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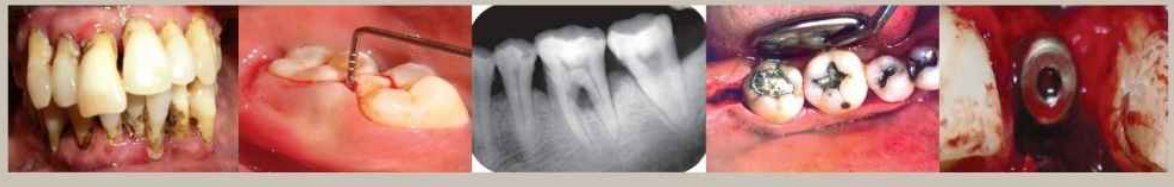
THIRD EDITION

Essentials of Clinical Periodontology and Periodontics



Shantipriya Reddy

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Essentials of Clinical Periodontology and Periodontics

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Essentials of Clinical Periodontology and Periodontics

THIRD EDITION

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Essentials of Clinical Periodontology and Periodontics

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To
My late grandfather, who had believed in my abilities,
it is because of whom I chose this profession
My grandmother, for her constant support
My father and mother, for always being there
My uncle for his continuous encouragement
My husband for his unique mentorship
My daughter and son for being so understanding

Preface to the Third Edition

I am extremely happy to present the third edition of *Essentials of Clinical Periodontology and Periodontics*. In planning the third edition, a firm commitment has been made to provide a thorough and complete text. An attempt has been made to revise and update all the chapters. The highlight of the edition is introduction of *KNOW MORE* section which contains latest additional information pertaining to each chapter.

Conscious attempt has been made not to disturb the clarity and simplicity of the main text which had gained immense popularity amongst the students. The edition also provides an add on, *Manual of Clinical Periodontics* which will guide the students in recording case history and performing clinical examination so as to arrive at a proper diagnosis and treatment planning. Also, special emphasis was given to instrumentation which will instruct the students in their clinical work. While describing all these, we have not only mentioned what steps are to be followed but also how to carry out these steps with the help of various illustrations and photographs.

I am grateful to all my colleagues and students who have helped me with their valuable suggestions, which in turn enabled me to write the edition in a more understandable fashion. I hope this book will help the students and practitioners in understanding periodontics in a simplified manner.

Shantipriya Reddy

Preface to the First Edition

The basic text when conceived in the year 2003 has been written in an attempt to make our understanding of periodontal disease accessible to the undergraduate students, general practitioners and dental hygienists. The *Essentials of Clinical Periodontology and Periodontics* is a learning textbook intended to serve the needs of several groups of dental care professionals and is written with credibility and readability maintained at every level. Undergraduate students especially will find it useful in integrating the concepts they have been taught in a more elaborate way. Clarity and simplicity in language has been my objective while writing this book. The organization of the chapters and the key points with review questions at the end of every chapter serve as a programmed-guide for the reader.

The text can be of help for the academicians to re-think the modes of presenting information and also as a model to test whether the students have grasped the concepts they have been taught and are able to use them in a practical manner.

All the efforts have been made to make the text as accurate as possible and the information provided in the text was in accordance with the standards accepted at the time of publication. In order to attain these goals, suggestions as well as critiques from many students and clinicians have been received and utilized.

Much of the style in the textbook is compact with adequate bibliographies. The reader is advised to use them to gain greater depth of knowledge. The way of periodontist is hard and the book will reflect the difficulty of that path. I hope that the textbook will fulfill all the requirements and expectations of the students and practitioners as this is a special branch of our profession. The field of periodontics remains a work in progress.

Shantipriya Reddy

Acknowledgments

Any book requires the help and assistance of others in order to be completed successfully. First and foremost, I would like to thank Dr Deepti Sinha for drawing the excellent diagrams inserted in the book. Special thanks must go to all my colleagues Dr Prasad MGS, Dr Amudha D and Dr Jaya for their contribution in writing *Manual of Clinical Periodontics*. I owe a deep sense of gratitude to all my postgraduate students (Dr Ravi Kumar Jirali, Dr Chaitali Agrawal, Dr Soumya Kambali, Dr Shweta Kumari, Dr Nirjhar Bhowmik, Dr Hrishikesh Asutkar) who have helped me relentlessly, while writing the edition. I would like to extend my special thanks to Dr Jeeth Rai, Dr Satish, Dr Keshav, Dr Shabeer, Dr Anilkumar for being so generous in helping me to collect the clinical photographs used in various editions.

My heartfelt thanks to Dr Srinivas K for writing the chapters *Desquamative Gingivitis* and *Oral Malodor* in the edition. I am also very grateful to Dr Deepak Daryani for providing me with some of the excellent photographs published in the edition. I would also like to thank Dr Ashwath (Smart design navigator) for helping me with animation photographs of various suturing techniques. The excellent cooperation of the publisher is also greatly acknowledged.

PROLOG

HISTORICAL BACKGROUND OF PERIODONTOLOGY

Various forms of gingival and periodontal diseases have affected the human race since the dawn of history. In the earlier historical records, almost all the writings have information regarding the diseases affecting oral cavity and majority of it is about periodontal diseases.

Early Civilizations

Summerians of 3,000 BC first practiced oral hygiene. Babylonians and Assyrians, who also have suffered from periodontal diseases, have treated themselves using gingival massage combined with various herbal medications. Research on embalmed bodies of the ancient Egyptians pointed out that periodontal disease was the most common of all the diseases. Medical writings of the time Ebers Papyrus had many references to gingival diseases and also contain various prescriptions for strengthening the teeth and gums.

The medical works of ancient India, Susruta Samhita and Charaka Samhita describe severe periodontal disease with loose teeth and purulent discharge from the gingiva and the treatment advised was to use a stick that is bitter for cleaning teeth.

Periodontal disease was also discussed in Ancient Chinese books. The oldest book written in 2,500 BC describes various conditions affecting oral cavity. Gingival inflammation, periodontal abscesses and gingival ulcerations are described in detail. They were among the earliest people to use the toothbrush to clean the teeth.

Middle Ages

The systematic therapeutic approach was not developed until the middle ages. This was a period of golden age of Arabic science and medicine. Avicenna and Albucasis made a refined, novel approach to surgical work. Albucasis had a clear understanding of calculus as etiology of periodontal disease and described the technique of removing it. He had also developed a set of scalers for removing calculus. He also wrote in detail on other treatment procedures like extraction of teeth, splinting loose teeth with gold wire, etc.

18th Century

Modern dentistry was developed in 18th century. Pierre Fauchard in 1678 who is rightly considered as Father of Modern Dentistry designed periodontal instruments and described the technique in detail. His book *The Surgeon Dentist* published in 1728 presented all aspects of dental practice (i.e. restorative dentistry, prosthodontics, oral surgery, periodontics and orthodontics). Fauchard wrote in that, confections and sweets destroy the teeth by sticking to the surfaces producing an acid. John Hunter (1728-93) known as an anatomist, surgeon and pathologist of 18th century wrote a book entitled *The Natural History of the Human Teeth* describing the anatomy of the teeth and their supporting structures with clear illustrations.

Thomas Berdmore (1740-85) known as *Dentist to His Majesty* published the Treatise in the disorders and deformities of the teeth and gums. He not only offered detailed descriptions of instrumentation but also stressed on prevention.

19th Century

A German born dentist, Leonard Korecker in his paper Philadelphia Journal of Medicine and Physical Sciences mentioned the need for oral hygiene by the patient, to be performed in the morning and after every meal using an astringent powder and a toothbrush.

Levi Spear Parmly was considered the Father of Oral Hygiene and the Inventor of Dental Floss. The name *Pyorrhea Alveolaris* was used to describe periodontal disease. John W Riggs was the first individual to limit his practice to periodontics and was considered the first specialist in this field. Periodontitis was known as *Riggs disease.* Several major developments took place in the second half of the 19th century starting the era called modern medicine.

The first was the discovery of anesthesia and second scientific breakthrough was made by Louis Pasteur who established the Germ Theory of Disease. The third scientific finding was the discovery of radiographs by Wilhelm Roentgen.

Also the late 19th century has witnessed a proper understanding of the pathogenesis of periodontal disease based on histopathologic studies. GV Black in 1899 gave the term gelatinous microbic plaque and described its relationship to caries. Xenophon recognized ANUG in 4th century BC Salomon Robicsek developed a surgical technique called gingivectomy.

20th Century

Early 20th century witnessed major changes in the treatment of periodontal disease. Gottlieb published extensive microscopic studies of periodontal diseases in humans. It was realized that, removal of calculus and other deposits was not enough. In addition, removal of periodontal pockets was necessary to control the disease.

Leonard Widman and Newman described flap surgery for the removal of periodontal pockets. Removal of bone was considered essential at that time.

After World War II, the focus was on periodontal research and this led to a better understanding of the pathological, microbiological and immunological aspects of periodontal disease.

The first workshop in periodontology was conducted in 1951. It was realized at that time, scientific methods should be introduced in the periodontal research. Subsequent workshops conducted in periodontics have witnessed significant scientific contributions in the field of periodontics.

JOURNALS OF PERIODONTOLOGY

The various journals of periodontology available are:

- The Journal of Periodontology by Robert J Genco.
- Journal of Periodontal Research by Isao Ishikawa, Jorgen Slots, Maurizio Tonetti
- Journal of Clinical Periodontology by Jan Lindhe.
- Periodontology 2000 by Jorgen Slots.
- International Journal of Periodontics and Restorative Dentistry in India by Myron Nevins.
- Journal of Indian Society of Periodontology.

Contents

Section 2(B): Surgical Therapy

Part I

The Normal Periodontium

1 Chapter

Anatomy and Development of the Structures of Periodontium

INTRODUCTION

The term periodontium arises from the Greek word *peri* meaning around and *odont* meaning tooth, thus it can be simply defined as the "tissues investing and supporting the teeth". The periodontium is composed of the following tissues namely alveolar bone, root cementum, periodontal ligament (supporting tissues) and gingiva (investing tissue).

The various diseases of the periodontium are collectively termed as *periodontal diseases.* Their treatment is referred to as *periodontal therapy.* The clinical science that deals with the periodontium in health and disease is called *periodontology.* The branch of dentistry concerned with prevention and treatment of periodontal disease is termed *periodontics* or *periodontia.*

EXTERNAL ANATOMIC FEATURES

The oral mucosa consists of three zones:

1. *Masticatory mucosa:* It includes the gingiva and the covering of the hard palate.
2. *Specialized mucosa:* It covers the dorsum of the tongue.
3. *Lining mucosa:* It is the oral mucous membrane that lines remainder of the oral cavity. Among all the structures of the periodontium, only the gingiva is visible clinically. The gingiva is divided anatomically into free or marginal, attached and interdental gingiva. The border or groove between marginal and attached gingiva is called as a free gingival groove, a shallow depression on the faciogingival surface that roughly corresponds to the base of the gingival sulcus. The junction between the attached gingiva and alveolar mucosa is called as *mucogingival line or junction* (Fig. 1.1).

The normal gingiva is pink in color (salmon coral pink) and accumulation of melanin pigmentation is normal. The surface of the gingiva exhibits an orange peel-like appearance referred to as *stippling.* In health, the gingiva completely fills the embrasure spaces between the teeth and is known as the *interdental gingiva.* In the posterior teeth, where the contact areas between the teeth are usually broad, the interdental gingiva consists of two papillae, facial and

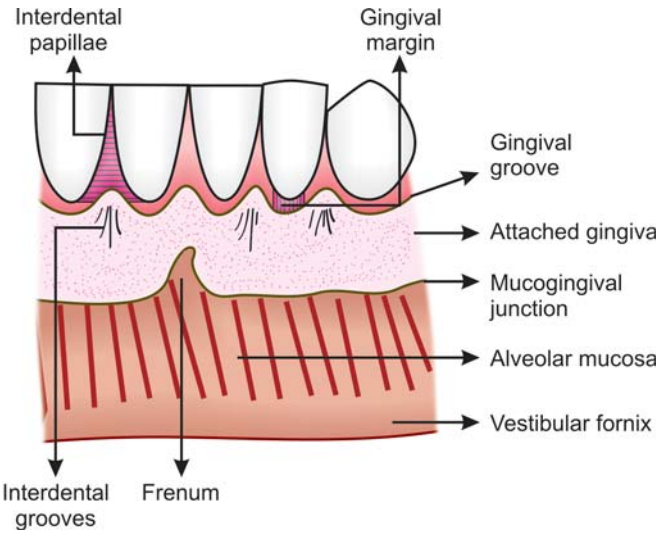

Fig. 1.1: Surface characteristics of the clinically-normal gingiva

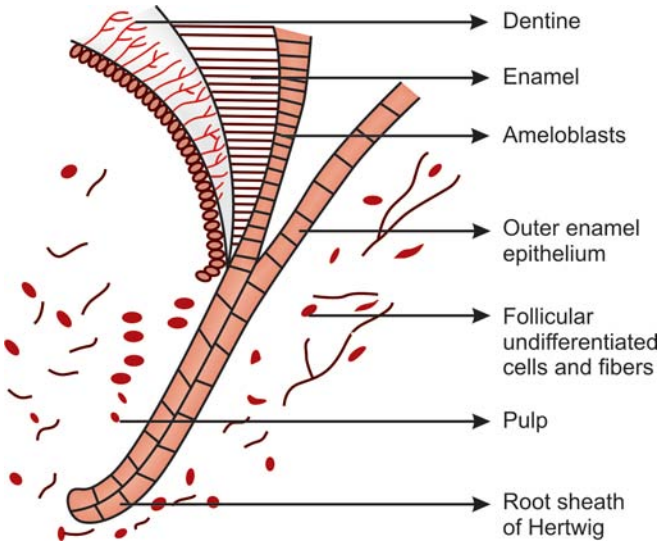

Fig. 1.2: Root sheath of Hertwig

lingual which are connected by the *col*. The significance of col is that, it is made up of nonkeratinized epithelium and hence represents the most frequent site for initiation of disease process.

DEVELOPMENT OF PERIODONTIUM

To understand the development of periodontal tissues one has to have a clear understanding of the root formation. Development of cementum and roots of the teeth starts once the formation of enamel is completed. The outer and inner epithelia together form the epithelial root sheath of Hertwig, which is responsible for determining the shape of the root.

Early Development of Cementum

The outer and inner epithelial layers become continuous (without stratum intermedium or stellate reticulum) in the area of the future cementoenamel junction and form a two-layered sheath, which grows into the underlying mesenchyme. The apical portion of the root sheath remains constant whereas the coronal portion, which is associated with dentin and cementum formation moves in the direction of the oral cavity. The root sheath bends horizontally at the level of future cementoenamel junction forming the epithelial diaphragm, following which the cervical opening becomes smaller (Figs 1.2 and 1.3).

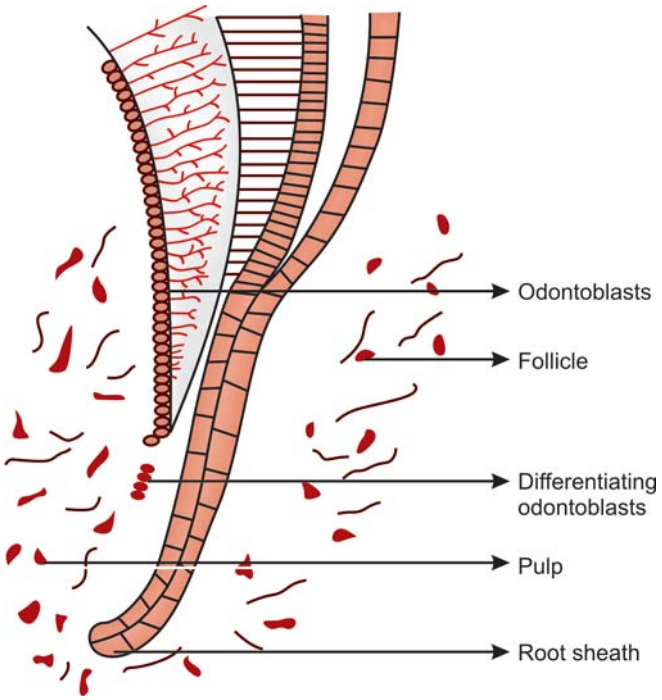

Fig. 1.3: Proliferation of root sheath and further dentine formation in an apical direction

Once the crown formation is complete the cells of the inner enamel epithelium loose their ability to form enamel and is called *reduced enamel epithelium*. They retain the ability to induce perimesenchymal cells to differentiate into odontoblasts and to proceed with the formation of predentin and dentin.

After the dentin formation is completed, certain changes occur in the root sheath. Recent studies have shown that, the epithelial cells of the root sheath produce a layer on the root dentin, which is 10 µm thick, has a hyaline appearance and contains fine granules and fibrils. This layer is called the *hyaline layer of Hopewell Smith* or *intermediate cementum*. The epithelial cells of the root sheath secrete enamel proteins such as *amelogenin or enameloids*. The root sheath at this stage becomes discontinuous and enables the surrounding follicular mesenchyme to come in contact with the amelogenin. These follicular cells then differentiate into cementoblasts and deposit the organic matrix of cementum on the root surface (Figs 1.4 and 1.5).

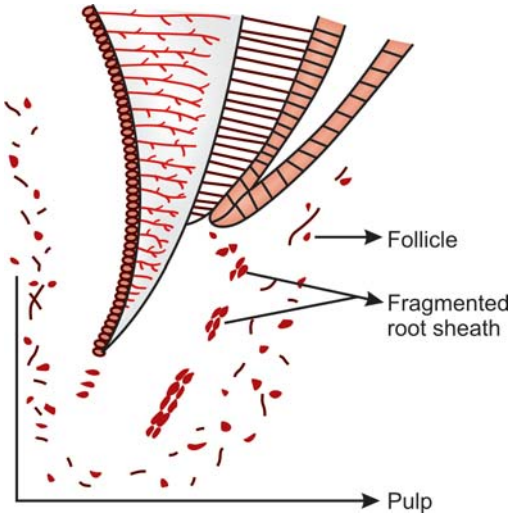

Fig. 1.4: Fragmentation of root sheath

Later Development of Cementum

Cementoblasts are cuboidal cells that are arranged on the outer surface of the hyaline layer. These cells are responsible for the deposition of the organic matrix of cementum, which consists of proteoglycan ground substance, intrinsic collagen fibers and is followed by subsequent mineralization of the organic matrix (Fig. 1.6).

Mineralization starts with the formation of a thin layer called *cementoid*. Mineral salts are derived from the tissue fluid containing calcium and phosphate ions and are deposited as hydroxyapatite crystals.

The disintegrated Hertwig's root sheath slowly moves away from the root surface and remain in the periodontal ligament as epithelial cell rests of Malassez. The periodontal ligament forms from the dental follicle soon after root formation begins. Before a tooth erupts, fibers from the follicle are incorporated in the cementum and they lie parallel to the root surface. Once the tooth erupts, the fibers are arranged in an oblique manner and are regarded as the precursor of the periodontal ligament fibers. As the cementum continues to increase in thickness, more fibers become incorporated into the cementum and eventually called as Sharpey's fibers, when periodontal ligament becomes established.

Alveolar bone forms around the periodontal ligament. With continuous bone deposition the periodontal ligament space gradually becomes narrower. The alveolar process

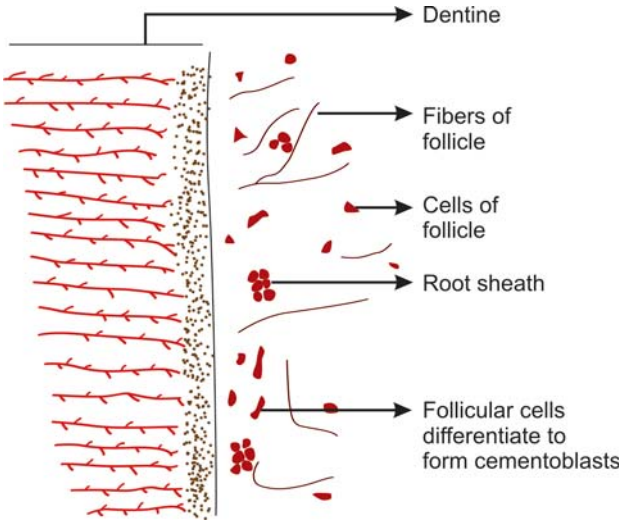

Fig. 1.5: Follicular cells and fibers contact dentine surface

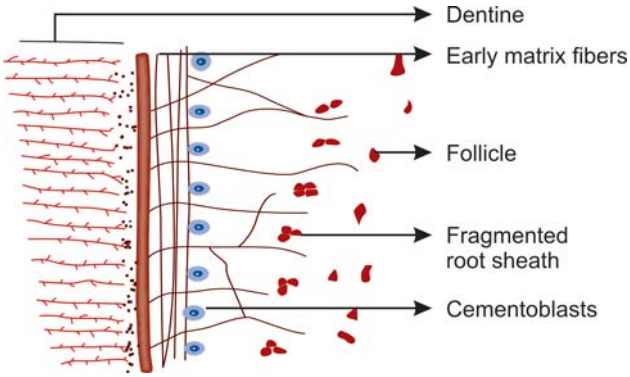

Fig. 1.6: Early deposition of matrix

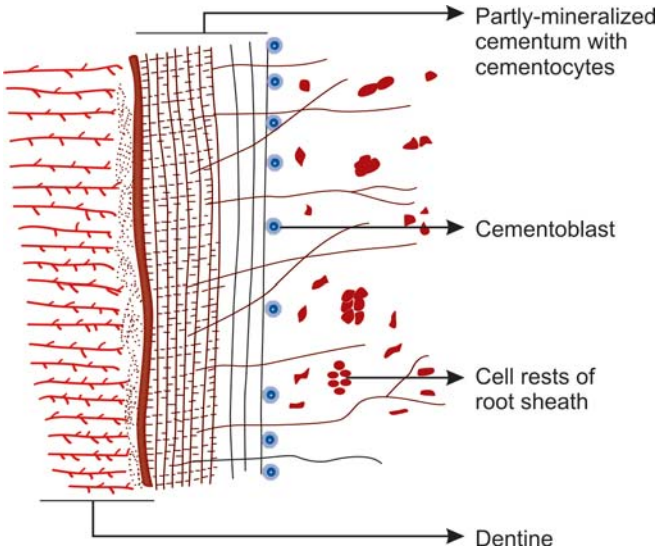

Fig. 1.7: Advanced formation of cementum

develops during the eruption of the teeth and cells responsible for bone formation are osteoblasts (Fig. 1.7).

Development of Junctional Epithelium (Fig. 1.8)

When the enamel formation is complete the ameloblasts become shorter, and they leave a thin membrane on the surface of the enamel called primary *enamel cuticle.* The inner enamel epithelium after laying down enamel reduces to a few layers of flat cuboidal cells, which is then called as *reduced enamel epithelium.* It covers the entire enamel surface extending till the cementoenamel junction. During eruption, the tip of the tooth approaches the oral mucosa leading to fusion of the reduced enamel epithelium with the oral epithelium. As the crown emerges into the oral cavity the former ameloblasts that are in contact with the enamel get transformed into *junctional epithelium.* Coronally, the junctional epithelium is continuous with the oral epithelium. As the tooth erupts, the reduced enamel epithelium grows shorter gradually. A shallow groove, the gingival sulcus may develop between the gingiva and the tooth surface.

Hence, the ameloblasts pass through two phases, in one they form enamel and in the other phase they help in formation of primary epithelial attachment or junctional epithelium. When the junctional epithelium forms from the ameloblasts it is called primary epithelial attachment. Junctional epithelium that forms after surgical therapy takes its origin from the basal cells of oral epithelium instead of ameloblasts. This is called secondary epithelial attachment.

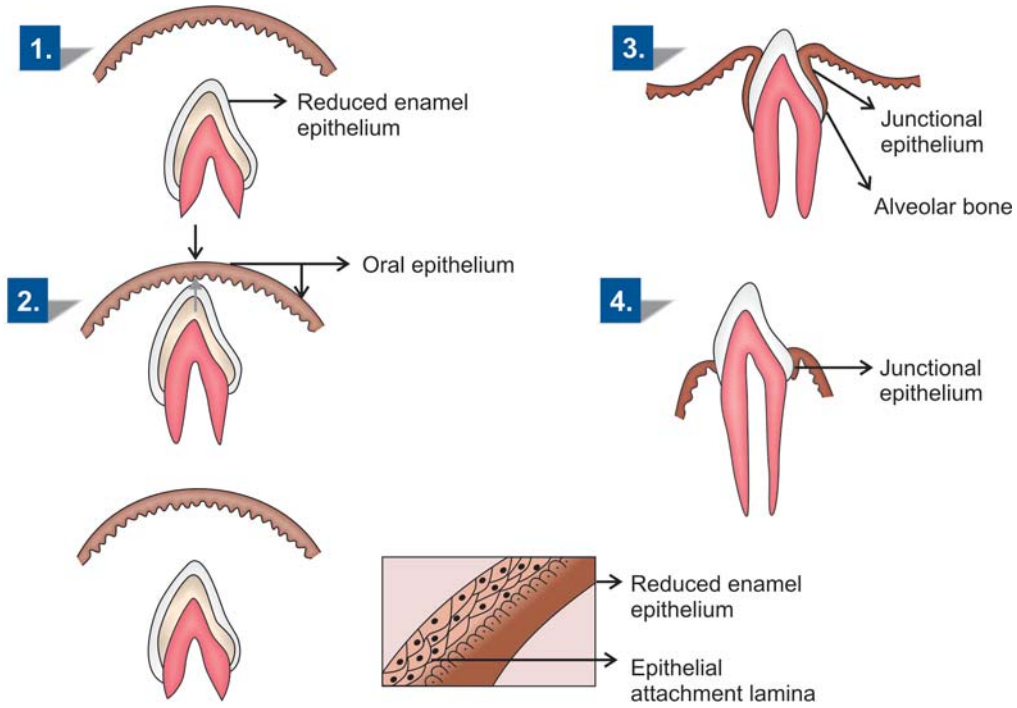

Fig. 1.8: Development of junctional epithelium

KEYPOINTS

1. The periodontium is composed of alveolar bone, root cementum, periodontal ligament (supporting tissue) and gingiva (investing tissue).
2. The gingiva is the only structure of the periodontium that is clinically visible and anatomically it can be divided into marginal, attached and interdental gingiva.
3. The junction between the marginal and attached gingiva is called free gingival groove, whereas the junction between the attached gingiva and alveolar mucosa is called as mucogingival junction.
4. Once the dentin formation is completed, the root sheath becomes discontinuous and allows the surrounding mesenchyme to come in contact with the products of the epithelial cells of the root sheath, i.e. amelogenin. These follicular cells then differentiate into cementoblasts, fibroblasts and osteoblasts by which cementum, periodontal ligament fibers and alveolar bone are deposited.
5. Fusion of the oral epithelium along with the reduced enamel epithelium gives rise to the junctional epithelium or epithelial attachment.

REVIEW QUESTIONS

1. Define periodontology and periodontics.
2. What are the parts of periodontium?
3. Describe the development of structures of periodontium.

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2 Chapter

Biology of Periodontal Tissues

INTRODUCTION

Periodontium is the functional unit of tissues supporting the tooth including gingiva, the periodontal ligament, the cementum and the alveolar process. The tooth and periodontium are together called as the *dentoperiodontal unit*. The main support of the tooth is provided by the periodontal ligament, which connects the cementum of the root to the alveolar bone or tooth socket, into which the root fits. The main function of the gingiva is to protect the surrounding tissues from the oral environment.

THE GINGIVA

Macroscopic Features

The gingiva is that part of the oral mucosa (masticatory mucosa) that covers the alveolar process of the jaws and surrounds the necks of the teeth. Anatomically, the gingiva

is divided into—marginal, attached and interdental gingiva (Fig. 2.1).

Marginal Gingiva or Free Gingiva or Unattached Gingiva

It is defined as the terminal edge or border of the gingiva surrounding the teeth in a collar-like fashion. In some cases, it is demarcated apically by a shallow linear depression called the *free gingival groove*. Though the marginal gingiva is well-adapted to the tooth surface, it is not attached to it (Fig. 2.2).

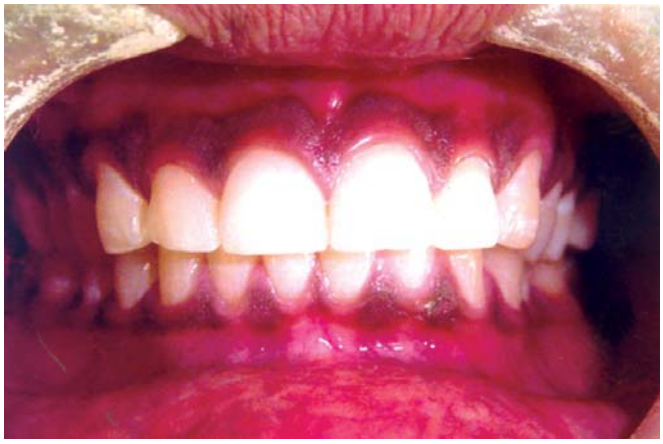


Fig. 2.1: Normal gingiva in health

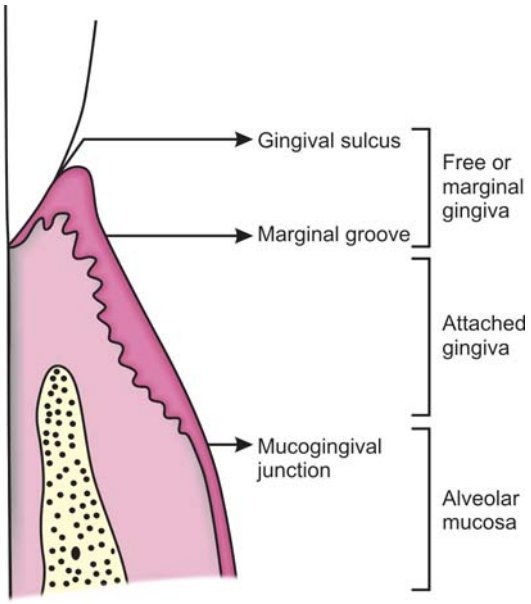


Fig. 2.2: Anatomic land marks of gingiva

Gingival Sulcus

It is defined as the space or shallow crevice between the tooth and the free gingiva, which extends apical to the junctional epithelium. It is V-shaped and barely permits the entrance of a periodontal probe. Under normal or ideal conditions it is about 0 mm (seen only in germ free animals). The so-called probing depth of a clinically-normal gingival sulcus in humans is 2 to 3 mm (Fig. 2.3).

Attached Gingiva

It is defined as that part of the gingiva that is firm, resilient and tightly-bound to the underlying periosteum of the alveolar bone. On the facial aspect it extends up to the loose and movable alveolar mucosa, from which it is demarcated by the mucogingival junction. The width of attached gingiva is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket.

It varies in different areas of the mouth, greater in the maxilla than mandible, least width in the mandibular first premolar area, greatest width in the maxillary incisor region. The width of attached gingiva increases with age and in supraerupted teeth.

Interdental Gingiva

Usually, occupies the gingival embrasure. There are three parts of interdental gingiva, facial papilla, lingual papilla and col, which is a valley-like depression that connects the

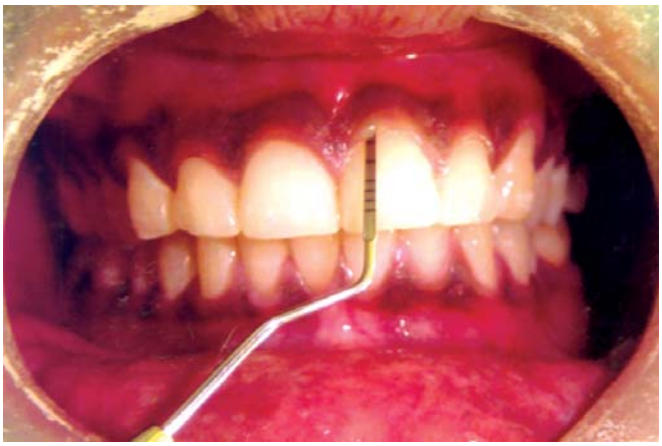


Fig. 2.3: Sulcus depth in healthy gingiva

facial and lingual papilla. The lateral borders and tips of the interdental papilla are formed by continuation of marginal gingiva and the intervening portion by the attached gingiva. In the presence of diastema the interdental papilla will be absent (Figs 2.4 and 2.5).

Microscopic Features

The gingiva consists of a central core of connective tissue covered by stratified squamous epithelium. Three types of epithelium exists in the gingiva.

1. The oral or outer epithelium (keratinized epithelium) (Fig. 2.6).
2. The sulcular epithelium
3. The junctional epithelium (nonkeratinized epithelium) (Fig. 2.7).

In the keratinized epithelium, the principal cell type is the keratinocyte, which can synthesize keratin. The process of keratinization involves a sequence of biochemical and morphological events that occur in a cell as it migrates from the basal layer towards the cell surface.

The nonkeratinized epithelium contains clear cells, which include melanocytes, Langerhans cells, Merkel cells and lymphocytes.

The oral epithelium has the following cell layers:

1. Basal layer (Stratum basale or Stratum germinativum).
2. Spinous layer (Stratum spinosum).
3. Granular layer (Stratum granulosum).
4. Keratinized cell layer (Stratum corneum).

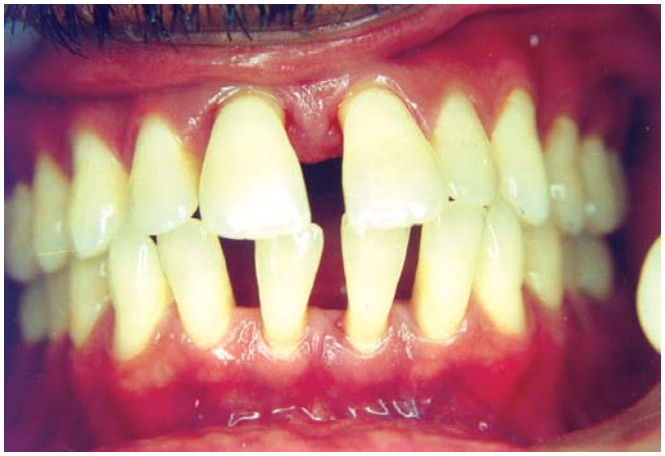


Fig. 2.4: Absence of interdental papilla in diastema cases

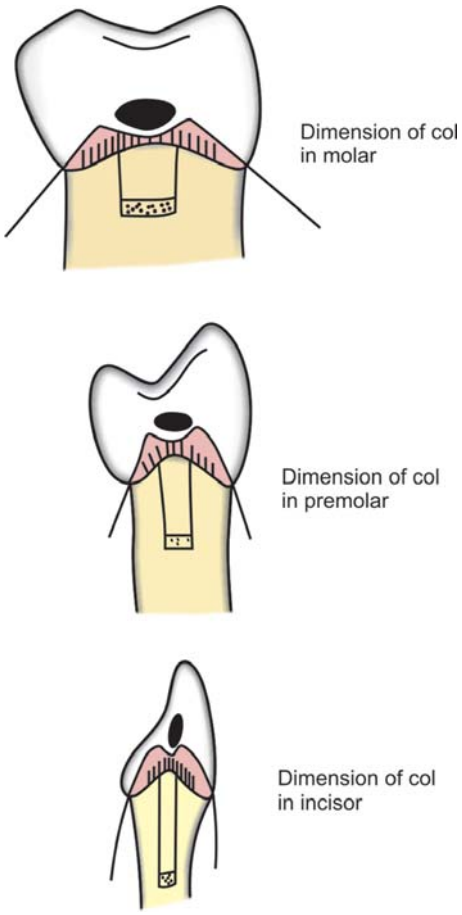


Fig. 2.5: 'COL' in various types of contacts

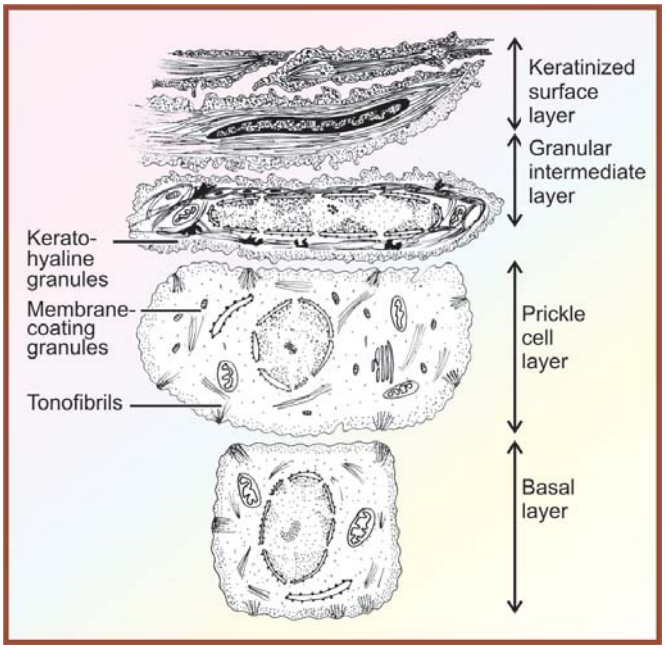


Fig. 2.6: Orthokeratinized epithelium

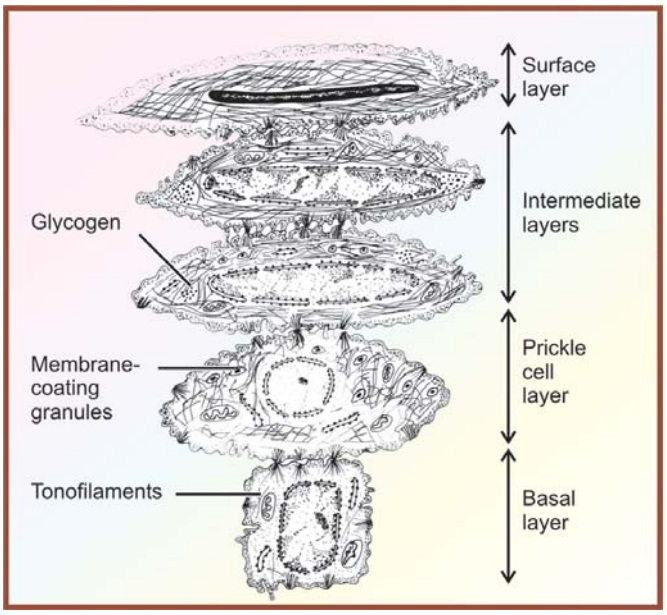


Fig. 2.7: Nonkeratinized epithelium

There are three distinct differences between the oral sulcular epithelium, oral epithelium and the junctional epithelium:

a. The size of the cells in the junctional epithelium is, relative to the tissue volume, larger than in the oral sulcular epithelium.
b. The intercellular space in the junctional epithelium is, comparatively wider than in the oral epithelium.
c. Granular layer, which is seen in the oral epithelium, is absent in sulcular and junctional epithelium.

Morphologic Characteristics of the Different Areas of Gingival Epithelium

Oral or outer epithelium: It covers the crest and outer surface of the marginal gingiva and the surface of the attached gingiva. It is keratinized or parakeratinized or combination of both. Keratinization varies in different areas in the following order: Palate (most keratinized), gingiva, ventral aspect of the tongue and cheek (least keratinized). The keratinized epithelium of the gingiva consists of four layers, namely stratum basale, stratum spinosum, stratum granulosum and stratum corneum.

The cells of the basal layer are either cylindrical or cuboidal and are in contact with the basement membrane. The basal cells possess the ability to divide, it is in the basal layer that the epithelium is renewed and therefore this is also called as stratum germinativum. When two daughter cells have been formed by cell division an adjacent "older" basal cell is pushed into the spinous cell layer and starts as a keratinocyte, to traverse the epithelium. It takes approximately one month for a keratinocyte to reach the outer epithelial surface where it becomes desquamated from the stratum corneum.

The basal cells are separated from the connective tissue by a basement membrane. In light microscopy this membrane appears as a zone approximately 1 µm wide and reacts positively to a PAS stain (periodic acid Schiff stain), which indicates the presence of carbohydrates (glycoproteins) in the basement membrane. In electron micrograph, immediately beneath the basal cell, an approximately 400 Å wide electrolucent zone can be seen which is called lamina lucida. Beneath the lamina lucida an electron dense zone – lamina densa is observed. From the lamina densa so-called anchoring fibrils project in fan-shaped fashion into the connective tissue. The epithelial cells facing the lamina lucida contain a number of electron dense, thicker zones called hemidesmosomes. The hemidesmosomes are involved in the attachment of the epithelium to the underlying basement membrane.

Stratum spinosum consists of large cells with short cytoplasmic processes resembling spines. Since they are arranged at regular intervals they give the cells a prickled appearance. The cells are attached to one another by numerous desmosomes (pairs of hemidesmosomes), which are located between the cytoplasmic processes of adjacent cells.

Composition of a desmosome: It consists of two adjoining hemidesmosomes separated by a zone containing electron dense granulated material (GM). In addition, outer and inner leaflets of the cell (OL, IL) and the attachment plaque (AP), represent granular and fibrillar material in the cytoplasm (GM) (Fig. 2.8).

Stratum granulosum: Electron dense keratohyalin bodies begin to occur, these granules are believed to be related to the synthesis of keratin. Here, there is conversion of cells

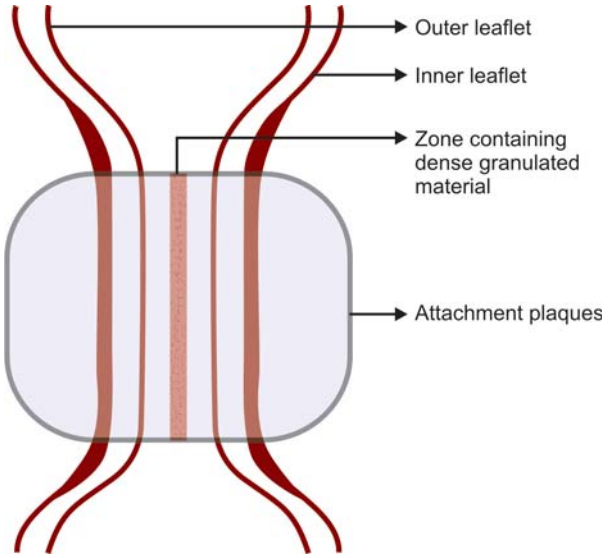


Fig. 2.8: Composition of a desmosome

to "acellular" structure bordered by a cell membrane indicating keratinization of the cytoplasm of the keratinocyte.

Stratum corneum: The cytoplasms of the cells in this layer are filled with keratin and the entire nucleus is lost. But in parakeratinized epithelia, the cells contain remnants of nuclei.

Keratinization is considered as a process of differentiation rather than degeneration. The keratinocytes while traversing from the basal layer to the epithelial surface undergoes certain changes.

a. From the basal layer to the granular layer both the number of tonofilaments in the cytoplasm and the number of desmosomes increase significantly.
b. In contrast, the number of organelles such as mitochondria, lamellae of rough endoplasmic reticulum and Golgi apparatus decrease.

Oral sulcular epithelium: The soft tissue wall of the gingival sulcus is lined coronally with sulcular epithelium, extending from the gingival margin to the junctional epithelium. It is made up of basal and prickle cell layer. The sulcular epithelium resembles the oral/gingival epithelium in all respects with the exception that it does not become fully keratinized. Although it contains keratinocytes they do not undergo keratinization. However, the surface cells are flattened and exhibit a tendency towards partial keratinization in response to physical stimulation. Normally, there are no regular rete pegs in sulcular epithelium but they form during the inflammation of the lateral wall.

Junctional epithelium: Denotes the tissue that joins to the tooth on one side and to the oral sulcular epithelium and connective tissue on the other. It forms the base of the sulcus. It has been examined in detail by several investigators, many hypothesis have been put forward to explain the mode of attachment of the epithelium to the tooth surface. Prior to the Gottlieb concept, it was believed generally, that the gingival soft tissues were closely opposed, but not organically united to the surface of the enamel. This concept was based on the clinical finding that the gingiva could be easily deflected from the tooth surface during instrumentation. However, experimental and clinical observations have led Gottlieb to the concept that the soft tissues of the gingiva are organically united to the enamel surface. He termed it as "epithelial attachment". Although it was accepted generally, the concept did not explain how exactly junctional epithelium attaches to the root surface (Fig. 2.9).

Waerhaug in 1952, based on his observations presented the concept of the "epithelial cuff, he concluded that the gingival tissues are closely apposed, but not organically united to the tooth surface. In 1962, Stern showed that the attachment to the tooth surface is through hemidesmosomes. This was supported by extensive studies conducted by Schroeder and Listgarten, they revealed that there is a structural continuity between the tooth surface and epithelium. These studies by Schroeder and Listgarten states that the epithelium-tooth interface was observed to be similar to the epithelium-connective tissue interface and that is by hemidesmosome and basal lamina. A basal lamina is always interposed between epithelial cells and crown or root surface, and the epithelial cells are united to the basal lamina by hemidesmosomes.

The junctional epithelium is attached to the tooth surface by internal basal lamina and to the gingival connective tissue by an external basal lamina. The internal basal lamina

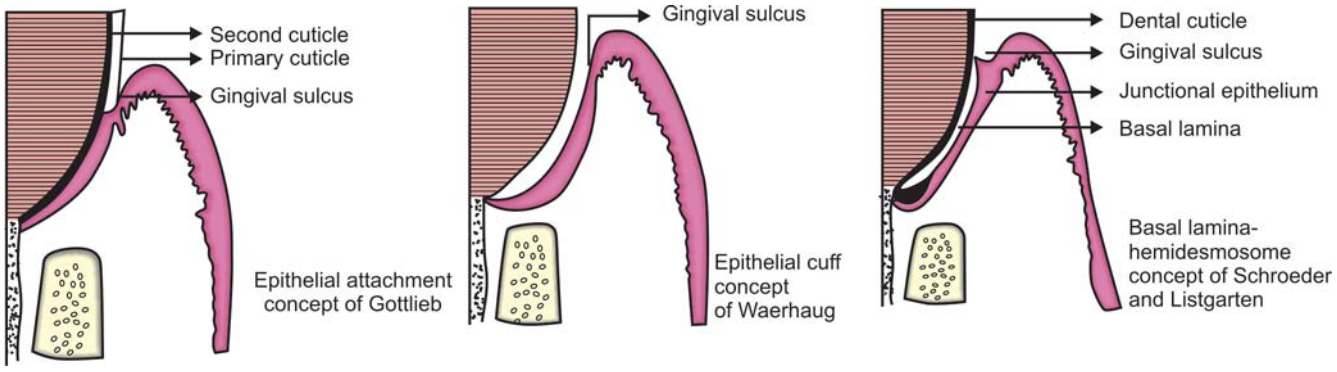


Fig. 2.9: Concepts of epithelial attachment

consists of a lamina densa (adjacent to the enamel) and a lamina lucida to which hemidesmosomes are attached. Various studies have also shown that junctional epithelial cells are involved in the production of laminin and play a key role in the adhesion mechanism. The attachment of the junctional epithelium to the tooth surface is reinforced by the gingival fibers. Hence the junctional epithelium and gingival fibers are considered as a functional unit, referred to as the dentogingival unit.

General structural features of junctional epithelium: It consists of a collar like band of stratified squamous non keratinizing epithelium. Thickness varies from three or four layers in early life and increases with age up to 15 to 20 layers at the base of the gingival sulcus, and only 1 or 2 cells at the most apical portion. The length of the junctional epithelium ranges from 0.25 to 1.35 mm. The cells are arranged into basal and suprabasal layers and they do not have granular layer or cornified layers. They exhibit unusual cytologic features and differ significantly from other oral epithelia. Three zones in junctional epithelium have been described, apical, coronal and middle. Apical is for germination, middle is for adhesion and coronal is permeable.

The basal cells are cuboidal or in some cases, flattened. They contain slightly more rough endoplasmic reticulum and lysosomal content. The content of mitochondria of the cells as they migrate toward and along the tooth surface decreases. Cells of the suprabasal layer especially those adjacent to the tooth surface exhibit complex microvillus formation and interdigitation. Junctional epithelial cells, especially those near the base of the gingival sulcus, appear to have phagocytic capacity.

The structural features of junctional epithelium indicate that it is highly permeable. Leukocytes and lymphocytes within junctional epithelium are seen even in clinically healthy gingiva. The densities of desmosomes that interconnect the cells are considerably less than that of oral epithelia and represents wider intercellular junctions.

Epithelium-Connective Tissue Interface

Histological sections have demonstrated that, the rete pegs of epithelial cells project deeply into the connective tissue leading to the formation of a series of interconnecting epithelial ridges. Basement membrane seems to form a continuous sheet that connects the epithelium and connective tissue. Electron microscope reveals a faintly fibrillar structure, called as the basal lamina, which is a part of the basement membrane. This structure has lamina lucida adjacent to the basal epithelial cells and lamina densa towards connective tissue. Basal lamina is produced by adjacent epithelial cells and is made up of collagenous proteins and proteoglycans binded together into a totally-insoluble complex. It also contains laminin and fibronectin. Fibrils measuring 20 to 40 nm in diameter seem to extend from the basal cells through the basal lamina and into the lamina propria of the connective tissue. These structures called as anchoring fibrils are supposed to bind the basal cells, basal lamina and connective tissue together (Fig. 2.10).

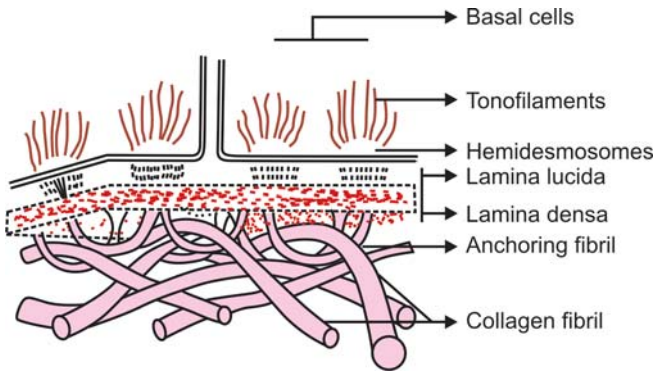


Fig. 2.10: Structure of the junction between epithelium and connective tissue

Supra-alveolar Connective Tissue

The connective tissue supporting the oral epithelium is termed as the lamina propria and for descriptive purpose it can be divided into two layers:

a. *The superficial papillary layer*—associated with the epithelial ridges.
b. *Deeper reticular layer*—that lies between the papillary layer and the underlying structures.

The term reticular here means net-like and refers to the arrangement of collagen fibers. In the papillary layer, the collagen fibers are thin and loosely-arranged and many capillary loops are present. The reticular layer is dominated by collagen arranged in thick bundles.

The lamina propria consists of cells, fibers, blood vessels embedded in amorphous ground substances.

I. *Cells*: Different types of cells present are:
   a. Fibroblast
   b. Mast cells
   c. Macrophages
   d. Inflammatory cells.
II. *Fibers*: The connective tissue fibers are produced by fibroblasts and can be divided into:
   a. Collagen fibers
   b. Reticulin fibers
   c. Oxytalan fibers
   d. Elastin fibers.

Collagen type I form the bulk of the lamina propria and provide the tensile strength to the gingival tissues. Type II collagen is seen in the basement membrane.

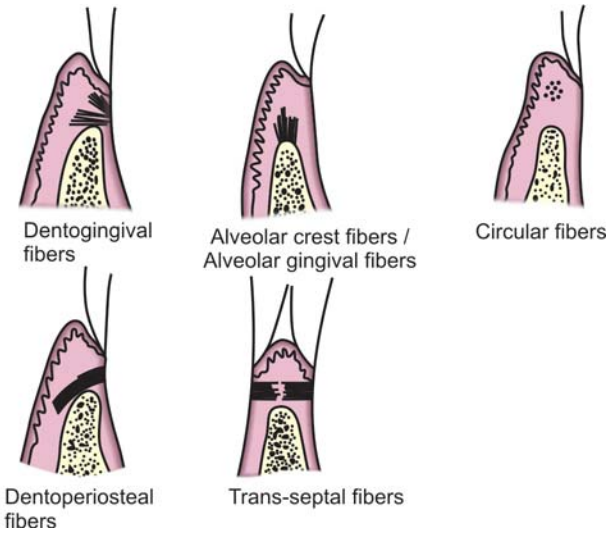


Fig. 2.11: The principal group of fibers

The functions of gingival fibers are the following:

a. It braces the marginal gingiva firmly against the tooth.
b. It helps to withstand the forces exerted by mastication.
c. It unites the free gingiva to the root cementum and the adjacent attached gingiva.

The arrangement of gingival fibers is described as *principal group* of five bundles and *secondary group* of minor fibers consisting of six sets (Fig. 2.11).

The principal group fibers are:

1. *Dentogingival fibers*: They project from the cementum in a fan-like conformation towards the crest and outer surface of the marginal gingiva. They provide support to the gingiva by attaching it to the tooth.
2. *Alveolar gingival fibers*: They extend from the periosteum of the alveolar crest coronally into the lamina propria. Their function is to attach the gingiva to the alveolar bone.
3. *Dentoperiosteal fibers*: They arise from the cementum near the cementoenamel junction and insert into the periosteum of the alveolar bone and protect the periodontal ligament.
4. *Circular fibers*: They surround the tooth in a cuff or ring like fashion and course through the connective tissue of the marginal and attached gingiva.
5. *Trans-septal fibers*: They are located interproximally, they extend from cementum of one tooth to the

cementum of the neighboring tooth. Their function is to protect the interproximal bone and maintain tooth-to-tooth contact.

Fibers of the secondary group (Fig. 2.12) are:

1. *Periosteogingival fibers*: They extend from the periosteum of the alveolar bone to the attached gingiva. They help to attach the gingiva to the alveolar bone.
2. *Interpapillary fibers*: They are seen in the interdental gingiva extending in a faciolingual direction and support the gingival papilla.
3. *Transgingival fibers*: These are seen in and around the teeth with in the attached gingiva. They maintain the alignment of teeth in the arch.
4. *Intercircular fibers*: They extend from the cementum on distal surface of a tooth splaying buccally and lingually around the next tooth and are inserted on the mesial surface.
5. *Intergingival fibers*: They are seen within the attached gingiva adjacent to the basement membrane extending mesiodistally. They provide support and contour for the attached gingiva.
6. *Semicircular fibers*: They extend from the mesial surface of a tooth to the distal surface of same tooth in a half circle.
7. *Oxytalan fibers*: They are present in all connective tissue structures of the periodontium. The function of these fibers is yet unknown.
8. *Elastin fibers*: Elastin fibers are only present in connective tissue of the gingiva and periodontal ligament. They are also seen in the connective tissue of alveolar mucosa in large numbers.

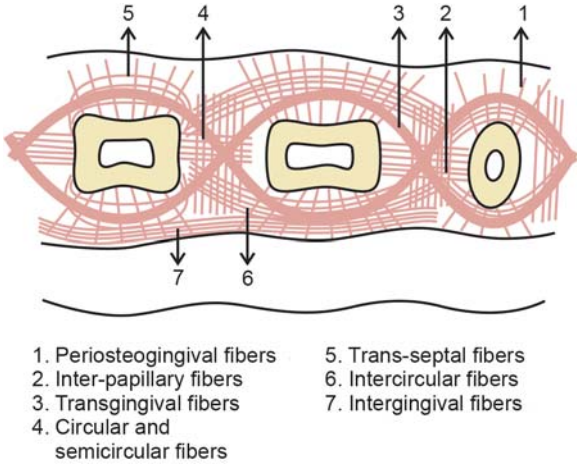


Fig. 2.12: Secondary group of fibers

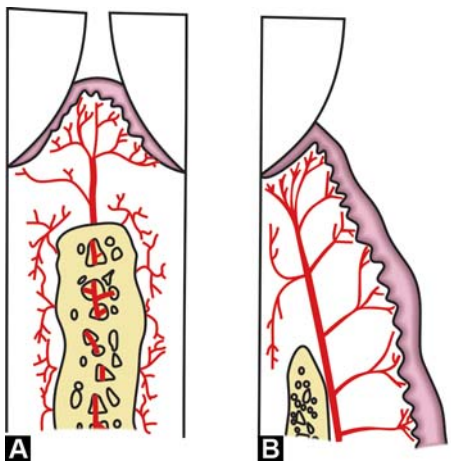
Extracellular matrix/ground substance: It is produced by fibroblasts, followed by mast cells and other components derived from the blood. The matrix is the medium in which the connective tissue cells are embedded and is essential for the maintenance of the normal function of the connective tissue. Thus, the transportation of water, electrolytes, nutrients, metabolites, etc. to and from the individual connective tissue cells occurs with in the matrix.

The main constituents of connective tissue matrix are protein polysaccharide matrix. These complexes are normally differentiated into proteoglycans and glycoproteins. The proteoglycans contains glycosaminoglycans, e.g. chondroitin sulfate, heparin sulfate, hyaluronic acid, etc. The proteoglycans act as a molecular filter and in addition, play an important role in the regulation of cell migration in the tissue. Due to their structure and hydration, the macromolecules exert resistance and hydration, towards deformation. Hence, when gingiva is suppressed, the macromolecule become deformed, when the pressure is eliminated the macromolecules regain their original form. *Thus, the macromolecules are of importance for the resilience of the gingiva.*

Blood Supply, Lymphatics and Nerves

Three major sources of blood supply to the gingiva (Fig. 2.13) has been described:

1. Supraperiosteal arterioles: Overlying the alveolar bone along the facial and lingual surfaces, sends branches to the surrounding tissue.



Figs 2.13 A and B: Blood supply to gingiva. Arterioles penetrating the interdental bone (A), supraperiosteal arterioles (B)

2. Vessels of the periodontal ligament: They extend into the gingiva and anastamose with the capillaries in the sulcus area.
3. Arterioles emerging from the crest of the interdental septa.

Lymphatic drainage of the gingiva brings in the lymphatics of the connective tissue papillae. It progresses to the periosteum of the alveolar process and then to regional lymph nodes (mainly submaxillary group).

Nerve supply to gingiva is derived from fibers arising from nerves in the periodontal ligament and from the labial, buccal and palatal nerves.

TOOTH-SUPPORTING STRUCTURES

PERIODONTAL LIGAMENT

Definition

Tooth-supporting structures include, the periodontal ligament, cementum and alveolar bone. The periodontal ligament is a connective tissue structure that surrounds the root and connects it with the bone. In the past, periodontal ligament has been described by many terms. Among these are desmodont, gomphosis, pericementum, alveolodental ligament and periodontal membrane. Since it is a soft connective tissue providing continuity between two mineralized connective tissues, the term periodontal ligament appears to be more appropriate.

In the coronal direction, the periodontal ligament is continuous with the lamina propria of the gingiva and communicates with the marrow spaces of the alveolar bone through Volkmann's canals.

Structure

The periodontal ligament space has the shape of an hourglass and is narrowest at the mid-root level. The width of periodontal ligament is approximately 0.25 mm ± 50 percent.

Cellular Composition

Cells of periodontal ligament are categorized as:

1. *Synthetic cells*
   a. Osteoblasts
   b. Fibroblasts
   c. Cementoblasts
2. *Resorptive cells*
   a. Osteoclasts
   b. Cementoclasts
   c. Fibroblasts
3. *Progenitor cells*
4. *Other epithelial cells*
   Epithelial cell rests of Malassez
5. *Connective tissue cells*
   Mast cells and macrophages.

Characteristics of a Synthetic Cell

1. Should be actively-synthesizing ribosomes
2. Increase in the complement of rough endoplasmic reticulum and Golgi apparatus
3. Large open faced or vesicular nucleus containing prominent nucleoli.

Osteoblasts: Covers the periodontal surface of the alveolar bone. Alveolar bone contains endosteum and a periosteum. A periosteum contains at least two distinct layers, cambium layer or cellular layer and a fibrous layer. A cellular layer is present on the periodontal surface of the alveolar bone.

Fibroblasts: It is the most prominent connective tissue cell (65% of total cell population). Periodontal ligament fibroblasts are phenotypically different from gingival fibroblasts. They consist of subtypes with distinct phenotypes and found to synthesize higher quantities of chondroitin sulfates and lesser quantities of heparan sulfate and hyaluronan sulfate. The main function of the fibroblasts is the production of various types of fibers and it is also instrumental in the synthesis of the connective tissue matrix. The fibroblast is a stellate or spindle-shaped cell which produce

- Collagen fibers
- Reticulin fibers
- Oxytalan fibers
- Elastin fibers

Various stages in the production of collagen fibers are as follows:

The first molecule released by fibroblasts is tropocollagen which contains three polypeptide chains interwined

to form a helix. Tropocollagen molecules are aggregated longitudinally to form protofibrils, which are subsequently laterally-arranged in parallel to form collagen fibrils. Collagen fibers are bundles of collagen fibrils.

Cementoblasts are seen lining the cementum.

Resorptive Cells

Osteoclasts: These are the cells that resorb the bone and tend to be large and multinucleated. The precursor cells of the osteoclasts are circulating monocytes. The characteristic features of osteoclasts are the plasma membrane of the cell lying adjacent to the bone that has been actively undergoing resorption is raised in characteristic folds and is termed as ruffled or striated border.

Fibroblasts: It must be made clear that the fibroblasts may be capable of both synthesis and resorption. The fibroblasts responsible for resorption contain fragments of collagen that appear to be undergoing digestion. The presence of these cells indicates resorption of fibers occurring during either disease or physiological turnover or remodeling of periodontal ligament.

Cementoclasts: The main observation in this is that cementum is not remodeled in the fashion of alveolar bone and periodontal ligament, but that it undergoes continual deposition during life. However, resorption of cementum occurs in certain circumstances and in these instance cementoclasts are located in Howship's lacunae.

Progenitor cells: Little is known about these cells. It is believed that, generally, as and when the need arises, the daughter cell after the division differentiates into the functional type of connective tissue cells.

Epithelial cell rests of Malassez: They are found close to cementum. These cells are first described by Malassez in 1884 and are remnants of the epithelium of Hertwig's epithelial root sheath. The epithelial cell rests persists as a network, strands, island or tubule like structures near and parallel to the surface of the root.

Electron microscope observation shows that the epithelial cell rests exhibits tonofilaments and they are attached to one another by desmosomes. The physiologic role of these cells is not known. When certain pathologic conditions are present, cells of the epithelial rests can undergo rapid proliferation and can produce a variety of cysts and tumors of the jaws.

Mast cells: These are relatively small, round or oval cell having a diameter of almost 12 to 15 μm. The cells contain numerous cytoplasmic granules with small or round nucleus. The granules have been shown to contain heparin and histamine. The physiologic role of heparin in mast cells does not appear to be clear. Whereas mast cell histamine plays a role in the inflammatory reaction and they have been shown to degranulate in response to antigen-antibody formation on their surface.

Macrophages may also be present in the ligament. They are capable of phagocytosis.

Extracellular Components

1. *Fibers:*
   - Collagen
   - Oxytalan
2. *Ground substance*:
   - Proteoglycans
   - Glycoproteins

Periodontal fibers: The most important elements of the periodontal ligament are the principal fibers. These fibers are collagenous in nature and are arranged in bundles, they follow a wavy course. The terminal portion of these principal fibers that insert into the cementum and bone are termed Sharpey's fibers. Collagen is a specific, high molecular weight protein to which a small number of amino acids are attached, the most important of which are glycine, proline and hydoxyproline. Collagen is synthesized by fibroblasts, chondroblasts, osteoblasts, odontoblasts and other cells. Several types of collagen have been demonstrated. The principal fibers are composed primarily of Type I collagen, whereas reticular fibers are made up of Type III collagen. Type IV collagen is seen in the basal lamina.

The principal fibers of periodontal ligament (Fig. 2.14) are arranged in six groups that develop sequentially in the developing root. They are:

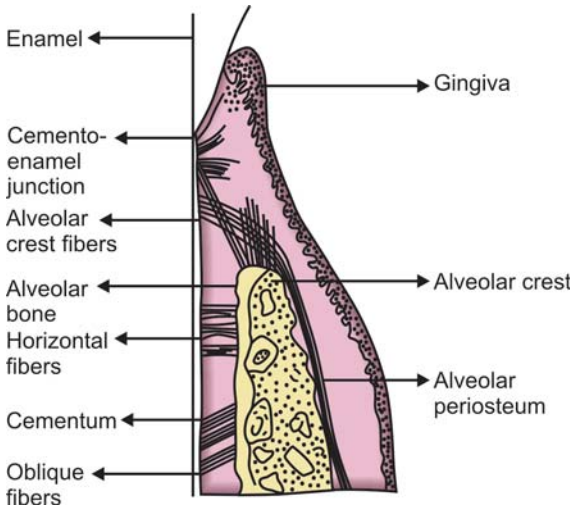


Fig. 2.14: Principal fiber groups in periodontal ligament

- Trans-septal group
- Alveolar crest, horizontal, oblique, apical and inter-radicular fibers.

Trans-septal group: They may be considered to belong to the gingiva because they do not have osseous attachment.

Alveolocrestal group: They extend obliquely from the cementum just beneath the junctional epithelium to the alveolar crest. Their function is to retain tooth in socket, resist lateral tooth movement and protect deeper periodontal ligament structure.

Horizontal group: Extend from cementum to the alveolar bone at right angles to the long axis of the tooth.

Oblique group: They are the largest group in the periodontal ligament; extend coronally in an oblique direction from the cementum to the bone. They resist axially directed forces.

Apical group: They originate from cementum of root apex, splaying apically and laterally into the bone of the alveolar fundus. Their main function is it prevents tooth tipping; resists luxation, protects blood, lymph and nerve supply to the tooth.

Inter-radicular fibers: Extends from cementum of bifurcation areas, splaying from apical into furcal bone. It resists luxation and also tipping and torquing.

Secondary fibers of periodontal ligament: In addition to the principal fiber groups, periodontal ligament contains other well-formed fiber bundles, that inter-digitate at right angles or splay around and between the regular fiber bundles. These fibers are associated with blood vessels and nerves of the periodontal ligament. Although periodontal ligament does not contain mature elastin, two immature forms have been described, the so-called oxytalan fibers and elaunin. It was suggested that, they provide elastic properties to periodontal ligament. There are also *reticulate fibers*, which are fine, immature collagen fibers with a lattice like arrangement.

In addition to the above fiber types, small collagen fibers arranged in all directions, forming a plexus have also been reported. They are closely-associated with principal fibers and are termed as the indifferent fiber plexus.

Ground substance: The space between cells, fibers, blood vessels and nerves in the periodontal space is occupied by ground substance. The ground substance is made up of two major groups of substances. Glycosaminoglycans such as hyaluronic acid, proteoglycans and glycoproteins such as fibronectin and laminin. It also has high water content (70%).

Development of Principal Fibers of Periodontal Ligament (Fig. 2.15)

The principal fibers develop in conjunction with the eruption of the tooth. The fibroblasts surrounding the developing root produce collagen fibers. These fibers are seen in the periodontal space without a specific orientation. As and when the tooth erupts, the orientation of the fibers alters.

1. First small, fine brush-like fibrils are seen arising from the root cementum and projecting into the periodontal ligament space.
2. Similar fibers are seen on the surface of the bone but only in thin, small numbers.
3. Later on, the number and thickness of fibers originating from the bone increase and elongate. They radiate towards the loose connective tissue in the midportion of the periodontal ligament.
4. The fibers originating from the cementum also increase in length and thickness and fuses with the fibers originating from the alveolar bone in the periodontal ligament space.

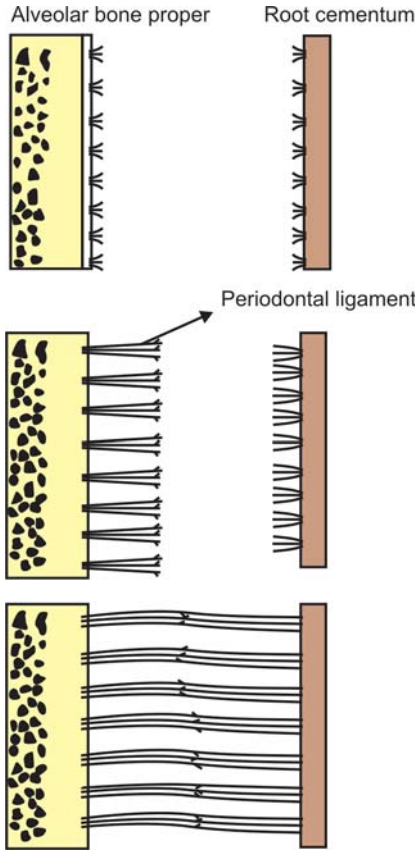


Fig. 2.15: Development of periodontal ligament fibers (principal)

5. They mature progressively towards the root apex as the eruption progresses. When the tooth, following eruption, reaches contact in occlusion and starts to function, the principal fibers become organized in bundles and run continuously from bone to cementum.

For long, it was believed that this middle portion where the splicing of fibers from cementum and bone takes place, it forms the intermediate plexus. These plexus were thought to play a significant role in orientation and adjustment of fibers during eruption and functional movement of teeth. But recent investigations have revealed that, in humans these plexus disappears once the fusion of cemental and osseous fibers are completed.

Structures Present in the Connective Tissue

1. Blood vessels
2. Lymphatics
3. Nerve innervation
4. Cementicles

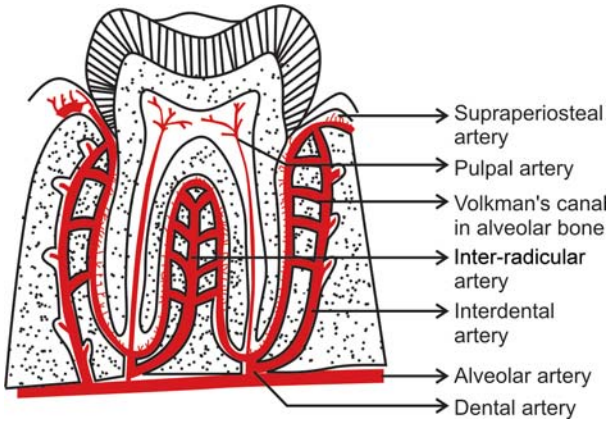


Fig. 2.16: Periodontal blood supply

Blood vessels: Periodontal ligament is supplied by branches derived from three sources dental, inter-radicular and interdental arteries (Fig. 2.16).

1. Dental artery before it enters apical foramen gives off branches to the periodontal ligament which also supplies the pulp.
2. The inter-radicular artery gives off branches in the alveolar process that supplies the periodontal ligament through the cribriform plate.
3. Interdental artery emerges from the crest of the alveolar bone and supplies the coronal part of the periodontal ligament.

Lymphatic vessels are seen to follow the path of blood vessels in the periodontal ligament.

Nerve supply: In an unerupted tooth, the developing periodontal ligament is supplied by fine, unmyelinated nerve fibers. Whether this persists after the tooth is erupted is not known. Periodontal ligament is mainly supplied by the dental branches of the alveolar nerve through the apical perforations of the tooth socket or from the cribriform plate. Many studies have proved that periodontal ligament is richly supplied by mechanoreceptors whose cell bodies are located in the trigeminal ganglion. These receptors provide sense of touch, pressure, pain, and proprioception during mastication.

Cementicles are calcified masses adherent to or detached from the root surface. They may be developed from calcified epithelial rests, calcified Sharpey's fibers, and calcified thrombosed vessels within the periodontal ligament.

Functions of Periodontal Ligament

The following functions of periodontal ligament have been explained:

1. Physical
2. Formative and remodeling
3. Nutritional and sensory function.

Physical Function

The physical functions of the periodontal ligament are:

a. Provides soft tissue "casing" in order to protect the vessels and nerves from injury due to mechanical forces.
b. Transmit the occlusal forces to the bone. Depending on the type of force applied, axial force when applied causes stretching of oblique fibers of periodontal ligament. Transmission of this tensional force to the alveolar bone encourages bone formation rather than bone resorption. But when horizontal or tipping force is applied the tooth rotates around the axis, at first the tooth movement is within the confines of the periodontal ligament. When a greater force is applied, displacement of facial and lingual plates may occur. The axis of rotation, in single-rooted teeth is located in the area between the apical and middle third of the root. In multirooted teeth, the axis of rotation is located at the furcation area.
c. Attaches the teeth to the bone
d. Maintains the gingival tissues in their proper relationship to the teeth.
e. Shock absorption resists the impact of occlusal forces.

Two theories have been explained for the mechanism of tooth support.

a. Tensional theory
b. Viscoelastic theory

Tensional theory: According to it the principal fibers of periodontal ligament plays a major role in supporting the tooth and transmitting forces to the bone. When forces are applied to the tooth, principal fibers unfold and straighten and then transmit the forces to the alveolar bone, causing elastic deformation of the socket.

Viscoelastic theory is based on the fact that, the fluid movement largely controls the displacement of the tooth, with fibers playing a secondary role. When the forces are transmitted to the tooth, the extracellular fluid is pushed from the periodontal ligament into the marrow spaces through the cribriform plate. After the depletion of the tissue fluids, the bundle fibers absorb the shock and tighten. This leads to blood vessel stenosis → arterial back pressure → ballooning of the vessels → Tissue replenishes with fluids.

Formative and Remodeling Function

Cells of the periodontal ligament have the capacity to control the synthesis and resorption of the cementum, ligament and alveolar bone. Periodontal ligament undergoes constant remodeling; old cells and fibers are broken down and replaced by new ones.

Nutritional and Sensory Function

Since periodontal ligament has a rich vascular supply it provides nutrition to the cementum, bone and gingiva. Periodontal ligament is supplied by nerve fibers that can transmit sensation of touch, pressure and pain to higher centers. The nerve bundle follows the course of blood vessel and enters the periodontal ligament from periapical area through channels from the alveolar bone. These bundles divide into single myelinated fibers, which later on lose their myelin sheath and end in one of the four types of neural termination.

- Free endings, carry pain sensations
- Ruffini-like mechanoreceptors located in the apical area
- Meissners corpuscles are also mechanoreceptors located primarily in the mid-root region.
- Spindle-like pressure and vibration endings, located mainly in the apex.

Pain sensation is transmitted by small diameter nerves, temperature by intermediate type; pressure by large myelinated fibers.

Clinical Considerations

The primary role of periodontal ligament is to support the tooth in the bony socket. Its thickness varies in individuals and in different teeth in the same person. Periodontal ligament is shaped like an hourglass and is narrowest in the middle region of the root and thus seems to be the fulcrum

of physiological movement. In connection with the physiological mesial migration of the teeth, the periodontal ligament is thinner on the mesial root surface than on the distal surface.

Due to acute trauma to the periodontal ligament or in accidental blows, many pathological changes will be produced, such as fracture or resorption of cementum and alveolar bone. Hence there will be loss of alveolar bone and widening of periodontal ligament, which results in the tooth becoming loose. When the trauma is removed repair usually will take place.

Orthodontic Tooth Movement

Depends on the resorption and formation of both bone and periodontal ligament. These activities can be stimulated by properly regulated pressure and tension. If the movement of the tooth is within physiological limits, the compression of the periodontal ligament on pressure side results in bone resorption, where as on the tension side bone apposition is seen. Application of the large forces results in the necrosis of periodontal ligament and alveolar bone.

If gingivitis is not controlled or treated, it will invariably extend to the periodontal ligament and bone and produces the destruction of the same. Once they are destroyed, it is difficult for them to regenerate and therefore the diseases of periodontal ligament are often irreversible.

ALVEOLAR BONE

Definition

It is that portion of the maxilla and mandible that forms and supports the tooth socket (alveoli). It is formed when the tooth erupts, in order to provide osseous attachment to the forming periodontal ligament and gradually disappears after the tooth is lost.

Parts of Alveolar Bone (Fig. 2.17)

Alveolar bone consists of the following:

1. Inner and outer cortical plate.
2. The bone lining the socket.
3. An interior portion of cancellous bone.

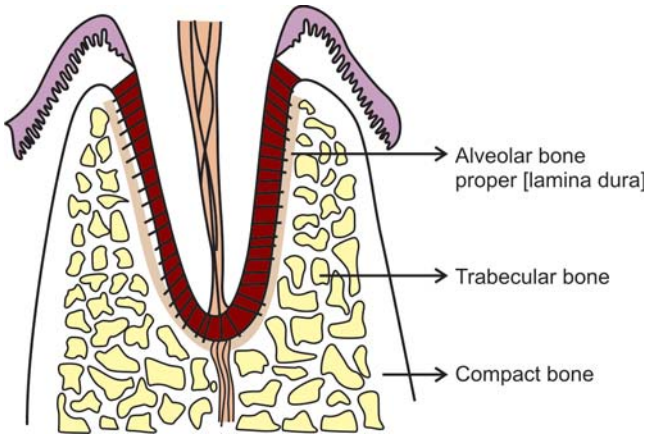


Fig. 2.17: Alveolar bone structure

The cortical plates consist of compact bone, in which the lamellae are often arranged circumferentially around blood vessels forming Haversian systems, which are the internal mechanisms that bring a vascular supply to bones that are too thick to be supplied only by surface vessels. The cortical plates and the bone lining the socket meet at the alveolar crest, usually 2 mm below the cementoenamel junction.

The bone lining the socket is also compact bone and can be known as any of the following:

1. *Bundle bone*, since bundles of Sharpey's fibers from the periodontal ligament are embedded in it.
2. *The cribriform plate*, because it is perforated by numerous vascular channels.
3. *Alveolar bone proper*, as it provides direct bony support for the teeth.
4. *Lamina dura*, which is radiographically seen as a dense plate.

Cancellous bone: It consists of narrow irregular bony trabeculae, which, by branching and uniting forms a network of spaces between the trabeculae. There is a considerable variation in the proportions of compact to cancellous bone in different regions of the jaws and in different tooth surfaces. For example: In relation to lower anterior teeth, i.e. lower incisors, there is a thin bone that consists of an outer cortical plate and the bone lining the socket with no intervening cancellous bone. In contrast, the buccal and interdental bone of molars is relatively thick and cancellous bone may predominate.

As the diameter of the tooth root gradually decreases in an apical direction, there is a corresponding increase in the thickness of alveolar bone with increase in cancellous bone being present. These variations are important to note because they influence the pattern and progression of bone loss in destructive forms of periodontal diseases.

Roentgenogram permits the classification of trabecular patterns of the alveolar process into two main types:

- *In type I*, the interdental and inter-radicular trabeculae are regular, horizontal and are arranged in a ladder-like pattern. (Common in mandible)
- *In type II*, irregularly arranged numerous interdental and inter-radicular trabeculae are seen most commonly in maxilla.

Composition of Alveolar Bone

It has two basic constituents:

a. The cells consist of osteoblasts, osteoclasts, and osteocytes.
b. Extracellular matrix consists of 65 percent inorganic and 35 percent organic matter.

The inorganic component is composed of minerals such as calcium, phosphate along with hydroxyl, carbonate, citrate and trace amounts of other ions, such as sodium, magnesium and fluorine. The minerals are in the form of hydroxyapatite crystals.

The organic matrix consists of 90 percent of type I collagen, with small amounts of non-collagenous proteins such as osteocalcin, osteonectin, bone morphogenetic proteins, proteoglycans, and glycoproteins.

Cellular Components

a. *Osteoblasts*: They are cuboidal cells with well-developed rough endoplasmic reticulum, a large Golgi apparatus, and secretory vesicles. It synthesizes bone matrix (osteoid), type I collagen and regulate its mineralization. Each osteoblast carries out a cycle of matrix synthesis after which it is either buried as an osteocyte or remains on the surface as a resting or inactive osteoblast. Osteoblasts are derived from the progenitor cells at sites of bone formation. These progenitor cells belong to the mesenchymal cell family. In contrast, osteoclasts originate from blood–borne monocytes.
b. *Osteoclasts*: They are large multinucleated giant cells. They originate from hematopoietic tissue and are formed by the fusion of mononuclear cells of asynchronous populations. Generally they are found in bay like depressions in the bone called *Howship's lacunae*. The part of the cell in contact with bone shows a convoluted surface and a ruffled border from which hydrolytic enzymes are believed to be secreted. The ruffled border is surrounded by a clear zone, which contains cytoplasm exclusively and is believed to be associated with binding of cells to the bone surface and isolation of areas of resorptive activity.
c. *Osteocytes*: Alveolar bone is formed during foetal growth by intramembranous ossification and consists of a calcified matrix with osteocytes enclosed within a space called lacunae. The osteocytes extend processes into canaliculi that radiate from the lacunae. The main function of these canaliculi is to bring oxygen and nutrients to the osteocytes through the blood and remove metabolic waste products.

The main cells responsible for bone resorption are osteoclasts, on rare occasions bone resorption by osteocytes has been reported which is called as *osteocytic osteolysis*.

Osseous Topography

The anatomy of the alveolar bone varies from patient to patient. Normally it conforms to the root prominence, with intervening depressions that taper towards the margin. The factors that affect the height and thickness of the facial and lingual bony plates are alignment of the teeth, angulation of the root to the bone and the occlusal forces.

When the teeth are in a labial version, the margin of the bone is located more apically and is thinned to a knife-edge as compared to the teeth in proper alignment. When teeth are in a lingual version, the facial bony plate is thicker than normal and the margin is blunt, rounded and horizontal rather than arcuate.

Fenestrations and Dehiscences (Fig. 2.18)

Fenestrations are isolated areas in which the root surface is covered only by the periosteum and gingiva, but in these

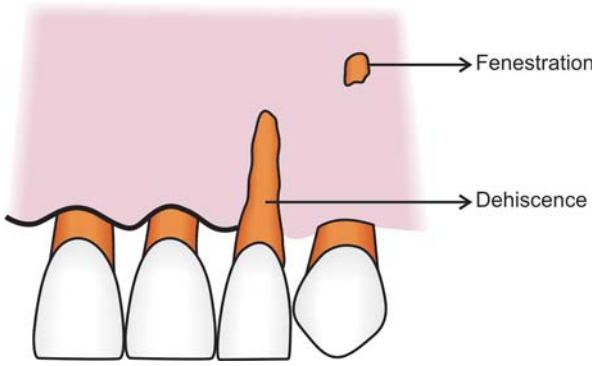


Fig. 2.18: Fenestration and dehiscence

situations, the marginal bone remains intact. When the marginal bone is also denuded, the defect is called dehiscence.

These defects are seen more often on the facial bone than on the lingual, and are more common in anterior teeth than on the posterior. The etiologies of these defects are not clear; some of the predisposing factors are root prominence, malposition, and teeth in labial version with thin bony plates. The diagnosis of these defects is important as it may affect the outcome of the surgical treatment.

Periosteum and Endosteum

The tissue covering the outer surface of bone is termed as periosteum, where as the tissue lining the internal bone cavities is called endosteum. The periosteum consists of two layers, the inner layer, next to the bone surface, consists of bone cells that have the potential to differentiate into osteoblasts and an outer layer which is more fibrous containing blood vessels and nerves.

The endosteum is composed of a single layer of osteoprogenitor cells and a small amount of connective tissue.

Remodeling and Resorption

During both embryonic bone development and the entire pre-adult period of human growth, bone is being formed very rapidly, primarily on the periosteal surface. Simultaneously, bone is being destroyed along the endosteal surface and at focal points along the periosteal surface (bone modeling).

Bone growth occurs by apposition of an organic matrix that is deposited by osteoblasts. Although the alveolar bone tissue is constantly changing in its internal organization, it retains approximately the same form from childhood through adult life. Haversian systems (osteons) are the internal mechanisms that bring vascular supply to bones too thick to be supplied only by surface vessels. Bone deposition by osteoblasts is balanced by resorption brought about by osteoclasts during tissue remodeling and repair.

Osteoblasts, lay down nonmineralized bone matrix called osteoid, while new osteoid is being deposited, the older osteoid becomes mineralized. Bone resorption is a complex process and appears as eroded bone surfaces, namely *Howship's lacunae*. It has been suggested that bone resorption at any site is a chemotactic phenomenon. This is, initiated by the release of some chemotactic factors like interleukin-1 and 6, which attract the monocytes to the target site. Osteoblasts release Leukemia inhibiting factor (LIF), which *coalesce* monocytes to form multinucleated osteoclasts, which then resorb bone.

During bone resorption three processes occur:

a. Decalcification
b. Degradation of matrix
c. Transport of soluble factors to the extra cellular fluid.

Since calcified matrix is resistant to proteases of all kinds, bone must first be decalcified. This is achieved at the ruffled border of the osteoclasts by secretion of some acids such as citric acid and lactic acid, which chelate bone, the low pH leads to increase in solubility of hydroxyapatite. The next step is degradation of matrix, takes place by collagenase enzymes and other proteases like cathepsin-B. Collagenolysis occurs outside the osteoclast. Finally, the break down products of bone are transported to the extracellular fluids and to the blood vascular system.

Tencate described the following sequence of events during bone resorption:

a. Attachment of osteoclasts to the mineralized bone surface.
b. Creation of a sealed acidic environment, which demineralizes the bone and exposes the organic matrix.
c. Degradation of exposed organic matrix by the action of enzymes such as acid phosphatase and cathepsine.
d. Sequestering of mineral ions and amino acids within the osteoclasts.

Blood Supply to the Bone

The vessels enter the interdental septa through nutrient canals together with veins, nerves, and lymphatics. The alveolar arteries sends off branches through periodontal ligament and enter the marrow spaces through the perforations in the cribriform plate.

Clinical Considerations

Bone although one of the hardest tissue of human body, is biologically a highly plastic tissue. Bone is resorbed on the side of pressure and apposed on the side of tension. It has been shown that on the pressure side there is an increase in the level of cyclic adenosine monophosphate (cAMP) in cells, which may play a role in bone resorption.

The most frequent and harmful change in the alveolar process is that which is associated with periodontal diseases. The bone resorption caused by periodontal diseases is usually symmetrical, occurs in episodic manner, and is both of the horizontal and vertical type. Once lost, this bone is very difficult to regenerate. Regeneration of just a few millimeters of bone that has been lost is the greatest challenge to the periodontists across the world.

CEMENTUM

Definition

Cementum is a calcified avascular mesenchymal tissue that forms the outer covering of the anatomic root. It provides anchorage mainly to the principal fibers of periodontal ligament. Two sources of collagen fibers can be found in the cementum:

1. Sharpey's (extrinsic) fibers which are formed by the fibroblasts.
2. Fibers belonging to the cementum matrix per se (intrinsic) produced by cementoblasts.

   Two types of cementum were described earlier:

   a. Acellular cementum/primary cementum (Fig. 2.19).
   b. Cellular cementum/secondary cementum.

The differences between the acellular and cellular cementum are given in Table 2.1.

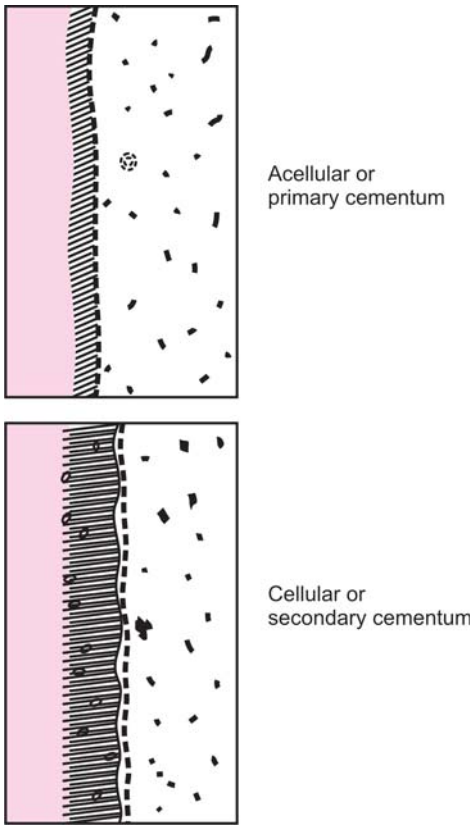


Fig. 2.19: Acellular/cellular cementum

Table 2.1: Differences between acellular and cellular cementum

Acellular cementum	*Cellular cementum*
a. Forms during root formation	Forms after the eruption of the tooth and in response to functional demands
b. Does not contain any cells	Contains cementocytes
c. Seen at the coronal portion of root	Seen at a more apical portion of root
d. Formation is slow	Deposition is more rapid
e. Arrangement of collagen fibers are more organized	Collagen fibers are irregularly arranged

Classification

Depending on location, morphology and histological appearance, Shroeder and Page have classified cementum as:

a. *Acellular afibrillar cementum* (AAC): It contains only the mineralized ground substance. It does not contain collagen fibers nor does it exhibit entrapped cemento-

cytes. It is a product of cementoblasts and is found almost exclusively on the enamel near the cementoenamel junction with a thickness of 1 to 15 µm.

b. *Acellular extrinsic fiber cementum* (AEFC): By definition it is composed primarily of Sharpey's fibers of periodontal ligament but does not contain cementocytes. Developmentally they come to occupy the coronal one half of the root surface. Its thickness is between 30 and 230 µm.
c. *Cellular mixed stratified cementum* (CMSC): It harbors both intrinsic (cementoblasts derived) and extrinsic (fibroblast derived) fibers and may contain cells. In humans it is seen in the apical third of the roots, apices and furcation areas. Its thickness varies from 100 to 1000 µm.
d. *Cellular intrinsic fiber cementum* (CIFC): It contains only intrinsic fibers secreted by cementoblasts and not by the periodontal ligament fibroblasts. In humans it fills the resorption lacunae.
e. *Intermediate cementum* (or) *the hyaline layer of Hope Well Smith*: It is an ill-defined zone extending from pre-cementoenamel junction to the apical 1/3rd of the root. It appears to contain cellular remnants of Hertwig's Sheath embedded in calcified ground substance. The significance of this layer is that, it contains enamel like proteins, which helps in attachment of cementum to dentin. It has been observed by many that, when this layer is removed during root planing procedure, the resultant reparative cementum that is formed will not be attached firmly on the dentin.

Functions

a. Primary function of cementum is to provide anchorage to the tooth in its alveolus. This is achieved through the collagen fiber bundles of the periodontal ligament, whose ends are embedded in cementum.
b. Cementum also plays an important role in maintaining occlusal relationships, whenever the incisal and occlusal surfaces are abraded due to attrition, the tooth supra erupts in order to compensate for the loss and deposition of new cementum occurs at the apical root area.

Composition

The cementum is composed of both inorganic (46%) and organic matter. The organic matrix is chiefly composed of 90 percent Type I collagen, 5 percent Type III collagen and noncollagenous proteins like enamel proteins, adhesion molecules like tenascin and fibronectin, glycosaminoglycans like chondroitin sulfate, dermatan sulfate and heparan sulfate which constitute the remaining organic matrix.

Thickness of Cementum

Formation of cementum is a continuous process, the formative rate of which varies throughout life. It is most rapid at the apical regions. At the coronal half the thickness varies from 16 to 60 µm (almost the thickness of hair) and at the apical third it varies from 150 to 200 µm. It is thicker in the distal surfaces as compared to the mesial surfaces and this can be explained by functional stimulation following mesial migration.

Hypercementosis or cemental hyperplasia is a prominent thickening of the cementum. It can be localized or generalized. It may appear as a generalized thickening of the cementum, with nodular enlargement at the apex or as spike like projections (cemental spikes). The etiology of hypercementosis is not very well understood. The spike like projections could be as a result of excessive tension from orthodontic appliance or occlusal forces. The generalized type may be associated with a variety of situations, like teeth without antagonists, in teeth with chronic pulpal and periapical infections. Hypercementosis of the entire dentition may be seen in patients with Paget's disease.

Cementoenamel Junction (Fig. 2.20)

At the cementoenamel junction three types of relationships may exist. In about 60 to 65 percent of cases, the cementum overlaps the enamel, in about 30 percent of cases, end-to-end relationship of enamel and cementum is seen and in 5 to 10 percent, the cementum and enamel fail to meet.

Cemental Resorption and Repair

Cemental resorption may be caused by local, systemic or idiopathic factors. Local conditions that contribute to

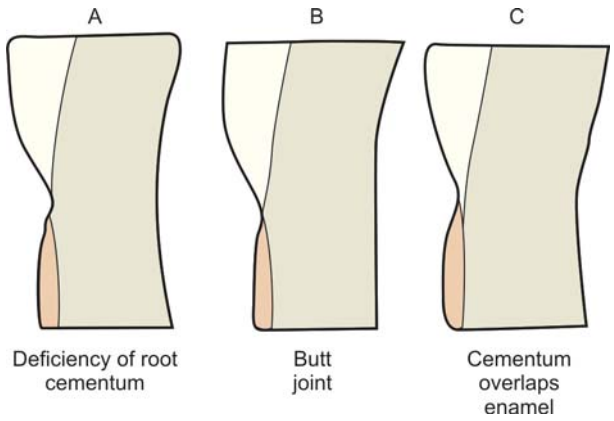


Fig. 2.20: Configuration of cementoenamel junction

cemental resorption are trauma from occlusion, orthodontic tooth movement, pressure from erupting teeth, cysts and tumors, teeth without functional antagonist, periapical disease and periodontal disease. Systemic conditions that may predispose to cemental resorption are calcium deficiency, hypothyroidism and Paget's disease.

The resorptive process may not necessarily be a continuous process; it may alternate with periods of repair and deposition, which can be demarcated by formation of reversal line. Remodelling of cementum requires the presence of viable connective tissue and can occur even in non-vital teeth.

Cementum is not exposed to the oral environment because it is covered by alveolar bone and gingiva. In cases of gingival recession and as a consequence of loss of attachment in pocket formation, cementum can become exposed to the oral environment. Once exposed, organic substances, inorganic ions and bacteria penetrate the sufficiently permeable cementum. Caries of the cementum may also develop.

