix has resulted in migration and proliferation of mesenchymal cells, differentiation into chondroblasts and chondrocytes, calcification of cartilage matrix, vascularization, resorption of calcified cartilage, and formation and mineralization of new bone matrix. This process appears similar to normal embryonic endochondral bone formation.

Osteoinductive factor (OIF) is a unique bone protein reported to induce the formation of ectopic cartilage and bone

when implanted in combination with TGF-βI or TGF-βII, but has no similarity of amino acid sequence with BMPs or other known growth factors.

Epidermal Growth Factor (EGF)

EGF is a small, heat-stable, single-chain polypeptide of 6,000 daltons best known for its ability to stimulate keratinization and epidermal growth. Cellular growth is the major result of EGF stimulation of a target cell, with EGF serving as a progression factor inducing competent cells to proceed with division. EGF sources include urine, saliva, Brunner's glands, blood, the central nervous system, and amniotic fluid. EGF binds to a surface receptor, the receptor-ligand complex is internalized, followed by the initiation of a tyrosine kinase cascade leading to the complex phosphorylation and increase in free intracellular Ca2+. The ligand-receptor complex internalization is followed by marked changes in cellular morphology, including rapid growth and division. Cells typically dedifferentiate during rapid growth and division until the influence of EGF is removed, at which time differentiation of the collagen producing fibroblasts resumes.

Monocyte-Derived Growth Factors (MDGF)

MDGF is a 40,000 dalton growth factor synthesized by macrophages and which acts on fibroblasts, smooth muscle cells, endothelial cells, and other mesenchymal cells. Secretion is enhanced if the macrophage is activated by lipopolysaccharide, concanavalin A, fibronectin, or phorbol esters.

Platelet-Derived Growth Factors (PDGF)

PDGF is a family of polypeptide growth factors consisting of a 2-chain polypeptide linked by disulfide bonds with a molecular mass ranging from 27,000 to 30,000 daltons. PDGF is derived from platelets, osteoblasts, activated macrophages, and some tumor cell lines of mesenchymal origin. It is found in serum and has been isolated from bone matrix. PDGF is known to stimulate bone DNA and protein synthesis as well as bone resorption. It serves as a powerful chemoattractant for smooth muscle cells, fibroblasts, and leukocytes and has major mitogenic effects in serum that are dependent upon the presence of other growth factors.

PDGF is stored in the alpha granules of platelets and is extravasated after injury and hemorrhage. Platelet activation and degranulation follow platelet exposure to thrombin or fibrillar collagen. Subsequently, PDGF directly recruits and activates neutrophils and monocytes, possibly in a concentration-gradient fashion. After initiation of this inflammatory phase of wound healing, it serves to activate mesenchymal cells essential to the proliferative phase, including endothelial cells and smooth muscle cells. In the remodeling phases of wound healing, PDGF stimulates secretion of the collagenase and extracellular matrix by associated fibroblasts.

Tumor Necrosis Factor α (TNF-α)

TNF-α is a typical cytokine with a wide range of cell regulatory, immune, and inflammatory properties. TNF-α also interacts and overlaps with other members of the cytokine network, inducing, enhancing, or inhibiting their action. It can stimulate the growth of various diploid fibroblasts and some tumor cell lines. TNF-α's mitogenic action is synergistic with epidermal growth factor (EGF), platelet-derived growth factor (PDGF), and insulin.

TNF-α is also cytotoxic for some cells independent of protein synthesis. Although the basis for susceptibility/resistance of cells to the cytotoxic action of TNF-α is unknown, it may be dependent on other cytokines or growth factors. TNF-α may be involved in the cytoxic action of lymphocytes and natural killer cells. TNF-α may also contribute to the host defense system by inducing a cellular antiviral state. It can protect cells from infection by both DNA and RNA viruses, but only a narrow spectrum of cells.

TNF-α is a powerful neutrophil activator which promotes adherence to endothelial cells or particulate matter, stimulating phagocytosis, respiratory burst activity, and degranulation. TNF-α also has immunoregulatory activity on T-cells and targets vascular endothelial cells promoting coagulation, inflammation, and immunity. It inhibits the activity of thrombomodulin, augments the secretion of inhibitors of plasminogen activators, and induces the synthesis and transient cell surface expression of tissue factor procoagulant activity. TNF-α greatly increases the expression of endothelial leukocyte adhesion molecules and Class I MHC antigens, and can alter the morphology of endothelial cells in vitro, changing their typical "cobblestone" to a more elongated form.

TNF-α's bone resorption potential is comparable to that of parathyroid hormone. It will also stimulate chondrocytes to degrade proteoglycans and will elicit the secretion of proteolytic enzymes, such as collagenase, from synovial cells and fibroblasts surrounding bone and cartilage. There is also evidence that sustained and/or systemic TNF-α production may contribute to the signs and symptoms of disease, including endotoxic shock and acute phase responses in infection and injury.

Tumor Necrosis Factor-β (TNF-β)

TNF-β is a lymphotoxin secreted by mitogen-stimulated lymphocytes with cytotoxic and cytolytic properties. Its biological activities are similar to those of TNF-α, stimulating the growth of non-transformed cells. TNF-β also stimulates bone resorption and bone cell replication.

Insulin-Like Growth Factor (IGF-I, also Somatomedin C and IGF-II)

IGF-I is a single chain polypeptide hormone weighing 7,500 daltons. It is synthesized and secreted in precursor form and is activated by proteolytic cleavage. IGF-I has a 47% homology with insulin, with 3 similar sulfide bridge

cross-links. Growth hormone stimulates the production of IGF-I by hepatic cells, fibroblasts, and fetal rat bone organ cultures. IGF-I stimulates cartilage growth, bone matrix formation, replication of pre-osteoblasts, and osteoblasts. It may also directly stimulate the cell which produces it (autocrine). IGF-I significantly increases alkaline phosphatase activity in osteoblastic cells, suggesting a role in stimulating cell differentiation. Both IGF-I and insulin are capable of similar effects reflecting cross-reactivity at the IGF-I and insulin receptors.

IGF-I transcripts have been reported in macrophages isolated from wounds suggesting IGF-I acts as a local messenger (paracrine factor) between cells in the wound environment. IGF-I alone has minimal effects on wound healing but in combination with PDGF can enhance the rate and quality of wound healing. Poorly controlled diabetics have increased levels of growth hormone but decreased levels of IGF-I, which may impact wound healing.

Fibroblast Growth Factor (FGF)

FGF is one of a group of similar growth factors which share an ability to bind heparin. They induce angiogenesis and mitogenesis of endothelial cells. Two subtypes, acidic fibroblast growth factor (aFGF), and basic fibroblast growth factor (bFGF) have been recognized. Each appears to be derived from a single gene with differences in the multiple similar growth factors within the same class resulting from post-translational processing. The mechanism of action of FGF is largely unknown, but it appears to be released from granules, where it may be stored in a preformed state.

Transforming Growth Factor-β (TGF-β)

TGF-β is a 2-chain polypeptide which is linked together by disulfide bonds. It has a molecular weight of 25,000 daltons. It exists as 3 different gene products: TGF-1, TGF-2, and TGF-3. TGF-β was originally identified because it could induce non-transforming cells to grow in soft agar, which is usually associated with anchorage-independent growth in malignant cells. TGF-1 is found in high concentrations in bone and in the alpha granules of platelets. It is secreted in a latent form by macrophages and activated in conditions such as the low pH of wound healing and bone resorptive environments. It is also present in high concentrations in endochondral growth plates, and at lower levels in diaphyses, epiphyses, and calvaria.

Generally, TGF-β has an inhibitory effect on cells of epithelial origin and usually stimulates cells of mesenchymal origin. TGF-β modulates extracellular matrix production and at the same time suppresses the matrix destruction by decreasing the amount of plasmin and collagenase produced. It may act by altering the cellular response to other growth factors, either at the receptor or postreceptor level. TGF-β can induce the expression of other growth factors, and stimulate the production of PDGF from mesenchymal cells.

Transforming Growth Factor-α (TGF-α)

TGF-α is a peptide with a molecular weight of 5,700 that acts synergistically to induce anchorage-independent growth of non-transformed NRK cells in vitro. It is not related to TGF-β, but is related to EGF and competes for the same receptors. The activation of macrophages may result in TGF-α gene expression and production. It is 42% homologous with EGF and stimulates fibroblasts, epithelial cells, and endothelial cells by binding to the EFG receptors, which mainly stimulate epithelium.

Literature Review

Terranova et al. (1987) evaluated new in vitro assay systems to test the potential chemotactic and proliferative activity of various biological response modifiers to dentin. One assay, using human periodontal ligament cells (PDL), was for specific cell migration (AFSCM) to dentin (either preconditioned with tetracycline [TCN] or unconditioned) treated with fibronectin (FN) and laminin (LM). Another assay, using epithelial cells, evaluated the ability of dentin-bound chemoattractants to stimulate directed movement and proliferation on TCN preconditioned dentin surfaces. These surfaces were incubated with 100 μg of FN, washed and then varying concentrations of endothelial cell growth factor (ECGF) and LM were added. Migration distances were determined by photographs quantifying the stained leading front cells. The authors concluded that assays for specific cell migration were useful in selecting potential biological response modifiers capable of promoting healing at the dentin-soft tissue interface.

Terranova and Wikesjö (1987) reviewed extracellular matrices and polypeptide growth factors as mediators of function of periodontium cells. Fibroblasts exist in a fibrous matrix composed of types I and III collagen, chondroitin sulfate proteoglycan, and fibronectin. Fibronectin binds fibroblasts to the matrix, and also binds to many different collagen types, heparin sulfate, fibrin, and other glycoproteins. Chondrocyte matrix contains type II collagen, chondroitin sulfate proteoglycan, link protein, hyaluronic acid, and chondronectin, which binds chondrocytes to the matrix. Chondronectin also attaches to proteoglycan, collagen, and cell surface receptors. Epithelial cells abut on a matrix of basement membrane containing type IV collagen, and a large heparin sulfate proteoglycan and laminin. Heparin sulfate forms a charged barrier in the basement membrane which prevents passage of proteins, while laminin constitutes 30% to 50% of the total protein in basement membranes.

Fibroblasts, chondrocytes, and epithelial cells produce and use different cell specific attachment factors. Laminin will not support fibroblast attachment, and fibronectin will not support epithelial cell attachment, and may be protective mechanisms. Endothelial cells and hepatocytes can use both fibronectin and laminin for attachment in vitro, and these growth factors can affect the phenotype of the cells.

Polypeptide growth factors (PGF) are released continuously to diffuse to target cells. They may represent a large family of regulatory factors, contributing significantly to wound healing. PGFs, neural elements, proximal contacts with heterologous and homologous cells, and interactions with extracellular matrix material all contribute to stimulation and control of cell growth. PGFs also may contribute to evolving chemotactic responses in targeted cells.

Connective Tissue Attachment Regeneration

Citric acid (CA) conditioning of instrumented root surfaces may contribute to new attachment in animals and humans, with animal models exhibiting better results. CA leads to partial surface demineralization and exposed collagen fibrils. Semi-porous membranes beneath gingival tissues have been used to exclude epithelial cell migration along the root surface, allowing fibroblast proliferation. Whereas epithelial cells normally migrate 10 times faster than fibroblasts, CA treatment of root surfaces followed by exogenous fibronectin application reverses this pattern. Application of exogenous extracellular matrix material and appropriate PGFs to prepared root surfaces may confer a selective advantage to gingival fibroblasts, osteoblasts, and PDL cells.

Sporn and Roberts (1988) completed a review of the multi-functionality of peptide growth factors. Peptides are now known to have a wider range of action than originally thought, with a broader cell response to them than previously believed. Interleukins were first defined as signalling molecules which controlled activities of cells of the immune system. IL-2, originally T-cell growth factor, is now known to be an important factor for B-cells and to increase the cytotoxic activity of monocytes. IL-4, originally B-cell growth factor, has been shown to be a potent stimulant of T-lymphocytes, and to control the maturation of granulocytes and macrophages. IL-1, first described as a substance produced by macrophages which acted on lymphocytes, also is a potent stimulator of epidermal keratinocytes, chondrocytes, and fibroblasts. Peptides originally isolated from non-immune cells and defined by their actions on non-immune cells now have been found to have potent effects on the immune system. In a single cell type, growth-factor action may change according to other substances that are present. TGF-β stimulates growth of certain fibroblasts in vitro in the presence of platelet-derived growth factors, but inhibits their growth if epidermal growth factor is present. The cellular mechanisms responsible for the multiple actions of a peptide ligand are unknown. A possible mechanism is the ability of a receptor for a specific peptide to alter either the cellular distribution or the binding affinity of the receptor for a second peptide growth factor, which is independent of any direct cross-reactivity of the peptides themselves. Also, receptor molecules can be multi-functional, having separate binding sites for two distinct ligands in the same receptor.

Sporn and Roberts concluded that growth factors form part of a complex cellular signalling language, in which individual peptides are the equivalent of characters in an alphabet or code. The information resides in the pattern, or set of regulatory peptide molecules, to which a cell is exposed.

Canalis et al. (1988) evaluated the role of growth factors and the regulation of bone remodeling. Growth factors act primarily as local regulators of cell growth and have important effects on cell replication and differentiation. Effects include increasing osteoblast populations capable of synthesizing bone matrix, stimulating cell replication and differentiated function, or affecting the availability of a factor, and the binding to its receptor. Three categories of growth factors include those synthesized by skeletal muscles, those isolated from bone matrix, and those synthesized by cells from adjoining tissues. Growth factors synthesized by skeletal cells include TGF, bone-derived growth factors (BDGF), IGF, and PDGF. BDGF is found in serum or on the surface of most mammalian cells and stimulates bone collagen and DNA synthesis. BDGF (2m) and molecules of the major histocompatibility complex interact with the receptors for hormones and growth factors and may modulate binding of other growth factors or hormones to their receptor; therefore, it may not be a growth factor in the classic sense. Growth factors isolated from bone matrix include TGF-β, 2m, IGF-1, PDGF, acidic fibroblast growth factor (aFGF), and basic fibroblast growth factor (bFGF); aFGF and bFGF are members of a family of polypeptides that include endothelial cell growth factor (ECGF). They are synthesized by multiple tissues and affect endothelial cell replication and neo-vascularization, and stimulate DNA synthesis and cell replication. They result in an increased bone cell population capable of synthesizing collagen and non-collagen protein. They have no direct stimulatory effect on cell function, and sometimes directly inhibit osteoblastic function. They are available only after cell injury or death, and have no effect on bone resorption. Growth factors synthesized by cells from adjoining tissues include cartilage and blood cell-derived factors. Somatomedin and bFGF-like factors have been isolated from cartilage. Blood-derived factors include monokines and lymphokines.

Lynch et al. (1989A) reported preliminary findings of studies using a combination of PDGF and IGF-1 to enhance the regeneration of periodontal structures. Using beagle dogs with 30 to 80% naturally occurring periodontal disease, 5 teeth received growth factors while 7 teeth served as controls. Following flap surgery and root planing, test teeth received 75 μl of an inert gel containing 1 μg each of purified human PDGF and recombinant IGF-1, while control teeth received the gel only. Two weeks post-surgery, block sections were taken and processed for histometric analysis. Control sites had a long junctional epithelium with no new cementum, while growth factor treated sites exhibited significant amounts of new cementum and

bone. The experimental sites averaged 926 μm of crestal new bone apposition, while the control sites averaged 71 μm. These preliminary results suggest that in vivo application of a combination of PDGF and IGF-1 may enhance periodontal regeneration.

In a study of skin wound healing in pigs where several growth factors were used, Lynch et al. (1989B) found that only the combination of recombinant PDGF-2 plus recombinant IGF-1 produced a dramatic increase in connective tissue regeneration and epithelialization in the absence of increased inflammation relative to the control. The optimal ratio for synergism between these 2 factors was 2:1 (by weight) respectively. Other factors evaluated included epidermal growth factor, TGF-α, TGF-β, and fibroblast growth factor. TGF-α was able to substitute for IGF-1 by acting synergistically with PDGF to promote collagen synthesis and fibroblast proliferation, resulting in a variable increase in epithelial thickness. TGF-β had the greatest individual effect, causing a significant increase in collagen synthesis and fibroblast proliferation. It also enhanced inflammation, abnormal epithelial differentiation, and decreased epithelial volume.

Early wound healing events of bone around press-fit titanium implants inserted with and without a combination of PDGF-β and IGF-1 were evaluated by Lynch et al. (1991A). Two female beagle dogs with edentulous quadrants received 2.0 mm × 6.0 mm sand-blasted titanium implants with 2 transverse 1.0 mm diameter holes in their apical sections. Twenty-four (24) implants were coated with methylcellulose gel containing 4 μg of both recombinant PDGF-β and recombinant IGF-1, while 8 implants received only the gel and 8 were untreated controls. Five quadrants with 20 implants were harvested at 7 and 21 days. At 7 days, the PDGF-β and IGF-1 treated sites had significantly increased percentages of bone-fill in contact with implant surfaces. At 21 days, the treated sites had significantly increased percentages of bone-fill in the peri-implant spaces.

Seyedin (1989) reported on the discovery and research associated with bone morphogenetic proteins (BMP). BMP are osteoinductive substances retained in a collagenous bone matrix and are responsible for the cascade of events leading to bone formation, which includes chemotaxis, proliferation, angiogenesis, bone formation, and differentiation. TGF-α and TGF-β in combination with osteoinductive factor (OIF) can induce massive amounts of ectopic endochondral bone development.

Graves and Cochran (1990) reviewed mesenchymal cell growth factors identifying them as multifunctional, affecting mitogenic activity, cell migration, and differentiation. Paracrine factors are those produced by 1 cell to stimulate another cell, while autocrine factors are those produced by a cell to stimulate itself. Competence growth factors are those that stimulate resting cells in G0 to enter the cell cycle at a point in G1 and enter the S phase. Progression factors are those needed to move the cell from G1 to S efficiently.

Lynch et al. (1991B) evaluated the short-term application of PDGF-β and IGF-1 as a means of enhancing periodontal regeneration. Using beagle dogs with naturally occurring periodontal disease with standardized osseous defects, clearance studies were completed. Teeth received either 10 ng of radiolabeled PDGF-β, 10 ng of radiolabeled IGF-1, or 3 μg of unlabeled recombinant-derived PDGF-β and IGF-1 in a methylcellulose gel carrier. The mean half-life was 3.0 hours for IGF-1 and 4.2 hours for PDGF-β. The clearance slowed after 10 hours but approximately 7% remained after 48 hours, and less than 4% remained after 96 hours, while no detectable label was found at 2 weeks. In vivo studies on the dogs evaluated periodontal wound healing, utilizing the application of a combination of 3 μg of recombinant PDGF-β and 3 μg of IGF-1 in a methylcellulose gel to the roots of diseased premolars. Contralateral roots received the gel alone, with sacrifice of the animals taking place at 2 and 5 weeks, followed by the completion of histologic evaluations. In the PDGF-β/IGF-1 treated sites, there was significantly increased bone height and area, and length of new cementum compared to controls at both 2 and 5 weeks. Mean height and area of new bone at 2 weeks were 0.96 mm and 1.57 mm^2, respectively, at growth-factor treated sites compared to 0.27 mm and 0.07 mm^2 for controls. The bone continued to increase in both height and area from 2 to 5 weeks. At 2 weeks, the length of new cementum was 0.04 mm and 0.82 mm for the control and growth-factor treated sites respectively. The mean percent defect fill was 6.9% in control sites and 40.6% in growth-factor treated sites. By 2 weeks in the growth-factor treated sites, osteoblast-like cells were present on the bone surface and surrounded by new bone matrix. At 5 weeks, numerous mitotic figures were present within the osteoblast-like cells. A normal PDL space was seen between new bone and cementum and there was no difference in ankylosis between the two groups.

Six dogs in the wound healing study received about 10 mCi of 99m-technetium MDP immediately prior to periodontal surgery, and at 2 and 4 weeks following surgery. Alveolar bone uptake around the teeth was compared to nuchal crest bone in each animal. The experimental sites exhibited bone-forming activity which was 2.0 times that of the controls at 2 weeks and 2.7 times the controls at 4 weeks.

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Section 12: Sutures—Materials and Methods

SUTURE MATERIALS

Types of Sutures

Properties of the ideal suture material should include handling ease, minimal tissue reaction, strength, and knot security. The material should also be absorbable, non-allergenic, non-electrolytic, non-carcinogenic, and withstand sterilization. Levin (1980) described the relative strengths and weaknesses of available suture materials as follows:

Braided Sutures. These (cotton, linen, polyester, and silk) are more pliable and flexible than monofilaments and have better knot security. Disadvantages include the tendency to collect bacteria, a "sawing" effect when pulled through tissue and fragmenting within the tissue (especially silk). This results histologically in a greater inflammatory reaction, though severe reactions are rarely seen within the first 10 days.

Monofilament Sutures. These sutures (steel, nylon, polypropylene) are generally stronger, more durable, and create less tissue reaction. Unfortunately, they are also more difficult to handle and have inferior knot-holding properties.

Absorbable Sutures. These materials are plain and chromic gut, plain and chromic collagen, polyglycolic acid, and polyglactin. Plain gut suture is probably the best choice when difficult or inconvenient removal is anticipated. It is more difficult to tie than silk, has inferior knot-holding properties, and forms a hard knot which may irritate tissues. Resorption usually occurs within 5 to 7 days as a result of slow hydrolysis. Chromic gut suture has chromic salts deposited on the outer surface or within the entire strand, providing greater resistance to absorption. It is difficult to handle and tie and does not rapidly resorb. Collagen, polyglycolic acid, and polyglactin sutures generally have less value in periodontal procedures due to delayed healing and handling difficulties (Table 1).

Silk (protein fiber) is the most commonly used multifilament suture material; this is attributed to its handling properties, strength, and minimal tissue irritation at 5 to 7 days (Meyer and Antonini, 1989A and 1989B). While surgical silk and nylon (ethilon) are generally considered to be non-absorbable suture materials, they, in fact, undergo very slow absorption. In response to local information, silk is completely absorbed in 1 to 2 years while nylon (amide polymer) is absorbed via slow hydrolysis.

Non-Absorbable Sutures. These include cotton, steel, polyester (mersilene, dacron), ethibond, propylene (prolene, surgilene), polyethylene and polybutester (elastic). These materials are primarily used in conjunction with general and vascular surgery (Meyer and Antonini, 1989A and 1989B).

TISSUE REACTION TO SUTURE MATERIALS

Lilly (1968) compared the reactions of oral tissues to several suture materials. Nine 4–0 suture materials were placed in the buccal mucosa and tongues of adult mongrel dogs which were sacrificed in groups of 2 at 1, 2, 3, 4, 6, 8, and 10 days post-placement. Block sections were processed for light microscopy and tissue reactions graded as mild, moderate, or severe. Steel and nylon (monofilament) initiated the least tissue reaction. Plain gut created a mild-to-moderate reaction (suture was not present at 8 or 10 days) and braided materials (polyester, dermal, cotton, and silk) resulted in similar reactions (29% to 65% judged severe at 8 to 10 days). Tissue reaction to linen was the most severe of any material studied. The author suggests that the more severe tissue reaction to the braided (multifilament) materials is due to a "wicking" action that may transmit bacteria and fluids to the depths of the wound.

Rivera-Hildago et al. 1991 compared the reactions of the oral tissues in dogs to teflon sutures versus silk sutures at 1, 3, 5, and 7 days. The inflammatory infiltrate increased from day 1 through day 7 for both materials with the infiltrate close to the sutures appearing to be comparable, while the infiltrate some distance from the lumen was more intense with silk. Overall, silk induced a greater inflammatory response than the teflon.

SUTURE NEEDLES/WOUND CLOSURE

Suture needles are made from stainless steel wire and have distinctive shape, size, point, and method of suture attachment. Basic shapes include straight, three-eighths circle, half circle, and five-eighths circle. The immediate needle return from the tissue when using a curved needle is an advantage, and the half circle is easier to use in confined locations. The needle diameter should match the suture size to minimize tissue damage. Needle points may be tapered or cutting, the latter being more useful for thick resistant tissues. Conventional cutting needles are triangular in cross-section with a cutting edge on the inside of the curve. There

Table 1. EXAMPLES OF ABSORBABLE SUTURES

Suture	Material	Absorption	Strength
Plain gut	Submucosa sheep intestine; serosa beef intestine	Body enzymes/macrophages; complete at 70 d	4 to 10 d
Chromic gut	Chromic salts + gut	Complete at 90 d	10 to 14 d
Polyglactin	Co-polymer of glycolide and lactide	Slow hydrolysis; complete at 60 to 90 d	20 to 30 d
Dexon	Homopolymer of glycolic acid	Slow hydrolysis Complete at 60 to 120 d	14 to 21 d

is a greater potential for severing a wound edge with this design. The reverse cutting needle has the third cutting edge on the outside of the needle curvature, minimizing tissue laceration. Sutures may be threaded through the needle or attached in the non-cutting end (swagged). Swagged needles are more expensive, but less time-consuming, and cause less tissue damage on penetration. The most popular needle is the three-eighths circle with a reverse cutting point (Meyer and Antonini, 1989).

Cyanoacrylates were introduced for dental (wound closure) purposes in 1965. The butyl and isobutyl forms of cyanoacrylate are the most acceptable in the oral cavity; the methyl form (super glue) is toxic to tissues. Cyanoacrylates are capable of cementing living wet tissues and are exfoliated in 4 to 7 days. McGraw et al. (1979) reported biometric and histometric results following a cyanoacrylate flap fixation in monkeys. Clinically, cyanoacrylate reduced flap fixation time by 10 to 15 minutes per quadrant, providing firm fixation of the conservative flaps used in this study. However, it may not adequately secure flaps reflected past the MGJ or provide the strength needed to resist muscle pull. The authors suggest a combination of cyanoacrylate and sutures in such instances. The authors concluded that the use of cyanoacrylate appeared to have no effect on probing depth, recession, or attachment level. Levin (1980) reported no adverse tissue reaction to this material in a review of 872 periodontal procedures in 725 patients. McGraw and Caffesse (1978) reported no evidence of cyanoacrylate under the tissue flaps, and there appeared to be less inflammation in the early stages of healing when compared to sutures. The authors caution that because cyanoacrylate is irritating to respiratory and ocular tissues, extreme caution must be employed during its use. This material is not currently approved by the FDA for intra-oral use.