KEYPOINTS

The Gingiva

1. The gingiva is that part of the oral mucosa that covers the alveolar process of the jaws and surrounds the necks of the teeth.
2. Anatomically it is divided into marginal, attached and interdental gingiva.
3. Microscopically, the gingival epithelium is divided into keratinized, i.e. oral epithelium and non-keratinized, i.e. junctional and sulcular epithelium.
4. The keratinized epithelium of the gingiva consists of four layers, namely stratum basale, stratum spinosum, stratum granulosum and stratum corneum.
5. The granular layer, which is seen in the oral epithelium is absent in sulcular and junctional epithelium.
6. Junctional epithelium is attached to the tooth surface by hemidesmosomes and basal lamina.
7. Lamina propria/gingival connective tissue consists of cells, fibers, and blood vessels, embedded in the extracellular matrix.

Tooth-supporting Structures

Periodontal Ligament

1. Periodontal ligament is a connective tissue structure that surrounds the root and connects it with the bone.
2. It consists of cells and extra-cellular substances.
3. Cellular components are categorized as synthetic cells, resorptive cells, progenitor cells, epithelial cells and connective tissue cells.
4. Extracellular matrix is composed of fibers like collagen, oxytalan and ground substance with proteoglycans and glycoproteins.
5. The principal fibers of periodontal ligament are arranged in six groups, i.e. trans-septal group, alveolar crest group, horizontal, oblique, apical and inter-radicular group.
6. In the connective tissue other structures like blood vessels, nerves, and lymphatics are also present.
7. Functions of periodontal ligament are explained under physical function, formative and remodelling function, nutritive and sensory function.
8. Finally any trauma to the periodontal ligament can result in the loss of alveolar bone and widening of ligament but once the trauma is removed, repair usually takes place.
9. When the inflammation extends from the gingiva to the periodontal ligament, destruction of periodontal structures will result, which is difficult to regenerate, hence the disease of periodontal ligament are often irreversible.

Alveolar Bone

1. Alveolar bone is that portion of the maxilla and mandible that forms and supports the tooth sockets.
2. It consists of inner and outer cortical plate, the bone lining the socket and an interior portion of cancellous bone.
3. It has two basic constituents (a) cells like osteoblasts, osteoclasts, and osteocytes, (b) extracellular matrix made up of 65 percent inorganic and 35 percent organic. Inorganic is composed of calcium, phosphate along with some trace elements. Organic matrix consists of 90 percent Type I collagen with small amounts of non-collagenous proteins such as osteonectin, osteocalcin, bone morphogenetic proteins, etc.

4. Fenestrations are isolated areas in which the root surface is covered only by periosteum and gingiva, but the marginal bone remains intact. When marginal bone is also involved, the defect is called dehiscence.
5. Bone resorption occurs by three processes—(a) decalcification, (b) degradation of matrix and (c) transport of soluble factors to the extracellular fluid.

Cementum

1. Cementum is a calcified avascular mesenchymal tissue that forms the outer covering of the anatomic root.
2. Acellular cementum forms during root formation and is seen at the coronal portion of the root, whereas cellular cementum forms after eruption of the tooth and is seen apically on the root.
3. The cementum consists of 46 percent inorganic matter and the rest 90 percent organic being, Type I collagen and the remaining consists of non-collagenous proteins.
4. Three types of relationships of cementum may exist at the cementoenamel junction. In 60 to 65 percent of cases, cementum overlaps the enamel, in 30 percent, edge-to-edge butt joint exists and in 5 to 10 percent, the cementum and enamel do not meet.

KNOW MORE ...

Gingiva

- Normally, the gingiva extends from marginal gingiva to the mucogingival junction but on the palatal surface it merges with the palatal mucosa. Hence there is no mucogingival line present on the palate.
- It was observed that, clinically a free gingival groove is present only in about 30-40 percent of adults and is positioned at a level corresponding to the level of cementoenamel junction (CEJ).
- The free gingival groove is most pronounced in the mandibular incisor and premolar regions on the buccal surfaces and least seen in the mandibular molar and maxillary premolar regions.
- Width of attached gingiva increases with age. Since the mucogingival junction remains stable throughout life, the increasing width of the gingiva may be due to the occlusal wear and supraeruption of the teeth.
- Functions of width of attached gingiva are: (a) It holds the marginal gingiva in position, (b) allows for proper deflection of food, (c) provides mechanical and functional stability.
- Microscopically in the keratinized epithelium, keratinocytes comprise about 90% of the total cell population. The oral epithelium contains the non-keratinocytes which are also called “clear cells” (Melanocytes, Langerhans cells, Merkel’s cells and inflammatory cells). In histologic sections the zone around their nucleus appears lighter than that in the surrounding keratin-producing cell. Hence they are named clear cells.
- Melanocytes are pigment synthesizing cells and are responsible for the production of melanin. The Langerhans cells are believed to play a role in the defence mechanisms of the oral mucosa whereas Merkel’s cells have been suggested to have a sensory function.

Periodontal Ligament

Progenitor cells are also seen in the cellular components of periodontal ligament. They are basically undifferentiated mesenchymal cells which have the capacity to differentiate into either formative cells or resorptive cells depending on the external signaling. Hence, presence of these cells in the periodontal ligament gives it the potential for regenerative phase of healing following periodontal disease.

Cementum

Development and acquired anomalies of cementum

Enamel projections: Most commonly seen in the mandibular furcation areas, when amelogenesis does not cease before the formation of root, there enamel projections may form over a portion of root.

Enamel pearls: They are globule like structures of enamel seen in the cervical portion of the root surface. When Hertwig’s root sheath fails to detach from the dentin surface, cementogenesis may proceed. This will result in large pearls.

Cementicles: They are globular masses of cementum, may be found lying either in periodontal ligament or attached to a root surface. They may have originated from calcified epithelial cell rests of Malassez or calcified Sharpey’s fibers.

Ankylosis: Fusion of the cementum and alveolar bone with obliteration of the periodontal ligament is termed as ankylosis. It occurs in teeth with cemental resorption which may represent a form of abnormal repair.

Alveolar Bone/Process

Bone marrow: In embryos and newborn, the bone cavities are filled with red hematopoietic marrow, which gradually undergoes physiologic changes and gets replaced by yellow inactive type of marrow. In the oral cavity mostly we see yellow type of marrow with occasional foci of red bone marrow.

REVIEW QUESTIONS

The Gingiva

1. Describe the microscopic and macroscopic features of gingiva.
2. Discuss the development, structure and mode of attachment of junctional epithelium.
3. Describe the blood supply to the gingiva.
4. Describe various gingival fiber groups with illustrations.

Tooth-supporting Structures

Periodontal Ligament

1. Describe the development, structure and composition of periodontal ligament.
2. Enumerate various principle fibers of periodontal ligament with the help of illustrations.
3. Discuss in detail the functions of periodontal ligament.

Alveolar Bone

Discuss the alveolar bone with reference to the following:

1. Structure, composition and parts of alveolar bone.
2. Fenestration and dehiscence.
3. Resorption and remodeling.

Cementum

Discuss cementum with reference to the following:

1. Structure and composition of cementum.
2. Cementoenamel junction.
3. Hypercementosis.
4. Classification.

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3 Chapter

Periodontal Structures in Aging Humans

GENERAL EFFECTS OF AGING

Skin

a. The dermis and epidermis are thinned
b. Keratinization is diminished
c. Blood supply is diminished
d. Degeneration of nerve endings occur
e. Capillaries appear to become more fragile which may result in hemangiomas after minor traumas
f. Tissue elasticity is decreased.

Bone

a. Undergoes osteoporosis with aging
b. The bone is rarified, trabeculae are reduced in number and cortical plates are thinned
c. Vascularity is reduced and lacunar resorption is more prominent
d. Increased susceptibility to fracture
e. With age water content of bone is reduced, the mineral crystals are increased in size and collagen fibers are thickened.

AGE CHANGES IN THE PERIODONTIUM (FIG. 3.1)

Gingiva and other Areas of the Oral Mucosa

1. Decreased keratinization.
2. Reduced or unchanged amount of stippling.
3. Decreased connective tissue cellularity.
4. Decreased oxygen consumption—which may reflect upon the metabolic activity.
5. Increased width of attached gingiva.
6. Greater amounts of intercellular substances.
7. Atrophy of the connective tissue with loss of elasticity.
8. Increase in the number of mast cells.

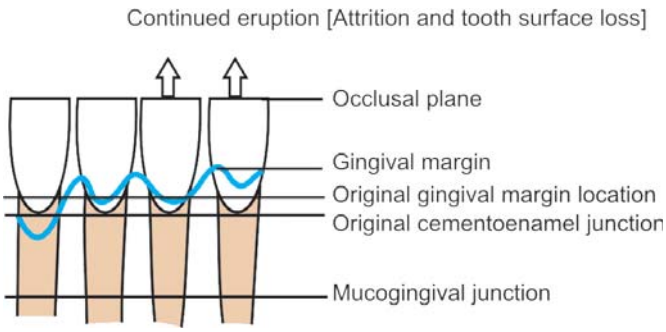

Fig. 3.1: Aging of the periodontium

Periodontal Ligament and Age Changes in the Periodontium

1. Decrease in vascularity.
2. Decrease in mitotic activity.
3. Decrease in fibroblasts.
4. Collagen fibers and mucopolysaccharides are decreased.
5. Increase in elastic fibers and arteriosclerotic changes are seen.
6. Both increase and decrease in the width of the periodontal ligament is seen. The periodontal ligament width is increased as a result of less number of teeth supporting the entire functional load and decrease in its width is associated with reduced strength of the masticatory musculature and continuous deposition of cementum and bone.

Changes in the Alveolar Bone and Cementum

1. Osteoporosis.
2. Decreased vascularity.
3. Reduction in metabolic rate and healing capacity.
4. Resorption activity is increased and the rate of bone formation is decreased.
5. Continuous deposition of cementum occurs with age and greater irregularity in the surface of both the cementum and alveolar bone facing the periodontal ligament is seen.

Bacterial Plaque and Immune Response (Fig. 3.2)

1. Plaque accumulation has been suggested to increase with age.

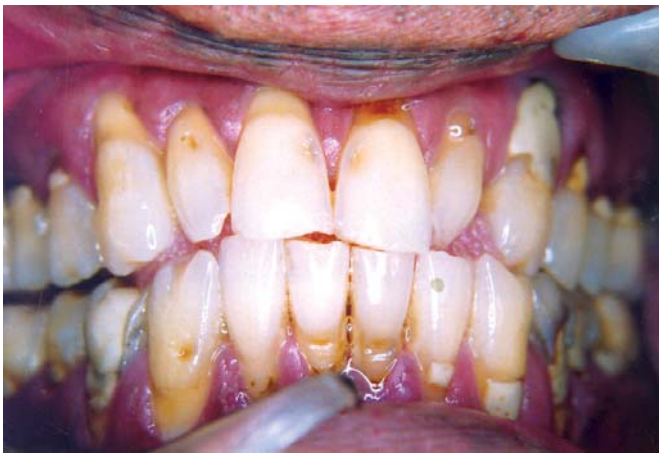

Fig. 3.2: Periodontal changes associated with aging

2. Some studies have shown a qualitative change in the subgingival flora, it has been speculated that a shift occurs in certain periodontal pathogens with age, including increase in number of enteric rods, *Porphyromonas gingivalis* and decreased role for *Actinobacillus actinomycetemcomitans*. Some age-related changes have been shown to affect the host response, but there is no evidence to show that these changes correlate with periodontitis in elderly patients.

EFFECTS OF AGING ON THE PROGRESSION OF PERIODONTAL DISEASES

The conclusions drawn from the various studies are strikingly consistent and show that, age has either no effect or provides a small and clinically insignificant increased risk of loss of periodontal support. Therefore, age has been suggested to be not a true risk factor but a background or an associated factor for periodontitis.

EFFECTS OF TREATMENT ON THE AGING INDIVIDUALS

The few studies that have been done so far have clearly demonstrated that in spite of certain changes in the periodontium with aging, no differences in response to nonsurgical or surgical treatment have been shown for periodontitis.

KNOW MORE ...

Age Changes in the Perodontium

With increasing age, the gingival connective tissue becomes coarser and denser.

Tissue culture studies of gingival fibroblasts from older individuals exhibits reduction in the rate of proliferation, decrease in the quantity and quality of proteoglycans with reduced protein and collagen production.

REVIEW QUESTIONS

1. Describe the age changes in the periodontium.
2. Role of aging on the progression of periodontal diseases.

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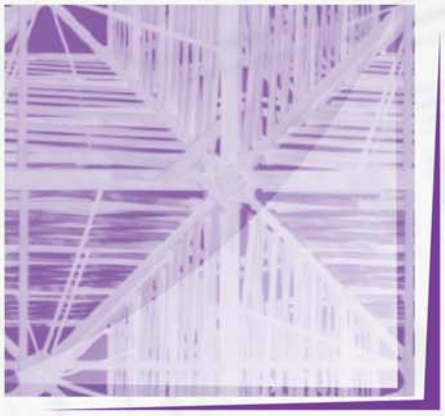

Part II

Classification and Epidemiology of Periodontal Diseases

4 Chapter

Classification Systems of Periodontal Diseases

- NEED FOR CLASSIFICATION
- CURRENT CLASSIFICATION SYSTEMS OF PERIODONTAL DISEASES
  - Gingivitis
  - Periodontitis
- SYNOPSIS OF TYPES AND CHARACTERISTICS OF GINGIVAL DISEASES
- SYNOPSIS OF TYPES AND CHARACTERISTICS OF PERIODONTAL DISEASES

NEED FOR CLASSIFICATION

- For the purpose of diagnosis, prognosis and treatment planning.
- To understand the etiology and pathology of the diseases of the periodontium.
- For logical, systematic separation and organization of knowledge about disease.
- Facts can be filed for future references.
- Helps to communicate among clinicians, researchers, educators, students, epidemiologists and public health workers.

CURRENT CLASSIFICATION SYSTEMS OF PERIODONTAL DISEASES

World Workshop in Clinical Periodontics (1988)

Gingivitis

- Childhood gingivitis
- Chronic (adult) gingivitis
- Acute necrotizing ulcerative gingivitis.

Periodontitis

- Adult Periodontitis:
  Possible subgroups:
  - High risk
  - Normal risk
  - Refractory periodontitis
- Early Onset Periodontitis:
  - Localized juvenile periodontitis
  - Rapidly progressive periodontitis
  - Prepubertal periodontitis
    - * Localized
    - * Generalized
  - Periodontitis associated with systemic diseases.

World Workshop in Clinical Periodontics (1989)

I. Adult periodontitis.
II. Early onset periodontitis.

- Prepubertal
  - Generalized or localized
- Juvenile
  - Generalized or localized
- Rapidly progressive periodontitis

III. Periodontitis associated with systemic diseases
- Down's syndrome
- Diabetes – Type I
- Papillon-Lefévre syndrome
- AIDS and other diseases

IV. Necrotizing ulcerative periodontitis

V. Refractory periodontitis.

Genco (1990)

- Periodontitis in adults.
- Periodontitis in juveniles
  - Localized form
  - Generalized form
- Periodontitis with systemic involvement
  - Primary neutrophil disorders
  - Secondary or associated neutrophil impairment.
  - Other systemic diseases
- Miscellaneous conditions.

Ranney (1993)

Gingivitis

- Gingivitis, plaque/bacterial induced
- Non-aggravated
- Systemically-aggravated by sex hormones, drugs, systemic diseases
- Necrotizing ulcerative gingivitis
- Systemic determinants unknown
- Related to HIV
- Gingivitis, non-plaque induced
- Associated with skin diseases, allergy and infections.

Periodontitis

- Adult periodontitis
  - Non-aggravated
  - Systematically-aggravated (neutropenias, leukemias, lazy leukocyte syndrome, AIDS, diabetes mellitus, Crohn's disease, Addison's disease).
- Early onset periodontitis:
  - Localized early onset periodontitis
  - Neutrophil abnormality
  - Generalized early onset periodontitis, neutrophil abnormality, immunodeficient
- Early onset periodontitis-related to systemic disease:
  - Leukocyte adhesion deficiency, hypophosphatasia, Papillon-Lefevre syndrome, neutropenias, leukemias, Chediak-Higashi syndrome, AIDS, diabetes mellitus type-I, trisomy-21, histiocytosis X, Ehlers-Danlos syndrome (type VIII)
- Early onset periodontitis, systemic determinants unknown
- Necrotizing ulcerative periodontitis
  - Systemic determinants unknown
  - Related to HIV
  - Related to nutrition
- Periodontal abscess

European Workshop in Periodontology (1993)

- Adult periodontitis
- Early onset periodontitis
- Necrotizing periodontitis

All the above-mentioned classification systems have been widely used by clinicians and research scientists throughout the world, unfortunately they have been many shortcomings including:

1. Considerable overlap in disease categories.
2. Absence of a gingival disease component.
3. Inappropriate emphasis on age of onset of disease and rates of progression.
4. Inadequate or unclear classification criteria.

Hence, the need for a revised classification system for periodontal diseases was emphasized and in 1999, the international workshop had considered designing a new classification system.

Classification of Periodontal Disease and Condition

By AAP 1999 (International Workshop for Classification of Periodontal Disease).

Table 4.1: Synopsis of types and characteristics of gingival diseases

Types of gingival disease	*Causes*	*Signs and symptoms*	*Treatment*
Gingivitis	Bacterial plaque, local plaque—retention factors	Gingival redness and swelling, bleeding, does not cause loss of clinical attachment	Debridement, plaque control, correct plaque retentive factors, supportive periodontal therapy
Acute necrotizing gingivitis	Bacterial plaque, may be associated with AIDS at any age	Pain, gingival redness, swelling, bleeding, necrosis of interproximal papilla	Debridement, plaque control, antimicrobial rinse, supportive periodontal therapy
Desquamative gingival disease	Skin diseases, lichen planus, pemphigus and cicatricial pemphigoid	Gingival redness, epithelial denudation, pain with trauma as on eating and brushing	Gentle plaque control, palliative and symptomatic therapy, supportive periodontal therapy
Gingivitis associated with systemic diseases	Manifestation of systemic diseases in gingiva	Dependent on systemic disease	Treatment of systemic disease, atraumatic plaque control, antimicrobial rinse, supportive periodontal therapy
Gingivitis associated with pregnancy	Bacterial plaque, local plaque, retentive factors, hormonal influence	Gingival redness and swelling, bleeding, pyogenic granuloma	Debridement and plaque control, supportive periodontal therapy, possible excision of pyogenic granuloma
Drug-induced gingival enlargement	Calcium channel blocking drugs, phenytoin, cyclosporine	Gingival enlargement	Debridement and plaque control, surgical excision, use of alternative medications, supportive periodontal therapy
Allergic reaction	Local allergens	Gingival redness and swelling	Identification and elimination of allergic agent
Herpetic gingivostomatitis	Herpes type I virus	Pain, vesicle formation, ulceration	Palliative and symptomatic therapy, antiviral medication
Gingival disease of specific bacteria or fungal origin	*Neisseria gonorrhoeae*, *Treponema pallidum*, streptococcal species, *Candida*, histoplasmosis	Varies according to infectious agent	Identification and elimination or control of infectious agent, appropriate chemotherapy

Gingival Disease (Table 4.1)

A. *Dental plaque-induced gingival diseases*: These diseases may occur on a periodontium with no attachment loss or on one with attachment loss that is stable and not progressing.

1. *Gingivitis associated with dental plaque only*:
   a. Without local contributing factors.
   b. With local contributing factors
2. *Gingival diseases modified by systemic factors:*
   a. Associated with endocrine system:

i. Puberty-associated gingivitis
ii. Menstrual cycle-associated gingivitis
iii. Pregnancy-associated
– Gingivitis
– Pyogenic granuloma
iv. Diabetes mellitus-associated gingivitis

b. Associated with blood dyscrasias:
i. Leukemia-associated gingivitis
ii. Others

3. *Gingival diseases modified by medications*:
a. Drug-influenced gingival diseases
i. Drug-influenced gingival enlargements.
ii. Drug-influenced gingivitis
– Oral contraceptive-associated gingivitis
– Others

4. *Gingival diseases modified by malnutrition*:
a. Ascorbic acid deficiency gingivitis
b. Others

B. *Nonplaque-induced gingival lesions*:

1. Gingival diseases of specific bacterial origin.
a. *Neisseria gonorrhoeae*
b. *Treponema pallidum*
c. Streptococcal species
d. Others

2. Gingival diseases of viral origin
a. Herpes virus infections
• Primary herpetic gingivostomatitis
• Recurrent oral herpes
• Varicella zoster
b. Others

3. Gingival diseases of fungal origin
a. *Candida* species infections; generalized gingival candidiasis
b. Linear gingival erythema
c. Histoplasmosis
d. Others.

4. Gingival lesions of genetic origin
a. Hereditary gingival fibromatosis
b. Others

5. Gingival manifestations of systemic conditions
a. Mucocutaneous lesions:
i. Lichen planus
ii. Pemphigoid
iii. Pemphigus vulgaris
iv. Erythema multiforme
v. Lupus erythematosus
vi. Drug induced
vii. Others
b. Allergic reactions:
i. Dental restorative materials
– Mercury
– Nickel
– Acrylic
– Others
ii. Reactions attributable to:
– Toothpaste's or dentifrices
– Mouthrinses or mouthwashes
– Chewing gum additives
– Foods and additives
iii. Others

6. Traumatic lesions (factitious, iatrogenic, or accidental)
a. Chemical injury
b. Physical injury
c. Thermal injury

7. Foreign body reactions

8. Not otherwise specified (NOS).

Chronic Periodontitis (Table 4.2)

a. Localized — Less than 30 percent of sites involved or,
b. Generalized — More than 30 percent of sites involved.
c. Slight — 1 to 2 mm clinical attachment loss.
d. Moderate — 3 to 4 mm clinical attachment loss and,
e. Severe — More than 5 mm clinical attachment loss.

Aggressive Periodontitis

a. Localized–slight, moderate or severe
b. Generalized.

Table 4.2: Synopsis of types and characteristics of periodontal diseases

Types of periodontal disease	*Causes*	*Signs and symptoms*	*Treatment*
Chronic periodontitis	Bacterial plaque, smoking, local plaque retentive factors such as dental calculus and faulty restorations	Overall slow progression with generalized periodontal pockets, bone and clinical attachment loss, may be generalized or localized	Plaque control, smoking cessation, scaling and root planing, correction of local plaque retentive factors antimicrobial chemotherapy, periodontal surgery, supportive periodontal therapy
Aggressive periodontitis	Bacterial plaque, superinfection with specific periodontal bacteria, possible impaired host response, smoking	Severe and rapid periodontal destruction possibly followed by periods of remission, may be generalized or localized	Specific, antimicrobial therapy based on microbial analysis, smoking cessation, debridement, possible periodontal surgery, supportive periodontal therapy
Refractory periodontitis of any type	Bacterial plaque, superinfection, with specific, periodontal bacteria, possible impaired host response, smoking	Progression of disease despite good conventional therapy and supportive periodontal therapy	Specific, antimicrobial, therapy based on microbial analysis, smoking cessation debridement, possible perio-dontal surgery, supportive periodontal therapy
Periodontitis as a manifestation of systemic diseases	Associated with disorders of the blood or blood forming organs such as neutropenia, leukemia or genetic disorders	Generalized and localized forms of severe destruction of bone and connective tissue tooth support	Treatment of systemic disease, atraumatic plaque control, antimicrobial rinse, supportive periodontal therapy
Juvenile periodontitis: localized and generalized	Probably major autosomal gene effect and infection with *Actinobacillus actinomycetemcomitans*	Localized juvenile, periodontitis: typically loss of bone support of first molars and incisors. Generalized juvenile periodontitis: generalized loss of support throughout dentition	Scaling and root planing, specific antimicrobial therapy based on microbial analysis, possible regenerative surgery, supportive periodontal therapy
Periodontitis associated with endodontic lesions	May be of endodontic or periodontal origin	Periodontal pocket extending to area of endodontic lesion	If primarily of endodontic origin, endodontic therapy alone, if primarily of periodontal origin, endodontic and periodontal therapy or extraction may be necessary

Contd...

Contd...

Types of periodontal disease	*Causes*	*Signs and symptoms*	*Treatment*
Periodontal abscess	Subgingival bacteria	Painful, acute swelling of periodontal tissues associated with deep periodontal pocket	Nonsurgical or surgical debridement, antibiotic therapy, regeneration of lost periodontal support is often a possibility
Acute necrotizing periodontitis	Immunocompromised, may be associated with HIV	Pain, rapid loss of bone and tooth support associated with gingival and bony necrosis	Debridement, atraumatic plaque control, analgesic medication, antimicrobial rinse, supportive periodontal therapy

Periodontitis as a Manifestation of Systemic Diseases

a. Associated with hematological disorders:
   i. Acquired neutropenia
   ii. Leukemias
   iii. Others
b. Associated with genetic disorders:
   i. Familial and cyclic neutropenia
   ii. Down's syndrome
   iii. Leukocyte adhesion deficiency syndrome
   iv. Papillon-Lefévre syndrome
   v. Chediak-Higashi syndrome
   vi. Histiocytosis syndrome
   vii. Glycogen storage disease
   viii. Infantile genetic agranulocytosis
   ix. Cohen syndrome
   x. Ehlers-Danlos syndrome (Types IV and VIII)
   xi. Hypophosphatasia
   xii. Others

Necrotizing Periodontal Diseases

a. Necrotizing ulcerative gingivitis
b. Necrotizing ulcerative periodontitis.

Abscesses of the Periodontium

a. Gingival abscess
b. Periodontal abscess
c. Pericoronal abscess

Periodontitis Associated with Endodontic Lesions

Combined periodontic—endodontic lesions.

Developmental or Acquired Deformities and Conditions

a. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis.
   i. Tooth anatomic factors
   ii. Dental restorations/appliances
   iii. Root fractures
   iv. Cervical root resorption and cemental tear
b. Mucogingival deformities and conditions around teeth:
   i. Gingival/soft tissue recession, facial or lingual surfaces, interproximal (papillary)
   ii. Lack of keratinized gingiva
   iii. Decreased vestibular depth
   iv. Aberrant frenum/muscle position
   v. Gingival excess:
      – Pseudopocket
      – Inconsistent gingival margin
      – Excessive gingival display
      – Gingival enlargement
   vi. Abnormal color

c. Mucogingival deformities and conditions on edentulous ridges:
   i. Vertical and/or horizontal ridge deficiency.
   ii. Lack of gingival/keratinized tissue
   iii. Gingival/soft tissue enlargement
   iv. Aberrant frenum/muscle position
   v. Decreased vestibular depth
   vi. Abnormal color
d. Occlusal trauma:
   i. Primary occlusal trauma
   ii. Secondary occlusal trauma.

KNOW MORE ...

Changes Made in the Classification Proposed by International Workshop (1999)

a. There is a separate section on gingival disease.
b. "Adult periodontitis" is being replaced with "chronic periodontitis" and "early onset periodontitis" with "aggressive periodontitis".
c. No separate entity for refractory periodontitis
d. Separate classification has been designed for "periodontitis as a manifestation of systemic diseases".
e. "Necrotizing ulcerative periodontitis" is replaced with "necrotizing periodontal diseases".
f. Additional categories like, "periodontal abscess", "periodontal endodontic lesions" and "developmental or acquired deformities" and conditions are included.

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5 Chapter

Epidemiology of Gingival and Periodontal Diseases

- DEFINITION, TYPES AND AIMS OF EPIDEMIOLOGY
- DEFINITION, USES AND CHARACTERISTICS OF AN INDEX
- VARIOUS INDICES USED TO STUDY PERIODONTAL PROBLEMS
  - To Assess Gingival Inflammation
  - To Measure Periodontal Destruction
  - To Measure Plaque Accumulation
  - To Measure Tooth Mobility
  - To Measure Calculus
  - To Assess Treatment Needs

EPIDEMIOLOGY

Definition

Study of health and disease in populations and how the states are influenced by heredity, biology, physical and social environmental ways of living.

Types of Epidemiologic Research

a. Descriptive studies
b. Analytical studies
c. Experimental epidemiology

Descriptive Studies

These are used to observe and document the occurrence, progression and distribution of a disease or condition in populations, in relation to host and environmental factors (when, where and who).

Measurement of disease: Incidence can be obtained from "Longitudinal studies" and prevalence from "cross-sectional" studies.

a. *Incidence rate*: This is defined as the number of new cases occurring in a defined population during a specific period of time.
b. *Prevalence rate*: This refers to all current cases (new and old) existing at a given point in time or over a period of time in a given population.

Two types are described:

a. Point prevalence
b. Period prevalence

Longitudinal Studies

The observations are repeated in the same population over a prolonged period of time by means of follow-up examinations.

Uses

i. To study the natural history of disease and its future outcome.
ii. For identifying the risk factors of disease.

iii. For finding out the incidence rate or rate of occurrence of new cases.

Disadvantage

They are difficult to organize and time consuming as compared to cross-sectional studies.

Cross-sectional Studies

It is the simplest form of an observational study. It is based on a single examination of a cross-section of population at one point of time (It is also known as prevalence study).

Uses

i. It is more useful for chronic (long-term diseases) as compared to acute (short-term) diseases.
ii. It gives information about the distribution of a disease in a population rather than its etiology.

Analytical Studies

It includes two distinct types:

a. Case control study
b. Cohort study

Case control or retrospective study: The study precedes backwards from effect to cause.

Cohort study: It is a prospective study (incidence study or longitudinal study), which is usually undertaken to obtain, additional evidence to refute and support the existence of an association between the suspected cause and disease.

Experimental Epidemiology

It is used to test hypothesis further by introducing a preventive or therapeutic agent and comparing the outcome in the test subjects with concurrent observations in control groups.

Aims of Epidemiology

Three main aims have been proposed:

a. To describe the distribution and size of disease problems in human populations.
b. To identify the etiological factors in the pathogenesis of disease.
c. To provide the data essential for the planning, implementation and evaluation of services for the prevention, control and treatment of disease.

INDEX

Definition

These are numerical values describing the relative status of the population on a graduated scale with definite upper and lower limits, which are designed to permit and facilitate comparisons with other populations and are classified by the same criteria and methods.

Purposes and Uses of an Index

For Individual Patients

An index can:

i. Provide individual assessment to help a patient to recognize an oral problem.
ii. Reveal the degree of effectiveness of present oral hygiene practices.
iii. Motivate the person in preventive and professional care for the elimination and control of oral disease.
iv. Evaluate the success of an individual and professional treatment over a period of time by comparing index scores.

In Research

An index is used to:

i. Determine the baseline data before the experimental factors are introduced.
ii. Measure the effectiveness of specific agents for the prevention, control and treatment of oral conditions.
iii. Measures the effectiveness of mechanical devices for personal care, such as, toothbrushes, interdental cleaning devices or water irrigators.

In Community Health

An index:

i. Can show the prevalence and trends of incidence, of a particular condition occurring within a given population.

ii. Provides a baseline data to show the existing dental health practices.
iii. Assesses the needs of a community and compares the effects of a community program and evaluates the results.

Characteristics of an Index

1. It should be simple to use and accurate.
2. It should require minimal equipment and expenses.
3. It should have clear-cut criteria, which are readily understandable.
4. It should be as free as possible from subjective interpretation.
5. It should be reproducible by the same examiner or different examiners.
6. Be amenable to statistical analysis, have validity and reliability.
7. Not require an excessive amount of time to complete.
8. Not cause patient discomfort or be otherwise unacceptable to a patient.

Indices Used to Assess the following Periodontal Problems

1. The degree of inflammation of the gingival tissues.
2. The degree of periodontal destruction.
3. The amount of plaque accumulated.
4. The amount of calculus present.
5. In addition, indices are developed to assess the treatment needs.

Indices Used to Assess Gingival Inflammation

a. PMA index by Schour and Massler.
b. Gingivitis component of the periodontal disease.
c. Gingival index by Loe and Sillness.
d. Indices of gingival bleeding.
   i. Sulcus bleeding index (SBI) of Mühlemann and Mazor.
   ii. Papillary bleeding index by Mühlemann.
   iii. Bleeding points index by Lennox and Kopczy K
   iv. Interdental bleeding index by Caton and Polson.
   v. Gingival bleeding index by Ainamo and Bay.

Indices Used to Measure Periodontal Destruction

a. Russell's periodontal index.
b. Periodontal disease index by Ramfjord.
c. Extent and severity index by Carlos and co-workers.
d. Radiographic approaches to measure bone loss.
   i. GBI—Gingival bone count index by Dunning and Leach.
   ii. PSI—Periodontal severity index by Adams and Nystrom.

Indices Used to Measure Plaque Accumulation

a. Plaque component of periodontal disease index by Ramfjord.
b. Simplified oral hygiene index by Greene and Vermillion.
c. Turskey-Gillmore-Glickman modification of the Quigley-Hein plaque index.
d. Plaque index by Sillness and Loe.
e. Modified navy plaque index.
f. Patient hygiene performance index by Podshadley and Haley.
g. Plaque weight.
h. Plaque free score by Grant, Stern and Everett.
i. Plaque control record by O'leary.

Indices Used to Measure Calculus

a. Calculus component of simplified oral hygiene index by Greene and Vermillion.
b. Calculus component of the periodontal disease index by Ramfjord.
c. Probe method of calculus assessment by Volpe and associates.
d. Calculus surface index by Ennever and coworkers.
e. Marginal line calculus index by Mühlemann and Villa.

Indices Used to Assess Treatment Needs

a. Gingival periodontal index.
b. Community periodontal index of treatment needs by Ainamo and associates.
c. PTNS—Periodontal treatment need system by Belini HT.

INDICES USED TO ASSESS GINGIVAL INFLAMMATION

Papillary Marginal Attachment (PMA) Index by Schour and Massler (1944)

The basic philosophy used in the development of the PMA index was very similar to the DMF index, i.e. the number of gingival units affected were counted rather than the severity of the inflammation. A gingival unit is divided into three component parts:

i. Papillary gingiva (P)
ii. Marginal gingiva (M)
iii. Attached gingiva (A)

The presence or absence of inflammation on each gingival unit is recorded as 1 or 0 respectively. The P, M, A numerical values for all the teeth are added separately and then added together to express the PMA index score per person. The developers of this index eventually added a severity component for assessing gingivitis, the papillary units (P) were scored on a scale of 0 to 5, and the marginal (M) and attached gingiva were scored on a scale of 0 to 3.

Gingivitis Component of the Periodontal Disease

The periodontal disease index (PDI) is similar to PI, in that; both are used to measure the presence and severity of periodontal disease. The PDI does so by combining the assessments of gingivitis and gingival sulcus depth on six selected teeth (# 3, 9, 12, 19, 25 and 28). This group of teeth frequently referred to as the Ramfjord teeth, have been tested as reliable indicators for the various regions of the oral cavity. Calculus and plaque are also examined to assist in formulating a comprehensive assessment of periodontal status.

A numerical score for the gingival status component of the PDI is obtained by adding the values for all the gingival units and dividing it by the number of teeth present. This index has been used in epidemiological surveys, longitudinal studies and clinical trials.

Gingival Index by Loe H and Sillness J (1963)

This index was solely developed for the purpose of assessing the severity of gingivitis and its location in four possible areas.

Method

The severity of gingivitis is scored on all surfaces of all teeth, or selected teeth, or on selected surfaces of all teeth, or, selected teeth. The tissues surrounding each tooth are divided into four gingival scoring units:

- Distal facial papillae,
- Facial margin,
- Mesial facial papillae,
- Entire lingual gingival margin.

A blunt instrument is used for recording the scores based on the following criteria:

0 — No inflammation
1 — Mild inflammation, no bleeding elicited on probing
2 — Moderate inflammation, bleeding on probing
3 — Severe inflammation

The scores around each tooth are added and divided by four to arrive at the score for that particular tooth. Total all the teeth scores and divide it by the number of teeth. This provides the gingival index score per person. The numerical values are correlated as follows:

0.1 to 1.0 — Mild gingivitis
1.1 to 2.0 — Moderate gingivitis
2.1 to 3.0 — Severe gingivitis

Indices of Gingival Bleeding

Sulcular Bleeding Index by Mühlemann and Son (1971)

The purpose of the index is to locate areas of the gingival sulcus that bleed upon gentle probing and thus recognize and record the presence of early inflammatory gingival disease. Four gingival units are scored systemically for each tooth: the labial and lingual marginal gingiva (M units) and the mesial and distal papillary gingiva (P units). The probe is held parallel with the long axis of the tooth and 30 seconds

after probing; scoring is done based on the criteria, which ranges from 0 to 5. Each of the four gingival units is scored 0 to 5. Scores for the four units are added and divided by four. Adding the scores of the undivided teeth and dividing them by the number of teeth can determine the sulcus-bleeding index.

Scoring criteria:

0 — Normal appearing gingiva, no bleeding upon probing
1 — No color or contour changes, but bleeding on probing
2 — Bleeding on probing, color change (reddening), no edema and contour changes
3 — Bleeding on probing, color change, mild inflammatory edema
4 — Bleeding on probing, color change, severe inflammatory edema.
5 — Spontaneous bleeding on probing, color change, very severe inflammatory edema with or without ulceration.

Papillary Bleeding Index by Mühlemann HR (1977)

It is based on bleeding elicited, following gentle probing of the interdental papilla. A blunt periodontal probe is carefully inserted into the gingival sulcus at the base of the interdental papilla on the mesial aspects, and then moved coronally to the papilla tip. This is repeated on the distal aspect of the same papilla. The intensity of any bleeding thus provoked will be recorded on a scale of 0 to 4.

Scoring criteria:

0 — No bleeding
1 — A single discrete bleeding point appears
2 — Several isolated bleeding points or a single fine line of blood appears
3 — The interdental triangle fills with blood shortly after probing
4 — Profuse bleeding occurs after probing, blood flows immediately into the marginal sulcus.

Bleeding Points Index by Lennox and Kopczy K

The index was developed to assess a patient's oral hygiene performance. It determines the presence or absence of gingival bleeding interproximally and on the facial and lingual surfaces of each tooth. A periodontal probe is drawn horizontally through the gingival crevice of a gradient, and the gingiva is examined for bleeding after 30 seconds.

Interdental Bleeding Index by Caton and Polson

The index utilizes a triangle-shaped toothpick made of soft, pliable wood to stimulate the interproximal gingival tissue. The interproximal cleaner is inserted horizontally between the teeth from the facial surface, depressing the interproximal papillae by up to 2 mm. The wooden cleaner is inserted and removed four times, and the presence or absence of bleeding within 15 seconds is noted. The score is determined by dividing the number of bleeding sites by the number of sites evaluated.

Gingival Bleeding Index by Ainamo and Bay

The index was developed as an easy and suitable way for the practitioner to assess a patient's progress in plaque control. The presence or absence of gingival bleeding is determined by gentle probing of the gingival crevice with a periodontal probe. The appearance of the bleeding within 10 seconds indicates a positive score, which is expressed as a percentage of the total number of gingival margins examined.

INDICES USED TO MEASURE PERIODONTAL DESTRUCTION

Russell's Periodontal Index by Russell AL (1956)

The index was intended to estimate deeper periodontal disease by measuring the presence or absence of the gingival inflammation and its severity, pocket formation and masticatory function.

All the teeth present are examined. All of the gingival tissues surrounding each tooth are assessed for gingival inflammation and periodontal involvement. Russell chooses the scoring values (0, 1, 2, 6, 8) in order to relate the stages of the disease in an epidemiological survey to the clinical conditions observed.

$$\text{PI score per person} = \frac{\text{Sum of individual scores}}{\text{Number of teeth present}}$$

Since only a mouth mirror and no calibrated probe or radiographs is used while performing the PI examination, the results tend to underestimate the true level of periodontal disease. The number of periodontal pockets without obvious supragingival calculus is also underestimated in the periodontal index.

Scoring Criteria

Score	*Criteria and scoring for field studies*	*Additional X-ray criteria followed in the clinical test*
0	*Negative:* There is neither overt inflammation in the investing tissues nor loss of function due to destruction of supporting tissues	Radiographic appearance is essentially normal
1	*Mild gingivitis:* There is an overt area of inflammation in the free gingiva, but this area does not circumscribe the tooth	
2	*Gingivitis:* Inflammation completely circumscribing the tooth, but there is no apparent break in the epithelial attachment	
4	Used when radiographs are available	There is early notch-like resorption of alveolar crest
6	*Gingivitis with pocket formation:* The epithelial attachment has been broken and there is a pocket. There is no interference with normal masticatory functions, the tooth is firm and has not drifted. There is horizontal bone loss involving the entire alveolar crest, up to half of the length of the tooth root	There is horizontal bone loss involving the entire alveolar crest, up to half of the length of the tooth root
8	*Advanced destruction with loss of masticatory function:* The tooth may be loose, may have drifted, may sound dull on percussion and may be depressible in socket	There is advanced bone loss, involving more than one-half of the length of tooth root, infrabony defects, widening of periodontal ligament, root resorption.

Clinical Conditions and Periodontal Scores

Clinical conditions	*Group PI scores*	*Stage of disease*
Clinically-normal supportive tissues	0 to 0.2	
Simple gingivitis	0.3 to 0.9	
Beginning of destructive–periodontal disease	0.7 to 1.9	Reversible
Established destructive periodontal disease	1.6 to 5.0	Irreversible
Terminal disease	3.8 to 8.0	Irreversible

Uses

i. Epidemiological surveys.
ii. More data can be assembled using periodontal index.
iii. Used in the National Health Survey (NHS).

Periodontal Disease Index by Sigurd P Ramfjord (1959)

This index is a clinician's modification of the Russell's periodontal index for epidemiological surveys of periodontal disease. Emphasis is placed on the recording of the attachment level of the periodontal disease relative to the cementoenamel junction.

Only six selected teeth are scored for assessment of the periodontal status of the oral cavity, which are 1, 6, 21, 24, 36, 41, 44. The first step is scoring of the gingival status. The gingiva around the teeth is dried superficially by gently dabbing it with absorbing cotton. Changes in color, consistency, contour; evidence of ulceration of gingiva is evaluated by a periodontal probe. The next step is recording of the crevice depth related to a cementoenamel junction. For this purpose a University of Michigan 0 probe is used. The end of probe should be placed against the enamel surface coronally to the margin of the gingiva and minimal force is applied in an apical direction maintaining tooth contact. The crevicular measurements are recorded in the following manner. Distance from free gingival margin to the bottom of the gingival crevice or pocket on the buccal and mesial aspect of the each tooth. The buccal measurements should be made at the middle of the buccal surfaces and the mesial measurement should be made at the buccal aspect of the interproximal contact area.

The scoring criteria ranges from 0 to 6. The PDI score for the individual can be obtained by adding the scores for each tooth examined and then, dividing by the number of teeth examined. The PDI score will range from 0 to 6.

Scoring Criteria

0 — Absence of inflammation
1 — Mild to moderate inflammatory gingival changes not extending all around the tooth
2 — Mild to moderately severe gingivitis extending all around the tooth
3 — Severe gingivitis, characterized by marked redness, tendency to bleed and ulceration
4 — Gingival crevice in any of the four measured areas (mesial, distal, buccal, lingual), extending apically to the CEJ, but not more than 3 mm
5 — Gingival crevice in any of the four measured areas extending apically, 3-6 mm from the CEJ
6 — Gingival crevice in any of the four measured areas extending apically more than 6 mm from the CEJ

Extent and Severity Index by Carlos and Coworkers

In this newer model, periodontal disease is viewed as a chronic process with intermittent periods of activity and remission that affects individual teeth and sites around the teeth at different rates within the same mouth. It uses a periodontal probe (NIDR probe) to determine attachment levels. The ESI score is a bivariate statistic. It expresses the percentage of sites that exhibit disease. ESI is based on probe measurements (at the mesiobuccal, interproximal and midbuccal locations on all teeth excluding molars and at the mesiobuccal interproximal and midbuccal of the mesial root of molars) at 14 sites in half of the maxillary arch and at 14 sites in the contralateral mandibular arch. Attachment level measurements are made using the Ramfjord criteria.

Radiographic Approaches to Measure Bone Loss

Gingival Bone Count Index by Dunning and Leach

This index records the gingival condition on a scale of 0 to 3 and the level of the crest of the alveolar bone.

The Periodontitis Severity Index by Adams and Nystrom

The index assesses the presence or absence of periodontitis as the product of clinical inflammation and interproximal bone loss determined radiographically using a modified schei ruler.

INDICES USED TO MEASURE PLAQUE ACCUMULATION

Plaque Component of Periodontal Disease Index by Ramfjord

The index is used on the six teeth selected by Ramfjord (teeth number 3, 9, 12, 19, 25 and 28) after staining with Bismarck brown solution. The criteria is to measure the presence and extent of plaque on a scale of 0 to 3, looking specifically at all interproximal facial and lingual surfaces of the index teeth. The scoring criteria is as follows:

0 — No plaque present.
1 — Plaque present on some but not all interproximal, buccal and lingual surfaces of the tooth.
2 — Plaque present on all interproximal buccal and lingual surfaces, but covering less than one half of the surfaces.
3 — Plaque extending over all interproximal, buccal and lingual surfaces, and covering more than one half of these surfaces.

Only fully-erupted teeth are scored and missing teeth should not be substituted.

$$\text{Plaque score of an individual} = \frac{\text{Total score}}{\text{Number of teeth examined}}$$

Uses

Suitable for:
a. Longitudinal studies of periodontal disease.
b. Epidemiological surveys.
c. Clinical trials of preventive or therapeutic agents.

Simplified Oral Hygiene Index by Greene and Vermillion (1964)

The OHI-S measures the surface area of the tooth that is covered by debris and calculus. It consists of two components:

a. Debris index-simplified (DI-S).
b. Calculus index-simplified (CI-S).

Scoring Criteria for DI-S

0 — No debris or stain present.
1 — Soft debris covering not more than one-third of the tooth surface or the presence of extrinsic stains without other debris, regardless of surface area covered.
2 — Soft debris, covering more than one-third but not more than two-thirds of the exposed tooth surface.
3 — Soft debris, covering more than two-thirds of the exposed tooth surface.

Scoring Criteria for CI-S

0 — No calculus present.
1 — Supragingival calculus covering not more than one-third of the exposed tooth surface.
2 — Supragingival calculus covering more than one-third, but not more than two-thirds of the exposed tooth surface or the presence of the individual flecks of subgingival calculus around the cervical portion of the tooth or both.
3 — Supragingival calculus covering more than two-thirds of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth, or both.

Method

Each component is assessed on a scale of 0 to 3. Only a mouth mirror and a Shepherd's Crook or sickle-type dental explorer, and no disclosing agent are used for examination. The six tooth surfaces examined are No 3, 8, 14, 24 (Facial surface) and No 19, 30 (Lingual surfaces). Each tooth surface is divided horizontally into gingival, middle and incisal thirds. For the DI-S, a dental explorer is placed on the incisal third and moved towards the gingival third and scores are awarded according to the criteria. The DI-S score per person is obtained by totaling the debris score per the tooth surface and dividing it by the number of surfaces examined. The CI-S assessment is performed by gently placing a dental explorer into the distal gingival crevice and drawing it subgingivally from the distal contact area to the mesial contact area. Scoring is done according to the criteria. The CI-S score per person is obtained by totaling the calculus scores per tooth surface and dividing it by the number of surfaces examined. The OHI-S score per person is the total of DI-S and CI-S scores per person. The clinical levels of oral cleanliness for debris that can be associated with groups (DI-S, CI-S) scores are as follows:

Good — 0.0 to 0.6
Fair — 0.7 to 1.8
Poor — 1.9 to 3.0

The clinical levels of oral hygiene that can be associated with group OH1-S scores are as follows:

Good — 0.0 to 1.2
Fair — 1.3 to 3.0
Poor — 3.1 to 6.0

Turesky-Gilmore-Glickman Modification of the Quigley Hein Plaque Index (1970)

Plaque is assessed on the facial and lingual surfaces of all of the teeth after using a disclosing agent. A plaque score per person is obtained by totaling all the plaque scores and dividing it by the number of surfaces examined. This system of scoring plaque is relatively easy to use because of the objective definitions of each numerical score. The strength of this plaque index is its application to longitudinal studies and clinical trials of prevention and therapeutic agents.

Scoring Criteria

0 — No plaque
1 — Separate flecks of plaque at the cervical margin of the tooth
2 — A thin, continuous band of plaque (up to 1 mm) at the cervical margin
3 — A band of plaque wider than 1 mm but covering less than one-third of the crown
4 — Plaque covering at least one-third but less than two-thirds of the crown
5 — Plaque covering two-thirds or more of the crown.

Plaque Index by Sillness and Loe (1964)

It is unique among the indices used for assessment of plaque because it ignores the coronal extent of plaque on the tooth surface area and assesses only the thickness of plaque at the gingival area of the tooth.

Method

The evaluation or scoring is done on the entire dentition or on selected teeth. The surfaces examined are the four gingival areas of the tooth, i.e. the distofacial, facial, mesiofacial and lingual surfaces. A mouth mirror, light source, a dental explorer and air-drying of the teeth and gingiva are used in the scoring of this index. Only plaque of the cervical third of the tooth is evaluated with no attention to plaque that has extended to the middle or incisal thirds.

Scoring Criteria

0 — No plaque in the gingival area
1 — A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be recognized only by running a probe across the tooth surface
2 — Moderate accumulation of soft deposits within the gingival pocket and on the gingival margin and/or adjacent tooth surface that can be seen by naked eye
3 — Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface.

The score for the area is obtained by totaling the four scores per tooth and dividing it by four. The plaque index score for the person is obtained by adding the plaque index scores per tooth and dividing by the number of teeth examined.

Modified Navy Plaque Index

Advantage

It is of value in assessing health education programs and the ability of the individuals to perform oral hygiene practices. A variation of the modified navy plaque index is the DMPI (Distal mesial plaque index), which places more emphasis on the gingival, interproximal areas of a tooth.

Procedure

Each tooth surface is divided into gingival, middle and incisal third. Gingival 3rd is divided into two halves (horizontally) both the gingival halves are again divided longitudinally into distal, middle and mesial thirds. The middle 3rd is divided into distal and mesial halves. The incisal 3rd is not subdivided, thereby emphasizing more of gingival two-thirds of the tooth.

$$\text{Modified navy plaque index} = \frac{\text{Total scores per tooth surface}}{\text{Number of surfaces examined}}$$

Tooth Mobility Indices

Miller's Index (1938)

1. The first distinguishable sign of movement.
2. The movement of the tooth which allows the crown to deviate within 1 mm of its normal position.
3. Easily noticeable and allows the tooth to move more than 1 mm in any direction or to be rotated or depressed in the socket.

Glickman's Index (1972)

- Grade I—Slightly more than normal
- Grade II—Moderately more than normal
- Grade III—Severe mobility faciolingually and/or mesiodistally combined with vertical displacement.

Patient Hygiene Performance Index (PHP) by Podshadley and Hadley

Advantages

i. It was the first index developed for the sole purpose of assessing an individual's performance in removing debris after tooth brushing instruction.
ii. It is easy to use and can be performed.
iii. For individual patient education.

Selection of Teeth and Surfaces

Six surfaces of the six teeth (3, 8, 18, 19, 24 and 30) are selected.

It records presence or absence of debris as 1 or 0 respectively.

$$PHP = \frac{\text{Total debris score}}{\text{No of teeth scored}}$$

Suggested Nominal Scale

Rating		
Excellent	—	score
Good	—	0.1 to 1.7
Fair	—	1.8 to 3.4
Poor	—	3.5 to 5.0

Plaque Weight

1. Sand-blasted standardized mylar foils attached to the lingual surface of lower anterior teeth are weighed on removal after a set time.
2. Removal of plaque directly from the tooth and subsequent weighing is less precise but simpler than the foil techniques.

Plaque-free Score by Grant, Stern and Everett

Purpose

To determine the location, number and percentage of plaque-free surfaces for individual motivation and instruction.

Selection of Teeth and Surfaces

All erupted teeth are included; four surfaces are recorded for each tooth (facial, lingual, mesial and distal).

Procedure

Apply disclosing agent and examine each tooth surface for evidence of plaque and record the surfaces in red color.

Papillary Bleeding on Probing

Total the number of small circles marked for bleeding. For a person with 32 teeth there are 30 interdental areas. The mesial or distal of tooth adjacent to an edentulous area is probed and counted.

Calculations

Plaque-free score = Total the number of teeth present and total the number of surfaces with plaque

$$\text{Plaque-free score} = \frac{\text{Number of plaque-free scores}}{\text{Number of available surfaces}} \times 100$$

Plaque Control Record by O'Leary

Similar to plaque-free score by Grant, Stern and Everett.

Interpretations

Although 0 percent is ideal, less than 10 percent has been suggested as a guideline in periodontal therapy. After initial therapy, when a patient reached 10 percent level of plaque control necessary periodontal and restorative procedures are initiated. In comparison, a similar evaluation using a plaque-free score record would mean a goal of over 90 percent or better plaque-free score before the surgical phase of treatment should be undertaken.

INDICES USED TO MEASURE CALCULUS

Calculus Component of the Simplified Oral Hygiene Index by Greene and Vermillion—Discussed in Plaque Index

Calculus Component of the Periodontal Disease Index by Ramfjord

Advantages

1. It has a high degree of examiner reproducibility.
2. It can be performed quickly and has the best application in epidemiological surveys and longitudinal studies.

Procedure

Six teeth are examined (3, 9, 12, 19, 25 and 28). Four surfaces are recorded (facial, lingual, mesial, distal) with the help of an explorer and score ranges from 0 to 3.

1. Supragingival plaque extending not more than 1 mm
2. Moderate amount of supragingival and subgingival calculus

3. Abundance of supragingival and subgingival calculus

$$\text{Calculus index for an individual} = \frac{\text{Total number of scores}}{\text{Number of teeth}}$$

Probe Method of Calculus Assessment by Volpe and Associates

Advantages and Disadvantages

It has been shown to possess a high degree of interexaminer and intraexaminer reproducibility. However, excessive training under a experienced investigator is required to master it.

Purpose

It is used for longitudinal studies.

Method

The lingual surfaces of six mandibular teeth are measured in millimeter divisions with the help of graduated periodontal probe.

Calculus Surface Index by Ennever and Coworkers

Objective

It is to determine rapidly whether a specific agent has any effect on reducing or preventing supragingival or subgingival calculus.

Four mandibular incisors are examined for the presence or absence by visual and tactile examination.

Scoring Criteria

1. Calculus not exceeding 0.5 mm in width or thickness
2. Calculus not exceeding 1 mm
3. Calculus exceeding more than 1 mm in width and thickness.

Marginal Line Calculus Index by Mühlemann and Villa

Purpose

To assess the supragingival calculus along the margins of the gingiva. It only scores the supragingival calculus formed in the cervical area along the marginal gingiva on the lingual side of the four mandibular incisors.

INDICES USED TO ASSESS TREATMENT NEEDS

Gingival Periodontal Index

It assesses three components of periodontal disease: gingival status, periodontal status, and collectively materia alba, calculus and overhanging restorations. The maxillary and mandibular arches are each divided into three segments: the six anterior teeth, the left posterior teeth, and the right posterior teeth.

Objective

The primary objective in using this index is to determine the tooth or surrounding tissues with the severest condition within each of the six segments. Each segment is assessed for each of the three components of periodontal disease described previously.

Specific Criteria

The specific criteria for the gingival status components of the GPI are as follows:

0 — Tissue tightly adapted to the teeth, firm consistency with physiologic architecture.

1 — Slight to moderate inflammation, as indicated by changes in color and consistency, involving one or more teeth in the same segment but not completely surrounding any one tooth.

2 — The above changes occur either singly or combined completely encircling one or more teeth in a segment.

3 — Marked inflammation, as indicated by loss of surface continuity (ulceration), spontaneous hemorrhage, loss of faciolingual continuity or any interdental papilla, marked deviation from normal contour, recession and clefts.

The area with the highest score determines the gingival score for the entire segment, and the gingival status for the oral cavity is obtained by dividing the sum of the gingival scores by the number of segments.

Community Periodontal Index of Treatment Needs by Ainamo and Associates

Procedure

The periodontal treatment needs are recorded for sextants. Third molars are not included except when they are functioning in place of second molars. The treatment need in a sextant is recorded only when two or more teeth are present and are not indicated for extraction. If only one functioning tooth is remaining in the maxilla, the jaw would be recorded as one sextant.

Missing sextants are indicated with a diagonal line through the appropriate box. The index teeth to be examined are as follows:

WHO Numbering			*American Equivalent*		
17, 16	11	26, 27	2, 3	8	14, 15
47, 46	31	36, 37	31, 30	25	18, 19

The worst findings from these teeth surfaces are recorded. WHO probe is used.

Criteria

Periodontal status:

a. Healthy periodontium
b. Bleeding observed directly or by using mouth mirror.
c. Calculus felt during probing, but entire blunt area of the probe is visible.
d. Pocket 4 or 5 mm
e. Pocket more than 6 mm

Treatment needs:

0 No treatment neede
1 Oral hygiene needs improvement
2 Oral hygiene improvement and professional scaling
3 Oral hygiene improvement, professional scaling and complex treatment.

Advantages

The value of CPITN is that it permits rapid examination of a population to determine periodontal treatment needs.

Disadvantages

1. A great deal of useful information is lost when only the worst score per sextant is recorded.
2. CPITN underestimates the pockets greater than 6 mm in older age groups and overestimates the need for scaling in younger age groups.

Periodontal Treatment Need System by Bellini HT

It attempts to place individuals into one of the four classes based on treatment procedures relative to time requirements. It considers the presence or absence of gingivitis and plaque, and the presence of pockets 5 mm or deeper in each quadrant of mouth.

Criteria for periodontal treatment need system is described in Table 5.1.

Table 5.1: Criteria for periodontal treatment need system

PTNS classification	*Unit*	*Plaque*	*Calculus and/or overhangs*	*Inflam-mation*	*Pocket depth*
Class-0	Mouth	No	No	No	No
Class-A	Mouth	Yes	No	Yes	<5 mm
Class-B	Quadrant	Yes	Yes	Yes	<5 mm
Class-C	Quadrant	Yes	Yes	Yes	>5 mm

KNOW MORE ...

1. Point prevalence

$$= \frac{\text{Number of all current cases at a given point of time}}{\text{Estimated population at the same time}} \times 100$$

Period prevalence

$$= \frac{\text{Number of all existing cases of specified disease at a given point of time}}{\text{Estimated population at risk}} \times 100$$

Other Tooth Mobility Indices

Prichard's Index (1972)

1. Slight mobility
2. Moderate mobility
3. Extensive movement in a lateral or mesiodistal direction combined with vertical displacement in the alveolus
4. Signs can be used for added refinement.

Wasserman's Index (1973)

1. Normal
2. Slight mobility—less than 1 mm of buccolingual movement
3. Moderate mobility—up to approximately 2 mm of buccolingual movement
4. Severe mobility—more than 2 mm of movement.

Nyman's Index (1975)

Degree 0 – Normal to less than 0.2 mm mobility
Degree 1 – Horizontal/mesiodistal mobility of 0.2 to 1 mm
Degree 2 – Horizontal/mesiodistal mobility of 1 to 2 mm
Degree 3 – Horizontal/mesiodistal mobility exceeding 2 mm and/or vertical mobility.

Flezar's Index (1980)

M_0 – Firm tooth
M_1 – Slight increased mobility
M_2 – Definite to considerable increase in mobility but no impairment of function
M_3 – Extreme mobility, a loose tooth that would be incomparable in function.

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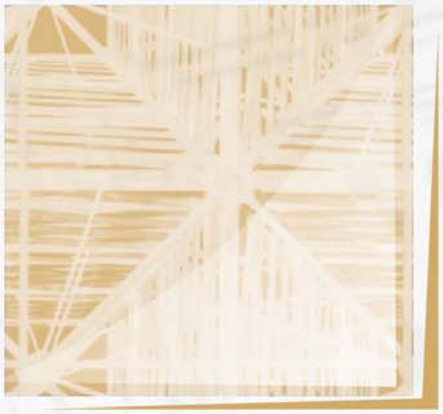

Part III

Etiopathogenesis

6 Chapter

Periodontal Microbiology (Dental Plaque)

- DEFINITION
- DENTAL PLAQUE AS A BIOFILM
- TYPES OF DENTAL PLAQUE
- COMPOSITION OF DENTAL PLAQUE
- FORMATION/DEVELOPMENT OF DENTAL PLAQUE
- STRUCTURAL AND MICROSCOPIC PROPERTIES OF PLAQUE
- CLINICAL SIGNIFICANCE OF PLAQUE
- MICROBIAL SPECIFICITY OF PERIODONTAL DISEASES
- WHAT MAKES PLAQUE PATHOGENIC
- MICROORGANISMS ASSOCIATED WITH PERIODONTAL DISEASES
- BACTERIA ASSOCIATED WITH PERIODONTAL HEALTH AND DISEASE

DEFINITION

Dental Plaque

Dental Plaque is an adherent intercellular matrix consisting primarily of proliferating microorganisms, along with a scattering of epithelial cells, leukocytes and macrophages.

Plaque

Plaque can also be defined as the soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed restorations. Dental Plaque is a host-associated biofilm.

Biofilms

Biofilms are defined as "Matrix—enclosed bacterial populations adherent to each other and or/to surface or interfaces (by Costerton). According to the recent data (Widerer and Charaklis 1989), biofilm is defined as the relatively undefinable microbial community associated with a tooth surface or any other hard, non-shedding material.

DENTAL PLAQUE AS A BIOFILM

Structurally, dental plaque is now considered to be a biofilm of complex and dynamic microbial community. It contains areas of high and low bacterial biomass interlaced with

aqueous channels of different sizes, which are the nutrient channels for bacterial colonization. The intercellular matrix forms a hydrated gel in which bacteria can survive and proliferate. Hence, biofilm adheres firmly to the tooth surface and is resistant to mechanical removal, as well as antibiotics.

TYPES OF DENTAL PLAQUE

Based on its relationship to the gingival margin, plaque is differentiated into two categories, supragingival and subgingival plaque (Table 6.1).

Supragingival plaque is further differentiated into:

Coronal plaque, which is in contact with only the tooth surface, and Marginal plaque, which is associated with the tooth surface at the gingival margin.

Subgingival plaque can be further differentiated into:

- Attached plaque
- Unattached subgingival plaque
- Attached plaque can be tooth, epithelium and/or connective tissue associated.

Supragingival Plaque (Fig. 6.1)

It can be detected clinically only after it has reached a certain thickness. Small amounts of plaque can be visualized by using disclosing agents. The color varies from grey to yellowish-grey to yellow. The rate of formation and location of plaque vary among individuals and is influenced by diet, age, salivary factors, oral hygiene, tooth alignment, systemic diseases and host factors.

Subgingival Plaque (Figs 6.2 and 6.3)

It is usually thin, contained within the gingival sulci or periodontal pocket and thus cannot be detected by direct observation. Its presence can be identified only by running the end of a probe around gingival margin (Table 6.2).

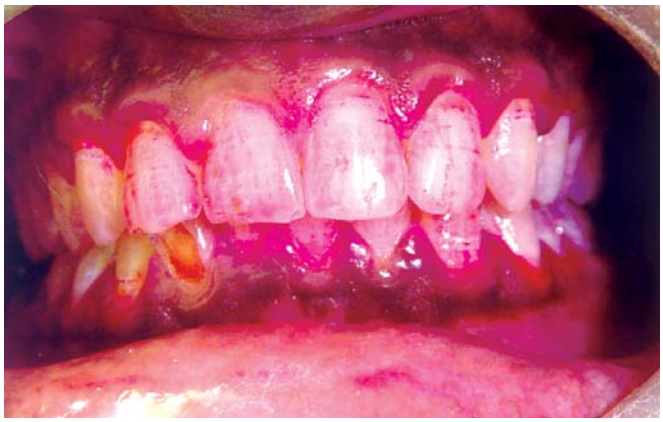

Fig. 6.1: Supragingival plaque

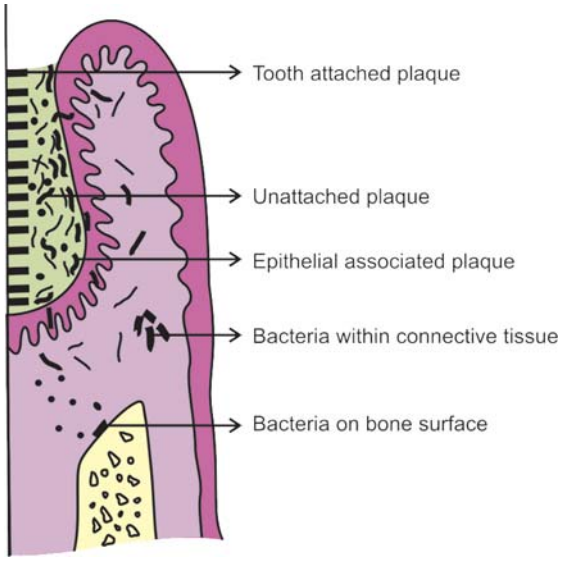

Fig. 6.2: Subgingival plaque bacteria associated with tooth surface and periodontal tissues

Tooth-associated Subgingival Plaque

The structure is similar to the supragingival plaque. The flora is dominated by Gram-positive cocci, rods, filamentous bacteria and some/few Gram-negative cocci and rods. This flora is associated with calculus formation, root caries and root resorption.

Table 6.1: Differences between supragingival and subgingival plaque

	Supragingival plaque	*Subgingival plaque*
1. Matrix	50% Matrix	Little or no matrix
2. Flora	Mostly Gram-positive	Mostly Gram-negative
3. Motile bacteria	Few	Common
4. Anaerobic/Aerobic	Aerobic unless thick	Highly anaerobic areas present
5. Metabolism	Predominantly carbohydrates	Predominantly proteins

Table 6.2: Characteristics of subgingival plaque

Sl. No.	*Tooth-associated subgingival plaque*	*Epithelium-associated plaque*
1.	Gram-positive bacteria predominates	Gram- positive and Gram-negative bacteria.
2.	Does not extend to junctional epithelium	Extends to junctional epithelium.
3.	May penetrate cementum	May penetrate epithelium and connective tissue.
4.	Associated with calculus formation and root caries	Associated with gingivitis and periodontitis.

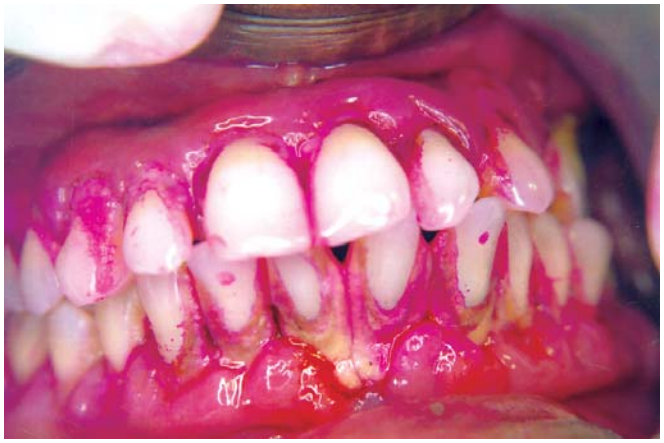

Fig. 6.3: Subgingival plaque and calculus

Epithelium-associated Subgingival Plaque

This type of plaque is loosely adherent because it lacks the interbacterial matrix and is in direct association with the gingival epithelium, extending from the gingival margin to the junctional epithelium. This plaque predominantly contains Gram-negative rods and cocci, as well as a large number of flagellated bacteria and *Spirochetes.*

Connective Tissue-associated Plaque

It is usually demonstrated in ANUG and localized aggressive periodontitis patients. The clinical significance is unclear. The unattached plaque can be seen anywhere. Thus, the tooth-associated subgingival plaque is most important in calculus formation, root caries and slowly progressive periodontal destruction, whereas unattached bacterial component is associated with rapid periodontal destruction.

COMPOSITION OF DENTAL PLAQUE

Bacteria + Intercellular matrix = Dental plaque

Bacteria make up approximately 70 to 80 percent of total material. One mg of dental plaque is estimated to contain 250 million bacteria. Other than bacteria, mycoplasma, fungi, protozoa and viruses may be present. The material among the bacteria in dental plaque is termed as inter-microbial/cellular matrix. It contains organic and inorganic portions. The organic matrix is composed of protein—polysaccharide complex produced by microorganisms. Carbohydrates in the form of levans (fructans) provides mainly energy while glucans (dextran) provide not only energy, but also act as the organic skeleton of plaque. Other carbohydrates are galactose and rhamnose. Glycoproteins provide the protein component and small amounts of lipids are also present. Inorganic components include, primarily calcium, phosphorus with small amounts of magnesium, potassium and sodium.

FORMATION/DEVELOPMENT OF DENTAL PLAQUE

Pellicle is the initial organic structure that forms on the surfaces of the teeth and artificial prosthesis. The first stage in pellicle formation involves adsorption of salivary proteins to apatite surfaces. This results from the electrostatic ionic interaction between hydroxyapatite surface which has negatively charged phosphate groups that interacts with opposite charged groups in the salivary macromolecules. The mean pellicle thickness varies from 100 nm at 2 hours to 500 to 1,000 nm.

The transition from pellicle to dental plaque is extremely rapid. The first components include mainly cocci with small number of epithelial cells and PMNL's, they form a monolayer within a few hours, and the attached bacteria proliferate and form small colonies of cocci. With time other types of microorganisms proliferate and form different microcolonies.

Hence, in dental plaque development, two adhesion processes are required. First, bacteria must adhere to the

pellicle surface and become sufficiently attached to withstand oral cleansing forces. Second, they must grow and adhere to each other to allow plaque accumulation.

Bacterial Adherence

During initial adherence, interactions occur mainly between specific bacteria and the pellicle. They are:

Bacterial Attachment via Electrostatic Interactions

Oral bacteria bear an overall net negative charge, negatively-charged components of the bacterial surface and negatively-charged components of pellicle become linked by cations such as calcium (Fig. 6.4).

Bacterial Attachment via Hydrophobic Interactions

These interactions are based on the close structural fit between molecules on the pellicle and bacterial surfaces. The nature of the hydrophobicity of the cell is not clearly known. The contributing factor might be lipoteichoic acid (LTA), which may provide a long hydrophobic area (Fig. 6.5).

Bacterial Attachment via Specific Lectin-like Substances (Fig. 6.6)

Lectins in the bacterial surfaces recognize specific carbohydrate structure in the pellicle and become linked.

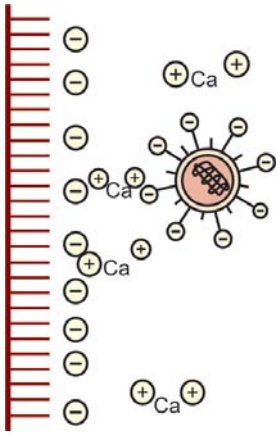

Fig. 6.4: Bacterial attachment via electrostatic interactions

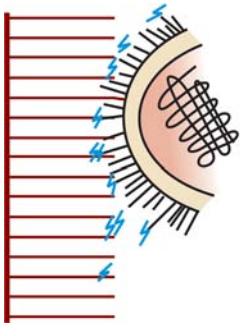

Fig. 6.5: Bacterial attachment via hydrophobic interactions

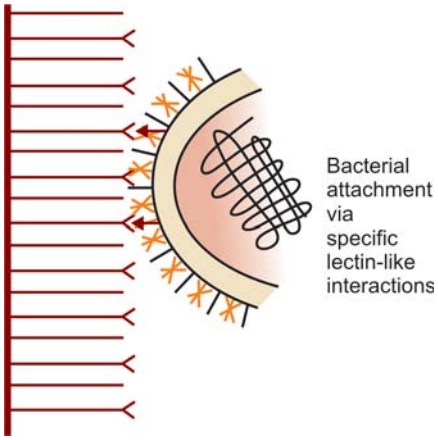

Fig. 6.6: Bacterial attachment via specific lectin-like interactions

Adhesion and attachment (Fig. 6.7) occurs between:

- Bacteria and clean tooth surface
- Bacteria and pellicle
- Bacteria and same species
- Bacteria and different species
- Bacteria and matrix.

Next step in plaque formation is:

Growth and Accumulation of Bacteria

Once the bacteria is adhered to the pellicle, subsequent growth leads to bacterial accumulation and increased plaque mass. Dental plaque growth (Fig. 6.8) depends on:

a. Growth via adhesion of new bacteria
b. Growth via multiplication of attached bacteria.

The initial bacteria that colonize the pellicle surface are mostly gram-positive facultative microorganisms such as

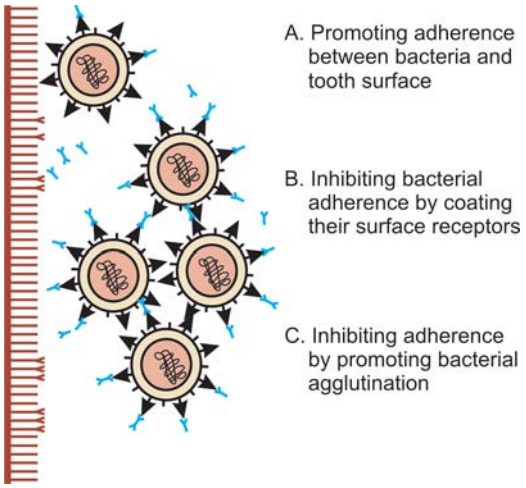

Fig. 6.7: Role of saliva in the development and maturation of dental plaque

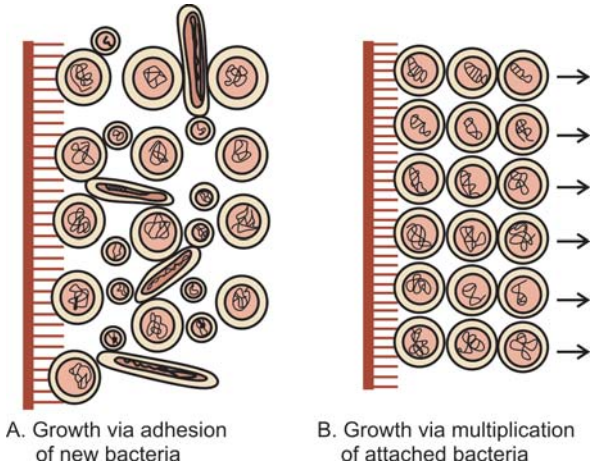

Fig. 6.8: Growth of dental plaque

Actinomyces viscosus and Streptococcus sanguis, as the plaque matures, secondary colonization *of Prevotella intermedia, Capnocytophaga, Porphyromonas gingivalis* takes place. This ability of bacteria to adhere to different species and genera of microorganisms is known as *co-aggregation* (Fig. 6.9).

STRUCTURAL AND MICROSCOPIC PROPERTIES OF PLAQUE

Supragingival Plaque

It is usually adherent to the tooth surface. It contains gram-positive cocci and gram-negative rods and filaments. The

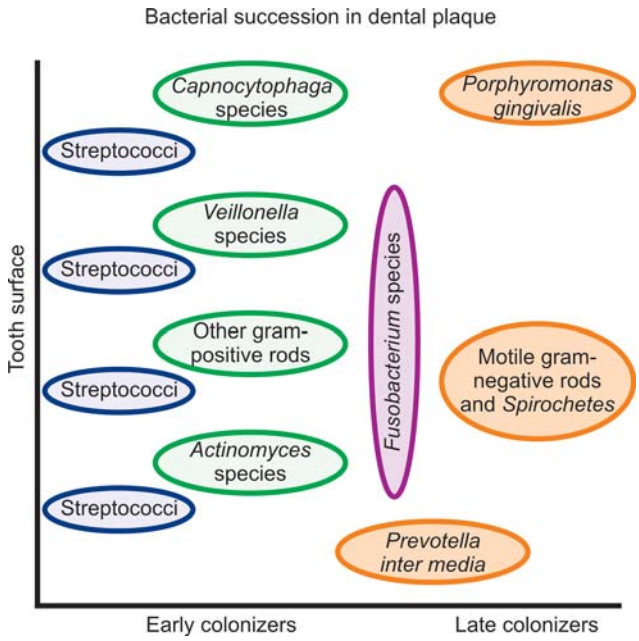

Fig. 6.9: Bacterial succession in dental plaque

morphologic arrangement of the flora in supragingival plaque described as *"corncob" formations*, characterized by central core consisting of rod-shaped bacterial cells, e.g. *Fusobacterium nucleatum* and coccal cells, e.g. streptococci which attaches along the surface of the rod-shaped cell.

The subgingival plaque differs from supragingival plaque, in that it contains many large filaments with flagella and is rich in *Spirochetes*. Tooth-associated plaque is similar to supragingival plaque; whereas tissue-associated plaque is covered with flagellated bacteria without a well-defined extracellular matrix and numerous bristle—brush formations. This arrangement is also called as "*test tube-brush*" formation characterized by large filaments that forms the long axis; and short filaments or gram-negative rods embedded in a amorphous matrix (Fig. 6.10).

CLINICAL SIGNIFICANCE OF PLAQUE

The microbial aggregations on the tooth surface if prevented from maturing may become compatible with gingival health. Supragingival plaque if allowed to grow and mature, may induce gingivitis and can lead to the formation of a microenvironment that permits the development of subgingival plaque. Therefore, supragingival plaque

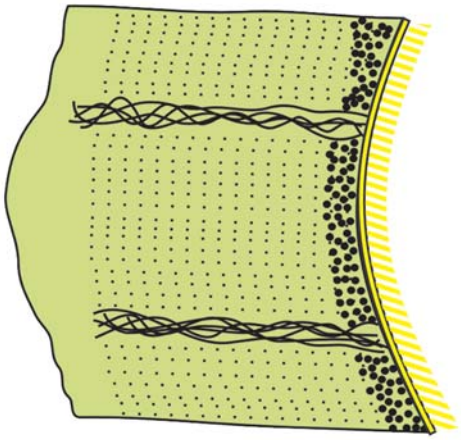

Fig. 6.10: Mature plaque diagram

strongly influences the growth, accumulation and pathologic potential of subgingival plaque, especially in the early stages of gingivitis and periodontitis.

Subgingival Plaque

In association with the presence of supragingival plaque, there are inflammatory changes that modify the anatomic relationships of the gingival margin and tooth surface. This result in enlarged gingiva, which increases the space for bacterial colonization and also protects bacteria from normal cleansing mechanisms. They derive nutrients from gingival crevicular fluid. Many of these microorganisms lack the adherence ability and utilizes supragingival plaque bacteria as a means of colonization of the subgingival area.

Electron microscopic studies have demonstrated the existence of an organic material called cuticle between the root surface and subgingival plaque. It is covered by a dense layer of microorganisms and is believed to be a remnant or secretory product of the junctional epithelial cells.

MICROBIAL SPECIFICITY OF PERIODONTAL DISEASES

Walter Loesche proposed the nonspecific and specific plaque hypothesis in 1976. The nonspecific plaque hypothesis states that it is the total bulk of plaque, which determines the pathogenicity rather than the individual species within it. In other words, all plaque is equally pathogenic. According to this, when only small amounts of plaque are present, the products released by this gets neutralized by the host. Similarly, large amounts of plaque would produce large amounts of noxious products, which would overwhelm the host's defenses. However, several authors have contradicted this concept. First, many patients have considerable amounts of plaque and calculus as well as gingivitis, but only a minority suffer from destructive periodontal disease even then in only few sites. This paradox might be explained by specific plaque hypothesis, which states that destructive periodontal disease is a result of specific microbial pathogens in plaque. Thus, although the amount of plaque present correlates well with disease severity, it correlates poorly in individual patients.

But it is the nonspecific plaque hypothesis, which forms the basis for virtually all the current modalities for treatment, and prevention, which relies on the principle of reducing plaque scores to a minimum. Thus, although the nonspecific plaque hypothesis has been discarded in favor of the specific plaque hypothesis, much clinical treatment is still based on the nonspecific plaque hypothesis.

Specific Plaque Hypothesis

It states that, not all plaque is pathogenic and its pathogenicity depends on the presence of certain specific microbial pathogens in plaque. This is based on the fact that, the specific microorganisms responsible for periodontal diseases release certain damaging factors that mediates the destruction of the host tissue. This concept was accepted easily due to the recognition of *Actinobacillus actinomycetemcomitans* as a possible pathogen responsible for localized juvenile periodontitis.

WHAT MAKES PLAQUE PATHOGENIC?

The following are the possible pathogenic mechanisms by which the plaque microorganisms can cause periodontal disease.

a. Physical nature of plaque.
b. Invasion of tissues by bacteria.
c. Release of toxic and inflammatory substances.
d. Role of bacterial specificity.

MICROORGANISMS ASSOCIATED WITH PERIODONTAL DISEASES

It is well accepted that a plaque bacteria is the primary etiologic agent in periodontal disease. Bacteria are seen in the oral cavity from birth to death. It is estimated that about 400 different species are capable of colonizing in the mouth. Counts in subgingival sites range from about 10^3 in healthy sulci to greater than 10^8 in deep periodontal pockets. It has not been possible to identify and study all the organisms present in the bacterial plaque, of nearly 400 species; only 30 of them are considered to be periodontopathic. Koch's postulates, generally used to identify the periodonto-pathogenecity of a microorganism, are not applicable in periodontal disease, as more than one organism is involved in periodontal diseases. Hence, Socransky (1977) had proposed the following criteria for identifying the possible causative organisms in periodontal diseases:

1. The number of etiologic organisms in the diseased site must be increased and conversely the number of organisms must be reduced or absent in healthy sites.
2. If the etiologic organism is eliminated or suppressed, the disease should stop.
3. Presence of specific antibodies to those microorganisms.
4. Presence of virulence factors associated with certain microorganisms (e.g. toxins, enzymes, etc.).
5. *In vitro* or animal experiments should be able to demonstrate the human disease process.

There are many speculations regarding the pathogens responsible for periodontitis whether they could be exogenous or components of indigenous flora. To explain this controversy, two theories have been proposed.

According to the first theory, periodontopathic organisms are a part of indigenous flora and they tend to overgrow during the disease progression. The second proposal is that, they are not components of indigenous oral flora, but are exogenous pathogens derived from outside sources. This concept hints that the quantity of plaque is not necessary for disease onset. Instead, the site should be contaminated with specific periodontopathogens.

Recent reports have demonstrated a possibility of viral etiology in periodontitis and implicated viruses are Epstein Barr virus, human cytomegalovirus and mixed herpes viral infections. Viral infection can contribute to periodontitis by altering the functions of neutrophils, macrophages and lymphocytes which in turn promotes the overgrowth of periodontopathic organisms in the subgingival flora. The other possibility is that, the viral infection can destroy the oral epithelial cells thus disrupting the barrier function of the periodontium.

Bacteria Associated with Periodontal Health and Disease

See Table 6.3.

Table 6.3: Reclassification of periodontal bacteria

Previous status	*Current status*
Wolinella recta	*Campylobacter rectus (C. rectus)*
Bacteroides gingivalis	*Porphyromonas gingivalis (P. gingivalis)*
Bacteroides intermedius	*Prevotella intermedia (P. intermedia)*
Bacteroides melaninogenicus	*Prevotella melaninogenica (P. melaninogenica)*
Bacteroides forsythus	*Tannerella forsythus*
Actinobacillus actinomycetemcomitans	*Aggregatibacter actinomycetemcomitans*

Health

- *Actinomyces* (*viscosus and naeslundii*)
- *Streptococcus* (*S. mitis and S. sangius*)
- *Veillonella parvula*, small amounts of gram-negative species are also found.

Chronic Gingivitis

Gram-positive (56%), Gram-negative (44%) organisms are found. Predominant gram-positive species include, *S. sangius, S. mitis, S. oralis, A. viscosus, A. naeslundii, Peptostreptococcus micros.*

Gram-negative Organisms

- *Fusobacterium nulceatum*
- *Prevotella intermedia*
- *Veillonella parvula* as well as *Haemophilus, Capnocytophaga* and *Campylobacter* species.

Pregnancy-associated Gingivitis

- *Prevotella intermedia.*

Acute Necrotizing Ulcerative Gingivitis

- *Spirochetes*
- *Prevotella intermedia.*

Adult Periodontitis

- *Porphyromonas gingivalis*
- *Bacteroides (Tannerella) forsythus*
- *Prevotella intermedia*
- *Campylobacter rectus*
- *Eikenella corrodens*
- *Fusobacterium nucleatum*
- *Actinobacillus actinomycetemcomitans*
- *Peptostreptococcus micros*
- *Treponema,* and
- *Eubacterium* species.

Viruses

- EBV-1 (Ebstein-Barr virus)
- HCMV (Human cytomegalovirus).

Localized Juvenile Periodontitis

- *Actinobacillus (Aggregatibacter) actinomycetem-comitans*
- *Porphyromonas gingivalis*
- *Eikenella corrodens*
- *Campylobacter rectus*
- *Fusobacterium nucleatum*
- *Bacteroides capillus*
- *Eubacterium brachy*
- *Capnocytophaga*
- *Herpes* virus.

Generalized Juvenile Periodontitis

- *Actinobacillus (Aggregatibacter) actinomycetem-comitans*
- *Porphyromonas gingivalis*
- *Prevotella intermedia*
- *Capnocytophaga*
- *Eikenella corrodens*
- *Neisseria.*

Refractory Periodontitis

- *Actinobacillus actinomycetemcomitans*
- *Bacteroides forsythus*
- *Porphyromonas gingivalis*
- *Prevotella intermedia*
- *Wolinella recta.*

Abscesses of the Periodontium

- *Fusobacterium nucleatum*
- *Prevotella intermedia*
- *Peptostreptococcus micros*
- *Bacteroides forsythus*
- *Porphyromonas gingivalis.*

KEYPOINTS

1. Dental plaque is defined as an adherent intercellular matrix, composed primarily of proliferating micro-organisms, along with a scattering of epithelial cells, leukocytes and macrophages.
2. Structurally dental plaque is now considered to be a biofilm of complex and dynamic microbial community.
3. Based on its relationship to the gingival margin, plaque is differentiated into two categories, supragingival and subgingival plaque.
4. Subgingival plaque can be, tooth-associated, epithelium-associated, connective tissue-associated and unattached plaque.
5. Dental plaque is mainly composed of bacteria and intercellular matrix.
6. In the formation of dental plaque first step is pellicle formation followed by bacterial adherence and growth and accumulation of bacteria.

KNOW MORE ...

Types of Infection

Several types of infections can be distinguished

a. *Endogenous infections* with bacteria that belong to the resident flora, e.g. skin, nose, oral cavity, intestinal and urinary tract.
Note: Members of the resident flora at one site may cause life-threatening infections in other organ system.

b. *Opportunistic infection* in a systemically or locally compromised host, the opportunistic organisms which are usually avirulent can become virulent.
c. *Exogenous infection* with microorganisms that are usually not members of the resident flora, e.g. among periodontopathogens, in particular some virulent clones of *A. actinomycetemcomitans* and *P. gingivalis* might be considered exogenous pathogens. However, it should be noted that bacteria alone cannot induce destructive periodontal disease.

Microbial Shifts from Health to Disease

- Gram-positive to gram-negative microorganisms
- Cocci to rods (and at later stage to spirochetes)
- Nonmotile organisms to motile organisms
- Facultative anaerobes to obligate anaerobes
- From fermenting to proteolytic species.

Principles of Intraoral Translocation, Transmission or Cross-infection of Bacteria

It has been substantiated that bacterial pathogens are transmissible within family members. This transmission differs from contagious disease which refers to the likelihood of a microorganism to cause disease in an uninfected host once transmitted from an infected host. This is supported by the finding of transmission of cariogenic microorganisms from mother to child.

On the basis of physical and morphological criteria, oral cavity has been divided into five major ecosystems (also called niches):

1. Intraoral, supragingival hard surfaces (teeth, implants, restorations and prosthesis).
2. Periodontal/peri-implant pocket (with its crevicular fluid, the root cementum or implant surface and pocket epithelium).
3. Buccal epithelium, palatal epithelium and epithelium of floor of the mouth.
4. Dorsum of the tongue.
5. Tonsils.

It has been seen that intraoral transmission (also called translocation or cross-infection) of bacteria occurs from one intraoral niche to another. It occurs through intraoral fluid and transport media (saliva) or transmitted via dental explorer during examination.

Certain examples have been cited to prove the transmission of periodontal and cariogenic pathogens within the oral cavity. Notable amongst these are:

a. Spread of *Streptococcus mutans* which was grown on dental implant to neighboring teeth.
b. Spread of *A. actinomycetemcomitans* via periodontal probes in patients with localized aggressive periodontitis.

The significance of this transmission or translocation of bacteria lies in reinfection of previously treated sites by bacteria from untreated sites or other intraoral niches. Hence, to prevent this a new treatment strategy called *one-stage, full mouth disinfection* was introduced by Leuven and group. This strategy consists of combination of following treatment approaches.

a. Full mouth scaling and root planing within 24 hours.
b. Subgingival irrigation of all periodontal pockets with 1 percent chlorhexidine gel.
c. Tongue brushing with antiseptic.
d. Mouthrinsing with an antiseptic to reduce bacteria in other intraoral niches.

Benefits of one-stage, full mouth disinfection over conventional quadrant-wise approach has better results in clinical attachment, pocket depth reduction and microbiological shifts.

Periodontal Vaccines

Periodontal disease can be considered as one of the most prevalent of the polymicrobial, chronic inflammatory diseases affecting humans. Despite intensive research in its treatment modalities, none have been completely successful in regenerating the lost hard and soft tissues. The advent of advanced molecular diagnostic techniques and a better understanding of the role of specific pathogens and the contributory role of the host immune response in the initiation and progression of periodontal disease have led to the development of periodontal vaccines. However, successful vaccine development that fully utilizes the current level of understanding has not yet occurred for human use.

Various forms of active and passive immunization methods have been tried. Although, most of the studies have yielded encouraging results none of these modalities of immunization have been able to be incorporated as a sole or complete 'vaccine' against periodontal disease for use in the human population. Thus, the current status of our understanding in the field of vaccines against periodontal disease is incomplete.

Key characteristics of specific periodontal pathogens

Porphyromonas gingivalis

Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative, anaerobic, nonmotile.	Coccal to short rod morphology. Possess fimbriae that facilitate adhesion and coaggregation.	6 forms have been identified based on capsule types.	Grows anaerobically with dark pigmentation on blood agar.	**Proteases [Gingipains]** types- rgpA, rgpB, kgp.	Destruction of immunoglobulins, complement factors Causes degradation of type I and IV collagen.
				Prolyl tripeptidyl peptidase- membrane enzyme	Causes degradation of host collagenase inhibitors.
				Periodontain	Acts on degraded proteins.
				Hemolysin	Causes agglutination and lysis of erythrocytes.
				Lipopolysaccharide	Degrades fibrinogen rapidly rendering host in a non clotting condition. Potent stimulator of osteoblasts and monocytes to release IL-1, PGE_2 and TNF-α.

Treponema denticola

Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative anaerobic highly motile microorganisms	Helical shaped, 5-15 μm in length and 0.5 μm in diameter. Irregular spirals. with peri-plasmic flagella.	*T. denticola, T. amylovorum T. maltophilum, T. medium, T. pectinovorum, T. socranskii, T. vincentii.*	Cultivated in broth media enriched with serum, trypticase, various fatty acids and growth factors. Addition of lecithin, ascorbic acid, and ammonium is required for optimum growth.	**Major surface proteins (Msp)**	• Causes metabolic inhibition of host cells-fibroblasts • Cell detachment, loss of cellular volume regulation, cytoskeletal disruption in epithelial cells
				Chymotrypsin like proteases (CTLP)	• Degradation of basement membrane; serum proteins, immunoglobulins • Activation of MMPs
				Others include: Outer sheath associated peptidases, trypsin like proteases	• Known to inhibit superoxide production in PMNs. • Also induces release of MMPs.
				Lipopolysaccharides	Causes stimulation of production of nitric oxide, tumor necrosis factor-α and interleukin- 1.
				Cystalysin	Degrades sulfur containing substances
				Hemolysins	Causes agglutination and lysis of erythrocytes

Contd...

Contd...

Bacteroides forsythus

Also known as *Tannerella forsythus*

Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative, anaerobic micro organisms.	Highly pleomorphic microorganism. • Exhibits spindle shaped or filament shaped morphology.	Various strains have been described on the basis of DNA component. Of which *T. forsythus* D-10 is most commonly found in supragingival and subgingival plaque samples.	Difficult to grow. Requires *atleast* 14 days to grow. Requires N-acetylmuramic acid for growth.	**Trypsin like proteases**	Degrades benzoyl-DL arginine naphthylamide. Also induces apoptotic cell death.
				Sialidases	Sialidases are known to degrade host glycoproteins and glycolipids.
				N- acetyl-β-glucosaminidase.	N-acetyl-β-glucosaminidase causes undermining of basement membrane in periodontal pockets.
				Cell envelope lipoprotein	Cell envelope lipoprotein stimulates gingival fibroblasts to produce IL-6 and TNF-α, and also nuclear factor κβ.

Actinobacillus actinomycetemcomitans

Also known as *Aggregatibacter actinomycetemcomitans*

Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative Capnophilic Nonmotile Saccharolyticus Occur singly, in clumps or pairs	Small, short (.4-1 μ) straight or curved rod morphology. Star shaped colonies-called Actinobacillus Fimbriae-peritrichous.	Serotypes based on heat stable surface antigen '*a* to *e*'. *a* and *b* most common, *c* is 10%, *in LJP 'b'* elevated.	Grows in serum or blood agar in anaerobic environment. Translucent colonies with star shaped internal morphology.	**Leukotoxins**	The pore forming property of leukotoxin leads to pathologic alterations in cell membrane of target cells.
				Immunosuppressive factors or cytolethal distending toxins	Heat labile toxin causing eukaryotic cell distension, cell cycle arrest, actin filament rearrangement and apoptosis. Also effects blastogenesis, antibody production, cytokine synthesis, neutrophil function, chemotaxis and phagocytosis.
				LPS	Causes bone resorption, platelet aggregation and necrosis of tissues. Stimulates production of interleukins and TNF.
				Collagenase	Destruction of collagen.
				Protease	Cleavage of immunoglobulin- IgG.

Contd...

Contd...

Prevotella intermedia					
Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative, anaerobic	Short rods with rounded ends. Possess fimbriae.	VPI 8944 like strains are related to gingivitis. VPI 4197 like strains (which include ATCC 25611) are related to periodontitis	Produces black pigmented colonies on blood agar.	**Hydrolases**	Degradation of immunoglobulins, heme sequestering proteins.
				Hemolysin	Lysis of RBCs
				LPS	Release of IL-1, IL-6, IL-8
				Interpain A	Inhibits complement by degradation of C3 which is common to all pathways.
				Cysteine protease	Degradation of hemoglobin.
Fusobacterium nucleatum					
Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-negative, anaerobic, nonmotile	Slender spindle shaped with tapered ends. Often show intracellular granules.	*F. fusiforme* *F. vincentii* *F. periodonticum*	Produces dark colonies on blood agar containing trypticase, peptone and serum.	**Toxic metabolites**	Induces apoptotic cell death. Also causes release of cytokines, elastase and oxygen radicals from macrophages.
Peptostreptococcus micros					
Bacterial properties	*Morphology*	*Strains/ Species*	*Culture and identification*	*Virulence factors*	*Actions*
Gram-positive, anaerobic bacteria. *Asaccharolyticus*, microaerophilic.	Oval to rod shaped morphology.	2 morphotypes have been described- rough and smooth.	Grown on agar enriched with horse or sheep blood.	**Cell wall**	Induces release of cytokines- TNF-α, IL-1β, IL-6, IL-8.

TNF–Tumor necrosis factor, LPS–Lipopolysaccharide, IL–Interleukins, MMP–Matrix Metalloproteinases

REVIEW QUESTIONS

1. Define plaque and describe the steps in formation of plaque.
2. What are the types and composition of dental plaque?
3. What is specific and non-specific plaque hypothesis?
4. Describe the role of plaque in periodontal disease.
5. What is the clinical significance of plaque?
6. What are differences between supra and subgingival plaque?

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7 Chapter

Calculus and other Etiological Factors

- CALCULUS
  - Definition
  - Types
  - Structure
  - Composition
  - Differences between Supra- and Subgingival Calculus
- OTHER CONTRIBUTING ETIOLOGICAL FACTORS INCLUDING FOOD IMPACTION

CALCULUS

Definition

Dental calculus is an adherent, calcified or calcifying mass that forms on the surfaces of teeth and dental appliances. It is covered on its external surface by vital, tightly adherent, nonmineralized plaque.

Types

Depending upon the position of calculus in relation to the marginal gingiva it is classified as:

1. Supragingival calculus
2. Subgingival calculus

Supragingival Calculus

It is the tightly adherent calcified deposit that forms on the clinical crowns of the teeth above the free gingival margin. Hence, it is clinically visible. It is also called as salivary calculus because it forms from the saliva.

Subgingival Calculus

As the name implies, it is that calcified deposits that is formed on the root surfaces below the free marginal gingiva. It is believed to be formed from the gingival exudate and hence called serumal calculus (Figs 7.1 and 7.2).

Structure

The deposits of supragingival calculus are usually whitish-yellow in color and can get stained by tobacco or food pigments, consistency is hard and clay-like. Since they derive the mineral salts from salivary secretions, they are most abundant on the lingual surfaces of lower anterior teeth, opposite Wharton's duct and Bartholin's duct and buccal aspects of maxillary molars opposite the Stenson's duct (Fig. 7.3).

Subgingival calculus is usually dark-brown or greenish-black in color and the deposits are firmly attached to the tooth surface. Since they are hard and firm, it cannot be removed easily. Unlike supragingival calculus, subgingival

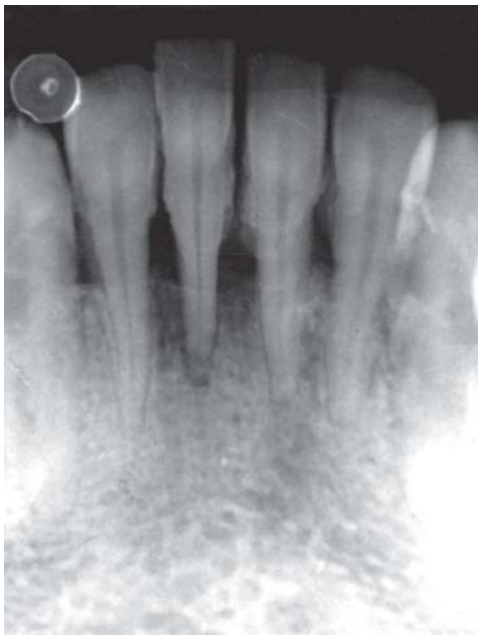


Fig. 7.1: Radiograph illustrating subgingival deposits in lower anterior and posterior teeth

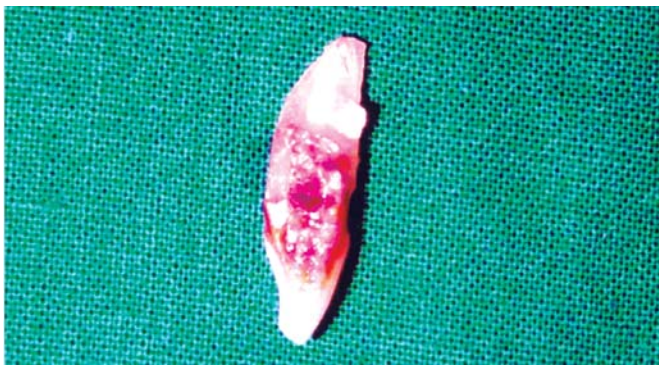


Fig. 7.2: Subgingival deposits on the root surface of extracted lower anterior tooth

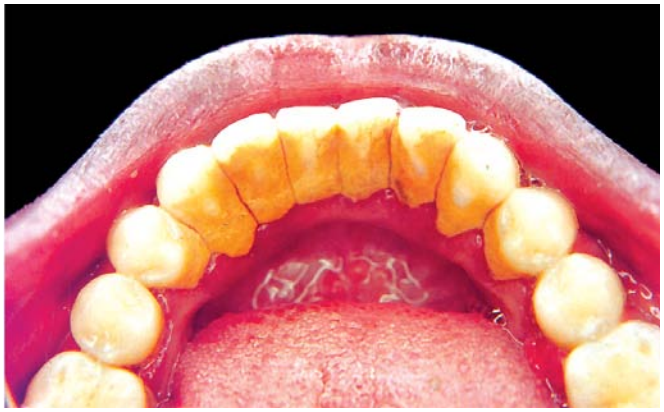


Fig. 7.3: Supragingival calculus on the lingual surfaces of lower anteriors

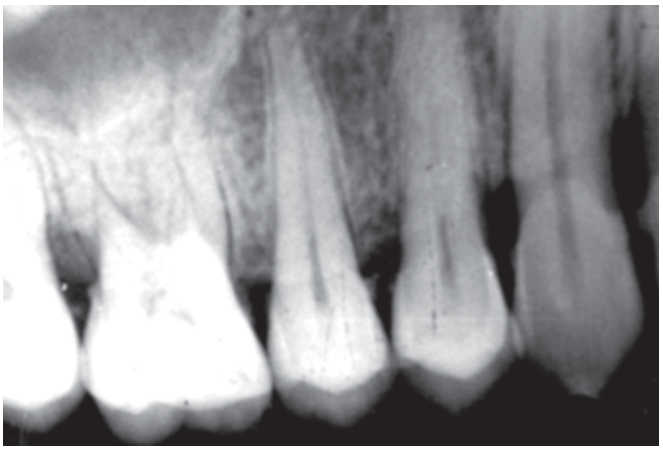


Fig. 7.4: Radiograph illustrating subgingival calculus in the interproximal areas

calculus can be found on any root surface with a periodontal pocket. Morphologically, it can appear in different forms, most commonly ring-like or ledge-like formations crusty, spiny or nodular deposits. Less frequently it can be seen as finger-like and fern-like formations (Fig. 7.4).

Composition

It consists of inorganic and organic components (Tables 7.1 and 7.2).

Trace amounts of zinc, strontium, bromine, copper, manganese, gold and aluminium are also seen. At least, two-thirds of inorganic component is crystalline in structure. The main crystal forms are: Hydroxyapatite, Magnesium whitlockite, Octacalcium phosphate and Brushite.

Table 7.1: Inorganic components

Component	*Dry weight (in percent)*
Inorganic	70-90
Calcium	27-29
Phosphorus	16-18
Carbonate	2-3
Sodium	1.5-2.5
Magnesium	0.6-0.8
Fluoride	0.003-0.04
Crystal forms	
Hydroxyapatite	58
Magnesium whitlockite	21
Octacalcium phosphate	12
Brushite	9

Table 7.2: Organic components	
Component	*Dry weight (in percent)*
Mixture of protein, polysaccharide complexes, desquamated epithelial cells, leukocytes and various microorganisms. Carbohydrate (consists of glucose, galactose, rhamnose, mannose)	1.9-9.1
Proteins	5.9-8.2
Lipids	0.2

Differences between Supragingival and Subgingival Calculus

See Table 7.3.

Attachment to the Tooth Surface

Four types of attachment of calculus to tooth surface have been reported.

1. Attachment by means of an organic pellicle.
2. Mechanical interlocking into surface irregularities such as resorption lacunae and caries.
3. Penetration of calculus bacteria into cementum.
4. Close adaptation of calculus under surface depressions to the gently sloping mounds of the unaltered cementum surface.

Calculus when embedded deeply in cementum may appear similar in morphology and thus has been termed as calculocementum.

Table 7.3: Differences between supra- and subgingival calculus	
Supragingival	*Subgingival*
Location—above the gingival margin	Deposits present below the margins of the gingiva
Color—white, yellow in color	Brown or greenish-black
Source—derived from salivary secretions	Formed from gingival exudate
Composition—more brushite and octacalcium phosphate less magnesium whitlockite	Conversely less brushite and octacalcium phosphate and more magnesium whitlockite
Salivary proteins are present	They are absent
Sodium content is lesser	Sodium content increases with the depth of the pocket

Formation of Calculus

Calculus is nothing but, dental plaque that has undergone mineralization. Calculus is formed by the precipitation of mineral salts, which can start between 1st and 14th day of plaque formation. In two days plaque can be 50 percent mineralized and 60 to 90 percent gets mineralized in 12 days. Calcification starts in separate foci on the inner surface of the plaque. These foci of mineralization gradually increase in size and coalesce to form a solid mass of calculus.

Calculus formation continues until it reaches maximum levels in about 10 weeks and 6 months, after which there is a decline in its formation, due to mechanical wear from food and from the lips, cheeks and tongue. This decline is referred to as reversal phenomenon.

Theories of Calculus Formation

It can be explained mainly under two categories:

1. Precipitation of minerals can occur from a local rise in the degree of saturation of calcium and phosphate ions, this is explained in,
   a. *Booster mechanism:* According to this theory, precipitation of calcium phosphate salts results from a local rise in the pH of the saliva. Factors such as loss of carbon dioxide and production of ammonia could lead to rise in pH.

      Other ways by which the precipitation of calcium phosphate salts can occur are:
   b. *Colloidal proteins in saliva* bind to calcium and phosphate ions thus producing a supersaturated solution. When saliva stagnates in the oral cavity, colloids settle and result in the precipitation of calcium and phosphorous salts.
   c. *Phosphatase* liberated from dental plaque, desquamated epithelial cells, or bacteria precipitate calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions.
2. Another concept that has been most widely held is "Epitactic Concept" (heterogenous nucleation). According to this, seeding agents induce small foci of calcification. These foci enlarge and coalesce to form

calculus. Hence, more appropriately called as heterogenous nucleation. The seeding agents in calculus is not clearly known, but suspected agents could be, intercellular matrix of plaque, carbohydrate protein complexes and plaque bacteria.

3. *Inhibition theory:* This theory considers the possibility of calcification occurring only at specific sites because, there exists an inhibiting mechanism at non-calcifying sites. Wherever calcification occurs, the inhibitor is either altered or removed. One such inhibiting agents could be pyrophosphate which prevents the initial nucleus from growing, by possibly 'poisoning' the growth centers of the crystal.

Pathogenic Potential of Calculus in Periodontal Diseases

Before 1960s the belief was that calculus was the principle etiologic factor in periodontal diseases. However, the current view is that the initial damage to the gingival margin in the periodontal disease is due to the pathogenic effects of microorganisms in plaque. However, the effect could get more pronounced by calculus accumulation because it further provides retention of more plaque microorganisms.

Hence, there is no doubt that, the mineralized deposits can—

a. Bring the bacterial deposits more closely to the supporting structures.
b. Interfere with the local self-cleansing defense mechanisms.
c. And also enable the patients to perform proper oral hygiene methods.

OTHER CONTRIBUTING ETIOLOGICAL FACTORS INCLUDING FOOD IMPACTION

Iatrogenic Factors

Faults in the dental restorations and prosthesis referred to as iatrogenic factors are common causes of gingival inflammation and periodontal destruction.

Faulty Restorations

Six characteristics of restorations are important from periodontal point of view.

a. Margins of restorations
b. Contours and overhanging dental restorations
c. Occlusion
d. Dental materials
e. Design of removable partial dentures
f. Restorative procedures themselves.

Biologic width is defined as the dimension of the soft tissue, which is attached to the portion of the tooth coronal to the crest of the alveolar bone. The biologic width is commonly stated to be 2.04 mm, which represents the sum of epithelial and connective tissue measurements. Hence, encroachment of the biologic width frequently leads to gingival inflammation, clinical loss of attachment and bone loss.

a. *Margins of restorations:* Subgingival restorations can contribute to periodontal diseases by,
   i. Providing ideal locations for the accumulation of plaque.
   ii. Changing the ecological balance of the gingiva to one that favors the growth of the disease associated organisms, at the expense of the health-associated organisms.
   iii. It was also demonstrated by Waerhaug et al (1978) that subgingival restorations are plaque retentive areas that are inaccessible to scaling instruments, hence greater chance of severe gingivitis and deeper pockets (Fig. 7.5).
b. *Contour of restorations/artificial crowns:* Over-contoured or improperly-contoured restorations tend to accumulate plaque and possibly prevent the self-cleansing mechanisms of the adjacent cheek, lips and

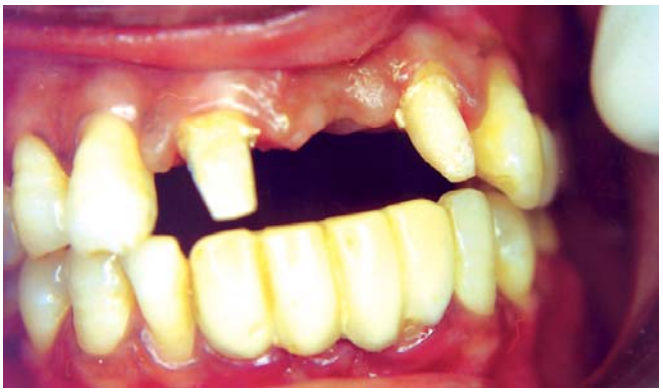


Fig. 7.5: Margins of restoration in relation to lower anterior teeth

tongue. Proper contours of the artificial crown are necessary for maintaining gingival health. In a review of periodontal prosthetic interactions, Becker and Kaldahl (1981) opined that buccal and lingual crown contours should be 'flat', not fat, usually <0.5 mm wider than the CEJ and that the furcation areas, should be "fluted or barreled out" to accommodate oral hygiene in these areas.

Overhanging dental restorations has been considered to be a contributing factor to gingivitis and possible periodontal attachment loss. Many authors have demonstrated radiographic bone loss adjacent to posterior teeth with overhanging restorations. The placement of the subgingival overhangs result in the changes in the associated microflora to that of one resembling the flora seen in chronic periodontitis. Hence, overhangs not only increase plaque mass but also increase the specific periodontal pathogens in the plaque (Fig. 7.6).

c. *Occlusion:* Poorly-constructed restoration will cause occlusal disharmonies that may be injurious to the supporting normal periodontal tissues. This type of tissue injury is called "primary trauma from the occlusion". Some of the examples are:
   i. Insertion of a "high filling".
   ii. Insertion of a prosthesis, replacement that creates excessive forces on abutment or antagonistic teeth.
   iii. The drifting movement or extrusion of the teeth into spaces created by unreplaced missing teeth or,
   iv. The orthodontic movement of teeth into functionally unacceptable positions.
d. *Dental materials:* In general, restorative materials are not by themselves injurious to the periodontal tissues. It is the rough, unpolished surfaces that favor plaque accumulation and contributes to periodontal diseases. Compared to all restorative materials that are available to the clinician, glass ionomer restorations and porcelain seems to retain less plaque and thus are more acceptable from periodontal point of view. Fluoride constantly leaking from the glass ionomer cement prevents the attachment of the bacteria to the pellicle and it also interferes with the metabolism and growth of bacteria, whereas highly-polished surfaces of porcelain inhibits plaque formation and permits its rapid removal too.

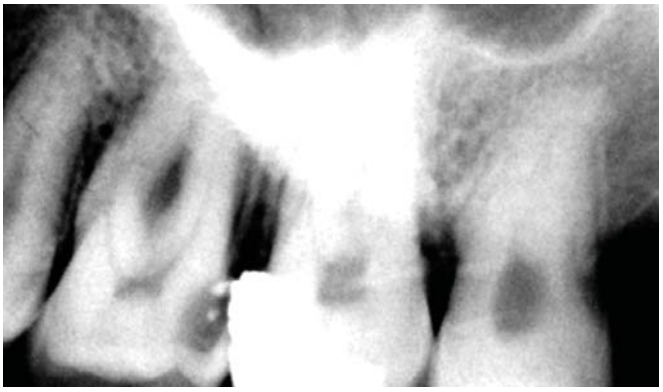


Fig. 7.6: Radiographic illustration of overhanging amalgam restoration

e. *Design of removable partial dentures:* Several studies have shown that after the insertion of partial dentures, there is an increase in the mobility of the abutment teeth within gingival inflammation and periodontal pocket formation. This is due to the increased plaque accumulation. The presence of removable partial dentures not only induces quantitative changes in plaque, but also qualitative changes by promoting the development of most pathogenic bacteria. Hence, from periodontal point of view, fixed prosthesis is more acceptable than the removable one.
f. *Restorative procedures themselves* can also cause destruction of periodontium. The use of the rubber dam clamps, copper bands, matrix bands and disks in such a manner as to lacerate the gingiva, results in the varying degrees of gingival inflammation.

Periodontal Problems Associated with Orthodontic Therapy

Orthodontic therapy may affect the periodontium by:

a. Favoring plaque retention and also modifying the gingival ecosystem.
b. Directly injuring the gingiva as a result of over extended bands which may also lead to forceful detachment of gingiva from tooth causing gingival recession.
c. Creating excessive and/or unfavorable forces on the supporting tooth structures causes necrosis of the

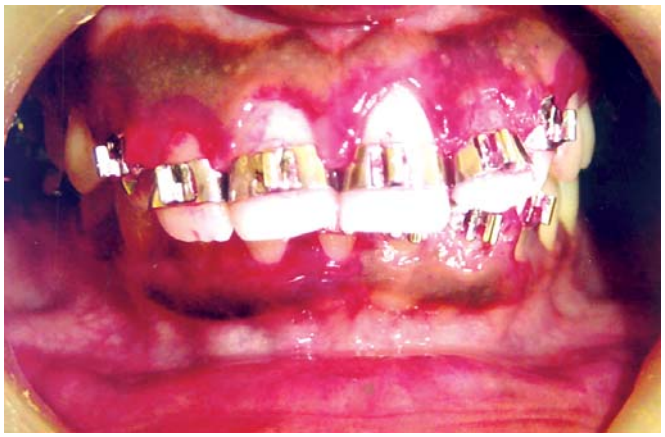


Fig. 7.7: Periodontal changes associated with orthodontic therapy

periodontal ligament and adjacent alveolar bone, excessive forces also increase the risk of apical root resorption (Fig. 7.7).

Food Impaction

It is the forceful wedging of the food into the periodontium by occlusal forces. Cusps that tend to forcibly wedge food interproximally are known as "Plunger cusps".

According to Hirschfeld food impaction can occur in the following conditions:

a. *Uneven occlusal wear:* It can lead to food impaction because deflection of food away from the proximal areas does not occur.
b. *Loss of proximal contact:* This is one of the most common cause for food impaction. It may be due to, periodontal disease, non-replaced missing teeth, proximal caries and abnormal biting habits.
c. Congenital morphologic abnormalities of teeth.
d. Improperly-constructed restorations.
e. *Lateral food impaction:* In addition to food impaction caused by occlusal forces, lateral pressure from the lips, cheeks, tongue may force food interproximally. This usually occurs when the gingival embrasure is enlarged by periodontitis or by recession.

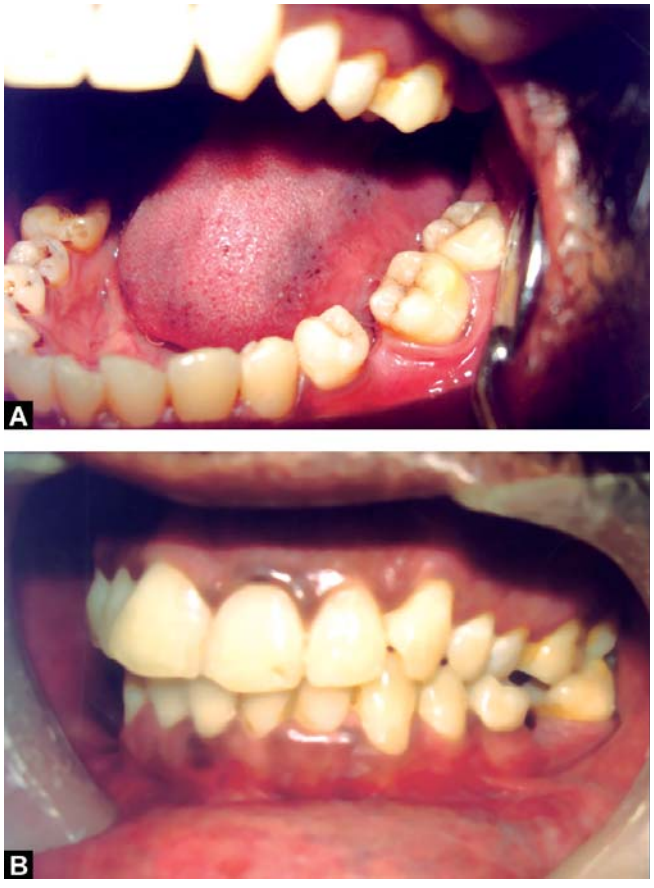
The following signs and symptoms may occur in the association with food impaction:

i. Feeling of pressure and urge to dig the material from between the teeth.
ii. Vague pain that radiates deep in the jaws.
iii. Gingival inflammation with bleeding and a foul taste in the involved area.
iv. Gingival recession.
v. Periodontal abscess formation.
vi. Varying degrees of inflammatory involvement of periodontal ligament, sensitivity to percussion.
vii. Destruction of the alveolar bone.
viii. Root caries.

Unreplaced Missing Teeth

Failure to replace extracted teeth initiates a series of changes that produce various degrees of periodontal diseases. The pattern of changes that may follow, failure to replace missing first molars is characteristic (Fig. 7.8). In extreme cases, it consists of the following:

a. The second and third molars tilt, resulting in a decrease in the vertical dimension.



Figs 7.8A and B: Sequelae of unreplaced missing first molar

b. The premolars move distally and the mandibular incisors tilt or drift lingually.
c. The anterior overbite is increased. The mandibular incisors strike the maxillary incisors near the gingiva or traumatize the gingiva.
d. The maxillary incisors are pushed labially and laterally.
e. The anterior teeth extrude because the incisal opposition has largely disappeared.
f. Diastema is created by the separation of the anterior teeth.

Loss of proximal contact relationships leads to,

Food impaction
↓
Gingival inflammation
↓
Pocket formation
↓
Followed by bone loss and tooth mobility.

Extraction of impacted third molars: Numerous clinical studies have reported that the extraction of third molars often results in the creation of vertical defects distal to the second molars and appears to occur more often in individuals older than 25 years than in those younger than 25.

Malocclusion (Figs 7.9 and 7.10A and B)

Depending on its nature, malocclusion exerts varied effect on the etiology of gingivitis and periodontal diseases (Fig. 7.9).

a. *Irregular alignment of teeth:* Makes plaque control difficult.
b. *Spacing between teeth:* Same sequelae following loss of proximal contact can occur.
c. *Facially-displaced teeth:* Can lead to gingival recession.
d. *Occlusal disharmony:* Results in injury to periodontium.
e. *Deep bite:* Inflammation of palatal mucosa.
f. *Open bite:* Leads to accumulation of plaque and periodontal atrophy.

Habits

Sorren has classified habits of significance in the etiology of periodontal diseases as follows:

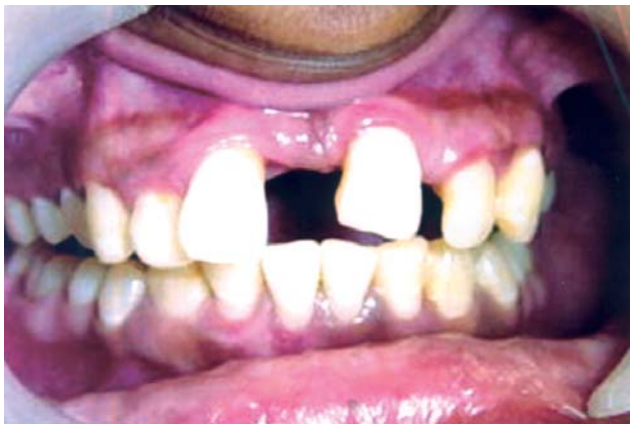
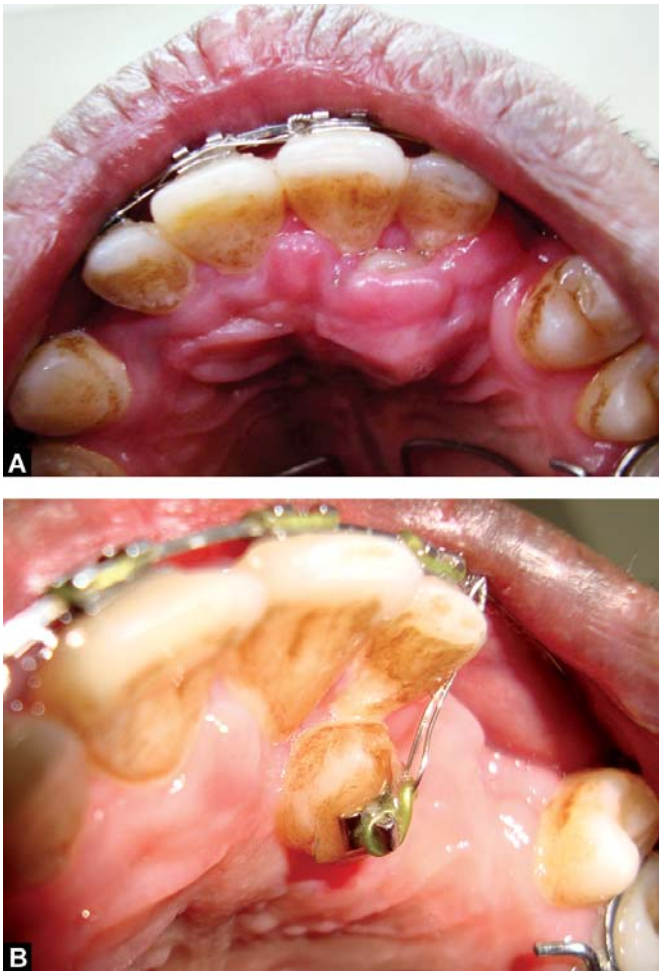


Fig. 7.9: Gingival changes associated with malocclusion



Figs 7.10A and B: (A) Canine impaction: Preoperative photograph, (B) Postoperative photograph

a. *Neurosis:* Such as lip biting and cheek biting which would lead to extrafunctional positioning of the mandible, others include tongue thrusting, fingernail biting and occlusal neurosis.

b. *Occupational habits:* Such as holding of nails in the mouth, e.g. carpenters, cobblers, etc.

c. *Miscellaneous habits:* Such as pipe or cigarette smoking, tobacco chewing, incorrect methods of tooth brushing, mouth breathing and thumb sucking.

I. *Mouth breathing:* Gingivitis is often associated with mouth breathing. The gingival changes include erythema, edema, enlargement and a diffuse shiny appearance on the exposed areas (Fig. 7.11).

II. *Tongue thrusting:* It is the persistent, forceful wedging of the tongue against the teeth. Instead of the dorsum of the tongue being placed against the palate with the tip behind the maxillary teeth during swallowing, the tongue is thrust forward against the anterior teeth.

Tongue thrusting causes excessive lateral pressure, which may be traumatic to the periodontium. It also causes spreading and tilting of the anterior teeth.

↓

Numerous secondary sequelae may develop from tongue thrusting. They include, change in the direction of the functional forces so that lateral pressure against the crowns is increased.

↓

Also interferes with food excursion and favors the accumulation of the food debris at the gingival margin.

Tongue thrusting is an important contributing factor in the pathologic tooth migration (Fig. 7.12).

III. *Use of tobacco:* The following oral changes may occur in the smokers:

1. Brownish, tar-like deposits and discoloration of tooth structure. (Due to the nicotine and its major metabolite, cotinine are deposited on the root surfaces).
2. Diffuse grayish discoloration and leukoplakia of the gingiva may occur (Fig. 7.13).
3. "Smokers palate" (nicotinic stomatitis), characterized by prominent mucous glands with inflammation of the orifices and a diffuse erythema or by a wrinkled, "cobble stone" surface, may occur (Fig 7.14).
4. Predisposition to acute necrotizing ulcerative gingivitis.
5. Delayed postsurgical healing.
6. Marked increase in gingival crevicular fluid flow.

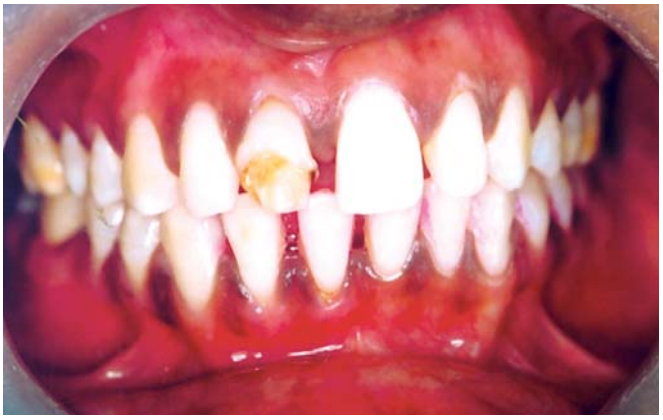


Fig. 7.12: Anterior overbite, pathologic migration associated with tongue thrusting

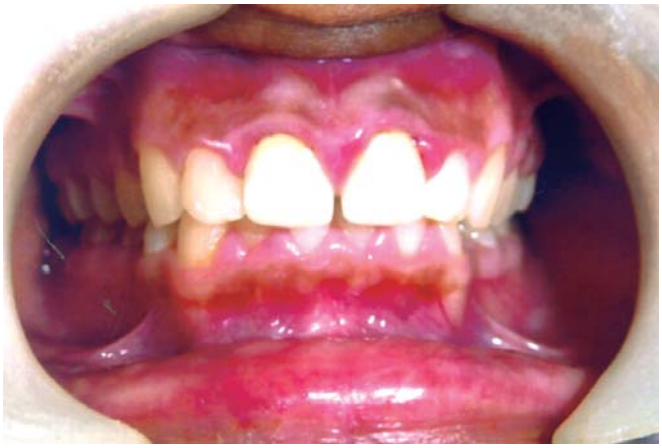


Fig. 7.11: Gingival changes associated with mouth breathing

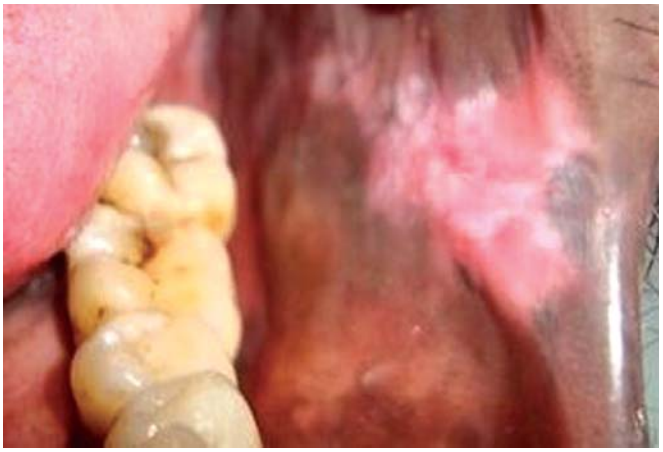


Fig. 7.13: Leukoplakia

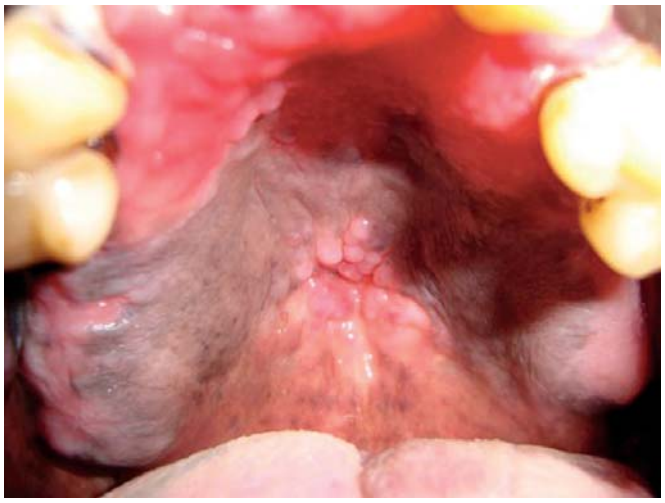


Fig. 7.14: Smoker's palate

7. More severe gingivitis and periodontitis have been reported in smokers.

   Special type of gingivitis, termed *gingivitis toxica* characterized by destruction of the gingiva and alveolar bone has been attributed to the chewing of tobacco.
8. Oral polymorphonuclear cells from smokers show reduced ability to phagocytose particles.

IV. *Toothbrush trauma:* Acute or chronic gingival changes.

Acute changes are:

1. Sloughing of the epithelial surface occurs along with denudation of the underlying connective tissue to form a painful gingival bruise.
2. Punctate lesions are produced by the penetration of the gingiva by the toothbrush bristles.
3. Painful vesicle formation in the traumatized areas is also seen.
4. Diffuse erythema and denudation of the attached gingiva throughout the mouth may be the striking sequelae of the overzealous brushing.

   The acute gingival changes noted can commonly occur when the patient uses a new brush.
5. Tooth bristles forcibly embedded and retained in the gingiva are a common cause of the acute gingival abscess.

Chronic toothbrush trauma results in:

1. Gingival recession with denudation of root surface
2. Often the gingival margin is enlarged and appears to be "piled up", as if it were molded in conformity with the strokes of the toothbrush.

Chemical irritation: Acute gingival inflammation may be caused by chemical irritation. The gingival changes range from simple erythema to painful vesicle formation and ulceration. The substances include, strong mouthwashes, dentifrices or denture materials, application of aspirin tablet to alleviate toothache and injudicious use of escharotic drugs.

Radiation: Patients with cancer of the oral cavity and adjacent regions who are treated with radiations initially develop erythema and desquamation of the oral mucosa including gingiva, which leads to ulcerations, infections and suppuration. The bone also undergoes degeneration. Radiation also includes atrophy of the salivary glands leading to xerostomia and changes in the oral flora predisposing to dental caries.

Occlusal neurosis or parafunctional habits: They are bruxism and clenching.

Bruxism: It is the clenching/grinding of the teeth when the individual is not chewing or swallowing.

Clenching: It is the closure of the jaws under vertical pressure.

Bruxism may lead to fracture of the teeth or dental restorations, tooth wear or uncosmetic muscle hypertrophy.

Two types of bruxism have been reported—Nocturnal Bruxism/Nonstress bruxists and diurnal bruxism/stress bruxists.

Clinical features of bruxism: Clinically you can diagnose bruxism by the presence of facet patterns. Sleep studies have shown that bruxism occurring during REM (rapid eye movement) sleep may be the most damaging. No association has been shown between bruxism and periodontitis or gingival inflammation.

Treatment of bruxism: Occlusal adjustment is contraindicated.

Maxillary stabilization appliance is advised, which is the most effective means of treating Bruxism. The main aim of this appliance is to protect the tooth surface and to

dissipate forces built up in the musculoskeletal system through the bruxism. This is more ideal to treat nocturnal bruxism than for correcting daytime clenching habits. It results in an immediate reduction in the masseter and temporalis muscle activity levels. The appliance should be readjusted in 2 to 4 weeks and thereafter over longer intervals bruxofacets should be observed in the follow-up visits and the surface should be burnished with a smooth, pumice impregnated rubber wheel.

KEYPOINTS

Calculus

1. Dental calculus is an adherent, calcified or calcifying mass that forms on the surface of the teeth and dental appliances.
2. Two types of calculus, supragingival and subgingival calculus is seen.
3. It is made up of organic and inorganic constituents.
4. Four types of attachments of calculus to the tooth surface has been reported.
5. Calculus formation takes place by the precipitation of mineral salts.
6. Theories that are explained in calculus formation are:
   a. Booster mechanism.
   b. Epitactic or heterogenous nucleation
   c. Inhibition theory
7. Finally, although a positive correlation exists between calculus and periodontal disease, it is plaque that shows a greater correlation with periodontal disease.

Other Contributing Etiological Factors including Food Impaction

8. Faults in the dental restorations and prosthesis are referred to as iatrogenic factors.
9. From the periodontal point of view, 6 characteristics of restorations are important:
   a. Margins of restorations.
   b. Contours and overhanging dental restorations.
   c. Occlusion.
   d. Materials.
   e. Design of removable partial dentures.
   f. Restorative procedures themselves.
10. Orthodontic therapy may affect the periodontium by, directly injuring the gingiva due to over extension of bands, favoring the plaque retention and changing gingival ecosystem and creating unfavorable forces on the supporting tooth structures.
11. Food impaction is defined as the forceful wedging of the food into the periodontium.
12. Bruxism is the clenching or grinding of the teeth when the individual is not chewing or swallowing. Clenching is the closure of the jaws under vertical pressure.

KNOW MORE ...

Calculus

The rate of calculus formation varies from person to person. For some, calculus may form faster than others. Accordingly they have been classified as heavy, moderate, mild and non-calculus formers. Various factors affecting the rate of calculus formation are—oral hygiene habits, accessibility for professional care, diet, age, frequency of professional scaling, medications and systemic diseases.

Other Contributing Etiological Factors

Stains

Pigmented deposits on tooth surface are called stains.

Discoloration of teeth can occur in three different ways:

- Stains adhering directly to tooth surface.
- Stains contained within the calculus and soft deposits.
- Stains incorporated within the tooth structure or restorative material.

Classification of Stains

Based on location

a. *Extrinsic stains:* They occur on the external surface of the tooth and may be removed by procedures like tooth-brushing, scaling and/or polishing.
b. *Intrinsic stains:* They occur within the tooth substance and cannot be removed by scaling and polishing.

Based on source

a. *Exogenous:* They develop or originate from sources outside the tooth. Exogenous stains may be extrinsic and stay on the outer surface of tooth or intrinsic and become incorporated within the tooth.
b. *Endogenous:* They develop or originate from within the tooth. Endogenous stains are always intrinsic and usually are discolorations of dentin reflected through enamel.

Extrinsic Stains

1. *Brown stain:* It is seen as thin, translucent, acquired, usually bacteria free, pigmented pellicle. It is seen in individuals who do not brush adequately or who use dentifrice with inadequate cleansing action.

   It is commonly seen on the buccal surface of maxillary molars and lingual surface of mandibular anterior teeth.

   The brown color is due to presence of tannin.
2. *Tobacco stain:* It is seen as tenacious dark brown or black deposit accompanied by brown discoloration of tooth substance. It is commonly seen on the lingual surfaces of teeth.

   Staining result from coal tar combustion products and from penetration of pits and fissures, enamel and dentin by tobacco by-products. The degree of staining is not necessarily proportional to amount of tobacco consumed, but depends to a considerable degree on pre-existent acquired coatings.

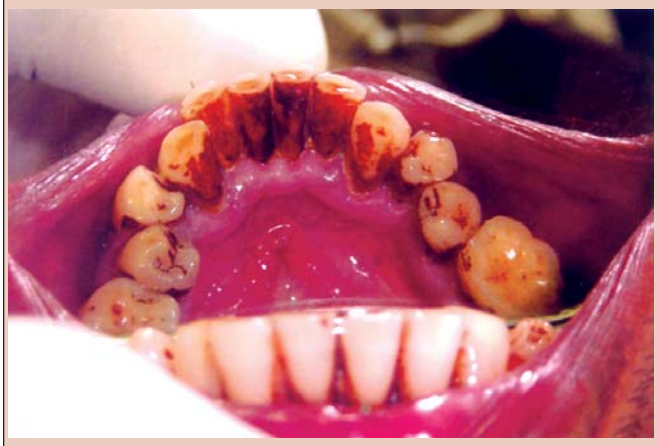


Fig. 7.15: Tobacco stains on the lingual surface of the lower anterior teeth

3. *Black stain:* It occurs as a thin black line on the facial and lingual surfaces of teeth along the gingival margin and as diffuse patches on proximal surfaces.

   It is firmly attached and tends to recur after removal. It is seen more common in women, children and individuals with excellent oral hygiene.

   It is caused by chromogenic bacteria namely *actinomyces species, prevotella melaninogenicus*.
4. *Green stain:* Green or greenish yellow stain is commonly seen in children. It is considered to be stained remnants of enamel cuticle. It is commonly seen on gingival half of maxillary anterior teeth. It is more frequent in boys (65%) than girls (63%).

   It is caused by fluorescent bacteria and fungi such as penicillin and aspergillus.
5. *Orange stain:* It is seen on both facial and lingual surfaces of anterior teeth. It is caused by chromogenic bacteria: *Serratia marcescens* and *Flavobacterium*.
6. *Metallic stains:* Metallic stains are caused by incorporation of metals and metallic salts and their products into acquired coatings. It is seen in industrial workers due to inhalation of metal containing dust.

   Commonly seen are copper stains seen as green stain; iron dust causes brown stain; manganese causes black stain; mercury causes greenish black stain; nickel causes green stain and silver causes black stains.
7. *Chlorhexidine stains:* Brown color staining of teeth and tongue has been noted following the use of chlorhexidine mouthrinse. Intensity of staining does not depend on concentration of chlorhexidine rinse. Following mechanisms have been suggested to cause staining:
   - Degradation of chlorhexidine resulting in the formation of parachloraniline
   - Catalysis of browning reaction of carbohydrates and amino acids by chlorhexidine
   - Denaturation of proteins resulting in the formation of sulphides
   - Precipitation of anionic dietary chromogens.

Intrinsic Stains

Stains that occur within the tooth can be caused by:

Exogenous sources

1. Restorative materials
   - Silver amalgam imparts grayish hue to the tooth
   - Copper amalgam produces bluish-green color.
2. Drugs
   - Stannous fluoride produces brown stain
   - Ammonical silver nitrate produces dark brown to black stain.

Endogenous sources

Example: Nonvital tooth

- Tetracycline administration during various stages of tooth development
- Developmental anomalies namely amelogenesis imperfecta, dentinogenesis imperfecta
- Enamel hypoplasia
- Dental fluorosis
- Erythroblastosis fetalis.

REVIEW QUESTIONS

Calculus

1. Define dental calculus. Describe the types and theories of formation of dental calculus.
2. What is the composition of dental calculus?
3. Differences between supragingival and subgingival calculus.

Other Contributing Etiological Factors including Food Impaction

1. What are the effects of overhanging restorations on periodontium?
2. What are the causes for the food impaction?
3. What is the sequelae following unreplaced missing teeth?
4. What are the effects of smoking on periodontium?
5. What are parafunctional habits?

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8 Chapter

Host Response: Basic Concepts

- ROLE OF SALIVA IN THE HOST DEFENCE
- GINGIVAL EPITHELIUM
- GINGIVAL CREVICULAR FLUID
- COMPLEMENT
- INFLAMMATORY CELL RESPONSE
- IMMUNOLOGICAL MECHANISMS
- IMMUNOLOGY OF PERIODONTAL DISEASE

INTRODUCTION

Health is not a static condition. It is a dynamic state in which the living and functioning organism or tissue remains in balance with a constantly changing environment. This constant process of readjustment to maintain a functional integrity is known as "Homeostasis". It is a well known fact that bacteria constitute an important part of environment and all the external surfaces in nature including living tissues are covered by bacteria, the skin, the gut and oral mucosa are of no exceptions. When different forms of life exist together there is competition for existence, hence various mechanisms have evolved to help one form to protect it from another called *Host defence mechanisms*.

The tissues of the periodontium are exposed to various environmental factors in the oral cavity. Over 300 species of bacteria have been isolated in the oral cavity. The periodontal tissues remain in a state of partnership (symbiosis) with most of the bacteria and only under certain circumstances do we suffer from their attack because, host defence system strikes a balance between the two.

The host responds to the attack of bacteria and its toxins at various levels.

ROLE OF SALIVA IN THE HOST DEFENCE

1. A vehicle for swallowing bacteria.
2. Inhibition of attachment of bacteria.
3. Bactericidal action by the peroxidase system.
4. Bactericidal action by lysozyme, lactoferrin and other factors.

Salivary Peroxidase System

SCN^- +	H_2O_2	→	HOSCN
(Thiocyanate from salivary glands)	(Generated by salivary glands, bacteria, neutrophils, etc.)	(Peroxidase enzyme)	Hypothio-cyanous acid, kills the bacteria)

Peroxidase is synthesized by salivary gland acini and secreted into the saliva, where it becomes bound to bacteria and thiocyanate is secreted into saliva by the ductal cells. Hydrogen peroxide is constantly secreted in low

concentration by bacteria, neutrophils and other host cells and is used by peroxidase to oxidize the thiocyanate to hypothiocyanous acid, which kills bacteria.

Lactoferrin: It is secreted by serous salivary gland, which binds iron, an important growth factor or requirement for many microorganisms. This action is bacteriostatic rather than bactericidal.

Lysozyme: It is an antimicrobial enzyme in the saliva secreted mainly by mucous salivary glands and it degrades mucopeptides in the cell wall of gram-positive bacteria, weakening the wall and causing lysis.

GINGIVAL EPITHELIUM

Gingival epithelium has three functions:

1. Epithelial cells are tightly attached to each other.
2. Keratinization to resist trauma.
3. Presence of permeability barriers.

GINGIVAL CREVICULAR FLUID

Gingival crevicular fluid functions are:

1. Washing nonadherent bacteria and their products out of the crevice.
2. Reducing the diffusion of plaque products into the tissues.
3. It also carries a steady supply of inflammatory mediators, protease inhibitors and host defence agents such as complement and antibody, into the crevice.

COMPLEMENT (FIG. 8.1)

The functions of complement are:

a. *Chemotaxis cellular activation:* Complement products released in this reaction attracts phagocytes to the site of infection, e.g. C_{3a} and C_{5a}.
b. *Opsonization:* Once they arrive at the site of infection the complement components coat the bacterial surface and allow the phagocytes to recognize the bacteria and thereby facilitating the bacterial phagocytosis, e.g. C_{3b}.
c. *Cytolysis:* Damage to the plasma membranes of the cells can lead to lysis of the cell, e.g. C_1-C_9.

The complement system comprises of nine major complement proteins which circulate in an inactive form and which, like the clotting system, are activated in an enzyme cascade.

C_1-C_9 causes cytolytic and cytotoxic damage to the cell.

Classical Pathway

The sequence is C_1, C_4, C_2, C_3, C_5, C_6, C_7, C_8, C_9.

Alternative Pathway

It is activated by antibodies of immunoglobulins and also by endotoxins. They can initiate the third component of the complement without starting from the beginning of the cascade.

The sequence is C_3, C_5, C_6, C_7, C_8, C_9.

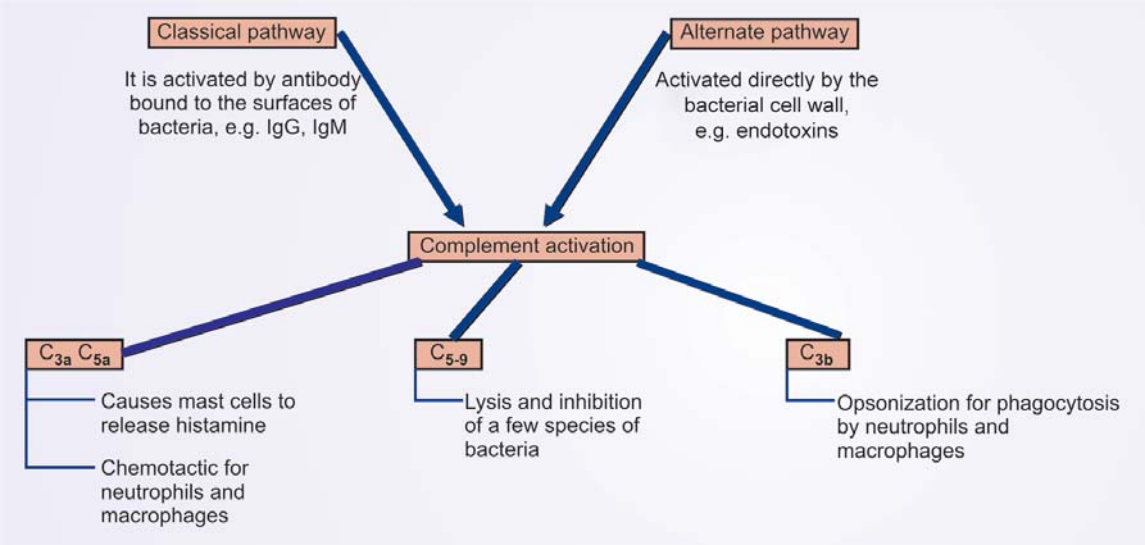


Fig. 8.1: Complement activation

THE INFLAMMATORY CELL RESPONSE

It involves emigration of neutrophils, macrophages and lymphocytes from the blood vessels into the tissues. Neutrophils and macrophages perform inflammatory functions whereas macrophages and lymphocytes perform immunological functions.

Neutrophils

They are the initial leukocytes seen in the gingiva. They exit the circulation and migrate into the junctional epithelium and gingival crevice, where they provide the first cellular host mechanism to control periodontopathic bacteria.

Functions of Neutrophils

Emigration and chemotaxis: Leukocytes normally travel along the center of the lumen of the blood vessel, but in inflamed tissues the blood flow is slowed by fluid exudation and they adhere more readily to endothelial cells, the mechanism is called "rolling" and "margination". When the neutrophils migrate across the endothelium it is called "diapedesis" and "interendothelial transmigration".

Hence, there are two phases of leukocyte endothelium adherence.

a. The selectin-dependent phase (primarily in rolling and margination).
b. The integrin-dependent phase (primarily in diapedesis).

The selectin-dependent phase: The various selectins are:

1. *L-selectin*—expressed on the surface of the leukocyte.
2. *P-selectin*—stored in the granules of endothelial cells (Weibel-Palade bodies).
3. E-selectin—expressed by endothelial cells.

P-selectin and E-selectin: They both strengthen the binding between the leukocyte and the endothelial cell and increase the number of leukocyte "rolling".

The integrin-dependent phase: (Leukocyte B_2-integrins): The three leukocyte integrins are sequestered in the specific granules of leukocytes,

- LFA-1—leukocyte function-associated antigen-1 CD_{11a}/CD_{18}
  (or)
  Mac-1/CD_{11b}/CD_{18}
- CD_{11c}/CD_{18}

These leukocyte B_2-integrins act as the molecular mediators of binding to the endothelial cells and their binding affinity can be increased or decreased as the leukocyte traverses the postcapillary venule.

Chemotaxis: It is the directed movement of a cell along a chemical gradient. The neutrophils are attracted by chemical signals from multiple sources, e.g. chemotaxins which include compounds such as complement fragments (C5a).

LTB4: It is secreted by mast cells, neutrophils macrophages, and also bacterial products including LPS and factors released by damaged tissues.

Chemotaxis requires phagocyte, which possess specific chemotaxin receptors, and the most well-studied chemotaxin receptor is the receptor for formylmethionyl peptides, known as the FPR (Formylmethionyl Peptide Receptor).

Stages in neutrophil chemotaxis (Fig. 8.2)

Stage I: Soluble products diffuse forming a concentration gradient.

Stage II: Resting neutrophils detect chemotaxins using surface receptors.

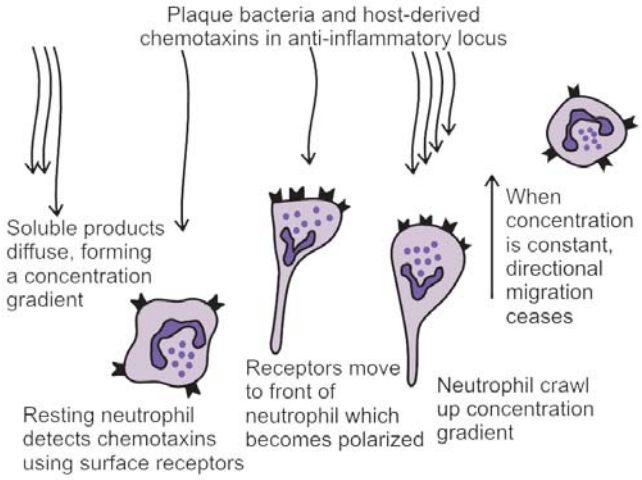


Fig. 8.2: Stages in neutrophil chemotaxis

Stage III: Receptors move to front of neutrophils, which becomes polarized.

Stage IV: Neutrophils crawl up to the concentration gradient.

Stage V: When concentration is constant, directional migration ceases.

II Phagocytosis (Fig. 8.3)

Once they arrive at the site of inflammation, the phagocytes have to recognize the infectious agent. This can be enhanced if the organism has been coated by C_{3b}. They may then attach to microorganism via their nonspecific cell surface receptors. After attachment the phagocytes proceed to engulf the microorganism by extending pseudopodia around it. Once inside, lysozymes fuse with the phagosome/phagocytic vacuole to form a *phagolysozyme* and the infectious agent is killed by a battery of microbiocidal mechanisms.

The main stages of bacterial killing by phagocytes:

For efficient phagocytosis, the particle should be coated with one or more host serum proteins. This process is called *opsonization* (meaning "to prepare for eating"). The two principal types of serum proteins referred to as opsonins are *IgG and* C_{3b}. There are receptors of these two proteins on the phagocytes. Hence, the opsonization could be either complement-dependent, antibody-dependent and the combination of both. Once the organism has been internalized, lysozymes fuse with the phagosome to form a phagolysozyme. Various killing mechanisms are activated, they are (Fig. 8.4):

A. Nonoxygen-dependent killing mechanisms.
B. Oxygen-dependent killing mechanisms.

Oxidative mechanism: Stimulation of phagocytic cells leads to an increase in cellular consumption of molecular

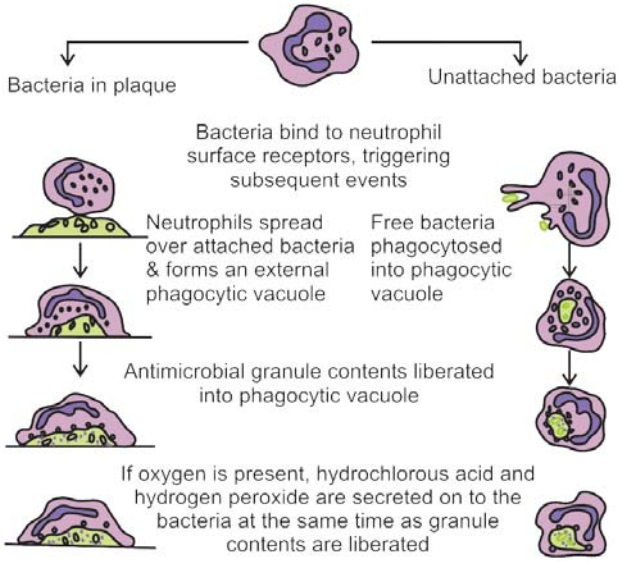


Fig. 8.3: Phagocytosis

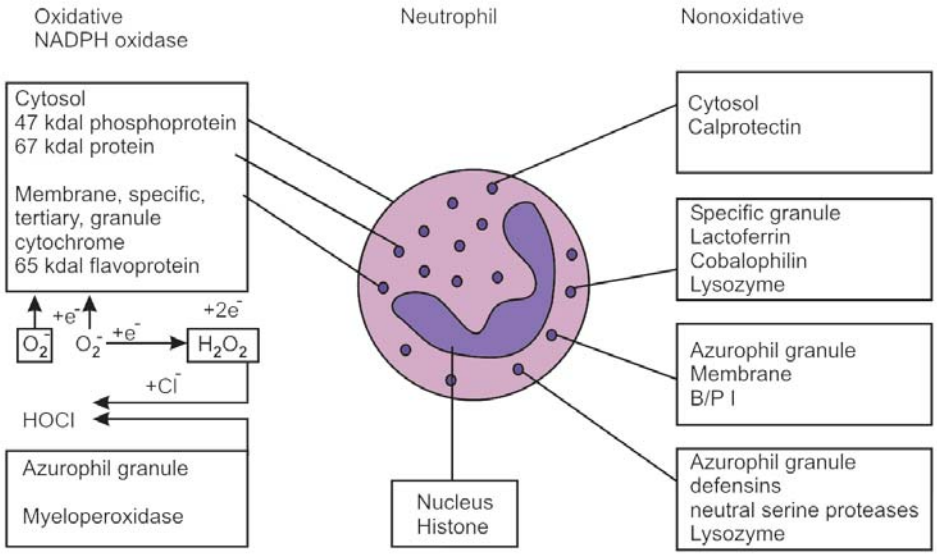


Fig. 8.4: Neutrophil: Oxidative and nonoxidative mechanisms for controlling microbes

oxygen, a process termed as the *respiratory burst.* This is associated with the generation of various oxygen metabolites, which are injurious to many species of microorganisms. The majority of the oxygen consumed by the phagocyte is converted directly to superoxide anion (O_2^-) through the action of a membrane—bound NADPH oxidase. Superoxide radicals inturn may undergo conversion to hydrogen peroxide (H_2O_2) either spontaneously or via superoxide dismutase and contribute significantly to microbicidal activity of the phagocytic cells. Additional oxidants, e.g. hypochlorous acid and toxic aldehydes are generated as a consequence of the interaction between hydrogen peroxide and azurophil enzyme (MPO). Hence, the oxygen-dependent bactericidal activity is further divided into:

- Myeloperoxidase—dependent
- Myeloperoxidase—independent.

Non-oxidative mechanisms: It appears to be based on the various components of the cell. Neutrophils contain three types of granules:

1. *Primary granules/azurophilic granules:* Myeloperoxidase, lysozyme, acid phosphatase, and acid hydrolases.
2. *Secondary/specific granules:* Lactoferrin, lysozyme, azurocidin.
3. *Tertiary granules:* Alkaline phosphatase, collagenase, and gelatinase.

Neutrophil Disorders Associated with Periodontal Diseases

1. Diabetes mellitus
2. Papillon-Lefevre syndrome
3. Down's syndrome
4. Chediak-Higashi syndrome
5. Drug-induced agranulocytosis
6. Cyclic neutropenia.

Periodontal Diseases Associated with Neutrophil Disorders

- Acute necrotizing ulcerative gingivitis (ANUG).
- Localized juvenile periodontitis (LJP).
- Prepubertal periodontitis (PPP).
- Rapidly progressive periodontitis (RPP).
- Refractory periodontitis (RP).

Functions of Macrophages in the Gingiva, Crevice and Pocket

Macrophages develop from blood monocytes, which emigrate into the tissues from the blood and are triggered to develop into mature macrophages by cytokines, other inflammatory mediators, bacterial products such as endotoxins.

Functions of macrophages (Fig. 8.5) includes:

1. Phagocytose and kill bacteria.
2. Remove damaged host tissue during inflammation.
3. And also trap and present antigens to lymphocytes for induction of immune responses.

Because these functions unite inflammation and immunity the macrophages play an important role in all bacterial infections.

Most of the above mentioned functions are carried out through the secretion of inflammatory mediators, including cytokines, prostaglandins, leukotrienes and complement components. Particularly important is the secretion of the cytokines and although other cells such as fibroblasts,

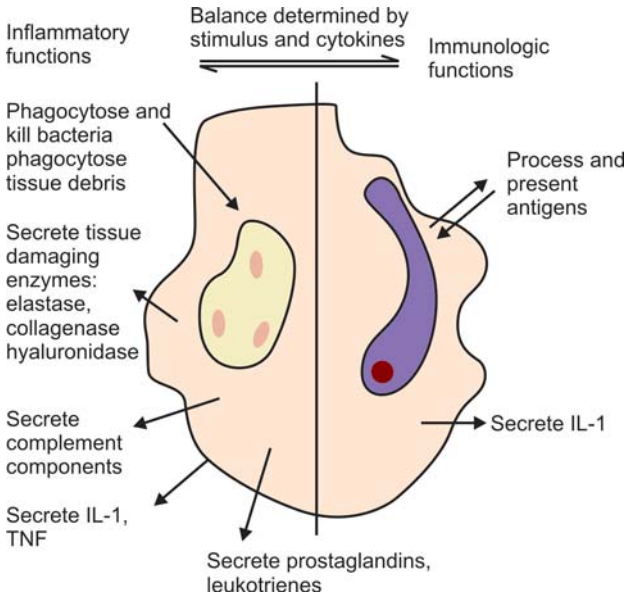


Fig. 8.5: Functions of macrophage in periodontal tissues

endothelial cells and keratinocytes also secrete cytokines, macrophages secrete the greatest quantities. The most significant cytokine in inflammation is interlenkin-1 (IL-1), which is a key mediator both in inflammation and immunity-induced by bacteria. Tumor necrosis factor (TNF) is also produced by macrophages. Both TNF and IL-1 have similar functions.

They increase inflammation by, releasing histamine from mast cells, attracting neutrophils and more macrophages into the tissues and by causing many other cells to release prostaglandins.

In summary, macrophages, like neutrophils are required for the effective host response, but can mediate a small amount of bystander damage. The damage could be caused by direct effect that is by secreting enzymes and toxins (similar to neutrophils) and indirectly by secretion of cytokines. In excess amounts IL-1 and TNF can have several damaging effects such as stimulation of bone resorption and tissue fibrosis.

Other cells such as mast cells, fibroblasts, endothelial cells, plasma cells and epithelial cells are also seen in gingival connective tissue during inflammatory response.

Lymphocytes (Fig. 8.6): Three types of cells are included,

1. *T-lymphocytes or T-cells:* Derived from the thymus and play a role in cell-mediated immunity.
2. *B-lymphocytes or B-cells:* Derived from liver, spleen and bone marrow. They are precursors for plasma cells and play a role in humoral immunity.
3. Natural killer (NK) cells.

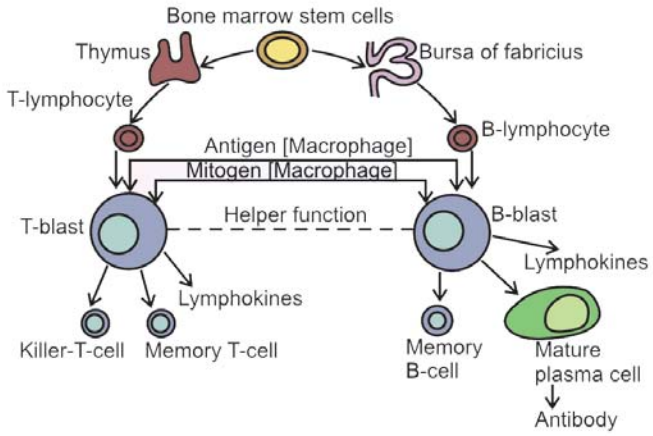


Fig. 8.6: Derivation and response of B and T-lymphocytes

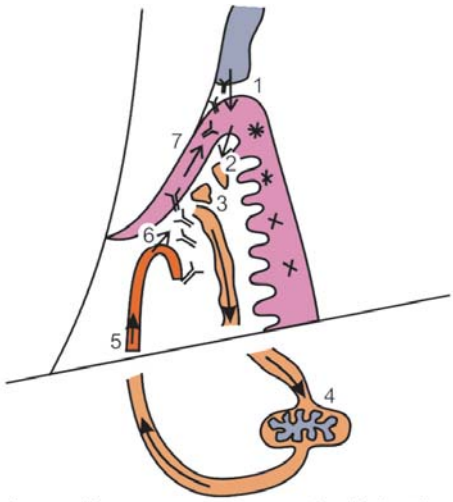
Different types of T-cells include:

Helper inducer T-cells (TH-cells) or CD4: They aid in the cellular response of the B-cells to differentiate into plasma cells and produce antibodies.

Suppressor-cytotoxic T-cells (TS-cells) or CD8: Stimulates cytotoxic and microbicidal activity of the immune cells. Subdivided into TH_1 TH_2 TH_0.

- TH cells release IL-2 and IFN (interferons)
- TS cells release interleukin: IL-4 and IL-5
- In adult periodontitis TH cells increase and TS cells decrease with increased gingival inflammation.

The humoral response to plaque (Fig. 8.7): Plaque bacteria and their soluble products such as enzymes and toxins carry out activation of the humoral response. Antigens, which pass into the tissues, are carried to the local lymph nodes, probably by macrophages, where they are presented to lymphocytes, which circulate continually



Systemic humoral immune response to microbial antigens within the gingival crevice region

1. Plaque antigens diffuse through the junctional epithelium.
2. Langerhans cells within the epithelium capture and process the antigens.
3. Antigen-presenting cells [macrophages & Langerhans cells] leave the gingiva in the lymphatics.
4. Antigen-presenting cells reach the lymph node and begin to stimulate lymphocytes to produce a specific immune response.
5. Periodontal microbe specific antibodies are produced by plasma cells within the lymph nodes & travel back to the gingiva via blood vessels.
6. Antibodies leave the circulation and are carried to the crevice in the transudate from the inflamed and dilated blood vessels.
7. Antibody action on microbes in the crevice can result in killing, aggregation, precipitation, detoxification, opsonization and phagocytosis of bacteria.

Fig. 8.7: Systemic humoral immune response

through the nodes and tissues. The lymphocytes, which recognize each individual antigen, are activated, undergo clonal expansion and differentiate into plasma cells, which secrete antibody under the control of helper and suppressor T-lymphocytes. Most of the lymphocytes and plasma cells remain in the lymph nodes and secrete antibodies into the bloodstream. The antibody predominantly is IgG, which can opsonize and activate the complement. Small amounts of IgM is also seen which is more of an activator of the complement and less of an effective opsonin. These antibodies pass into the gingival inflammatory exudates and then out into the gingival crevice in the crevicular fluid.

Possible mechanisms of action of antibodies in periodontitis:

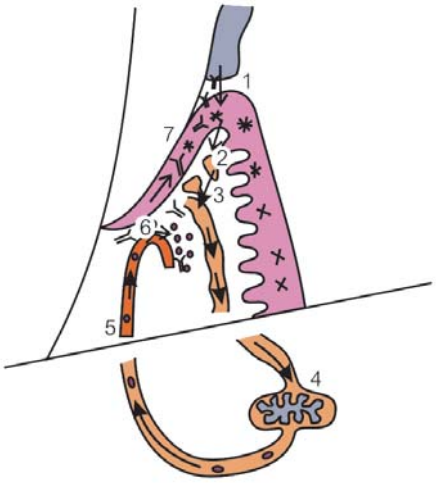
I. *Binding to bacteria, thus*:
   a. Opsonizing for phagocytosis
   b. Activating neutrophil enzyme secretion
   c. Coating bacteria and inhibiting attachment
   d. Activating complement and thus enhancing opsonization
   e. Directly inhibiting bacterial metabolism.

II. *Binding to soluble factors, thus:*
   a. Neutralizing toxins.
   b. Inhibiting enzymes.

The cell-mediated response in periodontal diseases: It is so called because it involves contact between cytotoxic T-cells and the target to be destroyed. These reactions are effective against persistent antigens, which are resistant to degradation, and the cells infected with viruses and tumor cells (Fig. 8.8).

IMMUNOLOGICAL MECHANISMS (FIG. 8.9)

They are stimulus—specific and differentiate between individual pathogenic species and sometimes-individual strains. Microorganisms and their products are recognized as being different from the host because they contain structures, which are not found in the human body. These so called antigens (antibody-generating) are first recognized by the lymphocytes and each lymphocyte is capable of recognizing only one foreign antigen and when it comes in contact with that antigen it is triggered to divide several times and therefore within few days there are many more



Local cellular immune response within the gingival crevice region and how this is invoked by microbial antigens and the mechanism by which pertinent periodontal immune cells traffic to the periodontium
1. - 4. Same as humoral immune response.
5. Periodontally-specific B cells and T cells proliferate within the lymph nodes and enter the bloodstream.
6. Periodontally-specific lymphocytes "home" back to the periodontium and locate within the tissues where they begin their humoral and cell-mediated immune functions.
7. Antibodies are produced locally by plasma cells which are controlled by type-2 T-helper cells. Cell-mediated immune activity is regulated by type-1 T-helper cells.

Fig. 8.8: Local cellular immune response

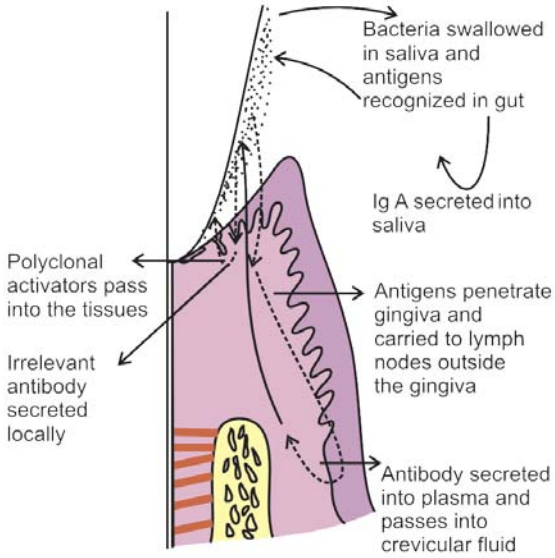


Fig. 8.9: Antibody production by salivary and humoral immune systems and by polyclonal B cell activation

cells with the same specificity. This amplification process is known as clonal expansion. This will result in the production of larger pool of cells, which are differentiated to protect the host either by humoral or cell-mediated mechanisms.

Humoral responses are carried out by lymphocytes, which differentiate into plasma cells and secrete antibody directed against the original antigen. Cell-mediated responses, in contrast, do not require antibody, but depend on the clonal expansion to provide large number of lymphocytes, which destroy targets directly. This direct effect of an immune reaction against a foreign antigen results in significant tissue damage is referred to as hypersensitivity reaction.

Type I: Anaphylactic Reactions

Two variations in the anaphylactic hypersensitivity may occur, depending on the route of the administration of antigen. If injected locally into the skin, the reaction is called cutaneous anaphylaxis.

If the antigen is injected intravenously it is called systemic or generalized anaphylaxis. The basic mechanisms of both types are similar (Fig. 8.10).

Mechanisms of Anaphylactic Hypersensitivity

Anaphylaxis occurs when two Ig E antibodies that are fixed to a mast cell or basophil react with the antigen through the Fab portion of the antibodies. This antibody-antigen reaction causes the release of the pharmacologically-active substances from the sensitized-cells.

These mediators released by the human mast cells include:

a. Histamine—increased capillary permeability.
b. Alpha 2-macroglobulins—collagenase activation.
c. SRS-A—smooth muscle contraction.
d. Bradykinin—increased permeability same as SRS-A. (Slow release substance of anaphylaxis).

Type II: Cytotoxic Reactions

In cytotoxic type, antibodies react directly with antigens tightly bound to cells. A cytotoxic reaction involving these cells will result in hemolysis. Cytotoxic antibodies are of

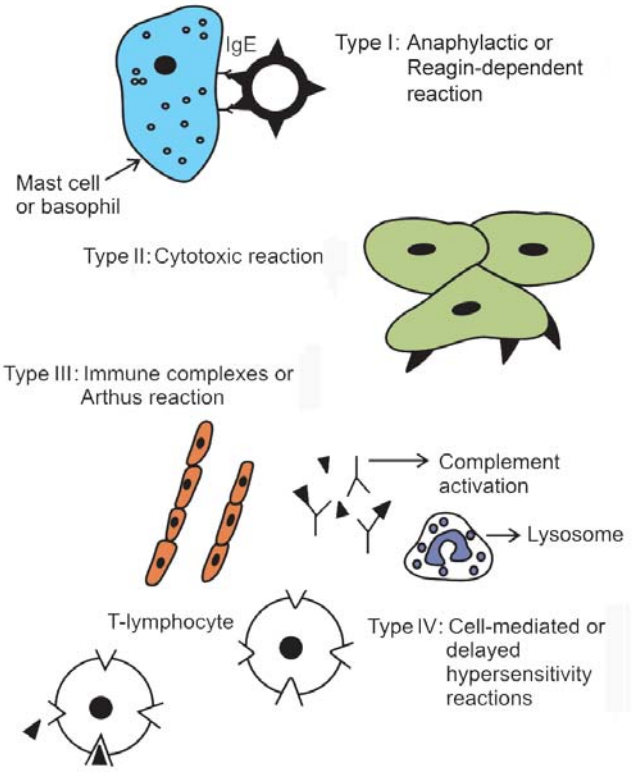


Fig. 8.10: Immunologic mechanism of tissue damage

IgG or IgM class. In addition to inducing cell lysis, cytotoxic antibodies may cause tissue damage by increasing the synthesis and release of lysozymal enzymes by cells (PMNs). The tissue in the vicinity of these enzymes will then be damaged.

Cytotoxic reactions are seen in the autoimmune diseases where antibodies react with body's own tissue components. For example, this occurs in pemphigus where antibodies react with cell membranes and pemphigoid antibodies react with the epithelial basement membrane. To date evidence suggests an important role for the cytotoxic reactions in the gingivitis and periodontitis.

Type III: Immune Complex or Arthus Reactions

When high levels of antigen are present and persist without being eliminated antigen-antibody (IgG or IgM) complexes precipitate in and around small blood vessels and with subsequent complement activation cause tissue damage at the site of the local reaction. Inflammation, hemorrhage and necrosis may occur. Tissue damage appears to be due to the

release of lysozymal enzymes from various cells such as neutrophils, mast cells, etc. This reaction is referred to as immune complex or Arthus reaction.

Type IV: Cell-mediated or Delayed Hypersensitivity

Cellular immunity does not include circulating antibodies but is based on the interaction of the antigens with the surface of T-lymphocytes.

IMMUNOLOGY OF PERIODONTAL DISEASE (FIG. 8.11)

Immune responses may be both beneficial and detrimental. Several components of the immune system are active in periodontal disease. These host variables may influence bacterial colonization, bacterial invasion, tissue destruction, healing and fibrosis (Tables 8.1 and 8.2).

Table 8.1: Influence of host responses on periodontal diseases

- *Bacterial colonization:* Subgingivally, antibody, complement in crevicular fluid inhibits adherence and co-aggregation of bacteria and potentially reduces their numbers by lysis.
- *Bacterial invasion:* Antibody-complement-mediated lysis reduces bacterial counts. Neutrophil-mediated lysis also reduces bacterial counts.
- *Tissue destruction:* Antibody-mediated hypersensitivity, cell-mediated immune responses, activation of tissue factors such as collagenase.
- *Healing and fibrosis:* Lymphocytes and macrophage produced chemotactic factor for fibroblasts, fibroblast-activating factors.

Table 8.2: Significant immune findings in periodontal diseases

Disease	*Immune response*
ANUG	• PMN chemotactic defects • Elevated antibody titers to *Prevotella intermedia* and intermediate-sized spirochetes
Pregnancy gingivitis Adult periodontitis	• No significant findings reported • Elevated antibody titers to the *Porphyromonas gingivalis* and other pathogens

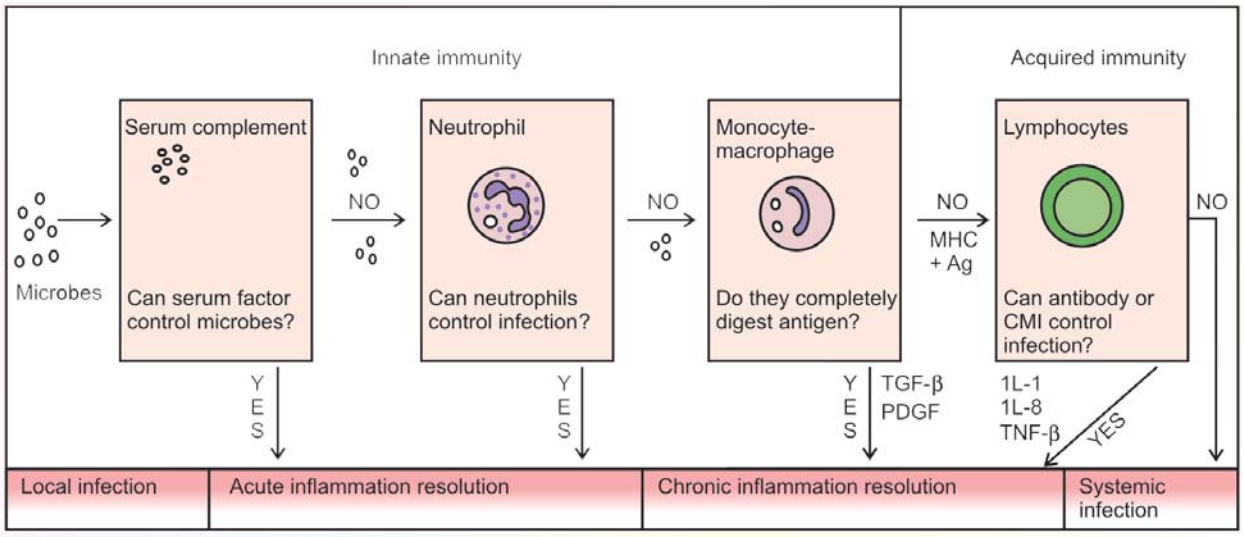
Contd.

Contd.

Disease	*Immune response*
	• Presence of immune complexes in tissues • Cell-mediated immunity to gingival bacteria
Juvenile Periodontitis:	
LJP	• Polymorphonuclear leukocytes chemotactic defect and depressed phagocytosis • Elevated antibody levels to *Actinobacillus actinomycetemcomitans* • Defect in GP_{110} receptors
GJP	• PMN chemotactic defect and depressed phagocytosis • Elevated antibody levels to *P. gingivalis*
Prepubertal periodontitis	• PMN and monocyte chemotactic defects
Rapidly-progressing periodontitis	• Suppressed or enhanced PMN, monocyte chemotaxis • Elevated antibody levels to several Gram-negative bacteria
Refractory periodontitis	• Reduced PMN chemotaxis

KEYPOINTS

1. Host defence is established at different levels.
2. Saliva acts as a vehicle for swallowing bacteria, inhibits the attachment of bacteria, and kills the bacteria by the peroxidase system, lysozyme and lactoferrin.
3. Functions of gingival epithelium are structural arrangement of epithelial cells, prevents bacterial entry, keratinization, resists trauma and also has a permeability barrier.
4. Complement functions are cellular activation, opsonization by C_{3b} and cytolysis (C_1-C_9).
5. The complement activation can be classical pathway with the sequence of C_1, C_4, C_2, C_3, C_5, C_6, C_7, C_8, C_9, alternative pathway C_3, C_5, C_6, C_7, C_8, C_9.
6. Various killing mechanisms of neutrophils are, non-oxygen-dependent mechanisms and oxidative mechanisms.
7. Macrophages phagocytose and kill bacteria, removes damaged host tissue during inflammation and also traps and presents the antigens to lymphocytes for induction of immune responses.
8. There are three types of lymphocytes, T-lymphocytes, B-lymphocytes and natural killer (NK) cells.



	Events	Protective mechanism
I	Microbes colonizing in the mouth	Inhibition of attachment by salivary antibody
	Continual Transmission of bacteria from environment	Washing effect of saliva
		Washing effect of crevicular fluid
II	Growth and division of bacteria in plaque	Detachment, phagocytosis and killing of bacteria by neutrophils
		Washing effect of gingival crevicular fluid
		Complement opsonization
III	Bacterial products diffuse into the tissues	Uptake and destruction by neutrophils and macrophages after binding to antibody
		Neutralizing or opsonizing antibody
		Outflow of crevicular fluid
		Epithelial barrier
IV	Bacteria entering into the tissues	Killing by complement, neutrophils and macrophages
		Opsonization by antibody, epithelial barrier
V	By stander tissue damage	
VI	Tissue repair and healing	

Fig. 8.11: Host defence against local and systemic infections of periodontal origin

KNOW MORE ...

Characteristics of inflammatory cells

Cells	*Features*	*Functions*
Granulocytes		
a. Neutrophils • Polymorphonuclear leukocytes	• Comprise 40-75% of total number of leukocytes • Diameter ranges from 10-15 µm • It has multilobed nucleus and granules containing proteases, myeloperoxidase, lysozyme, esterase, aryl sulfatase, acid and alkaline phosphatase.	• Forms first line of defense • Chemotaxis/cell mobilization • Phagocytosis • Oxygen/non-oxygen dependent killing of microorganisms.
b. Eosinophils	• Comprise 1-6% of total number of leukocytes • Measures about 10-12 µm in diameter • It has a bilobed nucleus • Consists of eosinophilic granules.	Involved in reactions to foreign proteins, antigen antibody reactions.
c. Basophils	• Comprise 1% of total number of leukocytes. • Diameter ranges from 10-15 µm • It has bilobed nucleus • Consists coarse basophilic granules.	Releases histamine and heparin through mast cells and mediates inflammatory reactions.
Agranulocytes		
d. Lymphocytes • B- lymphocytes • T- lymphocytes	• Comprise 20-45% of total number of leukocytes. • Lymphocytes in the peripheral blood measure about 9-12 µm in diameter. • It possesses a round or slightly indented nucleus with coarsely clumped chromatin network. • It has scanty basophilic cytoplasm. • Comprise 4-10% of total number of leukocytes	Involved in humoral immunity and cell-mediated immunity
e. Monocytes	• Largest mature leukocyte measuring 12- 20 µm. • It possesses a large central, oval, notched or indented or horse-shoe shaped nucleus. • It has abundant pale blue cytoplasm.	Functions through macrophages, same as that of neutrophils-comprise the second line of defense against microorganisms.

Immunoglobulins or Antibodies

These are glycoproteins which represent the components of adaptive immune system. These immunoglobulins (Ig) are produced by plasma cells in response to an antigen.

Structure

It is a "Y" shaped molecule, the tail end of the Y contains the ends of the heavy chains, "Fc fragment" i.e. complement binding site. The remainder of the "Y" contains light chains, "Fab" antibody binding site.

Functions of Immunoglobulins: IgG,A,M,D&E

Types of immunoglobulin	*Functions*
IgG	Only maternal immunoglobulin
Subclasses IgG1 to IgG4	Helps in phagocytosis by binding to microorganisms. Plays a role in most of the immunological reactions
IgA Subclasses IgA1, IgA2	Secreted in the body fluids. Attaches to the mucosal surfaces thus providing first line of defense against bacteria by preventing their penetration.
IgM	Provides primary immune response (synthesized in the fetus) More effective than IgG in opsonization and bactericidal action.
IgD	Unknown
IgE	Causes immediate hypersensitivity reactions.

REVIEW QUESTIONS

1. What are the function of saliva and gingival crevicular fluids?
2. Role of neutrophils in periodontal disease.
3. Describe the complement activation and functions of antibodies.

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9 Chapter

Trauma from Occlusion

PHYSIOLOGIC ADAPTIVE CAPACITY OF THE PERIODONTIUM TO OCCLUSAL FORCES

One must appreciate the dynamics of the periodontium to accommodate the forces exerted on the crown, which is called as *adaptive capacity*. This varies in different persons and in the same person at different times. This is mainly explained by four factors which mainly influence the effect of occlusal forces on the periodontium.

a. *Magnitude (the amount):* When it is increased the periodontium responds (a) with a thickening of the periodontal ligament, (b) an increase in the number and width of periodontal ligament fibers and (c) an increase in the density of the alveolar bone.
b. *Direction:* Changes in the direction causes reorientation of the stresses and strains within the periodontium (lateral or horizontal forces, torque or rotational forces are more likely to injure the periodontium).
c. *Duration:* Constant pressure on the bone is more injurious than intermittent forces.
d. *Frequency:* The more frequent the application of an intermittent force, the more injurious to the periodontium.

TRAUMA FROM OCCLUSION (TFO)

Definition and Terminology

According to Orban and Glickman et al (1968): Trauma from occlusion is defined as, when occlusal forces exceed the adaptive capacity of periodontal tissues, the tissue injury

results. This resultant injury is termed as trauma from occlusion.

WHO in 1978 defined trauma from occlusion as "damage in the periodontium caused by, stress on the teeth produced directly or indirectly by the teeth of the opposing jaw".

Other terms often used are, traumatizing occlusion, occlusal trauma, occlusal overload, periodontal traumatism, occlusal disharmony, functional imbalance and occlusal dystrophy. One must note that trauma from occlusion refers to the tissue injury, not the occlusal force. An occlusion that produces such an injury is called as traumatic occlusion.

Types

i. Depending on the onset and duration.
ii. Depending on the cause:
    a. Due to the alterations in the occlusal forces.
    b. Reduced capacity of the periodontium.
iii. Depending on the onset and duration:
    a. Acute trauma from occlusion (TFO).
    b. Chronic trauma from occlusion (TFO).

    Acute trauma from occlusion: Results from the abrupt changes in the occlusal forces, such as that produced by biting on a hard object, in addition, could also be due to iatrogenic factors (faulty restorations/prosthetic appliance).

    Chronic trauma from occlusion: As a result of the gradual changes produced in the periodontium due to the tooth wear, drifting movement, extrusion of the teeth combined with parafunctional habits such as bruxism and clenching.
iv. Depending on the cause (Table 9.1):

    Changes produced by primary trauma from occlusion are usually reversible, may be because, the supracrestal gingival fibers are not affected and thus prevents the apical migration of junctional epithelium.

In summary,

1. The criterion that determines whether an occlusion is traumatic is whether it produces periodontal injury, not how the teeth occlude.
2. Any occlusion that produces periodontal injury is considered traumatic.
3. Malocclusion is not necessary to produce trauma.

Signs and Symptoms

1. Clinical and,
2. Radiographic changes

Clinical Signs and Symptoms

a. *In acute situations:* Excessive tooth pain, tenderness on percussion, increased tooth mobility (hypermobility) is seen. In severe cases, periodontal abscess formation and cemental tears can be seen. Others such as presence of infrabony pockets, furcation involvement, attrition, pathologic migration may also be present.
b. *Fremitus test is positive.*
c. *Radiographic changes*
    i. Increase in the width of the periodontal ligament space often with thickening of the lamina dura along the lateral borders of the root, apical and bifurcation areas.
    ii. "Vertical" rather than horizontal destruction of the interdental septum.
    iii. Radiolucency and condensation of the alveolar bone.
    iv. Root resorption.

Histologic Changes

The response of tissues to increased occlusal forces is explained under three stages.

Table 9.1: Types of TFO: Depending on the cause

Primary trauma from occlusion	*Secondary trauma from occlusion*
It is a tissue injury, which is elicited around a tooth with normal height of periodontium. For example: Insertion of high fillings, insertion of the prosthetic replacement, orthodontic movement in functionally-unacceptable positions.	It is related to situations in which occlusal forces cause injury in a periodontium of reduced height. For example: Periodontitis.

- Stage 1: Injury
- Stage 2: Repair
- Stage 3: Adaptive remodeling of the periodontium.

Stage 1: Injury

When a tooth is exposed to excessive occlusal forces, the periodontal tissues are unable to withstand and hence they distribute, while maintaining the stability of the tooth. This may lead to certain well-defined reactions in the periodontal ligament and alveolar bone, eventually resulting in adaptation of the periodontal structures to altered functional demand. When the tooth is subjected to horizontal forces the tooth rotates or tilts in the direction of force. This tilting results in the pressure and tension zones, within the marginal and apical parts of the periodontium. Depending on the types of forces there can be many histologic changes (Table 9.2).

Stage 2: Repair

TFO stimulates increased reparative activity.

When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned-bony trabeculae with new bone. This attempt to compensate for lost bone is called buttressing bone formation which is an important feature of reparative process associated with trauma from occlusion (also occurs during inflammation or tumors).

Buttressing bone formation can occur within the jaw, called central buttressing and on the bone surface, called as peripheral buttressing. It usually occurs on the facial and lingual plates of the alveolar bone, if it produces a shelf-like thickening of alveolar bone it is referred as lipping.

Stage 3: Adaptive Remodeling of the Periodontium

If the repair process cannot keep pace with the destruction caused by occlusion, the periodontium may get remodelled in order to maintain the structural relationship. This may result in thickened periodontal ligament, angular defects in the bone with no pocket formation, loose teeth and increased vascularization.

In summary, histometric changes shown during these three stages are:

1. The injury phase shows an increase in areas of resorption and a decrease in bone formation.
2. The repair phase shows increase in areas of bone formation and decreased resorption.

Table 9.2: Histologic changes in periodontal tissues after injury

a.	*Slightly-excessive pressure* Changes are as follows: 1. Widening of the periodontal ligament. 2. Resorption of alveolar bone called as direct bone resorption. 3. The number of blood vessels are increased but the size is reduced.	*Slightly-excessive tension* 1. Elongation of periodontal ligament fibers. 2. Apposition of alveolar bone. 3. Blood vessels are enlarged and less.
b.	*Greater pressure* The changes in the tissues are as follows: 1. Compression of fibers producing areas of the hyalinization. 2. Injury to the cells like fibroblasts and other connective tissue cells leading to necrosis of areas of the ligament. 3. Changes in the blood vessels-breaking of vessel wall. 4. Increased resorption of alveolar bone. 5. Resorption of tooth surface.	*Greater/severe tension* 1. Causes widening of the periodontal ligament, thrombosis, hemorrhage, tearing of periodontal ligament. 2. Resorption of alveolar bone.
c.	*Pressure severe enough to force the root against bone* causes necrosis of periodontal ligament and bone. The bone is resorbed from viable periodontal ligament adjacent to necrotic areas and from marrow spaces, a process called undermining resorption or indirect bone resorption takes place. The furcation is the most susceptible area to injury due to excessive occlusal forces.	

3. Remodeling phase shows return of normal resorption and formation.

The animal experiments conducted to study the effect of traumatic occlusion on the periodontium had raised a lot of criticism. In these studies, the teeth were subjected to a unilateral horizontal force and the changes were observed. However, it has been suggested that in humans, the occlusal forces act alternatively in one and then in the opposite direction, which is termed as jiggling forces. It is important to note the difference in the forces because, unilateral horizontal force creates pressure and tension zones in the coronal and apical parts of the periodontium, whereas with jiggling forces pressure and tension zones occur on both sides of the jiggled tooth. Moreover, in humans the tooth rotates around a fulcrum which in single rooted teeth is located at the junction between the apical-third and the middle-third of the root. In multirooted teeth the fulcrum is located at the furcation area.

In the response to increased occlusal forces, the periodontal ligament gradually increases in width on both the sides of the tooth. This is associated with, (a) Inflammatory changes in the ligament tissue, (b) Active bone resorption. (c) Progressive mobility of the teeth. When the effect of the forces applied has been compensated by the increased width of the periodontal ligament space, the ligament no longer shows the above mentioned signs, except that the tooth remains hypermobile, but the mobility is no longer of the progressive type.

Other Properties

Effect of Insufficient Occlusal Force

It may also be injurious to periodontal tissues which results in the thinning of the periodontal, ligament, atrophy of the fibers, osteoporosis of alveolar bone and reduction in alveolar bone height. Hypofunction can result from an open bite relationship, absence of functional antagonists, or unilateral chewing habits.

Reversibility of Traumatic Lesion

Trauma from occlusion is reversible. When the injurious force is removed, the repair occurs. The presence of inflammation in the periodontium as a result of plaque accumulation may impair the reversibility of traumatic lesions.

Effect of Increased Occlusal Forces on Pulp

The effects on the pulp have not been established.

ROLE OF THE TRAUMA FROM OCCLUSION IN THE PROGRESSION OF PERIODONTAL DISEASE

The various studies conducted on animals and humans have shown changes produced by the pressure and tension sides of the tooth, with an increase in the width of the periodontal ligament and increased tooth mobility. None of these methods have caused gingival inflammation or pocket formation. This was explained by:

Glickman's concept (1965, 1967). He claimed that the pathway of the spread of plaque associated gingival lesion can be changed if the forces of an abnormal magnitude are acting on teeth harboring subgingival plaque. He has explained that, teeth which are nontraumatized exhibit suprabony pockets and horizontal bone loss, whereas teeth with trauma exhibit angular bony defects and infrabony pockets.