Human fibrin seal (HFS) contains fibrinogen, aprotinin, calcium chloride, fibronectin, fibrin, Factor XIII, thrombin, and platelet-derived growth factor. A dog study by Pini Prato et al. (1984) compared HFS to conventional silk sutures in the closure of periodontal flaps. Partial thickness flaps were reflected in 2 quadrants at healthy sites and immediately replaced and fixed with silk suture or HFS. Sutures were removed on the eighth postoperative day; block sections were obtained at 2 hours, and at 1, 3, 7, and 14 days. The authors state that flaps secured with HFS were more firmly adapted at 2 hours than the sutured flaps. Specimens from day 3 demonstrated absence of inflammation at the HFS side while the sutured side exhibited inflammation up to 7 days. At 14 days, the healing response was equal on both sides. In a subsequent study (Pini Prato et al., 1987), HFS was compared with sutures in humans receiving pedicle or free gingival grafts and modified Widman flap surgery. The authors reported more rapid tissue stabilization (30 seconds versus 4 to 5 minutes) and less hemorrhage at HFS sites. No differences among respective sites were observed at 14 to 22 days. Concerns include preparation time, expense, and "human" blood product derivation.

SUTURE TECHNIQUES

Dahlberg (1969) discussed 4 basic considerations of suturing: 1) use the smallest, least reactive material; 2) leave a minimum amount of suture under the flap; 3) maintain the suture close to the tissue; and 4) remove the suture as soon as possible (5 to 7 days). Regarding methodology, the interrupted suture should be used when tissue positioning is not a problem. The sling suture is used to position the flap at different levels around individual teeth. The continuous sling saves time in placement and the anchor suture is useful in positioning a single papilla. Finally, the vertical mattress suture is used when it is desirable to avoid suture placement beneath flap margins (e.g., osseous graft sites).

A study by Nelson et al. (1977) compared continuous sling and interrupted sutures for primary closure of mucoperiosteal flaps in 10 patients. Despite the authors' impression that interrupted sutures provided better flap adaptation, there was no difference between the 2 techniques with regard to attachment loss or recession.

Newell and Brunsvold (1985) described a vertical mattress suture for esthetic purposes in anterior regions of the mouth. If used with the "curtain procedure," a vertical mattress suture allows the palatal flap to adapt tightly to the underlying bone while retaining the facial papilla in its original position. According to the authors, long thin papillae are best treated with vertical mattress sutures while short wide papillae are best treated with horizontal mattress sutures.

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Section 13: Periodontal Dressings

DEFINITION

Periodontal Dressing (Pack): A protective material applied over the wound created by periodontal surgical procedures.

RATIONALE FOR USE

Reasons for using periodontal dressing include: 1) protect the surgical site from trauma; 2) enhance patient comfort by covering exposed bone and connective tissue; 3) keep debris out of the wound (does not prevent plaque formation); 4) can position and/or stabilize flaps and soft-tissue grafts; 5) prevent the proliferation of excess granulation tissue and exercise caution to avoid pushing the dressing in an apical direction resulting in an interproximal soft-tissue crater); 6) help retain osseous graft materials; and 7) aid in controlling post-operative bleeding in patients with coagulation disorders (Levin, 1980; Sachs et al., 1984).

PHYSICAL PROPERTIES

The original Ward's wonderpak consisted of zinc oxide-eugenol (ZOE) mixed with alcohol, pine oil, and asbestos fibers. Zinc oxide-eugenol dressings generally contain between 40 and 50% free-eugenol, which has been shown to cause tissue necrosis and delayed healing. ZOE dressings were popular due to their obtundent effect on sensitive dentin and connective tissue. Non-eugenol dressings may contain zinc oxide, various oils or fats, rosin, and bacteriostatic or fungicidal agents. ZOE dressings set with a hard, brittle consistency while non-eugenol dressings are more flexible. Tannic acid was originally added to dressings to facilitate hemostasis but has since been removed because of its associative potential for liver damage.

Barricaid is a visible light-cured periodontal dressing composed of polyetherurethane dimethacrylate resin, silanated silica, photo-initiator and accelerator, stabilizer, and colorant. The material is tinted pink, translucent, and tasteless and is packaged as a single component in a disposable syringe. It is highly viscous, easily positioned, and may be effectively handled following lubrication. The soft tissue and tooth surfaces should be dry to facilitate mechanical interlocking and adequate anchorage. While the cured material has adequate rigidity, it maintains enough flexibility to facilitate removal.

Cyanoacrylate has the advantage of eliminating sutures, providing immediate hemostasis, biodegradability, precise positioning of flaps, and protective barrier function. Delayed healing may occur if the material becomes embedded in tissue or beneath the flap. Despite short-term benefits, cyanoacrylate dressings apparently offer little advantage to long-term wound healing. Cyanoacrylate is not FDA approved for oral use.

All dressings are irritating to some degree with eugenol more irritating than non-eugenol dressings. Cell culture studies have demonstrated that cytotoxic components are found in all dressings. Cyanoacrylate appears better tolerated by tissues than conventional dressings and may speed early wound healing. This may be due to the reduction in plaque and debris accumulation when compared to conventional materials. The antimicrobial properties of dressings are of little therapeutic importance and potential drawbacks include sensitization, allergy, fungal overgrowth, and development of resistant strains (Levin, 1980; Sachs et al., 1984).

DRESSING VERSUS NO DRESSING

Dressings were used routinely in the past, especially during the era of gingivectomies and pushback procedures. With the advent of flap procedures and emphasis on post-surgical flap adaptation, dressing use has decreased. Jones and Cassingham (1979) compared healing following periodontal surgery (apically positioned flaps) with and without non-eugenol dressings in humans. Assessments of biopsies (inflammation), gingival crevicular fluid, gingival indices, and sulcular measurements revealed no difference between dressed and non-dressed sites. Postoperative pain was more severe with dressings. The authors imply no useful purpose of dressings following flap surgery. Using comparable assessments, Allen and Caffesse (1983) reported similar observations when comparing results following modified Widman surgery with and without surgical dressings. Sixty percent (60%) of patients preferred no dressing. Dressings contribute to plaque retention and may promote bacterial proliferation at the surgical site. It appears that the primary

concern should be flap adaptation, rather than the placement of a dressing.

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Section 14: Wound Healing

DEFINITIONS

Repair: Healing of a wound by tissue that does not fully restore the architecture or the function of the part.

Reattachment: To attach again. The reunion of epithelial and connective tissues with root surfaces and bone such as occurs after an incision or injury. Not to be confused with new attachment.

New Attachment: The union of connective tissue or epithelium with a root surface that has been deprived of its original attachment apparatus. This new attachment may be epithelial adhesion and/or connective tissue adaptation or attachment and may include new cementum.

Regeneration: Reproduction or reconstitution of a lost or injured part.

Guided Tissue Regeneration: Procedures attempting to regenerate lost periodontal structures through differential tissue responses. Barrier techniques, using materials such as expanded-polytetrafluoroethylene, polyglactin, polylactic acid, and collagen are employed in the hope of excluding epithelium and the gingival corium from the root surface in belief that they interfere with regeneration.

GENERAL

Wound healing involves many intricate mechanisms at the ultracellular and cellular level. However, the events which take place at the tissue level are of the utmost interest to the clinician and compose the main focus of this discussion

Epithelial Regeneration

Engler et al. (1966) studied the healing sequence of epithelialization following simple gingevectomy in monkeys using thymidine radiography. On a cellular level, mitosis within the basal cell and deep spinous layers of the epithelium provides cells for the process of epithelial migration. Notably, the replication rate for the junctional epithelium is about 5 days versus 10 days for gingival epithelium. Once the cells form, they are then expressed outward by a passive phenomenon. The source of epithelium required to cover a given wound is the epithelium peripheral to and adjacent to the wound site. Early in the healing process, epithelial cells begin to migrate (12 to 24 hours) over the wound by peaking at 24 to 36 hours. This movement proceeds at a rate of approximately 0.5 mm/day and takes place between the clot and the poly band which was established by 6 to 12 hours following surgery. Although the wounds were keratinized by 2 weeks, it took between 3 and 5 weeks for the new gingival sulcus to completely heal.

Connective Tissue

Collagen. In a radioautographic study of connective tissue healing following simple gingivectomy, Ramfjord et al. (1966) offered the following observations. Following clot formation and development of a PMN "poly band" at 13 hours, an acute inflammatory reaction (primarily macrophages) forms under the clot next to the poly band. This inflammatory reaction is responsible for the clearance of necrotic cells and provides an avenue for epithelial migration over the connective tissue and under the clot. Connective tissue matrix formation begins 1 to 2 days after surgery and peaks at 3 to 4 days. Collagenous maturation and functional orientation of the gingival CT required 3 to 5 weeks.

Bone. Wilderman (1964) reviewed the effects of bone exposure in periodontal surgery. Wound healing events accompanying temporary bone include presence of a fibrin clot beneath the flap and inflammation in the marrow spaces and Haversian canal. Granulation tissue was invading the clot at 4 days and bone resorption observed at days 4 to 8. Opposition of new bone occurred from days 10 to 21.

Cementum. Hiatt et al. (1968) studied repair following mucoperiosteal flap surgery and observed cementum formation as early as 3 weeks, which continued through the sixth month.

WOUND HEALING FOLLOWING NON-SURGICAL THERAPY

Following non-surgical therapy, the periodontium heals by formation of a long junctional epithelium. According to the most recent Glossary of Periodontal Terms (1992), this healing response represents new attachment which is a form of repair. Lindhe et al. (1978) characterized the effect of non-surgical therapy on gingival healing in beagle dogs. The junctional epithelium of healed tissues contained rete pegs and a much greater vascular density in the connective tissue subjacent to the junctional epithelium as compared to healthy gingiva which had not been inflamed. Caton and Zander (1979) used a ligature-induced periodontitis model in Rhesus monkeys to create periodontal defects which were then treated by scaling and root planing and soft tissue curettage. Healing was by the formation of a long junctional epithelium.

No new connective tissue attachment was observed.

Waerhaug (1978A, and 1989B) examined 39 teeth which were treated by scaling and root planing and removed by block section at 15 days to 7 months after treatment. Healing occurred by formation of a long junctional epithelium. Based on 2 samples, it was estimated that the subgingival plaque front had advanced at 2 μm per day. Waerhaug (1978B) also investigated the condition of root surfaces following subgingival plaque control. Eighty-four (84) teeth were extracted at various time intervals after subgingival scaling and root planing (11 cases were completed with flap access). After staining with toluidine blue, stereoscopic examination revealed that in pockets less than 3 mm deep, subgingival plaque removal was successful 83% of the time. Successful subgingival plaque removal was much less in 3 to 5 mm (39%) and > 5 mm pockets (11%). The author felt that these findings supported pocket elimination therapy.

The effect of postsurgical plaque control on bone regeneration in periodontal defects has also been investigated. Rosling et al. (1976) demonstrated that professional plaque control every 2 weeks (after modified Widman flap surgery) resulted in 80% bone fill (as measured using standardized radiographs) as compared to no bone fill or deepening of intrabony defects in control patients who received professional plaque control every 12 months post-surgery. In a later study using a ligature-induced periodontitis model in squirrel monkeys, Kantor (1980) reported reduction of the inflammatory cell infiltrate from 68% to 14% and 50% increase in bone fill after 10 weeks of professional subgingival and supragingival plaque control administered 3 times per week.

In addition to plaque control, the condition of the root surface may also affect non-surgical wound healing. Aleo et al. (1975) used an in vitro system to show that periodontally-diseased root surfaces inhibited attachment of gingival fibroblasts. Conversely, root surfaces scaled to remove diseased cementum or treated by phenol extraction allowed cell attachment to take place. The researchers concluded that either chemical or mechanical removal of toxic substances from the root surface is necessary to allow fibroblast attachment to the root surface.

GENERAL WOUND HEALING WITH SURGICAL THERAPY

General wound healing considerations in surgical therapy have included the nature of the epithelial and connective tissue attachment, effect of flap reflection on the underlying alveolar bone, and the relative merits of full thickness and partial thickness flap designs.

Frank et al. (1972) addressed reformation of a junctional epithelium after surgical therapy by examining a block section of 4 anterior teeth and associated gingiva which was removed 4 months after flap curettage. Electron microscopy demonstrated that following open flap curettage, the epithelial attachment reforms with the presence of hemidesmosomes and a basal lamina. No difference was seen in the epithelial attachment to cementum or dentin. Using mongrel dogs, Hiatt et al. (1968) investigated the strength of the flap attachment after full thickness flap reflection. At 2 to 3 days postsurgery, flap adaptation was mediated by fibrin adhesion which was strong enough to prevent downgrowth of epithelium if the flap was well adapted. Using a suture through the flap, 225 grams of tension was required to remove the flap. At 1 week, 340 grams was required to displace the flap and by 2 weeks 1,700 grams of force could not displace the flap. Epithelial attachment was reformed by 1 week and increased in strength at 2 weeks such that flap traction resulted in microscopic flap tears within the epithelium with epithelial cells adhering to the root. By day 14, new connective tissue attachment was observed at the root surface. Osteoclastic activity was observed up to 3 weeks (maximum bone loss was 1 mm) while the earliest osteoblastic activity was noted at 2 weeks. Osteoblastic activity restored the lost bone. Cementum formation occurred over 1 to 6 months. Retained vital cementum appeared to accelerate connective tissue attachment. Dentin surfaces which had been denuded of cementum by root planing all underwent resorption prior to new cementum formation.

Crestal bone resorption has been addressed in studies comparing the merits of full and partial thickness flap reflection. Wood et al. (1972) compared apically positioned full and partial thickness flaps in 9 patients. Using clinical photographs, measurements, and surgical re-entry (7 patients), it was determined that healing was delayed at 1 and 2 weeks with partial thickness flaps but no differences were seen by 1 month. Mean crestal alveolar bone loss was 0.62 mm for full thickness flap and 0.98 mm in areas treated by partial thickness flaps. Use of the partial thickness flap in areas of thin gingiva resulted in a very thin non-protective layer of connective tissue and significant osteoclastic activity was seen. The authors concluded that loss of crestal alveolar bone depends on the pre-operative thickness of the radicular bone and overlying gingiva and mucosa. Thus, partial thickness flaps are not indicated in areas of thin connective tissue. In contrast, Staffileno et al. (1966) studied the histologic repair of the periodontium in dogs following resection of a split thickness flap. Results demonstrated that split thickness flaps with periosteal retention produced minimal tissue destruction, rapid repair, slight alteration of the dentogingival junction, and maximum preservation of the periodontal supporting structures.

Vascular healing is also of importance to wound healing in general. Cutright (1969) examined the rate and pattern of vascular regeneration after incisional wounds in dog gingiva. At day 1, withdrawal and blockage of cut ends of vessels were observed at the wound periphery. At day 2, new sprouts and club-shaped stubs were observed at the bottom of the wound where a fibrin clot was found. At days 3 to 5, short capillary loops from cut vessel ends formed anastomoses of vessels and were of normal length by day

11. Vascular healing proceeded in an apical-incisal direction.

WOUND HEALING WITH RESECTIVE SURGICAL THERAPY

Sabag et al. (1984) studied the reformation of the epithelial attachment after gingivectomy in an animal model (Donjou rats). At day 1, the wound was covered by a blood clot and many neutrophils. Epithelial migration began after day 2 and after 5 days covered the proliferating cells and fibers of the underlying connective tissue. After 7 to 8 days, a new junctional epithelium was seen with evidence of attachment to the root surface by 12 to 14 days. In a similar study, healing was reported by Novaes et al. (1969) after gingivectomy procedures in dogs. Re-epithelialization was complete at 7 days and the normal thickness of the junctional epithelium was restored by 16 days. Connective tissue healing was complete by 23 days. The gingiva exhibited a normal shape and healing was complete at 38 days. In humans, surgical excision of the entire zone of gingiva by gingivectomy procedure heals with the regeneration of a thin band (average of 2.05 mm) of keratinized tissue (Wennstrom, 1983).

The long-term healing following flap and osseous surgery was studied by Wilderman et al. (1960). The histologic repair of human tissue following mucogingival flap and osseous surgery indicated that osteoblastic activity was still present at 1 year postsurgery. The initial crestal bone loss of 1.2 mm (range: 0.14 to 4.47 mm) was followed by 0.4 mm (range: 0.14 to 1.15 mm) of new bone apposition which resulted in an average reduction in alveolar crest height of 0.8 mm (range: 0.11 to 3.1 mm). Bone thickness was an important factor determining the amount of postoperative bone loss. Thick bone with marrow spaces exhibited less resorption and greater repair than thin bone.

WOUND HEALING IN MUCOGINGIVAL SURGERY

Denudation Procedures

Using a monkey model, Karring et al. (1975) studied the wound healing events of periosteal retention and denudation procedures designed to increase the zone of gingiva. Following both procedures, granulation tissue was observed to originate from residual periosteal connective tissue, PDL, bone marrow spaces, and the adjacent gingiva and alveolar mucosa. Bone resorption was generally more severe with the denudation procedure; however, greater amounts of loss were occasionally seen following periosteal retention. The transitional point between keratinized and non-keratinized epithelium corresponded to the junction between the connective tissue with and without regenerated elastic fibers, demonstrating the inductive influence of connective tissue on the overlying epithelium.

In a similar study of the denudation procedure, Wilderman et al. (1960) studied histologic wound healing of exposed alveolar bone in dogs. Differences in the anatomy of interdental and radicular bone appeared responsible for varying degrees of osteoclastic resorption seen. Where adequate marrow spaces remained interdentally, there was complete restoration of bone. In contrast, radicular areas showed 50% bone restoration, demonstrating functional repair with twice the fibrous attachment on new gingiva as compared to the original condition of epithelial attachment located more apically compared to interdental sites. The osteoclastic phase was present from 2 to 10 days while osteoblastic activity lasted for 10 to 28 days and peaked at 21 to 28 days.

Free Soft Tissue Autograft

Studies by James and McFall (1978) and Caffesse et al. (1979) have compared the healing of free soft tissue autografts placed on periosteum and denuded bone. Clinically, success rates were similar for the 2 types of recipient beds. Caffesse et al. (1979) reported delayed remodeling at grafts placed on bone while James and McFall (1978A) reported less shrinkage of grafts placed on bone (25% versus 50% on periosteum). James et al. (1978) performed a histologic comparison of wound healing between the 2 types of recipient sites. More marrow space to soft tissue communication occurred at "graft-to-bone" sites. Epithelial thickness was greater over free grafts placed on bone until 12 weeks, by which time no difference was seen. Grafts placed on bone exhibited less post-operative swelling, but there was no difference in the degree of inflammation. Bone resorption at the graft-to-bone sites allowed sufficient blood supply from the underlying marrow spaces.

Pedicle Grafts

Pedicle grafts have the advantage of a blood supply from the base of flap which can aid in wound healing by providing nourishment until the re-establishment of a vascular union with the recipient site. Sugarman (1969) reported 3 cases with human histologic evidence of the healing obtained with pedicle grafts and free soft tissue autografts. The full thickness, laterally positioned flap healed by new attachment consisting of junctional epithelium (1.0 to 1.6 mm), connective tissue attachment (0.1 to 3.2 mm), and areas of new cementum. Wilderman and Wentz (1965) presented the wound healing events of pedicle flaps in dogs. Four stages of healing were reported: 1) adaptation stage (0 to 4 days) when a fibrin clot containing neutrophils was present between the flap and the crestal bone; 2) proliferation stage (4 to 21 days) when granulation tissue invaded the fibrin clot, fibroblasts were present on the root surface (6 to 10 days), epithelium migrated apically (10 to 14 days), and an average of 1 mm of crestal bone was resorbed; 3) attachment stage (21 to 28 days) when collagen formation was visible, cementum formation occurred, and osteoblastic activity reached its peak; 4) maturation stage (28 to 180 days) showed new PDL fibers oriented perpendicularly to the root surface. New attachment consisted of a combina-

tion of long junctional epithelium (2.0 mm) and connective tissue attachment (2.1 mm).

Becker et al. (1986) investigated the repair of intrabony defects. Open debridement was performed, hard tissue measurements taken, and flaps were apically positioned to leave the margins of the flaps open adjacent to the treated sites. Re-entries were performed at an average of 14 months and results indicated that 34 of the 36 intrabony defects treated exhibited significant amounts of bone fill. Mean defect fill was 54.25% (2.55 mm) and defect resolution was due to the combination of crestal resorption and fill from the defect base and surrounding osseous walls.

Similar results were reported by Polson and Heijl (1978) following their treatment of intrabony defects; however, unlike Prichard (1983) and Becker et al. (1986), flaps were replaced with their margins at the pre-surgical level following open flap debridement. Re-entry at 6-8 months revealed defect remodeling consisting of 77% bone regeneration and 18% bone resorption. Osseous defect mean bone regeneration at re-entry was 2.5 mm. Two-and three- walled defects exhibited the same potential for osseous regeneration and initial mobility did not affect the regeneration potential.

Ellegaard et al. (1974) described a technique for attaining new periodontal attachment using free palatal or gingival grafts to retard epithelium migration in intrabony defects. The defect was exposed and debrided, a recipient bed for a free soft tissue graft prepared, a cancellous bone autograft placed, and the soft tissue graft inserted over the treated intrabony defect. The authors reported on 88 lesions treated with this procedure and evaluated at 3 and 6 months with clinical measurements and radiographs. The amount of new attachment was markedly greater with the soft tissue grafting technique than with a traditional flap procedure. Sixty percent (60%) of the defects treated using the soft tissue grafting technique exhibited 60% of the defects to fill completely, compared to 40% of defects showing some new attachment with conventional flap design. The practicality of this technique may be the reason for its lack of popularity as it requires a minimum of 3 different surgical sites.

Caton et al. (1980), using Rhesus monkeys, performed a histometric comparison of the following procedures: 1) modified Widman flap; 2) modified Widman flap with frozen autogenous red marrow and cancellous bone graft; 3) modified Widman flap and tricalcium phosphate; and 4) periodic root planing and soft tissue curettage. Animals were sacrificed after 1 year. All procedures resulted in healing by a long junctional epithelium with no new connective tissue attachment.

Although true regeneration was not achieved by any of the procedures compared in the study by Caton et al. (1980), studies have suggested that a long junctional epithelium may be as resistant to periodontal insult as connective tissue. Magnusson et al. (1983) used monkeys to compare histologically the effect of ligature-induced periodontitis on teeth with a known long junctional epithelial attachment and on teeth with a normal junctional epithelium. Results after 6 months demonstrated that a gingival unit with a long junctional epithelium responded to plaque infection in a manner similar to normal junctional epithelium. Similarly, in a shorter study (up to 20 days) Beaumont et al. (1984) reported that long junctional epithelial attachment formed in beagle dogs after flap surgery was no less resistant to plaque and its products than true connective tissue attachment. Although the study was relatively short, the authors noticed a trend toward replacement of the long junctional epithelium by connective tissue at longer time intervals.

Regeneration of lost periodontium is a process which is influenced by several different factors. Egelberg (1987) reviewed several clinical and laboratory studies which have investigated the role of factors such as the diseased root surface, the effect of various clinical regenerative techniques, supracrestal wound healing and healing of the periodontal ligament, and root resorption. Polson and Caton (1982) designed a study to evaluate the relative significance of a reduced periodontium and a diseased root surface in the formation of new bone, cementum, and periodontal ligament. The regenerative capacity of the reduced periodontium was evaluated by transplanting a tooth with a non-diseased surface into the reduced periodontium. The regenerative potential for the tooth with the diseased root surface was evaluated by transplanting it into the normal periodontium. Results revealed that the diseased root surface that had been placed into the normal periodontium was lined with epithelium interposed between root surface and alveolar bone. The normal root surface that had been placed into the reduced periodontium had connective tissue reattachment in the periodontal ligament and supracrestal region. It appeared that root surface alterations inhibited the potential for new connective tissue attachment and that the connective tissue in areas of a reduced periodontium possessed the progenitor cell populations necessary for this attachment formation.

Root surface alterations were again shown to inhibit connective tissue attachment in an investigation by Lindhe et al. (1984). The authors designed a study to examine if alveolar bone, located adjacent to a root surface deprived of its periodontal ligament and cementum layer, could stimulate the reformation of a connective tissue attachment. Using reimplanted incisors in monkeys, a histometric comparison of root planed teeth with either reduced buccal alveolar bone or normal bone and non-root planed teeth with reduced or normal bone was accomplished. A 6-month evaluation demonstrated that, irrespective of the presence or absence of alveolar bone, a fibrous reattachment failed to form on that part of the reimplanted teeth which had been deprived of their periodontal ligament. In those teeth where periodontal ligament and cementum were preserved, reattachment of connective tissue (CT) fibers occurred whether or not ad-

TABLE 1. WOUND HEALING

Study	Begin Migration	Epithelium Re-epithelialize	Reform JE	Connective Tissue Fibroblast Activity Began	Fibroblast Activity End	Bone Osteoclastic Phase	Osteoblastic Phase	Cementum Formation
Wilderman, 1960 (dogs), denudation procedure	2 days, granulated tissue from PDL, gingiva	Complete at 21 days			New CT covering at 14 days	2-10 days, undermining resorption peak 4 to 6 days	10-28 days, peak 21 to 28 days	
Staffileno, 1966, periosteal *retention*	2 days fibrin, clot, and PMN	Complete at 7 days	Complete at 14 days	4 days		Peak at 4 days over at 7 days	Begins at 7 days, *new bone at* 21 days	
Hiatt, 1968, mucoperiosteal flap			Complete at 7 days	Fibrin clot 2 to 3 days	14 days new CT at root surface	Up to 3 weeks	Begins at 14 days	1 to 6 months
Wood, 1972 full vs. PT flap						Crest reduction; full-0.62 mm PT-0.98 *mm*		
Novaes, 1969 (dogs) gingvectomy	Clot, first epithelial migration, 2 days	Complete at 7 days	16 days normal thickness	New CT at 7 days	Complete at 23 days			
Wilderman, 1965 pedicle flap	Clot, 2 to 4 days	Greatest rate 10 to 15 days		New CT at 4 days, cover root at 10 days	Increase CT formation at 28 days; *collagen* bundles from flap to root at 90 days	Present at 4 days, subsides by 14 days	Greatest at 21 to 28 days	Cementoid covered entire root surface at 28 days
Caffesse, 1979 FGG (Rhesus monkey)	At 7 days	Complete at 14 days		Periosteal bed 2 to 4 days Denuded bone none till 7 days				
James and McFall, 1978, on FGG* denuded bone bed		Epithelial variable thickness at 1 week		Capillary ingrowth from bone at 14 days	6 weeks-collagen fibers trapped in new osteoid	Resorption at 1 week, open marrow spaces	Thinner bone found at 2 to 4 weeks	

*Free gingival graft

jacent bone was present. It was concluded that alveolar bone adjacent to a root surface may have little influence on the biological conditions which determine whether periodontal healing results in CT reattachment or new attachment.