According to him, the periodontal structures are divided into two zones:

1. The zone of irritation and
2. The zone of co-destruction.

The zone of irritation includes the marginal and interdental gingiva, which is affected only by microbial plaque. In a plaque associated lesion, at a "nontraumatized" tooth, the inflammation spreads in an apical direction, first involving the alveolar bone and later the periodontal ligament area. Hence, there is an even (horizontal) bone loss.

The zone of codestruction includes the periodontal ligament, the root cementum and the alveolar bone, which are coronally demarcated by the transseptal and the dentoalveolar collagen fibers. The tissue in this region becomes the seat of a lesion caused by the trauma from occlusion. Here the spread of inflammation is from the zone of irritation directly down into the periodontal ligament and

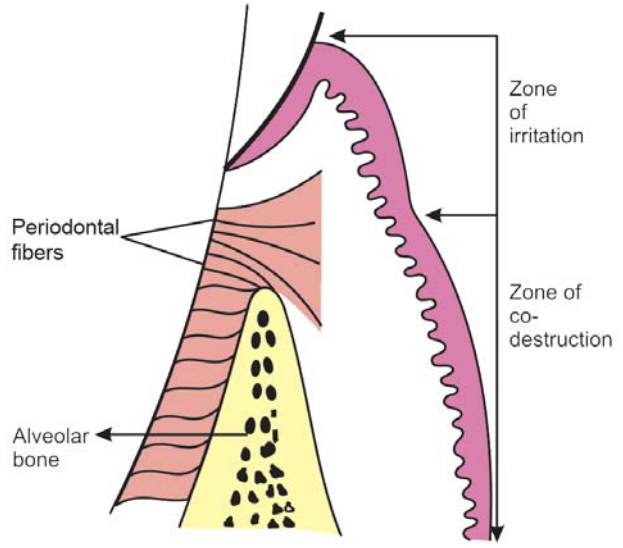
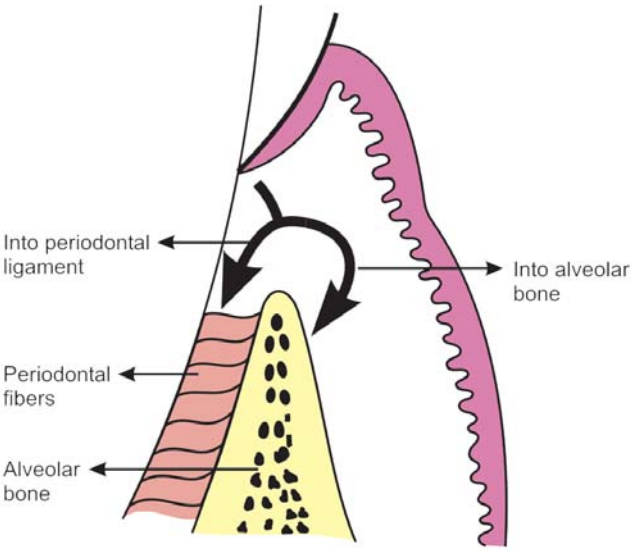


Fig. 9.1: Zone of irritation and zone of codestruction

hence angular bony defects with infrabony pockets are seen (Figs 9.1 and 9.2).

The summary of this concept is that trauma from occlusion is a codestructive factor of importance especially in situations where angular defects combined with infrabony pockets are found in one or several teeth.



The inflammatory lesion in the zone of irritation can, in teeth not subjected to trauma, propagate into the alveolar bone, while in teeth also subjected to trauma from occlusion, the inflammatory infiltrate spreads directly into periodontal ligament

Fig. 9.2: Pathway of inflammatory process

Waerhaug's concept: From his similar studies he concluded that angular defects and infrabony pockets occur often at periodontal sites of teeth not affected by trauma from occlusion. In other words, he refuted the hypothesis that trauma from occlusion played a role in the spread of a gingival lesion into the zone of codestruction. The loss of periodontium, according to Waerhaug was as a result of inflammatory lesions associated with subgingival plaque. He concluded that angular defects occur when the subgingival plaque of one tooth has reached a more apical level than the microbiota on the neighboring tooth, and when the volume of the alveolar bone surrounding the roots is comparatively large. This was also supported by Prichard (1965) and Manson (1976).

In conclusion, four possibilities can occur when a tooth with gingival inflammation is exposed to trauma.

1. Trauma from occlusion may alter the pathway of extension of gingival inflammation to the underlying tissues. Inflammation may proceed to the periodontal ligament rather than to the alveolar bone and the resulting bone loss would be angular with infrabony pockets.
2. It may favor the environment for the formation and attachment of plaque and calculus and may be responsible for development of deeper lesions.
3. Supragingival plaque can become subgingival if the tooth is tilted orthodontically or migrates into an edentulous area, resulting in the transformation of a suprabony pocket into an infrabony pocket.
4. Increased tooth mobility associated with trauma to the periodontium may have a pumping effect on plaque metabolites increasing their diffusion.

PATHOLOGIC TOOTH MIGRATION

Refers to tooth displacement that results when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease.

Pathologic migration occurs most frequently in the anterior region, but posterior teeth may also be affected. The teeth may move in any direction and the migration is usually accompanied by mobility and rotation. Pathologic migration in occlusal or incisal direction is called as "extrusion" (Figs 9.3 and 9.4).

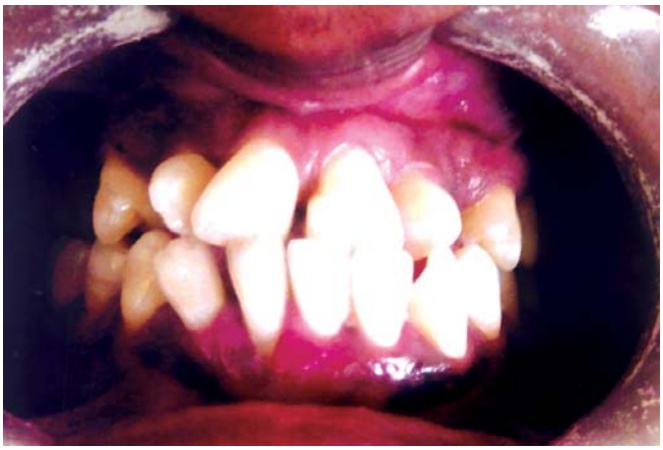


Fig. 9.3: Pathologic migration

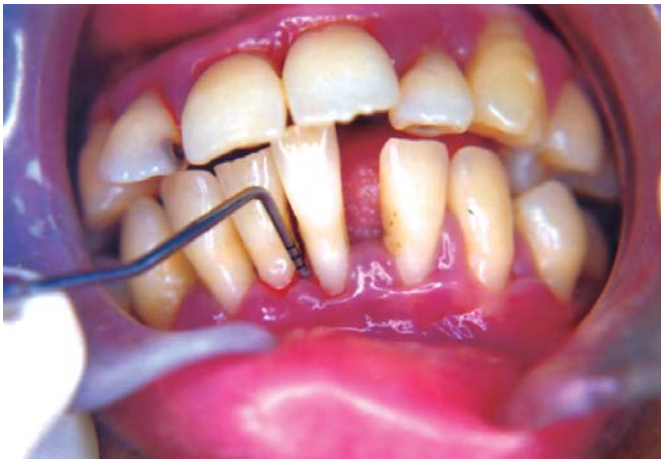


Fig. 9.4: Pathologic migration associated with tongue thrusting

Pathogenesis

Two major factors play a role in maintaining the normal position of the teeth.

1. The health and normal height of the periodontium.
2. The forces exerted on the teeth.

The Health and Normal Height of the Periodontium

A tooth with weakened periodontal support is unable to withstand the forces and moves away from the opposing force. It is important to understand that the abnormality in pathologic migration rests with the weakened periodontium. The force itself need not be abnormal. Forces that are acceptable to an intact periodontium become injurious when periodontal support is reduced. Pathologic migration may continue even after a tooth no longer contacts its antagonist.

Changes in the Forces Exerted on the Teeth

Changes in the forces may occur as a result of (a) unreplaced missing teeth, (b) Failure to replace first molars, or (c) other causes. These forces do not have to be abnormal to cause pathologic migration, if the periodontium is sufficiently weakened.

a. *Unreplaced missing teeth:* This leads to drifting of teeth into the spaces created by unreplaced missing teeth. Drifting differs from pathologic migration, in that it does not result from destruction of the periodontal tissues. However, it usually creates conditions that leads to periodontal diseases and thus the initial tooth movement is aggrevated by loss of periodontal support.
b. *Failure to replace first molars:* It consists of the following:
    1. The second and third molars tilt resulting in decrease in vertical dimension.
    2. The premolars move distally and the mandibular incisors tilt or drift lingually.
    3. Anterior overbite is increased.
    4. The maxillary incisors are pushed labially and laterally.
    5. The anterior teeth extrude due to disappearance of incisal apposition.
    6. Diastema is created by the separation of the anterior teeth.

OTHER CAUSES

1. *Pressure from the tongue:* It may either have a direct effect, that is, it may cause drifting of teeth in the absence of periodontal disease or may contribute to pathologic migration of the teeth with reduced periodontal support.
2. *Pressure from the granulation tissue of periodontal pocket:* It has also been listed as a contributing factor to pathologic migration. Usually tooth may return to their original position after pockets are treated, but if the destruction of the periodontium is more severe on one side of a tooth rather than the other, the healing tissue may pull the tooth in the direction of lesser destruction.
3. To summarize, trauma from occulsion does not have any:
    a. Effect on the supracrestal gingival tissue.

b. Human and animal studies have shown neither unilateral nor jiggling force can result in pocket formation.
c. Bone resorption and increased mobility in the absence of the pockets can be associated with trauma from occlusion.
d. In cases of teeth affected by periodontal disease, trauma from occlusion may aggravate the rate of progression of the disease and can also act as a co-factor in tissue destruction.

KEYPOINTS

1. When occlusal forces exceed the adaptive capacity of the periodontal tissues, tissue injury results. This resultant injury is termed as "trauma from occlusion".
2. Depending on the onset of duration trauma from the occlusion (TFO) may be divided into, acute trauma from occlusion, chronic trauma from occlusion. Depending on the cause, primary trauma from occlusion, secondary trauma from occlusion.
3. Primary trauma from occlusion is a tissue injury, which is elicited around a tooth with a normal height of periodontium whereas secondary trauma from occlusion is related to situations in which occlusal forces cause injury in a periodontium of reduced height.
4. Role of trauma from occlusion in the progression of periodontal disease is explained by two concepts, (a) Glickman's concept supports trauma from occlusion that is a co-destructive factor of importance, especially in situations where angular defects combined with infrabony pockets are found in one or several teeth where as Waerhaug's concept refutes this hypothesis and proposes that, angular defects and infrabony pockets occur often at sites not affected by trauma from occlusion. He proceeds to explain, that, angular defects occur, when the subgingival plaque of one tooth reaches a more apical level than the plaque on the neighboring tooth and also when the volume of the alveolar bone surrounding the roots is comparatively large.
5. Pathologic tooth migration refers to tooth displacement that results when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal diseases.

KNOW MORE ...

Various Types of Occlusal Forces

a. Normal or physiological occlusal forces which rarely exceeds 5N which is required to provide positive stimulus to maintain the periodontium.
b. Continuous forces/very low forces (e.g. orthodontic forces).
c. Impact forces-which are mainly high forces but of short duration, if it is beyond the adaptive capacity, may result in tooth fracture.
d. Jiggling forces are considered to be most traumatic, e.g. high fillings, premature contacts on crowns and fillings.

Clinical Indicators for Trauma from Occlusion

a. Mobility.
b. Fremitus test being positive.
c. Malocclusion.
d. Wear facets.
e. Tooth migration.
f. Fractured tooth/teeth.
g. Thermal sensitivity.
h. Muscle hypertonicity.

REVIEW QUESTIONS

1. Define trauma from the occlusion. Describe the clinical and radiographic changes associated with trauma from occlusion.
2. What is the role of trauma from occlusion in the progression of periodontal diseases?
3. What are the causes for pathologic tooth migration?
4. Differences between primary trauma from occlusion and secondary trauma from occlusion.

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10 Chapter

Role of Systemic Diseases in the Etiology of Periodontal Diseases

- DIETARY AND NUTRITIONAL ASPECTS OF PERIODONTAL DISEASE
- EFFECTS OF HEMATOLOGICAL DISORDERS ON PERIODONTIUM
- METABOLIC AND ENDOCRINE DISORDERS
- CARDIOVASCULAR DISEASES
- ANTIBODY DEFICIENCY DISORDERS
- OTHER SYSTEMIC DISEASES
- PSYCHOSOMATIC DISORDERS

INTRODUCTION

It is a well established fact that the primary etiological agent in periodontal disease is *bacterial plaque*. The toxins and enzymes produced by the bacterial plaque elicit inflammatory and immunologic changes in the periodontal tissues at both cellular and molecular levels. These responses can be affected by a variety of systemic factors that can alter the response of the tissue to plaque. Further more, certain systemic disorders can have a direct effect on the periodontal tissues and these represent the periodontal manifestations of systemic diseases. In general, these diseases do not initiate chronic destructive periodontitis but may accelerate its progression and increase tissue destruction.

DIETARY AND NUTRITIONAL ASPECTS OF PERIODONTAL DISEASE

The vitality of periodontal tissues, in both health and disease depends strongly on the essential nutrients. The epithelium of the dentogingival junction and the underlying connective tissues are the most dynamic tissues.

The majority of opinions and research findings point to the following:

1. Nutritional deficiencies produce changes in the oral cavity.
2. There are no nutritional deficiencies that by themselves cause gingivitis or periodontal pockets.

The Consistency of Diet

From the viewpoint of promoting and maintaining gingival and periodontal health, it is often stated that a firm and fibrous diet is more beneficial than an intake of soft and more loosely-textured food. Softer diets tend to produce greater deposits of plaque and an increase in plaque can be noticed, when the soft diet especially contains high proportion of sucrose. Diets that are predominantly fibrous are considered advantageous as they possess the ability to impart a natural cleansing action to the teeth and the periodontium.

A coarse diet, requires vigorous mastication and the plaque that forms approximately tends to be towards the cleansable buccal and lingual surfaces of the teeth. However, coarse and granular diets can predispose to a direct traumatic injury to the supporting tissues.

Protein Deficiency and Periodontal Disease

Proteins are constituents of the organic matrices of all the dental tissues including the alveolar bone. The integrity of the periodontal ligament is also dependent upon proteins (amino acid). Studies have indicated that on deprivation of proteins, extreme pathologic changes occur and there is marked degeneration of periodontal support.

Vitamins and Periodontal Disease

Vitamins are essential, biologically-active constituents of a diet which cannot be replaced by other dietary components.

Vitamin C

Its deficiency in humans results in scurvy, a disease characterized by hemorrhagic diathesis and retardation of wound healing.

Clinical Manifestations

1. Increased susceptibility to infections.
2. Impaired wound healing.
3. Bleeding and swollen gums.
4. Mobile teeth.

Histopathological Features

1. Defective formation and maintenance of collagen.
2. Retardation or cessation of osteoid formation and impaired osteoblastic function.
3. Increased capillary permeability.
4. Susceptibility to traumatic hemorrhage.
5. Hyporeactivity of contractile elements of the peripheral blood vessels.
6. Sluggishness of blood flow.

Possible Etiologic Relationships between Ascorbic Acid and Periodontal Disease

1. Low levels of Ascorbic acid influences the metabolism of collagen within the periodontium, thereby affecting the ability of the tissue to regenerate and repair by itself.
2. It interferes with bone formation leading to the loss of the alveolar bone.
3. Increases the permeability of oral mucosa to tritiated endotoxin and inulin.
4. Increased levels of Ascorbic acid enhances both the chemotactic and migratory action of leukocytes without influencing phagocytic activity.
5. Optimal level of Ascorbic acid is required to maintain the integrity of the periodontal microvasculature as well as the vascular response to bacterial irritation and wound healing.
6. Depletion of vitamin C may interfere with the ecologic equilibrium of bacteria in plaque and increases its pathogenicity.

Periodontal Features of Scurvy

The oral symptoms are that of chronic gingivitis which can involve the free gingiva, attached gingiva and alveolar mucosa. In severe cases, the gingiva becomes brilliant-red, tender and grossly swollen. The spongy tissues are extremely hyperemic and bleed spontaneously. In long standing cases, the tissues attain a dark blue or purple hue. Alveolar bone resorption with increased tooth mobility has also been reported.

Vitamin D Deficiency

Vitamin D is essential for the absorption of calcium from the gastrointestinal tract and the maintenance of calcium-phosphorus balance.

Radiographically, there is a generalized partial to complete disappearance of the lamina dura and reduced density of supporting bone, loss of trabeculae, increased radiolucency of the trabecular interstices and increased prominence of the remaining trabeculae.

Vitamin E

Evidence suggests that vitamin E acts as a antioxidant and plays an important role in maintaining the stability of cell membranes and protecting blood cells against hemolysis.

The possible role is based upon its ability to interfere with the production of prostaglandins.

Vitamin A

It is essential for normal functions of the retina, for growth, differentiation and maintenance of epithelial tissues and for bone growth and embryonic development.

Vitamin B-Complex

Oral disease is rarely due to a deficiency in just one component of the B-complex group. Oral changes common to—Vitamin B-complex deficiencies are gingivitis, glossitis, glossodynia, angular cheilitis and inflammation of the entire oral mucosa.

EFFECTS OF HEMATOLOGICAL DISORDERS ON PERIODONTIUM

Disorders of the blood and blood forming tissue can have a profound effect on the periodontal tissues and their response to bacterial plaque. The WBC disorders have the most pronounced effect on the periodontal tissues. Disorders of hemostasis can be classified according to the underlying defect. There can be a defect in the vascular constriction, platelet adhesion and aggregation, coagulation and fibrinolysis.

White Blood Cell Disorders

The WBC's disorders that affect the periodontium can be categorized as either a disorder of numbers or defect in function.

Neutropenias

a. Cyclic neutropenia.
b. Chronic benign neutropenia of childhood.
c. Benign familial neutropenia.
d. Severe familial neutropenia.
e. Chronic idiopathic neutropenia.

Cyclic Neutropenia

It is characterized by a cyclic depression of the PMN count in peripheral blood. The cyclic intervals are usually between 19 and 21 days. Clinical problems include pyrexia, oral ulceration and skin infections.

Periodontal manifestations include oral ulceration, inflamed gingiva, rapid periodontal breakdown, and alveolar bone loss. Bone loss is most obvious around the lower incisors and first permanent molars.

Treatment: Plaque control, supportive measures like antiseptic mouth wash, antimicrobial therapy has been proposed.

Chronic Benign Neutropenia of Childhood

The onset is usually between 6 to 20 months of age and in most patients, the condition is self-limiting.

The main periodontal feature is a bright-red, hyperplastic, edematous gingiva confined to the width of attached gingiva. The gingival tissues exhibit bleeding on probing and show areas of desquamation, varying degrees of gingival recession and pocketing are seen.

Treatment: Appropriate antimicrobial agent should be prescribed.

Benign Familial Neutropenia

It is transmitted as an autosomal dominant trait. The *periodontal manifestations* include hyperplastic gingivitis exhibiting edematous and bright-red appearance. There is marked bone loss around the first molars. The gingival tissues bleed profusely on probing.

Treatment: Plaque control and use of antimicrobial mouth washes.

Chronic Idiopathic Neutropenia

There is a persistent neutropenia from birth and is not cyclical. Clinical symptoms include persistent recurrent infections throughout the patient's life.

Periodontal manifestations include persistent severe gingivitis. The gingiva is cherry-red edematous and hypertrophic with occasional desquamation.

Treatment: Strict oral hygiene program, scaling and regular prophylaxis. Antiseptic irrigation and antibiotic prophylaxis are advisable before tissue manipulation.

Leukemia

It is a malignant disease caused by proliferation of WBC forming tissue, especially those in bone marrow. Acute leukemia is more frequent in people under 20 years of age. Chronic leukemia's occur in people over 40 years of age.

Periodontal Manifestations

The major manifestations being gingival enlargement, gingival bleeding and periodontal infections. The incidence and severity of these problems varies according to the type and nature of leukemia (Fig. 10.1).

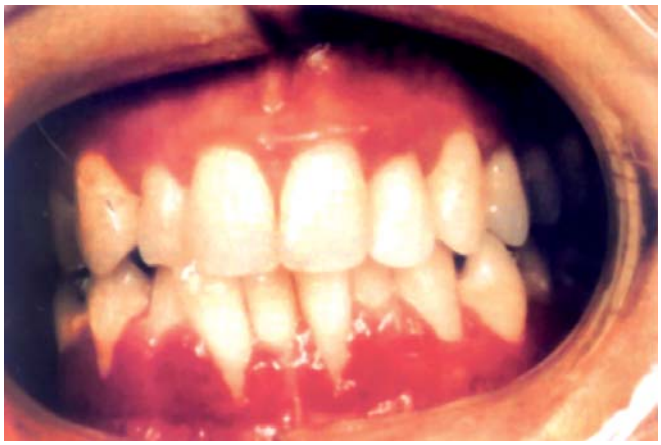

Fig. 10.1: Gingival changes associated with leukemia

a. Gingival enlargement is primarily due to a massive leukemic cell infiltration into the gingival connective tissue. The enlarged gingiva will hinder mechanical plaque removal; hence there will be an inflammatory component enhancing this enlargement.
b. Gingival bleeding is a common oral manifestation of acute leukemia. The bleeding is secondary to thrombocytopenia that accompanies leukemia.
c. Infections of the periodontal tissues secondary to leukemia can be of two types, either an exacerbation of an existing periodontal disease or an increased susceptibility of the periodontium to fungal, viral or bacterial infections.

Treatment Plan for Leukemic Patients

1. Refer the patient for medical evaluation and treatment.
2. Prior to chemotherapy, a complete periodontal plan should be developed.
   a. Monitor hematologic laboratory values.
   b. Administer suitable antibiotics before any periodontal treatment.
   c. Periodontal treatment consists of scaling and root planing, twice daily rinsing with 0.12 percent chlorhexidine gluconate is recommended. If there is irregular bleeding time, careful debridement with cotton pellets soaked in 3 percent hydrogen peroxide is performed.
3. During the acute phases of leukemia:
   a. Cleanse the area with 3 percent hydrogen peroxide (H_2O_2) or 0.12 percent chlorhexidine.
   b. Carefully explore the area and remove any etiologic local factors.
   c. Re-cleanse the area with 3 percent H_2O_2.
   d. Place a cotton pellet soaked in thrombin against the bleeding point.
   e. Cover with gauze and apply pressure for 15 to 20 minutes.
   f. Acute gingival or periodontal abscesses are treated by systemic antibiotics, gentle incision and drainage or by treating with 3 percent H_2O_2/0.12 percent chlorhexidine gluconate.
   g. Oral ulcerations should be treated with antibiotics and bland mouth rinses.
4. In patients with chronic leukemia, scaling and root planing can be performed but periodontal surgery should be avoided. Plaque control and frequent recall visits should receive particular attention.

Thrombocytopenic Purpura

It is characterized by a low platelet count, a prolonged clot retraction and bleeding time, and a normal or slightly-prolonged clotting time.

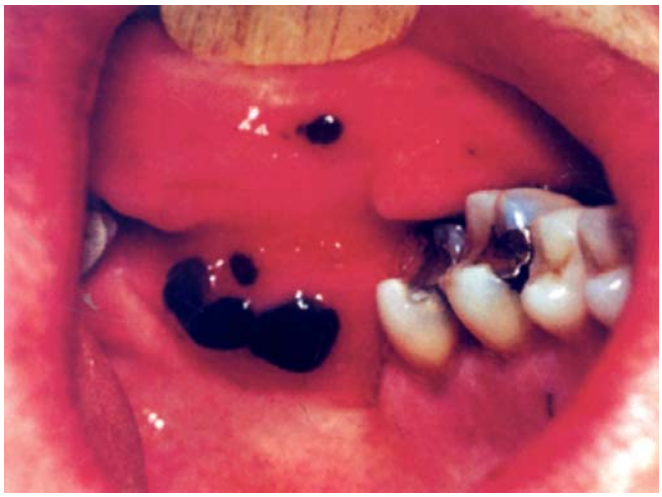

Fig. 10.2: Thrombocytopenic purpura with petechiae and hemorrhagic vesicles in the lining mucosa

Clinical manifestations include spontaneous bleeding into skin or from mucous membranes. Petechiae and hemorrhagic vesicles occur in the oral cavity. Gingiva is swollen, soft and friable. Bleeding occurs spontaneously (Fig. 10.2).

Treatment

1. Physician referral for a definitive diagnosis.
2. Oral hygiene instructions.
3. Prophylactic treatment of potential abscesses.
4. No surgical procedures are indicated unless platelet count is at least 80,000 cells/mm^3.
5. Scaling and root planing may be carefully performed at low platelet levels.

If surgery is indicated, it should be as atraumatic as possible, stents or thrombin-soaked cotton pellets placed interproximally, gentle hydrogen peroxide mouth washes and close postsurgical follow-up is recommended.

Disorders of WBC Function

Chédiak-Higashi Syndrome

It is a rare familial and often fatal disease which is transmitted as an autosomal recessive trait. PMNL's from patients with this syndrome show defective migration, defective chemotaxis, failure of postphagocytic degranulation and diminished intracellular bactericidal capacity.

Severe gingival inflammation appears to be a common finding in Chédiak-Higashi syndrome. The nature of the inflammatory changes may be plaque induced, secondary to infection or related to the underlying PMNL's defect.

Lazy Leukocyte Syndrome

The feature of the syndrome is a defect in leukocyte chemotaxis and random mobility. Marked gingivitis has also been described.

Chronic Granulomatous Disease

A genetically-transmitted disorder characterized by the inability of phagocytic cells to destroy certain infecting microorganisms.

Periodontal manifestations include marked, diffuse, gingivitis with an accompanying ulceration of buccal mucosa.

Red Blood Cell Disorders

There are many types of red blood cell disorders but only a few appear to have any effect on the periodontal tissues.

Aplastic Anemia

It is a bone marrow disorder characterized by a reduction in hematopoietic tissue, bone marrow is replaced with fat, and

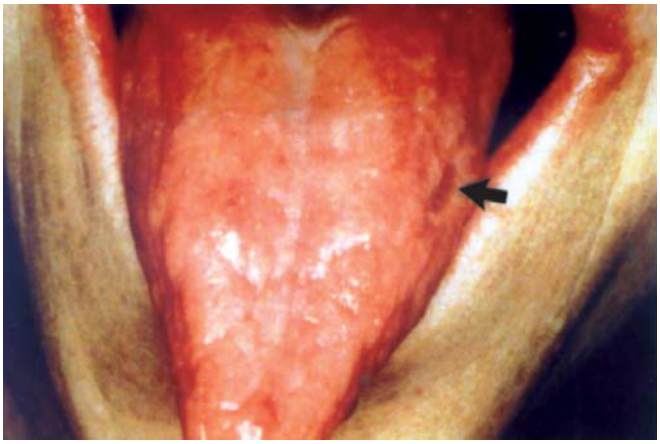

Fig. 10.3: Pernicious anemia

pancytopenia. Bleeding from the gingival margins appears to be a feature in these cases.

Fanconi's Anemia

This is a rare type of aplastic anemia characterized by a familial bone marrow hypoplasia that becomes manifested in the first decade of life. The periodontal manifestations being loss of several teeth, severe bone loss with pocketing greater than 10 mm. The gingiva will be bluish-red, bleed on probing, and shows suppuration on gentle pressure.

Sickle Cell Anemia

In this condition, the red blood cells undergoes sickling when subjected to hypoxia. Hence patients with sickle cell anemia are susceptible to infections. In some patients with sickle cell anemia, periodontal disease may provide a sufficient inflammatory response to precipitate a sickling crisis.

Acatalasia

It is caused by a lack of the enzyme catalase in many cells, especially the red blood cells and leukocytes. It causes hypoxia and necrosis of the gingival tissues. Severe periodontal destruction and gingival necrosis are seen.

METABOLIC AND ENDOCRINE DISORDERS

The endocrine glands produce hormones that control metabolism and maintain hemostasis. Diabetes mellitus is the main endocrine disorder that affects the periodontium. The sex hormone can alter the response of periodontal tissues to plaque. Disorders of the pituitary, thyroid and adrenal glands have little direct effect on the periodontal structures or in altering the host response to bacterial plaque.

Diabetes Mellitus and Periodontal Disease

Studies suggested that diabetic patient is more susceptible to periodontal breakdown, which is characterized by extensive bone loss, increased tooth mobility, widening of periodontal ligament space, suppuration and abscess formation (Fig. 10.4).

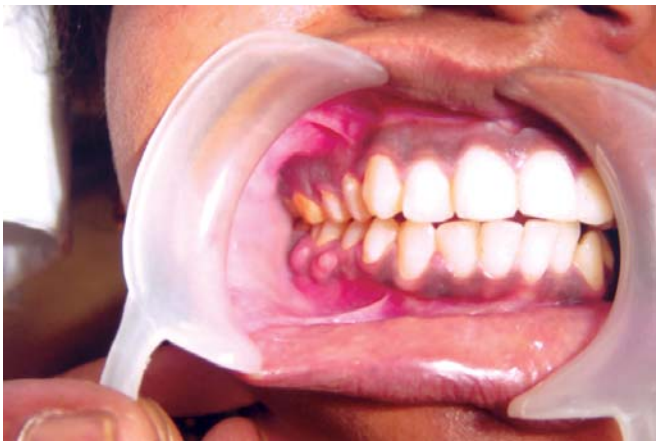

Fig. 10.4: Diabetic patient—multiple periodontal abscess

Pathogenesis

There are several underlying factors that accompany diabetes mellitus which may account for the apparent increased prevalence of periodontal disease in this condition. These factors can be considered under the following headings.

1. *Vascular changes*: Changes include thickening and hyalinization of vascular walls, PAS-positive, diastase-resistant thickening of capillary basement membranes, swelling and occasional proliferation of the endothelial cells, and splitting of capillary basement membrane. Diabetic-induced changes in the capillary basement membrane may have an inhibitory effect on the transport of oxygen, white blood cells, immune factors and waste products, all of which could affect tissue repair and regeneration.
2. *PMNL's function:* Impairment of PMN function is a feature of diabetes mellitus. Disorders include reduced phagocytosis and intracellular killing, impaired adherence and impaired chemotactic response. Suggested causes include inhibition of the glycolytic pathway with the PMNL's, abnormal cyclic nucleotide metabolism, which disrupts the organization of microtubules and microfilaments, or a reduction in leukocyte membrane receptors.
3. *Biochemistry of crevicular fluid:* Alterations in the constituents and flow rate of crevicular fluid have been

shown to be associated with diabetes. Cyclic AMP levels seems to be reduced in the diabetes group when compared with control.

4. *Changes in plaque microflora:* Studies have indicated that proteolytic activity has not been altered but hyaluronidase activity is lower in plaque from diabetes.

Treatment

a. Periodontal treatment in patient with uncontrolled-diabetes is contraindicated.
b. If suspected to be a diabetic, following procedures should be performed:
    1. Consult the patient's physician.
    2. Analyze laboratory tests, fasting blood glucose, post-prandial blood glucose, glycated hemoglobin, glucose tolerance test (GTT), urinary glucose.
    3. If there is periodontal condition that requires immediate care, prophylactic antibiotics should be given.
    4. If patient is a 'brittle' diabetic, optimal periodontal health is a necessity. Glucose levels should be continuously monitored and periodontal treatment should be performed when the disease is in a well-controlled state. Prophylactic antibiotics should be started 2 days preoperatively, Penicillin is the drug of first choice.

Guidelines

1. Clinician should make certain that the prescribed insulin has been taken, followed by a meal. Morning appointments are ideal, after breakfast, because of optimal insulin levels.
2. After any surgical procedures, postoperative insulin dose should be altered.
3. Tissues should be handled as atraumatically and as minimally (less than 2 hours) as possible. For anxious patients, if preoperative sedation is required, epinephrine concentration should not be greater than 1:1,00,000.
4. Diet recommendation should be made.
5. Antibiotic prophylaxis is recommended for extensive therapy.
6. Recall appointments and fastidious home oral care should be stressed.

Thyroid Gland

Hypothyroidism leads to cretinism in children and myxedema in adults. There are no notable periodontal changes.

Treatment

1. Patients with thyrotoxicosis and those with inadequate medical management should not receive periodontal therapy until the condition is stabilized.
2. Medications such as epinephrine, atropine and other pressor amines should be given with caution.
3. Hypothyroid patients require careful administration of sedatives and narcotics because of their diminished ability to tolerate drugs.

Pituitary Gland

Hyperpituitarism causes enlarged lips; localized areas of hyperpigmentation are seen along nasolabial folds. It is also associated with food impaction and hypercementosis is seen.

Hypopituitarism leads to crowding and malposition of teeth.

Parathyroid Glands

Parathyroid hypersecretion produces generalized demineralization of the skeleton. Oral changes include malocclusion and tooth mobility, radiographic evidence of alveolar osteoporosis, widening of the periodontal space and absence of lamina dura.

Treatment: Routine periodontal therapy must be instituted but the dental practitioner must be attuned to the oral and dental changes that occur.

Gonads

There are several types of gingival diseases in which modification of the sex hormones is considered to be either an initiating or complicating factor; gingival alterations are associated with physiologic hormonal changes with a predominant marked hemorrhagic tendency.

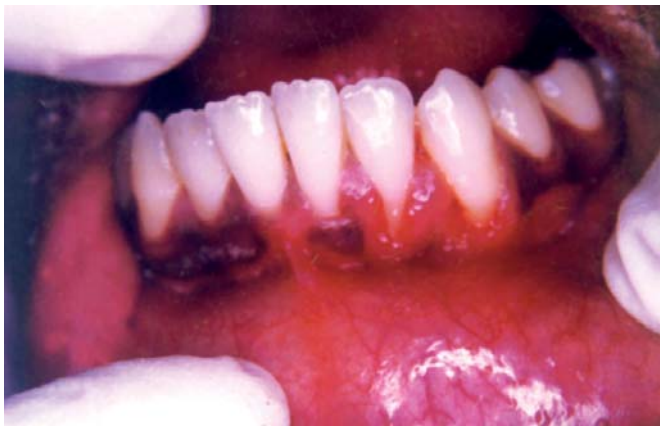

Fig. 10.5: Gingivitis in puberty with edema, discoloration and gingival enlargement

Gingivitis in Puberty (Fig. 10.5)

Pronounced inflammation, bluish-red discoloration, edema and enlarged gingiva may be seen.

Treatment: It is treated by scaling and curettage, removal of all sources of irritation and plaque control. In severe cases, surgical removal of enlarged tissue may be required.

Gingival Changes Associated with Menstrual Cycle

There is increased prevalence of gingivitis, bleeding gingiva. Exudation from inflamed gingiva is also increased, but the crevicular fluid flow is not affected. The salivary bacterial count is increased. No active treatment is required.

Gingival Diseases in Pregnancy (Fig. 10.6)

Pregnancy accentuates the gingival response to plaque. The severity of gingivitis is increased during pregnancy, beginning in the second or third month. It becomes more severe by the eighth month and decreases during ninth month.

Clinical Features

1. Pronounced base of bleeding.
2. Gingiva is bright-red to bluish-red.
3. Marginal and interdental gingiva is edematous, pits on pressure and sometime presents raspberry-like appearance.
4. It has been suggested that during pregnancy there is depression of maternal T-lymphocyte response.
5. Aggravation of gingivitis has been attributed principally to increased levels of progesterone which produces dilatation and tortuosity of the gingival microvasculature, circulatory stasis and increased susceptibility to mechanical irritation.
6. Increased crevicular fluid flow, pocket depth and mobility are also seen.

Treatment: Requires elimination of all local irritants that are responsible for precipitating gingival changes. Marginal and interdental gingival inflammation and enlargement are treated with scaling and root planing.

Treatment of tumor-like gingival enlargements consists of surgical excision, scaling and planing of tooth surfaces. In pregnancy emphasis should be on:

- Preventing gingival disease before it occurs.
- Treating existing gingival disease before it becomes worse.

Menopausal Gingivostomatitis

It occurs during menopause or in the postmenopausal period. Clinical manifestations include dry, shiny oral mucosa, dry burning sensation of oral mucosa, abnormal taste sensation described as salty, peppery or sour.

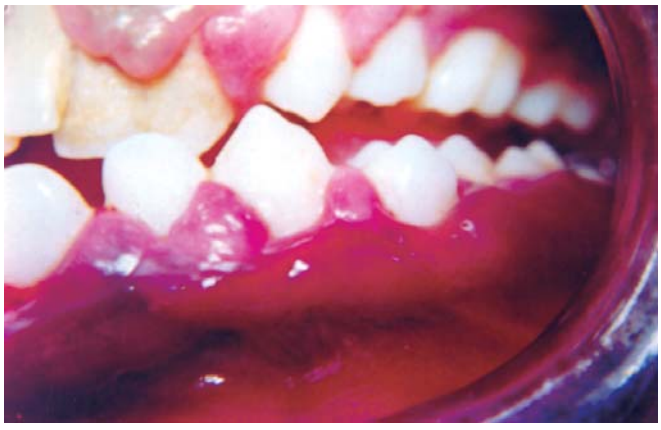

Fig. 10.6: Gingiva in pregnancy showing edema, discoloration and enlargement

CARDIOVASCULAR DISEASES

Arteriosclerosis

In aged individuals, arteriosclerotic changes in the blood vessels are characterized by, initial thickening, narrowing of lumen, thickening of media, and hyalinization of media and adventitia, with or without calcification are common.

Congenital Heart Disease

In cases of tetralogy of fallot, oral changes include a purplish-red discoloration of the lips and gingiva and sometimes severe marginal gingivitis and periodontal destruction. The tongue appears coated, fissured and edematous and there is extreme reddening of the fungiform and filliform papillae.

ANTIBODY DEFICIENCY DISORDERS

Acquired Immunodeficiency Syndrome

It is caused by a persistent HIV virus and is characterized by destruction of lymphocytes, rendering the patient susceptible to opportunistic infections including destructive periodontal lesions.

Clinical Manifestations

HIV gingivitis: In HIV gingivitis persistent, linear, easily bleeding, erythematous gingivitis has been described. Linear gingivitis lesions may be localized or generalized in nature. The erythematous gingivitis may be limited to marginal tissue, or extend into attached gingiva in a punctuate or a diffuse erythema or extend into alveolar mucosa. A severely destructive, acutely painful necrotizing ulcerative stomatitis has been reported.

HIV periodontitis: NUP (Necrotizing ulcerative periodontitis) is characterized by soft tissue necrosis and rapid periodontal destruction that results in marked interproximal bone loss. It is severely painful at onset.

Treatment: Recommended management for linear gingival erythema is as follows:

a. Instruct the patient to perform meticulous oral hygiene.
b. Scale and polish affected areas and perform subgingival irrigation with chlorhexidine.
c. Prescribe chlorhexidine gluconate mouth rinse.
d. Reevaluation and frequent recall visits.
e. Systemic antibiotics such as metronidazole or amoxicillin should be prescribed for patients with moderate to severe tissue destruction. Use of prophylactic antifungal medication should be considered.

In summary, treatment for necrotizing ulcerative stomatitis includes prescription of an antibiotic such as metronidazole or amoxicillin and use of an antimicrobial mouth rinse.

Treatment for necrotizing ulcerative periodontitis includes local debridement, scaling and root planing, irrigation with Betadine solution and establishment of meticulous oral hygiene, including home use of antimicrobial rinses. In severe NUP, antibiotic therapy is a must (metronidazole 400 mg thrice daily for 5 to 7 days).

OTHER SYSTEMIC DISEASES

Ingestion of metals such as mercury, lead, bismuth may result in oral manifestations owing to their intoxication or absorption without evidence of toxicity.

Bismuth Intoxication

Chronic bismuth intoxication is characterized by gastrointestinal disturbances, nausea, vomiting and jaundice

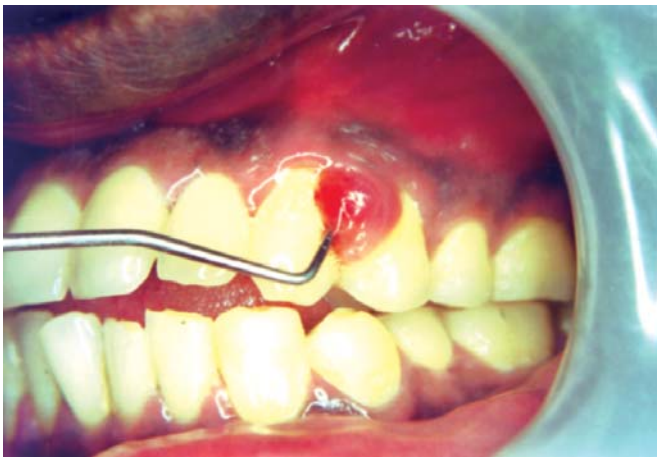

Fig. 10.7: Gingiva in pregnancy with localized gingival enlargement

as well as by an ulcerative gingivostomatitis. Generally pigmentation is accompanied by a metallic taste and a burning sensation of the oral mucosa. The tongue may be sore and inflamed. Urticaria, exanthematous eruptions of different types, bullous and purpuric lesions are among the dermatologic lesions that are attributed to bismuth intoxication.

It usually appears as a narrow, bluish-black discoloration of the gingival margin in areas of pre-existent gingival inflammation.

Lead Intoxication

Lead is slowly absorbed and toxic symptoms are not particularly definitive when they do occur. Among the oral signs are increased salivation, coated-tongue, peculiar sweetish taste, gingival pigmentation and ulceration. The pigmentation of gingiva is linear (Burtonian line), steel gray and associated with local irritation.

Mercury Intoxication

Gingival pigmentation in linear form results from the deposits of mercuric sulfide. The chemical also acts as an irritant which accentuates the pre-existing inflammation and commonly leads to notable ulceration of the gingiva and adjacent mucosa and destruction of underlying bone.

Other Chemicals

Phosphorus, arsenic, chromium, may cause necrosis of the alveolar bone with loosening and exfoliation of teeth.

PSYCHOSOMATIC DISORDERS

There are two ways by which psychosomatic disorders may be induced in the oral cavity, through the development of habits injurious to the periodontium and by the direct effect of the autonomous nervous system on the physiologic tissue balance.

However, under the conditions of mental and emotional stress, the mouth may subconsciously become an outlet for the gratification of basic drives in the adult. Gratification may be derived from neurotic habits, which are potentially injurious to the periodontium.

KNOW MORE ...

Diabetes and Periodontium

Various other factors contributing to the development of periodontal diseases:

The hyperglycemic state can result in:

- Reduced polymorphonuclear leukocyte (PMNL) function.
- Altered collagen metabolism which is associated with increased collagenase activity and decreased collagen synthesis.
- Excessive levels of advanced glycation end products (AGEs).
- Colonization of pathogenic subgingival flora.

REVIEW QUESTIONS

1. Describe the influence of systemic diseases on periodontium.
2. Explain the role of diabetes in periodontal disease.

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11 Chapter

Oral Malodor

Srinivas K

INTRODUCTION

Halitosis is a term used to describe noticeably unpleasant odor exhaled in breathing. It is a general term used to describe the unpleasant breath regardless of its sources, oral or non-oral. Whereas, oral malodor is the term especially used to describe the odor emanating from the oral cavity. Halitosis which is in synonym with breath malodor, foul breath and fetor oris or simply bad breath, affects a large proportion of population which may cause a significant social or psychological handicap to those suffering from it. This common disease has been ignored for too long by periodontologists even though the most common cause is related to the microbiota of the subgingival areas and the related tongue coating. The intensity of the bad breath differs during the day, (which may be due to the stress or fasting), eating certain foods (such as garlic, onions, meat, fish and cheese), smoking and alcohol consumption. Because the mouth is dry and inactive during the night, the odor is usually worse upon awakening (morning breath). Bad breath may also be persistent (chronic bad breath) which is a more serious condition, affecting at least 25% of population in varying degrees.

CLASSIFICATION OF HALITOSIS

Genuine Halitosis

- Physiologic halitosis
- Pathologic halitosis
  - Oral
  - Extraoral

Is an obvious malodor, with intensity beyond socially acceptable level is perceived.

Pseudohalitosis

Obvious malodor is not perceived by others, although the patient stubbornly complains of its existence, condition can be improved by counseling and simple oral hygiene measures.

Halitophobia

After treatment for genuine halitosis or pseudo-halitosis, the patient persists in believing that he/she has halitosis.

ETIOLOGY

At least 90% of all malodor originates from the oral cavity, whereas, the remaining 10% has systemic or local causes. Oral malodor is commonly the result of microbial putrefaction of food debris, cells, saliva and blood within the oral cavity. In particular, proteolysis of proteins to peptides and amino acids takes place. The resultant substrates with free **thiol** groups such as **cystein** and reduced **glutathionine**, rises to volatile sulfur compounds (VSCs), which are malodour substances. The most common physiological and pathological causes of halitosis are discussed below:

Causes for Physiologic Halitosis

a. Mouthbreathing.
b. Medications.
c. Aging and poor dental hygiene.
d. Fasting/starvation.
e. Tobacco.
f. Foods (onion, garlic, etc.) and alcohol.

Causes for Pathologic Halitosis

Oral and other contributing factors such as:

a. Periodontal infection: Odor from subgingival dental biofilm. Specific diseases like acute necrotizing ulcerative gingivitis and pericoronitis.
b. Tongue coating harbors microorganisms.
c. Stomatitis, xerostomia.
d. Faulty restorations retaining food and bacteria.
e. Unclean dentures.
f. Oral pathologic lesions like oral cancers, candidiasis.
g. Parotitis, cleft palate.
h. Aphthous ulcers, dental abscesses.

Systemic and Extraoral Factors

a. Nasal infections like rhinitis, sinusitis, tumors and foreign bodies.
b. Diseases of gastrointestinal tract (GIT) like hiatus hernia, carcinomas, Gastroesophageal Reflux Disorder (GERD).
c. Pulmonary infections like bronchitis, pneumonia, tuberculosis, and carcinomas.
d. Certain hormonal changes that occur during ovulation, menstruation, pregnancy and menopause.
e. Systemic diseases like diabetes mellitus, hepatic failure, renal failure, uremia, blood dyscrasias, rheumatologic diseases, dehydration and fever, cirrhosis of liver.

DIAGNOSIS OF HALITOSIS

a. **Review of Medical, Dental and Personal History.**
b. **Clinical examination:**
    i. *Intraoral examination:*
        1. Tongue coating.
        2. Evidence of mouthbreathing.
        3. Xerostomia: Dry mucosa.
        4. Other oral causes.
    ii. *Complete periodontal examination:*
        1. General personal care, state of oral hygiene.
        2. Probing for attachment levels, probing depths (Periodontal status).
        3. Evidence of neglect; past history of dental hygiene care.
c. **Measurement of oral malodor:** Patients should be instructed not to eat, chew, rinse or smoke for at least two hours before examination. Patients who are on antibiotics should be seen 2 weeks after discontinuation of medicines. The tests used to detect halitosis are as follows:
    i. *Subjective organoleptic method***:** This has been used as a bench mark for oral malodor measurement.
    ii. *Gas chromatography:* In order to assess oral malodor objectively, a portable industrial monitor

has been developed. These machines are specifically designed to digitally measure molecular levels of the three major Volatile Sulfur Compounds (VSCs) in a sample of mouth air (**hydrogen sulfide, methyl mercaptan and dimethyl sulfide)**. It is accurate in measuring the sulfur components of the breath and produces visual results in graph form via computer interface (Fig. 11.1).

iii. *Halimeters:* These machines measure the level of sulfide gas found in a persons breath. But it has certain drawbacks in clinical applications, some of the common sulfides such as mercaptan are not easily recorded and can be misrepresented in test results. The Halimeter is also very sensitive to alcohol, so one should avoid drinking alcohol or using alcohol containing mouth-washes for at least 12 hours prior to being tested (Figs 11.2A and B).

iv. *BANA Test (N-benzoyl-dL-arginine-2-naphthylamide)*: Some of the bacteria like *P. gingivalis, T. denticola* and *B. forsythus* produce waste products that are quite odiferous and as a result contribute in causing bad breath. These bacteria in question have the characteristic of being able to produce an enzyme that degrades the compound N-benzoyl-dL-arginine-2naphthylamide. When a sample of patient's saliva that contains these bacteria is placed within the BANA testing compound they cause it to breakdown. As a result of this degradation the test compound changes its color indicating a positive reaction.

v. *Chemiluminescence:* This test involves mixing a sample containing sulfur compound (VSCs) with the mercury compound and the resultant reaction causes fluorescence. This test is highly sensitive as it can measure even the low levels of sulfur compounds in the sample, which is in contradiction to testing with a halimeter.

Fig. 11.1: Gas chromatograph

A

B

Figs 11.2A and B: Halimeters

TREATMENT AND MANAGEMENT OF ORAL MALODOR

Treatment of oral malodor is a step-by-step problem solving procedure. Before commencing the treatment a clinician

must determine the source of malodor. The simplest way to distinguish oral from non-oral origin is to compare the smell from mouth and nose. If the origin is nasal or due to any other medical etiology they must be referred to a concerned specialist. The odor generating from the mouth often requires dental treatment. There are no standard and accepted protocols for the treatment of oral malodor, however, the possible protocols contains the basic elements including standard dental and periodontal treatment.

For genuine halitosis with oral causes, the treatment is as follows:

a. Reduction of anaerobic load by improving oral hygiene and periodontal health through basic dental care and if necessary incorporate advanced hygiene methods including oral irrigation and sonic or ultrasonic tooth brushes.
b. If oral malodor persists in spite of adequate conventional oral hygiene, tongue brushing should be advised.
c. Chemical reduction of oral microbial load includes rinsing or gargling with an effective mouthwash. One way to treat oral malodor associated with periodontitis is to combine regular periodontal treatment and a chlorhexidine mouth rinse. However, their long-term effect remains to be determined.
d. Another treatment strategy for oral malodor is conversion of volatile sulfur compounds by using various metal ions. Zinc (Zn^{++}) is an ion which bonds to the twice negatively charged sulfur radicals to reduce the expression of VSCs. Halita™ is a new solution containing 0.05% chlorhexidine, 0.05% cetyl pyridium chloride (CPC) and 0.14% zinc lactate with no alcohol has been more efficient than 0.2% chlorhexidine formulation in reducing the VSC levels. The special effect of Halita™ may result from the VSC conversion ability of zinc, besides its antimicrobial action.

SUMMARY

Halitosis affects a large proportion of population and may cause a significant social or psychological handicap to those who are suffering from it. Oral malodor can also reveal important diseases, if it can be analyzed accurately. VSC in periodontal pockets might be used as a predictor of periodontal diseases and also to monitor therapy sites. However, current available tools/tests are not applicable for achieving these goals due to lack of accuracy and objectivity.

The dental research community has long since ignored the subject of oral malodor. Recently, along with the growing public and media interest in oral malodor, dental professionals are becoming more aware of this problem. Let us hope that future research will overcome the problems related to diagnosing and treating oral malodor effectively.

KEYPOINTS

1. Halitosis is a term used to describe noticeably unpleasant odor exhaled in breathing.
2. Halitosis is classified into genuine halitosis, pseudo-halitosis and halitophobia.
3. Most commonly halitosis is as a result of problems originating from the oral cavity (90%). The remaining 10% is from systemic and non-oral origin.
4. Measurement of oral malodor is done by various methods like, organoleptic method, gas chromatography, Halimeters and BANA tests.
5. Treatment of oral malodor is a step-by-step problem solving procedure. It involves a complete recording of history, periodontal examination, and oral hygiene maintenance.
6. If oral malodor persists, chemical reduction of oral microbial load should be incorporated.

KNOW MORE ...

Specific character of breath odor can be related to a particular systemic malfunction

- A "rotten eggs" smell indicative of volatile sulfur compounds (VSCs).
- A sweet odor could be associated with liver insufficiency (accumulation of aliphatic acids).
- The smell of "rotten apples" associated with uncontrolled diabetes (accumulation of ketones).
- A "fish odor" associated with kidney insufficiency (accumulation of di- and trimethylamine).

REVIEW QUESTIONS

1. Describe the causes for oral malodor.
2. Enumerate various methods of treating oral malodor.

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12 Chapter

Pathogenesis of Periodontal Diseases

- ROLE OF BACTERIAL INVASION
- ROLE OF EXOTOXINS
- ROLE OF CELL CONSTITUENTS
- ROLE OF ENZYMES
- EVASION OF HOST RESPONSES
- THE HOST DERIVED BONE RESORBING AGENTS
- ROLE OF CYTOKINES

INTRODUCTION

Gingivitis and periodontitis are caused by bacteria that colonize the gingival crevice and attach to the tooth surface. The pathogenic potential of the bacteria within the plaque varies from gingiva site to site. Small amounts of plaque can be tolerated without causing periodontal disease, may be because of the host defence.

When bacteria and its products in the plaque increase beyond the threshold level of the host, then the balance shifts from health to disease. The mechanisms by which subgingival bacteria contribute to the pathogenesis of the periodontal disease are different. The pathogenesis of periodontal disease can be caused by subgingival bacteria alone or indirectly by evasion of immunologic host response. Direct effect can be by bacterial invasion, production of exotoxins, role of cell constituents, and production of various enzymes. The direct exposure or effect can be on the fibroblasts, epithelial cells, endothelial cells and inflammatory cells.

Junctional epithelium: It is the tissue most directly challenged by the pathogenic plaque bacteria. The microbial mass releases large quantities of metabolites like butyric acid and propionic acids which are toxic to periodontal tissues. The keratinocytes respond to these bacterial products by releasing cytokines and proinflammatory mediators. Interleukin (IL) and prostaglandin E_2 (PGE_2) and matrix metalloproteinases are released from the junctional epithelial cells. Neutrophils are present in the junctional epithelium as a part of host defence system. Activation of complement plays an important role in the earliest host response in the gingival crevice.

ROLE OF BACTERIAL INVASION

The properties that enable the bacterium to cause a disease are termed virulence factors, hence entry of bacterium itself (invasion) or of bacterial products into the periodontal tissues may be essential in the disease process.

The gingival sulcus and periodontal pockets are bathed in gingival crevicular fluid. Bacterial species that colonize

this region must attach to the available surfaces to avoid the displacement by the fluid flow. The surfaces available for attachment include the tooth or the root, the tissues and pre-existing plaque mass.

Bacteria that initially colonize the periodontal environment most likely attach to the pellicle or saliva-coated tooth surface, e.g. adherence of *A. viscosus* through fimbriae on the bacterial surface to a proline-rich protein found on the saliva-coated tooth surfaces.

Bacterial attachment to the pre-existing plaque is studied by examining the adherence between different bacterial strains (coaggregation), e.g. *A. viscosus* with *S. sanguis*. These are important in the colonization of the periodontal environment. The presence of bacteria in patients with ANUG was studied by Listgarten (1965). Microorganisms were recognized in the gingival connective tissue and in proximity to alveolar bone. Bacteria may also enter through ulceration in the pocket epithelium. In the Localized Juvenile Periodontitis (LJP), *A. actinomycetemcomitans* have been observed in gingival connective tissue. The presence of this organism within the tissues appears to make the disease more resistant to treatment and requires the use of antibiotics.

ROLE OF EXOTOXINS

A. actinomycetemcomitans produces an exotoxin referred to as leukotoxin because of its toxic effect on the human polymorphonuclear neutrophils (PMNs). The production of leucotoxin may enable *A. actinomycetemcomitans* to evade the host defence.

ROLE OF CELL CONSTITUENTS

Cell constituents of both gram-positive and gram-negative bacteria include endotoxins, bacterial surface components and capsular components.

Endotoxin or lipooligosaccharide (LOS) previously termed as lipopolysaccharide (LPS) is found in the outer membrane of all gram-negative bacteria. They are toxic substances affecting the tissues directly and through activation of the host responses. Its role in periodontal disease is, their ability to produce leukopenia, activate factor XII, activate the complement (C) system by the alternative pathway, lead to localized Schwartzman phenomena, with tissue necrosis occurring after two or more exposure to the endotoxin, may also have cytotoxic effects on the cells such as fibroblasts and induce bone resorption. These endotoxins can penetrate gingival epithelium. Bacteria can also produce fatty and organic acids such as butyric acid and propionic acids, amines, volatile sulphur compounds, indole, ammonia and glycans.

Peptidoglycan: A cell wall component may affect a variety of host responses including complement activation, immunosuppressive activity and stimulation of the reticuloendothelial system. It helps in the bone resorption and in stimulating macrophages to produce prostaglandin and collagenases.

ROLE OF ENZYMES

The bacterial enzymes that are capable of contributing to the disease process include collagenases, hyaluronidase, gelatinase, aminopeptidase, phospholipases and alkaline and acid phosphatases.

Destruction of periodontal tissues is by degradation of collagen. *P. gingivalis* and some strains of *A. actinomycetemcomitans* have been found to produce collagenase.

Bacterial hyaluronidase is capable of altering gingival permeability by allowing the apical proliferation of the junctional epithelium along the root surfaces. Hyaluronidase is found in high concentrations in periodontal pocket.

EVASION OF HOST RESPONSES (TABLES 12.1 TO 12.5)

Bacterial factors can also indirectly compromise the host tissues. These factors influence both the cellular and humoral immune responses.

For example,

1. Inhibition of PMNs by leukotoxin chemotactic inhibitors, decreased phagocytosis.
2. Immunoglobulins are inactivated by proteases.
3. Lymphocyte alterations
4. Endotoxicity
5. Catalase reaction.

Table 12.1: Influence of host response on periodontal disease

Aspect of disease	*Host factors*
1. Bacterial colonization	Subgingivally, antibody, complement in crevicular fluids inhibits adherence and coaggregation of bacteria and potentially reduces their numbers by lysis.
2. Bacterial invasion	Antibody-complement 1. Mediated lysis reduces bacterial counts. 2. Neutrophils as a consequence of chemotaxis, phagocytosis and lysis reduces bacterial counts
3. Tissue destruction	By antibody-mediated hypersensitivity, cell-mediated immune responses. Activation of tissue destruction factors such as collagenase.
4. Healing and fibrosis	Lymphocytes and macrophages produce chemotactic factors for fibroblasts, fibroblast activating factors.

Table 12.2: Impact of microorganisms on inflammation and immunity

Microbes and products	*Effects*
1. Microorganisms	• Activate complement • Activate neutrophils and macrophages. • Are antigenic.
2. Most peptides and proteins secreted by microorganisms	• Chemotactic for neutrophils and macrophages. • Are antigenic.
3. Enzymes	• Damage host cells. • Degrade connective tissue matrix. • Activate and degrade complement. • Degrade antibody. • Are antigenic.
4. Lipopolysaccharide	• Activate complement. • Damage host cells and alveolar bone resorption. • Is antigenic.
5. Polysaccharide plaque matrix and bacterial capsule	• Polyclonal B-cell activator. • Are antigenic.
6. Other toxins, acids, reducing agents	• Damage host cells. • Are antigenic.

Table 12.3: Mechanism which results in changes in epithelium

Mechanism	*Effects*
1. Bacterial toxins, e.g. endotoxins	• Cytotoxic to keratinocytes • Disruption of normal epithelial turnover and differentiation
2. Bacterial enzymes	• Damage to keratinocytes
3. Release of enzymes from neutrophils	• Damage of keratinocytes
4. Complement activation	• Cell damage
5. Production of TNF and interferon G by activated T-lymphocytes	• Decreased → keratinocyte proliferation
6. Production of IL-1 by macrophages	• Increased → keratinocyte proliferation

Table 12.4: Mechanisms which result in changes in connective tissue

Mechanisms	*Effects*
Bacterial toxins, e.g. endotoxins	• Toxic to fibroblasts • Decrease in collagen production
Bacterial enzymes, e.g. collagenase, hyaluronidase	• Degradation of extracellular matrix components.
Release of enzymes from neutrophils	• Damage to extracellular matrix components.
Complement activation	• Damage to fibroblasts • Decrease in collagen production
Production of IL-1 and TNF	• Increased secretion of collagenase and proliferations by fibroblasts
Production of TGF and Platelet derived growth factors (PDGF)	• Stimulation of fibroblast chemotoxin proliferation, matrix synthesis and attempts at repair.
Products of tissue damage	• Stimulation of fibroblast cells

THE HOST DERIVED BONE RESORBING AGENTS

Cytokines

- IL-1
- TNF

Table 12.5: Pathogenic immune reactions

Types	*Description*	*Definition*	*Examples*
Type-I	Anaphylactic	Antigen reacts with cells sensitized by IgE antibodies and release mediators.	Urticaria, hay fever, asthma, food allergies.
Type-II	Cytotoxic	Antibody reacts with cell-associated antigen usually, but not always, kills cells with help of complement or phagocytic cells.	Transfusion reaction auto-immune hemolytic anemia
Type-III	Arthus reaction	Antibody reacts with antigen in tissue spaces or blood stream to cause vasculitis, requires complement.	Serum sickness, Arthus reaction
Type-IV	Delayed hyper-sensitivity	Lymphocyte reacts with antigen. This reaction is mediated by lymphocytes, macrophages or their products.	Tuberculin reaction, con-act dermatitis, allograft rejection.

- TGF-β
- IL-6

Other Inflammatory Mediators

- Prostaglandins
- Leukotrienes

ROLE OF CYTOKINES

Meaning "cell protein" is used for molecules which transmit information or signals from one cell to another. Interleukins, growth factors, chemokines and interferon belong to the family of cytokines. They act on fibroblasts, macrophages, keratinocytes and PMNLs to release matrix metalloproteinases (MMPs) that degrade connective tissue matrix. However, most findings show that tissue destruction that occurs in periodontal disease is mainly due to host response to the bacteria and their products.

Important Cytokines Associated with Periodontal Disease

1. *IL-1*: It is produced predominantly by macrophages and lymphocytes. Fibroblasts, platelets and keratinocytes and endothelial cells also release IL-1. It upregulates adhesion molecules on endothelial cells, lymphocytes, neutrophils and monocytes. It activates T and B lymphocytes and promotes antibody production. IL-1α and IL-1β are potent stimulators of connective tissue destructions. It triggers the release of large quantities of prostaglandins E2 from fibroblasts and monocytes and stimulates secretion of matrix metalloproteinases.
2. *IL-2*: Monocytes and T lymphocytes produce IL-2. It stimulates T cells and enhances clonal expansion of beta cells into plasma cells.
3. *IL-4, IL-5 and IL-10*: They are produced by TH_2 cells and help in the activation of beta cells into plasma cells and down regulate monocytic response.
4. *IL-6*: It is released by lymphocytes, fibroblasts and monocytes. Lipopolysaccharide, IL-1 and tumor necrosis factor alpha upregulates the secretion of the IL-6. It is believed to be responsible for conversion of blood monocytes into osteoclasts.
5. *IL-8*: It is secreted by monocytes, keratinocytes and fibroblasts. It is a strong chemoattractant of PMNLs at low concentrations.
6. *TNF*: Tumor necrosis factor are produced by macrophages and release lymphotoxins. They stimulate the proliferation of osteoclast precursor cells and also activate the mature osteoclasts to resorb bone. It augments leukocyte chemotaxis, degranulation, adherence to endothelial cells and its ability to kill bacteria.
7. *Prostaglandin E_2*: Sources are macrophages and fibroblasts. IL-1 induces its production. It is a potent mediator of osteoclastic resorption.
8. *Matrix metalloproteinases* (MMPs): These are a family of enzymes capable of degrading connective tissue matrix. These enzymes are secreted in the latent form by fibroblasts, macrophages, keratinocytes and PMNLs, e.g. collagenase, gelatinase, stromalysin, matrilysin. IL-1 play an important role in upregulation of MMPs.

The leukotrienes, prostaglandins and related molecules are short range hormones that are produced by many cells; they exert their effect locally and are destroyed rapidly and spontaneously.

Prostaglandins and leukotrienes have been detected in biologically-active concentrations in the inflammatory exudates, leukotrienes C4, D4, and E4 known as slow reacting substances of anaphylaxis (SRS-A) are released from mast cells and basophils.

KNOW MORE ...

Various bacterial enzymes capable of degrading host tissue

They are collagenase, trypsin-like enzyme, phospholipase A, arylsulfatase, and neuraminidase.

In addition to immune cells, cells of the periodontium express **toll-like receptors (TLR)**. Signaling of these receptors results in innate immune responses including the release of various antibacterial peptides.

Matrix metalloproteinases (MMP) are a group of proteolytic enzymes found in neutrophils, macrophages, fibroblasts, epithelial cells and osteoblasts. They degrade extracellular matrix molecules such as collagen, gelatin and elastin.

REVIEW QUESTIONS

1. Describe the role of plaque bacteria in the pathogenesis of periodontal disease.
2. What are endotoxins?
3. What are cytokines?

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13 Chapter

Periodontal Medicine

- ERA OF FOCAL INFECTION
- PERIODONTAL DISEASES AND CORONARY HEART DISEASE/ATHEROSCLEROSIS
- EFFECT OF PERIODONTAL INFECTION
- PERIODONTAL DISEASE AND DIABETES MELLITUS
- ROLE OF PERIODONTITIS IN PREGNANCY OUTCOME
- PERIODONTAL DISEASE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE
- PERIODONTAL DISEASE AND ACUTE RESPIRATORY INFECTION
- PERIODONTAL MEDICINE IN CLINICAL PRACTICE

INTRODUCTION

Advances in science and technology over the last century have greatly expanded our knowledge of the pathogenesis of periodontal diseases. It is clear that certain systemic conditions may affect the initiation and progression of gingivitis and periodontitis.

The oral cavity is continuously challenged by opportunistic infections on one hand and the oral complications of systemic diseases and disorders occur on the other hand, thus an association between oral infections and systemic diseases has been suspected for centuries. The effect of oral health on the rest of the human body was proposed by the Assyrian's in the seventh century BC. In the 18th century a Pennsylvanian physician name Benjamin Rush quoted that arthritis could be treated in some people after they had extracted the infected teeth. But, over the past decade, growing scientific evidence suggests an exquisite association between oral infection (e.g. viruses, bacteria, yeasts) and systemic diseases (e.g. atherosclerosis, cardiovascular diseases, cerebrovascular diseases, premature low-birth infants and pulmonary diseases). Thus, the emerging evidence has shed light on the converse relationship between systemic health and oral health, i.e. the potential effects of periodontal disease on a wide range of organ systems. Thus, this chapter examines the emerging new evidence collected since the early 1990s implicating *periodontal infection as a risk factor for several systemic conditions*.

ERA OF FOCAL INFECTION

At the beginning of the twentieth century, medicine and dentistry were searching for reasons to explain why people became afflicted with a wide range of systemic diseases. Medicine at that time had very little insight into what caused diseases such as arthritis, pneumonia and pancreatitis, to name a few. Then, through the writings and lectures of principally two individuals, WD Miller and William Hunter, the concept that oral bacteria and infection were the likely cause of most of a person's systemic illness suddenly became very popular. For the next 40 years infections, especially

those originating in the mouth caused most of the man's suffering and illness. This era, which came to be known as the '*era of focal infection*' can be attributed primarily to a microbiologist in Philadelphia, Willoughby D. Miller and a London Physician, William Hunter.

'Focal infection' implied that there was a nidus of infection somewhere in the body, such as periodontitis, which via the bloodstream could affect distant sites and organs. Throughout the 1920s and 1930s, dentists and physicians believed that bacteria on the teeth and the resultant infectious diseases such as caries, gingivitis and periodontitis that followed were a 'focus of infection' that led to a wide variety of systemic problems. It became popular during this period to extract teeth as a means of ridding the body of oral bacteria and preventing and/or treating diseases affecting the joints as well as diseases of the heart, liver, kidneys and pancreas.

Hunter believed that teeth were liable to septic infection primarily due to their structure and their relationship to the alveolar bone. He stated that 'the degree of systemic effect produced by oral sepsis depended on the virulence of the oral infection and degree of resistance of the individual'. He also felt that oral organisms has specific actions on different tissues and that these organisms acted by producing toxins, resulting in low grade 'subinfections', which produced systemic effect over prolonged periods.

However, by 1940, medicine and dentistry were realizing that there was much more to explain a patient's general systemic condition than bacteria in his or her mouth. Dentists and physicians realized that: (1) extracting a person's teeth did not necessarily make the person better or make their disease go away, (2) people with very healthy mouths and no obvious oral infection developed systemic diseases, and (3) people who had no teeth and thus no apparent oral infection still developed systemic diseases.

By 1950, medicine was making great strides in discovering the true etiologies and dentistry was making great strides in the prevention as well as the treatment of caries and periodontal disease and so the era of 'focal infection' as a primary cause of systemic diseases finally came to an end.

However, it was not until the last decade of the twentieth century that dentistry and medicine again began to examine the relationship of oral infections as a risk for systemic disease and now there is a careful new look at periodontitis as a possible risk for systemic disease using discrete scientific levels of evidence.

PERIODONTAL DISEASE AND CORONARY HEART DISEASE/ATHEROSCLEROSIS

CHD and CHD-related events are a major cause of death. Myocardial infarction has been associated with acute systemic bacterial and viral infections and infarction is sometimes preceded by influenza like symptoms.

Is it possible that oral infections is similarly related to myocardial infarction?

Traditional risk factors such as smoking, dislipidemia, hypertension and diabetes mellitus do not explain the presence of coronary atherosclerosis in a large number of patients. Localized infection resulting in a chronic inflammatory reaction has been suggested as a mechanism underlying coronary heart disease in these individuals.

In cross-sectional studies of patients with acute myocardial infarction or confirmed CHD compared with age and gender matched control patients, myocardial infarction patients had significantly worse dental health than did controls. This association between poor dental health and myocardial infarction was independent of known risk factors for heart disease such as age, cholesterol level, hypertension, diabetes and smoking, because artherosclerosis is a major determinant of CHD-related events, dental health has also been related to coronary atheromatosus.

Mattila and colleagues performed oral radiographic examinations and diagnostic coronary angiography examinations on men with CHD. There was a significant correlation between the severity of dental disease and degree of coronary atheromatosus. This relationship remained significant after accounting for other known risk factors for coronary artery disease. Cross-sectional studies, thus suggest a link between oral health and coronary heart disease.

However, such studies cannot determine causality in this relationship. Rather, dental diseases may be indicators of general health practices for example, periodontal disease and CHD are both related to lifestyle and share numerous risk factors such as smoking, diabetes and low socioeconomic status. Bacterial infections have significant effects on endothelial cells, blood coagulation, lipid metabolism and monocyte macrophages. The research of Mattila and colleagues showed that dental infections were the only factors, other than classic and well-recognized coronary risk factors, that were associated independently with the severity of coronary arteriosclerosis.

This study and others in which periodontal condition is known to have preceded the CHD-related events, support the concept that periodontal disease is a risk factor for CHD, independent of other classic risk factors.

EFFECT OF PERIODONTAL INFECTION

Periodontal infection may affect the onset or progression of atherosclerosis and coronary heart disease through certain mechanisms. Periodontitis and atherosclerosis both have complex etiologic factors, combining genetic and environmental influences. The diseases share many risk factors and have distinct similarities in basic pathogenic mechanisms.

Ischemic Heart Diseases

Ischemic heart disease is associated with the process of atherogenesis and thrombogenesis. Increased viscosity of blood may promote major ischemic heart disease and stroke by increasing the thrombus formation.

Increased plasma fibrinogen is a recognized risk factor for cardiovascular events and peripheral vascular disease. Also elevated white blood cell count and coagulation factor VIII has been associated with risk of ischemic heart disease (Fig. 13.1).

Thrombogenesis

Platelet aggregation plays a major role in thrombogenesis, and most cases of acute myocardial infarction are precipitated by thromboembolisms. Oral organisms may be involved in coronary thrombogenesis since platelets selectively bind some strains of *Streptococcus sangius*, a common component of supragingival plaque and *Porphyromonas gingivalis*, a pathogen closely associated with periodontitis.

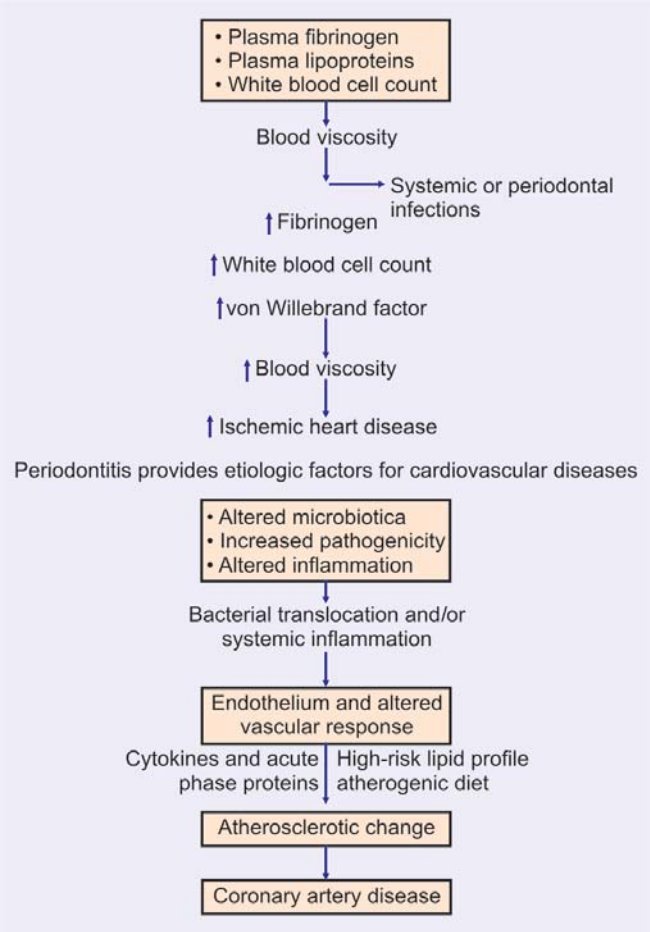

Fig. 13.1: Factors affecting blood viscosity in health

Daily activity: Routine daily activity such as mastication and oral hygiene procedures results in frequent bacteremia with oral organisms. The exposure time to bacteremias from routine daily chewing and tooth brushing is much greater than from dental procedures. Periodontal disease may predispose the patient to an increased incidence of bacteremias.

It has been estimated that about 8 percent of all cases of infective endocarditis are associated with periodontal or dental diseases without any preceding dental procedure.

Atherosclerosis (Fig. 13.2)

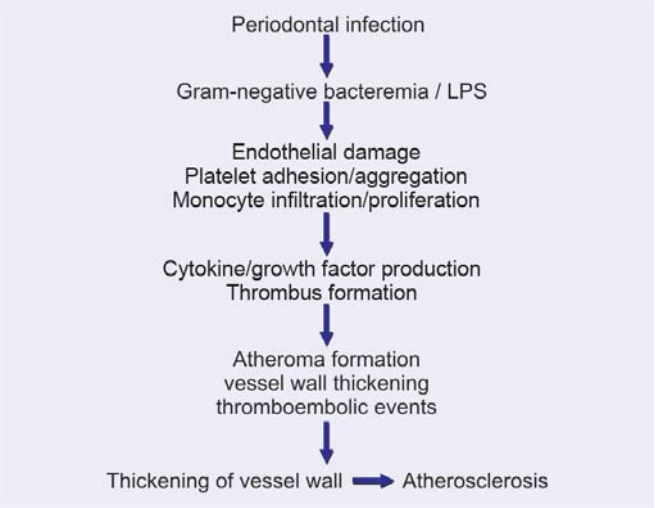

Fig. 13.2: Atherosclerosis

Periodontal Infection and Stroke

Ischemic cerebral infarction or stroke is often preceded by systemic bacterial or viral infections. In one study patients with cerebral ischemia were five times more likely to have had a systemic infection within 1 week before the ischemic event than were non-ischemic control subjects. Recent infection was a significant risk factor for cerebral ischemia and was independent of other known risk factors such as hypertension, history of a previous stroke, diabetes, smoking and coronary heart disease.

In case-control studies, poor dental health was a significant risk factor for cerebrovascular ischemia. In one study bleeding on probing, suppuration, subgingival calculus and number of periodontal and periapical lesions were significantly greater in male stroke patients than in controls.

Overall 25 percent of all stroke patients had significant dental infections compared with only 2.5 percent of controls. This study supports an association between poor oral health and stroke in men under age 50. In another study men and women's of age 50 and older who had a stroke had significantly more severe periodontitis and more periapical lesions as compared to the non-stroke control subjects.

PERIODONTAL DISEASE AND DIABETES MELLITUS

An understanding of effects of other infections is useful in determining the mechanisms by which the periodontal infection influences glycemia. Acute bacterial and viral infections have shown to increase insulin resistance and aggravate glycemic control. This occurs in individuals with and without diabetes. Systemic infection increases tissue resistance to insulin, preventing glucose from entering target cells causing elevated blood glucose level and requiring increased pancreatic insulin production to maintain normoglycemia.

It is possible that chronic Gram-negative periodontal infection may also result in increased insulin resistance and poor glycemic control.

In patients with periodontitis, persistent systemic challenge with periodontopathic bacteria and their products may act in a way similar to well-recognized systemic infection. This mechanism would explain the worsening of glycemic control associated with severe periodontitis. Periodontal treatment designed to decrease the bacterial insult and reduce inflammation might restore insulin sensitivity overtime, resulting in improved metabolic control. The improved glycemic control seen in several studies of periodontal therapy would support such a hypothesis.

ROLE OF PERIODONTITIS IN PREGNANCY OUTCOME

Periodontitis is a remote Gram-negative infection that may play a role in preterm low birth weight (Fig. 13.3) and pre-eclampsia.

Periodontopathic organisms and their products may have wide range effects, most likely mediated through stimulation of host cytokine production in target tissues. Animal studies suggest that remote reservoirs of Gram-negative organisms and their products may have a negative impact on pregnancy. *Porphyromonas gingivalis* implanted in subcutaneous chambers during gestation caused significant increase in fetal death and a decrease in fetal birth weight for those that remained viable, when compared with control animals that were not innoculated.

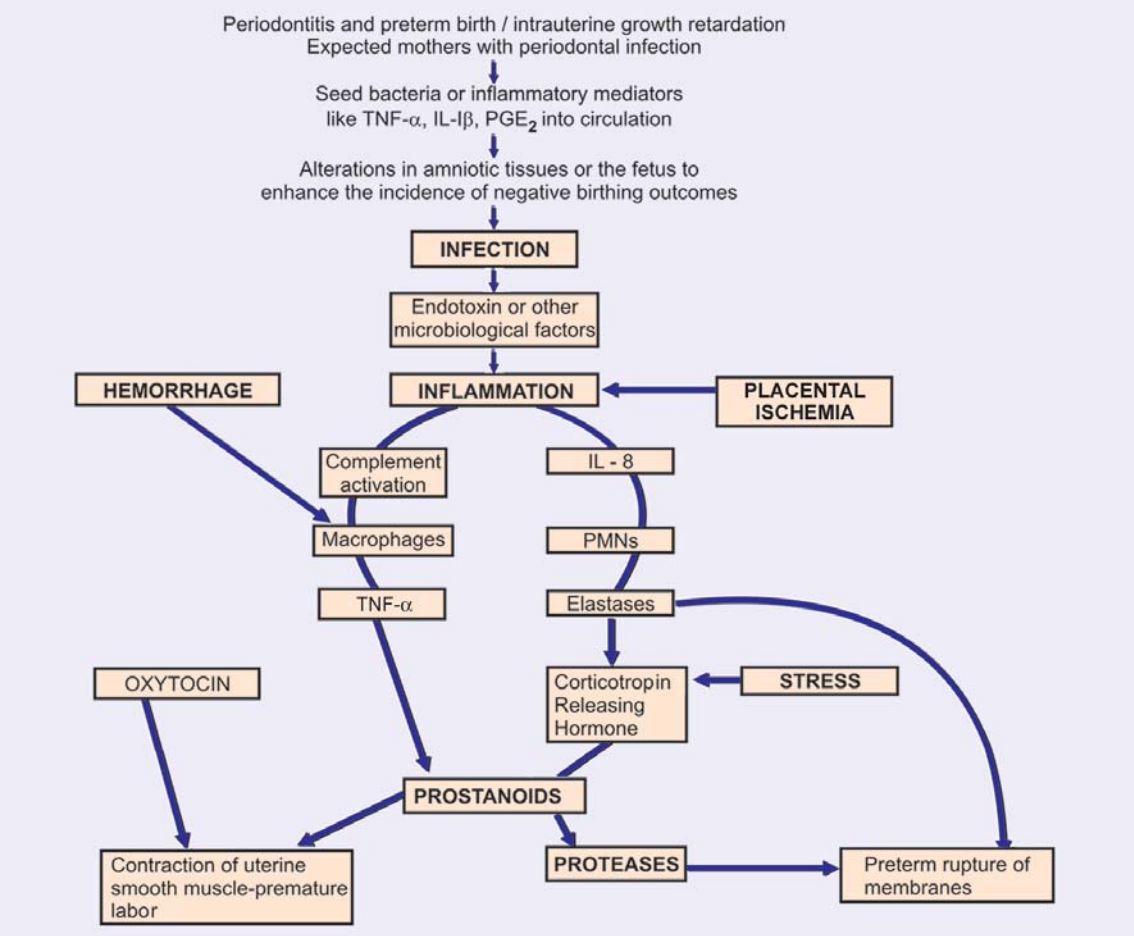

Fig. 13.3: Periodontitis and preterm birth/intrauterine growth retardation

There was a significant increase in TNF-α and PGE_2 levels. There was a significant correlation between TNF-α and PGE_2 levels, as well as fetal death and growth retardation. These data suggest that a remote, non-disseminated infection with *Porphyromonas gingivalis* may result in abnormal pregnancy outcome in this model.

PERIODONTAL DISEASE AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease is a disease state characterized by airflow obstruction due to chronic bronchitis or emphysema. Bronchial mucous glands enlarge and an inflammatory process occurs in which neutrophils and mononuclear inflammatory cells accumulate within the lung tissue.

Chronic obstructive pulmonary disease shares similar pathogenic mechanism with periodontal disease. In both diseases, a host inflammatory response is mounted in response to chronic challenge by bacteria in periodontal disease and by factors such as cigarette smoke in chronic obstructive pulmonary disease. The resulting neutrophil influx leads to release of oxidative and hydrolytic enzymes that cause tissue destruction directly.

In analyzing data from a longitudinal study of more than 1100 men alveolar bone loss was associated with the risk of chronic obstructive pulmonary disease. The increase in the risk was independent of age, smoking status and other known risk factors for chronic obstructive pulmonary disease. Individuals with poor oral hygiene have also been found to be at an increased risk for chronic respiratory

diseases such as bronchitis and emphysema. These associations remain to be confirmed by further research.

PERIODONTAL DISEASE AND ACUTE RESPIRATORY INFECTION

Pneumonia is an infection of lungs caused by bacteria virus, fungi or mycoplasma and is broadly categorized as either

- Community acquired, or
- Hospital acquired

Community acquired bacterial pneumonia is caused primarily by inhalation of infectious aerosols or by aspiration of oropharyngeal organisms. *S. pneumonia* and *Haemophilus influenzae* are most common, although numerous other species may occur, including anaerobic bacteria. To date, no associations have been found between oral hygiene or periodontal disease and the risk for acute respiratory conditions such as pneumonia in community dwelling individuals.

Hospital acquired (nosocomial) bacterial pneumonia has very high morbidity and mortality rate. The incidence of nosocomial pneumonia is highest in severely ill patients such as those in intensive care units or on ventilatory support.

Hospital acquired pneumonia is usually caused by aspiration of oropharyngeal contents. Oropharyngeal colonization with potential respiratory pathogens (PRPs) increase during hospitalizations and the longer the hospital stay the greater the prevalence of PRPs. PRPs are found predominantly in the gastrointestinal tract and may be passed through esophageal reflux into the oropharynx where they colonize. Subsequent aspirations may lead to pneumonia.

The PRPs may also originate in the oral cavity, with dental plaque serving as a reservoir of these organisms. Poor oral hygiene is common in the hospital and nursing home settings, especially in severely ill patients. PRPs are commonly isolated from supragingival plaque and buccal mucosa of patients in intensive care units than in outpatient setting. Thus, organisms that are not routinely found in dental plaque become plaque colonizers after prolonged hospitalizations. Subgingival plaque may also harbor PRPs and putative periodontal pathogens have been associated with nosocomial pneumonia. Furthermore anaerobic organisms from periodontal pockets may serve as the primary innoculum for suppurative respiratory disease such as pulmonary abscesses that have significant morbidity and mortality. Although considerable circumstantial evidence suggests that periodontal pathogens may cause acute nosocomial pulmonary infection, currently no published studies specifically demonstrate an increased risk of such infections in patients with periodontal disease.

PERIODONTAL MEDICINE IN CLINICAL PRACTICE

The concept of periodontal diseases as localized entities affecting only the teeth and supporting apparatus is over-simplified and needs to be revised. Rather than being confined to periodontium, periodontal diseases may have wide-ranging systemic effects. In most persons these effects may be relatively inconsequential or at least not clinically evident. However in susceptible individuals, periodontal infection may act as an independent risk factor for systemic disease and may be involved in the basic pathogenic mechanism of these conditions. Furthermore, periodontal infection may exacerbate existing systemic disorders.

Proper use of knowledge of relationship between periodontal disease and systemic health requires the dental professional to expand his or her horizons, to step back from the technically demanding aspects of dental art, and to recognize the oral cavity as one of the many interrelated organ systems. Patient education in this regard is also very important.

KEYPOINTS

1. Periodontal infection may affect the onset or progression of atherosclerosis and coronary heart disease through certain mechanisms.
2. Altered microbiota associated with deep periodontal pockets → bacterial translocations/or systemic inflammation → endothelial and altered vascular response → cytokines and acute phase proteins atherosclerotic changes → coronary artery disease (MI).
3. Studies conducted by Offenbacher has shown that women with severe periodontitis are 7.5 times more likely than women without periodontal disease to have an infant with preterm low-birth weight.

KNOW MORE ...

In periodontal medicine, evaluation of evidence could be derived from various types of studies

a. *Case reports* — provides relatively weak, anecdotal evidence.
b. *Cross-sectional study* — compares groups of subjects at a single point in time.
c. *Longitudinal study* — stronger than cross sectional study, follows groups of subjects over a period of time.
d. *Interventional trial* — strongest form of evidence examines effects of some interventions.

REVIEW QUESTION

1. Describe the role of periodontal disease as a risk factor for systemic diseases (Periodontal Medicine).

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14 Chapter

Smoking and Periodontal Diseases

- EFFECT OF SMOKING ON THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASE
  - Gingivitis
  - Periodontitis
- EFFECT OF SMOKING ON THE ETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASE
  - Microbiology
  - Immunology
  - Physiology
- EFFECT OF SMOKING ON THE RESPONSE TO PERIODONTAL THERAPY
- EFFECT OF SMOKING CESSATION

EFFECT OF SMOKING ON THE PREVALENCE AND SEVERITY OF PERIODONTAL DISEASE

See Table 14.1.

Table 14.1: Impact of smoking on periodontal diseases

Periodontal disease	*Impact of smoking*
Gingivitis	• Decreased gingival inflammation and bleeding on probing.
Periodontitis	• Increased prevalence and severity of periodontal destruction. • Increased pocket depth, attachment loss and bone loss. • Increased rate of periodontal destruction. • Increased prevalence of severe periodontitis. • Increased tooth loss. • Increased prevalence with increased number of cigarettes smoked per day. • Decreased prevalence and severity with smoking cessation.

EFFECT OF SMOKING ON THE ETIOLOGY AND PATHOGENESIS OF PERIODONTAL DISEASE

Microbiology

- Smokers had higher level of *Bacteroides forsythus*.
- Smokers do not respond to mechanical therapy and this is associated with increased level of *B.forsythus, A. actinomycetemcomitans* and *P. gingivalis* remaining in the pocket after therapy when compared to nonsmokers.
- *Eikenella nodatum, Fusobacterium nucleatum, S. vincentii, P. gingivalis, P. intermedia, Peptostreptococcus micros, Prevotella nigrescens, B. forsythus* were significantly more prevalent in current smokers than in nonsmokers and former smokers.

Immunology

- Altered neutrophil chemotaxis, phagocytosis and oxidative burst.
- Increased TNF-α, and PGE_2 in GCF.
- Increased production of PGE_2 by monocyte in response to lipopolysaccharides (LPS).
- IgG_2 level is reduced suggesting reduced protection against periodontal infection.
- Nicotine, a major component of tobacco adversely affect fibroblast function.
- Nicotine suppresses osteoblast proliferation while stimulating alkaline phosphatase activity.
- Tobacco products alter normal reparative and regeneration potential of periodontium.

Physiology

- Clinical signs of inflammation are less pronounced, due to alteration in the inflammatory response in smokers or due to alteration in vascular response of gingival tissues (Fig. 14.1).
- Decreased gingival blood vessels with increased inflammation.
- Decreased GCF flow and bleeding on probing with increased inflammation.

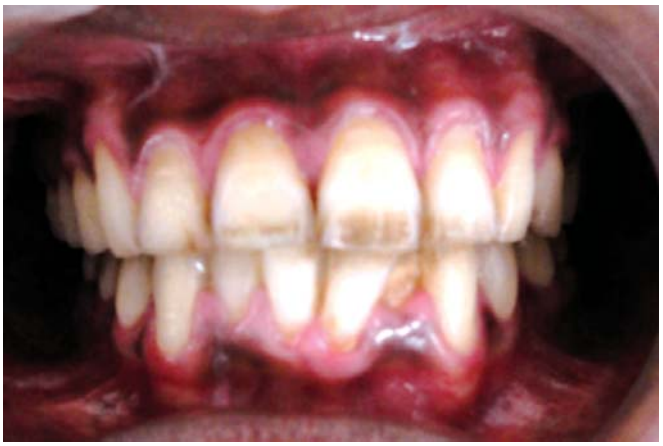

Fig. 14.1: Decreased signs of inflammation in smokers

- Decreased subgingival temperature.
- Increased time needed to recover from local anesthesia.

EFFECT OF SMOKING ON THE RESPONSE TO PERIODONTAL THERAPY

See Table 14.2.

EFFECT OF SMOKING CESSATION

- Gingiva of treated current smokers exhibit minimal redness and bleeding while brushing presumably

Table 14.2: Effect of smoking on periodontal therapies

Therapy	*Effect of smoking*
Non-surgical	• Decreased clinical response to scaling and root planing • Decreased reduction in pocket depth • Decreased gain in clinical attachment level. • Decreased negative impact of smoking with increased level of plaque control.
Surgery and implants	• Decreased pocket depth reduction post surgery. • Increased deterioration of furcation postsurgery. • Decreased gain in clinical attachment level, decreased bone fill, increased recession and increased membrane exposure following guided tissue regeneration (GTR). • Decreased pocket depth reduction after DFDBA allograft. • Decreased pocket depth reduction and gain in clinical attachment level after open flap debridement. • Conflicting data on the impact of smoking on implant success. • Smoking cessation should be recommended prior to implant.
Maintenance	• Increased pocket depth during maintenance. • Decreased gain in clinical attachment level.
Recurrent (refractory) disease	• Increased recurrent/refractory disease in smokers. • Increased need for retreatment in smokers. • Increased need for antibiotics in smokers to control the negative effect of periodontal infection on surgical outcome. • Increased tooth loss in smokers after surgical therapy.

because of immunosuppression or vascular effect of smoking.

- Several weeks following smoking cessation, gingival inflammation and bleeding on brushing occurs because of smoking cessation, gingiva loses its thick fibrotic appearance and assumes normal anatomy.

Therefore, smoking status should be considered in diagnosis, prognosis and treatment planning of periodontitis patients.

KEYPOINTS

1. In smokers there is decreased gingival inflammation and bleeding on probing. But, increased prevalence and severity of periodontal destruction.
2. There is increased levels of periodontopathic organisms namely, *B. forsythus*, *P. gingivalis*, *A. actinomycete-mcomitans* and others.
3. Increased production of inflammatory mediators.
4. Fibroblast function is diminished. It also alters normal reparative and regeneration potential of periodontium.
5. In general, there is increased need for retreatment in smokers.

KNOW MORE ...

Local effects of nicotine

a. Nicotine levels in GCF can be up to nearly 300 times that of nicotine plasma concentrations in smokers.
b. Impairs gingival blood flow which in turn affects the wound healing.
c. Products of nicotine forms a layer on root surface which can alter fibroblast attachment and hence decreased collagen production.
   – In an attempt to tobacco cessation, various nicotine replacement therapies have been introduced. Currently, FDA (The US Food and Drug Administration) approves nicotine chewing gum, nicotine lozenges, nicotine patches, nasal sprays and nicotine inhaler.

REVIEW QUESTION

1. What are the effects of smoking on periodontal disease?

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15 Chapter

Host Modulation in Periodontal Therapy

INTRODUCTION

Specific Aspects of Disease Pathogenesis as Potential Targets for Host Modulation

Considering the fact that periodontal disease is, as a result of host interaction between the plaque biofilm and host responses, the area of research is mainly directed at altering an individual's reaction to the bacterial challenge. Hence, various host modulatory therapies (HMT) have been developed or proposed to block the pathways responsible for periodontal tissue breakdown. In context to this, specific aspects of disease pathogenesis that have been investigated for modulation include,

a. Regulation of immune and inflammatory responses.
b. Excessive production of matrix metalloproteinases.
c. Arachidonic acid metabolism.
d. Bone metabolism.

Currently, one systemically administered agent is available for modulation of host, i.e. subantimicrobial dose doxycycline marketed as Periostat™ by collagens pharmaceuticals.

REGULATION OF IMMUNE AND INFLAMMATORY RESPONSES

Currently, it is believed that, small groups of periodontopathic microorganisms within the plaque biofilm are often associated with disease initiation and progression. Some of the strongly implicated organisms include *Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans (Aggregatibacter actinomycetemcomitans)* and *Bacteroides forsythus*. The disease initiation occurs by microbial challenge consisting of antigens, lipopolysaccharides (LPS) and other virulence factors, stimulates host responses thereby resulting in disease either limited to gingiva (gingivitis) or progresses to periodontitis.

Activation of host has both protective and destructive aspects. Protective aspects of host response include recruitment of neutrophils, production of protective antibodies and

possibly the release of various anti-inflammatory cytokines including TGF-β (transforming growth factor-β and interleukins (IL-4, IL-10 and IL-12). On the other hand perpetuation of the host response due to persistent bacterial onslaught may disrupt the homeostatic mechanism and result in release of mediators including proinflammatory cytokines, (e.g. IL-1, IL-6, TNF-α), matrix metalloproteinases (proteases) and prostaglandin E_2 (PGE_2), which can promote extracellular matrix destruction in the gingiva to stimulate bone resorption. However, there are other set of cytokines, which can suppress the action of pro-inflammatory cytokines, they include IL-4, IL-10, IL-11 and TGF-β. When administered for therapeutic purposes, these antagonists can reduce inflammation. However, there are certain unresolved issues regarding the cytokine modulation therapy like identifying an ideal method to maintain or inhibit cytokines for long periods and also understanding of the systemic implications associated with altering cytokine levels on tissue homeostasis.

Therefore, additional animal and human studies are required to apply it routinely in the treatment of periodontitis.

EXCESSIVE PRODUCTION OF MATRIX METALLOPROTEINASES (MMPs)

MMPs are a family of zinc and calcium dependent endopeptidases secreted or released by a variety of infiltrating cells like neutrophils, macrophages and resident cells like fibroblasts, epithelial cells, osteoblasts and osteoclasts found in the periodontium.

The major functions of MMPs are to degrade the constituents of the extracellular matrix like laminin, collagen, fibronectin, etc.

Role of MMPs in Human Periodontal Diseases

One hypothesis regarding periodontal disease pathogenesis is that host cells stimulated directly or indirectly by components of the plaque biofilm secrete MMPs, which are associated with altered connective tissue remodeling and alveolar bone resorption. Although, several periodontal pathogens (e.g. *P. gingivalis and A.actinomycetem comitans*) produce MMPs including collagenase. It is however, believed that endogenous MMPs are not the bacterial proteinases primarily responsible for tissue destruction. This further emphasizes the role of host modulatory approaches in periodontal therapy (Fig. 15.1).

Several synthetic MMP inhibitors are being studied in clinical trials. One such most extensively studied inhibitor are the family of tetracycline antibiotics, which can inhibit host derived MMPs by mechanisms independent of their antimicrobial properties. The development of host modulating therapy (HMT), utilizing tetracyclines primarily involves the use of a reduced dose of doxycycline (Periostat™ 20 mg bid). These doses reportedly do not exhibit antimicrobial effects, but can effectively lower MMP levels. This reduced dose has been referred to as subantimicrobial dose doxycycline (SDD).

In addition to use of SDD in host modulating therapy, 10 different chemically modified tetracyclines (CMTs) have been developed, 9 of which inhibits MMPs and do not possess antimicrobial properties. In animal models CMTs have been reported to reduce the progression of experimentally induced periodontitis, however inhibition of human periodontitis is still not established.

PRODUCTION OF ARACHIDONIC ACID METABOLITES

Another destructive pathway in the pathogenesis of periodontal disease is synthesis and release of prostaglandins and other arachidonic acid metabolites within periodontal tissues. Whenever, there is tissue damage due to bacterial and host factors the phospholipids in the plasma membrane of cells becomes available to phospholipase A_2, thereby results in production of free arachidonic acid. Arachidonic acid can be metabolized via the cyclooxygenase or lipoxygenase pathway. The final products of the cyclooxygenase pathway include prostaglandins, prostacycline and thromboxane, whereas the end result of the lipoxygenase pathway include leukotrienes and other hydroxyeicosatetraenoic acids. Elevated levels of PGE_2 and other arachidonic acid metabolites have been reported in crevicular fluid in patients with gingivitis and periodontitis.

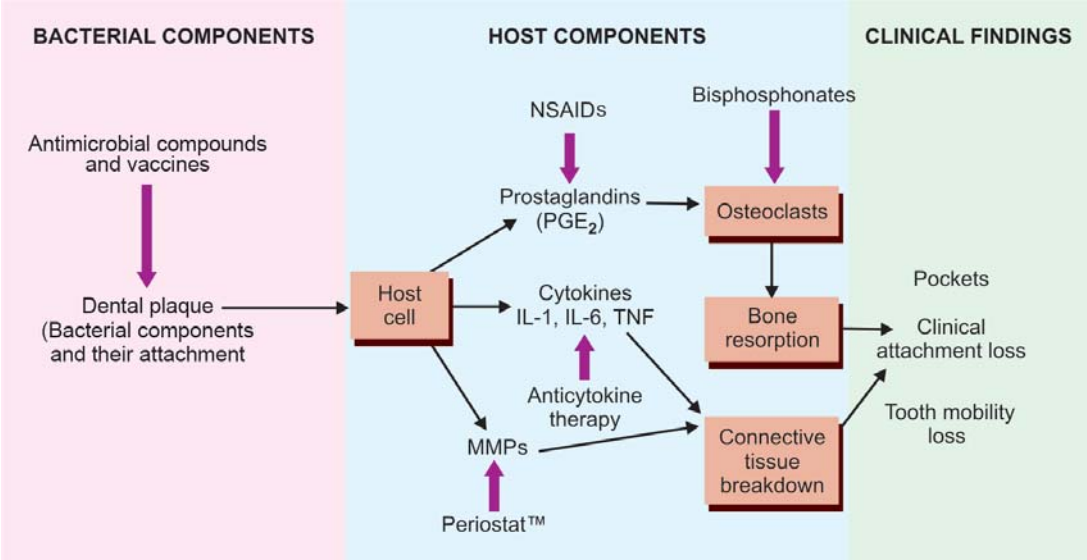

Fig. 15.1: Pathological events of periodontal diseases with various host modulatory approaches

Hence, one proposed approach to modulate host response is inhibition of enzymes responsible for the release of these destructive products.

Various *in vitro* studies have been conducted to assess the non-steroidal anti-inflammatory drugs (NSAIDs) as inhibitors of bone resorption. Multiple NSAIDs including indomethacin, flurbiprofen, ibuprofen, naproxen, meclofenamic acid and piroxicam have demonstrated their ability to reduce gingivitis and progression of periodontitis in animal models. Ketoprofen, an NSAID which can inhibit both cyclooxygenase and lipoxygenase pathways, has recently received a lot of attention. In humans, most of these NSAIDs have shown significant reduction in bone loss, however disease progression returned upon the withdrawal of the agent. Hence, topical administration of NSAIDs has been considered as an alternative method to deliver these agents. Various drugs that have been evaluated for topical administration include, ketorolac tromethamine rinse and S-ketaprofen dentifrice. However, further studies are required to determine the efficacy of these agents to provide clinically significant improvements, when used as adjuncts to scaling and root planing. Other agents that have been tried for topical use are lipoxins, which are a series of oxygenated arachidonic acid derivatives functioning as endogenous inflammatory mediators. In animal models, these lipoxins at a metabolically stable state blocked the neutrophil infiltration induced by *P. gingivalis* and also reduced PGE_2 levels. However, additional studies are required to demonstrate the role of lipoxins in the pathogenesis of periodontitis. Recently, a compound that has received interest as both antibacterial and anti-inflammatory agent is triclosan. Dentifrice containing sodium fluoride, triclosan and a copolymer has been tested. However, additional studies are required to examine the effect of this combination of drugs on periodontitis.

REGULATION OF BONE METABOLISM

Various drugs have been tried to inhibit the activity of osteoclasts. A new class of drugs used to manage osteoporosis, which may also have beneficial effects on the periodontium, are the bisphosphonates. These compounds through the mechanism of chelation of cations seem to inhibit MMP activity thereby inhibiting osteoclastic activity.

One of these drugs, alendronate has been evaluated in ligature induced periodontitis models. Although it inhibited loss of bone density in animal models, the human trials have shown minimal effects on clinical parameters. Further studies are required to evaluate the effectiveness of these drugs in the treatment of periodontal diseases.

KEYPOINTS

1. The current paradigm, for the etiology and pathogenesis of periodontitis includes the involvement of periodontal pathogens and destructive host responses.
2. To prevent the disease initiation and progression, mechanical and antimicrobial pharmaceutical agents have been used successfully.
3. For the long-term clinical management of periodontitis a novel adjunctive therapies such as host modulation has been introduced.
4. The use of HMT (Host Modulation Therapy) as an adjunct may be particularly useful in susceptible, high risk patients (e.g. smoking, diabetes, genetic predisposition) FDA has recently approved subantimicrobial dose doxycycline (Periostat™) for systemic administration as an adjunct to scaling and root planing in the treatment of chronic periodontitis.
5. The future holds much hope for application of HMT not only for managing the periodontitis patients, but also for the practice of periodontal medicine.

KNOW MORE ...

Host Modulators

Subantimicrobial dose doxycycline (Periostat®) is available at 20 mg dose which acts as a host modulator.

Mechanism of action involves direct inhibition of MMPs which indirectly reduces osteoclast activity and bone resorption.

Future host modulation agents could be Lipoxins, Resolvins and Probiotics (Lactobacilli and Bifidobacteria).

REVIEW QUESTION

1. Enumerate various host modulating agents in periodontal therapy.

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Part IV

Periodontal Pathology

16 Chapter

Defence Mechanisms of the Gingiva

DEFENCE MECHANISMS

A number of mechanisms operate to protect the body from attack by foreign bodies and toxins, including infections by bacteria. These mechanisms can be classified as:

1. Nonspecific mechanisms.
2. Mechanisms specific to invading foreign proteins called antigens which stimulate the immune system.

Nonspecific Protective Mechanisms

Bacterial Balance

The mouth as a whole and various zones in the mouth, including what has been called the '*crevicular domain*' can be viewed as an ecosystem in which a balance exists between different species of microorganisms, their flora and their tissues.

Surface Integrity

The surface integrity of skin and mucous membrane barrier, including the gingiva, is maintained by the persistent renewal of the epithelium from its base and desquamation of the surface layers. These two activities are balanced and this helps in maintaining a constant thickness of the epithelium. The efficiency of the surface barrier is enhanced by keratinization and parakeratinization. The junctional epithelium, although semipermeable, has a very high rate of cell turnover.

Surface Fluid and Enzymes

All vital surfaces are washed by fluids, which are capable of attacking foreign materials, e.g. gastric acid, lysozyme, saliva. Saliva bathes the oral mucosa and contains antibacterial substances. The gingival fluid exudates flow

through the junctional epithelium into the gingival crevice and this fluid contains phagocytic leukocytes and enzymes (Table 16.1).

Phagocytosis

Certain cells in the blood stream and in the tissues are capable of engulfing and digesting foreign materials. The most important phagocytic cells are polymorphonuclear neutrophils and the macrophages.

Macrophages are basically monocytes, which when moved into the tissues, mature and become macrophages, unlike polymorphonuclear neutrophils which have the capacity to undergo several divisions within the tissues, which progressively increase in number. While polymorphonuclear neutrophils are the main line of defence in acute infections, monocytes are more important in long-term chronic infections.

Phagocytosis is aided by a battery of nine related proteins known as *complement* which are activated by two systems, namely classical and alternative pathway. In the process of activation of the complement system, fragments are generated by cleavage of C_3, C_4 and C_5. These fragments are termed as C_{3a}, C_{4a} and C_{5a}, these take part in the defence mechanism, in the tissue fluids and are referred to as *anaphylotoxins*, since they can induce smooth muscle contraction, increase permeability of blood vessels and cause the release of histamine from mast cells and basophils.

C_{5a}, in addition, is chemotactic for neutrophils and monocytes. It augments cell adherence and causes degranulation of these cells, it also enhances arachidonic metabolism and stimulates production of toxic metabolites.

Table 16.1: Functions of saliva

Functions	*Salivary components*	*Probable mechanism*
Lubrication	Glycoproteins mucoids	Coating similar to gastric mucin
Physical protection	Glycoproteins mucoids	Coating similar to gastric mucin
Cleansing	Physical flow	Clearance of debris and bacteria
Buffering	Bicarbonate and phosphate	Antacids
Tooth integrity maintenance	Minerals glyco-protein pellicle	Maturation, remineralization, mechanical protection
	IgA	Control of bacterial colonization
Antibacterial action	Lysozyme	Breaks bacterial cell walls
	Lactoperoxidase	Oxidation of susceptible bacteria

The Inflammatory Reaction

The inflammatory reaction is stimulated by tissue injury and infection and leads to changes in the local microcirculation, which produces hyperemia, increased vascular permeability and the formation of a fluid and cellular exudate.

Specific Protective Mechanism

Animals (vertebrae) have a developed surveillance and attack system called the immune system. This can protect the body from bacteria, viruses and even cancer cells.

The system has three characteristics:

1. It can distinguish between itself and the enemy; thereby it does not attack parts that it recognizes as self. Sometimes it may go wrong and thereby results in certain diseases known as the *autoimmune diseases*.
2. The defences contain elements specific against any given antigen.
3. The system has a memory. The specific immune mechanism has two basic components:
   a. Humoral immunity
   b. Cell-mediated immunity

   Both arise from stem cells in the bone marrow.

SULCULAR FLUID

Anatomy of Gingival Crevice or Sulcus

The gingival sulcus is the shallow crevice or space around the tooth, bounded by the surface of the tooth on one side and the epithelium lining the free margin of the gingiva, on the other.

Sections of the marginal region of clinically healthy gingiva will show the presence of three types of epithelia, the oral or keratinized epithelium covering the gingival connective tissue, in continuation with the oral sulcular epithelium, which is not keratinized. It forms the soft tissue wall of the gingival sulcus and the junctional epithelium is in continuation with the oral sulcular epithelium. It is formed by a few strata of cells, with a long flat basal layer and a very small desquamating surface that forms the base of the gingival sulcus.

Normally, the depth of the gingival sulcus is zero, however 1.5 to 3.0 mm of the so called probing depth of the gingival sulcus may be considered normal. When the probing depth exceeds more than 3 mm, a new type of epithelium called the "pocket epithelium" appears. It is characterized by irregular ridges, ulceration, discontinuous basal layer and it is not attached to the tooth. Below this epithelium, a typical junctional epithelium is present; forming a short epithelial attachment, the designation of gingival pocket can be used. Finally, in the presence of periodontitis, pocket depth greater than 3 to 5 mm is seen, active bone resorption takes place, bacterial colonies are found situated very deep along the cementum surface.

Significance of Gingival Sulcus and Fluid

Brill and Krasse have clearly demonstrated that parenterally administered tracer material could be removed from the gingival sulcus by using 4 mm wide filter paper strips intracrevicularly. Brill reported various types of tissue damage using the same technique. Now new methods of collecting crevicular fluid are available which do not cause any irritation, for example extracrevicular technique and a simple procedure of drying a chronically inflamed gingiva by a blast of air.

Significance of Gingival Vasculature and Crevicular Fluid (Fig. 16.1)

The blood supply to marginal gingiva is by the vessels of both the periodontal ligament and oral mucosa, each consisting of arterioles and venules. In the presence of inflammation, the width and length of capillary and post-capillary venules increases, this results in twisting and looping of the vessels underlying sulcular and junctional epithelium. The vessels immediately below the sulcular epithelium and junctional epithelium are damaged in a flat layer. Since these epithelia do not possess ridges projecting into the connective tissue, their vasculature network is located in a very superficial position.

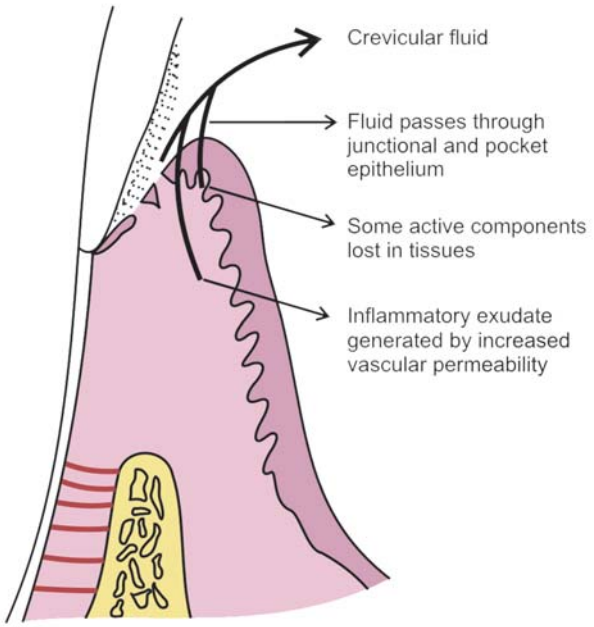

Fig. 16.1: The generation of crevicular fluid

The significance of such an arrangement in the mechanism of the production of gingival fluid was clearly demonstrated by Egelberg, who has demonstrated that the production of crevicular fluid is primarily related to an increase in the permeability of the vessels underlying junctional and sulcular epithelium.

Permeability of Sulcular and Junctional Epithelium

The main pathway for the transport of substances across the junctional and sulcular epithelia seems to be the intercellular spaces which form, 18 percent of the total volume of the junctional epithelium and 12 percent of that of the outer sulcular epithelium. Barriers to the passage of substances through junctional and sulcular epithelium are represented by the intercellular junctions and especially by the basement membrane.

In the presence of inflammation, enlargement of intercellular space of both junctional epithelium and sulcular epithelium along with partial destruction of the basal membrane results in the inward passage of foreign substances.

Variety of enzymes such as hyaluronidase and collagenase has the ability to alter the permeability properties of the junctional epithelium and the sulcular epithelium. They have the ability to penetrate even the intact junctional and sulcular epithelia. Nutritional deficiencies such as Ascorbic acid might also alter sulcular permeability and allow the passage of substances from the gingival sulcus into gingival connective tissue, like histamine, leucine, thymidine, phenytoin, peroxidase, albumin, dextran, carbon particles, endotoxins and others.

One can conclude that the epithelia covering the gingival sulcus, represents a relative barrier to the penetration of foreign material from the sulcus into the connective tissue. It is conceivable that plaque components, even of relatively high molecular weight could pass within the gingival connective tissue, when allowed, to accumulate in the sulcus.

Methods of Collection

It is usually collected from the anterior teeth (least contamination). A few techniques are available:

a. Absorbing paper strips
b. Sampling by means of micropipettes
c. Gingival washings
d. Other methods.

Absorbing Paper Strips

Two techniques are followed:

- *Intracrevicular*: The end of the paper strip is gently inserted into the pocket until minimum resistance is felt.
- *Extracrevicular*: The strip is placed at the entrance of the gingival crevice. This technique has subsequently never been used.

Evaluation of Amount of Fluid Collected

a. *Appreciation by direct viewing and staining*, was proposed by Egelberg and Attstrom. The strip was stained with an alcoholic solution of ninhydrin at concentration of 0.2 percent (gives blue or purple color). The stained area can then be measured with a transparent scale, calipers or calibrated magnifying glass.
b. *By weighing the strip*: The strip is weighed before collection of the sample within a sealed micro-centrifugation plastic tube and is also weighed immediately after the collection of the sample.
c. *Use of Periotron® (Fig. 16.2)*: This is the latest and standard method for measuring gingival fluid absorbed on paper strips. It was developed by Harco Electronics. HAR 600 is an electronic device whose functioning units are a pair of upper and lower counter parts which can be opened and closed in order to insert or remove the strip of filter paper. A moistened strip of paper when inserted between the two jaws will give a reading on the screen. HAR-6000 is the latest technique which was found to be sensitive in detecting small volumes of fluids as compared to the former two models.

Advantages

- It is a simple procedure. It can be viewed directly.
- Quantitative assessment of the fluid can be obtained.
- It seems to be compatible with subsequent chemical analysis.
- By using periotron, evaporation is kept to a minimum.

Disadvantages

- Contamination can occur. In case of evaporation of sample, it has to be repeated many times. It is not very reliable (Ninhydrin technique).

Fig. 16.2: Periotron

- Dislocation of the paper strip, thereby disturbing the integrity of the marginal tissues.
- When Periotron® is used, daily check on the reading accuracy should be performed; care should be taken to insert the paper strips into the machine in a standardized position for correct reading.

Sampling by Means of Micropipettes (Fig. 16.3)

Krasse and Egelberg were the first to utilize capillary tubing. This micropipette permits absorption by capillarity. Capillary tubes of standardized length and diameter are placed in the pockets, their content is centrifuged and analyzed.

Disadvantages

- The collection of fluid is difficult because the viscosity of the fluid makes aspiration through a pipette very difficult.
- Finally the recovery of the sample can also be very demanding.

Gingival Washings

There are two techniques that are available for the study of gingival fluid components.

First method: This was proposed by Tokamoli and Oppenheim; is based on the individual acrylic appliances.

Advantages

- Useful for longitudinal studies.
- Concentrations of various enzymes and the number of cells at the marginal area could be followed by this technique (polymorphonuclear leukocytes and epithelial cells).

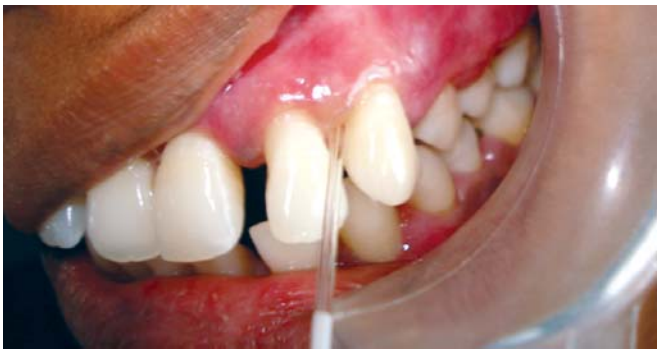

Fig. 16.3: Collection of GCF by micropipettes

- It permits collection of gingival fluid without disturbing the integrity of the marginal tissue.
- Contamination is least by this technique.

Disadvantages

- It is a complex procedure.
- It represents a dilution of crevicular fluid.

Second method: This was proposed by Skapski and Lehner. The procedure involves the ejection and re-aspiration of a known amount of the solution into a given interdental crevice.

Advantages

- It has an advantage of being useful for cases of clinically normal gingiva.
- It is useful for studying the number and functional state of cells and bacteria from the crevicular area.
- Total and differential leukocyte counts can be obtained.

Disadvantage

The technique does not permit absolute quantitative assessments, as the dilution factor cannot be determined.

Other Methods

- Plastic strips
- Platinum loops

The strips are placed along the long axis of the tooth or inserted into the sulcus and pressure is applied.

Volume of gingival fluid: The volume is calculated by using an isotope dilution method. The mean gingival fluid volume in spaces from molar teeth ranged from 0.43 to 1.56 µl. In anterior teeth, the volume was between 0.24 to 0.43 µl/tooth.

Challa Combe calculated an amount of 0.5 to 2.4 µl of fluid/day.

Composition of Gingival Crevicular Fluid (GCF) (Table 16.2)

A. *Cellular elements*
   1. Epithelial cells
   2. Leukocytes
   3. Bacteria

B. *Electrolytes*
   1. Sodium

Table 16.2: Composition of gingival crevicular fluid

	Source	*Action*	*Correlation with disease activity*
A. *Cellular elements*			
1. Epithelial cells	Junctional and sulcular epithelium.		Positive correlation is found.
2. Leukocytes	Dentogingival vessels	Phagocytosis and killing of micro-organisms.	Positive correlation only in some instances.
3. Bacteria	Oral cavity		Poor correlation between bacterial count in gingival fluid and periodontal parameters.
B. *Electrolyte*			
1. Sodium	Plasma and extracellular fluid.	• Genesis of plaque • Precipitation of proteins	Shows positive correlation significantly. Increases in the presence of inflammation.
2. Potassium	Plasma and extracellular fluid.	• Precipitation of mucoprotein along enamel surface.	Positive correlation between potassium concentration and the average pocket depth.
3. Calcium	Plasma and extracellular fluid		Positive correlation.
C. *Organic compounds*			
1. Carbohydrates (glucose, hexosamine and hexuronic acid)	Extracellular fluid		Glucose concentration is increased in inflamed tissues, hexosamine and hexuronic acid has no correlation with variation in gingival inflammation.
2. Proteins a. Immunoglobulins b. Complement	Plasma or synthesized locally Blood	Immune function 1. Control of inflammatory reaction 2. Elimination of antigen 3. Activation of cells 4. Preparation of microbes and foreign particles for phagocytosis. 5. Play a role in immune response.	Positive correlation.
3. Lipids	Serum		
D. *Metabolic and bacterial products*			
1. Lactic acid	Breakdown product of tissue		Positive correlation to both the clinical degree of inflammation and intensity of gingival fluid flow.
2. Hydroxyproline	Breakdown product of collagen		In presence of inflammation, the correlation tends to increase one month after surgery and return to baseline level postoperatively.
3. Prostaglandins	• Synthesized by most mammalian cells. • Are component of inflammatory reaction.	• Vasodilation • Bone resorption • Inhibition of collagen synthesis	It is positively correlated with disease activity.
4. Urea	Break down products of bacteria.	Elevates the pH of supragingival plaque in presence of gingivitis and periodontitis due to production of ammonia by microorganisms.	Urea concentration in gingival fluid decreases when gingival inflammation increases.
5. Endotoxins	These are lipopolysaccharides of cell wall of Gram-negative bacteria.	Highly toxic to gingival tissue	Positively correlated with the presence of varying degree of periodontal inflammation.
6. Cytotoxic substances (like H_2S)	Bacteria	Highly toxic metabolite (cytotoxic effect).	Positively correlated with gingival inflammation.
7. Antibacterial factors	Saliva	Prevents growth of bacteria	

Contd...

Contd...

	Source	*Action*	*Correlation with disease activity*
E. *Enzyme and enzyme inhibitors*			
1. Acid phosphatase	PMNLs and desquamating epithelial cells.	• Associated with connective tissue catabolism. • Attacks teichoic acid, one of the components of bacterial cell wall.	Negative correlation was found between the intercellular concentration of acid phosphatase and both the flow of gingival fluid and the percentage of bone loss.
2. Alkaline phosphatase	In gingival sulcus it is found in PMNLs.	Plays a role in calcification	Positively correlated with pocket depth.
3. Pyrophosphatase	• Plaque • Bacteria	Plays a role in calculus formation	Positive correlation.
4. β-glucuronidase	• It is a hydrolase found in azurophilic granules of PMNLs. • Bacterial plaque • Macrophages, fibroblast, endothelial cells.	Used as lysosomal marker	Positive correlation between the concentration of β-glucuronidase and the flow of gingival fluid and depth of periodontal pocket.
5. Lysozyme	PMNLs	• Bactericidal properties and also some detrimental effect upon epithelial cells. • Lytic effect of connective tissue, thereby contributing to formation of pocket.	Positively correlated with the severe periodontal destruction.
6. Hyaluronidase	Serum	• Widening of intercellular spaces in the junctional epithelium.	Significantly increases in presence of inflammation.
7. Mammalian proteases			
a. Cathepsin-D	Lysosomal enzyme seen in human mononuclear leukocytes.	Attacks various components of epithelium and connective tissue	Its concentration is positively correlated with periodontal destruction
b. Elastase	Azurophil granules of PMNLs	• Active upon elastin, proteoglycans, hemoglobin, fibrinogen and collagen. • Widening of epithelial intercellular spaces, partial destruction of basal membrane and loss of collagen.	Positively correlated with disease progression.
c. Cathepsin-G	Serine endopeptidase contained in azurophil granules of PMNs	• Hydrolyzes hemoglobin, fibrinogen, casein, collagen and proteoglycans.	Positive correlation.
d. Plasminogen activator (Streptokinase, urokinase)	Blood	• Fibrinolysis • Plays a role in inflammation • Essential for wound healing	Concentration increases as severity of periodontitis increases.
e. Collagenase	Specific granules of PMN's	• Collagenolytic activity	Higher concentration in chronically-inflamed gingiva.
f. Bacterial proteases	Bacteria	• Tissue damage	Positive correlation.
g. Serum proteinase inhibitor (α_2 macroglobulin and α_1 antitrypsin)	Plasma	• Modulates the activity of proteases in the tissue.	Positive correlation.
8. Lactic dehydrogenase	Bacteria	• Catalyzes the reversible reduction of pyruvate to lactate.	No significant correlation between total activity of lactic dehydrogenase in gingival fluid and any of the clinical parameters.

2. Potassium
3. Calcium

C. *Organic compounds*
1. Carbohydrates
2. Proteins
   - Immunoglobulins
   - Complement components
3. Lipids

D. *Metabolic and bacterial products*
1. Lactic acid
2. Hydroxyproline
3. Prostaglandins
4. Urea
5. Endotoxins
6. Cytotoxic substances
7. Antibacterial factors

E. *Enzyme and enzyme inhibitors*
1. Acid phosphatase
2. Alkaline phosphatase
3. Pyrophosphatase
4. β-glucuronidase
5. Lysozyme
6. Hyaluronidase
7. *Proteolytic enzymes*
   - Mammalian proteinases
   - Bacterial proteinases
   - Serum proteinase inhibitors
8. Lactic dehydrogenase

Clinical Significance

General Health and Gingival Fluid

A. *Gingival fluid flow and sex hormones*: Three groups of females are studied.
First group–during menstruation: There is increase in the gingival fluid flow because the sex hormones (estrogen and progesterone) cause increase in the gingival vascular permeability.
Second group–females on birth control pills: There is significant increase in the amount of exudate recorded.
Third group–females during pregnancy: The gingival exudates reached maximum values during the last trimester and decreased to minimum after delivery.

B. *Gingival fluid in diabetic patients*: The gingiva of diabetic patients significantly showed higher incidence of vascular modifications, which could be an increase in the width of the basal membrane of capillaries, small arteries and venules, resulting in higher production of gingival fluid. The exudates collected from the diabetic patients showed significantly more levels of glucose than that collected from healthy individuals.

Drugs in Gingival Fluid

Since the gingival fluid seems to be a characteristic feature of gingival inflammation, one could reasonably expect that when suitable drugs are given to a patient, it can be carried from the general circulation to the gingival sulcus or pocket by flow of the fluid.

Bader and Goldhaber (1966) were able to show that intravenous administered tetracycline in dogs rapidly emerges within the sulcus. It seems likely that a major pathway of entrance into oral cavity of systemically administered tetracycline is in the gingival sulcus. The concentration of drug seems to be five times higher in samples of gingival fluid as compared to the concentrations in serum; other drugs that have been detected in human gingival crevicular fluid are minocycline, erythromycin, clindamycin and metronidazole.

Influence of Mechanical Stimuli

Mechanical stimulation of the marginal gingiva, such as massage by means of a round instrument, causes a significant increase in the permeability of the blood vessels located below the junctional and sulcular epithelia. The sensitivity of gingival vasculature has led several investigators to find out whether certain usual powerful mechanical stimuli such as chewing or occlusal overload could influence the rate of gingival fluid production.

The effect of chewing was investigated by Brill (1959) in a group of 15 nurses aged 18 to 22 years, showing clinically healthy gingival margins. Each subject chewed a piece of paraffin for 10 minutes and samples of gingival fluid were recovered by the Brill technique. The amount of gingival fluid was shown to increase significantly under the influence of chewing. Even the minor stimulus in the

form of intrasulcular placement of paper strips increases the production of fluid.

Smoking: Smoking produces an immediate but transient increase in gingival crevicular fluid flow.

Periodontal Therapy and Gingival Fluid

Measurements of gingival fluid flow have been performed before and after different types of periodontal therapy.

Oral prophylaxis: It causes a decrease in the fluid flow, one week after oral prophylaxis and then slowly returned to pre-treatment values.

After surgical procedure: One week after gingivectomy there was a striking increase in the gingival fluid flow due to the increased inflammatory cells in the smear from the sulci. This increase was probably the result of the inflammatory reaction from the gingival trauma with the restoration of gingival integrity, a gradual drop in fluid flow occurred and the scores for the gingival fluid flow reached to minimum values, five weeks after gingivectomy.

KEYPOINTS

1. The structure and function of the gingival marginal area are now known with more precision. In a perfectly sound histologically normal gingiva, a few polymorphonuclear neutrophils can be seen migrating through the junctional epithelium while very little or no gingival fluid can be collected.
2. In a clinically healthy gingiva, a small area of infiltrated connective tissue can be seen and a very little fluid can be collected in the absence of irritation. In the beginning, the fluid seems to contain a low concentration of proteins and could represent interstitial liquid generated locally by an osmotic gradient as a result of an increased permeability of gingival venules; it may progress to a classical inflammatory exudate, containing higher amounts of total protein.
3. The junctional and sulcular epithelia are permeable to a variety of substances. Two methods have been proposed for the collection of material from the gingival sulcus, absorbent paper strips, capillaries, gingival washings and plastic strips or platinum loops are used. The first method, absorbant paper strips are most utilized, even for the quantitative investigations on the composition of gingival fluid. Approximate amount of fluid projected into the oral cavity is 0.5 to 2.4 ml/day.
4. It has been shown, that the absolute number of leukocytes increases with the intensity of the inflammatory process. More than 90 percent of the leukocytes are polymorphonuclear neutrophils and most of them possess the capacity of phagocytosis.
5. Various concentrations of ions in the gingival fluid were demonstrated. The sodium-potassium ratio of the fluid was proved to be positively correlated with the severity of periodontal destruction. The general organic composition of gingival fluid seems to be similar to that of serum. Various immunoglobulins and complement components were also demonstrated. Furthermore, the fluid contains metabolic products which are normally not found in serum or found only in minute concentrations.
6. In preliminary investigations, the pH of fluid was found to be around 7.54 to 7.89. The fluid contains a variety of enzymes, produced both by the cells of the host or by the bacteria.
7. The concentration of lysosomal enzymes in the fluid seems to increase in case of gingivitis and periodontitis, indicating a possible role of the enzymes in the pathogenesis of the lesions. It was also proved that, systemic administration of antibiotics such as tetracycline is found in a higher concentration in human gingival fluid as compared to serum.
8. In conclusion, one can say that the origin, the composition and the clinical significance of gingival fluid are now known with more precision and have significantly helped in the understanding of the pathogenesis of periodontal disease.

KNOW MORE ...

Role of Junctional Epithelium in Antimicrobial Defense

The antimicrobial substances produced by the junctional epithelium such as defensins, lysosomal enzymes, interleukins (IL-1, IL-6, IL-8) and TNF-α can form the first line of defense against microbial invasion into the tissues. The internal and external basal lamina can also act as a barrier against infective agents.

In normal individuals, 30,000 neutrophils per minute enter the oral cavity via gingival sulcus across the junctional epithelium. This flow of neutrophils is important to maintain normal periodontal health. These viable neutrophils present in the saliva are known as orogranulocytes. The oral leukocyte migratory rate index (OMRI) allows an objective assessment of periodontal health.

REVIEW QUESTIONS

1. Describe the functions and significance of gingival crevicular fluid.
2. Describe the methods of collection of gingival crevicular fluid.
3. What is the composition of gingival crevicular fluid?
4. Describe the various defence mechanisms of gingiva.

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17 Chapter

Gingival Inflammation

- STAGE I GINGIVITIS: THE INITIAL LESION
- STAGE II GINGIVITIS: THE EARLY LESION
- STAGE III GINGIVITIS: THE ESTABLISHED LESION
- STAGE IV GINGIVITIS: THE ADVANCED LESION

INTRODUCTION

Inflammation of gingiva is termed as gingivitis. The plaque microorganisms can exert its effect on periodontium by releasing certain products (e.g. collagenase, hyaluronidase, protease, chondroitin sulfatase) which can cause damage to the epithelial and connective tissue constituents. The intercellular spaces between the junctional epithelial cells are destroyed and may permit the bacterial products or bacteria themselves to gain access into the connective tissue (Fig. 17.1).

The sequence of events during the development of gingivitis can occur in four different stages (Table 17.1).

STAGE I GINGIVITIS: THE INITIAL LESION (FIG. 17.2)

Clinically, no visible changes are seen except presence of exudation of fluid from the gingival sulcus, hence this condition is called subclinical gingivitis.

The following features are observed in stage I gingivitis:

1. Classic vasculitis of vessels subjacent to the junctional epithelium.

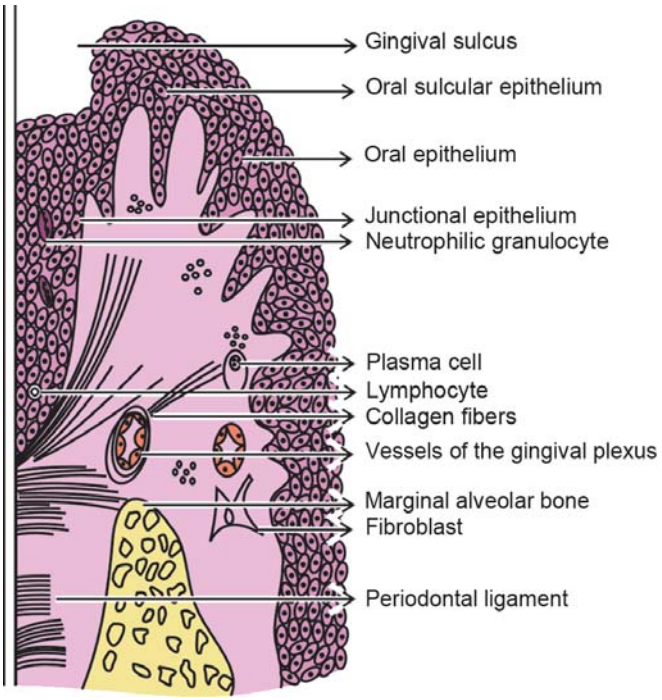

Fig. 17.1: Normal marginal gingiva

2. Exudation of fluid from gingival sulcus.
3. Changes in the coronal most portion of the junctional epithelium.

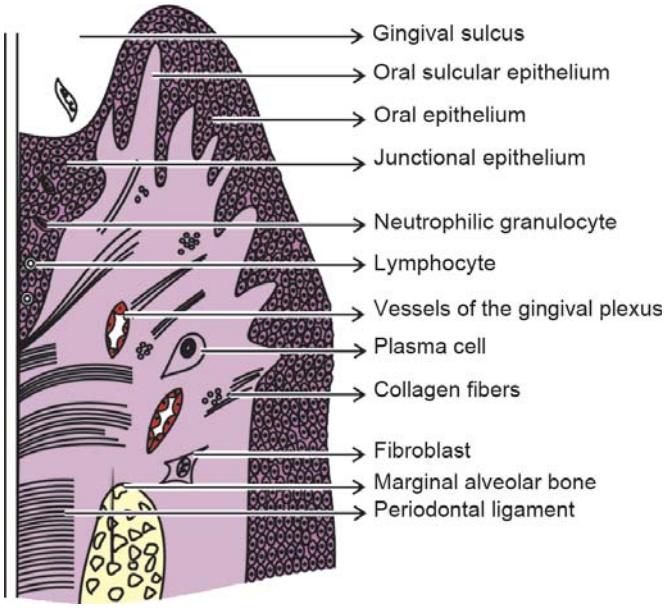

Fig. 17.2: Initial lesion

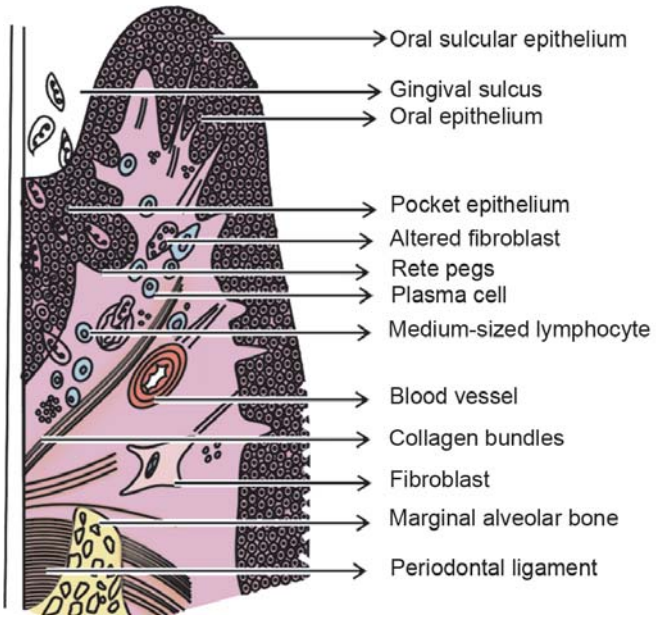

Fig. 17.3: Early lesion

4. Increased migration of the leukocytes into the junctional epithelium and gingival sulcus.
5. Presence of serum proteins.
6. Loss of perivascular collagen.

STAGE II GINGIVITIS: THE EARLY LESION (FIG. 17.3)

Clinically, erythematous gingiva and bleeding on probing may be evident. Microscopic features of the early lesion include:

1. All the changes seen in the initial lesion continue to intensify.
2. The junctional epithelium may begin to show the development of rete pegs or ridges.
3. Accumulation of lymphocytes (majority of them are T cells) beneath the junctional epithelium.
4. Further loss of collagen fiber network supporting the marginal gingiva.
5. Fibroblasts show cytotoxic alteration with a decreased capacity for collagen production.

STAGE III GINGIVITIS: THE ESTABLISHED LESION

Clinical changes include:

a. A bluish hue on the reddened gingiva due to impaired venous return.
b. The gingiva appears to be moderately to severely inflammed.

Microscopically

1. Predominant inflammatory cell types are plasma cells, which invades epithelium and also deep into the connective tissue, around the blood vessels and between the bundles of collagen fibres.
2. Proliferation, apical migration and lateral extension of the junctional epithelium is seen. Early pocket formation may or may not be present.
3. Further collagen destruction and continuing loss of connective tissue substance seen in the early lesion.
4. The following enzyme levels are said to be elevated in chronically inflammed gingiva, acid and alkaline

Table 17.1: Stages of gingivitis

Stage	*Vascular changes*	*Microscopic changes*	*Clinical changes*
1. Initial lesion (2-4 days)	Classic vasculitis subjacent to the junctional epithelium.	Presence of leukocytes (PMNs). Loss of perivascular collagen and presence of serum proteins. Changes in the coronal most portion of junctional epithelium.	Exudation of fluid from the gingival sulcus Subclinical gingivitis.
2. Early lesion (4-7 days)	Vascular proliferation	Rete pegs formation in the junctional epithelium, presence of lymphocytes (mostly T cells), loss of collagen, fibroblasts show cytoplasmic alteration.	Erythematous, gingival bleeding on probing.
3. Established lesion (14-21 days)	Same as early lesion, with blood stasis.	Proliferation,apical migration and lateral extension of junctional epithelium Atropic areas Plasma cells are predominant Further loss of collagen. Increased enzyme levels like acid and alkaline phosphatase, β glucuronidase and others.	Changes are seen in color consistency and surface texture. Bluish hue around the reddened gingiva.
4. Advanced lesion	Same as early and established lesions.	Persistence of features seen in established lesions. Extension of inflammation into deeper structures including alveolar bone and periodontal ligament. Presence of all types of inflammatory cells. Conversion of bone marrow into fibrous tissue.	Formation of periodontal pocket and its associated changes.

phosphatase, β-glucuronidase, aminopeptidase and others.

STAGE IV GINGIVITIS: THE ADVANCED LESION

The advanced lesion is also known as phase of advanced periodontal breakdown.

The following clinical and microscopic features are seen:

1. Persistence of features described in the established lesion.
2. Extension of the lesion into the alveolar bone and periodontal ligament leading to significant amount of bone loss.
3. Continued loss of collagen.
4. Formation of periodontal pockets.
5. Conversion of bone marrow into fibrous tissue.
6. Presence of almost all the types of inflammatory cells.

Progression from Health to Periodontitis

Clinically		*Histologically*
Pristine condition	→	Health
↓		↓
Clinically healthy	→	Initial lesion
↓		↓
Early gingivitis	→	Early lesion
↓		↓
Chronic periodontitis	→	Advanced lesion

KNOW MORE ...

The American Academy of Periodontology (AAP) defines gingivitis as an inflammation confined to the tissues of the marginal gingiva.

Pristine Gingiva

It is a state of superhealth where normal gingiva is free from significant accumulation of inflammatory cells. Hence histologically it may be described as pristine gingiva.

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18 Chapter

Clinical Features of Gingivitis

TYPES OF GINGIVITIS

- Depending on course and duration
- Depending on distribution.

Depending on the Course and Duration

- *Acute gingivitis* is of sudden onset and short duration and can be painful
- *Subacute* is a less severe phase of acute condition
- *Recurrent gingivitis* reappears either after treatment or disappears spontaneously
- *Chronic gingivitis* is slow in onset, of long duration, usually painless and the most commonly occurring gingival condition.

Depending on the Distribution

If the condition is involving a single tooth or group of teeth, it is called localized gingivitis, while generalized gingivitis involves entire mouth.

According to distribution, gingivitis could be marginal, papillary or diffuse. If the inflammation is limited to the marginal gingiva, the condition is termed as marginal gingivitis. In papillary gingivitis, the inflammation is limited to the interdental papilla. When the inflammation spreads to attached gingiva also, it is termed as diffuse gingivitis, i.e. involving marginal, papilla and attached gingiva. Papillary, marginal and diffuse gingivitis can occur as localized or generalized conditions (Figs 18.1 to 18.4).

CLINICAL FINDINGS

While examining the gingiva clinically, one must adapt a systematic approach. Close attention should be given to any tissue alterations, because they contribute to diagnosis. The gingiva is examined for the following characteristics, color, contour, consistency, size, position, severity of bleeding, surface texture (Summary of these features are discussed in detail in Table 18.1).

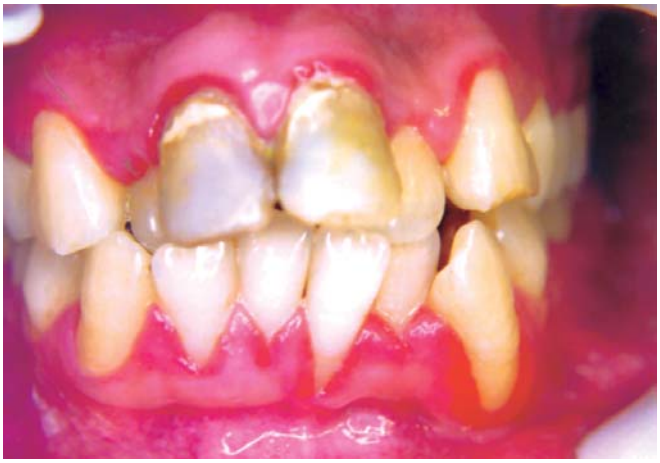

Fig. 18.1: Chronic gingivitis. The marginal and interdental gingiva are smooth edematous and discolored

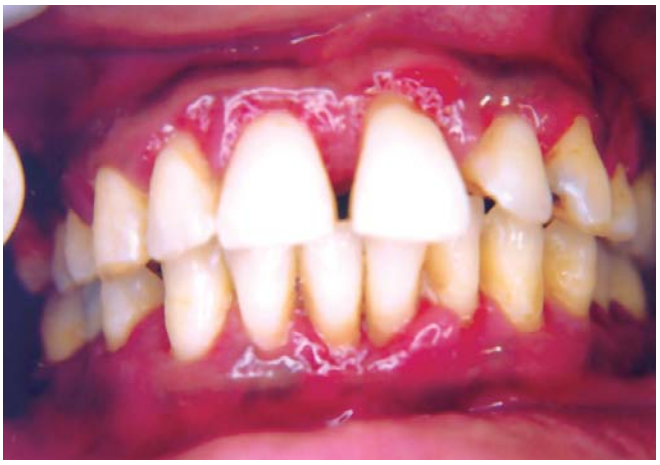

Fig. 18.3: Gingivitis—inflammatory type

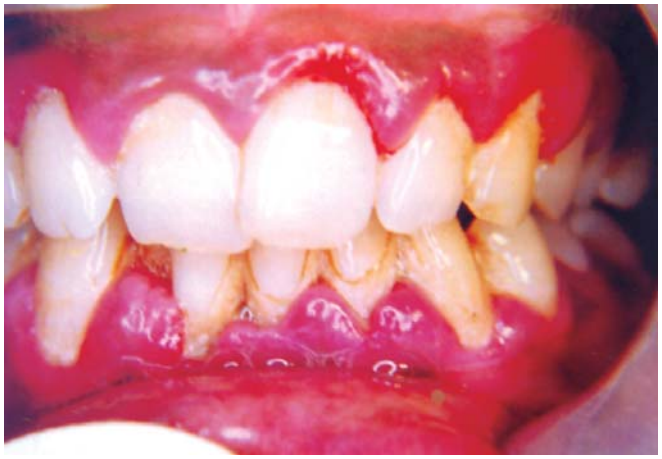

Fig. 18.2: Papillary gingivitis

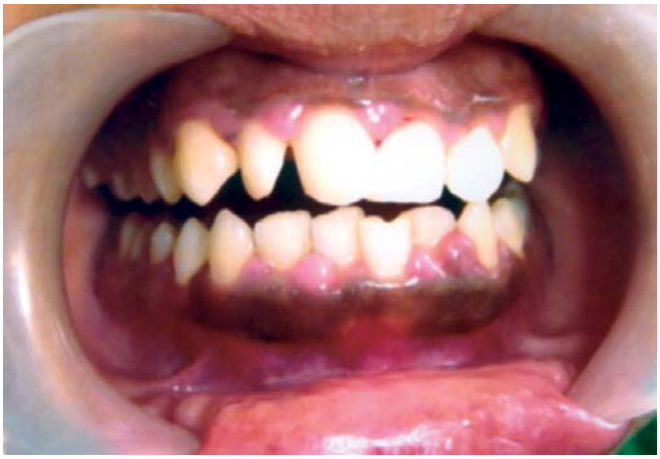

Fig. 18.4: Gingivitis—fibrotic type

Gingival Bleeding on Probing (Fig. 18.5)

1. Significance of gingival bleeding.
2. Etiological factors responsible for gingival bleeding.
3. Associated microscopic changes.

Significance of Gingival Bleeding on Probing

a. It is one of the earliest visual signs of inflammation.
b. It can appear earlier than color changes or any other visual signs of inflammation.
c. It also provides an additional advantage, by being a more objective sign that requires less subjective estimation by the examiner.

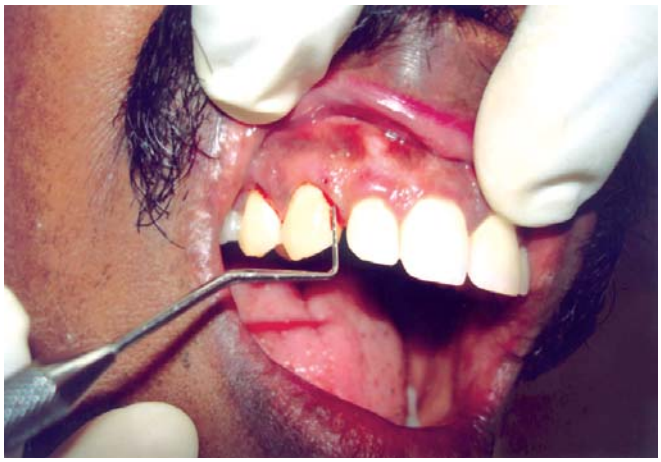

Fig. 18.5: Bleeding on probing

d. Gingival bleeding on probing also helps us to determine whether the lesion is in an active or inactive state. In inactive lesion, there will be little or no bleeding on probing, whereas active lesions bleed more readily on probing.
e. The severity and ease with which bleeding can be provoked indicates the intensity of the inflammation.

Etiological Factors Responsible for Gingival Bleeding on Probing

These are divided into:

a. Local factors:
   - Those factors that result in acute bleeding
   - Those factors that cause chronic or recurrent bleeding.
b. Systemic factors

Acute bleeding is caused due to:

1. Tooth brush trauma.
2. Impactation of sharp pieces of hard food.
3. Gingival burns from hot foods or chemicals.
4. In conditions such as acute necrotizing ulcerative gingivitis (ANUG).

Chronic bleeding: The most common causes are:

1. Chronic inflammation due to the presence of plaque and calculus.
2. Mechanical trauma, e.g. from tooth brushing, tooth picks or food impaction.
3. Biting into solid foods such as apples.

Systemic factors: include various systemic diseases such as:

1. Hemorrhagic diseases including, vitamin C deficiency, vitamin K deficiency, platelet disorders such as thrombocytopenic purpura, other coagulation defects such as hemophilia, leukemia and others.
2. Bleeding could also be as a result of excessive administration of drugs such as salicylates and anticoagulants such as dicumarol and heparin.

Microscopic Changes Associated with Gingival Bleeding on Probing

In plaque-induced gingival inflammation, the following histological changes are seen:

a. *In the epithelium:* Thinning and microulcerations of the sulcular epithelium is seen. Thinning of the epithelium could be due to the toxic substances released by plaque bacteria which destroys the intercellular junctions. Micro-ulcerations are as a result of bacteria trying to gain entry into the connective tissue by breaking through the epithelium or in response to inflammation, the neutrophils from the connective tissue cross the epithelial barrier to reach the site of infection, in doing so they cause ulceration in the epithelium or combination of both.
b. *In the connective tissue:* Dilation and engorgement of the capillaries takes place. Since the capillaries are engorged and closer to the surface which is already thinned and less protective, stimuli that are otherwise innocuous can cause rupture of the capillaries which may result in gingival bleeding.

Color Changes in the Gingiva

Color of the gingiva is an important clinical sign of gingival diseases. Normally gingiva appears to be "*coral pink*". The factors that are responsible for this are tissue vascularity, degree of keratinization and thickness of the epithelium. Generally color of the gingiva may change to red, to bluish-red, to pale-pink. When there is increased vascularity or reduced epithelial keratinization, the gingiva becomes more red. The color becomes pale when vascularization is reduced or epithelial keratinization increases. Venous stasis gives a bluish hue to the gingiva. Detailed description of the changes in color in health and disease is discussed in Table 18.1.

Changes in the color may start from the interdental papilla and later spread to marginal and attached gingiva. Systemically absorbed heavy metals may also cause gingival pigmentation, e.g. bismuth, arsenic, mercury, lead and silver. Abnormal melanin pigmentation of the gingiva may be observed in conditions like Addison's disease, Peutz-Jeghers syndrome, Albright's syndrome and von Recklinghausen's disease.

Changes in the Consistency of Gingiva

Normal gingiva exhibits a firm and resilient consistency. Factors that are responsible are cellular and fluid content

Table 18.1: Gingiva in health and disease

Gingival features	*In health*	*Factors responsible*	*In disease responsible*	*Factors Clinical changes*	*Disease conditions*
1. Color	Coral pink	• Vascular supply • Thickness and degree of keratinization of epithelium • Presence of pigment containing cells	Color change may be • Marginal • Diffuse • Diffuse or patch like.	Chronic gingivitis	• Vascular proliferation (Erythematous) • Reduction of keratinization owing to epithelium compression by inflamed tissues (Erythematous) • Venous stasis (Bluish red) • Tissue necrosis (Bluish red).
			Varying shades of red, reddish blue, deep blue.	Chronic gingivitis	
			Color changes from bright red erythema to • Shiny slate gray • Dull whitish gray.	Acute gingivitis • ANUG/HIV gingivitis • Herpetic gingivostomatitis • Chemical irritation.	
			Black line following the contour of margin.	Bismuth, arsenic and mercury pigmentation	Perivascular precipitation of metallic sulfides in subepithelial connective tissue, only in areas of inflammation due to irritated blood vessel.
			Bluish red or deep blue linear pigmentation (Burtonian line) Violet marginal line.	Lead pigmentation increased permeability Silver pigmentation Systemic diseases causing pigmentation	
2. Contour	Marginal gingiva: Scalloped and knife edged	• Shape of the tooth and thus alignment in the arch	Marginal gingiva becomes rolled or rounded. Interdental papilla becomes blunt and flat	Chronic gingivitis	Inflammatory changes
	Interdental papilla: Anterior: pyramidal Posterior: tent shaped	• Location and size of proximal contact • Dimensions of facial and lingual gingival embrasures	Punched out and crater like depressions at the crest of interdental papilla extending to the marginal gingiva	ANUG	
			Irregularly-shaped denuded appearance	Chronic desquamative gingivitis	
			Exaggerated scalloping	Ginginval recession	
			Apostrophe-shaped indentations extending from and into the gingival margins for varying distances on the facial surface	Stillman's cleft	Described by Stillman as a result of trauma from occlusion and by Box as a pathological pocket.
			Life saver like enlargement of the marginal gingiva (Canine and premolar facial region)	Mc Call's festoon	Enlargement of interdental papilla with no enlargement of marginal gingiva (Pseudocleft)
3. Consistency	Firm and resilient (except free margins)	Collagenous nature of lamina propria and its contiguity with the mucoperiosteum of alveolar bone. Cellular and fluid content of tissue.	• Soggy puffiness that pits on pressure	• Chronic gingivitis	• Infiltration by fluids and cells of inflammatory exudate
			• Marked softness and friability	• Exudative	• Degeneration of connective tissue and epithelium
			• Firm leathery	• Fibrotic	• Fibrosis and epithelium proliferation

Contd...

Contd...

Gingival features	*In health*	*Factors responsible*	*In disease responsible*	*Factors Clinical changes*	*Disease conditions*
			• Diffuse puffiness and softening • Sloughing: grayish flake like particle of debris • Vesicle formation.	Acute gingivitis	• Diffuse edema of acute inflammatory origin • Necrosis with pseudo membrane formation • Intercellular and intracellular edema.
4. Size	Normal	Sum total of the bulk of cellular and intercellular elements and their vascular supply Inflammatory type.	Increased	Gingival enlargement	Increase in fibers and decrease in cells – Non-inflammatory type. Increase in cells and decrease in fibers –
5. Surface texture	Stippling is present (viewed by drying)	• Due to attachment of the gingival fibers to the underlying bone. • Microscopically by alternate rounded protuberance and depressions in the gingival surface. (Papillary layer of connective tissue projects into the elevations).	Loss of stippling • Smooth and shiny • Firm and nodular • Peeling of surface • Leathery texture • Minutely nodular surface.	Gingivitis • Exudative chronic gingivitis • Fibrotic chronic gingivitis • Chronic desquamative gingivitis • Hyperkeratosis • Non-inflammatory gingival hyperplasia.	Due to destruction of gingival fibres as a result of inflammation
6. Position	1 mm above the cemento-enamel junction	• Position of tooth in the arch • Root bone angle • Mesiodistal curvature of tooth surface.	• Apically placed • Coronally placed.	Gingival recession Pseudopockets	• Tooth brush trauma • Gingival inflammation • High frenum attachment • Tooth malposition • Friction from soft tissue.
7. Bleeding on probing		Intact sulcular epithelium and normal capillaries	Present • Chronic recurrent, spontaneous bleeding or bleeding on slight provocation.	• Chronic gingivitis • ANUG Systemic diseases	Dilation and engorgement of capillaries and thinning or ulceration of sulcular epithelium

and collagenous nature of lamina propria. In disease conditions, it can be soggy and edematous or firm and leathery in consistency.

Changes in the Size of the Gingiva

Normal size depends on the sum of the bulk of cellular and intercellular elements and their vascular supply. In disease the size is increased which can be termed as gingival enlargement. The factors responsible for this are increase in fibers and decrease in cells as in non-inflammatory type. Whereas in inflammatory type there will be increase in cells and decrease in fibers.

Surface Texture

Under normal conditions, gingiva appears to be stippled (orange peel appearance) due to attachment of gingival

fibers to the underlying bone. Microscopically, alternate rounded protuberance and depressions in the gingival layer may give rise to stippled appearance. Stippling is absent in disease conditions, hence the gingiva may appear smooth and shiny.

Changes in the Position of Gingiva

Normally the gingiva is attached to the tooth at the cemento-enamel junction. In disease, the position can be shifted either coronally (pseudopocket) or apical to the cementoenamel junction (gingival recession).

Definition

Gingival recession is defined as the exposure of the root surface by an apical shift in the position of the gingiva.

Types

There are two types of recession, i.e. *visible*, which is clinically observable and *hidden*, which is covered by gingiva and can only be measured with probe. Gingival recession may also be *localized* or *generalized*.

Position of the gingiva can be actual or apparent (Fig. 18.6). Actual position is the level of epithelial attachment on the tooth, i.e. from the cementoenamel junction to the probable depth of the pocket, whereas apparent position is the level of crest of the gingival margin, i.e. from the cementoenamel junction to the gingival margin.

Classification of Gingival Recession

Two classification systems are available:

I. *According to Sullivan and Atkins*—Shallow-narrow, shallow-wide, deep-narrow and deep-wide.
II. According to PD Miller's—Class I, Class II, Class III and Class IV.

Class I: Marginal tissue recession that does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This can be narrow or wide.

Class II: Marginal tissue recession that extends to or beyond the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This can be narrow or wide.

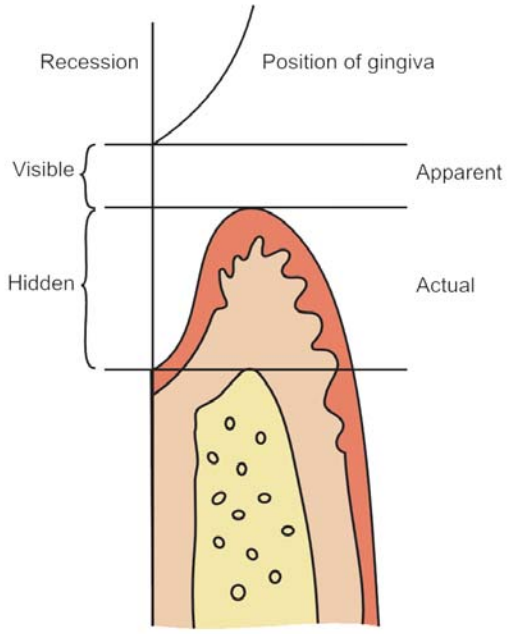

Fig. 18.6: Apparent and actual positions of gingiva

Class III: Marginal tissue recession that extends to or beyond the mucogingival junction. In addition, there is loss of bone and/or soft tissue in the interdental area or there is malpositioning of the tooth.

Class IV: Marginal tissue recession that extends to or beyond the mucogingival junction with severe loss of bone and soft tissue interdentally and/or severe malpositioning of the tooth.

- *Prognosis* of class I and II is good to excellent
- Class III–only partial coverage can be expected
- Class IV–Poor prognosis.

Etiology of Gingival Recession (Figs 18.7 to 18.11)

Plaque-induced gingival inflammation is the primary etiological factor responsible for gingival recession; next most common cause is faulty tooth brushing. Other secondary/contributing factors of gingival recession are broadly categorized (for convenience) as:

a. Anatomic factors
b. Habits

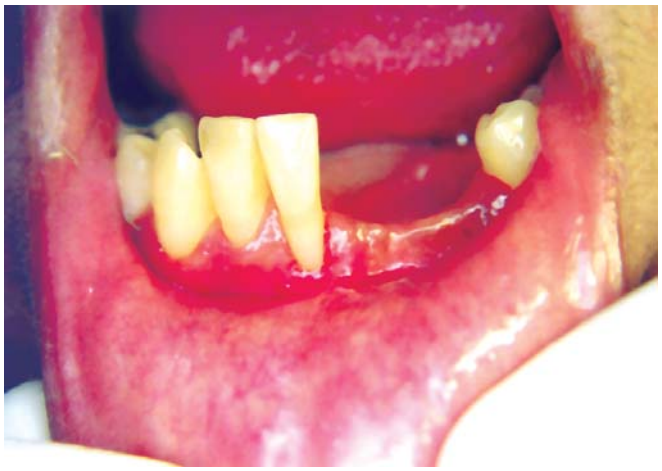

Fig. 18.7: Gingival recession due to inflammation

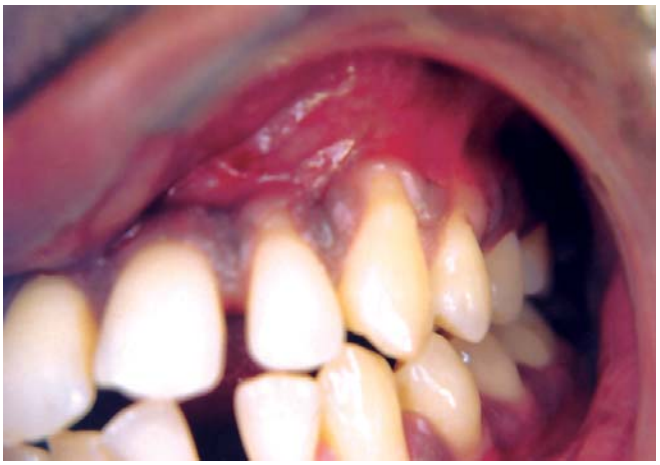

Fig. 18.10: Gingival recession associated with faulty tooth brushing

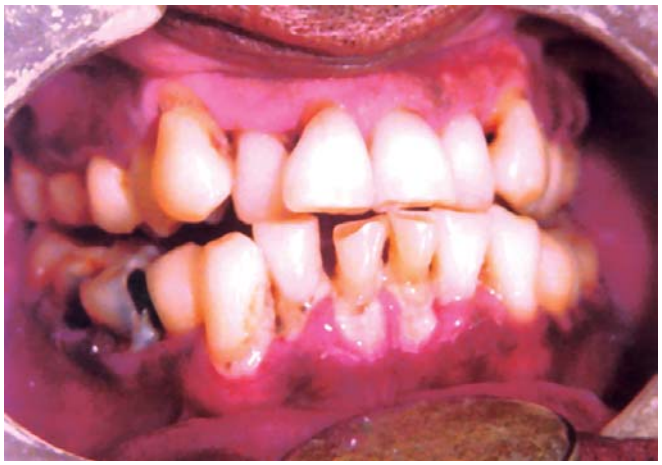

Fig. 18.8: Gingival recession associated with periodontitis

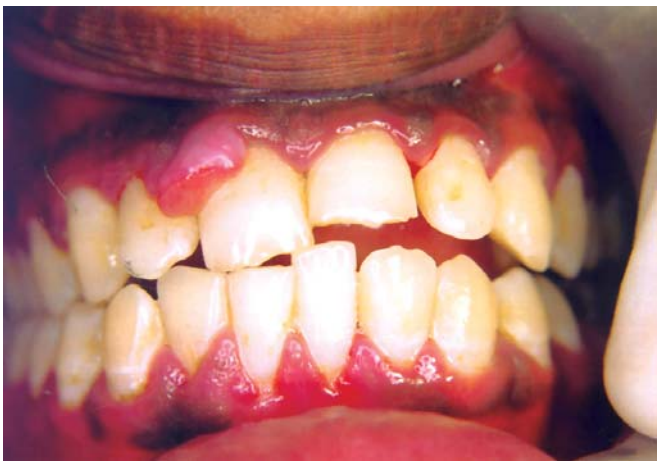

Fig. 18.11: Stillman's cleft in the lower gingiva

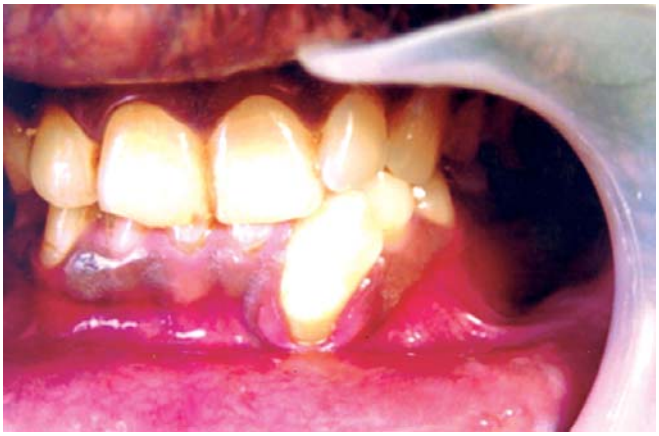

Fig. 18.9: Gingival recession associated with tooth malpositioning

c. Iatrogenic factors
d. Physiologic factors

a. *Anatomic factors include:*
   1. Tooth malposition or position of the tooth in the arch. When a tooth is labially placed, the periodontium on the labial aspect will be invariably thin. When this is exposed to any kind of trauma or frictional forces, gingival recession results.
   2. Presence of dehiscence and fenestrations.
   3. Gingival ablation from soft tissues like cheek, lips, etc.
   4. Root-bone angle and mesiodistal curvature of the tooth surface. In rotated and facially-displaced teeth,

bony plates are either thinned or shortened and recession results from repeated trauma to the thin periodontal tissues.

b. *Habits:* Faulty tooth brushing or brushing with hard bristles may lead to gingival recession. Recently it has been noted that there may be a positive relationship between smoking and recession. But the exact mechanism is not reported.
c. *Iatrogenic factors:* Primary trauma from occlusion has been reported to cause gingival recession. Orthodontic movement in a labial direction and improper restorations can lead to gingival recession.
d. *Physiologic factors:* Gingival recession was thought to be a physiologic process related to aging. However, this idea was discarded because there was no convincing evidence for a physiologic shift of the gingival attachment.

Clinical Significance of Gingival Recession

1. The exposed root surface may be extremely sensitive.
2. Hyperemia of the pulp may result due to gingival recession.
3. Interproximal recession creates oral hygiene problems thereby resulting in plaque accumulation.
4. Finally, it is aesthetically unacceptable.

Changes in Gingival Contour

Normally, marginal gingiva is scalloped and knife edged, whereas interdental papilla in the anterior region is pyramidal and posteriorly tent-shaped. The factors that maintain normal contour are, shape of the teeth and its alignment in the arch, location and size of the proximal contact and dimensions of facial and lingual gingival embrasures. In diseased conditions, the marginal gingiva may become rounded or rolled whereas interdental papilla can become blunt and flat. The various diseased conditions related to gingival contour is given in Table 18.1.

KEYPOINTS

1. Inflammation of the gingiva is termed as gingivitis.
2. Gingivitis can be classified depending on its course and duration and its distribution.
3. Gingiva can be examined for its color, contour, consistency, size, position, surface texture and gingival bleeding on probing.
4. Causes for gingival bleeding on probing could be local factors and systemic factors.
5. Normal color of gingiva is "*coral pink*". In disease it can change to red, bluish-red or pale-pink.
6. Normally, consistency of gingiva is firm and resilient. In diseased conditions, it can exhibit soft and edematous or firm and leathery consistency.
7. Contour is scalloped and knife edged, interdental papilla in the anterior region is pyramidal and posteriorly tent-shaped. In disease, it can become rounded or rolled, whereas interdental papilla can become blunt and flat.
8. Normal stippled gingiva may appear smooth and shiny. In diseased conditions, destruction of gingival fibers is responsible for loss of stippling.
9. Position of the gingiva can be actual or apparent. When position is shifted apically exposing the root surface, it is called as gingival recession.

KNOW MORE ...

Metallic Pigmentation

Heavy metals like bismuth, arsenic mercury, lead and silver absorbed systemically either from therapeutic use or occupational hazard may discolor the gingiva. Gingival pigmentation from systemically absorbed metals results from perivascular precipitation of metallic sulfides in the connective tissue and not as a result of systemic toxicity. It occurs only in the areas of inflammation and can be eliminated by treating the inflammatory changes without necessarily discontinuing medication.

REVIEW QUESTIONS

1. Describe in detail gingiva in health and disease.
2. What are the causes of gingival bleeding of probing?
3. Write about definition, types and etiology of gingival recession.

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2. Newman, Takei, Fermin A Carranza. Clinical Periodontology. 9th edn; WB Saunders Co, 2002.

19 Chapter

Gingival Enlargement

- CLASSIFICATION OF GINGIVAL ENLARGEMENTS
- INFLAMMATORY ENLARGEMENT
  - Acute Inflammatory Enlargement
  - Chronic Inflammatory Enlargement
- NONINFLAMMATORY GINGIVAL ENLARGEMENT
  - Phenytoin-induced Gingival Hyperplasia
  - Idiopathic Gingival Fibromatosis
  - Combined Enlargement
- ENLARGEMENT ASSOCIATED WITH SYSTEMIC DISEASE OR CONDITIONS
  - Conditioned Enlargement
  - Enlargement in Pregnancy
  - Enlargement in Puberty
  - Vitamin C Deficiency
  - Plasma Cell Gingivitis
  - Nonspecific Conditioned Enlargement (Granuloma Pyogenicum)
  - Systemic Disease Causing Gingival Enlargement
  - Granulomatous Diseases
- NEOPLASTIC ENLARGEMENT (GINGIVAL TUMOR)
  - Benign Tumors
  - Malignant Tumors
- FALSE ENLARGEMENT
  - Underlying Osseous Lesions
  - Underlying Dental Tissues

INTRODUCTION

Current clinical descriptive terminology used to describe increase in size of the gingiva is gingival enlargement and gingival overgrowth.

CLASSIFICATION

A. According to etiologic factors and pathologic changes, gingival enlargements could be listed out as:
   I. Inflammatory enlargement
      a. Chronic
      b. Acute
   II. Drug-induced enlargement
   III. Enlargements associated with systemic diseases
      a. Conditioned enlargement
         (i) Pregnancy
         (ii) Puberty
         (iii) Vitamin C deficiency
         (iv) Plasma cell gingivitis
         (v) Nonspecific conditioned enlargement (Granuloma pyogenicum)
      b. Systemic diseases causing gingival enlargements
         (i) Leukemia
         (ii) Granulomatous diseases
   IV. Neoplastic enlargement (Gingival tumors)
      a. Benign tumors
      b. Malignant tumors
   V. False enlargement

B. According to location and distribution, gingival enlargement can be classified as follows:

Localized : Gingival enlargement limited to one or more (group of) teeth.

Generalized : Entire mouth, the gingiva is enlarged
Marginal : Limited to the marginal gingiva
Papillary : Confined to the interdental papilla
Diffuse : Involves all the parts of the gingiva, i.e. marginal, attached and interdental gingiva
Discrete : Isolated sessile or pedunculated tumor-like enlargement

According to the Degree of Gingival Enlargement

Grade 0 : No sign of gingival enlargement
Grade I : Enlargement confined to the interdental papilla
Grade II : Enlargement involves papilla and marginal gingiva
Grade III : Enlargement covers three quarters or more of the crown

INFLAMMATORY ENLARGEMENT (TABLE 19.1)

It can be of two types:

a. Acute inflammatory enlargement
b. Chronic inflammatory enlargement

Acute Inflammatory Enlargement (Table 19.2)

Gingival abscess is a localized, painful, rapidly expanding lesion, that is usually sudden in onset.

Signs and Symptoms

a. It is a rapidly expanding lesion, which is usually limited to marginal gingiva or interdental papilla.
b. It appears as a red swelling with smooth shiny surface which is painful and the associated teeth are sensitive to percussion.
c. The lesion becomes fluctuant and pointed with a surface orifice from which purulent exudate may be expressed. If it is allowed to progress, the lesion may rupture spontaneously.

Etiology

It occurs as a result of bacteria, being carried deep into the tissues when foreign substances such as toothbrush bristle or fragments of food substance are forcefully embedded into the gingiva.

Histopathology

It consists of a purulent focus surrounded by a diffuse infiltration of polymorphonuclear leukocytes, edematous tissue and vascular engorgement. The surface epithelium is ulcerated and shows varying degrees of intra and extracellular edema, invaded by leukocytes.

Periodontal (lateral abscess) also produce enlargement of the gingiva, but they also involve the supporting periodontal tissues.

Table 19.1: Inflammatory enlargement

	Chronic inflammatory enlargement	*Acute inflammatory enlargement*
Etiology	Prolonged exposure to plaque and the factors that favor plaque retention.	From bacteria carried deep into the tissues. When a foreign object like a tooth brush or a lobster shell fragment is forcefully embedded into the gingiva.
Location and Distribution	Generally papillary or marginal gingiva. May be localized or generalized.	*Localized:* Marginal or papillary, e.g. gingival/ periodontal abscess.
Clinical Features	Life preserver like bulge around the involved tooth. Occasionally, occurs as a discrete mass which is sessile or pedunculated. Sometimes painful ulceration in the fluid between marginal and adjacent gingiva.	Painful, rapidly-expanding lesion of sudden onset. Within 24 to 48 hours, it becomes fluctuant and pointed with a surface orifice through which purulent exudate comes out.
Histopathology	Preponderance of inflammatory cells and fluid with vascular engorgement, capillary formation and degenerative changes.	Gingival abscess consists of a purulent focus in the connective tissue surrounded by a diffuse infiltration of polymorphonuclear neutrophils, edematous tissue and vascular engorgement.

Table 19.2: Acute inflammatory enlargement

	Gingival abscess	*Periodontal abscess*	*Periapical abscess*	*Pericoronal abscess*
Location	Localized swelling affecting the marginal and interdental gingiva	Usually affects the deeper periodontal structures including deep pockets, furcations and vertical osseous defects and located beyond the mucogingival junction	Usually seen near the root apex, i.e. in the mucogingival junction and alveolar mucosa	Seen near the incompletely erupted teeth
Etiology	Impactation of foreign objects in previously healthy sites	Periodontal pocket related to destruction by periodontitis	Due to dental caries involving the pulp and its extension into the periapical area	Plaque induced inflammation of the pericoronal flap, i.e. pericoronitis
Associated clinical findings	Gingiva appears to be red, swollen and extremely painful and sometimes impacted foreign object may still be embedded into the gingiva	• Associated with a periodontal pocket which may be either suprabony or infrabony. • Tooth elevation and mobility may be seen. • Tooth is tender on lateral percussion. • Pain is localized and patient can identify the offending tooth. • Affected tooth may be vital or sometimes non-vital. • May be associated with a fistula.	• Mostly deep carious involvement of tooth which is non-vital. • Pocket may or may not be present. Mobility is absent. • Tooth is tender on percussion. • Pain cannot be localized. • May be associated with sinus tract.	• Gingiva overlying the partially erupted or unerupted tooth. • Appears to be red, erythematous, swollen and extremely painful. • Swelling may interfere with occlusion. • Flap can be separated from the tooth with severe food impaction.
Radiographic features	No bone loss is evident	Bone loss is seen. Radiolucency along the lateral aspect of the root.	No bone loss. Periapical radiolucency seen.	Impacted tooth

Treatment of Gingival Abscess

- If the cause of the abscess is still present it should be removed.
- Drainage can be established by warm salt water mouthwashes used every 2 hours.
- If the lesion persists it can be curetted under local anesthesia or incised, if it is pointing.
- If it is persistent and severe systemic, antibiotic may be prescribed.
- Any residual pocketing can be removed by subgingival curettage or localized gingivectomy.

Chronic Inflammatory Enlargement (Fig. 19.1)

Types are:

- Localized
- Generalized
- Discrete/Tumor-like.

Localized/Generalized

- It originates as a slight ballooning of the interdental papilla or marginal gingiva.
- In the early stages, it produces a lifesaver-like bulge around the involved tooth and this bulge increases until it covers part of the crown.

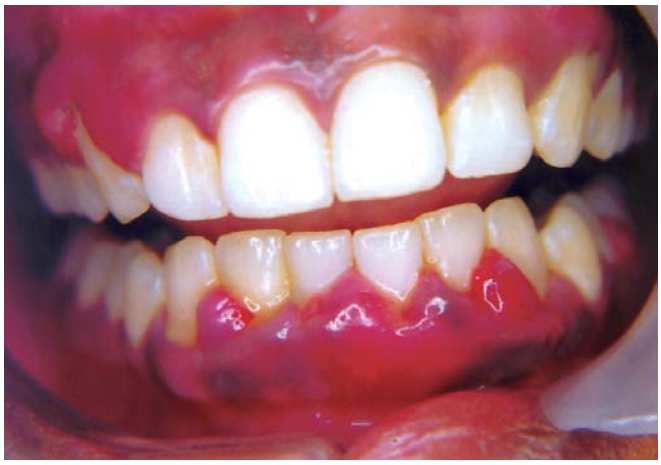

Fig. 19.1: Chronic inflammatory gingival enlargement localized to the lower anterior region

- It progresses slowly and painlessly unless it is complicated by acute infection or trauma.

Discrete/Tumor-like

Occasionally, they may occur as a discrete sessile or pedunculated mass resembling a tumor. It may occur in the interproximal/marginal/attached gingiva.

Histopathology

- Microscopically it can exhibit either exudative/proliferative features depending on the clinical lesion.
- The lesions are clinically deep red or bluish-red, are soft and friable with a smooth-shiny surface and bleed easily.
- They will have preponderance of inflammatory cells and fluid with vascular engorgement, new capillary formation and associated degenerative changes.
- Lesions that are relatively firm, resilient and pink will have a greater fibrotic component with an abundance of fibroblasts and collagen fibers.

Etiology

It is caused by prolonged local irritation. Following are the typical etiological factors; poor oral hygiene, abnormal relationships of adjacent teeth and opposing teeth, lack of tooth function, cervical cavities, overhanging margins of dental restorations, food impaction, irritation from clasps or saddle areas of removable prosthesis, nasal obstructions, habits such as mouth breathing and tongue thrusting.

In mouth breathers:

- Gingivitis and gingival enlargement are often seen.
- The gingiva appears to be red and edematous with a diffuse shiny surface at the exposed area.
- Maxillary anterior region is the common site and their effects are generally attributed to irritation from surface dehydration.

Treatment

a. Scaling and curettage: If the size of the enlargement does not interfere with the complete removal of deposits, the enlargement caused due to inflammation is treated by scaling and curettage.
b. Surgical removal: It is indicated for two reasons:
    i. In enlargement with significant fibrotic component that does not undergo shrinkage following scaling and curettage.
    ii. If the size of the enlargement interferes with the access to the root surface deposits.

Surgical techniques include:

a. Gingivectomy technique: The incision should be at least 1 to 2 mm coronal to the mucogingival line.
b. Flap operation.

NONINFLAMMATORY GINGIVAL ENLARGEMENT (FIBROTIC GINGIVAL ENLARGEMENT) (TABLE 19.3)

- This is produced by factors other than local irritation.
- This condition is uncommon and most of the cases occur due to drugs such as phenytoin, cyclosporine, nifedipine.
- In few cases, diltiazem, verapamil and sodium valproate can also result in fibrotic enlargement.

Phenytoin-induced Gingival Hyperplasia (Fig. 19.2)

Phenytoin is an anticonvulsant drug widely used in the control of epilepsy and other convulsive disorders. It is also used in trigeminal and glossopharyngeal neuralgias.

Table 19.3: Noninflammatory gingival enlargement

	Drug-induced fibrotic enlargement (Phenytoin, cyclosporine, nifedipine)	*Idiopathic gingival enlargement*
Etiology	Long-term therapy of the respective drug	Unknown, possible etiology may be hereditary
Location	Marginal and papillary, generalized	Diffuse enlargement and generalized
Clinical features	• Bead-like enlargement of facial and lingual gingival margins. • Massive tissue folds covering the crowns of teeth interfering with occlusion. • Appears to project from beneath the gingival margin. • Does not occur in edentulous spaces. • More severe in maxillary and mandibular anterior region. • It may occur in mouths with little or no plaque and may be absent in mouths with abundant deposits.	• Facial and lingual surface of maxillary and mandibular teeth are affected but involvement limited to either jaw. • Enlarged gingiva is pink in color, firm and leathery in consistency and has a characteristic pebbled surface. • Enlargement projects into the oral vestibule and jaw appears distorted.
Histopathology	• Hyperplasia of connective tissue and epithelium. • Abundance of amorphous ground substance. • Fibroblast to collagen ratio is equal to that of normal gingiva. • The connective tissue appears highly vascularized in cyclosporine-induced enlargement.	Increase in the amount of connective tissue and consists of densely-arranged collagen bundles and numerous fibroblasts.

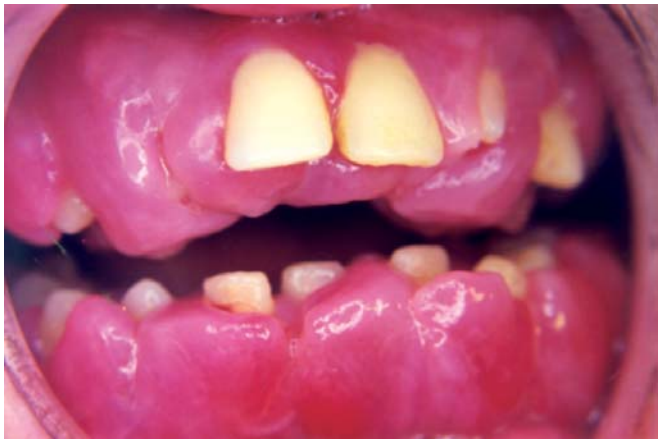

Fig. 19.2: Phenytoin-induced gingival enlargement

Clinical Features

- The overgrowth of the gingiva usually becomes apparent in the first three months after phenytoin dosage and is most rapid in the first year.
- Clinically, it starts as a painless, bead-like enlargement of facial and lingual gingival margins and interdental papillae.
- As the condition progresses, the marginal and papillary enlargement unite and develop into a massive tissue fold covering a considerable portion of the crown and may interfere with the occlusion.
- When uncomplicated by inflammation, the lesion is mulberry-shaped, firm, pale-pink and resilient with a minutely lobulated surface and no tendency to bleed.
- The enlargement characteristically appears to project from beneath the gingival margin, from which it is separated by a linear groove.
- The hyperplasia is usually generalized throughout the mouth, but is more severe in maxillary and mandibular anterior region.
- It occurs in areas in which teeth are present, but hyperplasia of the mucosa in the edentulous mouth has been reported, but is rare.
- The presence of the enlargement will result in a secondary inflammatory process that complicates gingival hyperplasia caused by the drug.
- Secondary inflammatory changes produce red or bluish-red discoloration and result in an increased tendency towards bleeding.

Histopathology

Several changes have been observed in both epithelium and connective tissue.

- The epithelium shows varying degree of acanthosis, with elongated, thin rete pegs/ridges that tend to divide at their ends.
- This can give rise to increased incidence of epithelial pearls.
- The degree of inflammation will determine the presence and extent of polymorphonuclear neutrophils in the gingival epithelium.
- The main change in the lamina propria is proliferation of fibroblasts and increase in the collagen production.

Pathogenesis

There are many theories as to why phenytoin causes gingival overgrowth. The most convincing at present is the direct effect of drug or metabolites on the gingival tissue.

Major metabolite of phenytoin is 5-parahydroxy phenyl or 5-phenylhydantoin (5-P-HPPH).

It is suggested that there are different subpopulation of fibroblasts in gingival tissue, some of which synthesize large amount of protein and collagen (high activity fibroblasts) and others which are only capable of low protein synthesis (low activity fibroblasts).

Hassell suggested that high activity fibroblasts become sensitive to phenytoin, with subsequent increase in collagen production.

Cyclosporine (Fig. 19.3)

- It is a fairly potent immunosuppressive agent used to prevent organ transplant rejection and to treat several diseases of autoimmune origin.
- It appears to be selectively and reversibly inhibit T helper cells, which play a role in cellular and humoral immune responses.
- In 30 percent of the cases, gingival growth was recorded.
- Clinically and microscopically the gingival hyperplasia induced by cyclosporine is similar to that, induced by phenytoin.

Nifedipine

- It is a calcium channel blocker that induces direct dilatation of coronary arteries and arterioles, improving oxygen supply to heart muscle.

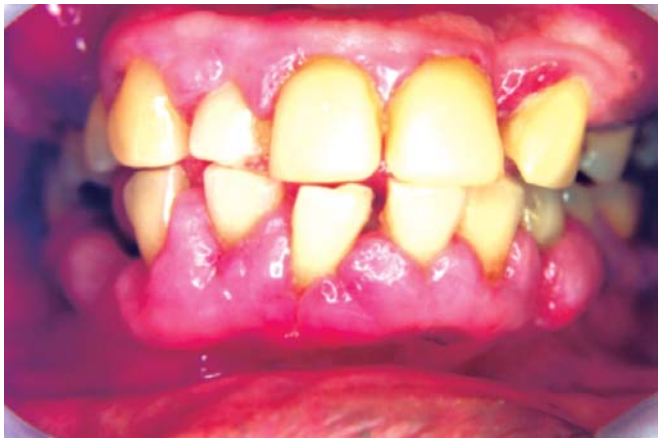

Fig. 19.3: Gingival enlargement associated with cyclosporine therapy

- It also reduces hypertension by dilating the peripheral vasculature.
- Gingival overgrowth occurs in about 20 percent of cases.

Treatment of Drug-associated Gingival Enlargement

Three different types of drugs are associated with gingival enlargement, namely anti-convulsants, calcium channel blockers and the immunosuppressants like cyclosporine. Treatment options are:

First Step

- Oral hygiene reinforcement, chlorhexidine gluconate rinses, scaling and root planing.
- Possible drug substitution. When it is attempted it is necessary to allow at least a period of 6 to 12 months between the discontinuation of the offending drug and the possible resolution of gingival enlargement.
- Professional recalls.

Second Step

If enlargement persists even after following the above mentioned approaches, surgical therapy is indicated. There are two surgical options available based on the features it presents:

- Small areas of enlargement with no attachment loss or bone loss and has good keratinized tissue, gingivectomy is the technique of choice.

- Large areas of enlargement with presence of osseous defects and limited keratinized gingiva, periodontal flap surgery may be indicated.

Idiopathic Gingival Fibromatosis (Fig. 19.4)

Other designated terms include, gingival mitosis, elephantiasis gingivae, diffuse fibroma, idiopathic fibromatosis, hereditary gingival hyperplasia and congenital familial fibromatosis.

Clinical Features

- The enlargement affects the attached gingiva as well as gingival margins and interdental papillae (diffuse).
- The gingiva is firm, pink and leathery in consistency and has a characteristic pebbled surface.
- In severe cases, the teeth are almost completely covered and the gingival enlargement projects into the oral vestibule.
- The jaws appear distorted because of bulbous enlargement of the gingiva. Secondary inflammatory changes are common at the gingival margin.

Histopathology

- The surface epithelium is thickened and acanthotic with elongated rete pegs.
- There is an increase in the amount of connective tissue, which is relatively avascular and consists of densely-arranged collagen fiber bundles and numerous fibroblasts.

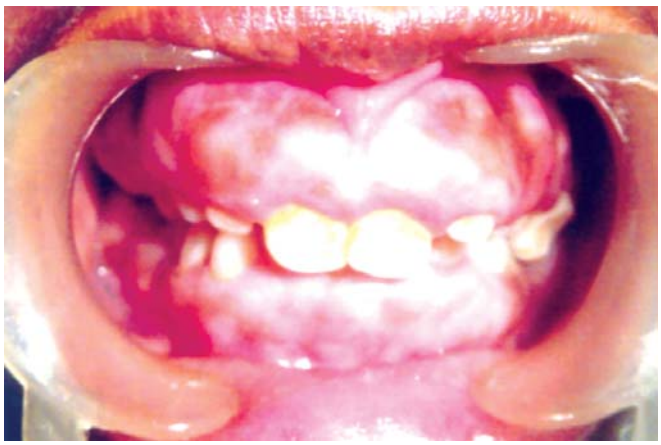

Fig. 19.4: Idiopathic hyperplastic gingival enlargement

Etiology

It is unknown. Some cases have hereditary basis. But the exact mechanism is not well-understood.

Gingival hyperplasia has been described in "tuberous sclerosis", which is an inherited condition characterized by a triad of epilepsy, mental deficiency and cutaneous angiofibromas.

Combined Enlargement

It results when gingival hyperplasia is complicated by secondary inflammatory changes. These changes occur when gingival hyperplasia produces conditions favorable for the accumulation of plaque and interferes with effective oral hygiene measures.

It consists of two components:

a. Primary or basic hyperplasia of connective tissue and epithelium, the origin of which is unrelated to inflammation.
b. Secondary complicating inflammatory component.

Treatment

Gingivectomy/gingivoplasty is indicated.