In a series of articles by Bowers et al. (1989A, B, and C), the relative influence of several factors on the regeneration of a new attachment apparatus (bone, cementum, and a functional periodontal ligament) in humans was evaluated. Teeth with advanced periodontal disease were treated, extracted after 6 months, and subjected to histologic examination. In Part I of the study, formation of new attachment was evaluated in two groups of teeth: 1) those treated by open debridement, crown removal, and submersion of the vital roots below the oral mucosa and 2) those treated by open debridement but not submerged. Results showed that a new attachment apparatus (mean 0.75 mm) was found in submerged defects and no evidence of a new attachment apparatus was seen in the non-submerged defects (long junctional epithelium only). In Part II, similar comparison was made by 2 different groups: 1) teeth treated using open debridement and placement of decalcified freeze-dried bone allograft (DFDBA) in a submerged environment and 2) teeth treated using open debridement and no DFDBA but also in a submerged environment. When results from these 2 groups were compared, it was determined that a significant difference was found in the amount of new attachment apparatus formation for grafted versus non-grafted sites (1.76 mm versus 0.76 mm) in a submerged environment. Finally, in Part III, only non-submerged teeth were evaluated and placed in 1 of 2 groups: 1) those teeth

TABLE 2. SEQUENTIAL WOUND HEALING EVENTS

Vascular regeneration Cutright, 1969 (incisional wound in dogs)	Day 1 Vessel contraction no cell labeling	Day 2 New vascular sprouts, club shaped ends	Day 3 Short capillary loops from anastomes of cut ends	Day 5 Increased capillary loops, dilitation of one end-venous differentiation	Day 7 Capillary loops reformed at wound edges	Day 9 Capillary loops about as high as normal gingiva, but not as dense	Day 11 Increased maturation of normal vasculature
Epithelial regeneration Sabag, 1984 (gingivectomy in rats)	Day 2 Migration of oral epithelium seen	Day 5 Epithelium covers cut CT, crevicular epithelium reformed	Days 7-8 JE stratification present	Days 9-11 JE thicker	Days 12 to 14 apical third of new JE joined to cemental surface, new epithelial attachment		
Granulation tissue Karring, 1975 (Rhesus monkeys)	Exposure of periosteum (periosteal retention procedure)	Days 1-2 Clot and PMN infiltrate	Days 4-5 granulation tissue formed from PDL space	1-3 weeks osteoclastic activity gingiva regenerated by 3 weeks	3-6 weeks osteoclastic activity subsided, epithelium thickened, well developed rate ridges	2 to 12 months variable amounts of alveolar crest resorption found	
Same	Exposure of bone (denudation procedure)	Thin fibrin clot over bone, many PMN, surface of bone necrotic	Some granulation tissue from PDL space bone resorption, especially at alveolar crest	Undermining resorption seen on PDL side of bone, granulation tissue extends from open marrow spaces	Osteoclastic activity subsided, new bone formation seen, thin epithelial covering over wound	Bone formation subsided, keratinized epithelium present, variable amounts of alveolar crest resorption	
Osseous surgery Wilderman, 1970	1 week CT proliferation	2 weeks continued CT proliferation, apical migration of epithelium	Crestal resorption averaged 1.2 mm, crestal apposition of 0.4 mm	Average bone loss 0.8 mm	2 months CT embedded into bone	3 months periosteum formed	

which were treated with open debridement and DFDBA and 2) those teeth which were treated with open debridement alone. Results revealed that grafted, non-submerged sites demonstrated 1.21 mm of new attachment apparatus while non-grafted, non-submerged sites healed by long junctional epithelium only. Free gingival grafts were used to enhance wound coverage and retard epithelial migration for both groups in Part III but these did not appear to enhance regeneration of a new attachment apparatus, new cementum, new connective tissue, or new bone in sites which were not treated using bone grafts. The role of free gingival grafts in DFDBA grafted sites could not be determined in this study.

Tables 1 and 2 summarize general wound healing events.

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CHAPTER 10. SUPPORTIVE PERIODONTAL THERAPY

DEFINITION

Supportive Periodontal Treatment (Periodontal Maintenance, Preventive Maintenance, Recall Maintenance): An extension of periodontal therapy. Procedures performed at selected intervals to assist the periodontal patient in maintaining oral health. These usually consist of examination, an evaluation of oral hygiene and nutrition, scaling, root curettage, and polishing of teeth.

TOOTH MORBIDITY/INCIDENCE

Pelton et al. (1954) evaluated tooth morbidity experience in adults. Data were obtained from the analysis of nearly 225,000 dental examinations of U.S. Public Health Service beneficiaries over a 5-year period. Of a possible 32 teeth with an average age of 41 years, 19.1 or 60% were decayed, missing, or filled (DMF). DMF increase with age, with missing teeth contributing the greatest weight to the total morbidity index after the age of 33. The number of decayed teeth and filled teeth increases with age and the number of unaffected teeth remaining in the mouth decreases. The number of teeth indicated for extraction increases with age, and the number requiring fillings decreases with age. This study reveals a DMF rate increases about 0.25 of a tooth per year over 50 years of an adult life. The component of the DMF rate due to missing teeth increases at 0.4 teeth per year. Patients under 35, primarily lost teeth due to caries, those above that age had periodontal disease as the controlling factor.

NATURAL HISTORY

In 1978A, Löe et al. published the initial observations in a longitudinal study on the initiation and progression of periodontal disease in man. The study design consisted of 1 group of 565 Norwegian students and academicians and another group of 480 Sri Lankan tea laborers. The periodontal state of the Norwegian group had good to excellent oral hygiene and mild gingivitis. Supra- and subgingival calculus was inconspicuous and untreated gingival caries rare. Slight attachment loss was seen in the youngest group and increased slowly with age, with a calculated rate of attachment loss of 0.05 mm per year during the 20s. The mean at 30+ year old was less than 1 mm. The Sri Lankan group showed poor oral hygiene, abundance of calculus, and generalized moderate to severe gingivitis. Caries and dental restorations were non-existent. Attachment loss was evident in the 15-year-olds and increased through the 20s. At 30+ years of age the tea laborers showed a mean loss of more than 3 mm and a great number in excess of 10 mm. A rate of attachment loss was calculated to be approximately 0.20 mm per year. The results of this investigation show that all stages of periodontal health and disease are present in these 2 populations.

In a second report, Löe et al. (1978B) discussed tooth mortality rates before 40 years of age by comparing the two populations. Both populations were examined 4 times during the study, a period of 6.25 years for the Norwegians and 7.5 years for the Sri Lankans. The 17-year-old Norwegians had 27.4 teeth with no major loss of teeth occurring during their 20s and 30s. As they approached 40, the mean number of teeth present was 27.1 and the mean mortality rate was 0.01 teeth per year. Sri Lankan 15-year-olds had 27 teeth present and the 40-year-olds had 25.6 teeth. The mean mortality rate ranged between 0.1 and 0.3 teeth per year.

A third report (Anerud et al., 1979) reviewed overall changes in gingival health and oral hygiene before 40 years of age. The overall gingival state of the 17-year-old Norwegians was good to excellent, with no significant increase of gingivitis to approximately 40 years of age. The 17-year-old Sri Lankans' gingival health was considerably poorer, with slight increases in GI levels occurring to age 40. In the Norwegians, 60 to 70% of all tooth surfaces had no visible plaque and oral hygiene continued to improve toward age 40. The Sri Lankans had clinically visible plaque covering almost all tooth surfaces at 15 years old, with no significant change occurring before 40.

UNTREATED DISEASE

Becker et al. (1979) examined 30 patients diagnosed with moderate to advanced periodontal disease, but who were not treated periodontally. The time interval between examinations ranged from 1.5 years to 9 years, 7 months (mean = 3.72 years). A total of 83 teeth were lost (10.6%), but a patient who lost 25 teeth was excluded, resulting in 58 teeth lost (7.7%), or 0.61 teeth per patient per year. Mandibular first and second molars were most frequently lost, followed by maxillary molars. Mandibular canines and incisors were most resistant loss. Teeth that were lost had deeper probing depths (PD) and greater mobility that other teeth, with greater PD at disto-lingual and mesio-lingual surfaces. An inverse relationship was observed between PD increase and patient age. The rate was slower for patients $>$ 44 years compared to younger patients. All patients had radiographic evidence of progressive bone loss.

Lindhe et al. (1983) studied 64 Swedish subjects with mild to moderate periodontal disease, monitoring attach-

ment levels over a 6-year period during which no periodontal therapy was received. Comparisons were made with 36 Americans who had a more advanced destructive periodontal condition and were observed at 1 year. At 3 and 6 years for the Swedish subjects, 1.6% of sites showed attachment loss of more than 2 mm, 57.4% of sites had no change and 0.5% of sites showed more than 2 mm of attachment "gain." Approximately 50% of sites that showed no change in the first 3 years, showed loss in the next 3 years. Attachment loss between the baseline and 3 and 6 years averaged ~ 0.2 mm per year. Of sites monitored in the American group, 102 sites (3.2%) exhibited more than 2 mm of additional attachment loss; 26% sites no change; and 138 (4.3%) sites showed a decrease in probeable attachment level. Significant progression of disease is an infrequent event, and demonstrated that sites with initially more advanced attachment loss were no more likely to show disease progression than areas with less attachment loss.

Buckley and Crowley (1984) examined 1,016 textile workers of whom 82 had no dental treatment other than tooth extraction in the 10-year period of the study. The average age was 27.0 and average tooth loss was 2.5 teeth per individual over the observed period. The variation in pattern on destructive periodontal disease in this study suggests that the disease is intermittent in nature and is not linear in time. Teeth most frequently lost were maxillary molars. Overall 6% of teeth initially free of periodontal disease (PI=0) were lost compared to 14% with destructive periodontal disease (PI=6). The rate of progression of established periodontal disease was similar for anterior and posterior teeth, while the onset of gingivitis was more pronounced in posterior teeth.

TREATED AND NOT MAINTAINED

Nyman et al. (1977) studied 25 patients with advanced periodontal disease who were treated by 1 of 5 procedures to eliminate probing depth. Patients were instructed once in oral hygiene, not recalled for maintenance, and were evaluated at 2, 6, 12, and 24 months. The plaque scores 6, 12, and 24 months ranged from 1.1 to 1.3. A similar pattern was seen in regard to the gingival scores. The initial probing depth was 5 to 6 mm and at 6 and 12 months the probing depths varied from 2.6 and 3.3 mm. After 24 months the average depth was approximately 4 mm. At the end of the study there was a significant loss of attachment for all 5 groups on the lingual (1.2 to 1.6 mm) and approximal (1.5 to 1.9 mm) surfaces. The authors concluded that periodontal surgery will fail in those patients with poor oral hygiene who are not seen on regular recall.

De Vore et al. (1986) assessed bone levels around individual tooth groups in 23 patients treated for periodontal disease and followed with infrequent maintenance (≤ 1 visit per year). Post-therapy radiographs were taken between 2.5 to 8.3 years. Bone loss was defined as a reduction in the alveolar crest of greater than 50% of the radiographic crown height which corresponds to approximately 4 mm in the posterior and 5 mm in the anterior teeth. Results showed increased bone loss and tooth loss when compared to initial presentation. Molar teeth were at more risk than incisors and canines and a lack of periodontal maintenance care and inadequate plaque control resulted in progressive bone loss following treatment.

TABLE 1. AVERAGE TOOTH LOSS RATE FOR TREATED AND UNTREATED PERIODONTITIS*

Study	Treatment Status	Tooth Loss Per Year	Years to Lose 1 Tooth
Lindhe et al. (1984)	Treated	0.02	43.5
Oliver (1968)	Treated	0.03	33.3
Hirschfeld and Wasserman (1978)	Treated	0.08	12.5
McFall (1982)	Treated	0.13	7.7
Nabers et al. (1988)	Treated	0.29	3.5
Becker et al. (1984)	Treated	0.11	9.1
	Treated and not maintained	0.22	4.5
	Untreated	0.36	2.8
Buckley and Crowley (1984)	Untreated	0.25	4.0 †

*Average tooth loss per patient divided by the average time in maintenance or observation.
†The rate of tooth loss is 2 to 12 times higher for untreated periodontitis.

Becker et al. (1984A) presented a retrospective report on 44 patients (1,117 teeth) treated for periodontal disease who received oral hygiene instruction, initial SR/P and 2 or more quadrants of pocket reduction therapy and who subsequently elected not to participate in the maintenance phase of treatment. The average time between examinations was 5.25 years, with a mean annual adjusted tooth loss rate of 0.22 (4.7%). The authors compare this to a mean adjusted tooth loss of 0.11 for treated and maintained patients and 0.36 for diagnosed and untreated patients. At re-examination there was a breakdown in health status of furcations, no reduction in probing depths, and worsened bone scores. It was felt that surgical intervention was of little value when there was an absence of the maintenance phase of periodontal therapy. Average tooth loss for treated and untreated periodontitis is shown in Table 1.

RESPONSE TO TREATMENT: SHORT-TERM (2 to 3 YEARS) STUDIES

A 3-year study of 1,248 patients by Suomi et al. (1971) tested the hypothesis that the development and progression of gingival inflammation and destructive periodontal disease is retarded by high levels of oral hygiene and maintenance. An experimental group received a high level of oral hygiene as well as a series of frequent oral prophylaxes, combined with OHI and dental health education. Patients in the control groups received only annual examinations, but they were

advised to continue with usual oral hygiene professional care. After 3 years, the increase in oral hygiene score was more than 4 times greater in the control when compared to experimental. Mean gingival inflammation scores were also greater in controls. The authors concluded that maintaining high levels of oral hygiene slows any progress of periodontal disease.

Nyman et al. (1975) treated 20 patients with pocket elimination surgery, who were divided into a test group who received oral hygiene instruction and professional cleaning every 2 weeks, and a control group who received tooth cleaning every 6 months. Examination was repeated 6, 12, and 24 months after the end of the treatment. The test group maintained good oral hygiene and had no further loss of attachment, while the control group experienced treatment failure with further loss of attachment. It was concluded that good oral hygiene and professional cleaning are essential for the success of periodontal surgical treatment.

In a 1976 study (Rosling et al.), 50 patients were randomly distributed into 5 groups treated by apically repositioned flap with or without resection of bony defects, Widman flap with or without elimination of the bony defects, and gingivectomy. After surgery, all patients received oral hygiene instruction and professional cleaning of the teeth once every 2 weeks during a 2-year period. Results indicated that this regimen prevented further destruction of the periodontal tissues, regardless of the surgical technique used for pocket elimination.

Axelsson and Lindhe (1981A and B) treated 90 patients with advanced periodontal disease and divided them into 2 study groups. All patients received detailed oral hygiene instructions, a scaling and prophylaxis, removal of ill-fitting margins of restorations, and surgery as needed. One group of patients was returned to the referring dentist while the other group entered a carefully-designed clinic maintenance care program. Results demonstrated that patients placed on a carefully designed recall program were able to maintain excellent oral hygiene standards and stable attachment levels over a 6-year period after treatment for periodontitis. The non-recall group lost on average 1.8 mm of attachment over the 6-year period. Patients who were not maintained in a supervised program were more prone to develop recurrent disease.

Westfelt et al. (1983) studied the significance of frequency of professional tooth cleaning for healing following periodontal surgery. Twenty-four (24) patients with moderate-advanced periodontitis were treated with modified Widman flap surgery and divided into 3 groups who received supportive periodontal care at 2, 4, or 12 week intervals for 12 weeks. After the first 6 months, recall was changed to every 3 months for all groups. At the 6-month exam, patients seen every 2 weeks had low numbers of inflamed gingival units and deep (> 3 mm) periodontal pockets; test patients seen less frequently exhibited an increasing number of inflamed gingival units and deeper pockets. Sites exhibiting attachment loss of > 1 mm was closely related to maintenance care. Group 3 had 3 times the number of sites with attachment loss than group 1. There were no significant changes in oral hygiene status, gingival condition, or the probing depth and attachment levels in the 3 groups between the 6 and 24 month re-examinations. Critical probe depth (CPD) values were also evaluated; i.e., the initial probing depth below which loss of clinical attachment occurred and above which attachment gain had resulted. The analysis showed that the CPD value was low in patients frequently recalled during healing phase, it increased in patients with less frequent intervals (Group 1 CPD = 4.4 ± 0.3 mm; group 2 CPD = 4.9 ± 0.3 mm; and group 3 CPD = 5.4 ± 0.7 mm).

RESPONSE TO TREATMENT: LONG-TERM (6 OR MORE YEARS) STUDIES

Oliver (1969) evaluated tooth loss in a group of 442 patients treated in private periodontal practice. Maintenance periods ranged from 5 to 17 years with an average of 10.1 years. Patients were seen on average every 4.6 months for their recall. Tooth loss due to periodontal disease was 178 of approximately 11,000 teeth available for therapy. This represents an average of less than one-half a tooth per patient over the 10-year period. Seventy eight percent (78%) of the patients did not lose any teeth and 11% lost only one tooth after therapy. Only 45 teeth were lost to caries or pulpal involvement, for 0.1 tooth per patient. Results indicated that periodontal disease can be effectively treated and that tooth loss due to periodontal disease can be prevented.

In a 1978 study by Hirschfeld and Wasserman 600 patients in a private practice were reexamined an average of 22 years (15 to 53) following their active treatment. Patients were divided on the basis of response to treatment into the following groups: well-maintained (WM) (lost 0 to 3 teeth) 499 patients (83.2%), downhill (D) (lost 4 to 9 teeth) 76 patients (12.6%), and extreme downhill (ED) (lost 10 to 23 teeth) 25 (4.2%). Tooth retention seemed more closely related to the case type. In general, the percentage of tooth loss was 7.1% (1,110 out of 15,666), and 31.4% for teeth with furcation involvement. Only 666 out of 2139 teeth that originally had been considered questionable were lost. The authors noted that periodontal disease is bilaterally symmetrical, with the mandibular, cuspids, and first bicuspids being most resistant and the maxillary second molars most susceptible to loss.

McFall (1982) reviewed long-term tooth loss in 100 treated patients with periodontal disease. The study population consisted of 77 well-maintained; 15 downhill; and 3 extreme downhill patients with a total of 2,627 teeth. During the maintenance period (an average of 19 years), 259 teeth (9.8%) were lost to periodontal disease and 40 (1.5%) lost for other causes. In the WM group 43.3% of teeth designated questionable were lost, an average of 0.68 teeth per patient. In

the downhill group, 60.4% of the questionable teeth were lost (6.1 teeth per patient), and the extreme downhill group lost 48.7% of the teeth deemed questionable (14.4 teeth per patient). Of a total of 163 maxillary and mandibular furcated teeth, 94 (56.9%) were lost: WM group losing 18 (27.3%), D group losing 68.9%, and ED losing 92.3%. Following surgical treatment, 131 out of 832 teeth were eventually lost (15.8%). By comparison 128 teeth not treated surgically were lost. More surgery was performed in the maxillary than mandibular arch. Molar teeth, particularly maxillary molars, represented the highest percentage of teeth lost following surgical treatment. Mandibular canines had the best survival rate in all groups. A bilateral pattern of osseous destruction was seen.

Meador et al. (1985) reviewed 620 patient records to determine the long-term effectiveness of periodontal therapy in a clinical practice. Cases (median treatment period 7.4 years) consisted of Type I and II patients treated nonsurgically, Type III patients treated by closed or open curettage or modified Widman flap (MWF), and Type IV patients treated by flap and osseous surgery. At 2-year reevaluation intervals, patients were classified as stable ideal (SI) probing depth (PD) up to 3 mm, no tooth loss; stable satisfactory (SS) PD up to one half pre-treatment levels, tooth loss up to 4 teeth; unstable retreatable (UR) increased PD and/mobility, radiographic bone loss, tooth loss; or unstable unsatisfactory (UU) severe increase in PD, mobility, radiographic bone loss, and tooth loss. Following treatment, the stable category included 71.93% of the patients (10.48% SI, and 61.45% SS), and the unstable category included 28.07% of patients (27.1% UR, and 0.97% UU). The authors concluded that moderate surgical procedures (open curettage, MWF) were at least as effective as osseous surgery, while non-surgical and closed curettage appeared to be less effective.

In a retrospective study, Goldman et al. (1986) assessed the effect of periodontal therapy on 211 patients maintained for 15 years or longer (15 to 34 years; average 22.2 years) at 3- to 6-month intervals. There were 131 (62%) subjects in the well-maintained group, 59 (28%) in the downhill group, and 21 (10%) in the extreme downhill group. There were 467 maxillary molar and bicuspid teeth and 169 mandibular molars that presented with radiographic evidence of furcation involvement. Of these, 201 maxillary teeth (43%) and 76 mandibular molars (45%) were lost during therapy. A total of 43.5% of the teeth initially diagnosed with a furcation involvement were lost. Molar teeth are most prone to loss and mandibular cuspids were most resistant to loss. The well-maintained group lost 16.9% of teeth with furcation involvement compared to 66% in the downhill and 93% in the extreme downhill group. A total of 13.4% of all teeth were lost over the mean period of 22 years. Seventy-two percent (72%) of all patients received surgery during active treatment and only a few cases required retreatment.

Lindhe and Nyman (1984) reported on the long-term maintenance of 61 patients treated for advanced periodontal disease. Patients with 50% or more of their periodontal support lost were given detailed oral hygiene instructions, scaling and root planing, and surgical elimination of periodontal pockets and then placed on a 3 to 6 month recall and followed for 14 years. During this time 92 to 99% of all sites maintained PD < 4 mm, while less than 1% of the sites developed probing depths > 6 mm. The mean attachment level was reduced from 6.1 mm to 5.4 mm and maintained at this level. However, attachment loss did occur at 16 in sites in 8 patients during the maintenance phase, 6 sites losing 5 mm or more. During the 14 years of maintenance, 30 of the 1,330 (2.3%) teeth were lost during the course of the study, 26 for periodontal reasons. Results demonstrated that treatment of advanced forms of periodontal disease resulted in clinically healthy conditions and that this state could be maintained by patients over a period of 14 years. A small number of sites lost a substantial amount of attachment at different times of the maintenance period but mean plaque and gingival indices did not prove helpful in monitoring the isolated sites.

Knowles et al. (1979) evaluated the results of periodontal treatment related to probing depth and attachment level. Following initial treatment 78 patients had half-mouth treatment with either subgingival curettage, modified Widman flap (MWF), or pocket elimination surgery. The patients were recalled every 3 months for a prophylaxis and patients were followed for 8 years. The results showed that the reduction in probing depth (PD) and the gain in attachment were greater for the deep pockets than the moderate pockets. The initial changes 1 year after treatment were sustained over the 8-year period: 1 to 3 mm PD got slightly deeper and lost about 1 mm of clinical attachment (CAL); 4 to 6 PD were reduced approximately 2 mm with a gain of 0.5 mm in CAL; 7 to 12 PD were reduced approximately 4 mm with a gain of 2 mm CAL. The reduction in probing depth following curettage was less than the 2 other procedures, and the gain in attachment following MWF was greater than the other 2 methods.

Becker et al. (1984B) also assessed long-term periodontal treatment and maintenance in a retrospective study on 95 patients (average age 46). The frequency of maintenance intervals was planned on an individual basis, with a median of 5.2 months. The average time between exams was 6.58 years with a loss of 6.21% of the total teeth (annual loss of 0.24/year). When hopeless teeth were adjusted for tooth loss was 2.94% (0.11/year). Of molars without furcations at first examination, 87.8% remained stable. Pooled patient means for probing depths were 3.787 mm initially and 3.409 at second exam. Fifty-five percent (55%) of the pockets between 4 to 6 mm were reduced to 1 to 3 mm at reexamination. No evidence of bone loss was seen in ~50% of the patients. Of teeth initially identified as hopeless, 80% were missing at the second examination; only 1.7% of the "good" teeth, and 25% of the questionable teeth were lost.

Results indicated that periodontal therapy and maintenance were successful in reducing moderate to deep periodontal pockets with minimal long-term bone loss. In the year after the study was completed, 22% of the patients had dropped out of the maintenance program.

In a similar study, Nabers et al. (1988) reported on 1,535 treated periodontal patients who averaged 12.9 years since completion of treatment. No teeth were lost due to periodontal disease in 1,371 patients and a total of 444 teeth were lost from a group of 164 patients, an overall tooth loss rate of 0.29 for the entire patient group. Initially, 26.5% were treated non-surgically and 73.5% were treated surgically. Although many patients developed recurrent periodontal problems during recall, only 15.9% of the 1,535 patients required surgical retreatment. Teeth originally given a doubtful prognosis often were responsible for recurrent problems and sometimes required extraction.

Wennstrom et al. (1993) examined periodontal conditions of adult, regular dental care patients. The 12-year study of 225 randomly selected patients offered annual preventive care at 12 community dental clinics in Sweden indicated an overall low incidence of tooth loss (0.4) and periodontal disease. A decrease in gingival scores from 15% to 4% was also observed, with no change in probing depth. The mean probing attachment loss during the 12 years was 0.5 mm. Tooth site analysis revealed that buccal sites had more loss of attachment than lingual and approximal surfaces. Radiographic assessment of the alveolar bone height revealed a mean longitudinal loss of 0.2 to 0.4 mm. The mean longitudinal changes were similar in all age groups, showing that therapy provided was equally effective in all age groups, although differences in rate of deterioration may be due to individual differences in environmental or disease conditions. Almost all patients (96%) had at least 1 site with $\geq$ 2 mm of attachment loss during the 12 years of follow-up.

EFFECT OF PLAQUE AND GINGIVITIS ON MAINTENANCE

Ramfjord et al. (1982) reviewed oral hygiene and maintenance of periodontal support. Seventy-eight (78) patients were treated and maintained with 3 month recalls over a period of 8 years. Variations in probing depth and attachment levels were related to individuals with plaque scores above and below the median. Results indicated personal oral hygiene, based on plaque scores, was not critical for maintenance of post-treatment probing depth and attachment levels in patients receiving professional tooth cleanings every 3 months over the 8 years. After 1 year, there was no indication that individuals with poor oral hygiene had any greater loss of attachment than those with good oral hygiene.