ENLARGEMENT ASSOCIATED WITH SYSTEMIC DISEASES OR CONDITIONS (TABLE 19.4)

The systemic diseases/condition can affect the periodontium by two different mechanisms:

a. Magnification of an existing inflammation initiated by dental plaque. These groups of diseases include some of the hormonal conditions (e.g. pregnancy and puberty), nutritional diseases such as vitamin C deficiency. Both belong to conditioned enlargement and some cases in which systemic influence is not identified—Conditioned enlargement.
b. Manifestation of systemic disease, independently of the inflammatory status of gingiva. This includes systemic

Table 19.4: Enlargement—associated with systemic disease/conditions

	Pregnancy: Marginal / Pregnancy tumor	Puberty	Vitamin C deficiency	Plasma cell gingivitis (Atypical gingivitis)	Nonspecific conditioned enlargement (Granuloma pyogenicum)	Leukemia
Etiology	• Altered tissue metabolism (increased estrogen and progesterone) • Pregnancy accentuates the *response to local*	Altered endocrine disturbance with local factors	Deficiency of vitamin C	Allergic in origin possibly to chewing gums, dentifrices or various dietary components	Trauma	Malignant neoplasms of leukocyte precursors irritants
Location and distribution	• Marginal • Incidence—10-70% Generalized. More prominent interproximally • Pregnancy tumor 1.8-5% (after third month of pregnancy) • Labial aspect of anterior teeth	Marginal and interdental facial gingiva is enlarged	Marginal gingiva is involved	Marginal and attached gingiva	Localized, discrete	*Incidence:* • Acute monocytic 66.7% • Acute myelocytic monocytic 18.7% • Diffuse or marginal • Localized or generalized
Clinical features	Gingiva is bright-red or magenta in color, soft and friable and has a smooth shiny surface. It has a tendency to bleed spontaneously • Discrete, mushroom-like, spherical mass flattened, spherical mass with sessile or pedunculated base that protrude from gingival margin or interproximal space • Generally dusky-red or magenta, has smooth, shiny surface with numerous deep-red pin point markings, painless, consistency varies from soft and friable to semifirm	• Prominent bulbous interproximal papillae, facial gingiva is enlarged, lingual gingiva is unaltered. • Features are similar to chronic inflammatory gingival disease	Gingiva is bluish-red, soft, friable has smooth, shiny surface. Tissue is spongy, hyperemic and bleeds spontaneously • Surface necrosis with pseudomembrane formation	• Red, friable, sometimes granular and bleeds easily • Located on oral aspect of attached gingiva	• Discrete spherical, tumor like mass with a pedunculated attachment to a flattened, keloid-like enlargement having a broad base • Bright-red or purple and can be friable or firm. Surface ulceration and purulent exudation	Bluish-red, sponge-like and friable, bleeds persistently on slight provocation or spontaneously. Gingival necrosis and pseudomembrane formation is seen.
Histopathology	Keratinized and stratified squamous epithelium, which is thickened with prominent rete pegs. Connective tissue shows numerous newly formed and engorged capillaries with edema and leukocyte infiltration Endothelial proliferation with capillary formation and associated inflammation	Chronic inflammation with prominent edema and associated degenerative changes	Epithelium undergoes thinning and shows spongiosis and may show severe atrophy. Connective tissue shows poorly-formed collagen fibers and many thin-walled and leaking blood vessels	• Oral epithelium is parakeratinized • Ultrastructurally shows signs of damage to spinous and basal layer • Connective tissue shows dense infiltrate of plasma cells	• Mass of granulation tissue with chronic inflammatory infiltrate and endothelial proliferation • Surface ulceration and exudation are common	• Chronic inflammation with mature leukocytes and connective tissue infiltrated with immature and proliferating leukocytes • Isolated areas of acute necrotizing inflammation with pseudomembranous meshwork of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes, bacteria

disease causing gingival enlargement and neoplastic enlargement.

Conditioned Enlargement

- Hormonal (Pregnancy, puberty)
- Nutritional (Vitamin C deficiency)
- Allergic

Local irritation is necessary for the initiation of this type of enlargement. Conditioned enlargement occurs when the systemic condition of the patient exaggerates the usual gingival response to dental plaque.

Enlargement in Pregnancy

Marginal Enlargement

- Results from the aggravation of previous inflammation and does not occur without the clinical evidence of local irritation.
- Pregnancy does not cause the condition. The altered tissue metabolism in pregnancy accentuates the response to local irritation.

Clinical features

- The enlargement is usually generalized and tends to be more prominent interproximally than on the facial and lingual surface.
- The enlarged gingiva is bright-red or magenta, soft and friable and has a smooth, shiny surface.
- Bleeding occurs spontaneously or on slight provocation.

Tumor-like Gingival Enlargement or Pregnancy Tumor

It is not a neoplasm but an inflammatory response to local irritation and is modified by the patient's condition. It usually appears after the first trimester but may also occur earlier.

Clinical features

- The lesion appears as a discrete mushroom-like flattened spherical mass that protrude from the interdental papilla or the gingival margin and is attached by a sessile or pedunculated base.
- It tends to expand laterally and pressure from the tongue and cheek increases its flattened appearance.
- Color—dusky red or magenta with smooth glistening surface that frequently exhibits numerous deep red, pin-point markings.
- Consistency—semifirm, but may have varying degrees of softness and friability.
- It is usually painless, unless complicated by either accumulation of debris under its margin or interference with occlusion—in which case, painful ulceration may occur.

Histopathology

- Both marginal, tumor-like enlargement consists of a central mass of connective tissue, the periphery of which is outlined with stratified squamous epithelium.
- The connective tissue consists of numerous, engorged capillaries and between the capillary network is a fibrous stroma with varying degrees of edema and leukocyte infiltration.
- The epithelium is thickened with varying degree of extra and intracellular edema. The epithelium also exhibits prominent rete pegs.
- Gingival enlargement in pregnancy is also termed as angiogranuloma in order to avoid implication of neoplasm. (Implicated in terms such as pregnancy tumors or fibrohemangioma).

Treatment

- The aim of the periodontal therapy for pregnant patient is to minimize the potential exaggerated inflammatory response related to hormonal alteration.
- Meticulous plaque control, scaling and root planing, polishing should be the only non-emergent periodontal procedures performed.
- The second trimester is the safest time in which treatment may be performed. However, long stressful appointment and periodontal surgical procedures should be postponed until postpartum.
- One must be aware of a condition, 'supine hypotensive syndrome' that occurs during the third trimester which is characterized by a decreased blood pressure, syncope and loss of consciousness. In view of this, the appointments should be kept short and the patient should be allowed to change the position frequently.

- Fully reclining position should be avoided as far as possible.
- Medication and radiographs should not be prescribed.
- In case of marginal and interdental enlargement, scaling and curettage can be performed.
- In case of a tumor-like enlargement, surgical excision is required which, if possible, should be postponed until postpartum. During pregnancy the lesion should be removed surgically only when it interferes with mastication and causes severe disfigurement and if the patient willingly wants to get it removed.

Enlargement in Puberty (Fig. 19.5)

Clinical Features

The enlargement is seen in both marginal and interdental papilla and is characterized by prominent bulbous interproximal papilla.

- Frequently only the facial gingiva is affected, because mechanical action of the tongue prevents accumulation of food on the lingual surfaces.
- Gingival enlargement during puberty has all the clinical features associated with chronic inflammatory gingival disease. It is the degree of enlargement and tendency to develop massive recurrence in the presence of relatively little local irritation that distinguishes pubertal gingival enlargement from uncomplicated chronic inflammatory gingival enlargement.

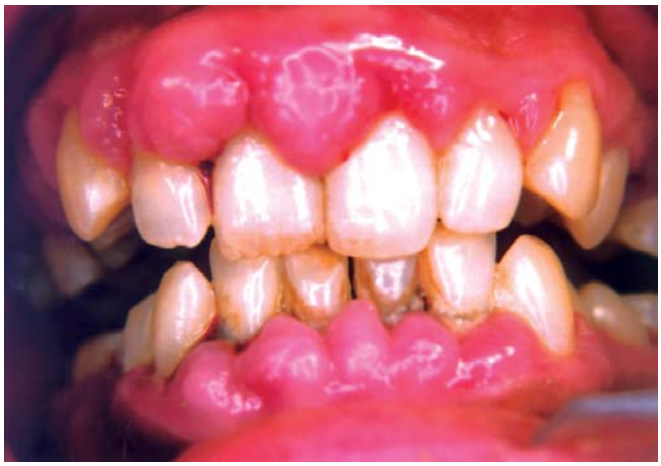

Fig. 19.5: Conditioned gingival enlargement in puberty

- Eleven to seventeen years of age showed a high prevalence of gingival enlargement. The gingival microbiology of children between the ages of 11 to 14 and their association with clinical parameters has implicated Capnocytophaga species in the initiation of pubertal gingivitis.

Histopathology

Same as chronic inflammatory enlargement.

Treatment

Scaling, curettage and oral hygiene instructions. Surgical removal may be performed in severe cases.

Vitamin C Deficiency

Acute vitamin C deficiency does not itself cause gingival inflammation but it causes hemorrhage, collagen degeneration and edema of gingival connective tissue. These changes modify the response of the gingiva to plaque.

Clinical Features

Gingival enlargement is marginal and is bluish-red, soft, friable and has smooth, shiny surface. Hemorrhage occurring either spontaneously or on slight provocation and surface necrosis with pseudomembrane formation are common features.

Histopathology

Areas of hemorrhage with engorged capillaries and marked diffuse edema, collagen degeneration and scarcity of collagen fibers and fibroblasts are striking features.

Plasma Cell Gingivitis

- It is also referred to as atypical and plasma cell gingivostomatitis and frequently consists of a mild-marginal gingival enlargement that extends to attached gingiva.
- Clinically, gingiva appears red, friable and bleeds easily. An associated cheilitis and glossitis have been reported.
- It is thought to be allergic in origin, possibly related to the components of chewing gum or dentrifices.

- Microscopically the connective tissue contains a dense infiltrate of plasma cells, that also extends to oral epithelium.

Nonspecific Conditioned Enlargement (Granuloma Pyogenicum) (Figs 19.6 and 19.7)

It is a tumor-like gingival enlargement that is considered to be an exaggerated conditioned response to minor trauma. The exact nature of the systemic conditioning factor has not yet been identified.

Clinical Features

- The lesion varies from a discrete, spherical tumor-like mass with a pedunculated attachment to a flattened, keloid-like enlargement with a broad base.

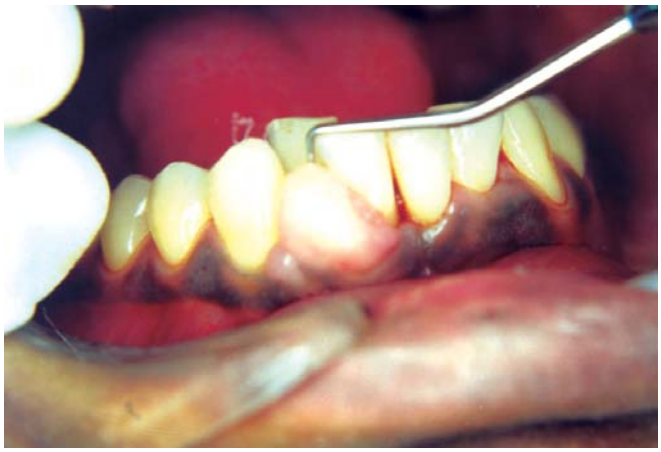

Fig. 19.6: Pyogenic granuloma in a young woman

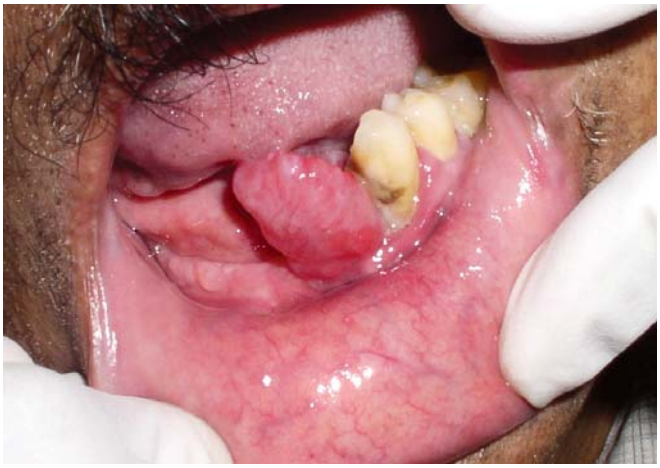

Fig. 19.7: Pyogenic granuloma (*Courtesy:* Dr Deepak Daryani)

- It is a bright-red or purple and either friable or firm, depending on its duration. In majority of the cases it presents with surface ulceration and purulent exudation.
- The lesion tends to involute spontaneously to become a fibroepithelial papilloma or persists relatively unchanged for years.

Histopathology

It appears as a mass of granulation tissue with chronic inflammatory cellular infiltration. Surface ulceration and exudation are common features.

Treatment

Consists of removal of the lesion along with the elimination of local irritating factors.

Systemic Disease Causing Gingival Enlargement

Leukemia

- The enlargement may be diffuse or marginal, localized or generalized. It may appear as an oversized extension of the marginal gingiva or a discrete tumor-like interproximal mass.
- The gingiva appears as bluish-red with a shiny surface.
- The consistency is moderately firm but there is a tendency towards friability and hemorrhage occurring either spontaneously or on slight provocation.
- ANUG may sometimes be seen.
- True leukemic enlargement occurs commonly in acute leukemia but may also be seen in subacute leukemia. It seldom occurs in chronic leukemia (Fig. 19.8).

Histopathology

It shows varying degrees of chronic inflammation with mature leukocytes and areas of connective tissue infiltrated with a dense mass of immature and proliferating leukocytes the specific nature of which varies with type of leukemia.

Isolated surface areas of acute necrotizing inflammation with a pseudomembranous meshwork of fibrin, necrotic epithelial cells, polymorphonuclear neutrophils and bacteria are frequently seen.

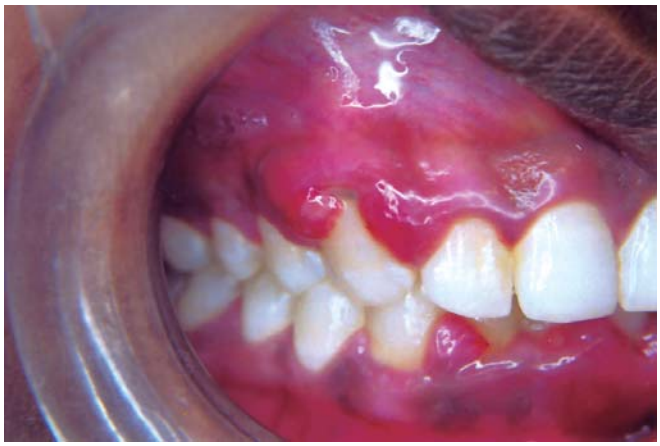

Fig. 19.8: Leukemic gingival enlargement

Treatment

- After the acute symptoms subside, attention is directed to correct the gingival enlargement. The rationale of treatment is to remove the local deposits and to control inflammatory component of the enlargement.
- The enlargement is treated by scaling and curettage which is carried out in stages under topical anesthesia. Oral hygiene procedures are extremely important in these cases.
- Antibiotics are administered systemically the evening before and for 24 hours after each treatment to reduce the risk of infection.

In leukemic patients the following instructions should be followed (in general):

(i) Refer the patient to the physician for medical evaluation and treatment. For this, close cooperation with the physician is required.

(ii) Prior to chemotherapy, a complete periodontal treatment plan should be prepared with the physician, because once the chemotherapy is started the patient will become immunosuppressed, thus increasing the risk of secondary infection.

The treatment plan for these patients is:

a. Monitor hematological lab values daily (bleeding time, clotting time, partial thromboplastin time and platelet count).

b. Administer antibiotics prior to any periodontal therapy.

c. Extract all hopeless, non-maintainable or potentially infectious teeth, at least 10 days prior to initiation of chemotherapy.

d. Thorough periodontal debridement (scaling and root planing) is done and oral hygiene instructions are given. If there is an irregular bleeding time, careful debridement with cotton pellets soaked in hydrogen peroxide may be performed around the neck of the teeth.

During acute phases of leukemia, patients should receive only emergency periodontal care.

A. *If there is a persistent gingival bleeding:* It should be treated as follows:

   i. Cleanse the area with 3 percent hydrogen peroxide.
   ii. Carefully explore the area and remove any etiologic local factors making sure to avoid gingival injury.
   iii. Recleanse the area with 3 percent hydrogen peroxide.
   iv. Place a cotton pellet soaked in thrombin against the bleeding point.
   v. Cover with a gauze and apply pressure for 15 to 20 minutes.
   vi. If oozing persists after the removal of the gauze and pressure, replace the cotton pellet saturated with 3 percent H_2O_2 firmly, then place a periodontal dressing over the area for 24 hours.

B. *NUG:* Follow routine treatment for NUG.

C. *Acute gingival or periodontal abscess:* They are usually associated with regional lymphadenopathy and systemic complications.

Treatment: It is as follows:

i. Systemic antibiotics.
ii. Gentle incision and drainage.
iii. Cleanse the area with cotton pellets saturated with 3 percent hydrogen peroxide.
iv. Apply topical pressure with gauze for 15 to 20 minutes.

D. *Oral ulceration:* It is treated with antibiotics and bland mouth rinses-topical rinses such as viscous xylocaine or promethazine hydrochloride syrup may be prescribed. Topical protective ointments and sharp irritational areas or appliances should be removed.

Oral moniliasis is common in leukemic patient and can be treated with nystatin suspensions.

In Chronic Leukemia

Scaling and root planing can be performed without complication, but an effort should be made to avoid periodontal surgery.

Before every procedure: Bleeding time should be measured. If any changes are seen, postpone the appointment and refer the patient to physician.

Plaque control and frequent recall intervals should be given attention particularly.

Granulomatous Diseases (Table 19.5)

Wegener's Granulomatosis

- It is a rare disease characterized by a granulomatous necrotizing lesion of the respiratory tract, including nasal and oral defects.
- The initial manifestation of Wegener's granulomatosis may involve the orofacial region and include oral mucosal ulceration, gingival enlargement, abnormal tooth mobility, exfoliation of teeth and delayed healing response.
- Clinically the enlargement appears reddish-purple in color and bleeds easily on stimulation. The etiology of Wegener's ganulomatosis is unknown, but the condition is considered as an immunologically mediated tissue injury.
- At one time the usual outcome was death from kidney failure but more recently the use of immunosuppressive drugs has produced prolonged remission.

Sarcoidosis

It is a granulomatous disease of unknown etiology. It starts in individuals in their twenties and thirties and can involve almost any organ, including the gingiva, where a red, smooth enlargement may occur.

NEOPLASTIC ENLARGEMENT (GINGIVAL TUMOR)

Benign Tumors of the Gingiva (Table 19.6)

- Fibroma
- Papilloma
- Peripheral giant cell granuloma
- Central giant cell granuloma
- Leukoplakia
- Gingival cyst.

Malignant Tumors of the Gingiva (Table 19.7)

- Carcinoma
- Malignant melanoma
- Sarcoma most commonly Kaposi's sarcoma
- Metastasis.

Table 19.5: Granulomatous disease

	Wegener's Granulomatosis (Multisystem Granuloma)	*Sarcoidosis*
Etiology	Immunologically-mediated tissue injury	Unknown, but may be impaired cell-mediated immunity
Location and Distribution	Papillary enlargement	Nonspecific
Clinical features	Shows oral mucosal ulceration, reddish purple gingival enlargements, bleeds on stimulation, abnormal tooth mobility, exfoliation of teeth, and delayed healing response	Red, smooth enlargement
Histopathology	Chronic inflammation with giant cells and micro-abscesses covered by thin acanthotic epithelium	Sarcoid granuloma consists of whorls of epithelioid cells and multinucleated Langerhans giant cells with peripheral mono-nuclear cells. Caseation and necrosis do not occur

Table 19.6: Benign tumors of gingiva

	Fibroma	*Papilloma*	*Peripheral giant cell granuloma*	*Central giant cell granuloma*	*Leukoplakia*	*Gingival cyst*
Etiology	Reaction to trauma or chronic irritation	Mostly due to papilloma virus Some unknown	Local injury	History of injury	Tobacco, chronic irritation, alcohol, syphilis, vitamin deficiency, hormones, candidiasis	• Remnants of dental lamina, enamel organ, epithelial islands of periodontal membrane • Traumatic implantation of epithelium
Location and distribution	Localized, diffuse and discrete	Localized, diffuse and discrete	Interdental or from gingival margin. Frequently on labial surface.	Mandible is more commonly involved than maxilla. More common in anterior segment and does not uncommonly cross midline	Buccal mucosa, commissures, alveolar mucosa, tongue, lips, hard and soft palate, floor of the mouth and gingiva	Mandibular bicuspid, cuspid incisor area
Clinical features	Slowly growing spherical mass that tends to be firm and nodular, but may be soft and vascular. Usually pedunculated	Hard, wart-like protuberance from gingival surface	Smooth, regularly outlined masses to irregularly-shaped multilobulated protuberance with surface indentation. Sessile or pedunculated, painless. Firm or spongy and color varies from pink to deep red or purplish blue. Ulcerations are sometimes seen.	Arise within the jaw and produces cavitation. No pain but slight discomfort. Slight to moderate bulging of jaw due to expansion of cortical plate is seen.	Varies from grayish-white, flattened scaly lesion to a thick irregularly-shaped keratinous plaque.	• Involves marginal or attached gingiva • Occurs in mandibular canine or premolar area. • Painless and causes erosion of bone with expansion.
Histopathology	Consists of bundles of interlacing collagen fibers interspersed with varying number of fibroblasts and small blood vessels. The surface is covered by stratified squamous epithelium. Areas of calcification are also seen.	It consists of many long thin, finger-like projections extending above the surface of mucosa, each made up of stratified squamous epithelium and containing a thin central connective tissue core.	It has numerous foci of multinuclear giant cells and hemosiderin particles and chronic inflammatory infiltrate in the connective tissue. Bone formation is occasionally seen. Overlying epithelium is hyperplastic with ulceration at the base.	• It consists of loose fibrillar connective tissue stroma with many proliferating fibroblasts and small capillaries. • Collagen fibers show whorled appearance. • Multinucleated giant cells are prominent. Foci of extra vasculated blood and osteoid are seen.	Thickening of epithelium with hyperkeratosis, acanthosis and some degree of dyskeratosis, inflammatory infiltrate of underlying connective tissues.	Cyst cavity lined by thin flattened epithelium with or without localized areas of thickening.

Table 19.7: Malignant tumors of the gingiva

	Squamous cell carcinoma	*Malignant melanoma*
Etiology	Chronic irritation, tobacco, alcohol, syphilis, nutritional deficiency.	Neoplasms of epidermal melanocytes. Sunlight exposure is possible etiological factor in cutaneous melanoma.
Location and distribution	Mandibular gingiva is more commonly involved than the maxillary gingiva. Attached gingiva is more frequently involved than free gingiva.	Palate, maxillary gingiva and alveolar ridge.
Clinical features	• Exophytic or ulcerative which appears as flat, erosive lesion. • Locally invasive to underlying bone or adjacent mucosa. • It may or may not be painful. • Attached gingiva is more frequently involved than free gingiva. • Metastasis is common.	Deeply-pigmented area at times ulcerated and hemorrhagic which increases in size.
Histopathology	Well-differentiated lesion which consists of sheets and nests of cells with origin from squamous epithelium. Nuclei of neoplastic cells are hyperchromatic. Mitotic figures are atypical. Individual cell keratinization or epithelial pearls are present. Shows rapidly dividing malignant cells. Resembles less to cells of origin.	• Radial growth phase of superficial spreading melanoma is characterized by presence of large, epitheloid melanocytes distributed in pagetoid manner. Vertical growth phase is characterized by proliferation of malignant epitheloid melanocytes into connective tissue. Macrophages and melanophages are present. Melanin pigment is scanty.

FALSE ENLARGEMENT (FIG. 19.9)

These are not true enlargements of gingival tissues but may appear as a result of increase in size of the underlying osseous or dental tissues.

Underlying Osseous Lesions

Enlargement of the gingiva due to enlargement of underling bone occurs most commonly in tori and exostosis but it can also occur in Paget's disease, fibrous dysplasia, cherubism, central giant cell granuloma, osteoma, osteosarcoma.

The gingiva usually presents with no abnormal clinical features except the massive increase in size in the specific area.

Underlying Dental Tissues

During the various stages of eruption of primary dentition, the gingiva may show bulbous, marginal distortion caused by the superimposition of the bulk of the gingiva on the normal prominence of the enamel in the gingival half of the crown. This enlargement has been termed as developmental enlargement.

In strict sense, this type of enlargement is physiologic. However, it may be complicated by marginal inflammation in which case alleviation of the marginal inflammation is sufficient rather than resection of the enlargement.

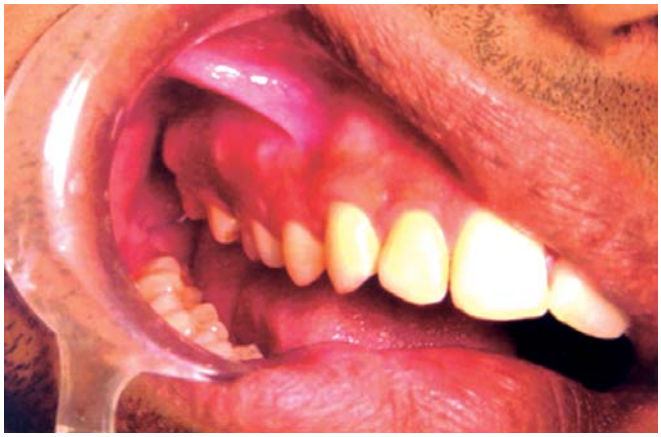

Fig. 19.9: False enlargement in the right upper posterior region

KEYPOINTS

1. Gingival enlargements can be classified according to, etiologic factors and pathologic changes, location and distribution and the degree of gingival enlargement.
2. Inflammatory enlargement can be of two types— acute type, e.g. gingival abscess and chronic type of enlargements.
3. Three categories of drugs have been identified to be associated with gingival enlargements, anticonvulsants, calcium channel blockers and immunosuppressants.
4. The drug-induced enlargements are differentiated from idiopathic enlargement, in the latter the enlargement involves all parts of the gingiva, hence called diffuse enlargement.
5. Treatment options for drug induced enlargements are—drug substitution and surgical therapy including gingivectomy or flap surgery.
6. Enlargements associated with systemic diseases can be:
   a. Magnification of an existing inflammation caused by dental plaque discussed as "conditioned enlargement" which includes some hormonal conditions (e.g. pregnancy and puberty), nutritional diseases such as vitamin C deficiency and some cases in which the systemic influence is not identified (nonspecific conditioned enlargement).
   b. Second category includes, manifestation of the systemic disease not dependent on the inflammatory status of the gingiva discussed as "systemic diseases causing gingival diseases" and "neoplastic enlargement (Gingival tumors)".

KNOW MORE ...

Various Groups of Drugs Associated with Gingival Enlargement

- Anticonvulsants such as Phenytoin, Valproic acid, Succinimides, Phenobarbitone and Ethosuximide
- Immunosuppressants like Cyclosporine and Tacrolimus
- Calcium channel blockers like Nifidipine, Nitrendipine, Amlodipine, Felodipine, etc.
- Other agents include Dilitiazem and Verapamil.

Syndromes Associated with Gingival Fibromatosis

- Gingival fibromatosis with Hypertrichosis
- Zimmerman-Laband syndrome
- Cross syndrome
- Murray-Puretic-Drescher syndrome
- Rutherford's syndrome
- Tuberous sclerosis
- Ramon's syndrome
- Sturge-Weber's syndrome

REVIEW QUESTIONS

1. Classify gingival enlargement. Discuss in detail clinical features, pathogenesis and treatment of drug-induced gingival enlargement.
2. What are the differences between gingival and periodontal abscess?
3. What is granuloma pyogenicum?

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20 Chapter

Acute Gingival Infections

- CLASSIFICATION OF VARIOUS ACUTE GINGIVAL LESIONS
- NECROTIZING ULCERATIVE GINGIVITIS
  - Terminology
  - Clinical Features
  - Intraoral/Extraoral Signs and Symptoms
  - Clinical Course
  - Etiology
  - Histopathology, Diagnosis and Treatment
- ACUTE HERPETIC GINGIVOSTOMATITIS
  - Clinical Features
  - Etiology
  - History
  - Histopathology
  - Diagnosis
  - Differential Diagnosis
  - Treatment
- PERICORONITIS
  - Definition
  - Types
  - Clinical Features
  - Complications
  - Treatment

CLASSIFICATION OF VARIOUS ACUTE GINGIVAL LESIONS

According to Manson

a. Traumatic lesions of gingiva:
   - Physical injury
   - Chemical injury

b. Viral infections:
   - Acute herpetic gingivostomatitis
   - Herpangina
   - Hand, foot and mouth diseases
   - Measles
   - Herpes varicella/zoster virus infections
   - Glandular fever

c. Bacterial infections:
   - Necrotizing ulcerative gingivitis
   - Tuberculosis
   - Syphilis

d. Fungal diseases:
   - Candidiasis

e. Gingival abscess

f. Aphthous ulceration

g. Erythema multiforme

h. Drug allergy and contact hypersensitivity

NECROTIZING ULCERATIVE GINGIVITIS (NUG)

Terminology

Necrotizing ulcerative gingivitis (NUG) is an inflammatory, destructive disease of the gingiva, which presents characteristic signs and symptoms (Fig. 20.1). Other terms used to describe this condition are:

- Vincent's infection
- Trench mouth
- Acute ulceromembranous gingivitis and others.

NUG (necrotizing ulcerative gingivitis) can cause destruction of the supporting structures. When it involves the bone it causes bone loss, the condition is referred as necrotizing ulcerative periodontitis (NUP). Necrotizing ulcerative gingivitis can occur in acute, subacute and recurrent forms.

Clinical Features (Fig. 20.1)

Necrotizing ulcerative gingivitis is characterized by sudden onset, sometimes may be followed by an episode of debilitating diseases or acute respiratory tract infections. Long hours of working without adequate rest and psychologic stress are also frequent features in the history of necrotizing ulcerative gingivitis.

Intraoral Signs and Symptoms

a. *Oral signs:* Lesions are characterized by punched out, crater-like depressions at the crest of the interdental papillae, subsequently involving marginal gingiva and rarely attached gingiva.
b. These craters are covered by grayish pseudomembranous slough, which is demarcated from the remaining of the mucosa by a pronounced linear erythema.
c. The ulcerations of necrotizing ulcerative gingivitis could be of two types—lateral ulceration and necrosis, deep ulceration and necrosis. This is related to the fact that the gingival tissues are supplied by two main sources of blood supply. Supraperiosteal vessels supplying the attached gingiva, lateral margins and lateral parts of the papillae, while intra-alveolar vessels supplying to the col and central portions of the papillae.

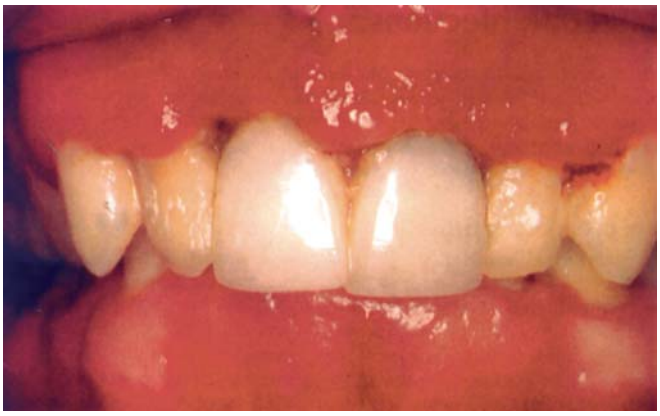


Fig. 20.1: ANUG (Punched out interdental papilla between two central incisors)

Lateral ulceration, which is (less common) involving primarily the buccal wall of the papillae, margins and possibly the attached gingiva, occurs in the distribution of the lateral blood supply. Deep ulceration involves necrosis of the tissues of the embrasure giving rise to typical truncated papillae, occurs in the distribution of the intra-alveolar vessels (more common).

d. Other signs include gingival hemorrhage or pronounced bleeding on the slightest stimulation.
e. Fetid odor and increased salivation.

Oral Symptoms

1. The lesions are extremely sensitive to touch.
2. Complains of a constant radiating, gnawing pain that is intensified by eating spicy or hot foods and chewing.
3. There is a metallic foul taste, and the patient is conscious of an excessive amount of "pasty saliva".

Extraoral Signs and Symptoms

In mild to moderate stages of the disease local lymphadenopathy and a slight elevation in temperature are common features. In severe, cases marked systemic complications such as high fever, increased pulse rate, leukocytosis, loss of appetite and general lassitude are common. Systemic reactions could be more severe in children. In rare cases, severe sequelae such as the following may occur, noma or gangrenous stomatitis, fusospirochetal meningitis, peritonitis, toxemia, and fatal brain abscess.

Clinical Course

It is indefinite, if left untreated, it may lead to destruction of the periodontium, and denudation of roots (necrotizing ulcerative periodontitis), combined with severe toxic systemic complications.

Horning and Cohen have described the following stages in the progression of necrotizing ulcerative gingivitis (NUG) (Table 20.1).

Etiology

Role of Bacteria

Plaut and Vincent introduced the concept that necrotizing ulcerative gingivitis is caused by a specific bacteria namely, a fusiform bacillus and a spirochetal organisms. More recently Loesche and colleagues described a constant and a variable flora associated with ANUG.

Constant flora is composed of fusospirochetal organisms and also *Bacteroides intermedius*. The variable flora consists of a heterogeneous array of bacterial types. These bacteriologic findings have been supported by immunologic data, increased IgG and IgM antibody titers to spirochetes (intermediate sized up to 90%) and *Prevotella intermedia* has been demonstrated. Electron microscopic studies have demonstrated three types of spirochetes, small, intermediate sized (maximum number up to 90%) and large spirochetes. The specific cause of necrotizing ulcerative gingivitis has not been established. The common opinion is that it is produced by a complex of bacterial organisms but requires underlying tissue changes to facilitate the pathogenic activity of the bacteria.

Local Predisposing Factors

Most important predisposing factors are:

i. Pre-existing gingivitis
ii. Injury to the gingiva
iii. Smoking

Deep periodontal pockets, pericoronal flaps are particularly vulnerable areas for the occurrence of the disease because they offer a favorable environment for the proliferation of the fusospirochetes and hence, they are called as incubation zones.

Areas of the gingiva traumatized by opposing teeth in malocclusion, such as the palatal surface behind the maxillary incisors and the labial gingival surface of the mandibular incisors are frequent sites of necrotizing ulcerative gingivitis.

Many investigators have reported a positive correlation between smoking and acute necrotizing ulcerative gingivitis. The possible reasons are:

- Direct toxic effect of tobacco on the gingiva.
- Vascular or other changes induced by nicotine or other substances.
- Smoking and necrotizing ulcerative gingivitis are both reflections of stress.

Systemic Predisposing Factors

Nutritional deficiency: A poor diet has been reported as a predisposing factor in NUG. Nutritional deficiencies such as vitamin B and vitamin C accentuate the response of gingival tissues produced by increased pathogenic flora. Several researchers have found an increase in the fusospirochetal flora in patients with nutritionally-deficient diets.

Table 20.1: Stages in the progression of NUG

Stages	*Involvement of the lesion*	*% of cases*	*Clinical conditions*
1.	Necrosis of the tip of the interdental papilla	93	NUG Necrotizing ulcerative gingivitis
2.	Necrosis of the entire papilla	19	NUG or NUP Necrotizing ulcerative gingivitis or necrotizing ulcerative periodontitis
3.	Necrosis extending to the gingival margin	21	Necrotizing ulcerative periodontitis
4.	Necrosis extending also to the attached gingiva	1	Necrotizing ulcerative periodontitis
5.	Necrosis extending into buccal or labial mucosa	6	Necrotizing stomatitis
6.	Necrosis exposing alveolar bone	1	Necrotizing stomatitis
7.	Necrosis perforating skin of cheek	0	Noma

Debilitating diseases: May predispose to the development of acute necrotizing ulcerative gingivitis. Such systemic disturbances are metallic intoxication, severe gastrointestinal disorders, blood dyscrasias such as anemia, leukemia and acquired immunodeficiency syndrome.

Psychosomatic factors: Appears to be important in the etiology of acute necrotizing ulcerative gingivitis. The mechanisms whereby psychological factors create or predispose to gingival damage have not been established, but an alteration in digital and gingival capillary responses suggestive of increased autonomous nervous activity has been demonstrated. Cohen and coworkers have suggested that a psychiatric disturbance may lead to activation of the hypothalamic pituitary adrenal axis. This results in elevation of serum and urine cortisol levels, which is associated with a depression of lymphocyte and polymorphonuclear leukocytes function that may predispose to necrotizing ulcerative gingivitis.

Histopathology

Microscopically, the lesions involve both epithelium and underlying connective tissue. The surface epithelium is destroyed and is replaced by a pseudomembranous mesh work of fibrin, necrotic epithelial cells, polymorphonuclear neutrophils and various types of microorganisms. This is the zone that appears clinically as the surface pseudomembrane. The underlying connective tissue is markedly hyperemic, with numerous engorged capillaries and dense infiltration of polymorphonuclear neutrophils. This acutely-inflamed hyperemic zone appears clinically as the surface pseudomembrane.

Relationship of Bacteria to the Characteristic Lesions

Listgarten and colleagues described four zones namely:

1. *Zone I—Bacterial zone:* It is the most superficial zone, consists of varied bacteria, including a few spirochetes of the small, medium-sized and large types.
2. *Zone II—Neutrophil-rich zone:* Contains numerous leukocytes predominantly neutrophils with bacteria including spirochetes of various types.
3. *Zone III—Necrotic zone:* Consists of a dead tissue cells, remnants of connective tissue fragments, and numerous spirochetes.
4. *Zone IV—Zone of spirochetal infiltration:* Consists of a well preserved tissue infiltrated with spirochetes of intermediate and large-sized without other organisms.

Diagnosis

Diagnosis is based on clinical findings. A bacterial smear may be used to corroborate the clinical diagnosis, but it is not necessary nor definitive because the bacterial picture is not appreciably different from the other conditions.

Differential diagnosis includes:

a. Gonococcal stomatitis
b. Agranulocytosis
c. Vincent's angina
d. Desquamative gingivitis
e. Necrotizing ulcerative gingivitis in leukemia
f. Necrotizing ulcerative gingivitis in AIDS
g. Streptococcal gingivostomatitis.

Treatment

1. *Non-ambulatory patient:* With symptoms of generalized systemic complications.
2. *Ambulatory patient:* With no serious systemic complications.

Treatment for Non-ambulatory Patients

Day 1:

a. Local treatment limited to gently removing the necrotic pseudomembrane with a pellet of cotton saturated with hydrogen peroxide (H_2O_2).
b. Advised bed rest and rinse the mouth every 2 hours with a diluted 3 percent hydrogen peroxide (H_2O_2).
c. Systemic antibiotics like penicillin or metronidazole can be prescribed.

Day 2: If condition is improved, proceed to the treatment described for ambulatory patients. If there is no improvement at the end of the 24 hours, a bedside visit should be made. The treatment again includes gently swab

the area with hydrogen peroxide, instructions of the previous day are repeated.

Day 3: Most cases, the condition will be improved, start the treatment for ambulatory patients.

Treatment for Ambulatory Patients

First visit: A topical anesthetic is applied and after 2 or 3 minutes the areas are gently swabbed with a cotton pellet to remove pseudomembrane and non-attached surface debris. After the area is cleansed with warm water the superficial calculus is removed with ultrasonic scalers. Patients with moderate or severe necrotizing ulcerative gingivitis and local lymphadenopathy, are placed on antibiotic regime of Amoxicillin 500 mg thrice daily, for penicillin-sensitive patients Azithromycin 500 mg once a day for three days or Metronidazole 200 mg or 400 mg twice daily for seven days.

Subgingival scaling and curettage are contraindicated at this time because of possibility of extending the infection to deeper tissues.

Instructions to the patient

1. Avoid smoking and alcohol.
2. Rinse with 3 percent hydrogen peroxide and warm water for every two hours.
3. Confine toothbrushing to the removal of surface debris with a bland dentifrice, use of interdental aids and chlorhexidine mouth rinse are recommended.

*Second visit:*Scalers and curettes are added to the instrumentarium. Shrinkage of the gingiva may expose previously covered calculus which is gently removed. Same instructions are reinforced.

Third visit: Scaling and root planing are repeated, plaque control instructions are given. Hydrogen peroxide rinses are discontinued.

Fourth visit: Oral hygiene instructions are reinforced and thorough scaling and root planing are performed.

Fifth visit: Appointments are fixed for treatment of chronic gingivitis, periodontal pockets and pericoronal flaps, and for the elimination of all local irritants. Patient is placed on maintenance program.

Further Treatment Considerations

1. Gingivoplasty.
2. Role of drugs (escharotic drugs and silver nitrate, hydrogen peroxide, sodium perborate).
3. Systemic antibiotics—only in patients with toxic systemic complications.
4. Supportive systemic treatment—copious fluid consumption and administration of analgesics and adequate bed rest.
5. Nutritional supplements—vitamin B/C supplements.

ACUTE HERPETIC GINGIVOSTOMATITIS (AHG) (FIGS 20.2 AND 20.3)

It is a viral infection of the oral mucous membrane caused by HSV I and II (Herpes simplex virus). It occurs most frequently in infants and children younger than 6 years of age but is also seen in adults.

Clinical Features

Primary Gingivostomatitis

Oral Signs

1. It appears as a diffuse, shiny erythematous, involvement of the gingiva and the adjacent oral mucosa with varying degrees of edema and gingival bleeding.
2. In its initial stage it may appear as discrete, spherical, clusters of vesicles dispersed in different areas, e.g. labial and buccal mucosa, hard palate, pharynx and tongue.

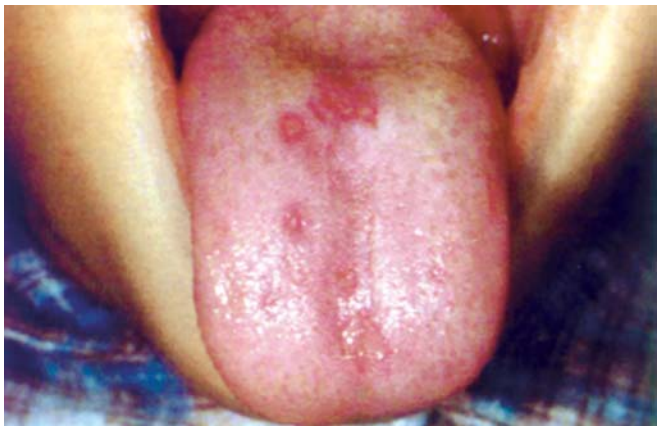


Fig. 20.2: Vesicles on the tongue in primary herpetic gingivostomatitis

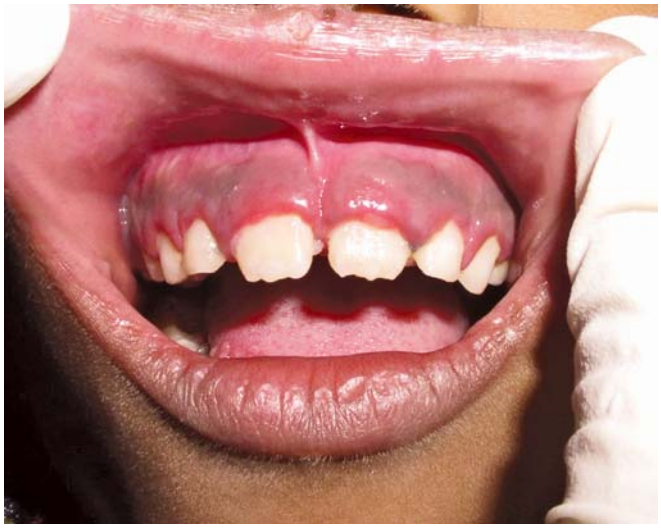


Fig. 20.3: Primary herpetic gingivostomatitis in a 10-year-old patient with diffuse erythematous involvement of the gingiva (*Courtesy:* Dr Deepak Daryani)

After approximately 24 hours the vesicles rupture and form painful shallow ulcers with scalloped borders and surrounding erythema.

3. Diffuse, edematous, erythematous enlargement of the gingiva with a tendency towards bleeding is seen.
4. The course of the disease is 7 to 10 days.

Oral Symptoms

1. Generalized soreness of the oral cavity which interferes with eating and drinking.
2. The ruptured vesicles are sensitive to touch, thermal changes and food.

Extraoral and Systemic Signs and Symptoms

There is a 1-3 day prodrome of fever, loss of appetite and myalgia. Cervical lymphadenopathy is present.

Recrudescent Oral HSV Infection

After the primary infection the virus remains latent in the nerve tissue. If reactivation occurs it causes Herpes labialis (cold sore). It is associated with prodrome of tingling and itching on the corners of lip followed by vesicle formation and ulceration (Fig. 20.4).

Etiology

Acute herpetic gingivostomatitis is caused by HSV (*Herpes simplex* virus) with a size of approximately 100 to 200 µm. When fully formed, it consists of a core containing genetic material (DNA) surrounded by a capsid. The capsid is made up of a hexagonal particle called capsomers. The capsid in turn is surrounded by a membrane or envelope. The fully grown virion penetrates the cell membrane, once inside, the virion loses, its coating and later it gains entrance into the nucleus. In the nucleus the DNA of the virus converts the host nucleus and codes the host DNA for the production of material necessary for viral replication.

Herpes viruses that cause orofacial diseases in humans are:

1. *Herpes virus type 1—(Infections above the waist)* oropharyngeal lesions responsible for acute herpetic gingivostomatitis (HGS) and cold sores.
2. *Herpes virus type 2—(Infections below the waist)*– Also affects the mouth with changing sexual practices
3. *Cytomegalovirus*—Severe Oral Ulcerations
4. *Varicella zoster virus*—responsible for chickenpox and herpes zoster (shingles)
5. *Epstein-Barr virus*—responsible for infectious mononucleosis and Hairy Leukoplakia
6. *Human herpes virus 8*—Kaposi's sarcoma.

Histopathology

Vesicles rupture to form a discrete ulceration, which appears to have a central portion of acute inflammation characterized

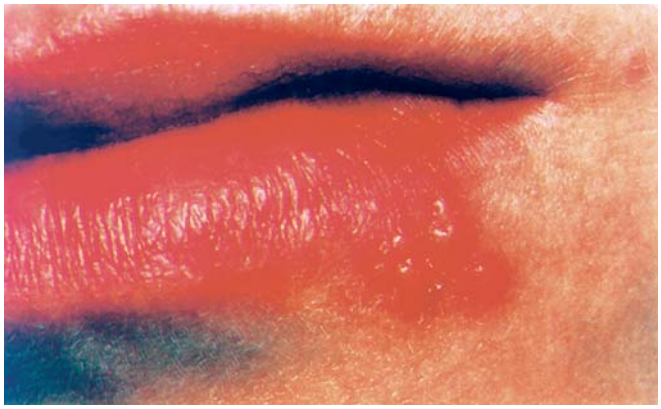


Fig. 20.4: Herpetic vesicles (*Herpes labialis*)

Table 20.2: Distinction between necrotizing ulcerative gingivitis and primary herpetic gingivostomatitis

Necrotizing ulcerative gingivitis (NUG)	*Primary herpetic gingivostomatitis (PHG)*
Etiology: Host bacterial interaction, mostly fusospirochetes	Specific viral etiology
Necrotizing condition	Diffuse erythema and vesicular eruptions
Punched out crater-like lesions affecting marginal gingiva. The lesions are covered with pseudomembranous slough, other oral tissues are rarely affected.	Vesicles rupture leaving slightly depressed oval and spherical ulcer. Diffuse involvement of gingiva, may include buccal mucosa and lips
Uncommon in children	Occurs more frequently in children
No definite duration	Duration of 7 to 10 days
Not contagious	Contagious

by ulceration and varying degrees of purulent exudate, surrounded by a zone rich in engorged blood vessels.

The microscopic picture of the vesicle is characterized by extra- and intracellular edema and degeneration of the epithelial cells. The cell cytoplasm appears to be liquefied and clear. Later the nucleus also degenerates. The vesicle formation results from fragmentation of the degenerated epithelial cells. Occasionally, round eosinophilic inclusion bodies are found in the nuclei of epithelial cells. These inclusion bodies may be a colony of virus particles, degenerated protoplasmic remnants of the affected cell, or a combination of both, called Lipschutz's bodies. Connective tissue is infiltrated by plasma cells. Smear obtained is Tzanks smear and the stain used is Giemsa's stain.

Diagnosis

It is usually established from the patients' history and the clinical findings. For confirmatory, tests the material may be obtained from the lesion and submitted to the laboratory.

1. HSV isolation by cell culture is the gold standard
2. Polymerase Chain Reaction from swabs obtained by scraping oral lesions
3. *Tzanck smear:* The material is obtained from the base of the lesion and smeared and stained. The finding of multinucleated cells with swelling, ballooning and degeneration is adequate for diagnosis.

Differential Diagnosis

1. Necrotizing ulcerative gingivitis (Table 20.2)
2. Erythema multiforme
3. Stevens-Johnson syndrome
4. Aphthous stomatitis (Canker sores).

Treatment

Primary Gingivostomatitis is treated with topical lignocaine for pain relief. Acyclovir at 15 mg/kg five times a day for 5-7 days reduces the duration of fever, halts the progression of lesions and reduces infectivity.

Herpes labialis can be managed with topical antiviral medications such as 5% acyclovir cream or 3% Penciclovir cream applied three to five times a day at the first sign of the lesion.

Recurrent Aphthous Stomatitis (RAS)

Recurrent aphthous stomatitis (RAS; aphthae; canker sores) is a common condition which is characterized by multiple recurrent small, round or ovoid ulcers with circumscribed margins, erythematous halo, and yellow or gray floors typically presenting first in childhood or adolescence.

The lesions may occur anywhere in the oral cavity, the buccal and labial mucosae are common sites. It's a painful lesion and may occur as a single lesion or as lesions scattered throughout the mouth. The duration of each lesion is 7 to 10 days.

Aphthous stomatitis may occur in the following forms:

Minor aphthae: Is the most common affecting about 80% of patients with RAS: ulcers are round or oval usually <5 mm in diameter with a gray-white pseudomembrane and an erythematous halo. The ulcers heal within 10-14 days without scarring.

Major aphthae: Is a rare severe form of Aphthous ulcer. Ulcers are oval and may exceed 1 cm in diameter. Ulcers persist for up to 6 weeks and often heal with scarring.

Herpetiform aphthae: Is the least common variety and is characterized by multiple recurrent crops of widespread small, painful ulcers. As many as 100 ulcers may be present at a given time, each measuring 2-3 mm in diameter.

Etiology

It is unknown. Major factors linked to RAS are genetic predisposition, Hematinic deficiencies, immunologic abnormalities, stress, food allergy and gastrointestinal disorders. Predisposing factors include hormonal disturbances, trauma, cessation of smoking and menstruation.

Treatment

Various medications have been used in the treatment of this condition including:

a. *Local applications:* Using topical lignocaine and benzocaine in mild cases. Topical steroids like Triamcinolone and Clobetasol application in severe cases shortens healing time.
b. *Systemic therapy:* Drugs like Colchicines, Pentoxifylline, Dapsone, short bursts of systemic steroids and Thalidomide have been used to reduce the number of ulcers and recurrences.

PERICORONITIS (FIG. 20.5)

Definition

It is an acute infection which refers to inflammation of gingiva and surrounding soft tissues of an incompletely erupted tooth. It occurs most frequently in the mandibular third molar area.

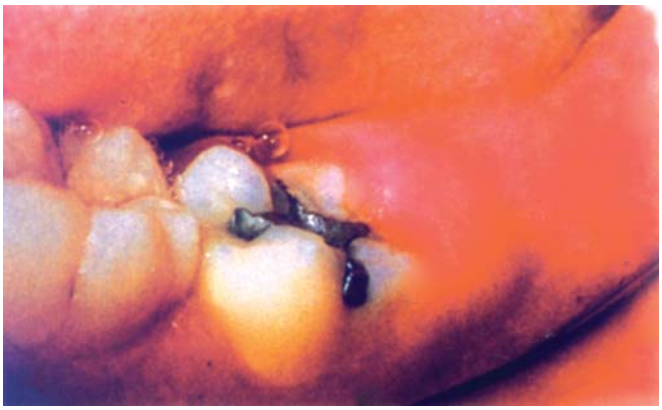


Fig. 20.5: Pericoronitis–third molar partially-covered by infected flap

Types

Acute, subacute or chronic.

Clinical Features

Signs and Symptoms

Include markedly red, edematous suppurating lesion that is extremely tender with radiating pain to the ear, throat and floor of the mouth.

The patient is extremely uncomfortable because of the foul taste and inability to close the jaws. In addition to the pain, swelling of the cheek in the region of the angle of the jaw is seen.

Acute Pericoronitis

It is identified by varying degrees of involvement of pericoronal flap as well as with systemic complications. An influx of inflammatory fluid and cellular exudates results in an increase in bulk of the flap which interferes with complete closure of the jaws. The flap is traumatized by contact with the opposing jaw and inflammatory involvement is aggravated.

Lymphadenitis is a common finding; the patient may also have toxic systemic complications such as fever, leukocytosis and malaise.

Complications

- The involvement may become localized, in the form of pericoronal abscess.

- If it occurs in a partly-erupted vital tooth it may give rise to cyst formation.
- It may spread posteriorly into the oropharyngeal area and medially into the base of the tongue, making it difficult for the patient to swallow.
- Depending on the severity there is involvement of the submaxillary, cervical, deep cervical and retropharyngeal lymph node.
- Peritonsillar abscess formation, cellulitis and Ludwig's angina are infrequent but nevertheless potential sequelae of acute pericoronitis.

Treatment (Figs 20.6A to E)

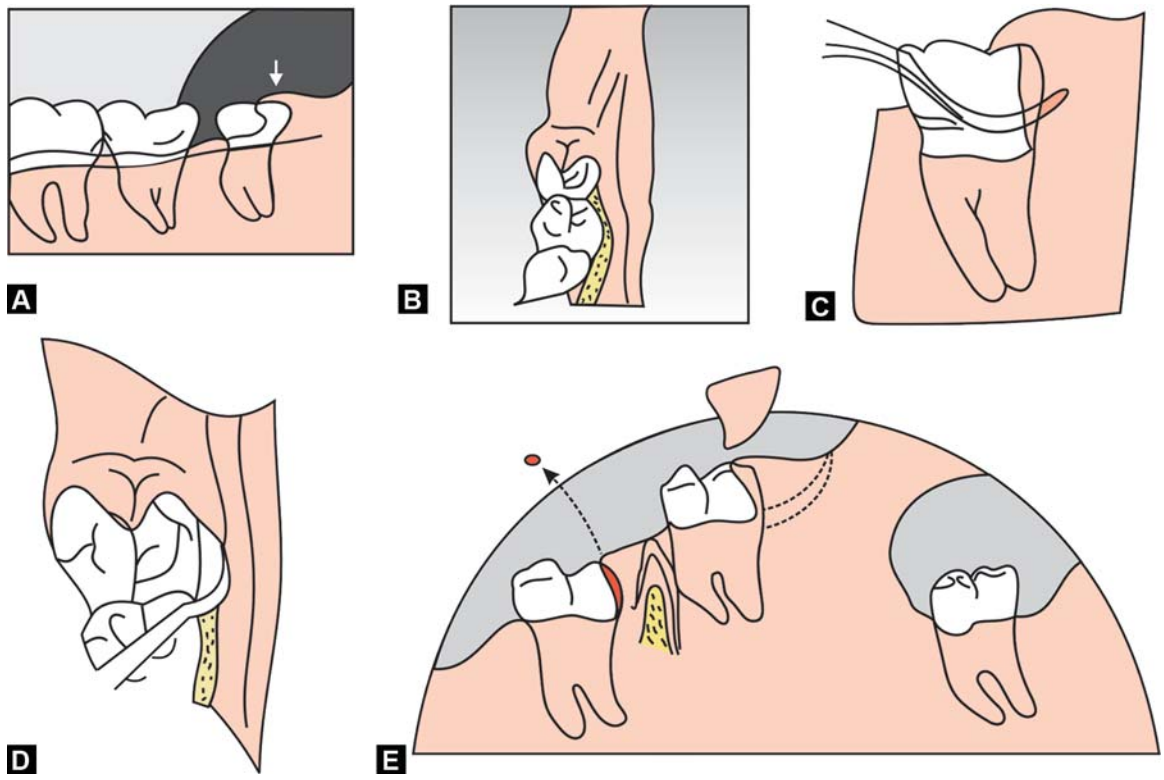
The treatment of pericoronitis depends on:

- Severity of the inflammation.
- The systemic complications, and
- The advisability of retaining the involved tooth.

First Visit

1. The area is gently flushed with warm water to remove superficial debris and exudate followed by application of topical anesthetic agent.
2. The flap is reflected with a scaler and the underlying debris is also removed and the area is flushed with warm water.
3. Instructions to the patient include hourly rinses with a solution of a teaspoonful of salt in a glass of warm water, rest, copious fluid intake and administration of systemic antibiotics, if toxic symptoms are present.
4. If the gingival flap is swollen and fluctuant an antero-posterior incision to establish drainage is made with a No. 15 bard parker blade, followed by insertion of 1/4th inch gauze wick.

In the next visit, determination is made as to whether the tooth is to be retained or extracted. This decision is governed by the likelihood of further eruption into a good functional position.



Figs 20.6A to E: Treatment of pericoronitis. (A, B) Pericoronal flap covering partially erupted lower IIIrd molar, (C, D) With the help of a scalor flap is reflected to cleanse the underlying debris, (E) A wedge-shaped incision is made to section the tissue

If it is decided to retain the tooth, the necessary surgical procedures are performed using a periodontal knife or electrosurgery. Under anesthesia, a wedge-shaped incision is made to section a tissue that includes the gingival flap with the tissue distal to the involved tooth as well.

After the tissue is removed, a periodontal pack is placed.

KEYPOINTS

1. Acute gingival lesions are classified as acute necrotizing ulcerative gingivitis (ANUG), primary herpetic gingivostomatitis, and pericoronitis.
2. ANUG is an inflammatory, destructive disease of the gingiva, which presents with characteristic signs and symptoms.
3. Oral lesions are characterized by punched-out, crater-like depressions at the crest of the interdental papillae, subsequently involving marginal gingiva and rarely attached gingiva.
4. These craters are covered by grayish pseudomembranous slough.
5. Patients with ANUG complain of a constant radiating, gnawing pain and metallic taste with excessive amount of pasty saliva.
6. Etiological factors responsible for ANUG are divided into:
   - Role of bacteria (fusospirochetal complex)
   - Local predisposing factors like preexisting gingivitis, injury to the gingiva and smoking.
   - Systemic predisposing factors like nutritional deficiency, debilitating diseases and psychosomatic factors
7. Primary herpetic gingivostomatitis is a viral infection characterized by diffuse erythema and vesicular eruptions.
8. Pericoronitis is an acute infection, which refers to inflammation of the gingiva and the surrounding soft tissues of an incompletely erupted tooth (Mostly in mandibular III molar area).

KNOW MORE ...

The term **transmissible** denotes a capacity for the maintenance of an infectious agent in successive passages through a susceptible animal host. Whereas the term **communicable** signifies a capacity for the maintenance of infection by natural modes of spread, such as direct contact through drinking water, food and eating utensils. Hence, a disease that is communicable is described as contagious.

REVIEW QUESTIONS

1. Classify acute gingival lesions; discuss the etiology, clinical features and treatment for acute necrotizing ulcerative gingivitis.
2. Differentiate between acute necrotizing ulcerative gingivitis and acute herpetic gingivostomatitis.
3. Pericoronitis—definition, clinical features and treatment.

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21 Chapter

Periodontal Diseases in Children and Young Adolescents

- ANATOMIC CONSIDERATIONS IN CHILDREN
  - Changes in Gingiva, Cementum, Periodontal Ligament, and Alveolar Bone
- HISTOPATHOLOGY OF GINGIVITIS IN CHILDREN
- MICROBIOLOGY OF PERIODONTAL DISEASES IN CHILDREN
- CLASSIFICATION OF PERIODONTAL DISEASES IN CHILDREN
  - Gingival Lesions
  - Types of Periodontitis
- PERIODONTITIS ASSOCIATED WITH SYSTEMIC DISEASE

INTRODUCTION

Periodontal disease consists of a group of infections affecting the gingiva and the supporting structures: cementum, periodontal ligament and alveolar bone. Gingival and periodontal changes in children could be due to the plaque and certain systemic diseases, which have a direct effect on the periodontium.

While the periodontal diseases are generally considered to affect the adults, children and adolescents can also manifest certain periodontal disorders, for example, prepubertal and juvenile periodontitis. This group of patients will benefit from an early periodontal evaluation and any necessary treatment.

The gingiva and periodontium of children differ in some respects from those of adults. Hence, periodontal diseases in children can be studied from anatomical, microbiological and cellular aspects.

ANATOMIC CONSIDERATIONS IN CHILDREN

Changes in Gingiva

1. The gingival tissues are more reddish due to a thinner epithelium, a lesser degree of cornification, and a greater vascularity.
2. The gingiva lacks the stippling, due to shorter and flatter papillae from the lamina propria. Normally, stippling appears at about 3 years of age and occurs in 35% of children between ages of 5 and 15 years.
3. The gingival margins appear to be rounded, rolled due to hyperemia and edema that follows eruption.
4. Greater sulcular depth, due to relative ease of gingival retraction.
5. The gingiva appears to be flabbier due to the lower density of the connective tissue in the lamina propria.

Cementum

Changes in the cementum include:

The cementum in the children is thinner and less dense and shows a tendency to hyperplasia of cementum apical to the epithelial attachments.

Periodontal Ligament

Changes in the periodontal ligament include:

The periodontal ligament in children is wider, has fewer and less dense fibers per unit area. It also has increased hydration with a greater blood and lymph supplies than in adults.

Alveolar Bone

Changes in the alveolar bone include:

1. In children, the lamina dura is thinner, fewer trabeculae and larger marrow spaces. There is also a smaller amount of calcification, greater blood and lymph supply and the crest of the alveolar bone appears flatter.
2. The contact points between the deciduous teeth are not as tight as those between the permanent dentition.

HISTOPATHOLOGY OF GINGIVITIS IN CHILDREN

1. Adult tissues show a greater density of plasma cells whereas in children, there were seen seven times as many lymphocytes as plasma cells.
2. Increased vascularity is seen.

MICROBIOLOGY OF PERIODONTAL DISEASES IN CHILDREN

Experimental gingivitis in children and the adults has shown in Table 21.1.

Table 21.1: Microbial species in children's and adult plaques

Species in greater numbers in children's plaque	*Species in adult plaques*
• *Leptotrichia* species • *Capnocytophaga* • *Selenomonas* species • *Bacteroides* species	• *Fusobacterium* • *Eubacterium*

CLASSIFICATION OF PERIODONTAL DISEASES IN CHILDREN

Gingival Lesions

1. Acute gingivitis
   - Herpetic gingivostomatitis
   - Necrotizing-ulcerative gingivitis
   - Candidiasis
2. Chronic marginal gingivitis
   - Plaque induced
   - Puberty gingivitis
3. Factitious gingivitis
4. Localized gingival recession

Periodontal Lesions

Early Onset Periodontitis

- Prepubertal
  - Localized
  - Generalized
- Juvenile
  - Localized
  - Generalized

PERIODONTITIS ASSOCIATED WITH SYSTEMIC DISEASE

- Papillon-Lefévre syndrome
- Ehler-Danlos syndrome
- Hypophosphatasia
- Chediak-Higashi syndrome
- Leukocyte adhesion deficiency
- Neutropenia
- Down's syndrome.

Gingival Lesions

Acute Herpetic Gingivostomatitis

It is a viral infection of the oral mucosa caused by herpes simplex virus. It occurs most frequently in infants and children younger than 6 years of age.

Clinical Features

I. Intraoral signs and symptoms
II. Extraoral signs and symptoms

Intraoral signs (Figs 21.1 and 21.2)

1. Gingiva appears to be diffuse red, erythematous, with varying degree of edema and gingival bleeding.

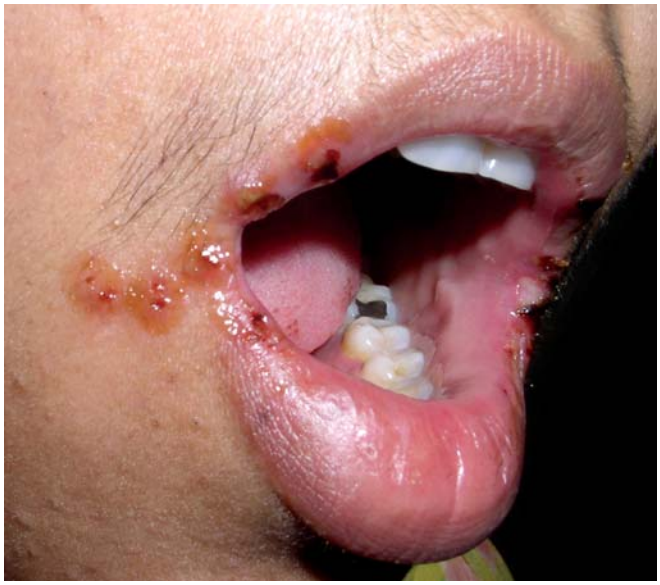

Fig. 21.1: Herpetic vesicles— late stage showing crusted lesions (*Courtesy*: Dr Deepak Daryani)

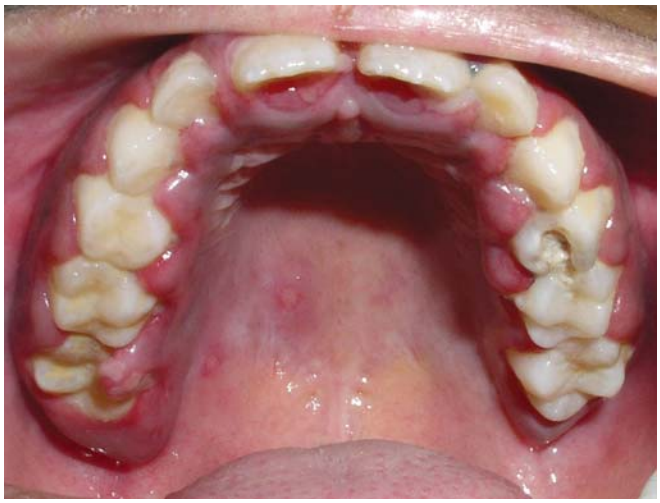

Fig. 21.2: Herpetic gingivostomatitis involving gingiva and palate (*Courtesy:* Dr Deepak Daryani)

2. In the initial stage, it appears to be discrete, spherical grey vesicles involving labial and buccal mucosa, soft palate, pharynx and tongue, approximately after 24 hours the vesicles rupture leaving painful ulcers.
3. The ulcers appear to be red, elevated with halo-like margins and a depressed yellowish or grayish-white central portion.
4. The course of the disease is 7 to 10 days.

Oral symptoms

1. Generalized soreness of the oral cavity, which interferes with the eating and drinking.
2. The ruptured vesicles are sensitive to touch, thermal changes and food.

Extraoral and systemic signs and symptoms

1. Involvement of the lips and face (*Herpes labialis*, cold sore)
2. Cervical adenitis, fever is as high as 101 to 105ºF and generalized malaise are common.

Treatment

a. *Local application*—using Talbot's iodine, zinc chloride 80 percent, riboflavin, thiamine, have been used. Chlortetracycline (Aureomycin) has been used successfully.
b. *Palliative treatment*—food debris and superficial debris is removed, relief of pain is obtained with 0.5 percent dyclonine hydrochloride mouthwash which has a topical anesthetic effect.
c. *Supportive treatment*—copious fluid intake, for relief of pain systemically administered aspirin is usually sufficient.

Acute Necrotizing Ulcerative Gingivitis (NUG)

Affects both children and adults, and is characterized by sudden onset. Sometimes following an episode of debilitating disease of acute respiratory tract infections.

Clinical features

- Intraoral signs and symptoms
- Extraoral signs and symptoms.

Oral signs

1. Two types of ulcerations are seen, lateral ulceration, deep ulceration and necrosis. It has been suggested that these patterns of necrosis are related to the fact that major blood supply to the two areas of gingiva is different. The gingival tissues gets blood supply from two main sources, i.e. supraperiosteal and intra-alveolar vessels.
2. Lateral ulceration is characterized by involvement of buccal wall of the papillae, margins and possibly the attached gingiva occurs in the distribution of lateral blood supply (less common). Deep ulceration involving primarily necrosis of the tissues of the embrasure, giving rise to the typical truncated papillae occurs in the distribution of the intra-alveolar vessels (comparatively more common).
3. These craters are caused by gray pseudomembranous slough demarcated from the remainder of the gingival mucosa by a pronounced linear erythema.
4. Other signs include—gingival hemorrhage or pronounced bleeding on slightest provocation.

Oral symptoms

1. The lesions are extremely sensitive to touch.
2. Constant radiating, gnawing pain that is intensified by eating spicy or hot food and chewing.
3. Metallic foul taste and the patient is conscious of an excessive amount of "pasty saliva".

Extraoral and systemic signs and symptoms:

In mild to moderate stages—local lymphadenopathy and slight elevation in temperature is seen.

In severe cases—high fever, increased pulse rate, leukocytosis, loss of appetite is seen.

In very rare cases, severe sequelae may follow:

- Noma or gangrenous stomatitis.
- Fusospirochetal meningitis.
- Toxemia and fatal brain abscess.

Etiology

a. Bacteriology consists of constant flora composed of fusospirochetal organisms, variable flora consists of spirochetes of varying sizes and *Bacteroides intermedius*.
b. Predisposing factors—local and systemic.

Local predisposing factors include:

- Pre-existing gingivitis, e.g. incubation zones.
- Injury to the gingiva, e.g. malocclusion
- Smoking.
  a. Direct toxic effect of tobacco
  b. Vascular or other changes induced by nicotine or other substances
  c. As a reflection of stress.

Systemic predisposing factors include:

- Nutritional deficiency.
- Debilitating diseases.
- Psychosomatic factors.

Treatment Should follow an orderly sequence.

First visit: Treatment is confined to the acutely involved areas, after applying topical anesthesia. The areas are gently swabbed to remove the pseudomembrane. Superficial calculus is removed with ultrasonic scaler. The patient is instructed to rinse the mouth every 2 hours with a glassful of an equal mixture of warm water and 3 percent hydrogen peroxide (chlorhexidine rinses are also recommended). In the presence of systemic symptoms penicillin or erythromycin or metronidazole is prescribed.

Second visit: Scaling is performed (After 1 to 2 days).

Third visit: After 1 to 2 days of second visit. Scaling and root planing are repeated with plaque control instructions, hydrogen peroxide rinses are discontinued, but chlorhexidine rinses can be maintained for 2 to 3 weeks.

Candidiasis: Acute Candidiasis (Moniliasis, Thrush)

It is the most common mycotic infection of the oral mucosa caused by *Candida albicans*. It is seen in three types of individuals; debilitated or immunosuppressed adults, infants and adults who have been on antibiotic therapy for sometime.

Oral lesions are described as four clinical types:

a. *Pseudomembranous type*—white curd-like plaques.
b. *Atrophic type*—usually seen on the dorsum of the tongue with erythema and papillary atrophy.

c. *Hyperplastic type*—hyperkeratosis of the epithelium with white plaques.
d. *Epidermal and perioral type*—scaling patches at the corner of the lips.

Treatment Current treatment is the use of the antimycotic agent like cotrimoxazole in the form of oral troches every 3 hours (for a total of 6 per day) for 7 to 10 days.

Chronic Candidiasis

It is a rare type of *C. albicans* infections resulting in a granulomatous lesion that begins in infancy or early childhood and may persist for several years.

Chronic Marginal Gingivitis

It is the most common type of gingival disease in childhood. Gingival color changes and swelling appear to be more common expressions of gingivitis in children than a bleeding and increased pocket depth.

Etiology

Plaque and calculus are most common causes. In children certain conditions predispose the gingivitis they include:

a. Gingivitis associated with tooth eruption is called as eruption gingivitis.
b. Partially-exfoliated, loose deciduous teeth frequently cause gingivitis.
c. Gingivitis occurs more frequently around malposed teeth.
d. Gingivitis is increased in children with excessive overbite and overjet, mouth breathing and nasal obstruction.
e. A higher prevalence and severity of gingivitis and gingival enlargement is found in the circum pubertal period. This form of gingivitis has been termed as pubertal gingivitis. There may be a gingival enlargement as a result of hormonal changes that magnify tissue response to the local irritants.

These inflammatory changes may persist during the time the primary tooth is in the mouth and with exfoliation may become more severe. These inflammatory lesions are usually nondestructive and do not progress to attachment loss. The treatment is plaque control instruction and debridement. The accumulation of plaque in children is probably more rapid than in adults, but the host response is generally not as intense. Calculus, in contrast, is found less frequently in children than in adults, but gradually increases as the child enters the teenage years.

Factitious Gingivitis (Gingivitis Artefacta)

It is of two types:

- Major form.
- Minor form.

Minor form results from the rubbing or picking the gingiva with finger nail (habitual). Major form is more severe and involves deeper peridontal tissues (psychological causes).

Localized Gingival Recession

It may be seen around individual teeth or groups of teeth. The recession may be seen in the presence or absence of inflamed gingiva depending on the local irritants. In children the position of the tooth in the arch is the most important cause, e.g. labially-positioned, tilted or rotated teeth and anterior open bite, the recession may be transitional phase in tooth eruption and may correct itself or it may be necessary to realign the tooth orthodontically.

Types of Periodontitis

Prepubertal Periodontitis

Prepubertal periodontitis occurs in localized and generalized forms.

Localized Prepubertal Periodontitis

Clinical features

- The age of onset is approximately 4 years.
- Plaque levels are usually low.
- Alveolar bone loss is rapid.
- Defect in neutrophil or monocyte functions has been reported.

Generalized Prepubertal Periodontitis

- Entire width of attached gingiva appears to be fiery-red.
- Gingival hyperplasia, cleft formation and recession.
- Rapid destruction of the alveolar bone.
- Systemic involvement like recurrent bacterial infections.
- Defects in polymorphonuclear leukocytes and monocytes.

Juvenile Periodontitis

Definition was given by Baer, who described it, "as a disease of the periodontium occurring in an otherwise healthy adolescents, which is characterized by a rapid loss of alveolar bone around more than one tooth of the permanent dentition". It exists in two forms:

a. Localized
b. Generalized.

Clinical features of localized juvenile periodontitis (LJP)

1. *Age and sex distribution*—between 11 and 15 years some studies show predilection for female patients.
2. *Distribution of lesions*—three types of involvement is seen:
   a. First molar and/or incisors.
   b. First molar and/or incisors with additional teeth (not exceeding 14 teeth).
   c. Generalized involvement.
3. The most striking feature is lack of clinical inflammation, despite the presence of deep periodontal pockets.
4. Small amounts of plaque is seen which rarely mineralizes to become calculus corrected.
5. Most common initial symptoms are mobility, migration of the incisors and first molars. Classically, a distolabial migration of the maxillary incisors with diastema formation occurs.

For LJP, classic distribution is the involvement of first molars and incisors with least distribution in the cuspid, premolar area. The reasons could be:

1. Production of opsonizing antibodies against Aa (*Actinobacillus actinomycetem comitans*).
2. Bacteria antagonistic to Aa may develop thereby decreasing the number of colonization sites.
3. Aa may lose its leukotoxin producing ability for unknown reasons.

Localization of the lesions could also be due to the defect in cementum formation [hypoplastic/aplastic cementum].

Pathogenesis of LJP is related to the interplay of several factors. These include the specific microbiology of subgingival plaque, defects in cementum, hereditary factors, impaired PMN functions and disorders of the immune system.

Microbiology of LJP: Two types of bacteria are considered to be pathogenic in LJP.

- *Actinobacillus actinomycetem comitans*
- *Capnocytophaga*

Virulence factors produced by *A. actinomycetemcomitans* are as follows:

a. *Leukotoxin*—destroys polymorphonuclear leukocytes (PMNs) and macrophages.
b. *Endotoxin*—activates host cells to secrete inflammatory mediators (PG's, IL1b, TNFa).
c. *Bacteriocin*—may inhibit the growth of beneficial species.
d. *Immunosuppressive factors*—may inhibit IgG and IgM production.
e. *Collagenase*—causes degradation of collagen.
f. *Chemotactic inhibition factors*—may inhibit neutrophil chemotaxis.

Radiographic findings:

1. Vertical/angular bone loss around, the first molars and incisors in an otherwise healthy teenagers is a diagnostic sign of classic Juvenile periodontitis (J.P.) "Arc-shaped" loss of alveolar bone extending from the distal surface of the 2nd premolar to the mesial surface of the 2nd molar is seen.
2. Bilateral symmetrical patterns of bone loss is seen [mirror-image pattern].

Immunologic findings: A large proportion of patients (70%) with LJP have a defect in PMN chemotaxis, which is cell-associated and depressed PMN phagocytosis, which is serum-associated. PMN show reduced cell surface receptors to the synthetic polypeptide chemotactic factors. N-formylmethionyl phenylalanine (FMLP) and the complement factor C5a, also have reduced amounts of surface glycoproteins GP-110 which is important for chemotactic response.

Clinical features of GJP: The age of diagnosis is between 20 and 30 years. Severe generalized bone loss is the characteristic feature. This may be restricted to upper or lower arch. Patients of GJP often show good plaque control and the extent of bone loss does not commensurate with the level of oral hygiene.

Treatment: In the past, the prognosis for juvenile periodontitis was considered to be poor.

Current therapy: Systemic tetracycline hydrochloride 250 mg q.i.d. for at least 1 week should be given in conjunction with the local mechanical therapy. Several reports have shown excellent bone fill in cases of LJP treated with tetracycline, flap surgery and placement of grafts.

In refractory cases, tetracycline resistant Aa has been suspected. In such cases a combination of amoxicillin and metronidazole has been suggested.

Periodontitis Associated with Syndromes

Papillon-Lefévre Syndrome

a. It is characterized by hyperkeratotic skin lesions and severe destruction of the periodontium.
b. These changes may appear before the age of 4 years.
c. Characteristic skin lesions are hyperkeratosis of localized areas on palms and soles, knees and elbows.
d. Periodontal involvement includes, early inflammatory changes that lead to bone loss and exfoliation of teeth. Primary teeth are lost by 5 or 6 years of age. The permanent dentition erupts normally but within few years the permanent teeth are also lost.

Ehlers-Danlos Syndrome

It is an inherited disorder affecting the connective tissues, the defect is in collagen molecular biology, but the nature of the defect is unknown. The syndrome is named after two clinicians, who described excessive joint mobility, skin hyperextensibility, easy bruising and peculiar scarring, which occur after skin wounds.

Oral and periodontal manifestations: The oral mucosa, gingival tissues, teeth and temporomandibular joints can all be affected by Ehlers-Danlos syndrome.

Oral mucosa

- It is often fragile and susceptible to bruising.
- Post-extraction hemorrhage can be a problem, due to fragility of blood vessels and defects in the supporting connective tissues.

Gingival tissues: These are often fragile and bleed readily on toothbrushing. Some forms of Ehler-Danlos syndrome (type VII) are reported to have advanced periodontal destruction.

Teeth: Teeth in Ehler-Danlos syndrome are fragile and fracture easily.

TMJ: Subluxation of temporomandibular joints have been reported.

Treatment

1. A thorough preventive program should be followed.
2. Due to the fragility of oral mucosa and gingiva, the periodontal therapy in Ehlers-Danlos syndrome should be as atraumatic as possible.

Down's Syndrome (Mongolism, trisomy 21)

It is a congenital disease caused by a chromosomal abnormality and characterized by mental deficiency and growth retardation.

The oral findings include presence of plaque, calculus, other local irritants, e.g. diastema, crowding of teeth, high frenum attachment and malocclusion.

Periodontal disease in Down's syndrome: include the formation of deep periodontal pockets associated with

plaque accumulation and moderate gingivitis, usually generalized but more severe in the lower anterior region (may be due to high frenal attachment). Acute necrotizing lesions are also common.

Two factors have been proposed to explain high prevalence and increased severity of periodontal destruction in Down's syndrome.

1. Reduced resistance to infections because of poor circulation (especially peripheral).
2. Defect in T-cell maturation and polymorphonuclear leukocyte chemotaxis.

Neutropenias: Destructive generalized periodontal lesions have been described in children with neutropenia.

Chédiak-Higashi syndrome (C-H syndrome): This is a rare syndrome characterized by recurrent bacterial infections. It exhibits oral ulcerations and rapidly destructive periodontitis.

Hypophosphatasia: This is a rare familial skeletal disease characterized by rickets, poor cranial formation, premature loss of primary dentition—particularly incisors. Patients have low levels of serum alkaline phosphatase. Teeth are lost with no clinical evidences of gingival inflammation and show reduced cementum formation.

Acute and subacute leukemia (Malignant Neoplasias of WBC Precursors): Accomplished by severe periodontal destruction.

Leukocyte adhesion deficiency: These cases are rare and begin during, or immediately after eruption of the primary teeth. Extreme acute inflammation and proliferation of gingival tissues with rapid bone loss are found. Profound defects in peripheral blood neutrophils and monocytes are seen, hence they are absent in gingival tissues. Patients with leukocyte adhesion disease also have frequent respiratory tract infection and sometimes otitis media.

KEYPOINTS

1. Various anatomic variations in the gingiva, periodontal ligament, cementum and alveolar bone may predispose to the disease of the periodontium.
2. Most common lesions in children are gingival lesions including acute gingivitis, chronic marginal gingivitis facititious gingivitis and localized gingival recession.
3. Periodontal lesions include, early onset periodontitis, prepubertal and juvenile periodontitis, periodontitis associated with systemic diseases.
4. Most of the signs of periodontitis are seen during adolescence, with primary prevention one may help to reduce the tooth loss.
5. Further investigations should be carried out whenever there is bleeding after gentle probing in the presence of relatively healthy gingiva, so that early onset periodontal lesions and prepubertal conditions can be ruled out.

KNOW MORE ...

Classification of Gingival Diseases in Childhood and Adolescents

1. Eruption gingivitis
2. Plaque induced gingivitis
3. Acute lesions:
   - HSV infection
   - Aphthous ulcers (recurrent in nature)
   - NUG
   - Acute candidiasis
4. Gingival enlargement associated with:
   - Puberty
   - Idiopathic
   - Drug induced and
   - Vitamin deficiency

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22 Chapter

Desquamative Gingivitis

Srinivas K

INTRODUCTION

The term chronic desquamative gingivitis was coined by Prinz in 1932 to describe a peculiar condition characterized by intense erythema, desquamation and ulceration of the free and attached gingiva. In 1960, Mc Carthy and colleagues suggested that desquamative gingivitis was not a specific disease entity, but a gingival response associated with a variety of conditions. Desquamative gingivitis is a generic term that may represent a variety of specific disease processes, but the clinical picture is indisputable. There may be threads or tags of loose necrotic epithelium, as the name suggests. Desquamative gingivitis involves not only marginal gingiva, as do most cases of gingivitis, but it also peels the attached gingiva often in a band-like fashion. The clinical description of desquamative gingivitis represents gingival manifestations of a variety of diseases.

CLASSIFICATION

A. *Dermatoses*

- Oral lichen planus
- Mucous membrane pemphigoid
- Pemphigus vulgaris
- Bullous pemphigoid
- Erythema multiforme
- Linear IgA disease
- Lupus erythematosus
- Epidermolysis bullosa aquisita
- Dermatitis herpetiformis

B. *Local hypersensitivity reactions to*

Sodium lauryl sulphate, mouthwashes, dental materials, drugs, cosmetics, chewing gum and cinnamon, etc.

Fig. 22.1: A case of pemphigus vulgaris presenting as desquamative gingivitis

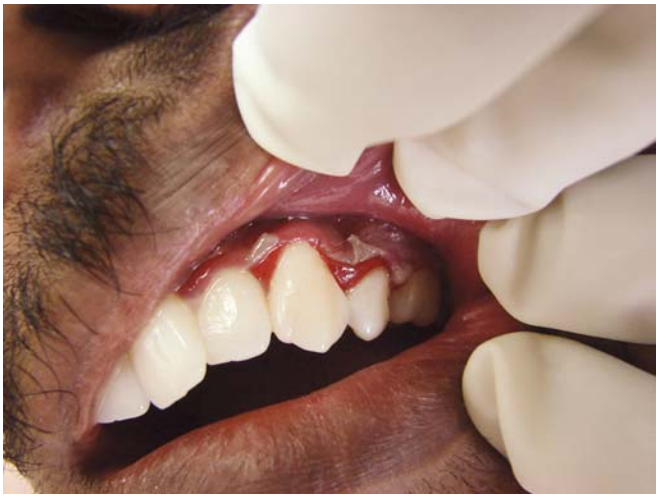

Fig. 22.2: Nikolsky's sign in a case of bullous pemphigoid

C. *Miscellaneous*

- Chronic ulcerative stomatitis
- Orofacial granulomatosis
- Plasma cell gingivitis

CLINICAL FEATURES

- Females are more frequently affected.
- Buccal aspect of anterior gingiva most commonly affected.
- The gingiva is fiery red, friable and desquamates easily (Fig. 22.1).
- Patients complain of soreness, especially when eating spicy or acidic food, and of bleeding and discomfort with toothbrushing.
- Lesions get aggravated by local plaque accumulation.
- A positive Nikolsky's sign where the surface epithelium "floats away" when lateral pressure is applied to the mucosa, may indicate vesiculobullous disorders (Fig. 22.2).
- The presence of white plaques or white striae indicate lichen planus (Fig. 22.3).

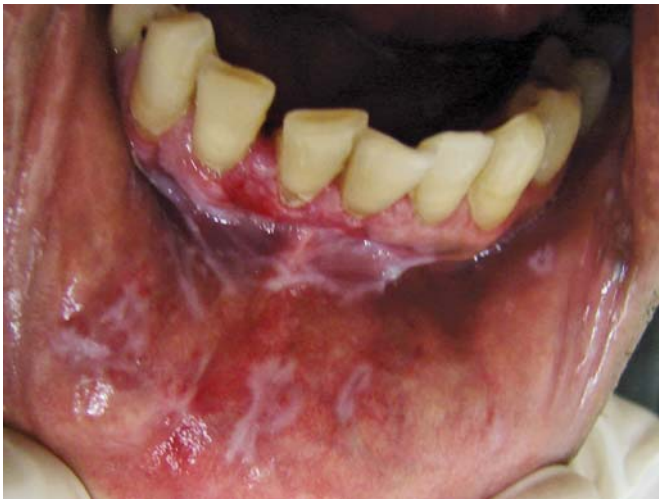

Fig. 22.3: A case of lichen planus—note the presence of white striae along with desquamation

HISTOPATHOLOGY

Microscopically, there will be a thin atrophic epithelium with little or no keratin at the surface. Acantholysis will be seen in bullous lesions and diffuse infiltration of chronic inflammatory cells in the underlying connective tissue in most of the cases. In most of the bullous diseases histopathology may not confirm the diagnosis and direct immunofluorescence will be required to arrive at a accurate diagnosis.

DIAGNOSIS

The success of any given therapeutic approach resides on the establishment of an accurate final diagnosis. The following approach helps us to elucidate the disease triggering desquamative gingivitis.

THERAPY

- Removal of underlying cause if known allergen or irritant is present
- Improvement of oral hygiene
- Treatment of underlying disease (Table 22.1)
- Local or systemic steroid or immunosuppressive treatment.

DISEASES CLINICALLY PRESENTING AS DESQUAMATIVE GINGIVITIS

LICHEN PLANUS

It is a relatively common, chronic dermatosis characterized by the presence of cutaneous violaceous papules that may coalesce to form plaques. Majority of the patients with oral lichen planus are middle aged.

Clinical Features

Oral Lesions

Oral LP almost invariably occurs as a bilateral disease and it involves the posterior buccal mucosa. Clinically, oral LP appears as radiating white or gray-velvety thread like lesion, which consists of papules in linear, annular or retiform arrangement. A tiny white elevated dot is present at the intersection of the white lines knows as 'Wickham's striae' or 'Honiton Lace' (Fig. 22.3).

Gingival Lesions

Oral LP is the main disorder associated desquamative gingivitis with up to 10 percent of patients having lesions restricted to the gingival tissue.

1. *Keratotic lesions:* Raised white lesions may present as groups of individual plaques, linear or reticular lesions.
2. *Erosive lesions:* These extensive erythematous areas with a patchy distribution may present as focal or diffuse hemorrhagic areas.
3. *Vesicular or bullous lesions:* These raised fluid-filled lesions are short lived on gingiva resulting in an ulceration.
4. *Atropic lesions:* Atrophy of the gingival tissues results in erythema confined to gingiva.

Histopathology

Microscopic criteria include hyperkeratosis, saw-toothed appearance to the rete pegs, liquefaction degeneration of basal cell layer, an eosinophilic band may be seen just benath the basement membrane and a dense subepithelial band shaped infiltrate of lymphocytes and macrophages is characteristic of the disease. Direct immunofluorescence demonstrates the presence of fibrinogen in the basement membrane.

Differential Diagnosis

- Lichenoid drug reaction
- Lupus erythematosis
- Cheek biting
- Hyperplastic candidiasis
- Contact hypersensitivity
- Speckled leukoplakia

Treatment (Fig. 22.4) and Prognosis

Corticosteroids are the single most effective group of drugs. Topical application and local injection of steroids have also been successful.

Addition of antifungal therapy typically enhances clinical results. The erosive, bullous and ulcerative lesions are treated with high potency topical steroid such as 0.05 percent

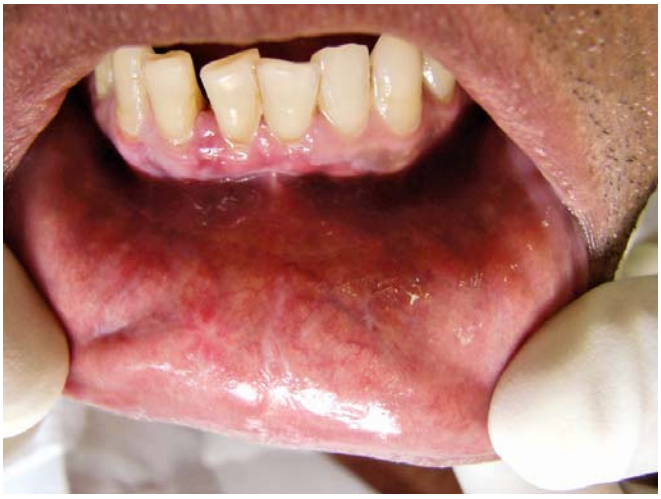

Fig. 22.4: Case of lichen planus shown in Figure 22.3 treated with topical steroid

Fluocinolone acetonide. Intralesional injection of triamcinolone acetonide (10 to 20 mg) has also been used successfully. Other treatment modalities are retinoids, hydroxychloroquine, cyclosporine and free gingival grafts.

CICATRICIAL PEMPHIGOID

It is a chronic autoimmune subepithelial disease primarily affecting the mucous membranes of patients over the age of 50. It is characterized by mucosal blister formation with subsequent scarring, while the oral and ocular mucosa are most often involved, other mucosal surfaces may also be affected.

Clinical Features

It tends to affect women more than men. The oral mucosal presentation ranges from erosion or desquamation of attached gingival tissues to large areas of vesiculobullous eruptions. Bullae are rarely seen because the blisters are fragile and short lived. Lesions are chronic and may heal with scarring. Gingival lesions appear as patchy red zones. Concomitant ulcers may be seen on marginal and attached gingiva. With chronicity, pain typically diminishes in intensity. *Nikolsky's sign* may also be seen.

Histopathology

It is a subepithelial or sub-basal clefting disorder. There is no evidence of acantholysis. Lamina propria is infiltrated by lymphocytes.

Differential Diagnosis

- Pemphigus vulgaris
- Atrophic lichen planus
- Discoid lupus erythematosus

Treatment and Prognosis

Topical or systemic corticosteroids are typically used. Prednisolone is used for moderate to severe disease because of its side effects which may outweigh benefits, highly potent topical steroids such as clobetasol, betamethasone dipropionate, fluocinoxide are used for gingival diseases, a custom made flexible mouthguard may be used. Rinsing with chlorhexidine is often a useful adjunct. Tetracycline and doxycycline are helpful in treating steroid resistant cases. In severe cases, immunosuppressive agents (azathioprine, cyclophosphamide, cyclosporine) may be occasionally added to the prednisolone regimen, but it often provides disappointing results. Very high dose may be required to achieve any significant results.

BULLOUS PEMPHIGOID

It is the most common blistering autoimmune disorder caused by binding of autoantibodies to specific antigens found in lamina lucida region of the basement membrane. It is often seen in elderly individuals. It usually presents with pruritis and bullous lesions.

Clinical Features

It is seen primarily in the elders, with the peak evidence in the seventh and eighth decades. The gingival lesions may be the only site of oral involvement that consists of generalized edema, inflammation and desquamation with localized areas of discrete vesicle formation.

Histopathology

Bullae are subepithelial in bullous pemphigoid and appear similar to those in cicatricial pemphigoid. Ultrastructurally the basement membrane is cleaved at the level of lamina lucida.

Treatment

Systemic corticosteroids are generally used to control this disease. Nonsteroidal immunosuppressive agents may also effectively control the disease process as well as reduce the side effects. A combination of tetracycline or doxycycline with niacinamide has provided some clinical success.

LINEAR IgA DISEASE

It is an uncommon subepithelial mucocutaneous disorder with predilection in women. It is characterized by deposition of IgA rather than IgG in the basement membrane. Clinically,

it represents as a pruritic papules and blisters at the site of trauma.

Oral Lesions

It consists of vesicles, painful ulcerations or erosions. The hard and soft palates are commonly affected. Rarely oral lesions may be the only manifestation before the presentation of cutaneous lesions.

Histopathology

Separation at the basement membrane is seen. Direct immunofluorescence demonstrates deposition of IgA.

Differential Diagnosis

- Erosive lichen planus
- Chronic ulcerative stomatitis
- Pemphigus vulgaris
- Bullous pemphigoid
- Lupus erythematosus.

Treatment

The primary treatment comprises combination of Sulfapyridine and Niacinamide. Small amounts of prednisone (10 to 30 mg) can be added.

DERMATITIS HERPETIFORMIS

It is a skin eruption. The cause is unknown, but most patients have an associated gluten-sensitive enteropathy.