In a companion paper, Morrison et al. (1982) reported on 78 patients in a 7-year longitudinal study which compared the effect of gingivitis on the maintenance of probing depth reduction and clinical attachment levels. For probing depths 1 to 3 mm and 4 to 6 mm there was no difference in pocket reduction maintenance. There was no difference in attachment response in 1 to 3 mm probing depths, and in 4 to 6 mm PD, lower gingivitis scores had better gain the first 2 years, but thereafter no difference was recorded. For 7 to 12 mm PD, lower gingivitis scores seemed to result in better probing levels and attachment gain for the first 3 years, but this was not maintained throughout the study. The authors concluded that the severity of recurrent gingivitis with 3-month recall and maintenance following therapy had little effect on probing depth and attachment level.

SUPPORTIVE THERAPY

According to Chace (1951), maintenance of the treated periodontal patient should be carefully considered and a definite routine established. Pertinent factors include patient education, oral hygiene reinforcement, full-mouth radiographs every 2 years, and 2 to 3 month recall intervals. The hygienist is an indispensable aid to the periodontist in preventive treatment, but a thorough examination of the occlusion and gingival crevice is necessary.

In a subsequent article, Chace (1967) indicated that the general dentist who participates in the maintenance of periodontal patients assumes far greater responsibilities than he does in the care of the average patient. This requires an understanding of periodontal pathology, basic periodontal procedures, techniques necessary for the use of fine curets, and time demands of preventive treatment. The general dentist must be aware of the tendency of the development of new periodontal lesions, just as the periodontist must be aware of patients' needs relative to total dental care.

Chace (1977) also suggested that patients treated for periodontal disease may be susceptible to recurrent periodontitis. Some patients tend to have recurrences despite exemplary care, necessitating retreatment. Reasons for regression must be thoroughly evaluated and may include oral hygiene regimen, surgical technique, occlusal factors, and systemic factors. Patients should be prepared psychologically and informed of the possibility of additional future treatment. Surgical retreatment should be performed if indicated, but if the deepened crevice does not bleed when probed and is not accompanied by bone loss surgery is not justified.

Based on epidemiologic studies, Schick (1981) noted that a maintenance program should provide adequate therapy for previously existing periodontal conditions. Initially, the patient should be provided with a thorough prophylaxis and complete reinforcement instructions in oral hygiene procedures every 3 months. The 3-month interval should be increased, maintained, or decreased depending on an evaluation of the stability of the supporting structures. Close monitoring will indicate the appropriate time interval for each individual patient, and if necessary retreatment determined for those areas that may be deteriorating.

Schallhorn and Snider (1981) reviewed practical management of periodontal maintenance and performed a time study to determine how the therapist's time is actually spent during a maintenance visit. The authors indicate that prevention of periodontal disease occurs at 3 levels: preventing the inception of disease; preventing progression of existing disease; and preventing recurrence of disease following treatment. The authors indicated 4 types of periodontal maintenance therapy (PMT): preventive PMT; trial PMT (allows time for decisions regarding definitive therapy); compromise PMT (i.e., palliative maintenance); and post-treatment PMT (provided to prevent disease recurrence). The authors state that most periodontal therapy should include a 3-month recall but that intervals may range from 1- to 6-months, with the typical appointment taking 52.61 minutes. Factors influencing the maintenance interval include oral hygiene, level of calculus formation, and various host factors.

Lang et al. (1986) evaluated bleeding on probing (BOP) as a predictor for the progression of periodontal disease in 55 patients following treatment and at least 4 years of maintenance at 4 to 5 month intervals. The incidence of bleeding on probing (BOP) during the last 4 recall visits was calculated for all sites of all teeth. Out of 7,704 teeth, 1,054 pockets were selected and subdivided into 5 groups according to the incidence of BOP of 4/4 and 3/4 were selected, interproximal sites with a BOP incidence of 2/4, 1/4, and 0/4 were chosen. These categories were grouped according to attachment level at the time prior to last 4 recall visits. Clinical attachment loss (CAL) was defined as $\geq$ 2 mm. Results indicated that pockets probing $\geq$ 5 mm had a significantly higher incidence of BOP and that 196 (2.5%) had sites BOP 75% to 100% of the time. Patients with 16% of more BOP sites had a higher chance of CAL. The percent of pockets with CAL was 30% when the incidence of BOP was 4/4; 14% when BOP was 3/4; 6% when BOP was 2/4; 3% when BOP was 1/4; and 1.5% when BOP was 0/4. Sensitivity and predictability calculations revealed that BOP is a limited, yet useful, prognostic indicator in clinical diagnosis for patients in maintenance phase. Initial therapy included debridement with OHI, followed by flap curettage procedures and preventive maintenance therapy. A total of 55 (12%) teeth were lost with an average survival rate of 8.8 years prior to extraction. The majority of teeth lost were maxillary second molars (38.2%), followed by maxillary first molars (25.5%), and mandibular second molars (16.4%). Tooth loss patterns were bilaterally symmetrical. The majority of the patients demonstrated above average compliance for oral hygiene and frequency of recall. The authors concluded that teeth with questionable prognosis can often be retained for many years with maintenance and compliance.

COMPLIANCE

Wilson et al. (1984) evaluated 961 patients from a private periodontal practice for compliance with suggested maintenance schedules over a period of 8 years. Patient compliance was encouraged by informing them of the importance of maintenance, notifying either by telephone or mail to schedule an appointment. Of these patients, only 16% complied with recommended maintenance schedules, erratic compliance was found in 49% of the patients, and 34% never reported for any maintenance therapy. It is suggested that as a result the patient's past history of compliance may modify the therapeutic approach employed.

Wilson reviewed compliance (1987A), noting that the medical literature suggests that patients with chronic illnesses tend to comply poorly, especially if the disease is not perceived to be threatening. The reasons for non-compliance are highly variable from fear, economics, and lack of compassion from the dental therapist. In periodontics, the focus is on the effectiveness of oral hygiene and on maintenance therapy. It is not that patients comply better when they are informed and positively reinforced, and when barriers to treatment reduced.

Wilson et al. (1987B) reported on a group of 162 maintenance patients from a previous study (Wilson et al., 1984) for tooth loss over a 5-year period. The group was divided into 2 groups, the compliant (58) and the erratic (104). All tooth loss had occurred in the erratic group (0.6) and it noted that the more often a patient presented for maintenance, the less likely he was to lose teeth.

Mendoza et al. (1991) identified 637 patients from their records as being compliant or non-compliant based on recommended SPT visits. Results indicated that there was no difference in compliance between sex, age, or disease severity. More non-compliant patients were smokers, whereas compliant patients had more periodontal surgery during treatment. Of the patients, ~ 30% failed to return for their first recall appointment and another 12% ceased SPT during the first year, resulting in an average non-compliance of 42.8% for the first year. Attrition rate decreased in subsequent years to average 10% of those remaining in each year, indicating that patients are more likely to remain compliant if they continue SPT at least 1 year. A questionnaire was sent to non-compliant patients, with 40% of the patients replying. The most common reason for their non-compliance was that a general dentist was attending to their needs; many considered SPT too expensive, while others thought they no longer required treatment.

Six-hundred and four (604) periodontal patients undergoing SPT were evaluated by Wilson et al. (1993) and the results of efforts at improving compliance in a private periodontal practice assessed. These efforts included attempts at simplifying compliance, maintaining records of compliance, informing patients of the consequences of non-compliance and attempting to identify non-compliers before active periodontal treatment. Results indicated that 32% were complete compliers, 48% were erratic, and 20% were non-compliers. The main finding of the 1993 study was an increase in complete compliance from 16% in the 1984

TABLE 2. RESPONSE PATTERNS OBSERVED IN STUDIES OF PERIODONTAL TREATMENT IN PRIVATE PRACTICE

Study	Response Pattern	Teeth Lost	Percentage of Patients	Number of Patients
Hirschfeld and Wasserman (1978)	WM	0-3	83.2	499/600
	DH	4-9	12.6	76/600
	EDH	10-23	4.2	25/600
McFall (1982)	WM	0-3	77.0	77/100
	DH	4-9	15.0	15/100
	EDH	10-23	8.0	8/100
Goldman et al. (1986)	WM	0-3	62.0	131/211
	DH	4-9	28.0	59/211
	EDH	10-23	10.0	21/211
Meador et al. (1985)	SI	0	10.48	65/620
	SS	1-4	61.45	381/620
	UR	>4	27.10	168/620
	UU	All	0.97	6/620

WM = well maintained; DH = downhill; EDH = extreme downhill SI = stable ideal; SS = stable satisfactory; UR = unstable retreatable; UU = unstable unsatisfactory

report (Wilson et al.) to 32%. This increase was at the expense of the non-compliant group, and largely due to the efforts of the office. It was suggested that increased recognition and better patient education can help reduce the problem of noncompliance.

Schmidt et al. (1990) studied patient compliance in 631 patients who had undergone active periodontal therapy, consisting of S/RP with (65.9%) and without periodontal surgery (34.1%). Results demonstrated excellent compliance (95%) to suggested maintenance recall. Both full (75 to 100%) and erratic compliers (< 75%) were able to maintain periodontal health over time, but erratic compliers required more surgical retreatment. Smokers exhibited poorer oral hygiene, more tooth loss, and deeper probing depth compared to non-smokers. The percentage of compliance did not appear to change over time; however, the less frequently patients were required to come for recall, the greater their compliance: ~70% of the patients were on 3-month recall and 30% were on 4-month. Patients who alternated recall with their general dentist maintained their periodontal health as well as patients seen only at the periodontist's office.

Patients' response patterns in private practice are shown in Table 2.

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CHAPTER 11. SPLINTING

DEFINITIONS

Splint: Any apparatus, appliance, or device employed to prevent motion or displacement of fractured or movable parts.

Dental Splint: An appliance designed to immobilize and stabilize loose teeth.

Occlusal Trauma: An injury to the attachment apparatus as a result of excessive occlusal force.

Primary O.T.: Injury resulting from excessive occlusal forces applied to a tooth or teeth with normal support.

Secondary O.T.: Injury resulting from normal occlusal forces applied to a tooth or teeth with inadequate support.

Occlusal Traumatism: Injury to the periodontium resulting from occlusal forces in excess of the reparative capacity of the attachment apparatus.

Traumatogenic: Capable of producing a wound or injury.

RATIONALE AND CLASSIFICATION

Lemmerman (1976) reviewed the rationale for splinting and defined a splint as an appliance for immobilization or stabilization of injured or diseased parts. Splints were classified as temporary, provisional, or permanent on the basis of duration and purpose. Temporary splints are those which are used less than 6 months during periodontal treatment and may or may not lead to other types of splinting. Provisional splints may be used from several months to years for diagnostic purposes, and usually lead to more permanent types of stabilization. Permanent splints are worn indefinitely and may be either removable or fixed. Lemmerman also described the concept of reversible and irreversible mobility. Reversible mobility is found in a relatively normal periodontium that is capable of recovery following therapy. Irreversible mobility is observed in a reduced periodontium. This type of mobility may be reduced, but cannot be eliminated. The rationale for splinting is based primarily upon the intended purpose. Other considerations include whether or not the periodontium is healthy or diseased and whether there is a need to prevent mobility or drifting. The author suggests that the rationale for splinting might include cases of post-acute trauma, prevention of drifting in normal dentitions during occlusal therapy, or to provide functional comfort by preventing mobility in diseased dentitions.

Ferencz (1991) reviewed splinting and noted that there is little rationale for splinting teeth manifesting primary occlusal trauma. Splinting during or after periodontal treatment is often useful in controlling the effects of secondary occlusal trauma and in instances where decreased mobility may improve comfort and function. Mobility may inhibit periodontal repair during therapy and therefore provides an additional rationale for splinting. The author further classified splints as short-term, provisional, and long-term. Short-term splints can be extracoronal (fixed or removable) or intracoronal (composite, wire with composite or amalgam). Provisional splints are designed to protect and stabilize teeth during therapy when definitive splinting with fixed restorations is planned later. Long-term splints may be fixed, removable, or a combination of both. According to the author, fixed splinting will provide the most effective means of long-term support.

Lindhe and Nyman (1977) presented an extensive review of the etiology and rationale for treatment of periodontal disease with a discussion of occlusal adjustment. The authors' main objectives for occlusal adjustment and splinting were control of progressive mobility and re-establishment of a narrow periodontal ligament space. They also felt that the presurgical phase of therapy may be useful in differentiating between occlusal trauma and infrabony pockets due to periodontal disease.

METHODS OF SPLINTING

Pollack and Ponte (1981) described methods of non-crown and bridge splinting, including specific techniques for non-precious metal splinting. These included an extracoronal acid-etch technique, pin composite splints, and pin amalgam splints. The extracoronal acid-etch technique uses 16-gauge stainless steel wire. The pin composite splints utilize TMS minim pins placed in the gingival floors of adjacent Class III preparations. The pins are bent across the contact areas and composite is placed and finished around the pins. The pin amalgam splint is similar to the composite technique, with TMS minim pins bent across the contact areas of two adjacent Class II amalgam preparations. Toffflemier retainers with interproximal cut-outs are placed around the teeth and amalgam is placed and carved, leaving the coronal aspects of the restoration bridging the contact area.

Chalifoux (1991) discussed splinting of anterior teeth using composite bonding, intracoronal wire, extracoronal mesh, metal bonding, and crown and bridge techniques. Composite bonding techniques use micro- or macrofilled composite with a standard acid-etch technique to bond contact points. It is indicated for slightly mobile teeth with few restorations, but has low overall strength. The intracoronal wire technique resembles the composite technique. A groove is prepared across the lingual tooth surfaces and 22-to 28-gauge wire is adapted into the groove and held into position with composite. This splint is used most often for maxillary teeth with

moderate mobility and small restorations. With the extracoronal mesh technique, wire mesh is adapted to the teeth and is bonded using composite. This splint is useful in areas with moderate mobility and provides moderate strength without being invasive tooth preparation. In the metal bonding technique, an acid-etched metal framework is constructed in the laboratory and bonded to the teeth. It is used in areas with moderate mobility and small restorations. Problems with this technique arise when 1 or 2 teeth become unbonded, compromising the splint. Fixed partial denture prostheses may be used for very mobile teeth, teeth with large restorations, or both.

Saravanamuttu (1990) described a non-rigid, intracoronal splint for periodontally involved teeth that have received orthodontic treatment. In this instance it was considered desirable for the teeth to have a degree of functional loading for the reformation of a healthy periodontium. The technique involved preparing a groove on the lingual surfaces of the teeth and adapting an annealed, multi-strand (flexible) orthodontic wire. The wire was secured to the teeth with composite, ensuring that the interproximal areas remained unbonded.

Gerstein and King (1975) reviewed the use of removable partial denture splints for the treatment of mobility. The appliance referenced was designed by Kratochvil and used specific design criteria to promote maximum stability and cleansability. Occlusal rests should extend completely through the center of the occlusal surface in a mesio-distal direction at a depth of 1.5 mm. Interproximal guide planes provide bracing and help eliminate tooth-prosthesis voids. Major connectors should be kept as far away from gingival margins as anatomically possible. Minor connectors should be strong and cleansable. The "I" bar clasp is recommended because it engages a more physiologic degree of undercut, is a cleaner clasp arrangement, and places less unfavorable forces on the abutment when torquing forces are produced by the partial denture. Removable splints were promoted due to better access for plaque removal when compared to fixed splints. Other advantages of the removable splints include less tooth preparation, pulpal trauma, chairside time, clinical effort, and expense.

Rudd and O'Leary (1966) determined that a specially-designed removable partial denture using multiple parallel guiding planes and bracing clasps with a rigid major connector could be used as a removable splint to stabilize periodontally weakened teeth. Such partial dentures require extremely meticulous preparation and technical expertise for "precision" design. Prior to periodontal therapy, the mean mobility of the 219 teeth tested was 0.0115 mm. This mobility was reduced to 0.0104 mm after periodontal therapy and further reduced to 0.0096 mm after 1 to 3 months of removable splint insertion. Although the time periods for the study were short, the authors concluded that precision designed partial dentures may be effective in stabilizing mobile teeth.

EFFECTS OF SPLINTING

Using five Rhesus monkeys, Glickman et al. (1961) studied the effects of splinting teeth in hyperocclusion. The authors observed that forces applied to 1 tooth in a splint were transmitted to all teeth within the splint. The direction of the initial force was maintained and comparable areas of the splinted periodontium were affected. The bifurcation and trifurcation areas were most susceptible to excessive force. Forces applied to non-splinted teeth were not transmitted to adjacent teeth and force sufficient to cause necrosis did not cause pocketing.

Mandel and Viidik (1989) used Vervet monkeys to study the effects of rigidly splinting anterior teeth that had been extruded 3 mm and replaced into the socket. Two weeks after post-extrusive healing, no significant differences were found between splinted and non-splinted teeth in terms of periodontal ligament (PDL) width or stress and strain values of the PDL. Within 2 weeks, the injured PDLs had regained 50 to 60% of the shear and strain values noted in non-injured teeth. Rigid splinting of the luxated teeth did not improve the mechanical properties of the PDL during healing.

Rateitschak (1963) studied the effect of initial preparation and occlusal adjustment on tooth mobility in 80 patients using the Muhlemann periodontometer with a deflective force of 500 gm. The author observed that if the initial mobility was greater than 0.2 mm, initial preparation and occlusal adjustment decreased tooth mobility up to 20%. Orthodontics or removable splints caused an initial increase in the mobility which returned to baseline by 2 years.

Nyman et al. (1975) studied 20 patients who had originally exhibited severe periodontal breakdown and extensive tooth loss. Extensive fixed bridgework was placed following periodontal therapy and the patients monitored for 2 to 6 years. No further bone loss was observed between the insertion of the fixed bridgework and the final examination. The authors reported no increase in PDL width of the abutments or changes in mobility.

Renggli et al. (1984) studied the use of telescoping bridges placed 3 to 4 months after surgical therapy that could be removed by the patient on a daily basis for access to hygiene. The authors noted that the telescoping bridges reduced mobility during an initial 4-week period when they were not removed and for an additional 6 weeks, when the bridges were removed daily. The mobility of non-splinted control teeth was also reduced at the 4- and 10-week periods. The reductions in mobility were not significantly different among the 2 groups. The authors suggest that the reductions in mobility may have been due to the establishment of a harmonious occlusion and not necessarily due to splinting.

Kegel et al. (1979) studied posterior tooth mobility following scaling and root planing, occlusal adjustment, and oral hygiene education in splinted and unsplinted teeth utilizing 7 patients in a split-mouth design. The splints were

removed at measurement times and then replaced during the experimental study which lasted 17 weeks. The authors found no significant difference between splinted and non-splinted segments with regard to tooth mobility, gingival bleeding, attachment level, or radiographic bone scores. Teeth that were initially more mobile received no significant benefit from splinting when compared to initially less-mobile teeth.

Galler et al. (1979) used a similar design to study the effects of splinting upon mobility during osseous surgery. Using maxillary teeth in a group of 10 patients, 1 quadrant was splinted following osseous surgery and the other was not. During a follow-up period of 24 weeks, it was noted that splinting had no effect on mobility at any time. An overall average of 0.6 mm of bone was removed per tooth with osseous resection and no significant correlations existed between the amount of bone removed and the change in tooth mobility for either pooled splinted or unsplinted teeth. Also, postoperative mobility seemed more dependent upon preoperative mobility than on the treatment method. Splinting had no effect on attachment level or alveolar bone height.

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CHAPTER 12. OCCLUSION

Section 1. Temporomandibular Disorders

DEFINITIONS

Temporomandibular Joint (TMJ): The connecting sliding hinge mechanism between the mandible and the temporal bone (base of the skull).

Temporomandibular Disorders (TMD): A group of disorders including myofascial pain-dysfunction syndrome (MPD), disk (meniscal) displacement with or without reduction (internal derangement), degenerative joint disease (osteoarthritis), rheumatoid arthritis, and other disorders of systemic origin, facial growth disharmonies, traumatic injuries, and neoplasms.

Internal Derangement: An abnormal relationship of the articular disk to the mandibular condyle, fossa, and/or articular eminence.

Meniscal Displacement with Reduction: The condition wherein the articular disk is located anterior or antero-medial to the condylar head when the jaw is closed. When the jaw is open the disk assumes the normal relationship to the condylar head and articular eminence.

Meniscal Displacement without Reduction: The condition wherein the articular disk is always located anterior or antero-medial to the condylar head regardless of whether the jaw is open or closed.

Myofascial Pain Dysfunction Syndrome (MPD): A group of symptoms including pain and dysfunction involving the muscles of mastication and upper body posture. Considered to be a psychophysiologic disorder.

BASIC ANATOMY

The human TMJ is functionally defined as a ginglymoarthrodial (hinge-glide) articulation. A brief synopsis of the joint's basic anatomy follows. The condyle, glenoid fossa, and articular eminence of the temporal bone comprise the bony boundaries of the TMJ. The condyle is elliptical in shape with its mediolateral diameter being greater than its anteroposterior diameter. It is oriented in the fossa with slight medial rotation. The joint is divided into upper and lower compartments by the disk which is attaches to the medial and lateral aspect of the condyle. The capsule of the TMJ consists of the strong lateral temporomandibular ligament and 2 weaker medial ligaments. The sphenomandibular ligament attaches to the lingula on the medial aspect of the mandible and the stylomandibular ligament attaches to the posterior border of the ramus (Figure 1).

The joint is innervated by the auriculotemporal nerve and masseteric branches of the trigeminal nerve (V.3). The blood supply derives from the superficial temporal, middle meningeal, and other associated branches of the internal maxillary artery.

Articular Disk (Meniscus)

The articular disk (meniscus) of the TMJ is a dense, pear-shaped fibrous structure with an inner concave and outer convex surface composed of 4 zones (Figure 2). These zones consist of a moderately thick anterior band, a thin narrow intermediate band, a posterior band which is the thickest area of the disk, and a retrodiskal zone (bilaminar zone) (Rees, 1954). The condyle articulates on the intermediate band.

The central (intermediate) area of the articular disk is avascular. The posterior attachment of the disk, sometimes referred to as the retrodiskal pad, contains numerous large vascular spaces and elastic tissue. The disk appears to have limited capacity for repair and the pathologic changes are usually degenerative (Blackwood, 1969).

Medially and laterally, the articular disk blends with the capsule (which surrounds the disk) and attaches to the condyle. Anteriorly, it attaches to the articular eminence, condyle, and superior head of the lateral pterygoid. Posteriorly, there are firm retrodiskal tissue attachments to the glenoid

Figure 1. Basic anatomy (articulation and capsule)

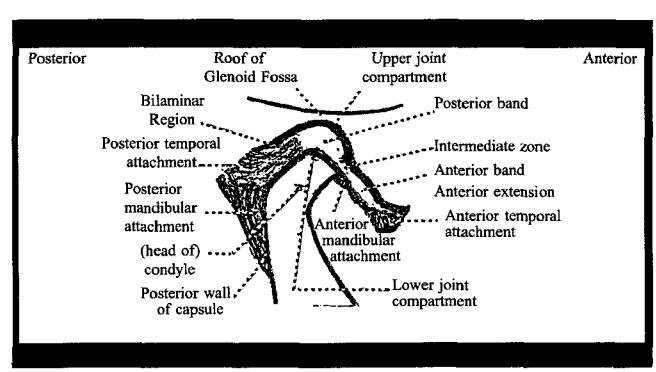
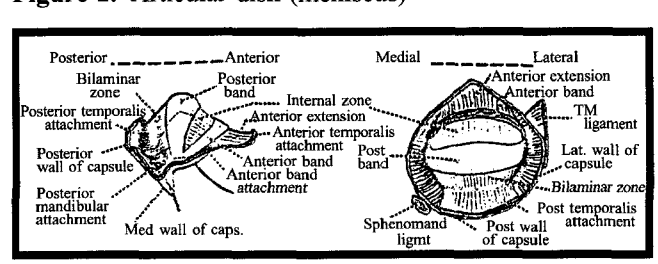


Figure 2. Articular disk (meniscus)



Post = posterior; Ant = anterior

fossa, squamo-tympanic suture, and condyle. Anteriorly, the capsule is reinforced by the temporomandibular ligament, a dense, collagenous ligament without elastic fibers, which functions to maintain contact between articular surfaces and limit extreme movement. No attachment is observed medially.

Mandibular Condyle Histology

There are 3 distinct cell zones in the cartilage of the mandibular condyle during growth: articular zone, proliferative zone, and hypertrophic zone. The proliferative and hypertrophic zones manifest cartilage replacement by mineralized bone, similar to other growing bones. The surface of the articular zone is renewed independent of the proliferative zone, and this difference is reflected in the response of this zone to some of the pathologic processes occurring in this area. With age, the articular zone becomes increasingly fibrous, the proliferative zone is reduced to a narrow band of cells, and the hypertrophic zone is replaced by fibrocartilage (Blackwood, 1969).

Articular Remodeling

Joint remodeling may be progressive, regressive, and peripheral. Progressive and peripheral remodeling constitutes the addition of tissue to the joint surfaces. Peripheral remodeling occurs primarily at the edges of the articular surfaces and occasionally involves the periosteum. Progressive and peripheral remodeling accompanies cellular activity in the proliferative zone, resulting in new cartilage formation and increasing vertical dimension. Regressive remodeling results from osteoclastic resorption of the subarticular bone and replacement with new cartilage and bone, lowering the vertical dimension. During remodeling, the articular surfaces of the joint remain intact and play a relatively passive role by adapting to the changes taking place in the tissues beneath (Blackwood, 1969).

PHYSIOLOGY

There is an average maximum forward movement of 8 mm between the condyle and meniscus, and an average maximum forward movement of 7 mm between the meniscus and temporal bone. As the condyle moves forward, the meniscus is displaced downward and forward, with forward movement due to pull of the lateral pterygoid muscle and attachments to the condyle. During movement, the condyle actually rotates on the disk. The disk is allowed to move forward 7 to 10 mm due to fibro-elastic connective tissue in the posterior attachment which "recoils" on closing (Rees, 1954). Movement between the lower joint structures is rotational while translation occurs between the structures of the upper joint (Okeson, 1991).

CLASSIFICATION OF TMJ DISORDERS

In 1934, Costen associated a number of symptoms, including a variety of ear and sinus problems (impaired hearing; stuffiness; tinnitus; dizziness; headache burning sensation of throat, tongue, and side of nose) with a dysfunction of the TMJ. Laskin (1969) noted that it took almost 20 years to clarify many of the inaccuracies of this theory which slowly developed into what was termed the "TMJ syndrome." This description eliminated many unrelated symptoms, but still grouped patients with pains of undetermined origin under the same diagnosis. In 1955, Schwartz identified a more definitive subgroup of patients who presented with painful, limited mandibular movement. He attributed this to spasms of the musculature and termed it "temporomandibular joint pain-dysfunction syndrome." Laskin indicated that Schwartz's studies greatly influenced the thinking in this area, resulting in the first major shift away from the narrow mechanical concept of occlusal etiology. Treatment principles included reduction of stress, pain and spasm, therapeutic exercise, and possible judicious occlusal alteration.