Clinical Features

It is a chronic disease typically seen in young and middle aged adults. Periods of exacerbation and remission characterize the disease. Lesions are usually symmetrical in their distribution, aggregated but are often individually disposed. In the oral cavity vesicles and bullae are evanescent. Subsequent to rupture, superficial nonspecific ulcers have a fibrinous base with erythematous margins. Lesions may involve both keratinized and nonkeratinized mucosae.

Histopathology

Collections of neutrophils, eosinophils and fibrin are seen at the papillary tips of dermis. Subsequent exudation at this location contributes to epidermal separation. A lymphophagocytic infiltrate is seen in perivascular spaces.

Treatment

It is generally treated with dapsone, sulfoxone and sulfapyridine. Because patients often have an associated enteropathy, a gluten-free diet may also be a part of the therapeutic regimen.

PEMPHIGUS VULGARIS

It is an autoimmune mucocutaneous disease characterized by autoantibodies to glycoprotein adhesion molecule desmoglien-3 resulting in intraepithelial blister formation.

Clinical Features

Skin lesions present as ulcers preceeded by bullae. Presentation of the lesions may initially be as fluid-filled bullae. Bullae rapidly rupture leaving a collapsed roof. This grayish membrane is easily removed with a guaze sponge, leaving a red, painful ulcerated base. Nikolsky's sign is seen. The incidence is equal in both sexes.

Histopathology

It represents the prototypical suprabasal or intraepithelial clefting morphology. It is the acantholytic lesion that features squamous epithelial cells lying free within the bulla or vesicle cavity. Tzanck cells are seen, characterized by nuclear enlargement and hyperchromatosis subsequent to formation of the suprabasal cleft. The intact basal layer remains attached to lamina propria.

Differential Diagnosis

- Bullous and cicatricial pemphigoid
- Erythema multiforme
- Bullous lichen planus
- Dermatitis herpetiformis.

Treatment

Disease control may be achieved with intermediate dose of steroid. For more severely affected patients, high dose of corticosteroid is used followed by a combined drug approach that includes alternate day prednisolone plus immunosuppressive agent such as azathioprine, methotrexate or cyclophosphamide.

DRUG REACTION OR ERUPTIONS

Drugs can act as an allergen either alone or in combination, sensitizing the tissues and then resulting in allergic reaction of skin and oral cavity. Stomatitis medicamentosa is characterized by eruptions in the oral cavity resulting from sensitivity to drugs that have been taken by mouth or parenterally. The local use of medicaments in the mouth is referred to as stomatitis venenata or contact stomatitis, examples are, aspirin burn or stomatitis due to topical penicillin. Clinically, drug eruption in the oral cavity could be:

- Vesicular or bullous—most commonly seen
- Pigmented or nonpigmented macular lesions

Erosions, followed by deep ulceration with purpuric lesions, may also occur in different parts of oral cavity with gingival being most commonly affected. Some of the compounds that may cause contact allergy in the gingiva are, mercurial compounds (e.g. amalgam) tartar control toothpastes (e.g. pyrophosphates) and cinnamon compounds can result in intense erythema of gingival tissue (plasma cell gingivitis). Elimination of the offending agent usually leads to resolution of the lesions within a week.

DISEASES CLINICALLY PRESENTING AS DESQUAMATIVE GINGIVITIS

It is listed in Table 22.1.

KEYPOINTS

1. Chronic desquamative gingivitis is characterized by intense redness and desquamation of the surface epithelium of marginal and attached gingiva.
2. Chronic desquamative gingivitis is the clinical manifestation of several different disorders, the clinical features are variable and diagnosis of the underlying condition is necessary for optimum management.
3. Removal of plaque is an important part of treatment. Mild cases can be managed with topical steroids and severe cases need systemic immunsuppression. Periodic recall and careful follow-up is the most important part of management.

KNOW MORE ...

Treatment of Desquamative Gingivitis

- Most symptomatic cases of desquamative gingivitis can be managed by topical steroids (Triamcinolone acetonide/clobetasol proprionate/fluocinolone acetonide with orabase)

  Lower the strength of the preparation as soon as the lesions heal and become asymptomatic should be lowered.
- A splint with spacer for applying topical steroids can be used for resistant cases.
- A combination of topical and systemic steroids is preferred for severe cases.
- Prednisolone is the most preferred systemic steroid and a dosage of 20-40 mg/day.

  The dose should be reduced by 5–10 mg/day gradually over a 2–4 week period (to prevent Hypothalamopituitary axis (HPA) suppression.
- Important—Always a topical antifungal like clotrimazole should be combined with steroid therapy to prevent secondary candidiasis.

REVIEW QUESTION

1. Classify desquamative lesions. Discuss in detail clinical features and treatment of desquamative gingivitis.

Table 22.1 Etiology, clinical features, investigations and treatment of diseases clinically presenting as desquamative gingivitis

Disease	*Etiology*	*Clinical features*	*Investigations*	*Treatment*
Oral lichen planus	Cell mediated (Unidentified epithelial antigen)	• White papular lesions and red erythematous or erosive areas can be a part of reticular, papular, plaque like, bullous and ulcerative patterns • Lesions confined only to gingiva (8-10%) may be entirely erythematous with no reticular or papular elements	HP— Hyperkeratosis; basal cell degeneration; subepithelial lymphocyte inflammatory infiltrate DIF— Fibrin, fibrinogen at BMZ	• Topical and systemic steroids • Topical tacrolimus or cyclosporine or systemic hydroxy chloroquine for unresponsive cases. • Surveillance for malignant transformation
Mucous membrane pemphigoid	Autoantibodies to basement membrane adhesion complex (BP180,α6β4 Integrin, Laminin 5 and 6)	• Intact vesicles of gingival or other mucosal surfaces but frequently appear as nonspecific erosions which spread slowly • Desquamative gingivitis may be the only manifestation of the disease	HP— Subepithelial vesicle formation; vacuolation in the basal lamina DIF— C3, IgG at BMZ	• Topical and systemic steroids • Immunosuppressive drugs like azathioprine, cyclophosphamide. Appropriate referral for extraoral involvement
Pemphigus vulgaris	Autoantibodies to glycoprotein adhesion molecules present on Desmosomes Desmoglien 3 and 1	• Bullae on noninflamed base which rapidly breaks to leave shallow irregular ulcers seen on buccal mucosa palate and gingiva • Thin layer of epithelium peels away in an irregular pattern leaving a denuded base • Nikolsky's sign positive	HP—Acantholysis; suprabasilar bullae; acantholytic keratinocytes (Tzank cells) DIF— IgG, IgA, IgM, Complement within the epithelial intercellular spaces	• Topical and systemic steroids • Immunosuppressive drugs like azathioprine, cyclophosphamide • Surveillance for relapses
Bullous pemphigoid	Autoantibodies to BP 180 and BP 230 found in lamina lucida region on the hemidesmosomes	• Bullae which breaks into ulcers seen in buccal mucosa • Gingival lesions consist of generalized edema, inflammation and desquamation with localized areas of discrete vesicle formation • Nikolsky's sign positive	HP— Subepithelial bullae formation (Lamina lucida); Eosinophil rich inflammatory infiltrate DIF—IgG at BMZ	• Topical and systemic steroids • Immunosuppressive drugs like azathioprine, cyclophosphamide • Dapsone/Combination of tetracycline and nicotinamide

Contd...

Contd...

Disease	*Etiology*	*Clinical features*	*Investigations*	*Treatment*
Erythema multiforme	Hypersensitivity reaction – Common inciting factors are HSV and drugs	• Erosions and ulcerations which are large and confluent • Severe crusting and bleeding of the lips	HP— Subepithelial or intra-epithelial vesiculation; Necrotic keratinocytes; Mixed inflammatory infiltrate in a perivascular distribution DIF— Fibrin C3 and cytoid bodies at BMZ	• Topical and systemic steroids • Acyclovir for HSV associated erythema multiformae
Linear IgA disease	Autoantibodies (IgA) to basement membrane	Blisters and ulcers of oral mucosa accompanied by desquamative gingivitis	HP— Subepithelial blister formation DIF— IgA at BMZ (Linear pattern)	• Topical and systemic steroids • Dapsone in severe cases
Lupus erythematosus	Prototype of autoimmune disease involving immune complexes	White radiating striae interspersed with erythematous lesions seen on gingiva, buccal mucosa tongue and palate	HP—Keratinocyte vacuolization, lamina propria edema, subepithelial PAS+ deposits, thickening of vascular basement membrane, lymphocytic perivascular infiltrate DIF— IgM, IgG and C3 at BMZ	• Topical and systemic steroids • Hydroxy chloroquine for mild cases. • Surveillance for extraoral involvement
Epidermolysis Bullosa Aquisita	Autoantibodies to type VII collagen – component of anchoring fibrils of basement membrane	• Bullae which break into ulcers seen in oral mucosa accompanied by widespread skin lesions, genital lesions, eye lesions and laryngeal lesions. • Nikolsky's sign positive	HP— Subepithelial Blister Formation DIF— C3 and IgG At BMZ (Thick linear pattern)	• Topical and systemic steroids • Immunosuppressive drugs
Dermatitis Herpetiformis	Immune mediated disease associated with gluten sensitive enteropathy	Vesicles and bullae which rupture rapidly to leave areas of superficial ulceration at any intraoral site	HP—Neutrophil accumulation in dermal papillae; papillary microabscesses form and progress to subepidermal vacuolization and vesicle formation	Dapsone, Sulfapyridine, gluten free diet

HP- Histopathology, DIF- Direct immunofluorescence, C3- Complement 3, BMZ- Basement membrane zone, ANA- Antinuclear antibodies

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23 Chapter

The Periodontal Pocket

- DEFINITION AND CLASSIFICATION
- CLINICAL FEATURES
  - Signs and Symptoms
- PATHOGENESIS
- HISTOPATHOLOGY
  - Changes in the Soft Tissue Wall
  - Microtopography of the Gingival Wall
  - Periodontal Pocket as Healing Lesions
  - Pocket Contents
  - Changes in Root Surface Wall
  - Periodontal Disease Activity
- RELATION OF LOSS OF ATTACHMENT AND BONE LOSS TO POCKET DEPTH
- DIFFERENCES BETWEEN SUPRABONY AND INFRABONY POCKETS
- PERIODONTAL CYST
- DETERMINATION OF POCKET DEPTH
- TREATMENT OF PERIODONTAL POCKETS

INTRODUCTION

A sulcus depth up to 3 mm is considered to be normal, provided the patient can maintain oral hygiene. If it is increased beyond 3 mm it is called as a *pocket*. The cause for this is mainly extension of inflammation leading to pathologic deepening of the gingival sulcus, and this marks the transition from gingivitis to periodontitis. Periodontitis is always preceded by gingivitis, but not all gingivitis progresses to periodontitis.

DEFINITION

"Pocket can be defined as deepening of the gingival sulcus." If this happens due to coronal migration of the marginal gingiva it is called as gingival or pseudo-pocket. Deepening due to apical migration of the junctional epithelium is referred to as "true pocket".

CLASSIFICATION (FIG. 23.1)

1. *Depending upon its morphology*
   a. Gingival/false/relative pocket.

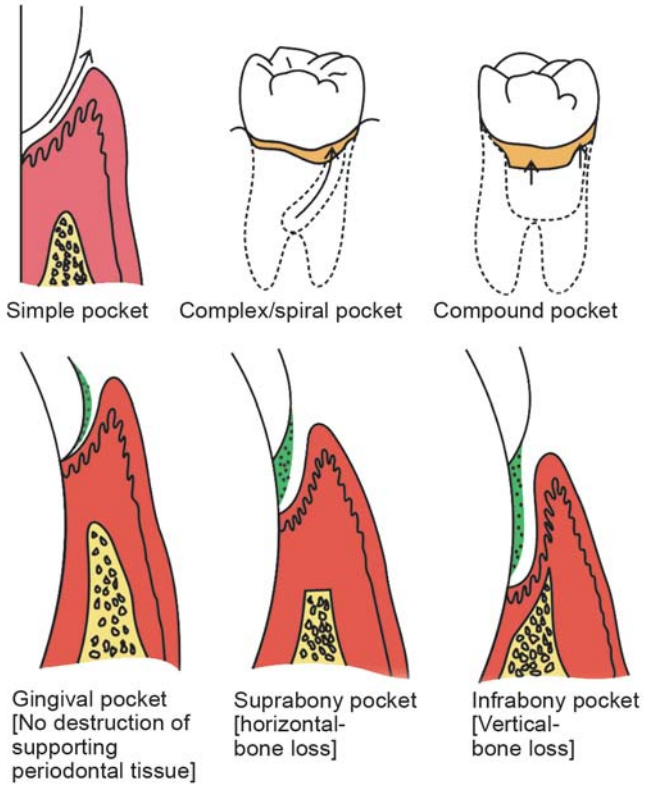


Fig. 23.1: Types of pockets

   b. Periodontal/absolute/true pocket.
   c. Combined pocket.
2. *Depending upon its relationship to crestal bone periodontal pockets are further classified as:*
   a. Suprabony/supracrestal/supra-alveolar pocket.
   b. Infrabony/intrabony/subcrestal/intra-alveolar pocket.
3. *Depending upon the number of surfaces involved*:
   a. Simple pocket—involving one tooth surface.
   b. Compound pocket—involving two or more tooth surfaces.
   c. Complex pocket—where the base of the pocket is not in direct communication with the gingival margin. It is also known as *spiral pocket*.
4. *Depending upon the nature of the soft tissue wall of the pocket*
   a. Edematous pocket.
   b. Fibrotic pocket.
5. *Depending upon the disease activity*
   a. Active pocket.
   b. Inactive pocket.

CLINICAL FEATURES

Signs

1. Enlarged, bluish-red marginal gingiva with a 'rolled' edge separated from the tooth surface.
2. A bluish-red vertical zone extending from the gingival margin to the alveolar mucosa.
3. A break in the faciolingual continuity of the interdental gingiva.
4. Shiny, discolored and puffy gingiva associated with exposed root surfaces.
5. Gingival bleeding, purulent exudate from the gingival margin.
6. Mobility, extrusion and migration of teeth.
7. The development of diastema where none had existed previously.

Symptoms

a. Localized pain or a sensation of pressure in the gingiva after eating, which gradually diminishes.
b. A foul taste in localized areas.
c. A tendency to suck material from the interproximal spaces.
d. Radiating pain "deep in the bone".
e. A "gnawing' feeling or feeling of itching in the gums.
f. The urge to dig a pointed instrument into the gums and relief is obtained from the resultant bleeding.
g. Patient complains that food "sticks between the teeth" or that the teeth "feel loose" or a preference to "eat on the other side."
h. Sensitivity to heat and cold; toothache in the absence of caries.

PATHOGENESIS (Figs 23.2A to G)

The first event in pocket formation is the laying down of a lawn of gram-positive bacteria on the supragingival tooth surface and its extension into the gingival sulcus.

The periodontal pockets are caused by micro-organisms and their products, which produce pathologic changes that lead to the deepening of the gingival sulcus.

As a result of inflammation, the following changes are seen in the junctional epithelium.

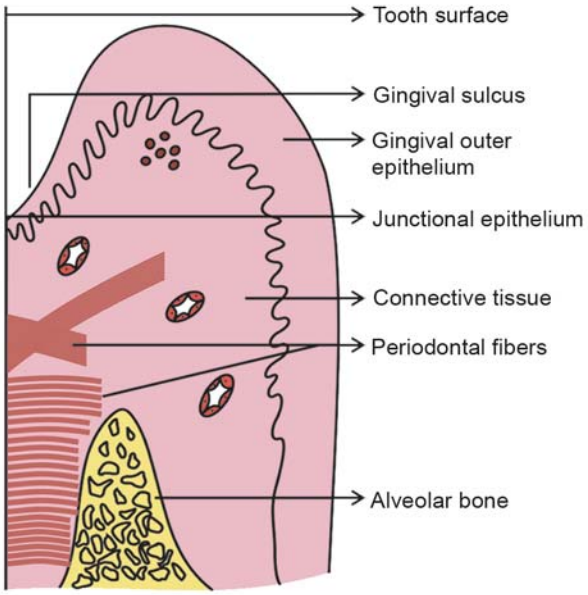


Fig. 23.2A: Schematic illustration of normal gingiva

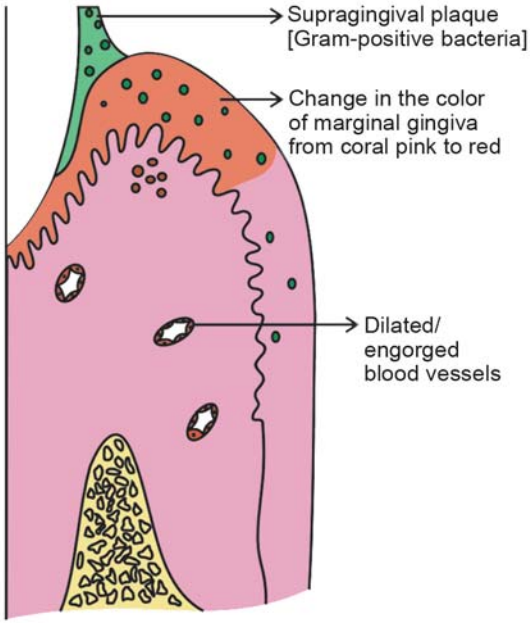


Fig. 23.2C: Extension of supragingival plaque into the gingival sulcus

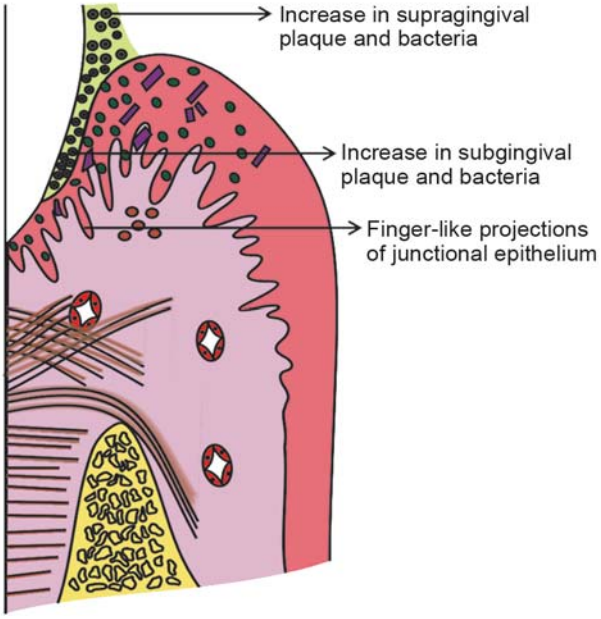


Fig. 23.2B: Accumulation of supragingival plaque

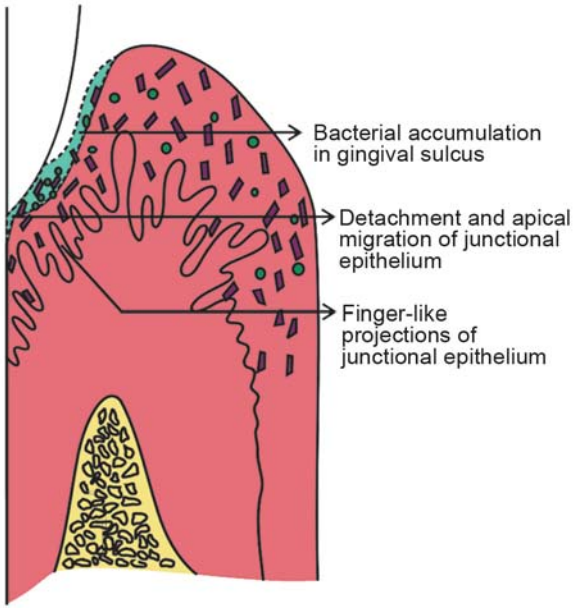


Fig. 23.2D: Detachment and apical migration of junctional epithelium

1. The junctional epithelium proliferates along the root in the form of finger-like projections.
2. The coronal portion of the junctional epithelium detaches from the root as the apical portion migrates. The epithelial cell detachment can take place due to:
   a. Enzymes released by bacteria
   b. Physical force exerted by rapidly growing bacteria
   c. Bacteria may also interfere with growth and synthetic activities of junctional epithelial cells and also with the normal maintenance of the attachment lamina.
   d. Exudate associated with the advancing bacteria may also be important.

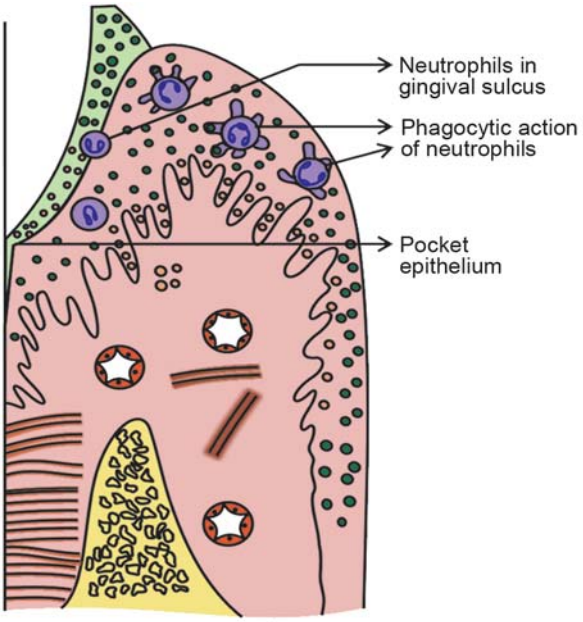


Fig. 23.2E: Phagocytic action of neutrophils

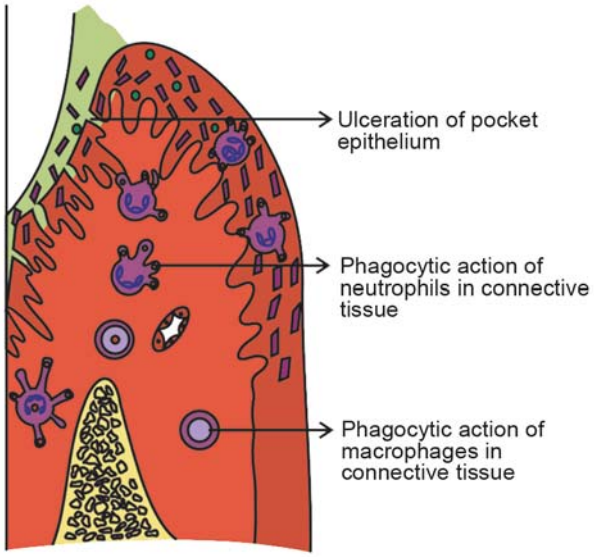


Fig. 23.2F: Ulceration of pocket epithelium

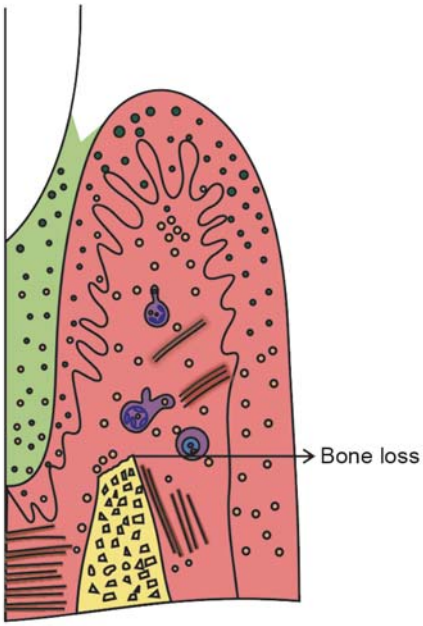


Fig. 23.2G: Periodontal pocket is established

Thus the sulcus base shifts apically and this will be replaced by pocket epithelium.

3. Under normal conditions a constant stream of neutrophils emigrate from the vessels of the gingival plexus through the junctional epithelium into the gingival sulcus and oral cavity. Most bacteria produce substances that chemotactically attract neutrophils. A chemical gradient of chemotactic agents seems to exist across normal, intact junctional epithelium and connective tissue. Neutrophils leaving the blood vessels are guided by this gradient towards the gingival margin and into the gingival sulcus. Under normal conditions, the transmigrating cells leave no trace of their passage and cause no damage. These neutrophils are the primary and first line of defence around the teeth; the epithelial barrier is the second.
4. Extension of plaque subgingivally causes an increase in the number of transmigrating neutrophils, which may be due to the increased concentration of chemotactic factors and other inflammation induced substances derived from the bacteria. These substances cause vasculitis. Neutrophils adhere to the endothelial lining and migrate into the connective tissue, but they still do not accumulate there. Instead they rapidly pass through the junctional or pocket epithelium to form a thick layer that covers the surface of the subgingival plaque. Upon arrival at the plaque surface, the neutrophils are viable partly, but not completely functional. Their role is to

limit further extension and spread of bacteria by phagocytosis and killing. This may be inhibited by lipopolysaccharides and leukotoxin. A constant battle occurs at the neutrophil plaque interface. Usually the normal neutrophil activities are sufficient to limit the extension of plaque, however increasing growth rate of bacteria, swamps the neutrophil system and permits tissue destruction to occur.

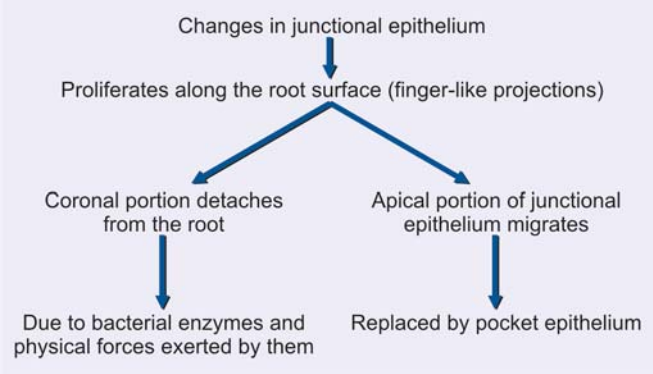
5. Assuming the pocket formation proceeds either because of aggressive growth and action of bacteria or because of ever increasing number of neutrophils that transmigrate in constant streams through the junctional epithelium and pocket epithelium causes open communication between the pocket and connective tissue (by disrupting the epithelial barrier).

   Ulceration of this sort is the second major event in pocket formation. Once the epithelial barrier is breached, the gradient of chemotactic agents released by the pocket bacteria may be disrupted. As a result the neutrophils no longer have a guidance systems, to direct them from the vessels through tissues and into the pocket; they remain in the connective tissue moving randomly. Also because of the rupture of the epithelial barrier, the connective tissue become flooded with the bacterial substances, and bacteria may enter the connective tissue.
6. The neutrophils now encounter these substances within the connective tissue rather than outside in the pocket. The neutrophils become activated and undertake phagocytosis with resultant release of lysosomal enzymes, collagenases and other substances (PGE_2) that cause extensive tissue damage.

   As soon as the bacterial substances have entered the connective tissue many systems other than the neutrophils are activated like macrophages lymphocytes and complement system.
7. When the epithelial barrier is re-established the chemotactic gradient is formed again and the destructive process subsides. If this barrier is not re-established, tissue destruction continues and alveolar bone is resorbed. A periodontal pocket is now established.

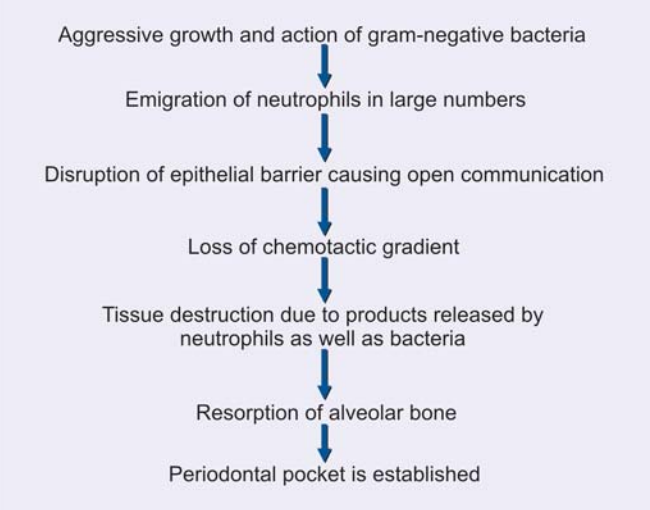
Summary of Pathogenesis

First Event



Colonization of Gram-positive bacteria supragingivally and its extension into the gingival sulcus and conversion of Gram-positive aerobes to Gram-negative anaerobes.

Second Event



HISTOPATHOLOGY

During the initial stages of pocket formation the changes that takes place are described in stages I, II, III gingivitis. Once the pocket is formed the following microscopic features are present.

Changes in the Soft Tissue Wall

- The blood vessels are engorged and dilated.
- The connective tissue is edematous and densely infiltrated with plasma cells (80%), lymphocytes and

a scattering of polymorphonuclear leukocytes (PMNLs).

- Special note should be made of the fact that extension of the junctional epithelium along the root requires the presence of healthy epithelial cells, because of this it is reasonable to assume that the degenerative changes occur along the lateral walls of the pocket.

The changes are as follows:

1. The epithelium along the lateral wall of the pocket presents striking proliferative and degenerative changes.
2. The epithelial projection extends deep into the connective tissue and also extends further apically than the junctional epithelium.
3. The epithelium is infiltrated with leukocytes and other inflammatory cells.
4. Degeneration and necrosis of the epithelium leading to ulceration of the epithelium and exposure of the underlying connective tissue.
5. Bacterial invasion along the lateral and apical areas of the pocket. Some bacteria traverse the basement lamina and invade the subepithelial connective tissue.

The following morphotypes of bacteria have been identified, filamentous rods and coccoid organisms with predominant Gram-negative bacteria. Increased numbers of Langerhans' cells were found in the epithelium.

Microtopography of the Gingival Wall of the Pocket

Under scanning electron microscope the following areas have been noted:

1. Areas of relative quiescence.
2. Areas of bacterial accumulation—mainly cocci, rods, filamentous rods with a few spirochetes.
3. Area of emergence of leukocytes.
4. Area of leukocyte—bacterial interaction.
5. Areas of intense epithelial desquamation.
6. Areas of ulceration with exposed connective tissue.
7. Areas of hemorrhage with numerous erythrocytes.

The transition could be postulated as follows:

Bacteria accumulate in the previously quiescent areas, triggering the emergence of leukocytes and the leukocyte-bacterial interaction. This leads to intense epithelial desquamation and finally to ulceration and hemorrhage.

Periodontal Pocket as a Healing Lesion

It is characterized by interplay of destructive and constructive tissue changes.

The destructive changes are characterized by fluid and cellular inflammatory exudates and by the associated degenerative changes initiated by the plaque bacteria.

The constructive changes consist of the formation of blood vessels in an effort to repair the tissue damage caused by inflammation. The balance between destructive and constructive changes determines clinical features such as color, consistency and surface texture of the pocket wall.

Pocket Contents

It consists of debris principally containing micro-organisms and their products, (like enzymes, endotoxins and other metabolic products) dental plaque, gingival fluid, food remnants, salivary mucin, desquamated epithelial cells and leukocytes. If purulent exudate is present, it consists of living, degenerated and necrotic leukocytes (PMNLs), living and dead bacteria, serum and a scanty amount of fibrin. Pus formation is a common feature in periodontal disease but it is only a secondary sign.

Changes in the Root Surface Wall

1. *Structural changes*:
   a. Presence of pathologic granules
   b. Areas of increased mineralization
   c. Areas of demineralization/root caries
2. *Chemical changes*: The mineral content of exposed cementum is increased. The following minerals are increased in diseased root surfaces: calcium, magnesium, phosphate, fluoride and others. Hence, a highly increased resistant calcified layer to decay is formed. This can also be harmful if the adsorbed products are toxic.

3. *Cytotoxic changes*: Bacterial penetration into the cementum can be found as deep as cementodentinal junction. In addition, bacterial products such as endotoxins have also been detected.

The following zones can be found in the base of a periodontal pocket (Fig. 23.3):

1. Cementum covered by calculus where all the changes described earlier takes place.
2. Attached plaque which covers calculus (100 to 500 u)
3. The zone of unattached plaque.
4. The zone where the junctional epithelium is attached to the tooth. This zone, which is normally more than 500 µ is usually reduced to less than 100 µ in periodontal pocket.
5. Apical to the junctional epithelium there may be a zone of semidestroyed connective tissue fibers.

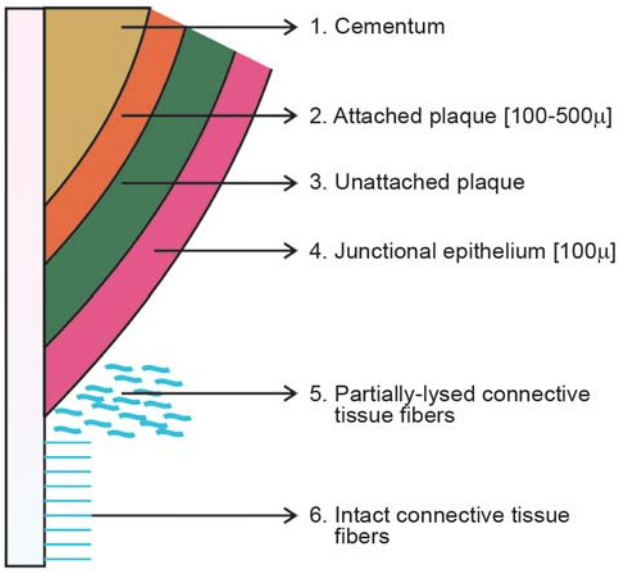


Fig. 23.3: Zones in the base of the periodontal pocket

Periodontal Disease Activity

a) Periods of exacerbation / Periods of activity	b) Periods of remission quiescence / inactivity
Bone and connective tissue attachment are lost as pocket deepens. Clinically - active periods show bleeding, either spontaneously or with probing and greater amounts of attachment loss, presence of exudation or pus.	Reduced inflammatory response, little or no loss of bone and connective tissue attachment.

Relation of Loss of Attachment and Bone Loss to Pocket Depth (Fig. 23.4)

Pocket of same depth may be associated with different degree of attachment loss. Pocket of different depth may be associated with same amount of attachment loss.

Area between the base of the pocket and the alveolar bone is always constant. The radius of action of the plaque bacteria is 0.5 to 2.7 mm.

PERIODONTAL CYST

- It is an uncommon lesion that produces localized destruction of the periodontal tissues along the lateral root surface.
- Most common in the mandibular canine—premolar area. The following possible etiologies have been suggested:
- Odontogenic cyst caused by proliferation of the epithelial rests of Malassez.
- Lateral dentigerous cyst (retained in the jaw after tooth eruption).
- Primordial cyst of supernumerary tooth germ.
- Stimulation of epithelial rests of the periodontal ligament by infection from a periodontal abscess or from the pulp through an accessory canal.

Usually asymptomatic but may present as a localized tender swelling. Radiographically seen at the site of the root as a radiolucent area bordered by a radiopaque line (can not be differentiated radiographically from periodontal abscess).

Determination of Pocket Depth

- Probing depth measurement (Fig. 23.5).
- Clinical detection of attachment loss.
- Clinical detection of suprabony and infrabony pockets.

Clinically, probing depth measurement is recorded from the crest of the marginal gingiva to the probable depth of the pocket. Whereas attachment level is measured from cementoenamel junction to the probable depth of the pocket. To differentiate between pseudo and true pocket, attachment level measurement should be considered.

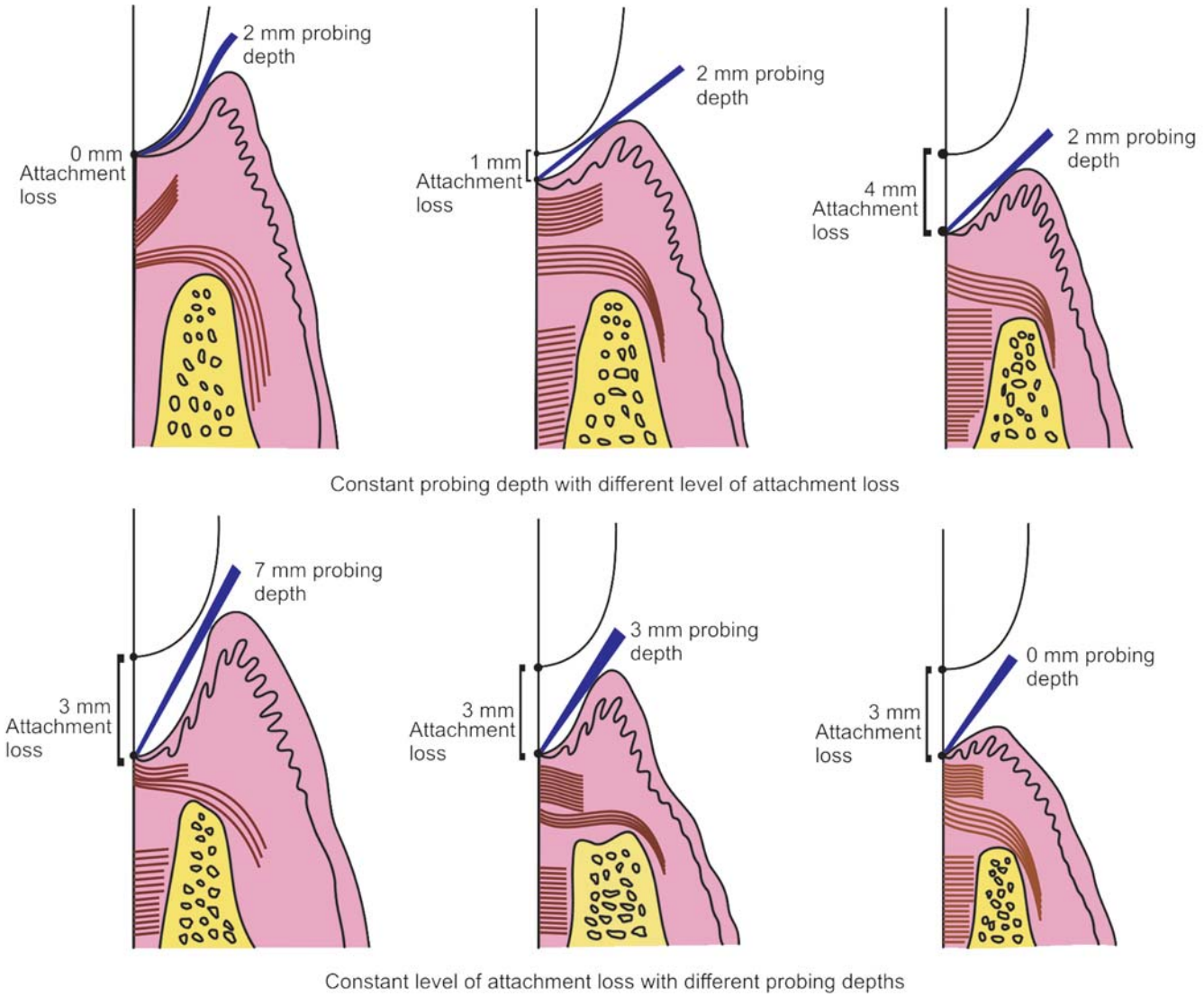


Fig. 23.4: Relation of loss of attachment

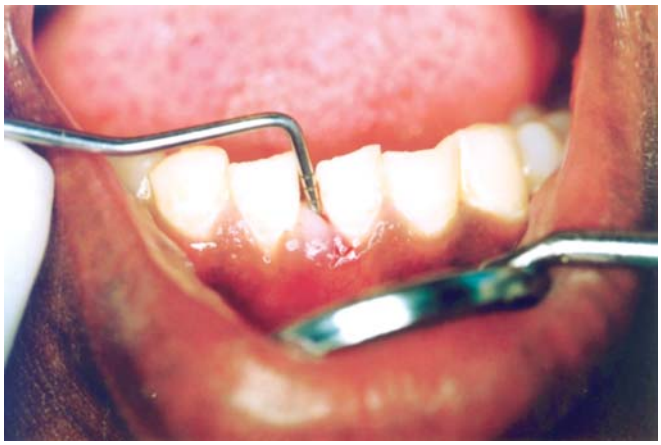


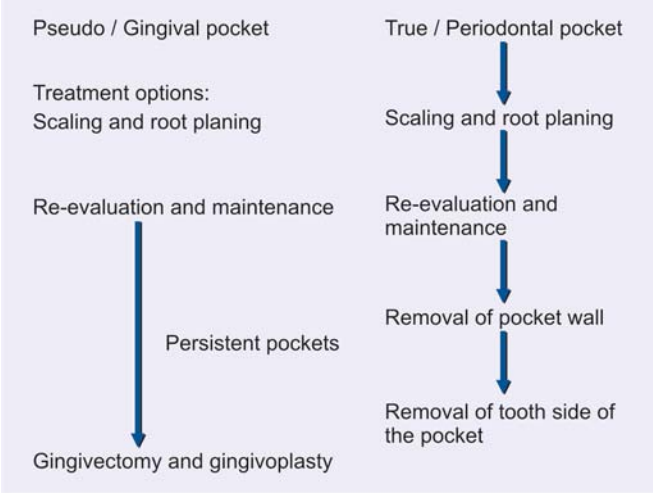
Fig. 23.5: Probing of a pocket

Clinical detection of attachment level is done by first identifying the cementoenamel junction (CEJ) with the probe tip (since junctional epithelium is attached at the CEJ), if the probe passes beyond the point of CEJ then it is considered to be a true pocket.

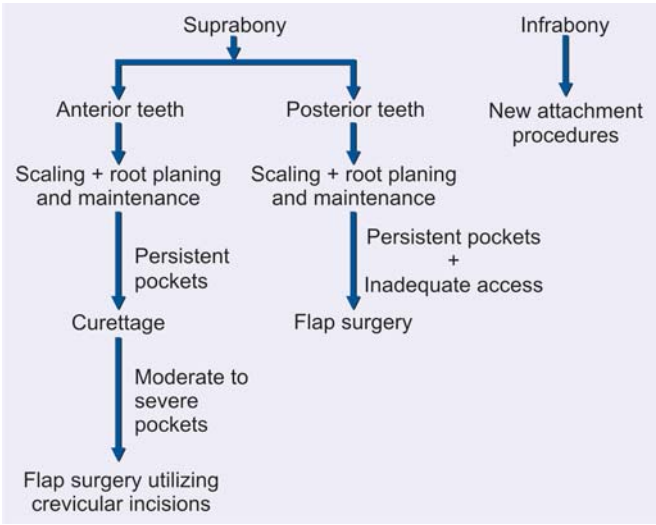
While probing, soft tissue resistance on lateral pressure indicates suprabony pocket and hard tissue resistance indicates infrabony pocket.

TREATMENT OF PERIODONTAL POCKET

I. *Treatment of pocket depends on the type of pocket*



II. *Treatment of suprabony and infrabony pockets*



III. *Treatment of pockets can also be classified under three main headings.*

a. *New attachment techniques*: It offers ideal result by reuniting the gingiva to the tooth at a position coronal to the base of pre-existing pocket. Here all the structures of lost periodontium are restored. Following are the techniques for new attachment:
   - Non-graft associated new attachment procedures.
   - Graft associated new attachment procedures.
   - Combined techniques.

b. *Removal of pocket wall by,*
   1. Retraction or shrinkage, e.g. scaling and root planing.
   2. Surgical removal by gingivectomy or by means of an undisplaced flap.

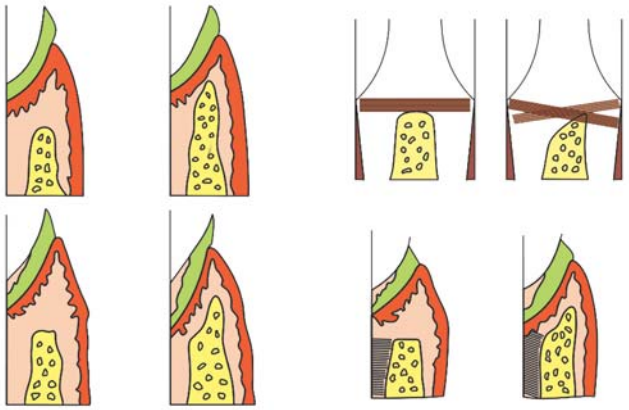


Fig. 23.6: Differences between suprabony and infrabony pockets

   3. Apical displacement of pocket wall by apically displaced flap.

c. *Removal of the tooth side of the pocket*, by tooth extraction or partial tooth extraction such as hemisection or root resection.

Table 23.1: Differences between suprabony and infrabony pocket (Fig. 23.6)

Suprabony pockets	*Infrabony pockets*
a. The base of the pocket is coronal to the crest of alveolar bone.	a. The base of the pocket is apical to the crest of the alveolar bone.
b. The pattern of bone destruction is horizontal	b. The pattern of bone destruction is vertical/angular
c. Interproximally, the transeptal fibers are arranged horizontally.	c. Interproximally, the transeptal fibers are arranged in an oblique pattern, extending from cementum below the pocket over to the cementum of the adjacent tooth.
d. On the facial and lingual surfaces, the periodontal ligament fibers follow normal horizontal-oblique course between the tooth and bone.	d. On facial and lingual surfaces the periodontal ligament fibers follow the angular pattern of the adjacent bone.

KEYPOINTS

1. Pockets are defined as the pathologic deepening of the gingival sulcus.
2. They can be pseudo or true pockets; simple, compound or complex pockets; suprabony or infrabony pockets.
3. Microscopically, in a periodontal pocket certain changes are observed in both the soft tissue wall and the root surface.

4. Clinically, true pocket can be identified by recording the attachment level (which is measured from the cementoenamel junction to the probable depth of the pocket).
5. Pockets can be treated by: (a) new attachment techniques, (b) removal of the pocket wall; (c) removal of the tooth side of the pocket.
6. Only infrabony pockets can be treated successfully by various regenerative procedures.

KNOW MORE ...

Significance of Pus Formation

Pus is a common feature of periodontal diseases but it is only a secondary sign. It merely reflects the nature of the inflammatory changes in the pocket.

A periodontal abscess is a localized purulent infection of the periodontal tissues which may lead to destruction of periodontal ligament and alveolar bone. It is also referred to as a lateral or parietal abscess. Periodontal abscess can be classified.

a. According to location
   - Abscess along the lateral aspect of the root
   - Abscess in the soft tissue wall of the deep periodontal pocket

b. According to onset and course
   - Acute periodontal abscess
   - Chronic periodontal abscess

c. Depending on number
   - Single and multiple periodontal abscess

Causes of Periodontal Abscess

Mainly divided into:

I. Abscess related to periodontal diseases.
II. Periodontal abscess caused by other factors (Non-periodontitis related abscess).

Periodontal abscess may occur in the following ways:

1. Deep extension of infection from a periodontal pocket into the supporting tissues and localization of suppurative inflammatory process along the lateral aspect of the root.
2. Lateral extension of inflammation from inner surface of a pocket into the connective tissue of the pocket.
3. The existence of tortuous pockets, with cul-de-sac that eventually becomes isolated and may favour abscess formation.
4. Incomplete removal of calculus during treatment of a periodontal pocket may lead to occlusion of the pocket orifice due to shrinkage of gingival wall and a periodontal abscess may form in the sealed-off portion of the pocket.
5. Treatment with systemic antibiotics along with root planing in advanced periodontal patients may result in abscess formation. Other factors that may cause periodontal abscess include:
   a. Impactation of foreign bodies especially related to oral hygiene aids (toothbrush bristle, piece of dental floss) have been termed as "oral hygiene abscess"
   b. Perforation of the lateral wall of root in endodontic therapy. (Differences between gingival abscess, periodontal abscess and periapical abscess is discussed in detail in Chapter 'Gingival enlargement').

Treatment options for periodontal abscess are:

a. Drainage through pocket retraction or incision.
b. Non-surgical therapy including scaling root planing and antimicrobial therapy.
c. Periodontal surgery.
d. Extraction of the offending tooth.

Antibiotic options for acute, periodontal abscess are:

- Amoxicillin 500 mg three times a day for 3 days.
- Clindamycin – 600 mg loading dose subsequently 300 mg four times a day for 3 days.
- Azithromycin, 500 mg four times a day for 3 days.

REVIEW QUESTIONS

1. Define, classify periodontal pockets, and describe the pathogenesis and treatment of a periodontal pocket.
2. Describe the various pocket elimination procedures.
3. Describe the histopathology of a periodontal pocket.
4. What are the root surface changes associated with a periodontal pocket?
5. Differentiate between suprabony and infrabony pockets.

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24 Chapter

Bone Loss and Patterns of Bone Destruction

- NORMAL ANATOMY OF ALVEOLAR BONE
- MECHANISM OF BONE FORMATION AND BONE DESTRUCTION
  - Local Factors
  - Systemic Factors
- FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE
- BONE DESTRUCTION PATTERNS IN PERIODONTAL DISEASE
- PREVALENCE AND DISTRIBUTION OF BONE DEFECTS IN MODERATE ADULT PERIODONTITIS

INTRODUCTION

Osseous defects are those defects, which are formed as a result of destruction of alveolar bone due to periodontal disease. The normal height of alveolar bone is at cemento-enamel junction and this height is maintained by physiologic equilibrium between bone formation by osteoblasts and bone loss by osteoclasts, which in turn are regulated by local and systemic influences.

NORMAL ANATOMY OF ALVEOLAR BONE (FIG. 24.1)

Alveolar bone is that part of jaw bone that surrounds and supports the teeth. It has facial and lingual cortical plate or compact bone between which cancellous bone is sandwiched.

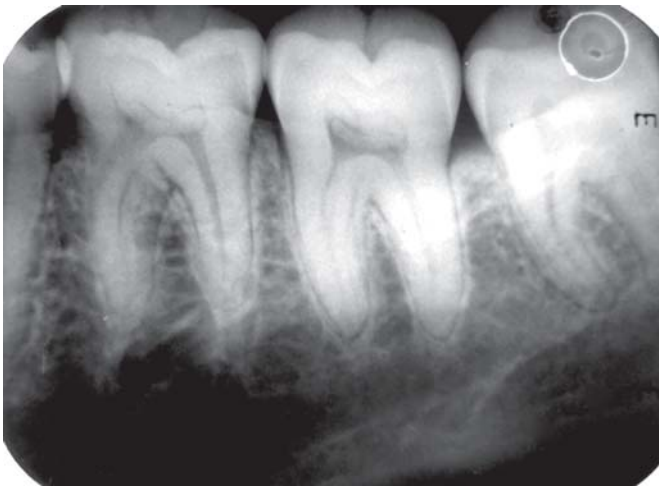

Fig. 24.1: Normal anatomy of alveolar bone

It is partially tooth-dependent and hence it is resorbed once the tooth is extracted. The shape, size, thickness varies

in different regions of the same mouth. The margins of the alveolar crest run parallel to the cementoenamel junction at a remarkably constant distance of 1 to 2 mm.

MECHANISM OF BONE FORMATION AND BONE DESTRUCTION

Osteoblasts are the primary cells responsible for the synthesis of the bone matrix, which subsequently undergoes calcification. Initially, uncalcified matrix, called osteoid, is formed and this is mineralized as a result of deposition of crystals of hydroxyapatite.

Bone destruction in periodontal disease is caused by local factors and systemic factors.

Bone destruction in periodontal disease is not a process of bone necrosis. It involves the activity of the live cells along the viable bone. Tissue necrosis and pus if present are seen in the soft tissue walls of the periodontal pocket but not along the resorbing margins of the underlying bone.

Local Factors

Local factors could be:

a. Chronic gingival inflammation
b. Trauma from occlusion
c. Combination of both

Role of Chronic Gingival Inflammation

It is the most common cause for bone destruction in periodontal disease. It is believed that inflammation spreads from the gingiva into the deeper tissues along two pathways (This marks the transition from gingivitis to periodontitis).

The transition from gingivitis to periodontitis is associated with changes in the composition of bacterial plaque or resistance of the host. The lesion presents with most pathogenic bacteria, inflammatory cell infiltrate, the lesion becoming more progressive and destructive with the conversion of T-lymphocyte to B-lymphocytic lesion.

Pathway of spread of inflammation could be,
Interproximally:

a. From the gingiva
↓
Bone
↓
Periodontal ligament

b. From the gingiva
↓ (Less common; seen in trauma from occlusion)
Periodontal ligament

Facially and lingually:

a. From the gingiva along the outer periosteum
↓
Into the bone

b. From the gingiva
↓
Into the periodontal ligament

When the inflammation reaches the bone by extension from the gingiva, it spreads into marrow spaces and it replaces it with a leukocytic and fluid exudate, new blood vessels and proliferating fibroblasts. Multinuclear osteoclasts and mononuclear phagocytes are increased in number and bone surfaces are lined with cone-like resorption lacunae. In the marrow spaces, resorption proceeds within, causing first thinning of the surrounding bony trabeculae and enlargement of marrow spaces, followed by destruction of bone and reduction in bone height. Around the resorption areas, normally fatty bone marrow is partially or totally replaced by a fibrous type of marrow.

In summary, the changes in the bone could be as follows:

Gingival inflammation
↓
Marrow spaces
↓
Replaced by leukocytes and fluid exudates,
new blood vessels and proliferating fibroblasts
↓
Increase in osteoclasts and mononuclear cells
↓
Thinning of bone trabeculae and enlargement
of the marrow spaces
↓
Destruction of the bone and reduction in bone height
↓
Replacement of fatty bone marrow with the fibrous type
(around the resorption areas)

The following are the possible pathways by which bone destruction is caused by extension of gingival inflammation (by Hausmann):

1. Direct action of plaque products on bone progenitor cells to release osteoclasts.
2. Plaque products act directly on bone, destroying it through a non-cellular mechanism.
3. Plaque products stimulate gingival cells, to release mediators which in turn induces progenitor cells to differentiate into osteoclasts.
4. Stimulates gingival cells to release agents that destroy bone by direct chemical action without osteoclasts.
5. Plaque products act as co-factors in bone resorption.

It has been hypothesized that two cell types are responsible for bone resorption:

1. *Osteoclast*: Removes the mineral portion of bone.
2. *Mononuclear cells*: Plays a role in organic matrix degradation.

Both are found near the resorbing bone.

Bone Destruction Caused by Trauma from Occlusion Alone

Trauma from occlusion in the absence of inflammation can cause the following changes:

- Increased compression and tension of periodontal ligament.
- Increased osteoclasis of alveolar bone and necrosis of periodontal ligament.

These changes are reversible, if offending forces are removed. However, persistent trauma from occlusion results in funnel-shaped bony defects.

Systemic Factors

Local and systemic factors regulate the physiologic equilibrium of bone. When there is generalized tendency towards bone resorption, bone loss is initiated by a local inflammatory process that may be magnified. This systemic influence on the response of alveolar bone has been termed as the *bone factor concept* in periodontal diseases. In recent years, a lot of studies focussed on the possible relationship between periodontal bone loss and osteoporosis. Osteoporosis is a physiologic condition of postmenopausal women, resulting in loss of bone mineral content and microstructural bone changes. Periodontal bone loss may also occur in generalized skeletal disturbances (e.g. hyperparathyroidism, leukemia, etc.) by mechanism that may not be related to the usual periodontal bone destruction.

Pharmacological Agents and Bone Resorption

These include prostaglandins and their precursors and osteoclast activating factors, all of which are present in inflamed gingiva. Complement can also induce bone resorption by enhancing the synthesis of prostaglandins. Prostaglandins are synthesized by fatty acid precursors such as arachidonic acid and is controlled by cyclo-oxygenase pathway. Flurbiprofen (NSAID) is a potent inhibitor of cyclo-oxygenase pathway of arachidonic acid metabolism which retards the rate of bone loss.

Radius of Action

Some of the authors suggested that, locally produced bone resorption factors may have to be present in the proximity of the bone surface to be able to exert their action. On the basis of Waerhaug's measurements, it was postulated that there is a range of effectiveness of about 1.5 to 2.5 mm within which bacterial plaque can induce bone loss, beyond 2.5 mm there is no effect. Interproximal angular defects can appear only in spaces wider than 2.5 mm because narrower spaces are destroyed completely, Large defects far exceeding 2.5 mm can be seen in certain conditions, like localized juvenile periodontitis and Papillon-Lefevre syndrome, may be caused by the presence of bacteria within the tissues.

Rate of Bone Loss

Loe and associates found the rate of bone loss on an average to be about 0.2 mm a year for facial surfaces and about 0.3 mm a year for proximal surfaces, when periodontal disease is allowed to progress untreated.

Periods of Destruction

Periodontal destruction occurs in an episodic, intermittent pattern characterized by periods of activity and exacerbation followed by periods of remission and quiescence. The destruction results in loss of collagen and alveolar bone and deepening of periodontal pocket.

The reasons for the onset of destructive patterns are not totally understood but the following theories have been offered:

1. Bursts of activity is associated with subgingival ulceration and an acute inflammatory reaction resulting in loss of alveolar bone.
2. Bursts of activity coincides with a predominantly T-lymphocyte lesion to one with predominance of B-lymphocyte plasma cell infiltrate.
3. Periods of exacerbation is associated with an increase in loose, unattached motile, gram-negative, anaerobic pocket flora and period of remission coincides with the formation of a dense, unattached, non-motile, gram-positive flora.
4. Presence of antibodies.

FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE

Normal Variation in Alveolar Bone

It can affect the osseous contours produced by periodontal disease. The anatomic features that substantially affect the bone destructive pattern in periodontal disease include the following:

- Thickness, width and crestal angulation of the interdental septa
- Thickness of facial and lingual alveolar plates
- The presence of fenestrations and dehiscences
- Increased thickness of alveolar bone margins to accommodate functional demands
- The alignment of the teeth, root trunk anatomy.

For example, angular bone defects cannot form in thin facial and lingual alveolar plates, which have little or no cancellous bone between the outer and inner cortical layers. In such an instance the entire crest of the bone is destroyed and the height of bone is reduced.

BONE DESTRUCTION PATTERNS IN PERIODONTAL DISEASE

Horizontal Bone Loss

It is the most common pattern of bone loss in periodontal disease. The bone is reduced in height but the bone margins remain roughly perpendicular to the tooth surface (Fig. 24.2).

Vertical or Angular Defects (Figs 24.3 and 24.4)

They are those that occur in an oblique direction, leaving a hollowed out trough in the bone alongside the root, the base of the defect is located apical to the surrounding bone. In most situations angular defects are accompanied by infrabony pockets.

Angular defects are classified on the basis of number of walls present as:

- One-walled or hemiseptal defect—One wall is present
- Two-walled defect—Two walls are present

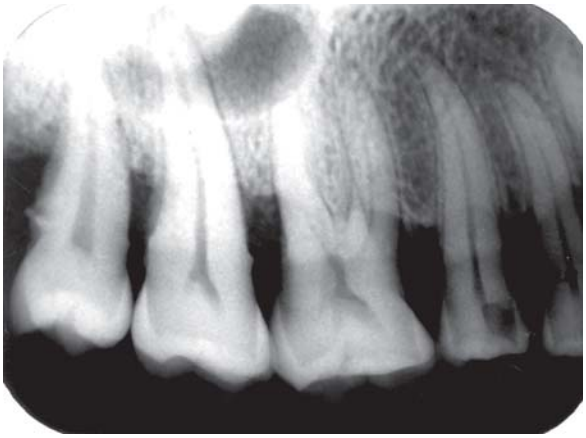

Fig. 24.2: Radiographic illustration of horizontal bone loss

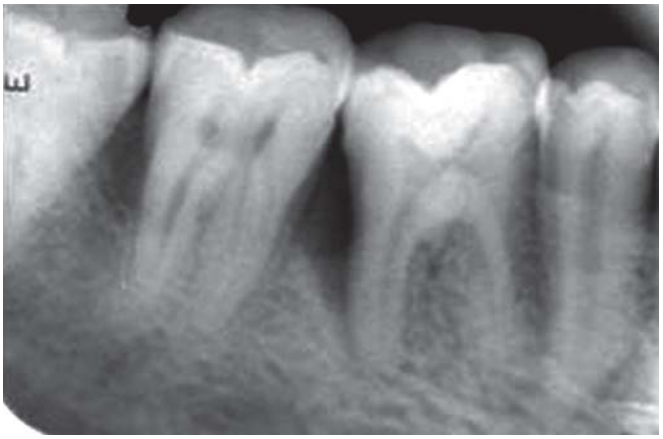

Fig. 24.3: Vertical bone defect in relation to lower first molar

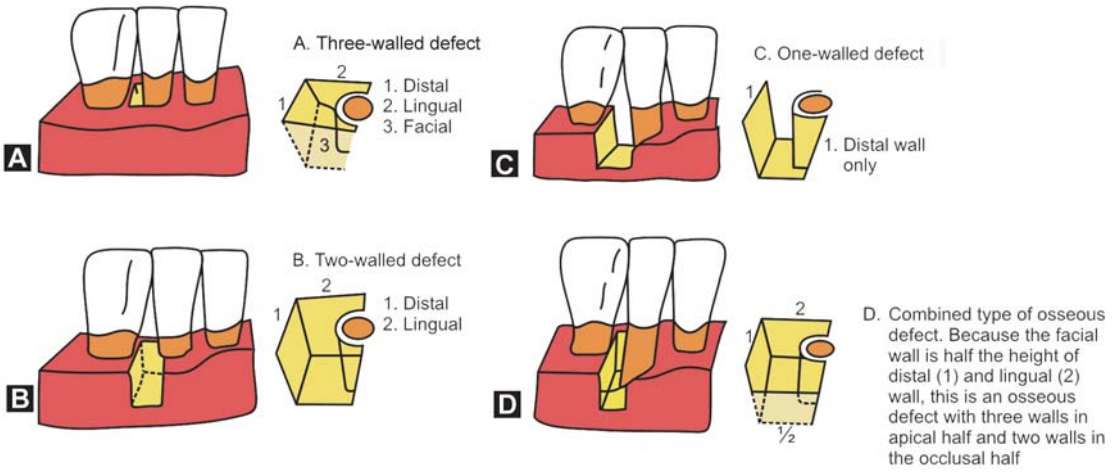

Fig. 24.4: Types of angular defects

- Three-walled or intrabony defect—Three walls are present (more common on mesial surfaces of upper and lower molars)
- *Combined osseous defect*—The number of walls in the apical portion of the defect are greater than that in its occlusal portion. Radiographs may help upto some extent to locate vertical defects, but the best would be surgical exposure of the defect.

Osseous Craters (Fig. 24.5)

They are concavities in the crest of the interdental bone confined within the facial and lingual walls. It is found to make up two-thirds of all mandibular defects, can be diagnosed by transgingival probing.

The following reasons have been suggested for the high frequency of interdental craters.

- Interdental areas are more prone to the accumulation of plaque and are more difficult to clean
- The normal flat or even concave faciolingual shape of the interdental septum in lower molars may favor crater formation
- Vascular patterns from the gingiva to center of the crest may provide a pathway for inflammation.

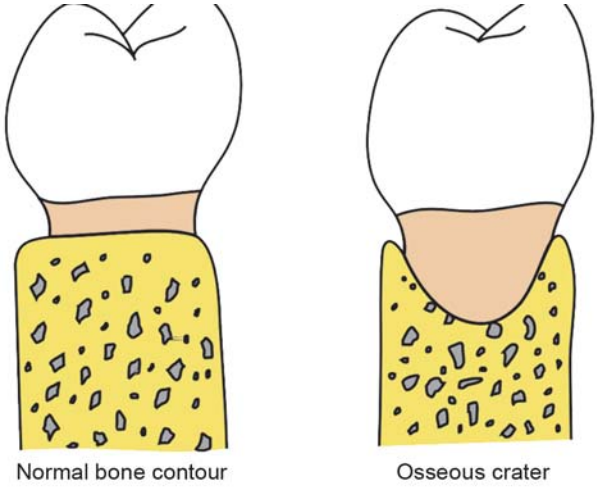

Fig. 24.5: Diagrammatic representation of an osseous crater in a faciolingual section between two lower molars

Bulbous Bony Contours (Fig. 24.6)

They are bony enlargements caused by exostoses, adaptation to function or buttressing bone formation. They are found more frequently in the maxilla than mandible.

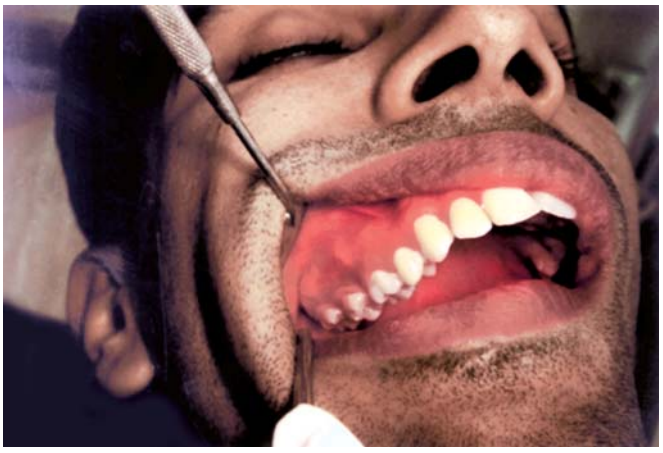

Fig. 24.6: Bony exostosis in the buccal aspect

Reversed Architecture

These defects are produced by loss of interdental bone, including the facial and lingual plates without concomitant loss of radicular bone, thereby reversing the normal architecture (more common in maxilla).

Ledges

They are plateau-like bone margins caused by resorption of thickened bony plates.

Furcation Involvements (Fig. 24.7)

It refers to the invasion of the bifurcation and trifurcation of multirooted teeth by periodontal disease. The mandibular first molars are most common sites and least common are maxillary premolars.

The role of trauma from occlusion in the etiology of furcation involvement is controversial; others include presence of enamel projections into the furcation, presence of accessory pulpal canals. Diagnosis is made by careful probing with Naber's probe and radiograph of this area is helpful, but can be obscured by various factors like, angulation of the beam and radiopacity of adjacent structures.

PREVALENCE AND DISTRIBUTION OF BONE DEFECTS IN MODERATE ADULT PERIODONTITIS

Different classifications of bone defects have been proposed:

I. *Goldman and Cohen (1958)*

According to morphology of bone defects, can be classified as:

- One-walled defect
- Two-walled defect
- Three-walled defect
- Combined defect

II. *Glickman (1964)* classified bony defects as:

1. Osseous/interdental craters
2. Hemiseptal defects
3. Infrabony defects
4. Bulbous bone contours (more seen in maxillae and are enlargement of bone due to exostoses, buttressing bone formations).

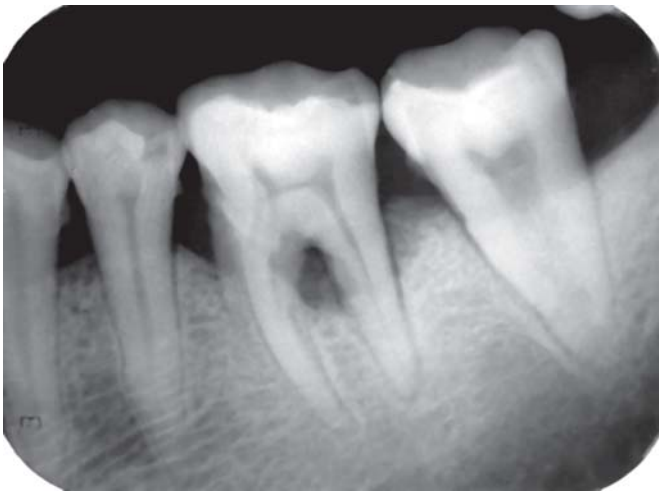

Fig. 24.7: Furcation involvement in relation to mandibular first and second molar (Grade-II)

5. Inconsistent margins and ledges (plateau-like bony margins)
6. Reversed architecture.

III. *Prichard (1967)* expanded this classification and included furcation involvement, anatomic aberrations of the alveolar process, i.e. thick marginal ledges, exostoses and tori, dehiscence and fenestrations.

There is a high prevalence of bony defects in posterior segments (due to thicker bone). Thin bone leads to horizontal defects. In the posterior segments, percentage of osseous defects are more in the mandible. Interdental crater is the most common defect in the molars and hemisepta presents lower proportion.

KEYPOINTS

1. Osseous defects are those defects which are formed as a result of destruction of alveolar bone due to periodontal disease.
2. Osteoblasts are the primary cells responsible for the synthesis of bone matrix, which later on undergoes calcification.
3. Bone destruction in periodontal disease is caused by osteoclasts and mononuclear cells and is mediated by local and systemic factors.
4. Local factors, that are responsible for bone destruction are:
   a. Chronic gingival inflammation
   b. Trauma from occlusion
   c. Combination of both.

5. Bone destruction patterns in periodontal disease are:
   a. Horizontal bone loss.
   b. Vertical or angular defects.
   c. Osseous craters.
   d. Bulbous bony contours.
   e. Reversed architecture.
   f. Ledges.
   g. Furcation involvement.

KNOW MORE ...

Bone Destruction in Periodontal Disease

- Bacteria mediated — LPS, lipoteichoic acid, lipoproteins and others.
- Host mediated — prostaglandins, leukotrienes, cytokines and others.
- Combination of both.

Diagnosis of Osseous Defects

a. Clinical examination — transgingival probing.
b. Radiographs — not very reliable, cannot reveal the extent of involvement and presence/absence of bony walls.
c. Surgical exposure — during flap operations, it is the only reliable method for determining the true architecture of a bony defect.

REVIEW QUESTIONS

1. Describe the mechanism of bone destruction in periodontal disease.
2. Describe the various patterns of bone loss in periodontal disease.

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25 Chapter

Chronic Periodontitis

- DEFINITION
- DIAGNOSTIC CRITERIA
  - Clinical Features
  - Microbiological Features
  - Radiographic Features
- TYPES BASED ON DISEASE DISTRIBUTION AND SEVERITY
- NATURE OF DISEASE PROGRESSION
- RISK FACTORS FOR DISEASE
- GENERAL CONCEPT FOR ETIOLOGY

INTRODUCTION

Chronic periodontitis was previously known as *adult periodontitis* or *slowly progressive periodontitis*.

DEFINITION

Chronic periodontitis occurs as a result of extension of inflammation from the gingiva into the deeper periodontal tissues. It has recently been defined as "an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss".

DIAGNOSTIC CRITERIA FOR CHRONIC PERIODONTITIS (FIGS 25.1 AND 25.2)

Clinical Features

1. Age of onset is usually 30 to 35 years.
2. The disease is usually generalized, although some areas are more deeply involved than the other areas.
3. No consistent pattern of distribution of lesion is seen, except that they are usually not isolated to one or two sites.
4. Highly acute inflammatory sites are not seen, mostly gingiva appear to be slight to moderately swollen and color may range from pale-red to magenta.

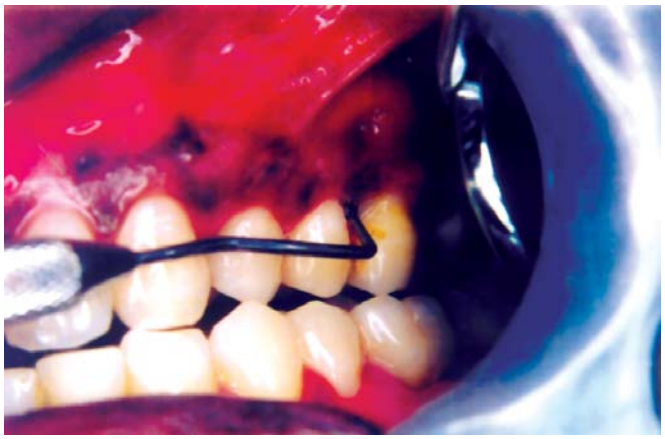

Fig. 25.1: Chronic periodontitis in a 45-year-old male

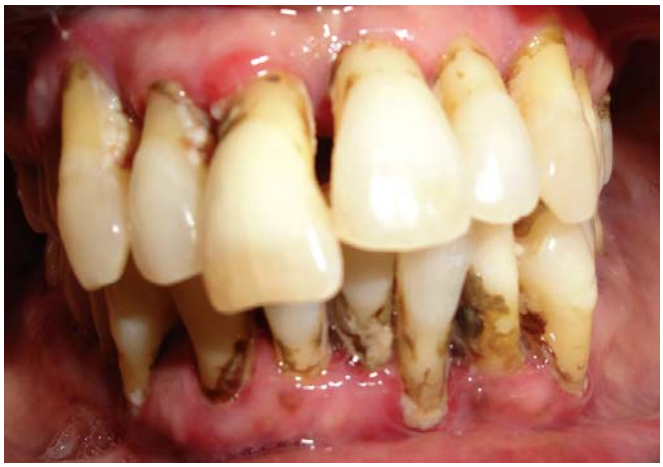

Fig. 25.2: A case of chronic periodontitis

5. Loss of stippling, blunt or rolled gingival margins and flattened or cratered papillae may be seen.
6. Spontaneous bleeding and inflammation related exudate from the pockets may also be found.
7. When the pocket occludes it may result in abscess formation.
8. Pocket depths are variable and both suprabony and infrabony pockets can be found.
9. Conditions that enhance plaque accumulation like open interdental contacts, defective restorative margins and malposed teeth may be frequently seen.
10. The amount of microbial deposits are consistent with severity of the disease.
11. Tooth mobility is seen in advanced cases.
12. No serum neutrophil/monocyte abnormalities are seen.

Microbiological Features

Causative organisms of chronic periodontitis are:

- *Porphyromonas gingivalis (P. gingivalis)*
- *Prevotella intermedia (P. intermedia)*
- *Capnocytophaga*
- *A.actinomycetemcomitans (A.a)*
- *Eikenella corrodens (E. corrodens)*
- *Campylobacter rectus (C. rectus)*

Radiographic Features (Fig. 25.3)

Pattern of bone loss observed in chronic periodontitis may be vertical or horizontal. When attachment loss and bone loss on one tooth surface is greater than that on an adjacent surface is referred to as vertical bone loss and is usually associated with angular bony defects and infrabony pockets. When attachment loss and bone loss occur at a uniform rate on majority of tooth surfaces it is called horizontal bone loss and is generally associated with suprabony pockets.

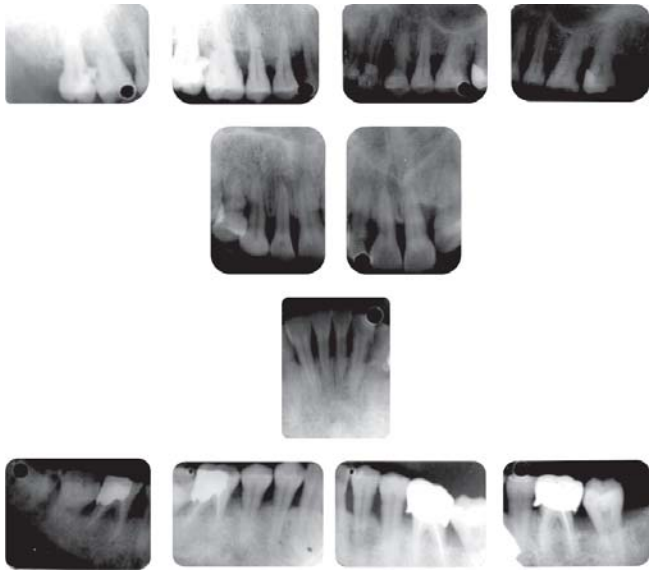

Fig. 25.3: Radiograph showing generalized bone loss

TYPES

Types are based on the following:

Disease Distribution

Localized periodontitis: Periodontitis is considered localized when less than 30 percent of the sites assessed in the oral cavity demonstrate attachment loss and bone loss.

Generalized periodontitis: It is considered generalized when more than 30 percent of the sites assessed in the oral cavity demonstrate attachment loss and bone loss.

Disease Severity

Slight (Mild) Periodontitis

1. Periodontal destruction is generally considered slight when there is not more than 1 to 2 mm of clinical loss of attachment.
2. Usually generalized involvement.

3. Minimal furcation invasions with little or no mobility are observed.
4. Bone loss is minimal (less than 20% of total attachment).

Moderate Periodontitis

It is considered moderate when there is:

1. 3 to 4 mm of clinical attachment loss.
2. Early to moderate furcation involvement with slight to moderate tooth mobility.
3. Bone loss up to 40 percent of the total periodontal attachment on the tooth.

Severe Periodontitis

1. When attachment loss is 7 mm or more, the condition is severe.
2. Furcation involvement up to grade III.
3. Excessive tooth mobility.
4. Bone loss more than 40 percent—both horizontal and angular bony defects are observed.

NATURE OF DISEASE PROGRESSION

The rate of disease progression in chronic periodontitis is slow, but sometimes may be modified by systemic or other underlying factors. Though onset can occur at anytime, because of its slow progression it usually becomes clinically significant in the mid 30s or later. Several models have been proposed to describe the rate of disease progression.

1. Continuous paradigm.
2. Random burst theory.
3. Asynchronous multiple burst hypothesis.

Continuous paradigm implies slow, continuous and progressive destruction of periodontium. This type of progression has been reported in longitudinal studies, not responsive to treatment.

The Random Burst Theory proposes that the progression of disease occurs at short periods of active destruction, which are followed by periods of remission that randomly occur with respect to time and site in an individual. An example for this is chronic adult periodontitis.

In Asynchronous Multiple Burst Model, the tissue destruction occurs at a definite period of time in one's life, then it passes into a state of remission as in juvenile periodontitis.

RISK FACTORS FOR DISEASE

a. *Local factors*: These include plaque and plaque retentive factors. Plaque attached to the tooth and gingival surfaces at the dentogingival junction is considered to be the primary etiologic factor in chronic periodontitis. *P. gingivals*, *B. forythus* and *Treponema denticola* are frequently associated with chronic periodontitis. Plaque retentive factors are those that facilitate plaque accumulation or prevent the removal of plaque by routine oral hygiene procedures. They play an important role in the development of chronic periodontitis because they allow plaque microorganisms to be in close proximity to periodontal tissues. Some of these factors include, calculus, subgingival and/or overhanging margins of restorations, deep carious lesions that extend subgingivally, crowded or malaligned teeth and root surface irregularities (Fig. 25.4).

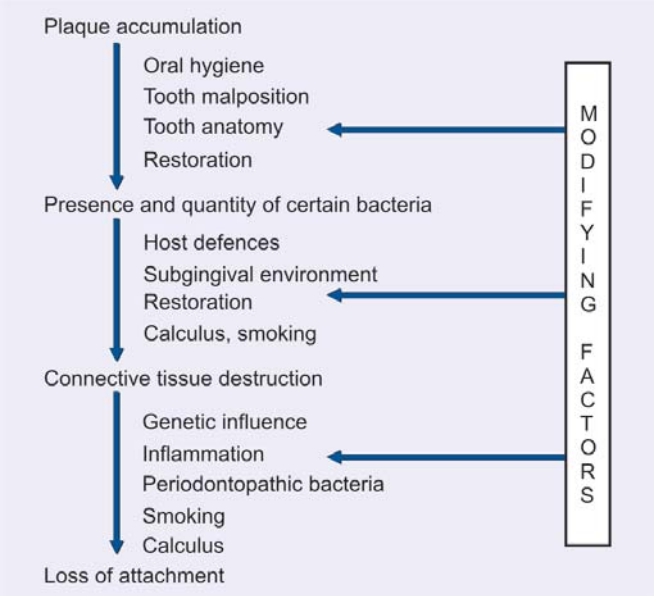

Fig. 25.4: Local factors in chronic periodontitis

b. *Systemic factors*: The role of systemic factors in periodontal diseases can influence the host response and increase the rate of progression of periodontal disease. Diabetes mostly type II, a non-insulin dependent diabetes mellitus is considered to be one of the most important systemic condition that can increase the extent and severity of periodontal disease (Fig. 25.5).

c. *Environmental or behavioral factors*: Smoking is one such factor that presently is receiving a lot of attention. It has been shown that when combined with plaque it induces periodontitis. It can result in greater attachment loss, bone loss, furcation involvement and deeper pockets. There is abundant evidence suggesting that emotional stress may also influence the extent and severity of chronic periodontitis.

d. *Genetic factors*: Genetic basis for periodontal disease is based on the recent studies that have demonstrated periodontal destruction among the family members and different generations within a family. Although no clear determinants have been described for chronic periodontitis, a genetic predisposition may be observed in aggressive periodontal breakdown in response to accumulation of plaque and calculus.

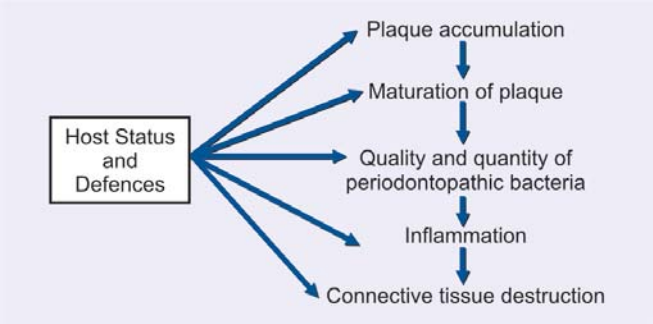

Fig. 25.5: Role of host status and defence mechanisms in influencing the progression of peridontal diseases

GENERAL CONCEPT FOR ETIOLOGY OF CHRONIC PERIODONTITIS

The key points regarding the etiology of chronic periodontitis are enumerated here:

KEYPOINTS

1. Chronic periodontitis is recently defined as an "infectious disease resulting in inflammation within the supporting tissues of the teeth, which is characterized by progressive attachment loss and bone loss."
2. Depending on the disease distribution it is classified as, localized periodontitis, i.e. less than 30 percent of the sites showing attachment loss and bone loss. It is generalized when more than 30 percent of sites demonstrate attachment loss and bone loss.
3. Depending on the disease severity it can be described as mild, moderate and severe forms.
4. Three models have been proposed to describe the rate of disease progression:
   a. Continuous Paradigm,
   b. Random Burst Theory,
   c. Asynchronous Multiple Burst Hypothesis.
5. Various risk factors for chronic periodontitits are:
   a. Local factors like plaque and plaque retentive factors.
   b. Systemic factors—systemic diseases like diabetes.
   c. Environmental factors like smoking.
   d. Genetic factors—no clear determinants.

KNOW MORE ...

Various Treatment Considerations in Chronic Periodontitis

I. *Non-surgical therapy*

- Initial therapy (scaling and root planing)
- Antimicrobial therapy—as an adjunct to routine periodontal therapy
- Instructions, reinforcement, evaluation of plaque control records
- Removal of all the factors contributing to plaque accumulation, e.g. correction of ill-fitting appliances, overcontoured crowns, overhanging restorations, etc.

II. *Surgical therapy*

A variety of surgical procedures could be implemented:

1. Periodontal flap surgery
2. Pocket elimination procedures
3. Regenerative therapy including bone grafts and barrier membranes.

REVIEW QUESTIONS

1. Describe the types, etiology and clinical features of chronic periodontitis.
2. What are risk factors for chronic periodontitis?

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26 Chapter

Aggressive Periodontitis

INTRODUCTION

Aggressive periodontitis is characterized by the rapid loss of attachment and bone loss occurring in an otherwise clinically healthy patient with the amount of microbial deposits inconsistent with disease severity and familial aggregation of diseased individuals. Aggressive periodontitis was formerly classified as early onset periodontitis, i.e. localized juvenile periodontitis (LJP) has been changed to localized aggressive periodontitis; generalized aggressive periodontitis was previously classified as generalized juvenile periodontitis (GJP) and rapidly progressive periodontitis (RPP).

LOCALIZED AGGRESSIVE PERIODONTITIS/ LOCALIZED JUVENILE PERIODONTITIS

Historical Background

- In 1923, Gottlieb reported a case as diffuse atrophy of alveolar bone characterized by the loss of collagen fibers in the periodontal ligament and loss of alveolar bone.
- In 1928, Gottlieb attributed this condition to the inhibition of cementum formation and termed the disease as cementopathia.
- In 1938, Wannenmacher described incisor, first molar involvement and called the disease as periodontitis

marginalis progressiva. Unlike others, he considered this as an inflammatory disease.

- In 1940, Thoma and Goldman used the term paradontosis and reported that the initial abnormality was located in the alveolar bone rather than in the cementum.
- In 1942, Orban and Weinmann introduced the term periodontosis and on the basis of autopsy case, described three stages in the development of the disease.

Stage 1: Involves the degeneration of principle fibers of the periodontal ligament, which induces cessation of cementum formation and resorption of alveolar bone. In this stage, tooth migration occurs without detectable inflammatory involvement.

Stage 2: The lack of periodontal fibers results in the rapid proliferation of the junctional epithelium along the root and earliest signs of inflammation appear.

Stage 3: It is characterized by progressive inflammation and the development of deep, infrabony periodontal pockets.

Most of these studies considered 'periodontosis' as a degenerative disease caused by unknown systemic factors. Other investigations denied the existence of a degenerative type of periodontal disease and attributed the changes observed to trauma from occlusion.

In 1996, the World Workshop concluded that periodontosis as a degenerative entity was unsubstantiated and the term should be eliminated from periodontal nomenclature.

The term juvenile periodontitis was introduced by Chaput and colleagues in 1967 and by Butler in 1969. In 1971, Baer defined it as 'a disease of 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. The amount of destruction is not commensurate with the amounts of local irritants'.

A more recent definition by Genco et al in 1986 describes localized juvenile periodontitis as a disease occurring in otherwise healthy individuals under the age of 30 years with destructive periodontitis localized to the first permanent molars and incisors not involving more than two other teeth.

Generalized juvenile periodontitis is defined as destructive periodontitis in individuals under the age of 30 years affecting more than fourteen teeth, i.e. generalized to an arch or an entire dentition.

Clinical Features

Age and Sex Distribution

Affects both the sexes and is seen mostly between puberty and 20 years of age. Some studies show predilection to female patients.

Distribution of Lesions

Three areas of localization of bone loss have been described:

1. First molar and/or incisors.
2. First molar and/or incisors + additional teeth (not exceeding 14 teeth).
3. Generalized involvement.

For localized juvenile periodontitis, classic distribution is in the first molars and incisors with least destruction in the cuspid, premolar area.

Limitations of destruction to certain teeth could be for the following reasons:

- Production of opsonizing antibodies against *A. actinomycetemcomitans* called *burn-out* phenomenon.
- Bacteria antagonistic to *A. actinomycetemcomitans* may develop, thereby decreasing the number of colonization sites.
- *A. actinomycetemcomitans* may loose its leukotoxin producing ability for unknown reasons.
- Localization of the lesions could also be due to the defect in cementum formation (hypoplastic/aplastic cementum).

Clinical Findings (Figs 26.1 and 26.2)

1. The most striking feature is lack of clinical inflammation despite the presence of deep periodontal pockets.
2. There is a small amount of plaque, which forms a thin film on the tooth and rarely mineralizes to become calculus.

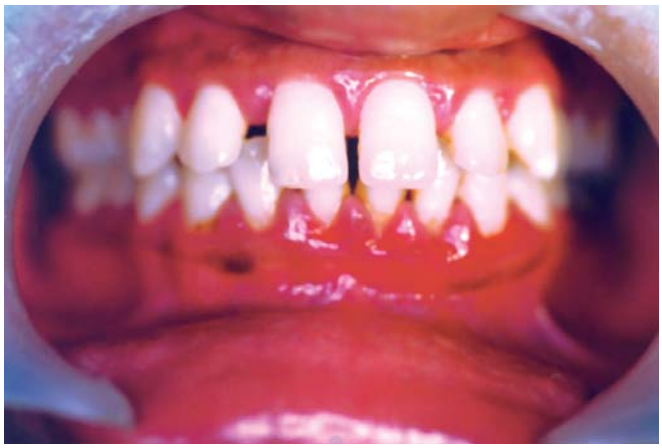

Fig. 26.1: Generalized aggressive periodontitis in a 30-year-old patient

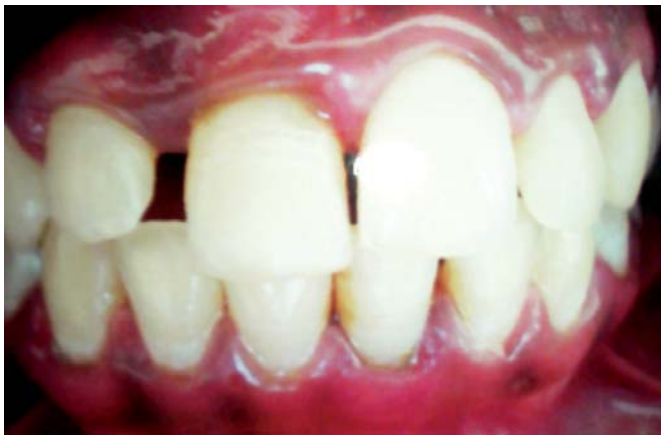

Fig. 26.2: Clinical appearance of localized aggressive periodontitis

3. Most common initial symptoms are mobility and migration of first molars and incisors. Classically, a distolabial migration of the maxillary incisors with diastema formation occurs, lower incisors rarely migrate compared to upper incisors, all changes followed by sequelae of migration are seen.
4. As the disease progresses, other symptoms like root surface sensitivity, deep dull radiating pain, periodontal abscess formation and regional lymph node enlargement may occur.

Radiographic Findings (Figs 26.3 and 26.4)

- Vertical or angular bone loss around the first molars and incisors in an otherwise healthy teenagers is a diagnostic sign of classic juvenile periodontitis. The pattern appears to be, "Arc-shaped loss of alveolar bone extending from distal surface of 2nd premolar to mesial surface of 2nd molar."
- Frequently, bilaterally symmetrical patterns of bone loss occurs, called as "mirror image pattern".

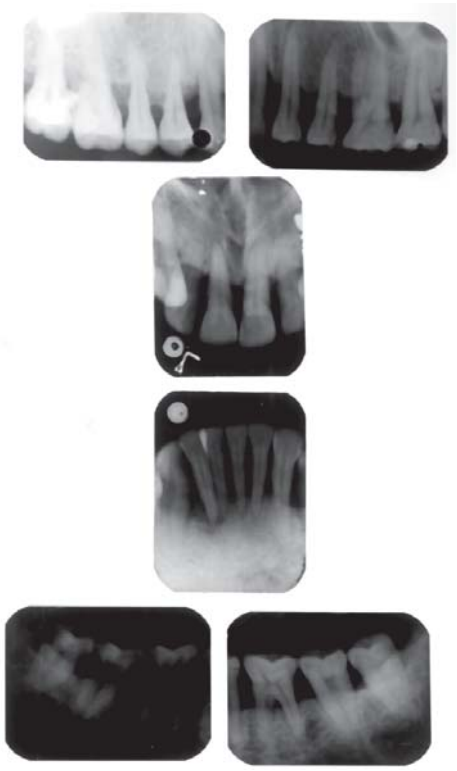

Fig. 26.3: Radiograph showing progressive bone loss

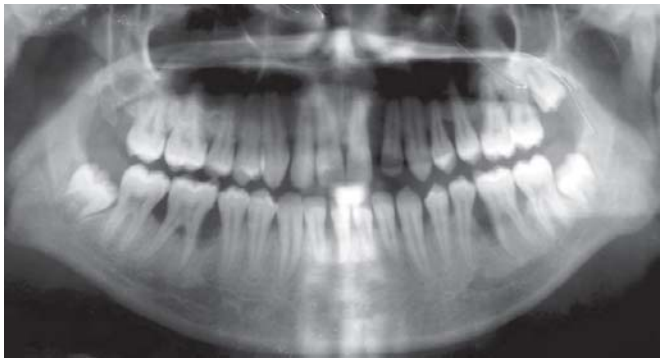

Fig. 26.4: Radiographic appearance of localized aggressive periodontitis

Pathogenesis of aggressive periodontitis is due to an interplay of several factors, these include the specific microbiology of subgingival plaque, defects in cementum, hereditary factors, impaired PMNs function and disorders of the immune system.

Histopathology/Microscopic Features

These are the same as those seen during pocket formation.

- Like ulcerated pocket epithelium.
- Accumulation of various inflammatory cells in the connective tissue mainly leukocytes, plasma cells and small number of lymphocytes and macrophages.
- Electron microscopic studies of juvenile periodontitis revealed bacterial invasion of connective tissue that reaches the bone surface.
- The flora involves *A. actinomycetemcomitans*, *Capnocytophaga sputigena* and others.

Bacteriology

Two types of bacteria are considered to be pathogens in localized aggressive periodontitis— *A. actinomycetemcomitans* and *Capnocytophaga*. *A. actinomycetemcomitans* is a short, facultatively anaerobic, non-motile Gram-negative rod.

Virulence factors associated with *A. actinomycetemcomitans* are:

Factors	*Significance*
Leukotoxin	Destroys polymorphonuclear leukocytes and macrophages.
Endotoxin	Activates host cells to secrete inflammatory mediators (prostaglandins, interleukin's 1 and 3, tumor necrosis factor-α).
Bacteriocin	May inhibit IgG and IgM production
Collagenase	Causes degradation of collagen
Chemotactic inhibition factors	May inhibit neutrophil chemotaxis

Immunology

Immune defects that have been implicated in the pathogenesis of localized aggressive periodontitis are functional defects of polymorphonuclear leukocytes/monocytes, thereby it impairs the chemotactic attraction of PMNLs (polymorphonuclear leukocytes) to the site of infection.

Treatment

Prognosis is no more considered as poor for patients with aggressive periodontitis. The following treatment has been tried in the past with varying results:

1. *Extraction*: Extraction of involved teeth especially first molars results in uneventful healing.

   Transplantation of developing third molars into the sockets of previously extracted 1st molars has been tried but with limited success.
2. *Standard periodontal therapy*: Includes scaling, root planing, curettage, flap surgery with/without bone grafts, root amputation, hemisection, occlusal adjustment and strict plaque control has been tried.