In 1983, the American Dental Association convened a special conference of TMJ experts to establish guidelines regarding the examination, diagnosis, and management of temporomandibular disorders (TMD). It was this committee's opinion that temporomandibular disorders should discontinue such terms as "temporomandibular joint dysfunction" and use descriptive terminology. The group agreed that TMD could be classified under three broad headings: those occurring primarily in the muscles of mastication, the TMJ, or related areas mimicking temporomandibular disorders (Griffiths, 1983).

In 1987 and 1989, ADA-sponsored workshops on TMD discussed controversial issues, including TMD classifications. At the 1989 conference (McNeil et al., 1990), TMD was defined as a cluster of related disorders of the masticatory system with common features such as pain, headaches, earaches, joint sounds, and limited mandibular motion. The workshop seemed to approve a classification established by the International Headache Society and the American Academy of Craniomandibular Disorders. The major headings included TMD (disk displacement with and without reduction, hypermobility, dislocation, synovitis, capsulitis, osteoarthritis, osteoarthrosis, and ankylosis) and craniofacial muscle disorders. Currently, however, the masticatory muscle disorders (myofascial pain, myositis, spasm, reflex splinting, hypertrophy, etc.) usually fall under the heading of TMD as well.

INTRINSIC TMJ-ASSOCIATED DISORDERS

An important aspect of diagnosis of TMD includes differentiating disorders originating from intracapsular sources (internal derangements) from those of extracapsular sources (i.e., muscles). Internal derangements or disk-interference disorders, as classified by Okeson in 1991, include: disk displacement or dislocation with reduction; disk dislocation without reduction; adhesions; and alterations in form.

Stockstill and Mohl (1991) reviewed evaluation of TMJ sounds including diagnostic analysis and clinical implications. Joint sounds are often caused by internal derangements but also may be present in "normal" joints as well

as other conditions such as TMJ laxity or condylar movement beyond the eminence. Clicking is the most common joint sound and is caused by the posterior movement of an anteriorly positioned disk or the passage of the condyle against the posterior aspect of the disk during reduction of the disk during jaw opening. Reciprocal clicking produces a similar sound near maximum intercuspation when the disk recoils anteriorly. The authors note that these 2 clicks do not occur at the same jaw position. Crepitus is a grating sound caused by rubbing of irregular articular surfaces or dry synovial surfaces of the joint often associated with degenerative joint disease. The variability in joint sounds and the lack of characterization of sounds for particular disorders limit the importance of TMJ noises unless other signs and/or symptoms of TMD are present.

In 1972, Farrar discussed the diagnosis and treatment of anterior dislocation of the disk indicating that 5% of the patients with TMJ complaints manifest anterior dysfunction. Clinically, there was limitation of opening (24 to 32 mm) with deviation toward the affected side. He stated pain was variable and not necessary for diagnosis. Loss of posterior tooth support on the affected side was a common finding. He felt transcranial radiographs were beneficial in assessing condylar position and joint spaces and thus useful in diagnosis. The 1983 President's Committee concluded such imaging for assessing internal derangements was not reliable and should not be routine (Griffiths, 1983). McNeil et al. (1990) suggest that TMJ imaging is only indicated if a recent or progressive pathological joint condition exists and is not indicated for joint sounds in the absence of other TMD signs and symptoms. Interpretation of TMJ imaging remains a problem. Although technological advances allow imaging using tomography, computed tomography, and magnetic resonance imaging, none of these techniques can predictably differentiate between adaptation and pathologic changes. Prior to the expense and radiation of most imaging techniques, a high probability should exist that the findings of the examination will aid significantly in diagnosis and treatment selection (Dixon, 1991). Farrar's treatment recommendations included manual jaw manipulation to reduce recent disk displacement. If the displacement was painful or had existed for days or weeks, a flat, hard, acrylic bite plane was worn for a few days and then an anterior repositioning splint to move the mandible forward until the disk returned to its normal position (recapturing the disk). This was followed by occlusal equilibration or reconstruction in 4 to 6 weeks.

Anterior repositioning appliances are still being used, although not without controversy. Only 25 to 36% of the patients had long-term reductions of pain and dysfunction (i.e., clicking). Posterior open bites were found to be a problem and occlusal equilibration or rehabilitation was costly, time consuming, and difficult. "Stepping back" the mandible to the original occlusal position to avoid these procedures often proved effective in recapturing the disk. For these reasons, anterior repositioning appliances should be used with discretion and only if initial treatment with a stabilization appliance (splint) proves unsuccessful (Okeson, 1991).

DISORDERS ASSOCIATED WITH THE MUSCLES OF MASTICATION

In 1969, Laskin stated that the diagnosis of pain dysfunction syndrome is based on 6 signs or symptoms: 1) pain of unilateral origin; 2) muscle tenderness; 3) TMJ clicking or popping; 4) limited jaw function; 5) lack of tenderness in the TMJ when palpated via the external auditory meatus; and 6) absence of organic changes in the TMJ. Guralnick et al. (1978) also listed these symptoms, considering the first 4 as "cardinal signs" of myofascial pain dysfunction (MPD). DeBoever (1980) included ear symptoms, noting their presence in 25 to 37% of the patients, with jaw deviation to the affected side upon opening. The controversy over the importance of occlusion versus the impact of psychosocial stress persists; however, most authorities have discounted the role of occlusal discrepancies. In 1976, Rugh and Solberg supported a psychological component to TMJ pain and dysfunction, but studies were unable to correlate TMD with 1 specific personality trait. There is relatively strong evidence, however, that MPD patients exhibit more emotional anxiety than the average population. Grzesiak (1991) speculated that a unique psychological profile may predispose for MPD, citing a study in which 62% of TMD patients sought care after a significant life event caused a stress response that aggravated a physical condition. Increased masticatory muscle activity, tension, and pain have been related to periods of subjective stress.

BRUXISM

Bruxism is defined as the clenching or grinding of the dentition during non-functional movements of the masticatory system. During sleep, it is termed nocturnal bruxism and during waking hours is called diurnal parafunction. Diurnal parafunction also includes oral habits such as nail biting, finger sucking, tongue thrusting, and chewing on various objects. Most individuals demonstrate signs of bruxism but only 5 to 20% are aware of their parafunctional activity. Simply relying on the patient's history may not be sufficient for diagnosis of nocturnal bruxism. The occlusal wear pattern in a bruxer will result in facets that align while wear facets associated with mastication do not. Nocturnal bruxism has been related to periods of daytime emotional and physical stress and is currently, considered a stress-related sleep disorder. Evidence indicates there is no direct relationship with occlusal interferences, although the etiology is probably multi-factorial and occlusion could play a minor role in certain cases (Attanasio, 1991). Tooth wear, fractured cusps, injury to the periodontium with hypermobility of teeth, hypercementosis, and pulpitis may result from bruxism. Painful masticatory musculature with limitation in mandibular motion is common as is hypertrophy of

the masseters and muscle tension headache pain. Initial therapy should rely on reversible procedures including interocclusal appliances, stress management, physical therapy, pharmacological muscle relaxants, and/or occlusal adjustment (Attanasio, 1991).

CONSERVATIVE TREATMENT FOR TMD

Treatment for TMD should start conservatively, be reversible, and proceed based on need. Early treatment may include patient education and counseling, habit management, physical therapy, bio-feedback, bite-splint appliances, short-term analgesics, and/or muscle relaxants. The diagnosis and etiology will be the determining factor of therapeutic choice. The influence of placebo effect should be noted when treating TMD patients (McNeil et al., 1990).

Greene and Laskin (1983) compared the initial and long-term results of conservative, reversible therapy to 10 previously published papers, regarding irreversible procedures. Patients (175) treated for MPD were interviewed by phone, 1 to 11 years (5-year mean) following completion of treatment. Treatment modalities were variable (oral appliance, biofeedback, psychological counseling, medication, transcutaneous nerve stimulation, placebo), but all were reversible and required some degree of patient cooperation and participation. Immediately following treatment, 74% had been greatly improved, 25% had minor or no improvement, and 1% worsened. Subsequently, 42 patients had sought further treatment. Of this group, 43% reported no additional improvement. However, this group reported gradual improvement after discontinuing therapy. When phoned, 90% of the patients were doing well (53% asymptomatic, 37% minor symptoms), 8% were no better than before treatment, and 2% were worse. In general, patients expressed satisfaction. Two major conclusions resulted from this study. First, those who initially responded well to conservative treatment continued to do well for an extended period of time (whether they received real or placebo treatment). Second, more than half the patients who initially failed to respond to treatment eventually improved. The success rate of this study is similar to previously reported long-term studies. This demonstrates general effectiveness of MPD treatment with no single treatment modality required for success. The authors concluded that irreversible treatments are not necessary for success and are inappropriate.

Clark (1984) reviewed the 5 splint designs including: 1) soft resilient appliances; 2) anterior bite plane hard acrylic splints; 3) full coverage hard acrylic appliances; 4) dynamic exercise or spring-loaded appliances to place opening traction forces on the TMJ of patients with ankylosis, fibrosis and adhesions; and 5) repositioning splints. The author also identified and explained 5 splint theories: 1) occlusal disengagement; 2) restored occlusal vertical dimensions; 3) maxillomandibular realignment; 4) TMJ repositioning; and 5) cognitive awareness. No conclusive and controlled testing of these theories has been accomplished.

Baragona and Cohen (1991) presented a concept of long-term orthopedic appliance therapy. One form of therapy uses a maxillary splint at night and a mandibular splint during the day. Gradual withdrawal of the splint is begun after 1 to 3 asymptomatic months. If patients cannot function without some form of craniomandibular stabilization, if there is a return of painful symptomatology, or if the patient is not a candidate for more aggressive, irreversible treatment, splint therapy may continue indefinitely.

SURGICAL THERAPY

According to Hoffman et al. (1991), approximately 1 to 3% of the population requires or seeks surgical treatment for dysfunctional TMD. Basic indications for arthroscopic surgery include internal joint derangements, condylar hypermobility secondary to intra-articular disorders, foreign bodies, degenerative disease, disk perforation, or radiographic evidence of intra-articular diseases. Treatment includes lysing of adhesions, stretching the fibrotic lateral capsule, and releasing fibrotic attachments of the capsule to the lateral eminence. The joint is lavaged and inspected for mobility. Eminoplasty and plasty of the disk may be accomplished. Physical therapy should commence within 48 hours of arthroscopy. More specific indications for open arthroplasty include osteophytes on the condyle or fossa, ankylosis, implant failure, or cranial perforation. Disk removal may be necessary with or without replacement with alloplastic materials, cartilage grafts, dermis grafts, or temporal fascia. The authors suggest that clinical success in treating joint derangements does not depend on repositioning the disk, but rather, mobilization of joint tissues and removing fibrous tissue to improve disk mobility

SYSTEMIC PROBLEMS AND CONDITIONS WHICH MIMIC TMD

TMD is responsible for most orofacial pain (Fricton, 1991), but the differential diagnosis of chronic orofacial pain includes a wide variety of other disorders. Chronic pain is pain of 6 months' duration, is not self-limiting, serves no biologic purpose, is often therapeutically nonresponsive, and is frequently intensified by psychologic factors (Pertes and Heir, 1991; Grzesiak, 1991). In contrast, acute pain is temporary in nature, often self-limiting, serves a biologic signal function, has a specific cause, and responds to therapy (Fricton, 1991; Pertes and Heir, 1991). The patient with chronic pain often suffers from depression and anxiety and may develop dependence on medications, health care professionals, and surgical procedures. Dependence on family and friends can result in low self-esteem. Grzesiak (1991) describes 4 psychologic models of symptom formation in chronic pain (psychodynamic, behavioral, cognitive-behavioral, and cognitive-psychological). The author indicates that the psychological factors are so intermingled with biologic factors that a multidisciplinary approach to ad-

dress both areas is essential for effective management of chronic TMD patients.

Differential diagnosis of chronic orofacial pain may be complex. Pertes and Heir (1991) proposed 3 major pain categories. On the basis of history and physical examination, the primary diagnostic pain category and tissue system affected may be identified. These pain categories are described as somatic, neurogenic, and psychogenic. The 6 tissue systems affected include the following: extracranial, intracranial, musculoskeletal, vascular, neurologic, and psychological. A synopsis of chronic oro-facial pain based on these tissue systems is described briefly in the following summary (Pertes and Heir, 1991; Graff-Radford, 1991; Austin and Cubillos, 1991).

Extracranial. This includes dental pain of odontogenic origin and sinusitis. Dental pain must be differentiated from non-dental causes such as sinusitis, myofascial trigger points, vascular headaches, neuropathies, neuritis, atypical odontalgia, and cardiac pain.

Intracranial. Pain secondary to an intracranial neoplasm, aneurysm, hematoma, hemorrhage, or edema. For patients over 40 years of age with any new persistent headache or a change in chronic headache pattern, a referral to a physician is advised even though this is an uncommon cause of orofacial pain.

Musculoskeletal. This is deep somatic pain that may be caused by MPD or internal derangements. Diagnosis is aided by palpation of trigger points. Cervical spine disorders are also included in this group.

Vascular. These disorders include a variety of headaches including migraine, cluster (Horton's cephalgia), and tension headaches. Temporal arteritis (TA) is a vasculitis characterized by inflammation and destruction of the vessel wall which may be caused by cell-mediated immune mechanisms. TA is often associated with headaches and ocular complaints. Average age of onset is 70 years with a 2:1 female predilection.

Neurologic. This pain is caused by a functional abnormality within the nervous system. Trigeminal neuralgia (tic douloreux, Father Giles' disease) involves the trigeminal nerve (usually the mandibular branch) and is categorized as idiopathic or symptomatic. Idiopathic trigeminal neuralgia (TN) is characterized by paroxysmal, unilateral facial pain confined to the distribution of the trigeminal nerve. The pain is initiated by stimulating trigger zones through touch, talking, chewing, or yawning. The pain is described as brief flashes of excruciating pain lasting a few seconds to a few minutes. Spontaneous remissions are possible. If evidence of a structural cause, neurologic deficit, or other cranial nerve involvement exists, then TN is referred to as symptomatic. Treatment is pharmacologic (tegretol, baclofen, or phenytoin) or surgical (Austin and Cubillos, 1991). Other neurologic disorders include glossopharyngeal neuralgia, deafferentation pain syndromes (atypical odontalgia, trauma neuromas, and neuritis), and paroxysmal neuralgia. Horton's cephalgia (or cluster headache) has also been included in this category (Lazar et al., 1980). These cluster headaches are characterized by unilateral severe pain which begins in the retro-orbital, forehead, and maxillary regions. These begin suddenly and may increase in intensity for 15 to 180 minutes. Males are affected 6 times more than females.

Psychological. Emotional and psychological factors are the primary cause of the pain complaint with no apparent organic basis.

Miscellaneous Problems

Blackwood (1969) described other problems which may mimic TMD. These include degenerative arthritis, rheumatoid arthritis, growth disturbances, disk lesions, and osteoarthritis. A brief summary of the author's descriptions of these conditions follows.

Degenerative arthritis (DA). This condition may affect up to 100% of patients, especially in weight-bearing joints (knee) of 80- to 95-year-olds, with less incidence reported for non-weight bearing joints. DA is seen in the TMJ in approximately 40% of individuals age 40 and older. Degenerative changes are seen in the bone and cartilage. Ankylosis is seldom the result of degenerative arthritis.

Rheumatoid arthritis (RA). RA is rarely observed initially in the TMJ. However, when present, it usually occurs bilaterally. The active disease involves inflammation of synovial tissues with infiltration of plasma cells and lymphocytes resulting in joint deformity. Little repair is observed histologically.

Growth disturbances. These usually manifest as changes associated with secondary hyperparathyroidism, chronic leukemia, and achondroplasia. All impair endochondral bone formation. Local disturbances in growth may also be seen.

Disk lesions. Perforations may be associated with osteoarthritis or attributed to unknown cause. Tears of the posterior attachment will result in a forward displacement of the disk. Histologic changes are mostly degenerative.

Osteoarthrosis. In 1973, Toller described this degenerative disease of unknown etiology. The 130 cases surveyed in his report were derived from 1,573 cases of TMJ lesions and accounted for 8% of the total. Major signs and symptoms include pain on jaw movement which is often more severe later in the day, limitation of movement, crepitus (not clicking), tenderness over the condyle, radiographic changes at first examination, and, less frequently, aching on the side of the face. The majority of patients had intact dentitions with adequate occlusal support. Females were affected 6 times as often as males and age of onset ranged from 20 to 80+ years, peaking in the fourth to fifth decades. Patients diagnosed with osteoarthrosis did not present with signs of arthritis. Only 5% had a relevant history of trauma. Radiographic changes using a transpharyngeal technique revealed either loss of lamina dura or a cupping excavation of the condyle around the point of contact with the articular

eminence. Although radiographic changes were not always evident at the first visit, a diagnosis of osteoarthrosis was not confirmed until radiographic changes were observed. As severity of symptoms increased or persisted, radiographic changes usually became more severe. Within 2 to 3 years, radiographs revealed rounding of sharp edges and remineralization of the articular surface which often formed at an oblique angle and was reduced in size. Diagnosis of osteoarthrosis should rule out pain dysfunction syndrome, osteoarthritis, and other erosive diseases of bone such as neoplasms. Normally the disease is self-limiting with most cases being symptom-free within 2 years. Treatment is empirical and may include pain alleviation (pain medication, massage, heat), correction of dental deficiencies, and establishment of proper vertical dimension. Crepitus often persists but should not cause concern in a symptom-free patient. While the value and safety of intra-articular steroid injection is questionable, the author recommends a single injection of Prednisolone (25 mg) for relief of acute pain. For cases which do not respond to conservative treatment and in which the patient has intractable pain and lack of function, a condylectomy might be considered. Of 19 cases treated surgically, 13 had excellent results, 3 improved, and 1 was a failure.

SUMMARY

TMD is responsible for most orofacial pain. Characteristic signs include joint noise, muscle tenderness, pain, and limitation of mandibular motion. Common symptoms include facial pain, headache, joint noise, and jaw function difficulties. Multiple factors have a role in TMD including parafunctional habits, stress, trauma, and malocclusion. MPD and disk displacements are the 2 most common TM disorders (Fricton, 1991). MPD is a regional muscle pain disorder characterized by muscle tenderness, slight limitation in range of motion, and local and referred pain. TMJ disk displacements are characterized by progressive stages of clinical dysfunction. Joint alterations usually consist of an anteriorly displaced disk caused by trauma, laxity of the ligaments, and changes in the fluid environment of the joint. Displacement is often associated with TMJ capsulitis, pain, tenderness, and joint swelling. TMD screening should be part of the routine dental examination for all patients but TMJ imaging is indicated only if recent or progressive pathologic joint condition exists based on the clinical examination and history. Imaging is not indicated for joint sounds in the absence of other TMD signs and symptoms. Goals of therapy should include pain reduction, restoration of normal jaw function, and reduction of need for future health care. Treatment should start conservatively and progress as needed.

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Section 2. Occlusion: Theory

REVIEW

Weisgold (1969) reviewed the major concepts of occlusion from the 1920s through the 1960s. A summary of his report appears below. Readers are referred to the 1969 article for full references. In the 1920s, Stillman and McCall recognized a relationship between gingival disease and traumatic occlusion, associating excessive force with the presence of gingival clefts or linear depressions (McCall's festoons, Stillman's clefts). Orban (1939) suggested that traumatized tissue may be at an increased risk for inflammation and that inflamed tissue would yield more easily to trauma, but still stressed the existence of 2 separate periodontal lesions. In a rat model, Macapanpan and Weinmann (1954) noted that trauma caused damage to the periodontal ligament (PDL) and that the inflammatory exudate was directed into the PDL. Glickman and Smulow (1957, 1962) believed that presence of inflammation and occlusal trauma could lead to angular bone destruction. Akiyoshi and Mori (1967) evaluated autopsy specimens and suggested that gingival inflammation from the interdental papilla may extend through transeptal fibers and follow the course of the blood vessels into the interdental septum and the PDL, even if there was no evidence of occlusal trauma. In the late 1920s, gnathologic theories were described, with the true hinge axis being proposed by McCollum et al. Intercuspation in the terminal hinge position was stressed. Schuyler, in the 1930s and 1940s, proposed guidelines of occlusal adjustment, advocating the elimination of balancing side contacts. Youdelis and Mann (1965) also supported elimination of balancing side contacts based on retrospective observations that molar teeth associated with such contacts had greater bone loss, mobility, and pocket depths when compared to molars without balancing contacts.

Posselt (1952) reported that over 90% of subjects had maximum intercuspation anterior and inferior to the retruded position, and that maximum jaw opening required both translatory and rotary movements of the condyle. Jankelson (1953) observed that tooth contact occurs during mastication and swallowing and that centric relation is present during mastication. In the 1960s, Ramfjord advocated occlusal adjustment in centric relation to eliminate bruxism. It was during this same period that Pankey et al. introduced "long centric" as an occlusal relationship.

CENTRIC OCCLUSION VERSUS CENTRIC RELATION

Adams and Zander (1964), using experimental bridges with miniature radio transmitters, questioned the use of centric relation as a desired intercuspation. The authors placed switches at the intercuspation position and lateral to intercuspation. Muscle activity of the temporalis and masseter muscles was also recorded using electromyography. The subjects chewed 3 foods on their left side only, right side only, and in their usual manner. Chewing sequences were analyzed for the number of chewing strokes, number of tooth contacts for each chewing motion, duration of tooth contact, and relation of tooth contact to muscle activity. All subjects presented tooth contacts lateral to intercuspation with all test foods and all 3 chewing motions. Contact frequency increased for lateral and intercuspal positions as chewing progressed. The longest intercuspational contact occurred during swallowing.

Pameijer et al. (1969) placed a telemetric device in a fixed denture appliance to investigate the occurrence and frequency of single and gliding tooth contacts during chewing, swallowing, and bruxism. The authors reported that most chewing and swallowing occurred in the centric occlusion position, with lateral excursions atypical of the chewing cycle. They also noted that bruxism consisted of repetitive grinding tooth contacts which differed from the haphazard pattern recorded during mastication.

Carwell and McFall (1981) evaluated the incidence and position of tooth contacts using chin point manipulation, bilateral mandibular manipulation, and chin point guidance with an anterior jig. Three groups of 10 patients each were analyzed using 2 of the 3 techniques. Assessments included centric relation contacts, anterior and lateral components of the slide from centric relation to centric occlusion, and nonfunctional contacts in lateral excursions. The authors also examined the periodontal status and classified the occlusal relationship as Angle's type, cuspid protected, and group function. The chin point jig technique identified the initial point of contact most accurately, but all techniques correctly identified the location of centric relation contacts. Twenty-six (26) of 30 patients had an Angle's Class I occlusion, 14 of 30 had bilateral cuspid-guided occlusion, 8 of 30 had bilateral group function, and 8 of 30 had a combination of cuspid protected and group function relationships. Maxillary and mandibular first premolars were involved with the greatest percentage of centric relation contacts. A slide was present from centric relation to centric occlusion in 90% of patients. Balancing side interferences were present in 21 of 30 patients with first and second molars involved 78% of the time, and second molars only in 56%. Teeth associated with centric relation contacts were not significantly different periodontally from control teeth.

O'Leary et al. (1972) examined tooth mobility in cuspid-protected and group-function occlusions. Mean tooth mobility was higher in the cuspid-protected group for every tooth type.

Ramfjord and Ash (1981) reviewed the role of occlusion relative to the etiology and treatment of periodontitis. The authors indicated that trauma from occlusion (TFO) did not initiate gingivitis or periodontitis, or have a role in disease progression. Active trauma tended to accelerate bone loss and pocket formation depending on the presence of local irritants and inflammation. Hypermobility, without additional signs or symptoms, was not an absolute indication of TFO.

A diagnosis of TFO should be supported by evidence of active injury over time with continued or increasing mobility, persistent discomfort, and radiographic evidence of bone or root resorption. It was noted that a widened PDL was not always indicative of TFO or hypermobility, but may represent physiologic adaptation or past TFO that was self-limiting. Increased mobility with a reduced periodontium did not usually lead to further loss of support. Bruxism could cause primary or secondary TFO, with increased significance depending on level of periodontal support. Marginal ridge discrepancy and plunger cusp's role in the etiology of periodontal disease is not firmly established and neither is the relationship between Angle's malocclusion and periodontal disease. Malocclusion may indirectly affect periodontitis in the presence of poor plaque control, and gingival trauma may result from tissue impingement. Plaque control must be initially established, and occlusal factors addressed subsequently. The authors suggested that long-term splinting was indicated if mobility was found to interfere with the health and comfort of the patient or if the mobility was progressive.

Wasson (1988) described functional occlusion in orthodontic patients and the relationship of the teeth to condylar movements. Changes in occlusion include the angle of incisal guidance, cusp-fossa relationships, and the direction of the occlusal plane. It is suggested that in order to prevent temporomandibular joint disorders, muscular dysfunction, and excessive occlusal wear, the rules of functional occlusion should be followed. The neuromuscular avoidance systems allow the mandible to avoid prematurities during movement. However, this system is absent during sleep, resulting in parafunctional habits which may be detrimental due to the duration of activity of muscles and leverage-producing direction of the applied forces. The most desirable lever in normal jaw function is one which produces work at the end of the lever arm with a condylar fulcrum, reducing the level of stress. To achieve a sound occlusal result in orthodontic patients, the authors suggest: 1) maximum intercuspation with the mandible in centric relation; 2) immediate anterior disclusion during functional movements; and 3) stable intercuspation allowing opposing cusps to pass without interference.

Burgett et al. (1992) conducted a randomized trial of occlusal adjustment in 50 periodontal patients treated with scaling and root planing or modified Widman flap (MWF) surgery. Following 2 years of quarterly maintenance, mean attachment levels were better in the occlusal adjustment group (significant only in the 4- to 6-mm probing depth) than the unadjusted group, and were better in patients undergoing scaling and root planing than those undergoing modified MWF surgery. Probing depth reduction was greater (not significantly) in patients treated with occlusal adjustments than those not adjusted. Modified Widman flap surgery was more effective at reducing mean probing depths than scaling and root planing.

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CHAPTER 13. INTERDISCIPLINARY CONSIDERATIONS

Section 1. Periodontic-Endodontic Considerations

DIAGNOSIS

One of the more perplexing problems confronting the patient and periodontist is the periodontic-endodontic lesion (PEL). Diagnosis of these lesions is based on an accurate, comprehensive history and a thorough examination (including vitality testing, radiographs, and root fracture assessment). It may be difficult, if not impossible, to determine the nature and chronology of a periodontic-endodontic lesion.

Hiatt (1977) has suggested that such lesions be considered endodontic in nature for treatment planning purposes, since endodontic therapy alone may resolve the lesion. However, resolution of the defect is highly dependent on the primary source and the chronicity of the lesion; treatment may eventually involve both endodontic and periodontal treatment (Benenati et al., 1981).

Pitts and Natkin (1983) reported on the diagnosis and treatment of vertical root fractures, acknowledging their diagnostic challenges. Many signs of a vertical root fracture are similar to those associated with pulpal necrosis. If possible, visual observation of the crack is preferred using such diagnostic aids as dye, fiberoptic lights, or fine explorers. Often a definitive diagnosis can only be derived following surgical exposure or extraction. Radiographic evidence of root fracture is usually absent, but may present as a "parapulpal" radiolucent line, separation of root fragments, extrusion of filling material, or radiolucency around the root apex in a halo-like configuration. While coronal fractures may manifest as a periodontal-like lesion, the presence of a narrow, step-like radiolucency is highly suggestive of a tooth fracture. Additional diagnostic signs of a vertical root fracture include external resorption along the fracture line and loosening of retrofill amalgams. Treatment is directed at eliminating the fracture while maintaining the maximum amount of tooth structure. Depending on the size and location of the fracture, treatment may range from root resection to extraction.

CLASSIFICATION OF PERIODONTIC-ENDODONTIC LESIONS

Simon et al. (1972) provided a classification of periodontic-endodontic lesions based on possible etiology, diagnosis, and prognosis.

Primary Endodontic Lesions. These lesions may appear concurrently with drainage from the gingival sulcus area and/or swelling in the buccal attached gingiva. They are periodontal only in that they pass through the periodontal ligament area. In reality, they are sinus tracts resulting from pulpal disease. Radiographically, different levels of bone loss may be apparent depending on the avenue of sinus tract formation. Diagnosis may be facilitated by inserting a gutta percha point or silver cone into the tract and taking a radiograph to determine the origin of the lesion. When the pulp does not respond to an electric vitalometer or thermal tests, a necrotic pulp may be the offender. In addition, a minimal amount of calculus or plaque formation is usually encountered when probing. These lesions will usually heal with endodontic therapy alone.

Primary Endodontic Lesions With Secondary Periodontic Involvement. If the primary endodontic problem remains untreated, it may be affected secondarily with periodontal breakdown. The treatment and prognosis of the tooth are altered when plaque or calculus is detected on the affected root surface. Such teeth require endodontic and periodontal therapy. The prognosis depends primarily on the periodontal therapy, assuming that the endodontic needs have been met.

Primary Periodontic Lesions. These lesions generally manifest calculus at varying distances along the affected root surface. The pulp responds vitally to endodontic testing procedures and prognosis depends on the effectiveness of the periodontal therapy.

Primary Periodontic Lesions With Secondary Endodontic Involvement. As periodontal lesions progress apically, lateral or accessory canals may be exposed to the oral environment and contribute to pulpal necrosis. Resulting lesions may be radiographically indistinguishable from primary endodontic lesions with secondary periodontic involvement. Teeth undergoing periodontal therapy that do not respond as anticipated should be pulp tested. Prognosis depends on the periodontal care once endodontic therapy has been accomplished. These lesions will not respond to periodontal treatment alone.

"True" Combined Lesions. These lesions occur where an endodontically-induced periapical lesion exists on a periodontally-diseased tooth. The radiographic intrabony defect presents when the respective lesions merge along the root surface. These clinical and radiographic features are indistinguishable from the other lesions previously described which have secondary involvement. Periapical healing may

be anticipated following successful endodontic therapy. Periodontic aspects may or may not respond to periodontal treatment, depending on the severity of involvement.

Hiatt (1977) provided a classification of periodontic-endodontic lesions, relating appropriate treatment and prognosis based on the primary etiology. Lesions of pulpal origin with associated periodontal involvement of short duration can be expected to resolve following endodontic therapy. Incomplete root fractures and periodontal lesions of short duration (i.e., periodontal abscess) with secondary pulpal disease primarily require treatment of the periodontium. Independent pulpal and periodontal lesions may merge into combined lesions and carry a poor prognosis similar to pulpal lesions which evolve into periodontal lesions following treatment. The major determinant of successful treatment of periodontic-endodontic lesions is the chronicity of the periodontal component.

INTERRELATIONSHIPS OF THE PULP AND PERIODONTIUM

Sharp (1977) and Benenati et al. (1981) have discussed the relationship between periodontium and pulp. Embryologically, dental pulp originates from the dental papilla, while periodontal ligament and cementum originate from the dental follicle. These structures are mesodermal in origin and are initially separated by the epithelial root sheath (Hertwig's epithelial root sheath). Lateral root canals are formed when odontoblasts do not produce dentin and cementum in areas of blood vessels and nerves that have penetrated the epithelial root sheath, establishing a communication between the pulp and periodontal ligament (PDL). The authors suggest that accessory canals are primary channels through which microorganisms may move between pulp and periodontium. Although the percentage of lateral canals varies, the authors feel the frequency of this relationship warrants concern. Sharp (1977) suggests that periodontal disease does not affect the pulp until the lesion has extended to the root apex.

Several studies have attempted to examine the effect of the periodontium on the pulp. Mazur and Massler (1964) histologically examined the pulps of 106 caries-free teeth extracted for periodontal reasons. No relationship was observed between the amount of periodontally-exposed root and degenerative changes in the pulp. In a separate group of patients, the pulps of periodontally involved teeth were compared to homologous, non-periodontally involved teeth on the other side of the arch, or on the same side but in the opposite jaw. All teeth were caries-free and unfilled. No relationship could be established between morphologic changes in the pulp and sites with periodontal involvement. Langeland et al. (1974) provided support for the effect of periodontal disease on the pulp. The authors examined pulp tissue from 60 periodontally-involved teeth in an attempt to correlate the presence of bacterial plaque at the entrance of lateral or main canals in the presence of an inflammatory pulpal response. While the presence of inflammatory cells and pulpal calcification (true pathosis) occurred more frequently as the plaque front moved apically, as long as the principal (apical) canal was not seriously involved, the entire pulp did not necrose (despite involvement of one or more lateral canals and/or dentinal tubules).

Czarnecki and Schilder (1979) also evaluated the degree of pulpal pathosis associated with periodontal disease. When pulps of periodontally involved teeth were examined, 6 of 34 exhibited pulpal pathology. All of these teeth had carious lesions or extensive restorations. Caries-free teeth had no histologic evidence of true pulpal pathology regardless of their periodontal condition. The authors concluded that pulpal health is unaffected by the presence or severity of periodontal disease. Teeth receiving previous periodontal therapy were excluded from this study since it was felt that periodontal treatment may induce pulpal changes. Torabinejad and Kiger (1985) histologically evaluated 25 teeth from the same patient which exhibited varying degrees of periodontal disease. The teeth were vital, had few restorations, and no histologic evidence of pulpal pathosis. Although calcifications were present, this was not considered pathologic.

The effects of periodontitis and periodontal therapy on the pulp were assessed by Bergenholtz and Lindhe (1978) using experimental ligature-induced periodontal disease in the monkey model. After induction of periodontal disease, no further treatment was performed in 1 group of teeth, while remaining teeth were subjected to gingivectomy followed by scaling and root planing (with an attempt at complete cementum removal). Following plaque accumulation for 2, 10, and 30 days, secondary dentin and inflammation were observed in the pulp tissue of all groups (including non-treated controls), tending to occur adjacent to exposed root surfaces. However, pulps in the majority of roots (health, periodontitis, and periodontitis with root planing) exhibited no pathology. Based on this relatively short-term study, the authors concluded that periodontitis and scaling/root planing do not predictably lead to pulpal changes.

Effects of extensive periodontal and prosthetic treatment on dental pulps were examined by Bergenholtz and Nyman (1984) who retrospectively examined 672 teeth 4 to 13 years after treatment for severe periodontal disease. The authors reported that abutment and non-abutment teeth displayed similar periodontal breakdown; however, endodontic complications resulting from pulpal necrosis occurred more frequently in abutment teeth (15% versus 3%), suggesting that prosthetic treatment may be a causative factor in pulpal disorders. The assumption that the periodontal status could have played a role as a causative factor for pulpal involvements was countered by the observation that pulpal necrosis of unidentified etiology occurred almost exclusively in teeth serving as bridge abutment.

EFFECTS OF ENDODONTIC THERAPY ON PERIODONTAL THERAPY

Another area of controversy in the periodontic-endodontic literature relates to connective tissue attachment potential in endodontically treated teeth. Diem et al. (1974) addressed this question using endodontically treated teeth denuded of cementum in 6 Rhesus monkeys. Four teeth were used per animal: 1) an untreated control; 2) pulp extirpated and unfilled; 3) pulp extirpated and a camphorated parachlorophenol (CMCP) paper point placed in the canal; and 4) pulp extirpated and canal filled with gutta percha. Attachment loss was surgically created by removing 3 to 5 mm of bone from facial surfaces of the teeth. No relationship was found between the amount of connective tissue/alveolar bone regeneration and canal status, indicating cementum would form on roots of endodontically-treated teeth denuded of cementum. Interestingly, mean cementum regeneration was highest in teeth with CMCP medicated canals. Unfortunately, this experiment did not duplicate in vivo conditions, since the lesions were surgically created immediately after endodontic therapy. More recently, Dunlap et al. (1981) examined the ability of human gingival fibroblasts to attach to root-planed dentin of endodontically and non-endodontically treated, periodontally-involved teeth. Teeth were extracted and longitudinally sectioned following cementum removal on one side of the root. Root fragments were then incubated with fibroblasts in a tissue culture system for 72 hours prior to staining and evaluation. Fibroblasts grew on all root planed segments and none of the unplaned segments. No difference was detected between vital and endodontically treated teeth. The authors concluded that teeth treated endodontically and those with vital pulps should respond to new attachment procedures equally well and that delaying endodontic therapy until periodontal surgery is completed is not warranted. These findings differ from those of Sanders et al. (1983) who evaluated the use of freeze-dried bone allografts in human osseous defects. Results suggested that a significantly lower percentage of allografts placed in defects adjacent to endodontically obturated teeth resulted in complete or greater than 50% osseous regeneration as compared to those placed adjacent to non-obturated teeth of unknown pulpal status (33% versus 65%). While the authors concluded there is need for greater attention to osseous defects associated with endodontically obturated teeth, they felt differences were at least in part due to continuing endodontic pathology associated with inadequate obturations.

ANATOMIC CONSIDERATIONS—ACCESSORY CANALS

It is felt that in order for the periodontium to affect the pulp there must be a means of interaction. Accessory canals may provide one such avenue. Gutmann (1978) evaluated the prevalence, location, and patency of accessory canals in the furcation regions of first and second molars. Under external vacuum, safranin dye was placed in the pulp chamber and forced through the tooth. Upon evaluating the external furcation for dye penetration, 28.4% (29.4% mandibular/27.4% maxillary) of the teeth exhibited patent accessory canals in the "furcation region" (defined as the actual furcation plus an area 4 mm extending apically along the internal aspect of the root surfaces). In addition, 10.2% of the teeth had lateral accessory canals, with little difference noted between mandibular and maxillary molars. Ability of the pulp and periodontium to communicate via dentinal tubules was also evident, especially where cementum was denuded. Although this study demonstrated the prevalence of accessory canals, it is quite different from the in vivo scenario where living cells (odontoblasts) can exclude material from dentinal tubules. The author emphasized that the mere presence of accessory canals does not imply that pathosis will spread from one entity to another. While necrotic tissue and bacterial plaque present in accessory canals may not severely affect the pulp, they may tend to perpetuate periodontal furcation lesions making therapeutic success impossible.

Since periodontally affected teeth have more exposed root surface area due to attachment loss than other teeth, Kirkham (1975) investigated the incidence of accessory canals adjacent to periodontal pockets by injecting a radiopaque solution into the pulp chambers of 100 extracted periodontally-involved teeth. Upon radiographic examination, 23% of the teeth exhibited one or more lateral canals. Most of the canals were located in the apical third of the root. No accessory canals were observed in the furca (although the furcation was not specifically examined). The frequency of lateral canals in mandibular premolars (53.5%) and mandibular molars (44.5%) was considerably higher than that of any other tooth group. Maxillary molars and mandibular incisors had the lowest percentage of teeth with accessory canals. Only 2 of 100 teeth demonstrated an accessory canal within a periodontal defect. Although 1 of these teeth showed histologic signs of pulpal necrosis, it was impossible to determine the exact origin of the periodontic-endodontic lesion.

The ability of bacteria to traverse tooth structure was demonstrated by Adriaens et al. (1988) who histologically examined 21 extracted, caries-free human teeth and observed bacterial invasion of radicular cementum and dentin. Bacteria were found in the lumina of dentinal tubules, lacunar defects in cementum, and in spaces between remnants of Sharpey's fibers and the structures in cementum where these fibers inserted. Although bacterial invasion of the dentinal tubules was generally limited to the outer 300 μm, bacteria were detected on the pulpal wall in 2 teeth. Bacteria were never observed in intertubular dentin. The authors suggested that dentinal tubules and lacunae may serve as bacterial reservoirs from which pulpal pathosis or re-

colonization of treated root surfaces could occur. In general, these studies indicate that the periodontium does not predictably affect the dental pulp. When it does exert an effect, it most likely occurs via lateral canals (apical third and furcation) and in previously compromised pulps (through periodontal or restorative treatment).

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Section 2. Periodontic-Orthodontic Relationships

INTRODUCTION

Since orthodontic tooth movement may affect the surrounding periodontium, it is important to consider the interrelationships between orthodontic and periodontal therapy when such multidisciplinary treatment needs exist.

Gryson (1965) presented a review of the orthodontic literature concerning the response of the periodontal ligament (PDL) to orthodontic forces of various magnitude. Strong forces literally crush the PDL on the pressure side as a result of direct contact between the root and bone. No movement occurs until adjacent bone is resorbed (undermining [rear] resorption) and the PDL regenerates. Moderate forces (in slight excess of capillary pressure) cause strangulation of the PDL and a delay in bone resorption. Light forces (less than capillary pressure [20 to 25 mm Hg]) merely cause ischemia in the PDL and tooth movement proceeds continuously with concomitant bone resorption and formation. The author concludes that although intermittent forces seem to offer the best chance of complete physiologic tooth movement, continuous light forces provide a compromise that is more time efficient and results in no permanent tissue damage. Retention of orthodontically repositioned teeth is necessary to allow tissues to reorient to their new position. Rotations are the most difficult movement to retain and are most effectively managed by early correction, over-rotation, and transeptal fiberotomy.

ORTHODONTIC-PERIODONTIC RELATIONSHIPS

Keesler (1976) presented another review of the interrelationships between periodontics and orthodontics, noting that with the possible exception of hypofunction and severe overbite with impingement, there is no evidence that orthodontic correction of malocclusion will enhance or detract from periodontal health. Possible exceptions include severe overbite with impingement and hypofunction. Plaque retention and oral hygiene habits are the primary factors in periodontal disease with tooth position playing a minor role. Orthodontic treatment in adults should be approached with the understanding that compared to young individuals, adults lack skeletal growth potential, have decreased osteoblastic-osteoclastic activity, and have increased potential for tooth mobility with orthodontic movement. Initial periodontal therapy should be accomplished prior to orthodontic movement. The author suggests waiting 6 to 9 months after tooth movement before proceeding with periodontal surgical procedures. Improvement in periodontal health may be achieved by moving (e.g., tipping) affected teeth into a greater volume of bone or adjacent osseous defects. The author suggests that uprighting of molars should be approached cautiously due to the potential for furcation exposure and that movement into recent extraction sites should be avoided.

Orthodontic therapy may be directed toward correction of spacing and/or crowding of teeth. Silness and Roynstrand (1984) evaluated the relationship between spacing and dental health in 15-year-old children, reporting that children with more spacing had less plaque, gingivitis, probing depth, and interproximal restorations than those with minimal spacing. In a subsequent study, Silness and Roynstrand (1985) evaluated periodontal health adjacent to

aligned and non-aligned interproximal surfaces in the same population. Patients with fewer non-aligned surfaces had a more favorable periodontal condition (less plaque, gingivitis, and probing depth) than patients with many non-aligned surfaces. Likewise, the greater the number of non-aligned surfaces present, the greater the number of interproximal restorations.

Orthodontic therapy has been utilized in attempts to improve the health of teeth with significant periodontal destruction. Brown (1973) examined the clinical and histological effects of molar uprighting on existing periodontal osseous defects in 5 patients. Molars with mesial osseous defects were uprighted for 90 to120 days, and stabilized for 3 months. A single molar which served as the control was not moved orthodontically but was scaled and root planed (S/RP) bi-weekly. All teeth were extracted via block section and examined histologically. Following uprighting, the gingival tissues exhibited decreased inflammation, a more apical location, and reduction in probing depth of 3.5 mm. The control tooth showed little improvement. Clinically and radiographically, there was a reduction in probing depth of 3.5 mm and a mean bone loss of 0.5 to1.0 mm for the uprighted molars.

The effect of tooth movement into adjacent osseous defects was studied in a monkey model by Polson et al. (1984). Intrabony defects were induced by placement of ligatures and elastics followed by restoration of health by S/RP and maintenance. The experimental teeth were moved up to 6 mm into the osseous defects over a 3-month period and were retained for 2 months prior to collection of block sections. Histologic evaluation of the pressure side showed narrowing of the angular defect while the tension side demonstrated conversion of the intrabony pocket to a suprabony pocket. On both the pressure and tension sides, epithelium extended to the level of root planing with no evidence of new connective tissue (CT) attachment. The authors concluded that teeth with a reduced but healthy periodontium may be moved orthodontically without detrimental effect on the attachment level.

Van Venrooy and Yukna (1985) evaluated the effects of orthodontic extrusion of single-rooted teeth with severe surgically-created defects in beagle dogs. Ligature-induced periodontitis resulting in loss of 1/3 to 1/2 of the periodontal support was followed by orthodontic extrusion using elastics with 20 to 25 grams of force over a 14 to 21 day period. Compared to non-extruded control teeth, extruded teeth exhibited shallower probing depths, less gingival inflammation, no bleeding on probing, a wider PDL, greater width of cementum, and increased crestal bone height. The authors suggested that the positive changes observed in extruded teeth may have resulted from conversion of the subgingival plaque to a supragingival plaque with decreased pathogenicity.

Conflicting findings have been published regarding the effect of labial movement of incisors on facial alveolar bone. Batenhorst and Bowers (1974) reported the clinical and histologic changes associated with facial tipping and spontaneous extrusion of mandibular incisors in monkeys. They noted an increase in width of the facial attached gingiva with no alteration in position of the mucogingival junction. The epithelial attachment maintained a close relationship to the CEJ on all surfaces except the facial where the epithelial attachment was longer and more apically located. As teeth moved facially, alveolar bone apposition occurred on the interproximal and lingual surfaces while dehiscences formed on the facial surfaces. Facial movement and extrusion also resulted in a parallel arrangement of connective tissue (CT) fibers along the facial root surfaces rather than a perpendicular arrangement as seen on the interproximal and lingual surfaces.

In a similar study, Wingard and Bowers (1974) used a different monkey model in an attempt to create dehiscences or fenestrations on the facial surfaces. Mandibular central incisors were tipped facially 2 to 5 mm while untipped lateral incisors served as controls. They reported no significant difference in mean alveolar bone level between experimental and control teeth; furthermore, no dehiscences or fenestrations were observed on tipped teeth. Histologically, there was thinning of the PDL and facial alveolar bone on the tipped teeth, with frontal resorption and enlarged narrow spaces consistent with undermining resorption. Results of this study agree with other authors who have concluded that compensatory bone formation occurs in order to maintain a tooth's normal supporting apparatus when the tooth is moved facially with proper forces.

Karring et al. (1982) studied the effect of facial movement of incisors on the supporting periodontal tissues in beagle dogs. In this study, meticulous oral hygiene was consistently performed and the tissues were maintained in a state of health. Maxillary incisors were initially moved in a facial direction over 5 months, resulting in formation of dehiscences extending halfway down the roots. The incisors on one side were moved back to their original position over 5 months and specimens were evaluated after a final 5-month retention period. Teeth that were moved facially and retained in that position failed to demonstrate repair of the dehiscences and exhibited CT fibers paralleling the root surface. Incisors that were moved back to their original position after facial displacement demonstrated complete regeneration of alveolar bone with CT fibers inserting perpendicularly into new bone and cementum. In the absence of clinical inflammation, the apical termination of the junctional epithelium (JE) in the orthodontically treated teeth was at the cementoenamel junction (CEJ), and no loss of CT attachment was observed.

ORTHODONTIC MOVEMENT IN THE PRESENCE OF PLAQUE

Ericsson et al. (1977) compared the effects of orthodontic forces with tipping/intrusive components on healthy and

plaque-infected periodontal tissues in dogs. Periodontal defects were created by placement of copper bands and were surgically corrected prior to tooth movement. This resulted in a reduction of healthy periodontium. Orthodontic forces were applied bilaterally over 6 months with plaque accumulation allowed on one side and oral hygiene procedures accomplished on the other. Clinically, there was a slight gain of attachment in plaque-free teeth and a slight loss in plaque-infected teeth. Histologically, while there was a trend for plaque-infected teeth to have a loss of attachment, there was no statistically significant difference in the level of attachment between the 2 groups of teeth. There was significantly more inflammation in the tissues adjacent to plaque-infected teeth and intrabony pocket formation was frequently associated with these teeth. The authors suggest that intrusive forces may have shifted the supragingival plaque to a subgingival location, resulting in intrabony pocket formation and loss of attachment. However, the data from this study indicate no difference in the CT attachment levels between plaque-free and plaque-infected teeth.

Since the majority of orthodontic patients are relatively young, the orthodontist may encounter patients with localized juvenile periodontitis (LJP). Folio et al. (1985) studied the clinical and microbiological effects of orthodontic therapy on 2 LJP and 2 post-LJP patients. The patients were placed on the "Keyes technique" oral hygiene regimen and were monitored clinically and by phase contrast microscopy. The authors observed increased levels of spirochetes, motile rods, and PMNs in all subjects within 1 to 6 months of appliance placement, prompting follow-up care including oral hygiene instruction, subgingival irrigation, S/RP, and/or systemic antibiotics. One episode of this treatment resulted in reduction or elimination of these organisms for the duration of the study (up to 78 weeks). Conclusions were that orthodontic therapy may aggravate plaque-induced diseases resulting in further breakdown and that periodontally compromised teeth may be successfully treated with orthodontics if excellent plaque control is maintained. It should be noted that phase contrast microscopy was used to monitor the patient's oral hygiene effectiveness, rather than the pathologic flora of LJP.

To evaluate the long-term impact upon the periodontium, Trossello and Gianelly (1979) performed a retrospective study comparing the status of 30 females who had received multibanded fixed orthodontic therapy at least 2 years previously with 30 age-matched controls. The only statistically significant differences between the 2 groups were related to root resorption and mucogingival defects. The orthodontically treated patients had a higher prevalence of root resorption (17% versus 2%) and a lower prevalence of mucogingival defects (5% versus 12%). Root resorption was most common in maxillary incisors followed by mandibular incisors. While not statistically significant, the orthodontically treated patients also had more crowding of tissue and loss of alveolar bone where extraction spaces were closed and slightly greater crestal bone loss overall. Because only minor differences were encountered, the authors concluded that effects of orthodontic treatment on the periodontium are minimal. Another study to evaluate the long-term effects of orthodontic therapy was performed by Polson and Reed (1984), in which cross-sectional assessment of radiographic alveolar bone levels in 104 patients who had completed orthodontic therapy at least 10 years previously were compared with 76 matched controls who had no orthodontic treatment. Overall, they found no significant difference in alveolar crest levels between the 2 groups, with one exception. In the orthodontically treated patients, the alveolar crest on the distal surfaces of the molar teeth was located at a more coronal level than in non-orthodontic controls. This may have resulted from intrusion of the molars secondary to orthodontic treatment.

MUCOGINGIVAL CONSIDERATIONS

The treatment of mucogingival defects may involve orthodontic and periodontal therapy. Boyd (1978) reviewed the indications for and sequence of mucogingival therapy with respect to orthodontic intervention. He suggested that mucogingival defects in the absence of malocclusion-malalignment should be treated early to avoid further breakdown. However, he suggests that preoperative orthodontic intervention may improve or even eliminate gingival recession when malocclusion is a contributing factor. The author recommended that orthodontic consultation should be obtained when the: 1) involved area is related to a shearing effect of one tooth on another (e.g., deep overbite/crossbite with tripping of gingival tissue); 2) involved tooth may be elected for extraction due to tooth size discrepancy; and, 3) tooth with the mucogingival defect is in labioversion (lingual movement of the tooth may correct the mucogingival defect without surgery).

ORTHODONTIC ROTATION

The periodontist is frequently called on to assist in the retention of orthodontically repositioned teeth. Tooth rotation is generally simple to achieve but difficult to retain. It is theorized that stretching of the gingival fiber apparatus during rotation is followed by recoil of the fibers during the retention phase, with resultant relapse of tooth malposition. Edwards (1970) tattooed the attached gingiva and alveolar mucosa around orthodontically rotated teeth in 12 patients. Following rotation and 8 weeks of mechanical retention, experimental teeth received a circumferential fiberotomy (number 11 blade placed into sulcus to and below the crest of bone). Control teeth received no surgical procedures. During tooth rotation, the tattooed fibers deviated in the direction of rotation. Upon release of mechanical retention, all control teeth demonstrated relapse with deviation of fibers in the direction of relapse. Conversely, teeth which received fiberotomies did not relapse. Within 20 to 40 hours post-fiberotomy, tattooed fibers had returned to the original pre-rotation position. Thus, Edwards demon-

strated that fiberotomy relieves post-rotation tensional forces in the fiber apparatus, allowing recoil of the fibers without relapse of tooth malposition.

ORTHODONTIC EXTRUSION AND FIBEROTOMY

During the forced-tooth eruption, the adjacent periodontium usually accompanies the root coronally. The coronally displaced tissues usually necessitate surgical crown lengthening to provide adequate clinical crown for the final restoration. Pontoriero et al. (1987) and Kozlovsky et al. (1988) have reported using a supracrestal fiberotomy procedure to sever the supracrestal gingival fibers during rapid extrusion of tooth. Pontoriero et al. recommend the procedure weekly, Kozlovsky et al. every 2 weeks. Both have reported case studies which have successfully avoided the need for crown lengthening surgery following extrusion. The supracrestal fiberotomy is believed to eliminate the tensile stress upon the alveolar crestal bone preventing crestal bone deposition. Following an intra-sulcular incision which parallels the cemental surface and engages bone-to-bone, the root is then thoroughly planed to the level of the alveolar crest. Berglundh et al. (1991) performed extrusions on 5 beagle dogs with and without a fiberotomy (at 2-week intervals) with histologic evaluation. The authors concluded that extrusion combined with a fiberotomy limits displacement of the gingival and supracrestal tissues coronally, and limits crestal bone apposition, but does not completely prevent the coronal migration of those tissues.

Advantages of the technique include: 1) ease and quickness of the procedure; 2) possibly a shorter retention period post-extrusion; 3) direct inspection of the extruding sound tooth structure preventing over- or under-treatment; and 4) possible elimination of the need for a crown lengthening procedure following extrusion.

ORTHOGNATHIC SURGERY

As orthognathic surgical procedures have become more commonplace, interest has grown concerning the effect of such therapy on the periodontium. Foushee et al. (1985) evaluated 24 patients who had received advancement genioplasty with or without maxillary/mandibular osteotomy. Width of keratinized and attached gingiva was determined pre- and postoperatively. Following surgery, there was a significant decrease in the width of keratinized and attached gingiva in mandibular incisors and premolars. The initial width of keratinized and attached gingiva was unrelated to the susceptibility for recession after surgery. Of the 24 patients, 10 had post-treatment recession: 4 had slight localized recession (0.5 mm per site), and 6 had more severe and generalized recession (range of 0.5 to 3.0 mm). Since these patients received orthodontic treatment between the initial evaluation and surgery, it is difficult to determine if the recession resulted from the orthodontic or orthognathic treatment.

IMPLANTS AND ORTHODONTICS

Higuchi and Slack (1991) reported the placement of 10-mm implant fixtures placed bilaterally in the third molar areas of 7 adult patients. The implants were allowed to integrate for 4 to 6 months, and were subsequently used as posterior anchorage (up to 400 g) for protraction and retraction. After completion of orthodontic treatment, the fixtures were placed in a non-functional state beneath the soft tissues. Measurements performed on the cephalometric radiographs revealed no movement of the implant fixtures. Fixture placement in the mandibular third molar area was described as difficult, and interference with the maxillary soft tissue and dentition was also reported.

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Section 3. Periodontic-Prosthodontic-Restorative Interactions

INTRODUCTION

The periodontium is the foundation which largely determines the function, esthetics, and longevity of the dentition. Factors impacting restorative/prosthodontic treatment include esthetics, function, and periodontal health. Periodontal health should be the foremost of these factors as the infringement of a restoration on the physiologic dimensions of the periodontium and/or the interference with plaque removal will potentially affect esthetics and function.

BASIC CONCEPTS

Dimensions of the Periodontium in Health

Garguilo et al. (1961) described the dimensions of the dento-gingival junction using human cadaver specimens. A range of dimensions was noted with the connective tissue attachment being most consistent with a mean measure of 1.07 mm and the epithelial attachment most variable with an average of 0.97 mm. The sulcus depth averaged 0.69 mm. This dento-gingival dimension has been referred to as the biologic width.

Biologic Width/Physiologic Dimension

Ingber et al. (1977) discussed the maintenance of the biologic width when restoring fractured or carious teeth where marginal infringement on the dento-gingival junction was imminent. The authors concluded that "a minimum dimension of 3 mm coronal to the alveolar crest is necessary to permit healing and proper restoration of the tooth." Violation of this width potentially leads to adverse periodontal reactions including inflammation and alveolar bone loss. Ramfjord (1988) questioned the surgical creation of a 2- to 3-mm biologic width apical to a proposed restoration margin via ostectomy. He felt that bone should be removed to the minimum extent needed to ensure access for margin placement, but it may be better to ". . . let nature determine the biologic width over the coming years, with the patient maintaining adequate oral hygiene."

Maynard and Wilson (1979) described the following 3 aspects of the physiologic dimension: intracrevicular, subcrevicular, and superficial. The authors discussed the implications of restorative encroachment on the subcrevicular physiologic dimension or biologic width. They also noted that when "intracrevicular restorative margins" are placed at sites of insufficient gingival (or marginal tissue) width and/or thickness, "marginal tissue recession," apical migration of the attachment apparatus or both may result. The authors recommended 5 mm of keratinized tissue (3 mm attached and 2 mm free) and a minimum crevicular physiologic dimension of 1.5 to 2.0 mm when marginal coverage by free gingiva is dictated by esthetics.

TISSUE-RESTORATIVE INTERACTIONS

Overhanging Dental Restorations

Overhanging dental restorations can provide a niche for plaque accumulation, hinder oral hygiene, cause mechanical irritation, impinge on the interproximal embrasure space, and encroach upon the biologic width, leading to inflammation and potential periodontal destruction. Waerhaug (1976) hypothesized that overhangs extend the sphere of microbial influence. The prevalence of overhanging dental restorations range from 25% to 76% for restored surfaces and 32% to 90% for patients (Brunsvold and Lane, 1990). Even when identified, overhangs are often difficult to remove or the replacement restoration has an overhang. Pack et al. (1990) reported that 61% of teeth with overhanging restorations on pre-treatment radiographs had residual overhangs post-treatment.

Gilmore and Sheiham (1971) radiographically (Schei ruler) identified 0.22 mm proximal alveolar bone loss associated with overhangs in posterior teeth compared to non-restored proximal surfaces. Clinically, higher periodontal disease index scores were associated with the presence of overhangs. Similarly, Jeffcoat and Howell (1980) reported that medium and large overhangs were associated with greater radiographic bone loss compared to contralateral teeth without overhangs in 100 patients.

Lang et al. (1983) reported the clinical and microbiological effects of subgingival restorations with or without overhangs. In a crossover design, gold onlays extending 1 mm subgingivally were placed in 10 patients (20 sites). Half of the onlays had clinically perfect margins, the other half had 0.5 to 1.0 mm overhangs. The onlays were placed for 8 to 24 weeks and were subsequently switched (i.e., crossed-over) for 12 to 27 weeks. No oral hygiene was performed on the proximal surfaces of the onlays. Overhang surfaces were associated with bleeding on probing and a 1 to 2 mm increase in probing depth (without any loss of attachment). The overhanging restorations were accompanied by a shift in the subgingival microflora similar to that found in chronic adult periodontitis. This included increased proportions of Gram-negative anaerobic bacteria, black-pigmented *Bacteroides*, and an increased anaerobe:facultative ratio.

Highfield and Powell (1978) examined the effect of posterior amalgam overhang removal on periodontal health. Eighty (80) overhangs received 1 of 4 treatments consisting of: 1) no treatment; 2) overhang removal; 3) professional plaque control every 2 weeks without overhang removal; or 4) overhang removal and professional plaque control every 2 weeks. Three-month results indicated that the group

receiving overhang removal and professional plaque control had the greatest improvement in gingival index and bone score. Removal of plaque only or overhangs only failed to result in a dramatic improvement.

Effects of Preparation

The effects of margin preparations which violate the biologic width were demonstrated in a report by Carnevale et al. (1983). Marginal and interproximal connective tissue was removed, exposing interproximal crestal bone in dogs. Teeth received one of the following margin preparations which extended to the alveolar crest: 1) chamfer; 2) feather edge; 3) shoulder; or 4) no preparation. Histological examination revealed complete healing by 90 days with the experimental sites demonstrating approximately 1 mm of crestal bone resorption while the control sites had no bone loss or attachment loss. Tooth preparation for crowns with crevicular margins can be accomplished relatively atraumatically with judicious treatment. Dragoo and Williams (1982A and 1982B) prepared human teeth scheduled for extraction. The teeth were subsequently removed in block section with the adjacent soft tissue. Histological examination indicated that tooth preparation preceded by retraction cord placement sustained minimal soft tissue trauma. Teeth prepared without retraction cord which subsequently received either retraction cord, electrosurgery, or rotary gingival curettage sustained various degrees of soft tissue damage.

Margin Placement

When margins violate the biologic width, potential exists for attachment loss and apical migration of the junctional epithelium. Parma-Benfenati et al. (1986) placed amalgam restorations at or 4 mm coronal to the alveolar crest in dogs following partial thickness flap reflection. Flaps were subsequently apically positioned. Twelve-week histological results demonstrated approximately 5 mm of bone loss in thin osseous septa adjacent to the crestal restoration margins. In a similar study design, Tal et al. (1989) placed amalgam restorations with facial margins at the alveolar crest following mucoperiosteal flap access. Control sites received mucoperiosteal flap reflection but no restorations. At 57 weeks the experimental sites demonstrated a mean of 3.16 mm marginal recession, 1.17 mm bone loss, and 0.90 mm connective tissue attachment. Control sites presented 0.46 mm marginal recession, 0.15 mm bone loss, and 4.47 mm connective tissue attachment. Although the post-study "biologic width" apparently changed from the pre-study measurements, attachment loss was obviously related to the violation of the biologic width.

Dragoo and Sullivan (1982A and 1982B) examined the effects of subgingival margin placement using acrylic temporary crowns on human teeth scheduled for removal. Shoulder preparations with a gingival bevel were followed by placement of temporary crowns. Crown margins terminated at the length of the gingival bevel or shoulder. Clinically, crowns with short margins appeared to heal more favorably. Histologic examination of teeth and adjacent soft tissue removed at 4 weeks indicated that crowns with the long margins gave the most ideal healing response. This was attributed primarily to their ability to prevent soft tissue from collapsing onto the shoulder and compromising complete seating of the permanent restoration. Flores-de-Jacoby et al. (1989) studied the effect of crown margin location on periodontal health and bacterial plaque morphotypes (dark-field microscopy) at 6 to 8 weeks and 1 year after restoration placement. The plaque index, gingival index, probing depths, and sulcus fluid flow were significantly higher for subgingival-margin sites compared to gingival or supragingival margins. Plaque from the subgingival margin group also had significantly lower counts of cocci and higher counts of spirochetes, fusiforms, rods, and filaments as compared to gingival or supragingival margins. The authors concluded that subgingival margins are associated with higher bacterial accumulation and less favorable plaque composition. Supragingival margins were most favorable while there was no clear indication that gingival margins were harmful. Stetler and Bissada (1987) investigated the interaction of subgingival crown margins in areas of narrow (< 2.0 mm) and wide (≥ 2.0 mm) zones of keratinized tissue in 58 teeth. The authors assumed that areas showing recession in proximity to crown margins signified that the original crown margin was subgingival. Narrow zones of keratinized tissue had a significantly higher gingival index (GI) than subgingival margins with a wide zone of gingiva. The gingival index did not differ between narrow and wide zones of gingiva in areas without subgingival margins The authors recommended augmenting the zone of keratinized tissue if subgingival margin placement is planned in an area with < 2 mm of keratinized tissue and where optimal oral hygiene cannot be anticipated.

Reasons for intracrevicular placement of margins include removal of caries or faulty restorations, development of adequate retention, prevention of root sensitivity, and/or esthetics. When placement of intracrevicular margins is not indicated, every attempt should be made to keep margins supramarginal; there is no assurance that they will remain in the desired position. Valderhaug and Birkeland (1976) evaluated 98 patients with crowns and fixed partial dentures over the course of 5 years. Initially, 59% of crown margins were located intracrevicularly; after 5 years, only 35% remained in this location. Mean probing depth and GI increased and attachment loss was greater adjacent to teeth with intracrevicular margins, while teeth with supragingival margins had decreased probing depths, gingival indices, and less attachment loss.

Carnevale et al. (1990) retrospectively evaluated 510 single unit crowns (350 molars, 139 premolars, and 21 anterior teeth) that had been re-prepared during flap and osseous surgery at least 1 year previously. Non-restored teeth were used as controls. The crown margin locations were

categorized as supragingival, subgingival, or at the gingival margin and were evaluated for clinically significant differences with respect to plaque, gingivitis, and probing depth. The 109 patients in the study had been treated for moderate to advanced periodontal disease and were seen on a 1 to 6 month recall interval. They found no statistically significant differences between the restored versus non-restored teeth relative to plaque and gingival inflammation. Crowns terminating at the gingival margin had the highest percentage of GI scores of 0, followed by subgingival and supragingival margins. At re-examination, 95.5% of the teeth had probing depths less than 3 mm; 4.1%, 4 to 5 mm; and 2 teeth ≥ 5 mm. Less than 1% bled upon probing.

Crown Lengthening

Ross and Garguilo (1982) discussed the restorative alveolar interface (that area of the root surface located between the alveolar crest apically and the free gingival margin coronally) and felt that crown lengthening procedures should include modification of this area of root surface and provide adequate room for crown preparation and re-establishment of the biologic width. The authors suggested that these modifications would enhance the contours of the final restoration and facilitate hygiene and maintenance therapy.

Bragger et al. (1992) evaluated the changes in periodontal tissue immediately and 6 months after crown lengthening surgery on 43 teeth in 25 patients. The surgeries created a 3-mm distance between the restoration margins and the osseous crest. Treatment proceeded after 3 weeks of healing, with clinical measurements recorded at baseline, 6 weeks, and 6 months. Osseous crest reductions of 1 mm, 2 mm, and 3 to 4 mm were observed 32%, 21%, and 4% of the time, respectively. Mean apical soft tissue recession was 1.32 mm immediately after suturing. No further significant change in mean soft tissue position accompanied healing. However, on a site basis, 33% of the sites had 1 to 3 mm of coronal soft tissue displacement and 29% of sites had 1 to 4 mm of recession between 6 weeks and 6 months. No further changes in attachment loss occurred after the initial 6 weeks of healing, nor did probing depths change. The study emphasized the need to delay margin placement in areas of esthetic concern up to 6 months following crown lengthening surgery.

Temporary Restorations

Waerhaug (1980) created cavity preparations which extended subgingivally in monkeys and dogs. The preparations were subsequently filled with self-curing acrylic resin, zinc oxide and eugenol, or gutta percha. Histological observation 13 to 283 days after restoration placement indicated initial plaque formation at the tooth-restoration interface which spread over the restoration and eventually over the tooth surface apically. Even in the absence of plaque, submarginal gingivitis accompanied restorations. Attachment loss of more than 0.2 mm was invariably associated with apical migration of the subgingival plaque (1.0 mm at 18 days). Vigorous toothbrushing was effective up to 0.7 mm below the gingival margin, suggesting that submarginal extension of restorations should be limited to no more than this distance.

Osseous Resective Surgery

Guilbert et al. (1988) reviewed restorative treatment for patients with severe periodontal disease who were treated with osseous resective surgery for pocket reduction. Prognostic considerations included patient age, systemic condition, patient behavior, clinical form of the disease, disease rate of progression, tooth anatomy, malocclusion, and habits. The strategic value of individual teeth was evaluated by comparing anterior and posterior and left and right segments. Molars and canines were assigned a value of 3; second molars, second premolars and centrals, 2; and first premolars and laterals, 1. These values were decreased by 1 if the tooth had 50 to 80% bone loss, Class I furcation invasions, or mobility. With > 50% bone loss and more involved furca, the strategic value was reduced by 2. Each segment had to score ≥ 3 for a fixed prosthesis to have a favorable prognosis. According to the authors, esthetic considerations for osseous resective surgery include increased crown length, with the lip frame, lip line, and anterior overbite requiring consideration. Treatment plans should include initial preparation; caries control and defective restoration repair; pathological tooth migration correction; provisional stabilization; endodontics; surgical periodontics, postsurgical endodontics; clinical and radiographic re-evaluation at 12 weeks; final restorative phase and final periodontic, endodontic, and prosthetic evaluations prior to final cementation. The authors felt that the final prosthesis should be divided into segments of ≤ 6 units, occlusal forces should be directed along the long axis of the teeth, and initial cementation should be temporary (3 months) followed by re-evaluation.

OTHER PERIODONTAL-PROSTHODONTIC RELATIONSHIPS

Soft Tissue

The environment associated with pontic placement is a prime determinant of long-term success of a fixed partial denture. If soft tissue form and surface characteristics are deemed unacceptable, corrections should precede fabrication of the restoration (Hunt, 1980). Circumstances permitting, pontics should be placed over keratinized tissue rather than alveolar mucosa. Ridge augmentation may be accomplished by internal connective tissue grafts, free soft tissue onlay-autografts, or ridge transposition. When the ridge is covered by excessive amounts of soft tissue, ridge reduction can be accomplished by gingivoplasty or internal soft tissue wedge reduction (e.g., tuberosity reductions). Osseous sur-

gery may be indicated when a bony portion of ridge is covered by a thin layer of soft tissue. Ridge reduction surgery may be required to increase the vertical clearance between the residual ridge and opposing occlusion. Surgery (vestibuloplasty-free soft tissue autograft) may also be required in areas where shallow vestibules complicate oral hygiene or predispose to adverse interactions between the soft tissue and pontics associated with fixed or removable prostheses.

Allen (1988) described mucogingival treatment techniques to enhance anterior tooth esthetics. He recommended having the gingival margins on incisors peak slightly distal to the midline of the teeth. Central incisors, with an average length of 11 to 12 mm, should be 1.5 mm longer than laterals. These recommendations should take into account whether full coverage restorations are to be utilized with root exposure avoided if restorations are not planned.

Crown Contour

Eissmann (1971) discussed physiologic design criteria for effective restorative function, comfort, and hygiene. The author advocated axial forms that provided protection and stimulation. Protective contours were described as convex (prominences) while stimulatory contours were concave (sluiceways, embrasures). Protective convexities relate to clinical crown length, decreasing in prominence as the distance from the occlusal table to the free gingival margin increases.

Physiologic tooth contouring is directed at minimizing plaque retention by exposing the largest possible area of the clinical crown to cleansing by food flow patterns, musculature, and mechanical oral hygiene devices. Overcontouring causes plaque accumulation and inflammation and is potentially more detrimental to the periodontium than undercontouring (Youdelis et al., 1973).

Supragingival and subgingival contours should have a flat emergence profile or angle (Kay, 1985). The subgingival contour is an extension of this relationship into the sulcus. The character and dimension of the gingival tissues are the primary variables affecting subgingival contours. Thin friable tissue is less tolerant of subgingival restorative invasion and is more susceptible to shrinkage and marginal recession.

Becker and Kaldahl (1981) emphasized access for oral hygiene and suggested guidelines for crown contours. The guidelines included: 1) "Flat," not "fat" buccal and lingual contours: the normal bucco-lingual contour of teeth without caries is flat with a bucco-lingual bulge, usually $<$ 0.5 mm wider than the cemento-enamel junction. 2) Open embrasures: embrasures should be wide enough to allow adequate room for the papilla and accessibility for cleaning. 3) Contact areas should be in the coronal third of the crown and buccal in relation to the central fossa. This creates a large lingual embrasure for optimum health of the lingual papilla. 4) Exposed furcations should be "fluted" or "barreled out."

Pontics

Stein (1966) examined 500 pontics to determine associated soft tissue reactions of various pontic designs and materials. Ridges with visible inflammation were termed "involved," while those without visible signs of inflammation were considered "uninvolved." All ridges exhibited histologic signs of connective tissue infiltration with the involved areas demonstrating more superficial and severe infiltration. When tissue was excised from the residual ridge, a transient reduction of 1 mm in tissue height occurred; however, the original ridge height returned within 1 year regardless of whether a pontic was placed. When polished ridge-lap pontics were placed, 90% produced visible inflammation of mucosa regardless of the material (gold, porcelain, acrylic); furthermore, daily flossing under the pontic aggravated the problem. The author concluded that pontic design was more important than the material used in the pontic construction. The ideal design should have pinpoint, pressure-free contact on the facial slope of the ridge, and all surfaces should be convex, smooth, and highly polished or glazed. This configuration has been termed the "modified ridge-lap" pontic. This pontic design offers the most favorable balance between comfort, support, and hygiene, but may appear unesthetic anteriorly. Becker and Kaldahl (1981) recommend the modified ridge-lap design posteriorly and the ridge-lap facing design anteriorly.

Overdentures

Johnson and Sivers (1987) discussed periodontal considerations for overdentures. Selection of abutment teeth is based on prosthodontic and periodontal considerations, including bone support and architecture, width of attached gingiva, tooth mobility, root anatomy, and tooth position. A minimum of 5 to 6 mm of bone support is suggested. A greater width of attached gingiva may be necessary when the tissue is subjected to mechanical stresses and plaque accumulation accompanying the prosthesis. Mobility patterns are often improved by reducing the crown to root ratio during abutment preparation. Molars and furcated maxillary premolars make poor abutment choices due to concavities, grooves, and possible furcation invasions. However, resected teeth may be suitable abutments. Periodontal surgery may be necessary to reduce pockets, augment attached gingiva (keratinized tissue), and increase vestibular depth where indicated.

Maintenance is essential to the long-term success of overdenture abutments. Hygiene adjuncts using end-tufted brushes and daily application of fluoride are beneficial. Overdenture abutments generally have an increase in gingivitis, and patients with poor oral hygiene and sporadic professional maintenance frequently experience increased caries and attachment loss at overdenture abutments.

Longitudinal Evaluation of Periodontal-Prosthetic Treatment

Nyman and Lindhe (1979) longitudinally evaluated combined periodontal and prosthetic treatment of patients with advanced periodontal disease. Participants included 251 patients with dentitions devoid of 50% or more of the periodontal support who had received periodontal surgery and prosthetic rehabilitation. Initial clinical and radiographic evaluations were completed following treatment and annually for 5 to 8 years. Low plaque and gingival index scores were maintained over the 5 to 8 year period. No additional attachment loss occurred and bone levels were maintained for all types of fixed partial dentures, including cantilevers. This study suggests that periodontal tissues surrounding fixed partial denture abutments do not react differently from tissues around non-abutment teeth. It should be noted that supragingival margins and excellent oral hygiene were consistently observed in the study population.

Nyman and Ericsson (1982) randomly selected 60 fixed partial dentures (FPD) from the previous study at the 8 to 11 year point for further evaluation. Radiographic assessments of the root surface area, percentage of root in bone, normal PDL area for pontics, and percent of abutment to pontic PDL surface area were determined. Only 5 FPDs met Ante's law, with the PDL surface area of the abutments being equal to or greater than the calculated area of the pontic root surface. The bone heights for all the FPDs were unchanged over the observation period. None of the FPDs failed due to periodontal reasons over the 5 to 8 year period, while 26 of 332 failed as a result of loss of retention, fracture of the abutment teeth, or fracture of the bridgework.

Silness (1980) reviewed selected investigations of periodontal health adjacent to fixed prostheses, examining the concepts that had emerged, and relating these to actual clinical practices. The review included 342 individuals with 357 bridges that had been in place up to 6 years. The patients were divided into 2 groups. Group 1 consisted of 197 subjects who had received periodontal treatment and were given oral hygiene instructions prior to prosthodontic treatment. Group 2 was comprised of 145 subjects who had not received these instructions. Further subgroups and sub-studies were devised to evaluate the distributional pattern of plaque, gingivitis, pocket formation, periodontal effects of the crown margins, influence of full and partial crowns, the relationship between the pontic and the periodontal condition, and the effect of splinting adjacent teeth. Abutment teeth were compared to uncrowned contralateral control teeth. The PI, GI, and PD affected interproximal surfaces more than buccal surfaces, with no difference between abutments and controls. Group 1 values were lower than Group 2 values. Bridges did not change the distribution of plaque and periodontal disease. Subgingival margins showed higher PI, GI, and PD values than margins above the gingival margin, which, in turn were better than margins even with the gingiva. The authors suggest that the subgingival zone should be as smooth as possible in order to avoid harmful tissue reactions; splints should only be used when retainer margins are supragingival and embrasures facilitate cleaning; and pontics should be convex in all directions.

In 38 patients who had received removable partial dentures (RPDs) 8 to 9 years previously, Chandler and Brudvik (1984) evaluated clinical parameters and caries rates, comparing the results with those recorded at the 1 to 2 year periods. Teeth were categorized as abutments, indirect abutments (with rest seat), and non-abutments. Thirty-three (33) of 44 RPDs were still serviceable, with occlusal function rated fair to good. Of the 291 teeth included in the study, 8 were lost in 5 patients. All were mandibular teeth and 3 were lost as a result of periodontal disease. Approximately 50% of the caries occurred on surfaces covered by the RPDs, but no significant differences were observed between the 3 groups of teeth. Probing depths were significantly increased for all 3 groups when compared to the pre-insertion depths, but no significant differences were observed between the groups. The abutment teeth had significantly greater increases in mobility when compared with the 2 time periods. Although no significant differences in gingival inflammation were reported for the 2 time periods, by 8 to 9 years, more gingival inflammation was present in regions covered by the RPDs. Alveolar bone level changes were not significant between either the time periods or the groups. These results of this study indicate that long-term dental health may be maintained in patients wearing RPDs.

Bergman et al. (1985) also reported that RPDs did not compromise long-term dental health. They completed a 10-year longitudinal study of 30 patients who had been treated restoratively, periodontally (as required), and prosthetically with RPDs. An attempt was made to place crown margins supragingivally when possible. Conventional RPDs were designed and fabricated to keep denture bases, clasps, and bars as far from the gingiva as possible. Recalls for oral hygiene and necessary restorative treatment were maintained. Clinical parameters including oral hygiene, GI, PD, mobility, alveolar bone levels, decayed and filled surfaces, and prosthetic concerns, were recorded at day 0, and at 1, 2, 4, 6, and 10 years. During the 10-year follow-up period, no changes were observed relative to the plaque and gingival indices, probing depth, and mobility. Small, insignificant differences were found in proximal marginal alveolar bone levels for direct abutment teeth with distal-extension RPDs. The number of surfaces at risk for decay or restoration that were restored increased from 50.5% to 54.2% over the 10-year period, with an average of 1 new surface per patient with caries. Restoration service time averaged approximately 8 years before replacement. The authors concluded that with an effective preventive dentistry program, RPDs will not adversely impact the progression of periodontal disease or carious lesions.

SUMMARY

One of the primary goals of restorative therapy is the re-establishment of function commensurate with contour and design that will facilitate the long-term maintenance of periodontal health. Careful attention to detail relative to the effects of crown contour, margin placement and pontic design on the surrounding soft tissue is essential if this goal is to be achieved.

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CHAPTER 14. IMPLANTS

DEFINITIONS

Osseointegration: A direct contact, on the light microscopic level, between living bone tissue and an implant.

Biointegration: A bonding of living bone to the surface of an implant which is independent of any mechanical interlocking mechanism.

Peri-Implantitis: A term used to describe inflammation around a dental implant and/or its abutment.

CLASSIFICATIONS

Lekholm and Zarb (1985) proposed classifications for residual jaw shapes and bone resorption patterns following extraction. A classification was also proposed for associated bone quality. A brief description of these classifications follows:

Jaw Shape-Bone Resorption Pattern: 1) most of the alveolar ridge is present; 2) moderate residual ridge resorption has occurred; 3) advanced residual ridge resorption and only basal bone remains; 4) some resorption of basal bone has started; and 5) extreme resorption of basal bone has taken place.

Bone Quality: 1) homogenous compact bone; 2) thick layer of compact bone surrounds a core of dense trabecular bone; 3) thin cortical bone with dense trabecular bone of favorable strength; and 4) thin layer of cortical bone with low density trabecular bone.

IMPLANT/TISSUE INTERFACE

The implant/soft tissue interface is similar to that present in the natural dentition, with a functional junctional epithelium containing basal lamina and hemidesmosomal attachments. McKinney et al. (1985) suggested that the dense linear body of the basal lamina was composed of glycoproteins produced by fibroblasts and that junctional epithelial cells secrete laminin, resulting in a basal lamina as the epithelium migrates down the implant surface. Although Jansen et al. (1985) reported that this attachment was only associated with hydroxyapatite-coated implants, ultrastructural studies have revealed a similar attachment to titanium (Gould et al., 1981). Following exposure of titanium to air or water, a very stable 3 to 5 Å thick surface oxide layer has been demonstrated. Epithelial cell attachment to this surface oxide layer (Kasemo and Lausmaa, 1985). Brunette (1988) studied the orientation of epithelial cells in grooved titanium surfaces and observed that migration and cell orientation follow the axis of the grooves. The author suggested that horizontal grooving of the non-screw titanium surface (titanium collar-abutment cylinder) may impede apical epithelial migration.

While a lack of an absolute biologic attachment between the implant and surrounding connective tissue has been suggested (Stallard, 1985), in vitro fibroblast attachment to titanium surfaces has been demonstrated by Dmytryk et al. (1990). In addition, circumferential connective tissue fibers have been observed in association with the implant post (James, 1976). Ultrastructural evaluation of the human connective tissue-implant interface revealed bundles of collagen, often directed towards the implant surface, with a 20-nm thick proteoglycan layer between the connective tissue and the titanium oxide surface. No evidence of toxic or foreign body reaction has been seen between the implant-connective tissue interface (Donley and Gillette, 1991). Fiber thickness and orientation are thought to be dependent on the functional load placed on the implant (Stallard, 1985). Fibroblast orientation has been found to differ, depending on the texture of the titanium surface. Inuoe et al. (1987) found no distinct cellular orientation when cells migrated onto porous titanium surfaces. However, Lowenberg et al. (1987) reported a more favorable orientation of cells to porous surfaces when compared to smooth surfaces. Schroeder et al. (1981) noted perpendicular connective tissue fiber attachment into rough plasma-sprayed titanium surfaces. Application of tensile strength removed the sprayed surface from the implant while fiber attachment to the plasma-sprayed titanium surface was maintained.

Osseointegration has been observed between the endosteal-titanium implant interfaces. Sections viewed with electron microscopy have revealed a proteoglycan layer (containing calcified tissue) in direct contact with the titanium oxide surface. The proteoglycan layer is 40 to 200 Å thick (Albrektsson, 1985). In addition, true bonding between titanium and bone has been demonstrated by Steinemann et al. (1986) and Buser et al. (1990). Van der Waal's bonding, hydrogen bonding, and covalent and ionic bonding have been observed between the biomolecular and implant surface (Kasemo and Lausmaa, 1985). The biocompatibility of titanium implants was demonstrated by Buser et al. (1990) when formation of a distinct layer of cementum was observed on the implant surface. In a 12-month study, plasma-sprayed titanium implants were placed in monkeys. Non-submerged hollow cylinder implants were placed in areas of retained root tips. In sites where retained roots directly contacted the implant bed, cementum apposition was noted on titanium surfaces. The collagen fibers of the periodontal ligament were attached perpendicularly to the implant surface and extended into the opposing bone.

Osseointegration also occurs with hydroxyapatite (HA) coated implants (Meffert et al., 1987). The author suggested that only hydroxyapatite, and not titanium, was capable of

true bonding to bone. Bagambisa et al. (1990) reported that an even carpet of multilayered osteoblasts covered the surface of HA implants, with bone infiltrating the porous surface. Hydroxyapatite was not osteoinductive but did act as a nucleation site for osteoid material. Bone formation occurred through epitaxial crystal growth.

CLINICAL CHARACTERISTICS

Marginal tissue response to titanium implant was evaluated by Adell et al. (1986) and Lekholm et al. (1986A). Lekholm et al. reported a relation between plaque and gingivitis and between gingivitis and probing depths, while Adell and colleagues did not. The Lekholm study was cross-sectional and the Adell study was longitudinal. Lekholm et al. measured gingivitis (80%) by bleeding on probing while Adell et al. measured gingivitis (15 to 20%) based on visual signs of inflammation. In the Adell study, plaque was present in only 25 to 30% of the implant sites while 54% in the Lekholm study had plaque. Lastly, the Adell study population was composed of edentulous patients whereas 8 of 20 of Lekholm patients were partially edentulous. Both groups reported increased recession, with the same mean bridge to gingiva distance (3.2 mm). Attached gingiva was present in 65% (Adell) and 51% (Lekholm) of all buccal and lingual surfaces. Probing depths were generally low, with none > 5 mm in the Adell group and 15% > 6 mm in the Lekholm study. Adell et al. reported 0.9 mm bone loss the first year and 0.05 mm annually for the next 2 years (based on radiographic findings). Both groups reported minimal histological inflammation, with no inflammation in 49% of the biopsies and slight inflammation in 33% (combined results). Currently, mobility and radiographic bone loss represent the most reliable methods of detecting implant failure (Newman and Flemmig, 1988).

MICROBIOLOGY

The implant microflora are derived from the natural flora of the oral cavity (Heimdahl et al., 1983). Bacterial adherence to enamel and titanium seem to differ, with titanium exhibiting a 5-fold decrease in adherence of *Actinomyces viscosus* and a slight decrease in adherence of *Streptococcus sanguis* (Wolinsky et al., 1989). Rams et al. (1984) noted that bacteria from healthy edentulous implant sites were composed primarily of non-motile coccoid cells (64.2%), filamentous rods, and minimal numbers of spirochetes (2.3%). "Corncob" formations were also commonly seen. In a study evaluating colonization of newly exposed titanium implants, Mombelli et al. (1988) reported no significant changes in the proportions of microorganisms over a 6-month period. Eighty percent (80%) of the cultivated bacteria were Gram-positive facultative cocci. The authors concluded that in health, the subgingival implant microbiota were similar to that of the natural healthy dentition. Apse et al. (1989) compared the implant microflora in edentulous and partially edentulous patients, noting greater numbers of motile forms and less black-pigmented *Bacteroides* and wet spreaders in the edentulous group. In the partially edentulous group, there was no significant predilection for any type of bacteria at either the implant or tooth sites. The authors suggested that the differences between edentulous and partially edentulous implant sites may be the result of contamination of the peri-implant sites by pathogens from periodontal pockets. These findings are in agreement with Lekholm et al. (1986B) who also reported a similar microbial composition adjacent to natural teeth and titanium fixtures. Non-motile rods, filaments, and fusiforms comprised 50% of the microflora in partially edentulous healthy implant and tooth sites. The remainder was comprised of cocci (25%) and motile rods (25%). Few spirochetes, and no *Actinobacillus actinomycetemcomitans, Bacteroides gingivalis*, or *Prevotella intermedia* were noted around implant sites.

As inflammation and probing depths (> 5 mm) increase, elevated levels of spirochetes and decreases in coccoid cells are noted (Rams et al., 1984). An increase in the number of Gram-negative anaerobic flora is observed, with equal proportions of *Bacteroides, Fusobacterium*, and vibrios (Newman and Flemmig, 1988).

Failing implants have been associated with a florium which differs from that seen in health. Rams et al. (1983) evaluated the microbiota around 3 failing ceramic implants in edentulous patients. An increase in spirochetes (31 to 56%) and motile rods (15 to 31%) with a decrease in coccoid cells (19 to 31%) was reported. "Brush forms," composed of spirochetes and non-motile rods, were also noted. Small and intermediate size spirochetes were observed with electron microscopy. Mombelli et al. (1988) reported similar findings, noting a decrease in cocci and an increase in spirochetes, *Fusobacterium*, and *Actinomyces*. The microflora associated with failing implants are very similar to that of periodontal disease. The composition of implant-associated plaque was consolidated and presented in chart form by Newman and Flemmig (1988) (See Table 1).

LONG-TERM STUDIES

When interpreting long-term implant results, the reader should consider the criteria for success, type of implant system used, site of implant placement (maxillary or mandibular), and edentulous status (partially or fully edentulous). The criteria for success may differ between studies, with many of the earlier studies not including implants that failed, but the prostheses were retained or implants left sleeping as failures. More recent studies have determined success based on a lack of mobility and lack of peri-implant radiographic radiolucency. Albrektsson (1986) proposed the following criteria for evaluation of implant success: 1) no clinical mobility; 2) no radiographic peri-implant radiolucencies; 3) < 0.2 mm annual bone loss following the implant's first year of service; and

TABLE 1. SUBGINGIVAL PLAQUE COMPOSITION IN OSSEOINTEGRATED TITANIUM FIXTURES

Stable Implants
Streptococcus sanguis (6.9%)
Streptococcus mitis
Streptococcus acidominimus
Peptostreptococcus
Peptococcus
Actinomyces viscosus (4%)
Actinomyces naeslundi
Veillonella parvula
Fusobacterium nucleatum (6.5%)
Non-pigmented *Bacteroides*
Black-pigmented *Bacteroides*
Prevotella intermedia (0.9%)
Campylobacter
Vibrios
Motile rods
Spirochetes
Curved rods
Failing Implants
Black-pigmented *Bacteroides*
Prevotella intermedia (5.7%)
Capnocytophaga
Fusobacterium ssp (15.3%)
Spirochetes
Motile rods
Surface translocating bacteria
Curved rods
Staphylococcus aureus
Pseudomonas aeruginosa
Klebsiella pneumonia
Enterobacter cloacae

Taken from Newman and Flemmig, *J Dent Ed* 1988;52:737. Reproduced with permission.

4) lack of pain, infection, paresthesia, or violation of the mandibular canal.

The Branemark implant system has been extensively evaluated with multiple long-term studies from various investigators and centers. Adell and co-workers (1981) reported 5 to 9 year single-center success rates of 91% for the mandible and 81% for the maxilla. Albrektsson et al. (1988) performed a multi-center study which included placement of 8,139 consecutively placed implants in edentulous patients. They noted a 5 to 8 year success rate of 99.1% in the mandible and 84.9% in the maxilla. Seventy-eight percent (78%) of the mandibular failures occurred during the first year and 13% during the second year. More recently, Ahlquist et al. (1990) evaluated 269 implants over a 2-year period. The authors reported success rates of 97% in the mandible and 89% in the maxilla. Average bone loss during the first year was 1 mm for the mandible and 1.6 mm in the maxilla. During the second year, the mandibular sites lost an average of 0.04 mm and maxillary sites 0.1 mm. Implant placement in partially edentulous patients was evaluated by van Steenberghe et al. (1990). This 9-center study, which included 558 consecutively placed implants, showed a 1-year success rate of 96%. Reasons for loss or unaccounted for implants included failure to integrate (3.4%), patient withdrawal (2%), prosthodontic reasons (1.1%), and failure during prosthodontic treatment (0.2%). In summary, Branemark implants in both edentulous and partially edentulous patients have a success rate greater than 90%.

The Integral (Calcitek) hydroxyapatite-coated titanium system was evaluated by Kent et al. in 1990. This 5-year longitudinal follow-up study assessed 772 hydroxyapatite implants (Integral and IMZ) in 229 patients. The criteria for success were based on whether or not the implants were removed or left sleeping. Reasons for implant removal included lack of integration at stage 2, > 50% bone loss at stage 2, lack of bone support, loss of bone during function, malposition, and psychiatric reasons. Of the 745 implants placed, 28 were removed and 1 was left as a sleeper, for a success rate of 94.6%. This compares well with Golec (1990) who noted a 93.7% success in the 2,249 implants studied over a 5-year period.

The IntraMobile Cylinder (IMZ) implant system was originally introduced in 1978. In 1984, a plasma-sprayed hydroxyapatite (HA)-coated surface was introduced. Kirsch and Ackerman (1989) published the results of a 10-year study and reported an overall success rate of 97.8%. Implant failure was defined as failure to integrate during primary healing, implant mobility after functional restoration, or soft tissue sequelae. Examples included bone loss, pain, and change in clinical parameters around the implant (i.e., GI, bleeding, exudate). While the 10-year data included evaluation of both types of implants (i.e., titanium plasma-sprayed and HA-coated), only the 4-year data for the modified (HA-coated) IMZ implants are included. A total of 804 of these implants were placed in 333 patients, with 72.9% placed in partially edentulous areas. Two of the 804 implants failed, for a success rate of 99.8%. These findings are in agreement with Babbush et al. (1990) who reported a 97 to 98% 10-year success rate in the 3,436 implants placed. These results are in contrast to Kent et al. (1990) who noted a 63%, 5-year success rate for 27 HA-coated IMZ implants.

The Core-Vent implant system has been evaluated by several researchers with contradictory results. Malmquist and Sennerby (1989) noted success rates ranging from 58 to 77%. Albrektsson and Lekholm (1989) reviewed multiple implant systems. In addition to those mentioned above, the authors also reviewed the ITI and sapphire implant systems. Success rates of 85 to 92% over a 1- to 92-month time frame was reported for the ITI implant system (Schroeder et al., 1988), while the success rate was 77.7% for sapphire implants (Koth et al., 1988).

DIAGNOSIS AND PROGNOSIS

When evaluating patients for implant placement, a multidisciplinary approach is necessary to ensure maximal benefit from the therapy provided. A thorough medical history

should be taken to rule out immediate anesthetic and surgical risks, psychologic and psychiatric risks, medical threats to long-term retention, and long-term deleterious effects of implants on health (Matukas, 1988). Adell et al. (1981) proposed absolute contraindications for implant placement which included pregnancy, hemophilia, granulocytopenia, steroid use, prophylactic antibiotics, brittle diabetes mellitus, Ehlers-Danlos syndrome, osteoradionecrosis, radiation, renal failure, organ transplantation, anticoagulation therapy, hypersensitivity, fibrous dysplasia, and regional enteritis.

In addition to a thorough medical history, a comprehensive dental examination should be performed. Radiographic examination should include a panoramic, periapical, or occlusal film, cephalographs, tomograms, or computed tomography scan. In dentate patients, a periodontal examination should be performed and periodontal disease controlled prior to implant placement. Variables which can affect implant success should be assessed. These include bone type, dental arch (maxilla versus mandible), implant location (anterior versus posterior), anatomical variations, presence of natural dentition, implant type, and operator expertise. Taken together, these variables may aid in determining prognosis. Jaffin and Berman (1991) noted that the quality of bone was the single greatest determinant in predicting fixture failure. Type IV bone had fixture loss rates of 44% for the maxilla, 37% for the posterior mandible, and 10% for the anterior mandible. This was in contrast to type I, II, and III bone with fixture loss rates of 3.6% for the maxilla, 6.8% for the posterior mandible and 1.2% for the anterior mandible. van Steenberghe et al. (1990) also associated implant failures with quality of bone, with 22% of patients with type IV bone patients having one or more implant failures. The authors reported gender (failure: males, 13%; females, 7%) and small fixture size (failures: 7 mm, 10.7%; 10 to 13 mm, 5.9%; 15 mm, 0%) as factors associated with implant failures. Ahlqvist et al. (1990) noted increased bone resorption in anteriorly placed implants (versus posterior) and in patients with minor preoperative alveolar bone resorption. Another factor which may affect implant success is whether or not the patient is edentulous. Because implants placed in dentate patients harbor the same bacteria as the natural teeth, the likelihood of periimplant breakdown may be increased.

Implant placement in irradiated patients has not been without controversy. Matukas (1988) stated that implant placement in a site with a history of radiation > 4,000 rads is contraindicated and Adell et al. (1981) listed radiation as an absolute contraindication. However, Albrektsson et al. (1988) reported 100% success rate in the mandible (56 implants) and 88% in the maxilla (16 implants) when Branemark implants were used. These rates compare well with non-irradiated patients, supporting the use of implants in selected radiation cases.

SURGICAL TECHNIQUES

Soft Tissue Management

The soft tissue management during placement and uncovering of the implant should include a review of proper access and the anatomy of the implant site. In a review of implant surgical techniques, Moy et al. (1989) discussed the three most common flap designs for stage one surgery: These include the labial split-thickness flap, mid-ridge flap and lingual-palatal off-ridge flap. In addition, a split-thickness palatal flap (Krauser, 1989) or an overlapped flap (Langer and Langer, 1990) may be used.

The labial split-thickness flap was described by Moy et al. (1989) as providing the "best access for visibility, ease of placement of implants, and accommodation for surgical stents." This procedure is generally used in the mandibular anterior region. A superficial semilunar incision is made laterally on the mucosa from canine to canine at two-thirds the sulcular (vestibular) depth. The fibers of the mentalis and the underlying periosteum are then incised with the blade placed perpendicular to the labial plate. Blunt dissection is performed subperiosteally, proceeding posteriorly until the mental foramen is palpated. Once the mental nerve is visualized and retracted, the dissection continues in a coronal direction until the lingual aspect of the ridge is visualized. Care must be taken to avoid excessive facial dissection, as it may lead to coronal movement of the flap, thus decreasing vestibular depth. Following implant placement, closure of the flaps is achieved in layers to ensure complete closure and to reduce the possibility of hematoma formation. This flap design works well everywhere except in the maxillary anterior region, where esthetics is of concern. The main disadvantage of this procedure includes occasional opening of the incision with subsequent delayed healing in the vestibular area (Krauser, 1989).

In the mandible, the mid-ridge flap is used when there is concern about injury to the mental nerve. Reflection of the flap is limited, thereby preventing visualization of the complete anatomy of the ridge. Because of limited access, osteoplasty is difficult to perform. In addition, cases with knife-edge ridges have increased incidence of dehiscences with this flap approach (Moy et al., 1989).

The lingual/palatal off-ridge flap is often used in the maxillary anterior region. This design permits adequate access for visualization and for use of a surgical stent, because the blood supply is limited on the palatal and healing often occurs by secondary intention. In addition access for closure is more difficult with this flap design (Moy et al., 1989).

In the split-thickness palatal flap technique (Krauser, 1989), a full-thickness flap is reflected from the palate to the facial, which continues as a split-thickness dissection on the buccal. According to the author, this provides a biologic seal over the labial periosteum which can be especially useful in cases where possible facial perforation of the implant is anticipated. With the palatal approach, the

blood supply to the facial pedicle flap is limited, and flap necrosis with hematoma formation often develops.

Another surgical flap modification for stage 1 surgery is the overlapped flap design described by Langer and Langer (1990). A partial thickness flap is reflected either from the buccal or palatal aspect of the ridge. In the mandible, the buccal approach is the technique of choice. In the maxilla, the palatal approach is used. With the palatal approach, the split-thickness flap becomes thicker as it goes apically until bone is reached, in an effort to preserve the flap blood supply. Two beveled vertical incisions are then made on the inner flap, to facilitate flap reflection. Vertical incisions are placed on the outer flap as needed. The full-thickness double flaps are then reflected, providing visualization of the ridge. Following implant placement, the flaps are adapted and sutured in place with vertical or horizontal mattress sutures. In order to prevent contamination of the fixtures, the sutures should not completely penetrate the underlying flap. Superficial tissue necrosis may occur if sutures are placed too tightly or if inadequate pressure is placed and a hematoma develops under the flaps.

Stage 2 surgery requires much less access and can be accomplished one of two ways. A punch may be used to remove the tissue over the cover screw or a linear incision made. The punch technique does not allow for soft or hard tissue recontouring. In addition, the minimal keratinized tissue present at some sites may be compromised with this procedure. An alternative technique is the use of a full-thickness linear incision over the cover screw. This provides access for thinning the soft tissues or recontouring the bone, while preserving keratinized tissue. No sutures are required for the punch technique. A continuous sling with interproximal mattress suture works well with the linear incision technique (Moy et al., 1989). In addition, keratinized tissue may be restored and esthetics enhanced with periodontal plastic surgical techniques (Israelson and Plemons, 1993).

Implant Placement

During implant placement undue trauma to the osseous structures should be avoided. The critical temperature above which bone will necrose is 47°C. Handpiece speed should be controlled and irrigation provided to prevent irreversible damage to bone. Tapping of screw type implants is performed at low speeds (15 rpm) to remove enough bone (0.125 mm) for a tight fixture fit. With screw-type implants, bicortical stabilization of the implant is desired. Overall success is dependent on the quality of the bone at the implant site. Screw-type implants are often placed in less than ideal angulations to achieve bicortical stabilization. With HA-coated cylinders and screws, lamellar-type bone formation can be expected even in spongy bone (Krauser, 1989). The length of the implant has been previously considered more important to the success of the implant than the diameter (van Steenberghe et al., 1990; Krauser, 1989). However, "wide" fixtures are now available which facilitate biocortical stabilization and provide equvalent or increased surface area.

Regenerative Procedures

Favorable results have been reported with regenerative procedures adjacent to implants. Dahlin et al. (1989) evaluated the use of expanded polytetrafluoroethylene (ePTFE) membrane over exposed (3 to 4 threads) newly placed implants in the rabbit tibia. By the sixth week, all of the exposed surfaces on the test sites were filled (3.8 mm) with new bone of uniform thickness. The control sites (no membrane) averaged only half as much bone fill (2.2 mm), with the bone becoming thinner coronally. Becker and Becker (1990) presented 4 implant cases where ePTFE implant augmentation material was used to enhance bone formation in extraction sites and over dehiscences of newly placed titanium implants. The augmentation material was left in place until the second stage surgery (6 to 8 months). Regeneration was noted in 3 out of 4 cases (fourth case still pending), with complete defect fill (3 mm) in both dehiscence defect sites and fill of 6 of the 8 threads in the extraction site. Histologic evaluation of the ePTFE material revealed bone woven into the membrane.

Mucosal Grafts

Soft tissue complications arising around implants include soft tissue proliferation, retractable margins, inadequate vestibular depth for hygiene, and peri-implant mucositis. While conservative treatment should be performed to eliminate these conditions, gingival grafting may be indicated if these measures are not successful. Where recession involves only one area, the vestibule is of normal depth and there is adequate donor tissue adjacent to the recipient site, a contiguous gingival graft may be considered (Horning and Mullen, 1990). Free gingival grafts are indicated when there is inadequate donor tissue adjacent to the recipient site or when the vestibular depth is minimal or inadequate. With the free gingival grafts, the mucosa should be periodically cut back during the healing phase to prevent coronal migration over the denuded connective tissue at the grafted site.

MAINTENANCE

While the reported long-term success rate for implants is good, it is important to monitor the patients and periodically evaluate and debride the implant. Maintenance intervals may vary depending on the patient's ability to maintain the area. However, 6 months is the maximum, with 3 months being the average. Orton et al. (1989) described the dental professional's role in implant monitoring and maintenance. While it is important to document clinical parameters such as probing depth, clinical attachment level, bleeding on probing, and plaque and gingival indices, their prognostic value is currently unknown. Progressive changes

in probing depths are more important than absolute depths. Mobility is a sign of implant failure. Periodic radiographs should be taken to evaluate for loss of implant integration (radiolucency) or excessive horizontal bone loss. With Branemark fixtures, the mean horizontal bone loss during the first year is approximately 1 mm and 0.1 mm/year thereafter (Albrektsson and Lekholm, 1989). Radiographs should be taken after the second-stage surgery at yearly intervals for the first 3 years to ensure proper fit of the abutment. When removing accretions from the implant, care must be taken not to damage the surface. Scratches on the titanium surface may result in increased plaque accumulation, corrosion, and a decrease in cell spreading (Fox et al., 1990). Rapley et al. (1990) evaluated various instruments and materials to determine the surface changes produced in titanium abutments. The following were evaluated: rubber cup, rubber cup with flour of pumice, air abrasive, interdental tapered brush, Eva yellow plastic tip, soft nylon toothbrush, universal plastic scaler, ultrasonic scaler, and a stainless steel scaler. Following instrumentation, the abutments were viewed with electron microscopy. Instrumentation with the interdental brush, Eva plastic tip, rubber cup, air abrasive, soft nylon toothbrush, or plastic scaler did not alter the implant surface. The rubber cup with the flour of pumice resulted in a smoother surface than the control. The air abrasive system produced a surface with dark discolorations, possibly indicative of surface corrosion. Metal scalers appeared to gouge the titanium surface and produced significant vertical grooving. The air abrasive caused severe roughening, which was readily evident at the macroscopic level. These findings are in agreement with Fox et al. (1990) who used a helium neon laser to evaluate implant roughness. Surfaces received 30 vertical strokes in a 2 mm area. Greater roughness was noted in surfaces treated with metal scalers (titanium curet > stainless steel) than those treated with plastic scalers or untreated controls (plastic scalers similar to control). A subsequent study by the same group (Dmytryk et al., 1990) reported cell attachment to be impaired in titanium surfaces scaled with metal instruments. The following hierarchy of fibroblast attachment was found: plastic curet > untreated control > titanium scaler > stainless steel scaler. This study suggests that other factors in addition to surface roughness may affect cell attachment since the titanium scaler which produced greater surface roughness than the stainless steel scaler did not affect cell attachment as much as the stainless steel scaler. In theory, metal scalers other than titanium cause corrosion, obliterating the titanium oxide surface layer and impairing cell attachment. These results suggest that metal scalers and instruments such as ultrasonic scalers or the air abrasive should not be used on titanium surfaces since damage to the titanium-oxide surface will occur. However, plastic scalers and rubber cup polishing with flour of pumice will maintain or enhance the titanium implant surface.

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