   However, response is unpredictable and frequent maintenance visits are a must.
3. *Antibiotic therapy*: Several authors reported successful results using antibiotics as adjuncts to standard therapy.
   - Genco and coworkers reported scaling and root planing and tetracycline 250 mg qid for 14 days every 8 weeks.
   - Several other investigations have also noticed excellent bone fill in cases of localized juvenile periodontitis treated with tetracycline, flap surgery and placement of grafts.

Current Approach to Therapy

- In almost all cases, systemic tetracycline hydrochloride 250 mg qid for atleast a week should be given in conjunction with local mechanical therapy. If surgery is indicated, systemic antibiotics are advised with patient instructed to begin taking the antibiotic approximately 1 hour before surgery.
- Doxycycline 100 mg/day may also be used.
- Chlorhexidine rinses should be prescribed.
- In refractory cases, tetracycline resistant *Actinobacillus* species have been suspected. In such cases, a combination of amoxicillin and metronidazole has been suggested.

GENERALIZED AGGRESSIVE PERIODONTITIS

Previously classified as Generalized Juvenile Periodontitis (GJP) and Rapidly Progressive Periodontitis (RPP).

Generalized aggressive periodontitis is usually characterized by 'generalized interproximal attachment loss affecting atleast three permanent teeth other than first molars

and incisors.' Patients with generalized aggressive form may exhibit minimal amounts of microbial plaque associated with the affected teeth, i.e. quantitatively, the amount of plaque seems to be inconsistent with the amount of periodontal destruction; qualitatively, most pathogenic organisms may be associated, e.g. *Porphyromonas gingivalis*, *A. actinomycetemcomitans*, and *Bacteroids forsythus*.

Clinical Characteristics

a. *Age and sex distribution*: It affects persons between puberty and 35 years (but may be older). No sex discrimination is seen.
b. *Distribution of lesion*: No specific pattern is observed, all or most of the teeth are affected.
c. *Two types of gingival responses*, may be seen in generalized aggressive periodontitis. One is severe, acutely inflamed tissue which is often proliferating, ulcerated and fiery red, spontaneous bleeding and suppuration are commonly seen. In the other cases, the gingival tissue may appear pink and free of inflammation but deep pockets can be demonstrated by probing.
d. Some of the patients may have systemic manifestations such as weight loss, mental depression and general malaise.

Radiographic Findings

No definite pattern of distribution occurs but the radiographic picture can range from severe bone loss associated with the minimal number of teeth, to advanced bone loss affecting the majority of teeth in the dentition.

Risk Factors for Aggressive Forms of Periodontitis

Microbiologic Factors

A. actinomycetemcomitans has been implicated as the primary pathogen associated with this disease. Microscopically, the lesions of localized aggressive periodontitis have revealed bacterial invasion of connective tissue that reaches the bone surface. These invading bacteria have been identified as *A. actinomycetemcomitans*, *Capnocytophaga sputigena*, *Mycoplasma* sub-species and *Spirochetes*.

Immunologic Factors

Some of the immune defects that have been implicated in the pathogenesis of localized aggressive periodontitis are:

a. Approximately 75 percent of patients with localized aggressive periodontitis (LAP) have dysfunctional neutrophils, which are seen as decreased in the chemotactic response to several chemotactic agents, including the complement component C5a, N-formyl-methionyl leucylphenylalanine (FMLP) and leukotriene B4. The defect is also associated with a 40 percent deficiency in glycoprotein, GP110, on the neutrophil surface.
b. Patients with LAP demonstrate a strong antibody response to *A. actinomycetemcomitans* which explains the limitation of the infection. In LAP the dominant serum antibody is IgG2 type which is specific to antigens of *A. actinomycetemcomitans*.
c. In generalized form of aggressive periodontitis diverse microbial patterns including organisms associated with chronic periodontitis have been implicated. Host response is often characterized by defects in either neutrophils or monocytes.

Genetic Factors

Some of the above mentioned immunologic defects seen in aggressive periodontitis may have a genetic basis, i.e. familial clustering of neutrophil abnormalities may be seen. It has been suggested by some authors that, a major gene plays a role in aggressive periodontal disease, which could be transmitted through an autosomal dominant mode of inheritance.

Environmental Factors

Smoking is one of the factors that can influence the extent of destruction seen in young patients. Especially, smokers with generalized aggressive periodontitis exhibit more

number of teeth affected by loss of clinical attachment than non-smokers with generalized aggressive periodontitis.

KEYPOINTS

Juvenile Periodontitis

It is defined as "a disease of 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. The amount of destruction is not commensurate with the amount of local irritants.

1. Previously classified as generalized juvenile periodontitis (GJP) and rapidly progressive periodontitis (RPP).
2. Usually affects individuals under the age of 30, but older patients may also be affected.
3. They usually produce a poor antibody response to the pathogens present.
4. Two types of gingival responses may be seen, one is a severe form, which is characterized by acute inflammatory changes in the gingival tissue. Other cases, the gingival tissue may appear pink, free of inflammation but in the presence of deep pockets.
5. There is no specific pattern for distribution of lesions, radiographically again no definite pattern of distribution seen.

KNOW MORE ...

Treatment of Aggressive Periodontitis

It also includes full mouth disinfection which has been proposed by Quirynen et al. Since it was observed that *A. actinomycetemcomitans* has the ability to translocate from one person to another and from site to site, full mouth disinfection was implemented. It includes the following steps:

a. Full mouth scaling and root planing (in 2 sessions within 24 hours).
b. Brushing the dorsum of the tongue with an antimicrobial agent (1% chlorhexidine gel) for one minute.
c. Mouthrinsing with antimicrobial agents.
d. Home irrigation systems.

Other Treatment Options

- Local drug delivery system.
- Host modulation and photodynamic therapy.

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27 Chapter

Necrotizing Ulcerative Periodontitis, Refractory Periodontitis and Periodontitis as a Manifestation of Systemic Disease

- NECROTIZING ULCERATIVE PERIODONTITIS
  - Non-AIDS type
  - AIDS associated
- REFRACTORY PERIODONTITIS
  - Etiology
  - Clinical Features
  - Treatment
- PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASE
  - Papillon-Lefévre Syndrome
  - Chédiak-Higashi Syndrome
  - Down's Syndrome
  - Hypophosphatasia
  - Neutropenia
  - Leukocyte Adhesion Deficiency

NECROTIZING ULCERATIVE PERIODONTITIS (NUP)

Necrotizing ulcerative periodontitis occurs as a result of extension of necrotizing ulcerative gingivitis into the periodontal structures, leading to loss of attachment and bone loss. There are two types of necrotizing ulcerative periodontitis described, based on its relationship to acquired immunodeficiency syndrome (AIDS): Non-AIDS type necrotizing ulcerative periodontitis and AIDS-associated necrotizing ulcerative periodontitis.

Non-AIDS Type Necrotizing Ulcerative Periodontitis

Clinical Features

Since necrotizing ulcerative periodontitis occurs after repeated attacks of necrotizing ulcerative gingivitis (NUG), all the characteristic clinical features of necrotizing ulcerative gingivitis are seen, i.e.

i. Ulceration and necrosis of gingival margin, which gets covered by a pseudomembranous slough.
ii. The ulcerated margins are surrounded by an erythematous halo.
iii. The lesions are extremely painful and bleed spontaneously.
iv. Localized lymphadenopathy, fever and malaise. These lesions, especially in long-standing cases, can extend to the deeper periodontal structures resulting in deep, crater-like osseous lesions especially in interdental areas. Such cases are identified as necrotizing ulcerative periodontitis (NUP), most striking feature of this condition is absence of deep conventional pockets associated with deep interdental osseous craters. This is because the necrotizing and ulcerative

properties of the gingival lesion destroys the marginal epithelium, resulting in total destruction of the marginal tissue leading to recession.

AIDS-Associated Ulcerative Periodontitis

(Discussed in detail elsewhere in this book)

Gingival and periodontal lesions of HIV positive patients appear to have similar findings that are seen in non-AIDS-associated necrotizing ulcerative periodontitis patients, in addition, they may exhibit certain complications such as:

1. Large areas of soft tissue necrosis with exposure of bone and sequestration of bone.
2. Sometimes these lesions may extend onto the buccal vestibule or the palate and become necrotizing stomatitis.

REFRACTORY PERIODONTITIS

According to American Academy of Periodontology, refractory periodontitis has been defined as "those cases which do not respond to any treatment provided, whatever the thoroughness or frequency". It should be differentiated from the cases of recurrent periodontitis, in which, after remission of the disease, recurrence follows due to inadequate plaque control either by the patient or clinician. Hence recurrent periodontitis, which is recurrence of the disease due to incomplete treatment should be differentiated from refractory cases whose reasons for not responding to adequate treatment, in certain cases, is still not clearly understood (Table 27.1).

Etiology

Risk Factors Responsible for Refractory Cases

a. Abnormal host response.
b. Resistant strains of pathogenic periodontal microflora.
c. Failure to eliminate plaque-retentive factors, such as furcation involvement, irregular root surface, palato-gingival groove, etc. which may in turn interfere with complete plaque removal.
d. On the other hand, smoking and systemic diseases may result in generalized lesions, which may not respond favorably to treatment.

Specific microorganisms have been identified in lesions of refractory periodontitis. Haffajee et al. (1988) reported three major microbial complexes in refractory periodontitis cases.

1. *B. forsythus, F. nucleatum* and *C. rectus.*
2. *S. intermedius, P. gingivalis* and *P. micros.*
3. *S. intermedius* and *F. nucleatum.*

Clinical Features

In the classification proposed by the American Academy of Periodontology (1999), the term refractory periodontitis has been removed as a single entity due to the diversity of clinical conditions and treatments under which periodontal therapy fails to arrest the progression of periodontitis. Therefore, the participants of the workshop have concluded that, rather than a single disease entity, the 'refractory'

Table 27.1: Distinction between recurrent and refractory periodontitis

Disease	*Recurrent periodontitis*	*Refractory periodontitis*
Definition	Sites are successfully treated but disease returns, may refer to site/patients.	Sites do not respond to conventional therapy; usually refers to patients but may refer to sites.
Phase of therapy	May be because of inadequate therapy during maintenance/no maintenance.	May be because of inadequate therapy during active treatment/other factors.
Etiology	May be because of re-infection, with microbes that were suppressed but not eliminated. Re-infection with eliminated organisms or new bacteria.	May be because of infection with tissue invasive microbes that cannot be eliminated with conventional therapy or because of immunoincompetence.
Immune system	Immunocompetent.	May not be immunocompetent.
Antibiotic therapy	Not usually needed.	Usually needed.

designation could be applied to all forms of periodontitis, e.g. refractory chronic periodontitis, refractory aggressive periodontitis and others.

Hence, refractory periodontitis in no way differs from other forms of periodontitis. Magnusson et al. (1991) noticed no changes in the amount of plaque in relation to the sites that have been treated, however, the sites that showed attachment loss had also showed persistent bleeding on probing. The refractory cases can be identified either by presenting new areas of attachment loss or progressive attachment loss in already treated sites.

Treatment

Antimicrobial therapy along with mechanical debridement has been found to be effective in reducing the microbial population at the refractory sites.

Antibiotic therapy aims to reinforce mechanical periodontal therapy and also helps the host defence system by killing subgingival pathogens that remain after conventional mechanical periodontal therapy. Many antibiotics have been used to treat the refractory sites (Table 27.2).

In addition to this, intrasulcular irrigation with 10 percent povidone-iodine solution and chlorhexidine solution have also been used successfully.

In cases of localized lesions, local drug delivery system has been tried successfully. The advantages of this are smaller doses and minimal side effects. These local therapies are available in the form of gels, fibers or chips.

Table 27.2: Drugs used to treat refractory sites

Drug	*Dosage*
Tetracycline hydrochloride	250 mg of qid
Amoxicillin	250 mg + 125 mg thrice daily
Clavulanate potassium (Augmentin)^{R}	for 2 weeks
Clindamycin hydrochloride	150 mg of qid for 1 week
Combination of the above drugs, i.e. metronidazole/ amoxicillin, metronidazole doxycycline are also used.	

Another approach in treating refractory periodontitis is through modulation of host by sub-antimicrobial doses of doxycycline or non-steroidal anti-inflammatory drugs (NSAIDs) in conjunction with conventional therapy. The sub-antimicrobial or low dose doxycycline helps to prevent the periodontal destruction by controlling the production of collagen and gelatinase, whereas flurbiprofen, indomethacin and naproxen may reduce the production of inflammatory mediators during chronic periodontal disease.

PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASE

Severe periodontitis has been observed in patients who exhibit either defective numbers of neutrophils or defective neutrophil function. Some of the conditions associated with defective neutrophils are:

a. Papillon-Lefévre syndrome.
b. Chédiak-Higashi syndrome.
c. Down's syndrome.
d. Hypophosphatasia.
e. Neutropenia.
f. Leukocyte adhesion deficiency.

Papillon-Lefévre Syndrome (Fig. 27.1)

1. This is characterized by hyperkeratotic skin lesions and severe destruction of the periodontium.
2. These changes may appear before the age of 4 years.
3. Skin lesions are—hyperkeratosis of localized areas on palms, soles, knees and elbows.
4. Periodontal involvement is early inflammatory changes that lead to bone loss and exfoliation of teeth. Primary teeth are lost by 5 or 6 years of age. The permanent dentition erupts normally but within few years the permanent teeth are also lost.

Chédiak-Higashi Syndrome (CH Syndrome)

This is a rare syndrome characterized by recurrent bacterial infections. It exhibits oral ulceration and rapidly destructive periodontitis. Chédiak-Higashi syndrome is a hereditary disease with defects in both neutrophils and monocytes.

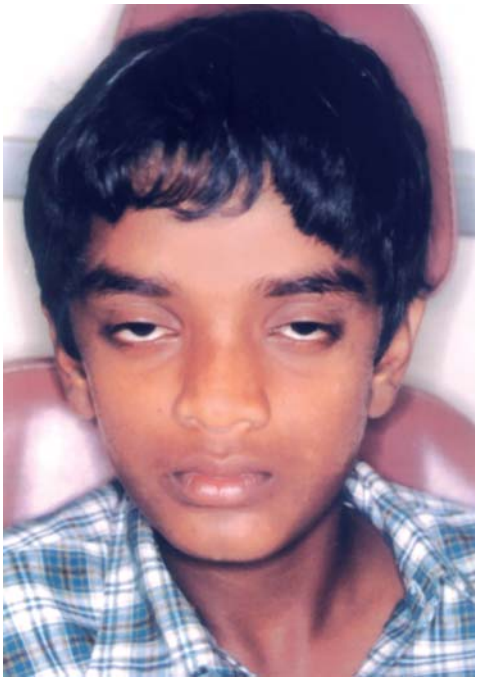

Fig. 27.1: A case of Papillon-Lefévre syndrome

Down's Syndrome (Mongolism, Trisomy 21)

This is a congenital disease caused by a chromosomal abnormality and is characterized by mental deficiency and growth retardation.

The oral findings include presence of plaque, calculus, and other local factors, like diastema, crowding of teeth, high frenal attachment and malocclusion.

Periodontitis in Down's syndrome include the formation of deep periodontal pocket associated with plaque accumulation and moderate gingivitis, usually generalized but more severe in the lower anterior region; marked recession is also seen in this region (may be due to high frenal attachment). Acute necrotizing lesions are also common (Fig. 27.2).

Two factors have been proposed to explain high prevalence and increased severity of periodontal destruction in Down's syndrome:

1. Reduced resistance to infections because of poor circulation (especially peripheral).
2. Defect in T-cell maturation and polymorphonuclear leukocyte chemotaxis.

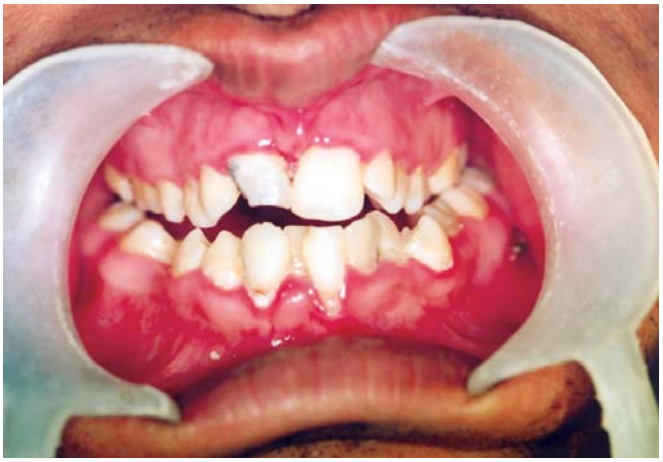

Fig. 27.2: Gingival changes associated with Down's syndrome

Hypophosphatasia

This is a rare familial skeletal disease characterized by rickets, poor cranial formation, premature loss of primary dentition particularly incisors. Patients have low level of serum alkaline phosphatase. Teeth are lost with no clinical evidence of gingival inflammation and show reduced cementum formation.

Neutropenia

It is a condition where the circulating neutrophils are reduced, i.e. less than 1,500 per ml. Destructive generalized periodontal lesions have been described in children with neutropenia. Neutropenia could be inherent or acquired. Certain drugs and infections can lead to reduction in number of neutrophils.

Leukocyte Adhesion Deficiency

These cases are rare, and begin during or immediately after eruption of the primary teeth. Extreme acute inflammation and proliferation of gingival tissues accompanied by rapid bone loss is found, profound defects in peripheral blood neutrophils and monocytes are seen. Hence they are absent in gingival tissues. Patients with LAD (Leukocyte adhesion deficiency) also have frequent respiratory tract infections and sometimes otitis media.

KEYPOINTS

1. When ulcerative gingivitis extends deeper into the periodontal structures, it results in loss of attachment and bone loss. This condition is termed as necrotizing ulcerative periodontitis (NUP).
2. It can be of two types, based on its relationship to acquired immunodeficiency syndrome (AIDS). Non-AIDS type NUP and AIDS-associated NUP.
3. A non-AIDS type occurs as a result of repeated attacks of NUG (Necrotizing ulcerative gingivitis).
4. AIDS-associated NUP (Necrotizing ulcerative periodontitis) shares most of the features of non-AIDS type, but with larger areas of soft tissue necrosis with exposure of bone and sequestration of bone fragments.
5. Refractory periodontitis is defined as, "those cases which do not respond to any treatment, provided, whatever the thoroughness or frequency." It should be differentiated from recurrent periodontitis cases, in which, after the remission of the disease, recurrence follows due to inadequate plaque control either by the patient or the clinician.
6. According to the classification proposed by AAP (American Academy of Periodontology, 1999) the term refractory periodontitis has been removed as a single entity.
7. Many systemic conditions associated with defective number or function of neutrophils exhibit severe periodontitis. Some of the conditions are, Papillon-Lefèvre syndrome, Chediak-Higashi syndrome, Down syndrome, hypophosphatasia, neutropenia and leukocyte adhesion deficiency.

KNOW MORE ...

Treatment of Necrotizing Ulcerative Periodontitis

- Local debridement of lesions with scaling and root planing
- Oral hygiene instructions
- The use of ultrasonic instrumentation with copious irrigation may be helpful in flushing of the deep lesions
- Locally applied antimicrobials, systemic antibiotics and analgesics should be considered to alleviate the infection and pain.

REVIEW QUESTION

1. Describe the features of necrotizing ulcerative periodontitis.

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28 Chapter

AIDS and the Periodontium

- HIV OPPORTUNISTIC INFECTIONS
- CLASSIFICATION OF PERIODONTAL DISEASES ASSOCIATED WITH HIV INFECTION
  - Linear Gingivitis
  - Necrotizing Ulcerative Gingivitis
  - Necrotizing Ulcerative Periodontitis
  - Necrotizing Stomatitis
  - CDC Surveillance Care Classification
- MOST COMMON ORAL AND PERIODONTAL MANIFESTATIONS OF HIV INFECTION
- MANAGEMENT

HIV OPPORTUNISTIC INFECTIONS

Most of the opportunistic infections seen in HIV-positive patients are caused by, protozoan, fungal, or viral pathogens. This is because the effective immune defence for these pathogens is the cell-mediated response which is impaired by HIV infection. Of the various types of oral lesions found in the HIV-positive population, the most destructive and problematic are those of bacterial origin. Bacterial infections seen in HIV-positive patients include diseases caused by encapsulated or enteric bacteria such as *Campylobacter*, *Klebsiella*, *Salmonella* and *Streptococcus*.

CLASSIFICATION OF PERIODONTAL DISEASES ASSOCIATED WITH HIV INFECTION

Four distinct disease types are seen,

1. *HIV-associated gingivitis (HIV-G):* A distinctive linear inflammation is seen around the gingival margin with possible punctate erythema extending throughout the width of the attached gingiva that may occur in the presence of excellent oral hygiene.
2. *HIV-associated periodontitis (HIV-P):* Characterized by rapid loss of attachment, connective tissue destruction and deep bone pain.
3. HIV-necrotizing gingivitis *(HIV-NG).*
4. *Necrotizing stomatitis (NS):* In which spontaneous sequestration of interdental bone along with extensive soft tissue necrosis occurs.

Since it was felt that the prefix 'HIV' was over-descriptive and caused potential, ethical and legal problems with confidentiality, the new classification has dropped the term HIV from individual disease titles.

- HIV-G has been changed to *linear gingivitis.*
- HIV necrotizing gingivitis has been changed to *necrotizing ulcerative gingivitis* (NUG).
- HIV-P has been changed to *necrotizing ulcerative periodontitis* (NUP).
- Necrotizing stomatitis.

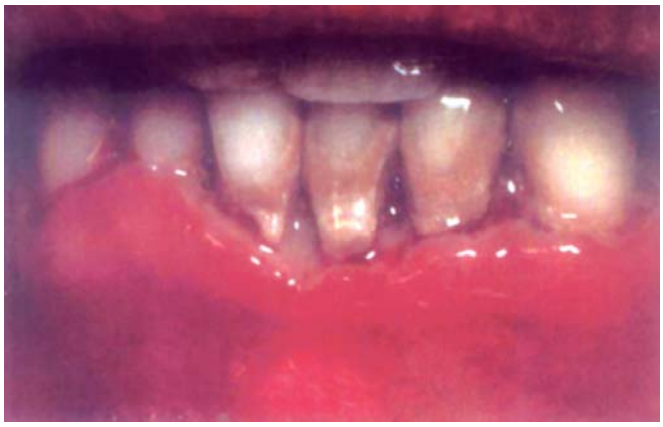

Fig. 28.1: A case of necrotizing ulcerative periodontitis

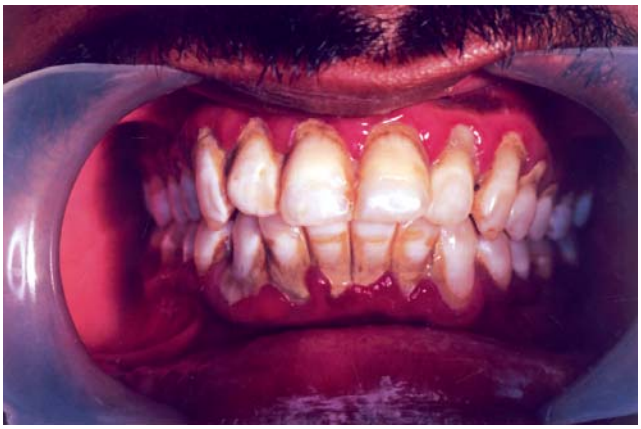

Fig. 28.2: Marginal gingival erythema and early necrosis of the interdental papilla

Linear Gingivitis

It is characterized by:

- Marginal linear erythema across the attached gingiva generally involving all the teeth
- Punctate lesions appear to coalesce giving the entire gingiva a bright-red appearance
- Spontaneous bleeding or bleeding on probing
- The amount of supragingival plaque is not proportional to the amount of erythema
- No ulceration, no loss of attachment is seen
- Does not respond to the removal of plaque by intensive scaling, root planing and plaque control measures.

Necrotizing Ulcerative Gingivitis (NUG)

- Sudden onset, bleeding on toothbrushing.
- Pain and characteristic halitosis.
- The gingiva appears fiery-red and swollen and yellow to grayish necrosis is observed on the tip of the interdental papilla and margins of the gingiva.
- Mostly anterior gingiva is affected and normally limited to the soft tissue of the periodontium.

Necrotizing Ulcerative Periodontitis (Figs 28.1 and 28.2)

- Severe pain, localized soft tissue necrosis, ulceration and interproximal cratering
- Not associated with deep pocket formation but instead there is a loss of crestal bone coinciding with soft tissue destruction
- Rapid horizontal bone loss in the absence of severe gingival inflammation has been reported
- Tooth mobility is a common feature
- Associated with severe immune suppression with CD4+ cell count below 200 cells/mm^3.

Oral Lesions Associated with HIV Infection

Group I	*Group II*	*Group III*
Oral lesions strongly associated with HIV infection 1. Candidiasis 2. Hairy leukoplakia 3. Non-Hodgkin's lymphoma 4. Kaposi's sarcoma 5. Periodontal diseases • LGE: Linear gingival erythema • NUG: Necrotizing ulcerative gingivitis • NUP: Necrotizing ulcerative periodontitis	Lesions less commonly associated with HIV infection 1. Salivary gland diseases 2. Melanotic hyperpigmentation 3. Viral infection 4. Bacterial infections 5. Necrotizing stomatitis	Lesions seen in HIV infection 1. Recurrent aphthous stomatitis 2. Osteomyelitis 3. Sinusitis 4. Fungal lesions other than candidiasis 5. Cytomegalovirus infection 6. Bacterial infections 7. Exacerbation of apical periodontitis

Necrotizing Stomatitis

- Extensive soft tissue and bony necrosis with sequestration.
- It resembles noma and cancrum oris and represents the most severe form of periodontal infection seen in association with HIV.

All of the above mentioned conditions may occur in isolation or in combination in any one patient. A common feature of these periodontal diseases which distinguishes them from conventional periodontal conditions are, a lack of response to the removal of plaque and to the patients maintenance of good oral hygiene.

CDC Surveillance Care Classification (1993)

AIDS patients have also been grouped as follows:
Category A: Includes patients with acute symptoms or asymptomatic diseases, along with individuals with persistent generalized lymphadenopathy, with or without malaise, fatigue or low grade fever.
Category B: Patients have symptomatic conditions such as oropharyngeal or vulvovaginal candidiasis, herpes zoster, oral hairy leukoplakia, idiopathic thrombocytopenia or constitutional symptoms of fever, diarrhea and weight loss.
Category C: Are those with outright AIDS as manifested by life-threatening conditions identified by CD4+T4 lymphocyte levels of less than 200 cells/mm^3.

MOST COMMON ORAL AND PERIODONTAL MANIFESTATIONS OF HIV INFECTION

1. *Oral hairy leukoplakia*
   - Found on lateral borders of tongue
   - Caused by Human Papillomavirus
   - Keratotic, asymptomatic area with vertical striations giving a corrugated appearance
   - When dried appears hairy and does not rub off
2. *Oral candidiasis manifested as*
   - Pseudomembranous (thrush) candidiasis
   - Erythematous candidiasis
   - The hyperplastic candidiasis
   - Angular cheilitis
3. *Kaposi's sarcoma*: Multifocal, vascular neoplasm manifest as nodules, papules or non-elevated macules that are usually brown, blue or purple in color.
4. *Bacillary angiomatosis*: It is an infectious vascular, proliferative disease. It appears as red, purple or blue edematous soft tissue lesions that may cause destruction of periodontal ligament and bone.
5. *Oral hyperpigmentation*
6. *Atypical ulcers and delayed healing.*

MANAGEMENT

Step I: Thorough medical and dental history (should be kept confidentially).
Step II: Periodontal therapy

TREATMENT OF LINEAR GINGIVAL ERYTHEMA AND NECROTIZING ULCERATIVE GINGIVITIS

- Medical history and appropriate medical consultation.
- Scaling of affected areas and oral hygiene instructions.
- Intrasulcular irrigation using 10 percent povidone iodine.
- 0.12 percent chlorhexidine mouth-rinse twice daily.
- Antifungal agents like nystatin oral suspension and clotrimazole.

Disadvantages of oral antifungal agents are, lack of patients compliance because of strong, sweet flavor and also high sucrose content in them preventing its use for long term (risk of rampant tooth decay).

Hence, systemic antifungal agents are recommended. Ketoconazole (may cause liver toxicity), fluconazole–200 mg tablets once and twice daily (most preferred).

- Follow-up one day and one week post-initial therapy.
- Recall every 4 weeks until periodontal condition is stable, then every 3 to 6 months.

Treatment of Necrotizing Ulcerative Periodontitis

- Medical history
- Scaling of affected areas under local anesthesia
- Remove necrotic bone and soft tissue
- Perform 10 percent povidone iodine irrigation

- Oral hygiene instructions
- 0.12 percent Chlorhexidine mouth-rinse
- Systemic analgesics
- Consider systemic antibiotic such as metronidazole
- Antifungal agents
- Follow-up (1 day to 4 weeks, 1 to 6 months)

Treatment of Necrotizing Stomatitis

Same steps followed + protect lesion with mouthguard if possible.

KEYPOINTS

1. *Four distinct types of periodontal diseases associated with HIV infection are:*
   a. HIV-associated gingivitis (HIV-G)
   b. HIV-associated periodontitis (HIV-P)
   c. Necrotizing stomatitis (NS)
   d. HIV-necrotizing gingivitis (HIV-NG)
2. *Oral lesions associated with HIV infection could be classified under*
   Group I: Oral lesions strongly associated with HIV infection.
   Group II: Lesions less commonly associated with HIV infection.
   Group III: Lesions seen in HIV infection.
3. *Most common oral and periodontal manifestations of HIV infections are*:
   a. Oral hairy leukoplakia
   b. Oral candidiasis
   c. Kaposi's sarcoma
   d. Bacillary angiomatosis
   e. Oral hyperpigmentation
   f. Atypical ulcers and delayed wound healing

KNOW MORE ...

Acquired Immunodeficiency Syndrome (AIDS)
It is a disease due to infection with the human immunodeficiency virus.

Role of Antiretroviral Drugs in the Management of HIV/AIDS
The agents that are developed so far act at different stages of the life cycle of HIV:
a. They can block the binding of virus to the target cell.
b. They can block the viral RNA cleavage.
c. One that inhibits enzyme reverse transcriptase.

Testing for HIV Antibodies
a. Screening test → ELISA (Enzyme Linked Immunosorbent Assay)
b. Confirmatory test → Western blot assay
→ Polymerase chain reaction.

REVIEW QUESTION

1. What are the various periodontal manifestations of HIV infection?

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29 Chapter

Diagnosis of Periodontal Diseases

PERIODONTAL DIAGNOSIS

Proper periodontal diagnosis is essential for successful treatment. Hence, diagnosis should first determine whether disease is present; then identify its type, extent, distribution and severity and finally provide an understanding of the underlying pathologic processes and their cause. *Hence, diagnosis may be defined as, identifying disease from an evaluation of the history, signs and symptoms, laboratory tests and procedures*.

PRINCIPLES OF DIAGNOSIS

This includes:

Sensitivity—refers to the ability of a test or observation to detect the disease whenever it is present.

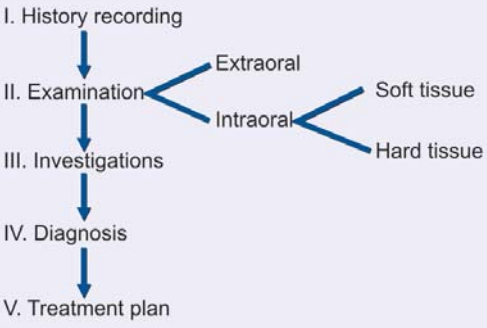
Specificity—refers to the ability of a test or observation to clearly differentiate one disease from another.

Predictive value—refers to the probability of the test results.

CLINICAL DIAGNOSIS

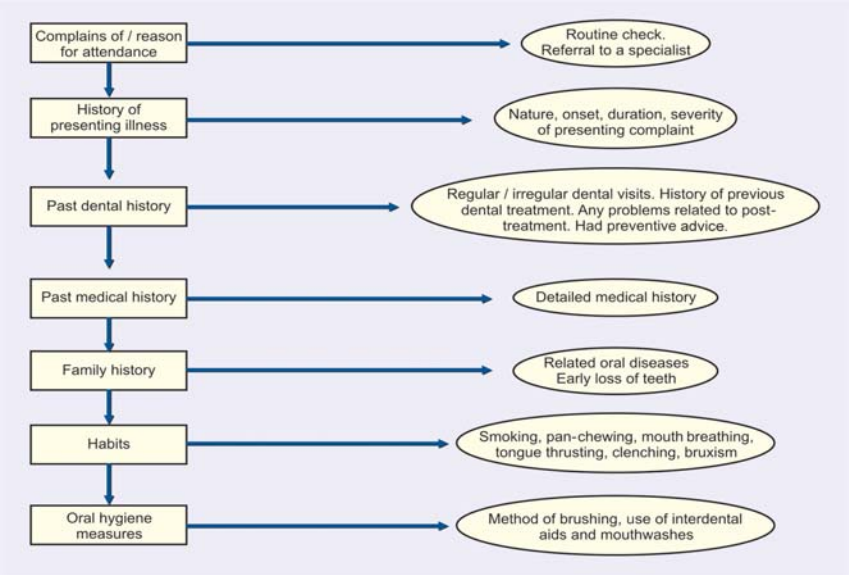
Periodontal diagnosis is mainly based on careful analysis of the case history and recording clinical findings with the help of various aids. One must remember that the importance should be given to the patient who has the disease and not simply the disease itself. The clinical findings are recorded in a systematic manner, when pieced together should provide a meaningful explanation.

Key Stages of Periodontal Diagnosis

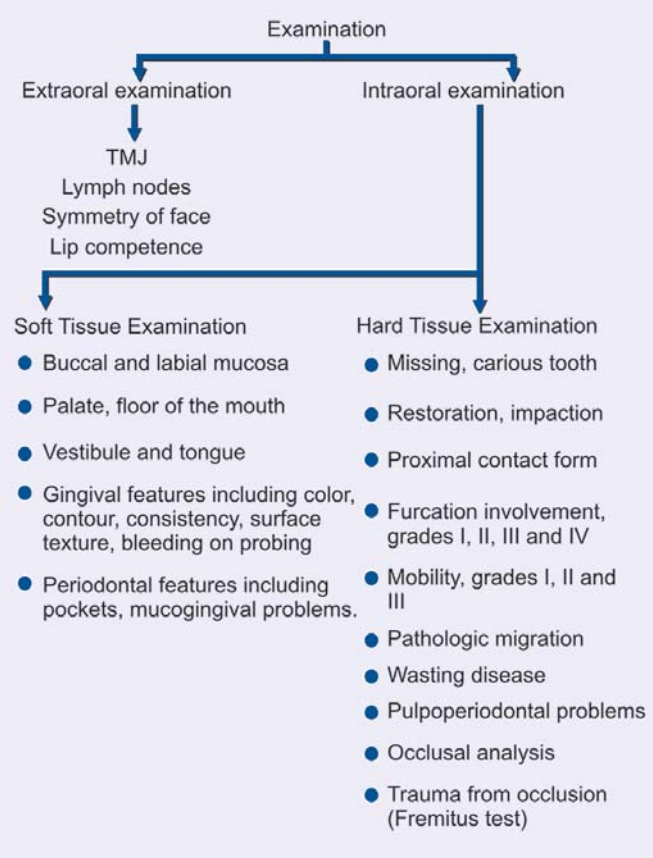


Clinical Diagnosis with Detailed Case History Recording

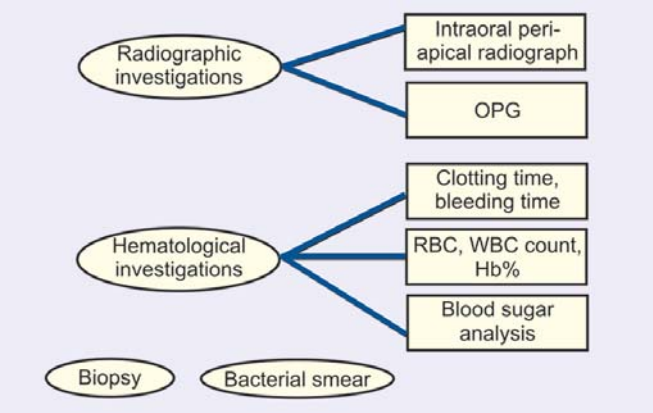
History Recording



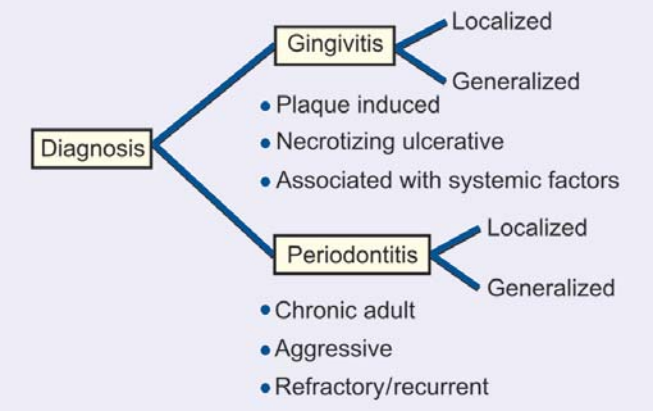
Examination

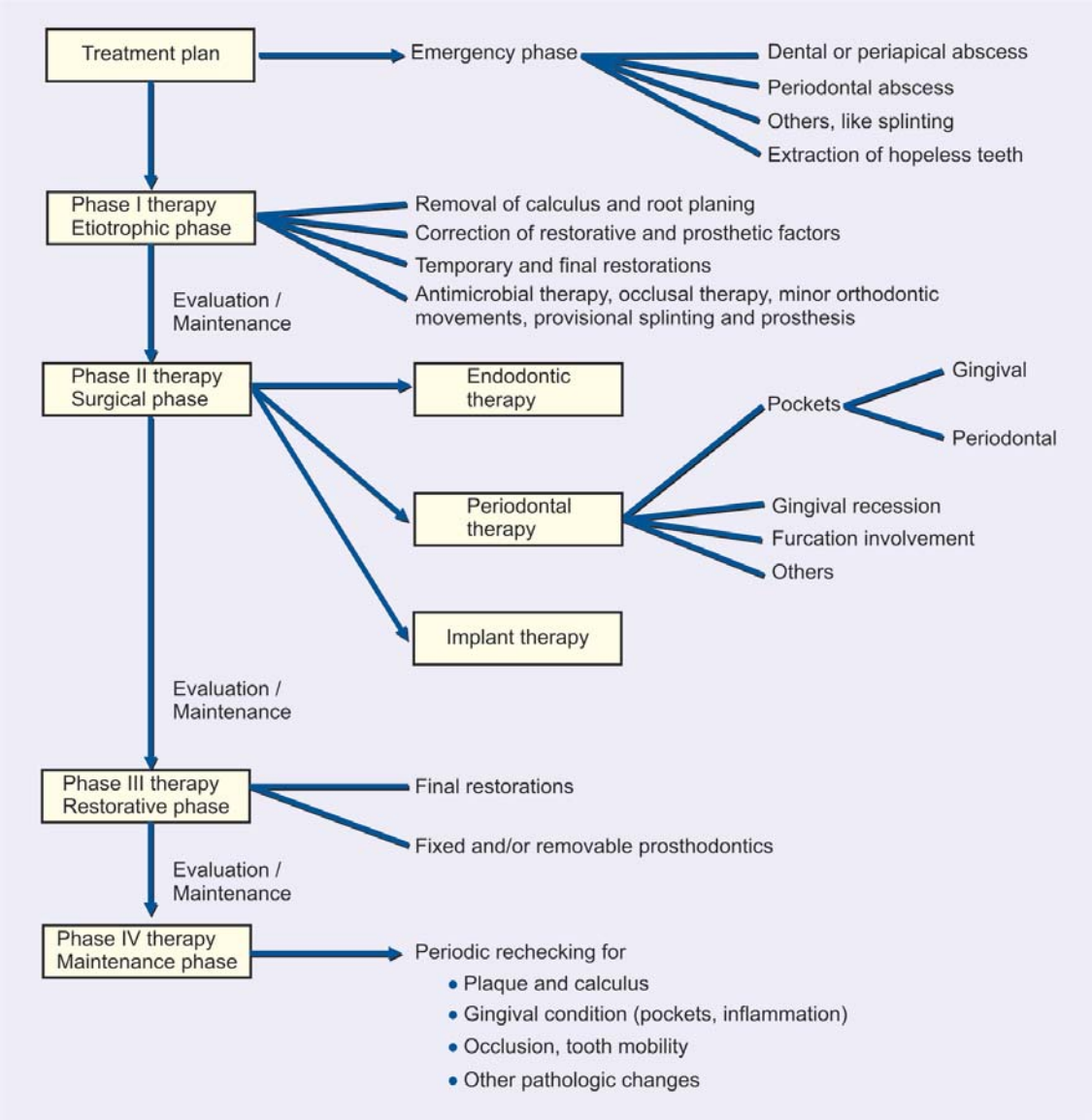


Investigations



Diagnosis





CASE HISTORY PROFORMA

Name: OP No:
Sex: Age:
Address: Occupation:
Date:

Chief Complaint

History of present illness:
Past dental history:
Medical history:
Family history:

Drug allergy:
Habits:

Clenching	[ ]	Tonguethrusting	[ ]
Mouthbreathing	[ ]	Pan-chewing	[ ]
Bruxism	[ ]	Cigarette/pipe-smoking	[ ]
Chronic biting of cheek/tongue	[ ]	Tobacco chewing	[ ]

Oral Hygiene Methods

1. Toothbrushing:
   Age of brush:

Type of brush:
Methods:
Horizontal [ ] Vertical [ ]
Circular [ ]
Frequency of brushing:
Estimate of time spent:

2. Dental floss [ ]
3. Toothpicks [ ]
4. Interproximal brushes [ ]
5. Mouthwashes [ ]
6. Others specify [ ]

Clinical Examination

Extraoral

Symmetry of face:
TMJ:
Tenderness:
Clicking:
Jaw deviation:
Lymph nodes:
Submental:
Submandibular:
Cervical:

Intraoral

Lip seal:
Halitosis:

Soft Tissue:
Buccal/labial mucosa:
Palate:
Floor the mouth:
Lips:
Vestibule:
Tongue:

Hard Tissue (Teeth):
Missing teeth:
Carious teeth:
Restored teeth:
Loss of proximal contact:
Crowding of teeth:
Wasting disease:
Attrition:
Abrasion:
Erosion:
Fillings/restorations
Tender on percussion
Pulp/periapical problem:
Pathologic migration

Occlusal Analysis:
Angles classification:
Overbite and over jet:
Openbite:
Premature contact:
Crossbite:
Plunger cusps:
Edge to edge bite:
Prematurities:

Gingival Status

	Upper right posterior *Upper left posterior*	*Upper anterior*
Color		
Contour		
Size		
Consistency		
Stippling		
Position		
Bleeding on probing		
Exudation		

	Lower right posterior *Lower left posterior*	*Lower anterior*
Color		
Contour		
Size		
Consistency		
Stippling		
Position		
Bleeding on probing		
Exudation		

Investigations

Radiological Investigations (IOPA/OPG)

Horizontal bone loss:
Vertical bone loss:
Furcation involvement:
Endodontic treatment/overhanging restorations:
Periapical pathology
Impacted/supernumerary/embedded teeth:
Caries:
Crown/root-ratio:

Special Investigations

Biopsy:
Bacterial smear:

Other Investigations

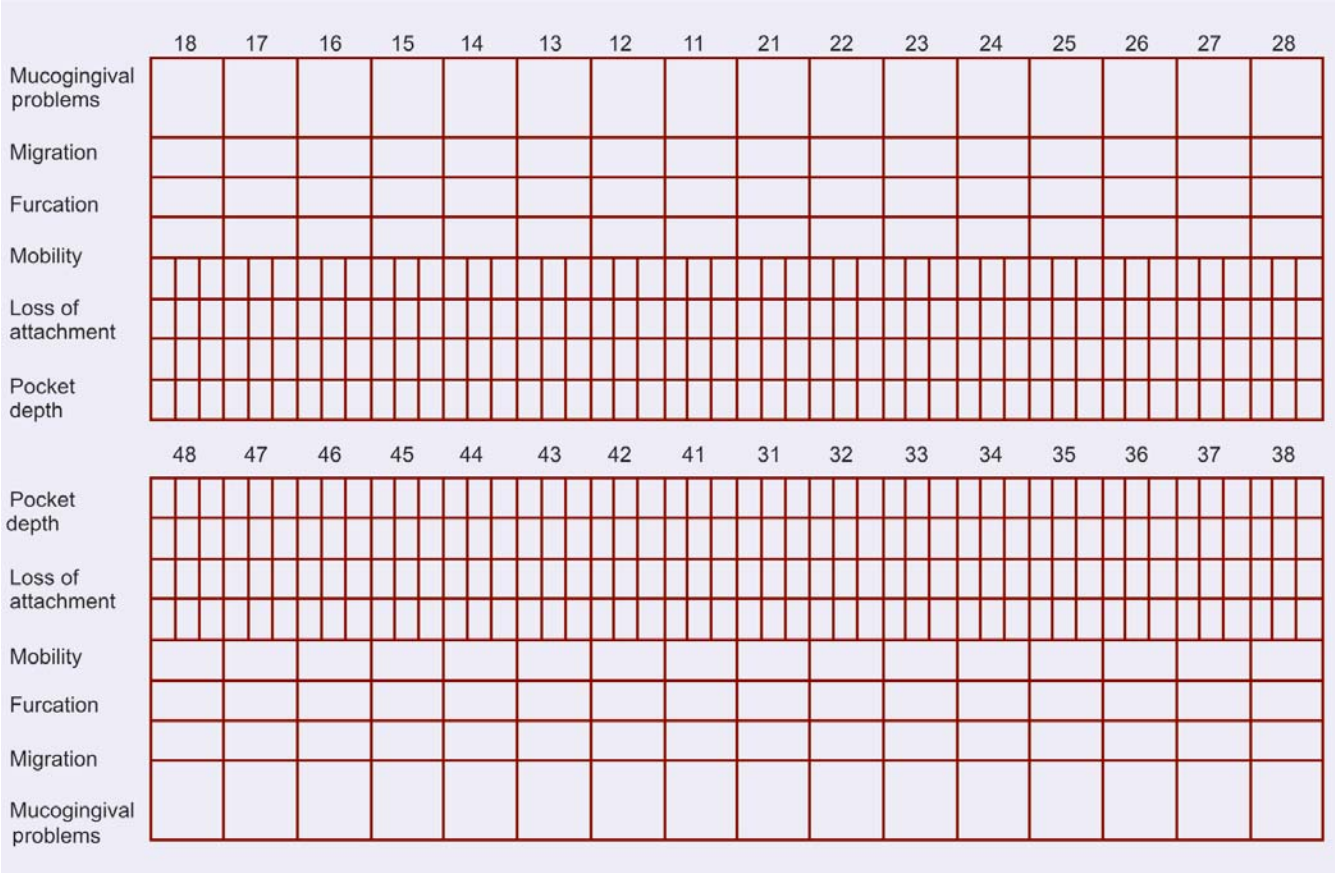
Diagnosis

Prognosis

Individual:
- Good [ ]
- Fair [ ]
- Poor [ ]

Overall:
- Good [ ]
- Fair [ ]
- Poor [ ]

Surgical Assessment



Treatment Plan

Preliminary Phase/Emergency Phase

Phase I therapy (Etiotropic phase)

Phase II therapy (Surgical phase)

Phase III therapy (Restorative phase)

Phase IV therapy (Maintenance phase)

KNOW MORE ...

Periodontal Examination

It includes, measurement of probing depths, clinical attachment loss, furcation involvement, mobility, trauma from occlusion and pathologic tooth migration (given in detail in Chapter No. 9).

Factors that can Influence the Accuracy of Probing

- Physical qualities of a probe
- Angulation of the probing
- Force applied during probing
- Inflammatory status of the tissue
- Availability of a reference point.

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30 Chapter

Determination of Prognosis

- DEFINITION, DIFFERENCES BETWEEN PROGNOSIS AND RISK
- DETERMINATION OF A PROGNOSIS
- FACTORS FOR DETERMINATION OF PROGNOSIS
  - Overall Clinical Factors
  - Systemic/Environmental Factors
  - Local Factors
- RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS
- RE-EVALUATION OF PROGNOSIS AFTER PHASE I THERAPY

DEFINITION OF PROGNOSIS

It is a prediction of the probable course, duration and outcome of a disease based on a general knowledge of the pathogenesis of the disease and the presence of risk factors for the disease.

It is established after the diagnosis is made and before the treatment plan is established. Prognosis is often confused with the term risk. The differences between prognosis and risk are given in Table 30.1.

Table 30.1: Differences between prognosis and risk

Prognosis	*Risk*
i. It is the prediction of the duration, course and the termination of disease and its response to treatment.	i. Deals with the likelihood that an individual will get a disease in a specified period.
ii. Prognostic factors are characteristics that predict the outcome of disease once the disease is present.	ii. Risk factors are those characteristics of an individual that put them at increased risk for getting a disease.

There are certain situations where risk factors and prognostic factors are similar, for example, patients with diabetes and smokers are more at risk for developing periodontal diseases, and once they acquire it, they are considered to have a poor prognosis.

DETERMINATION OF PROGNOSIS

It is based on the factors to be considered while determining the prognosis. Prognosis may be:

i. *Excellent prognosis*: No bone loss, excellent gingival condition, good patient cooperation, no systemic/ environmental factors.

ii. *Good prognosis*: One or more of the following: adequate remaining bone support, possibilities to control etiologic factors and establish a maintainable dentition, adequate patient co-operation, no systemic/ environmental factors or if present are well-controlled.

iii. *Fair prognosis*: One or more of the following: less than adequate remaining bone support, some tooth mobility,

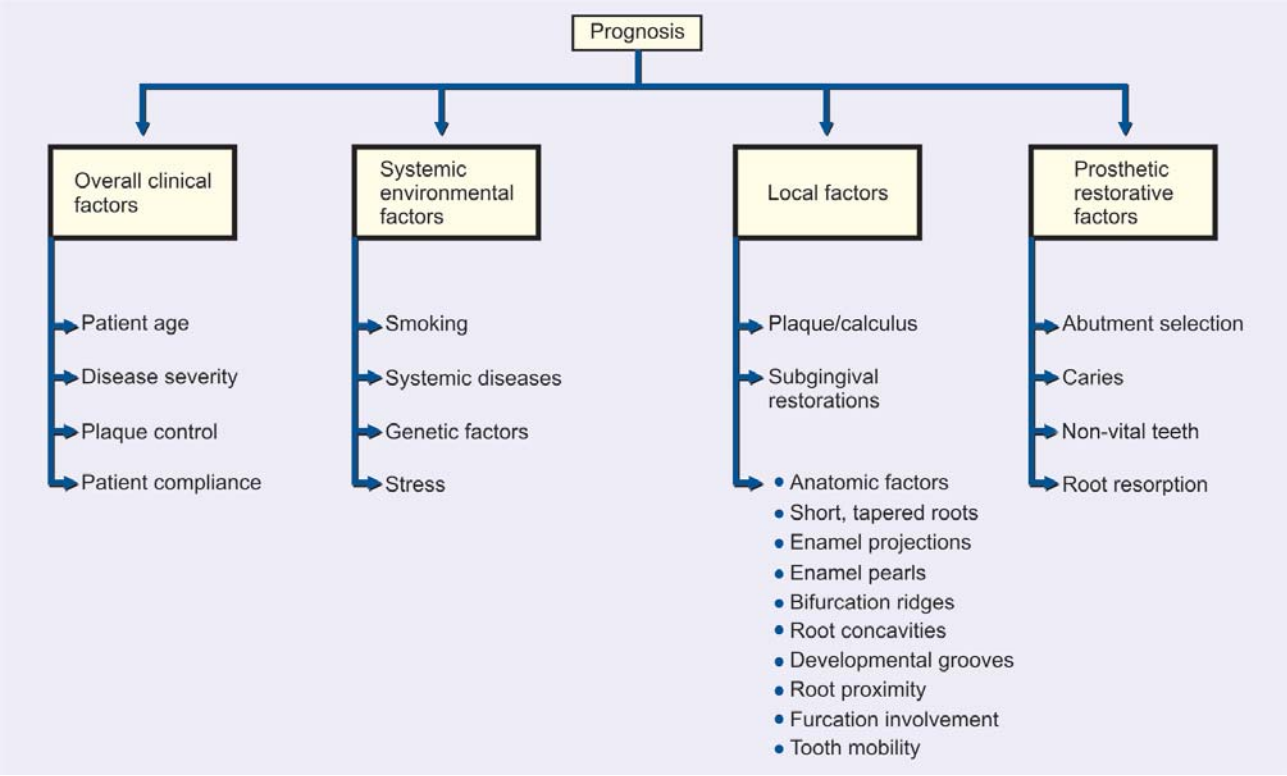

Fig. 30.1: Factors for determination of prognosis

grade I furcation involvement, adequate maintenance, acceptable patient co-operation, presence of limited systemic/environmental factors.

iv. *Poor prognosis*: One or more of the following: moderate to advanced bone loss, tooth mobility, grade I and II furcation involvements, doubtful patient co-operation, difficult to maintain areas, presence of systemic/environmental factors.

v. *Questionable prognosis*: One or more of the following: advanced bone loss, grade II and III furcation involvements, tooth mobility, inaccessible areas, systemic/environmental factors.

vi. *Hopeless prognosis*: One or more of the following: advanced bone loss, non-maintainable areas, extraction indicated, presence of uncontrolled systemic/environmental factors.

It is advisable to establish a provisional prognosis until phase I therapy is completed and evaluated. The provisional prognosis allows initiating treatment of teeth having a doubtful outlook. The re-evaluation phase allows to assess the tissue response to scaling, oral hygiene and root planing and also the use of chemotherapeutic agents where indicated.

Factors for Determination of Prognosis (Fig. 30.1)

i. Overall clinical factors.
ii. Systemic/Environmental factors.
iii. Local factors.
iv. Prosthetic/restorative factors.

Overall Clinical Factors

a. *Patient age*: In two patients with comparable levels of remaining connective tissue attachment and alveolar bone, the prognosis is better in the older of the two. In younger patient, the prognosis is not as good because of the increased periodontal destruction in a shorter time frame. The cause may be aggressive type of periodontitis or the progression of disease may be increased due to systemic diseases or smoking.

b. *Disease severity*: It is determined by recording the patient's past history of periodontal disease; for this the following variables should be carefully recorded:
   i. Pocket depth.
   ii. Level of attachment.
   iii. Degree of bone loss.
   iv Type of bony defect.

These can be determined by clinical and radiographic evaluation.

   i. *Pocket depth*: This is not necessarily related to bone loss. A tooth with deep pockets and little attachment and bone loss has a better prognosis than one with shallow pockets and severe attachment and bone loss.
   ii. *Level of attachment*: Reveals the approximate extent of root surface that is devoid of periodontal ligament; the radiographic examination reveals the amount of root surface still invested in bone. Prognosis is adversely affected if the base of the pocket is close to the root apex. The presence of apical disease as a result of endodontic involvement also worsens the prognosis.
   iii. *Degree of bone loss*: The prognosis can also be related to the height of the remaining bone. The height of the remaining bone is usually somewhere in between making bone level assessment alone insufficient for determining the overall prognosis.
   iv. *Type of bony defect*: The prognosis for horizontal bone loss depends upon the height of the existing bone. The prognosis for angular, intrabony defects depends upon the contour of the existing bone and the number of osseous walls. The chance to regenerate bone in vertical bony defect is excellent as compared to horizontal bony defects.

When greater bone loss has occurred on one surface of a tooth, the bone height on the less involved surfaces should be considered when determining the prognosis. Because greater the height of bone in relation to other surfaces, the center of rotation of the tooth will be nearer the crown which results in favorable distribution of forces to the periodontium and less tooth mobility. No heroic attempts should be made to retain hopelessly involved tooth because it may indirectly affect the health of adjacent teeth.

c. *Plaque control:* Plaque is the primary etiological factor for periodontal disease. Therefore, effective removal of plaque is important for the success of periodontal therapy and to the prognosis.
d. *Patient compliance and co-operation:* The prognosis for patients with gingival and periodontal disease is dependent on patient's attitude; desire to retain the natural teeth, willingness and ability to maintain good oral hygiene. If the patient is unwilling or unable to perform adequate plaque control and receive periodic maintenance checkups, the treatments to be considered are:
   1. Refuse to accept the patient for treatment.
   2. Extract teeth that have a hopeless or poor prognosis and perform scaling and root planing on the remaining teeth.

Systemic/Environmental Factors

a. *Smoking*: It is the most important environmental risk factor affecting the development and progression of periodontal disease. There is a direct relationship between smoking and the prevalence and incidence of periodontitis. It even affects the healing potential of the periodontal tissues. Therefore, the prognosis in patients who smoke and have slight to moderate periodontitis is generally fair to poor. But with the cessation of smoking, the prognosis may be upgraded from fair to good in case of slight to moderate periodontitis and poor to fair in case of severe periodontitis.
b. *Systemic disease/condition*: Studies have shown that patients with type I and type II diabetes have increased severity of periodontitis than in those without diabetes. Prognosis in these cases is dependent on patient compliance relative to both their medical and dental status well-controlled diabetics.

In patients with uncontrolled diabetes, the prognosis is questionable when surgical periodontal treatment is required. In well-controlled diabetic patients with mild

to moderate periodontitis, who comply well to recommended instructions, respond well and hence have good prognosis. Similarly, patients with incapacitating conditions and other systemic disorders can affect the progression of disease, hence correction of systemic problem may improve the prognosis. Newer automated oral hygiene devices like electric tooth brushes may help patients with Parkinson's disease to maintain oral hygiene and thereby improve their prognosis.

c. *Genetic factors:* Genetic polymorphisms in the interleukin–1 (IL-1) genes, resulting in increased production in IL1–β, have been associated with a significant increase in risk for severe, generalized, chronic periodontitis. Genetic factors also appear to influence serum IgG_2 antibody titers and the expression of Fcγ RII receptors on the neutrophil, both of which may be significant in aggressive periodontitis. Other genetic disorders such as leukocyte adhesion deficiency type I can influence neutrophil function, creating an additional risk factor for aggressive periodontitis.

   Detection of genetic variations that are linked with periodontal disease can potentially influence the prognosis in several ways. First, early detection of patients at risk due to genetic factors, can lead to early implementation of preventive and treatment measures. Second, identification of genetic risk factors during the course of treatment can influence treatment recommendations such as the use of adjunctive antibiotic therapy. Finally, identification of young individuals who are identified as being at risk because of the familial aggregation seen in aggressive periodontitis can lead to the development of early intervention strategies. Early diagnosis, intervention and/or alterations in the treatment regimen may lead to an improved prognosis for the patient.

d. *Stress*: Physical and emotional stress as well as substance abuse alter the patients ability to respond to periodontal treatment.

Local Factors

a. *Plaque/calculus*: It is the most important local factor in periodontal diseases. In most cases, having a good prognosis is dependent on the ability of the patient and the clinician to remove these etiologic factors.

b. *Subgingival restorations*: Contribute to increased plaque accumulation, increased inflammation and increased bone loss. Overhangs can have negative impact on the periodontium. The amount of destruction is mainly dependent upon the size of these discrepancies and the amount of time they have been present. In general, a tooth with a discrepancy in its subgingival margins has a poorer prognosis than a tooth with well-contoured, supragingival margins.

c. *Anatomic variations/factors*: Anatomic factors that predispose the periodontium to disease include short, tapered roots with large crowns, cervical enamel projections (CEP) and enamel pearls, intermediate bifurcation ridges, root concavities and developmental grooves, root proximity, furcation involvement and tooth mobility.

   Prognosis is poor for teeth with short, tapered roots and relatively large crowns because of disproportionate crown to root ratio and reduced root surface area available for periodontal support. The periodontium may be injured by occlusal forces.

   Cervical enamel projections (CEP) are flat, ectopic extensions of enamel that extend beyond the normal contours of the cemento-enamel junction. They may also extend into furcations, most likely found on buccal surfaces of maxillary second molars. Enamel pearls are larger, round deposits of enamel that can be found in furcations or other areas of root surface. They are less frequent than CEP, the presence of these enamel pearls on the root surface interferes with the attachment apparatus and may prevent regenerative procedures from achieving their maximum potential. Therefore, it has a negative effect on the prognosis of individual teeth.

   Scaling and root planing is a fundamental procedure in periodontal therapy. Anatomic factors that decrease the efficiency of this procedure have a negative impact on prognosis. Root concavities exposed through loss of attachment can vary from shallow flutings to deep depressions. These concavities increase the attachment

area and produce a root shape that may be more resistant to torquing forces, and also create areas that can be difficult for dentist and patients to clean.

Other anatomic factors that present accessibility problems include developmental grooves, root proximity and furcation involvements. The presence of any of these can worsen the prognosis. The developmental grooves, which initiates on enamel can extend to a significant distance on the root surface. It provides plaque-retentive areas that are difficult to clean by the instrument. Root proximity results in interproximal areas that are difficult for the clinician and patient to access. Furcation areas are also difficult to access.

d. *Tooth mobility*: The principal causes are loss of alveolar bone, inflammatory changes in the periodontal ligament and trauma from occlusion. Tooth mobility caused by inflammation and trauma from occlusion may be correctable but tooth mobility caused by loss of alveolar bone is not likely to be corrected. The stabilization of tooth mobility through the use of splinting may have a beneficial impact on the overall and individual tooth prognosis.

Prosthetic/Restorative Factors

The overall prognosis requires general consideration of bone levels and attachment levels to establish whether teeth can be saved for functional and aesthetic purposes or to serve as abutments for prosthesis. When few teeth remain, the prosthodontic need become more important and sometimes periodontally treatable teeth may have to be extracted if they are not compatible with the design of the prosthesis. Teeth that serve as abutments are subjected to increased functional demands. A tooth that has undergone endodontic treatment post is more likely to fracture when serving as a distal abutment supporting a distal removable partial denture. Special oral hygiene measures should be instituted in these areas.

Caries, non-vital teeth and root resorption: Teeth with extensive caries should be adequately restored and endodontic therapy should be considered before undertaking periodontal treatment. Extensive idiopathic root resorption or root resorption as a result of orthodontic therapy, jeopardises the stability of teeth and adversely affects the response to periodontal treatment. The periodontal prognosis of treated non-vital teeth is not different from that of vital teeth. New attachment can occur to cementum of both non-vital and vital teeth.

OVERALL VERSUS INDIVIDUAL TOOTH PROGNOSIS

Prognosis can be divided into overall and individual tooth prognosis.

Overall Prognosis

It is determined for the whole dentition and it addresses some important questions such as—

- Should treatment be undertaken?
- Is treatment likely to succeed?
- When prosthetic replacement is needed, are the remaining teeth able to support the added burden of the prosthesis?

Factors influencing the overall prognosis are:

- Patient's age
- Current severity of disease
- Systemic factors
- The presence of plaque, calculus and other local factors
- Patient compliance
- Smoking
- Prosthetic possibilities.

Individual Tooth Prognosis

It is determined for a single tooth and it is determined after the overall prognosis. Individual tooth prognosis is affected by overall prognosis. For example, in a patient with a poor overall prognosis, the dentist likely would not attempt to retain a tooth that has a questionable prognosis because of local conditions.

Factors Influencing the Individual Tooth Prognosis

- Plaque and calculus
- Subgingival restorations

- Anatomic factors
  - — Short, tapered roots
  - — Cervical enamel projections
  - — Enamel pearls
  - — Bifurcation ridges
  - — Root concavities
  - — Developmental grooves
  - — Root proximity
  - — Furcation involvement.
- Prosthetic and restorative factors
  - — Abutment selection
  - — Caries
  - — Non-vital teeth
  - — Root resorption
- Mobility

RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS

Factors such as patient's age, severity of disease, genetic susceptibility and presence of systemic disease are all important criteria in the diagnosis of the condition. These are also important in developing a prognosis (Table 30.2).

Prognosis for Patients with Gingival Disease

Dental Plaque-induced Gingival Disease

- *Gingivitis associated with dental plaque only*: The prognosis for patients with gingivitis associated with only dental plaque is good, provided all local irritants are eliminated, i.e. other local factors contributing to plaque retention are eliminated, gingival contours conducive for the preservation of health are attained and the patient co-operates by maintaining good oral hygiene.
- *Plaque-induced gingival diseases modified by systemic factors*: The systemic factors which influence the inflammatory response to bacterial plaque include endocrine related changes associated with puberty, menstruation, pregnancy and diabetes and the presence of blood dyscrasias, prognosis for these patients depends on not only control of bacterial plaque but also on control or correction of the systemic factors.
- *Plaque-induced gingival diseases modified by medications*: Drug-influenced gingival enlargement is often seen with phenytoin, cyclosporin, nifedipine and oral contraceptive-associated gingivitis. Plaque control alone does not prevent the development of the lesions and surgical intervention is usually necessary to correct the alterations in gingival contour. Continued use of the drug causes recurrences of the enlargement even following surgical intervention. Therefore, prognosis is dependent on whether the patient's systemic problem can be treated with an alternative medication. In oral contraceptive associated gingivitis, frank signs of gingival inflammation can be seen in the presence of relatively little plaque. Prognosis in these patients is dependent not only on the control of dental plaque, but also on the likelihood of continued use of the oral contraceptive.
- *Gingival diseases modified by malnutrition*: Most of the clinical studies have disproved the importance of malnutrition in developing gingival disease. Prognosis in these cases may be dependent on the severity and duration of deficiency and on the likelihood of reversing the deficiency through dietary supplementation.

Nonplaque-induced Gingival Lesion

It can be seen in patients with a variety of bacterial, fungal and viral infections. Prognosis is dependent on elimination of the source of infectious agent.

Prognosis for Patients with Periodontitis

See Table 30.3.

Table 30.2: Distinction between condition and prognosis

	Condition	*Prognosis*
i.	Dermatological disorders like lichen planus, pemphigoid, pemphigus vulgaris, erythema multiforme, lupus erythematosis.	It is linked to management of the associated dermatologic conditions.
ii.	Allergic, toxic and foreign body reactions, Mechanical and thermal trauma.	

Table 30.3: Distinction between chronic and aggressive periodontitis

Chronic periodontitis	*Aggressive periodontitis*
i. Slowly progressive disease associated with local environmental factors. ii. Can be localized or generalized. iii. In case of not advanced attachment loss, prognosis is generally good. But inflammation has to be controlled through good oral hygiene and removal of local plaque-retentive factors. iv. In cases of severe disease with furcations involvement and increasing clinical mobility/ who are non-compliant with oral hygiene, prognosis is down graded from fair to poor.	i. Rapidly progressive disease with minimal/no local factors with increased level of Aa and P gingivalis. ii. Can be localized or generalized. iii. Two common features are observed. a. Rapid attachment loss and bone destruction in an otherwise clinically healthy person. b. A familial aggregation. iv. Localized type, which occurs around the age of puberty and is localized to first molars and incisors, if diagnosed early can be treated conservatively with oral hygiene instructions and systemic antibiotic therapy. Resulting in excellent prognosis. In advanced cases, the prognosis is still good if the lesions are treated with debridement, local and systemic antibiotics and regenerative therapy. v. In generalized type, also seen in young patients with generalized interproximal attachment loss and poor antibody response. Secondarily aggravated by cigarette smoking does not respond well to conventional periodontal therapy, therefore prognosis is often fair, poor or questionable and the use of systemic antibiotics should be considered.

Periodontitis as a Manifestation of Systemic Disease

It can be divided into two categories:

i. Associated with hematological disorders such as leukemia and acquired neutropenias.
ii. Associated with genetic disorders such as familial and cyclic neutropenia, Down syndrome, hypophosphatasia, Papillon-Lefévre syndrome.

In both these cases, prognosis may be fair to poor and is mainly dependent on the treatment of systemic disease.

Necrotizing Periodontal Disease

Necrotizing periodontal disease can be divided into necrotic diseases that affect the gingival tissues [NUG] and necrotic diseases that affect deeper tissues of the periodontium, resulting in loss of connective tissue attachment and alveolar bone [NUP]. In these [NUG] cases, the predisposing factor is bacterial plaque. The disease is further complicated by both presence of secondary factors such as acute psychological stress, tobacco smoking and poor nutrition, all of which can contribute to immunosuppression. With the control of both the bacterial plaque and the secondary factors, the prognosis is good.

In necrotizing ulcerative periodontitis cases the treatment is not only reducing local and secondary factors but also in dealing with the systemic problem. Prognosis depends on management of disease.

RE-EVALUATION OF PROGNOSIS AFTER PHASE I THERAPY

A frank reduction in pocket depth and inflammation after phase I therapy indicates favourable response to treatment and is suggestive of a better prognosis and in vice versa cases the overall prognosis may be unfavourable.

KEYPOINTS

1. Prognosis is defined as the prediction of the probable course, duration and outcome of a disease based on a general knowledge of the pathogenesis of the disease and the presence of risk factors for the disease.
2. Prognosis is established after the diagnosis and before the treatment plan.

3. Prognosis may be excellent, good, fair, poor, questionable, and hopeless.
4. Prognosis can be divided into, overall prognosis and individual tooth prognosis.
5. Factors that are to be considered while determining the prognosis are:
   a. Overall clinical factors like age of the patient, disease severity, systemic restorative and environmental factors and local factors, patient compliance and prosthetic possibilities.
   b. Factors influencing individual tooth prognosis are– plaque/calculus, subgingival restorations, caries, non-vital teeth and root resorption.

REVIEW QUESTIONS

1. Define prognosis and describe the factors influencing overall prognosis.
2. Describe the factors to be considered for determining prognosis.

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31 Chapter

Related Risk Factors Associated with Periodontal Diseases

- DEFINITION
- RISK FACTORS
- RISK DETERMINANTS/BACKGROUND CHARACTERISTICS
- RISK INDICATORS
- RISK MARKERS/PREDICTORS
- CLINICAL RISK ASSESSMENT
  - Demographic Data
  - Medical History
  - Dental History
  - Clinical Examination

DEFINITION

Risk is the probability that an individual will develop a specific disease in a given period which may vary from one individual to another. Risk assessment involves identifying elements that either may predispose a patient to developing periodontal disease or may influence progression of disease that already exists.

RISK FACTORS FOR PERIODONTAL DISEASE

Risk factors may be environmental, behavioral or biologic factors that, when present, increases the likelihood that an individual will develop the disease. Following are the risk factors:

1. *Tobacco smoking*: A direct relationship exists between smoking and prevalence of periodontal disease. Smoking has a negative impact on response to therapy.
2. *Diabetes*: Diabetes is a risk factor for periodontitis. Prevalence and severity is higher in diabetes than in those without diabetes.
3. *Pathogenic bacteria and microbial tooth deposits*:
   - Quantity of plaque present on teeth is not of major importance. Composition or quality of the plaque biofilm is of importance.
   - In terms of plaque, three specific bacteria have been identified as etiologic agents:
     a. *Actinobacillus actinomycetemcomitans*
     b. *Porphyromonas gingivalis*
     c. *Bacteroides forsythus*
4. *Anatomic factors*: Such as furcations, root concavities, developmental grooves, cervical enamel projections, enamel pearls and bifurcation ridges. All predispose to periodontitis, as they harbor bacterial plaque and present a challenge to clinician during instrumentation.

Presence of calculus: It serves as a reservoir for bacterial plaque.

RISK DETERMINANTS/BACKGROUND CHARACTERISTICS FOR PERIODONTAL DISEASE

It is defined as those risk factors that cannot be modified.

1. *Genetic factors:* Genetic factors influence clinical measures of gingivitis probing pocket depth, attachment loss and interproximal bone height.
   - A specific interleukin-1 (IL-1) genotype has been associated with severe chronic periodontitis.
   - Immunologic alterations such as neutrophil abnormalities are under genetic control.
   - Genetics plays a role in regulating the titre of protective IgG_2 antibody response to A. *actinomycetemcomitans* in patients with aggressive periodontitis.
2. *Age:*
   - Both the prevalence and severity of periodontal disease increases with age.
   - Attachment loss and bone loss seen in elderly individuals is a result of prolonged exposure to other risk factors over a longer period of time.
   - Changes related to old age such as intake of medications, decreased immune functions and altered nutritional status may increase susceptibility to periodontitis.
3. *Gender:*
   - Males have more attachment loss than females and have a poorer oral hygiene, therefore more males are prone to periodontal diseases.
4. *Socioeconomic status:*
   - Poor oral health is seen in lower socioeconomic status. This can be attributed to:
     1. Decreased dental awareness.
     2. Decreased dental visits.
5. *Stress:* Incidence of ANUG increases during stressful situations.
   - Emotional stress may interfere with normal immune function.
   - There is an apparent link between psychosocial factors and risk behavior such as smoking, poor oral hygiene and chronic periodontitis.
   - Individuals with financial strain, distress, depression and inadequate coping mechanisms have more loss of attachment.

RISK INDICATORS FOR PERIODONTAL DISEASE

Risk indicators are probable or putative risk factors that have been identified in cross-sectional studies but have not been confirmed through longitudinal studies.

Following are the risk indicators:

1. *HIV/acquired immunodeficiency syndrome:* It has been hypothesized that immune dysfunction associated with high HIV infection and AIDS increases the susceptibility to periodontal disease, though the evidence is not conclusive.
2. *Osteoporosis:* Osteoporosis does not by itself initiate periodontitis, there is reduced bone mass in osteoporosis, this may enhance the progression of periodontal disease.
3. *Infrequent dental visits:* Some studies have shown increased risk for severe periodontitis in patients who have not visited dentist for 3 or more years (although age factor also plays a role).

RISK MARKERS/PREDICTORS

These are associated with increased risk for disease, but do not cause the disease. These factors also are identified in cross-sectional and longitudinal studies. A risk factor that can be used to predict the future course of disease, is known as a risk marker.

1. Previous history of periodontal disease.
2. Bleeding on probing: Bleeding on probing along with increased pocket depth may serve as an excellent predictor for future loss of attachment.

CLINICAL RISK ASSESSMENT FOR PERIODONTAL DISEASE

It is done by careful evaluation of the following:

Demographic Data

Age

- Duration of exposure to the risk elements
- Postmenopausal women
- Evidence of aggressive disease

Gender

- Males
- Frequency of care and preventive practices

Socioeconomic Status

- Dental awareness
- Frequency of care

Medical History

The following conditions either predispose or make the patient more susceptible to periodontitis.

- Diabetes
- Tobacco smoking
- HIV/AIDS
- Osteoporosis
- Stress

Dental History

- Genetic predisposition to aggressive disease.
- Previous history of periodontal disease.
- Frequency of dental care.

Clinical Examination

- Plaque accumulation
- Calculus
- Bleeding on probing
- Extent of loss of attachment
- Tooth examination
  - Plaque retentive areas
  - Anatomic factors
- Restorative factors
- Once a risk patient is identified and a diagnosis is made, treatment plan may be modified accordingly.

KEYPOINTS

1. *Risk factors* may be environmental, behavioral or biologic in nature that when present increases the likelihood of a disease, e.g. smoking, diabetes, presence of pathogenic bacteria.
2. *Risk determinants* are those risk factors that cannot be modified, e.g. age, gender, socioeconomic status, stress.
3. *Risk indicators* are probable or putative risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies, e.g. AIDS, osteoporosis, infrequent dental visits.
4. A *risk factor* that can be used to predict the future course of disease, is known a *risk marker*, e.g. previous history of periodontitis, bleeding on probing.
5. In conclusion, risk assessment may have a role at two levels, one involving identification of factors that may predispose to developing periodontal disease, secondly, may influence the progression of already existing disease.

KNOW MORE ...

Hill's Criteria

In order to confirm a risk factor as a part of the casual chain, Hill's criteria are usually quoted:

- Strength of association between factor and disease.
- Consistency of association in different populations.
- Temporal sequence: A factor must precede outbreak of the disease (principle of cause and effect).
- Specificity of association.
- Dose-response effect.
- Biological plausibility.
- Experimental evidence.

REVIEW QUESTIONS

1. Define risk, risk factors, risk indicators, risk markers and risk determinants.
2. Enumerate various risk factors for periodontal disease.

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32 Chapter

Various Aids including Advanced Diagnostic Techniques

- AIDS USED IN CLINICAL DIAGNOSIS
  - Periodontal Probes
  - Conventional Probes
  - PSR
- AIDS USED IN RADIOGRAPHIC DIAGNOSIS
  - Orthopantomograph
  - Xeroradiography
  - Advanced Radiographic Techniques
- AIDS IN MICROBIOLOGICAL DIAGNOSIS
  - Identification of Bacteria
  - Speciation Techniques
- AIDS IN IMMUNOLOGICAL DIAGNOSIS
  - Immunofluorescence
  - Latex Agglutination
  - ELISA
  - Flow Cytometry
- BIOCHEMICAL DIAGNOSIS
  - Prostaglandins
  - Collagenase
- OTHER DIAGNOSTIC AIDS
  - BANA Test
  - FSEIA
  - PCR

AIDS USED IN CLINICAL DIAGNOSIS

a. Millimeter probe for gingival bleeding.
b. Measurement of gingival crevicular fluid flow with the help of a filter paper. Newer method is by use of a periotron 6000.
c. Measurement of temperature by pressure-sensitive probes. Periotemp™ probe (Abiodent™).
d. Mouth odors—olfactometer.
e. Tooth mobility—mobilometer/periodontometer.
f. PSR (Periodontal screening and recording).

Periodontal Probes

Uses of Probes

1. Periodontal probes are used to measure the pocket depth.
2. Quantification of bacterial plaque and gingival inflammation.
3. Determination of mucogingival relationship.
4. Measurement of gingival recession.
5. Location of calculus.
6. Identification of tooth irregularities.

7. Identification of tissue characteristics.
8. Determination of bleeding tendency.
9. Evaluation of bone support in the furcation areas of bifurcated and trifurcated teeth.

Types of Conventional Periodontal Probes (Fig. 32.1)

1. Marquis color coded probe: Calibrations are in 3 mm sections.
2. The University of North Carolina-15 probe (UNC-15): 15 mm long and markings are at 1 mm and color coding at the 5th, 10th and 15 mm.
3. The University of Michigan O probe with Williams markings (at 1, 2, 3, 5, 7, 8, 9) 4 and 6 missing.
4. The Michigan 'O' probe with markings at 3, 6, and 8 mm.
5. The WHO probe, which has a 0.5 mm ball at the tip and millimeter markings at 3.5, 8.5 and 11.5 mm and color coding from 3.5 to 5.5 mm.
6. Furcation areas can best be evaluated with the curved, blunt Naber's probe.

Periodontal probes may be divided into:

First generation probes are conventional, hand held probes, e.g. conventional periodontal probes.

Second generation probes are the pressure-sensitive probes. It has been shown that, with forces up to 30 grams, the probe tip remains within junctional epithelium and forces up to 50 grams are necessary to diagnose osseous defects. This probe did solve many of the problems of the conventional probes, but lacked tactile sensitivity.

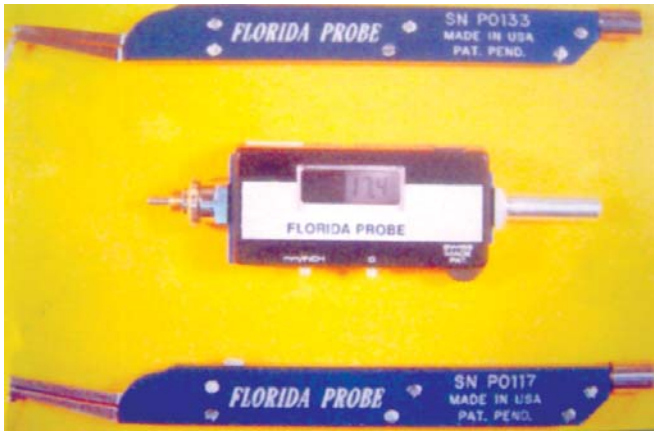

Fig. 32.1: Florida probe

Third generation probes are the computerized probes. Gibbs et al designed Florida probe. Other examples are—Foster Miller probe, Toronto automated probes, which can detect the cementoenamel junction.

Limitations of Conventional Probes

1. Probing depth obtained with periodontal probe does not coincide with the histological pocket depth, because the probe normally penetrates the coronal level of the junctional epithelium.
2. Another limitation is related to the reproducibility, which has been correlated with the variation in the probing force.

Other factors that are likely to influence clinical measurement of attachment level include intra and inter-examiner reliability, patient discomfort, accuracy of probe markings and anatomical variations in tooth contours or position.

Limitations of all Automated Controlled Force Probes

1. Reduced tactile sense of the operator.
2. Increased patient discomfort.
3. Presence or absence of inflammation often produced inaccurate measurement.

Periodontal Screening and Recording (PSR)

It is designed for easier and faster screening and recording of the periodontal status of a patient or a group of population. It uses a specially designed probe that has a 0.5 mm ball tip and is color-coded from 3.5 to 5.5 mm.

The patient's mouth is divided into six sextants—maxillary right quadrant, left quadrant and anteriors, mandibular left and right quadrants and anteriors, and at least six points around each tooth is examined.

The deepest finding is recorded in each sextant, according to the following code.

Code 0: In the deepest sulcus of the sextant, the probes colored band remains completely visible, gingival tissue is healthy and does not bleed on gentle probing. No calculus or defective margins are found. These patients require only appropriate preventive care.

Code 1: The colored band remains completely visible in the deepest sulcus of the sextant. No calculus or defective margins are found but some bleeding after gentle probing is found. Treatment for these patients include subgingival plaque removal and appropriate oral hygiene instructions.

Code 2: The probe's colored band is still completely visible, but there is bleeding on probing. Supragingival or subgingival calculus and/or defective margins are found. Treatment includes plaque and calculus removal, correction of plaque retentive margins of restorations and oral hygiene instructions.

Code 3: The colored band is partially-submerged. This indicates the need for a comprehensive periodontal examination and charting of the affected sextant to determine the necessary treatment plan. If two or more sextants score code 3, a comprehensive full mouth examination and charting is indicated.

Code 4: The colored band completely disappears in the pocket, indicating a depth greater than 5.5 millimeters. In this case, a comprehensive full-mouth periodontal examination, charting and treatment planning are needed.

Code:* When any of the abnormalities are seen, an asterisk (*) is entered, in addition to the code number, (for example, furcation involvement, tooth mobility, mucogingival problem or gingival recession extending to the colored band of the probe).

AIDS USED IN RADIOGRAPHIC DIAGNOSIS

Radiographs are used to obtain a visual image of the bone support around a tooth or dental implant. The radiographic image is the result of X-ray beam passing through the area of interest and exposing the silver halide emulsion on the radiographic film.

The most commonly used radiographs in periodontal diagnosis are transmission radiographs. Transmission radiographs, including periapical and bite-wing films are used to detect the amount of bone loss in any type of periodontitis. They should be used with less exposure to avoid any cervical burnout effect.

Though there are many *advantages* of radiographs there is also equal number of *disadvantages*:

1. Thirty to sixty percent of the mineral content of the bone must be lost to visualize the change in the radiographic image, hence though very specific, lacks sensitivity.
2. Actual damage is more extensive than radiographs.
3. Radiographs are a two-dimensional representation of a three-dimensional anatomy.

Techniques are available to minimize this source of distortion. First a long cone should be used (because parallel rays will minimize distortion).

Secondly, the use of parallel positioning devices helps to standardize the relationship between film, object and X-ray source, e.g. RINN XPR.

Disadvantage: Only a limited view of the osseous crest is available. Hence the use of extended cone projection instruments is recommended.

Bite-wing: It is an often forgotten radiograph in periodontal diagnosis. On a bite-wing radiograph we can visualize both maxillary and mandibular teeth along with the interdental alveolar bone. Thus, it can provide us with the vital information regarding the presence of local irritating factors, e.g. calculus.

Orthopantamograph (OPG)

When compared with IOPA radiograph, they have a tendency to underestimate minor bone changes. The major disadvantages of OPG are magnification, unsharpen and distortion. However, the clarity of panoramic image obtained from digital machines are superior to analogue machines.

Xeroradiography

It does not involve wet chemical processing or the use of a dark room. Instead of X-ray film, xeroradiography uses a uniformly charged selenium plate held in a light tight cassette. Exposure to X-ray and adequate processing produces a real image on opaque paper, which is viewed by reflected light.

Advantages: Less expensive, edge enhancement.

Advanced Radiographic Techniques

Techniques have been developed to enhance the ability to "see" small changes over a period of time in the bone. They are:

Iodine-125 Absorptiometry

A nonradiographic method to analyze the periodontal bone mass changes. It is based on the absorption by bone of a low energy gamma beam, originating from a radioactive source of 125-1. This method has shown to measure bone changes with a high degree of accuracy and precision.

Disadvantage: Technical considerations limit the use of this system on posterior sites. To overcome this, photo-densitometric analysis has been developed.

Photodensitometric Analysis

A beam of light is passed onto the radiographic film and the image is shown on an aluminium scale and then it transforms the density readings into millimeter of aluminium equivalents. It is mainly developed to evaluate bone resorption especially in furcation areas. This technique mainly enables the clinician to detect the variations in the bone density that cannot be detected by visual inspection.

Digital Radiography

This is useful in detecting small changes in hard tissues that occur between examinations. The purpose of digital subtraction radiography is to remove all unchanging structures from a set of two films and to display only the area of changes in periodontal defects.

Substraction Radiography

Two radiographs are taken and the changes are noted depending on their gray levels.

Digital Substraction Radiography

Digitization is done before subtraction, i.e. serial radiographs are converted into digital images. These images are superimposed and are used on a video screen. Light areas indicate bone gain and dark areas indicate bone loss.

Computer Assisted Densitometric Image Analysis (CADIA)

In this technique, parallelization errors can also be corrected and values of difference are shown between two X-rays. A video camera measures the light transmitted through a radiograph and the signals from the camera are converted into gray levels. The images can be stored in the computer.

Computerized Tomography

Unlike conventional radiography, which is a two-dimensional representation of a three-dimensional object. Computed tomography gives an exact picture of the bone levels in coronal, axial and sagittal plane by which all the osseous defects can be visualized accurately.

Nuclear Medicine Bone Scan

This involves the detection of changes in bone metabolism—hence can detect the earliest stage of bone loss. A bone seeking radiopharmaceutical diphosphonate compound is injected intravenously and after a waiting period, the uptake by the bone is measured by the semiconductor probe radiation detector. This technique has the ability to detect bone changes before structural alterations occur.

AIDS IN MICROBIOLOGICAL DIAGNOSIS

It is based on the concept of bacterial specificity. These microbiological tests may have the potential not only to diagnose various forms of periodontal diseases but also to determine the sites which are at a higher risk of undergoing active destruction.

Predictive treatment model: It is a combination of clinical and microbiological parameters to predictably recommend specific therapy.

Microbiology and Disease Progression

Because bacteria are the causative agents in periodontal diseases it makes sense to look for specific bacteria as indicators of disease activity.

Identification of Bacteria

1. *Direct examination*—microscopy
   - Light
   - Dark field

2. *Culture and sensitivity assay*
   i. *Culture techniques*
      - Aerobic
      - Anaerobic
   ii. *Speciation techniques*
      - GLC (Gas liquid chromatography)
      - DNA homology

Direct microscopy: Specimens are viewed directly under the light. They are of two types:

a. *Light microscopy:* Under this stained or unstained specimens can be read.
   Gram's staining: Differentiates Gram-positive and Gram-negative organisms. Gram-positive appears violet. Gram-negative appears pink under the microscope. This may be important because it differentiates between health and disease.
b. *Dark field and phase contrast microscopy:* Fresh, unstained samples are examined. It uses a special condenser in which the light rays are either reflected or refracted off bacterial cell surface. So the outline of the bacterium is dark against the light background in phase contrast microscopy and light against a dark background in dark-field microscopy.

Advantages of direct microscopy: It is quick, easy and inexpensive means of screening a microbial sample for major morphotypes.

Disadvantages

- Inability to identify species.
- Specimens have to be examined as soon as they are collected from the patients.

Culture methods: These are used for cultivation and identification of organisms, then to determine its susceptibility or resistance to various antimicrobial agents.

Types of specimens are:

- Blood samples.
- Mucosal surfaces.
- Periodontal pockets.

Subgingival plaque sampling methods (Fig. 32.2) are:

- Nickel-plated curettes
- Scalers

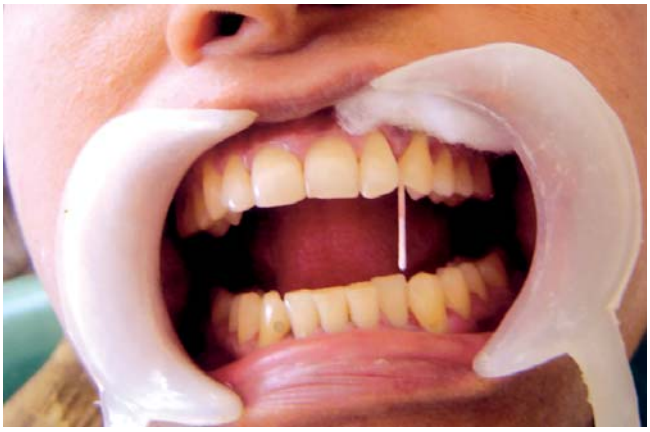

Fig. 32.2: Microbial sampling method

- Paper points
- Irrigation
- Surgical excision

For the identification of anaerobes from the clinical material, many artificial media and culture techniques are available.

Different Kinds of Media

Supportive media: Only allows growth of non-fastidious organisms.

Enriched media: Encourages the growth of organisms.

Nonselective media: Permits the growth of most oral micro-organisms without specific inhibitory agents.

Selective media: Contains dyes, antibiotics that are inhibitory to all organisms except those being sought.

Different Culture Techniques

a. *Jar technique*: Removes air/oxygen within the jar and is replaced by oxygen-free gas containing 80 to 90 percent nitrogen, 5 to 10 percent hydrogen and 5 to 10 percent carbon dioxide.
b. *PRAS*: Pre-reduced anaerobically sterilized roll tubes contain a medium which is boiled to remove the dissolved air and is then flushed with oxygen-free gas.
c. *Anaerobic chamber techniques*.
d. *Enzyme reduction technique*: It contains certain enzymes which can sweep the oxygen out.

Speciation Techniques

a. Gas Liquid Chromatography (GLC): In which various metabolic products of anaerobes are studied which are unique enough to serve as markers for identification.
b. *DNA probes* in the identification of periodontal pathogens: It is based on the ability of DNA to hybridize or bind to the complementary strands of DNA having the exact base sequence.

Procedure: The plaque is first denatured to obtain single strain bacterial DNA and then incubated on a membrane such as nitrocellulose. The specific labeled DNA probe is incubated on the membrane to allow hybridization and then washed off. The plaque sample contains complementary DNA; hybridization of the two single strains takes place, which can be visualized via the label of the probe.

METHODS/AIDS IN IMMUNOLOGICAL DIAGNOSIS

Immunofluorescence

This method permits the identification of specific bacteria in bacterial smears.

Antiserum: A serum that contains antibody or antibodies, it may be obtained from an animal that has been immunized either by injecting the antigen into the body or by infecting with microorganisms containing the antigen.

Direct Immunofluorescence (Fig. 32.3)

Antiserum to a microorganism is conjugated to fluorescein. The conjugate is incubated on a clinical smear containing the microorganisms and then washed off. The antigen antibody reactions take place and organism is visualized by its fluorescent outline, when observed under a fluorescent microscope, if the microorganism is not present, it appears dark with no fluorescence.

Indirect Immunofluorescence (Fig. 32.4)

It is a two step procedure. Antiserum to the microorganism is incubated on the clinical smear and washed off, then a conjugate of a fluorescent dye and an antiserum to the first antisera are incubated and then washed off.

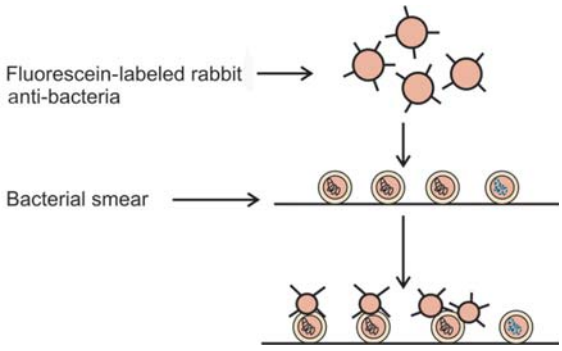

Fig. 32.3: Direct immunofluorescence

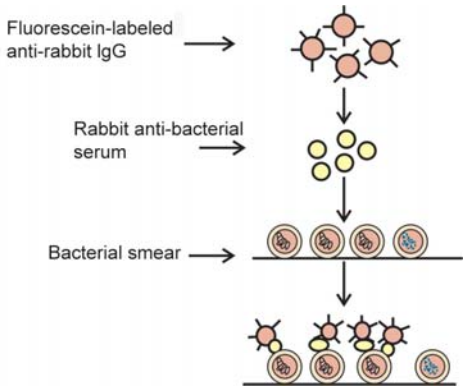

Fig. 32.4: Indirect immunofluorescence

It not only identifies but also quantifies the percentage of the pathogens in the latex smear.

Others are:

- Enzyme-linked immunosorbent assay (ELISA).
- Flow cytometry.

Latex Agglutination (Fig. 32.5)

It is based on the binding of protein to latex. Latex beads are coated with species specific antibody and when these beads come in contact with the microbial cell surface, antigens cross-linking occurs and its clumping/agglutination is made visible within 2 to 5 minutes.

Enzyme-linked Immunosorbent Assay (ELISA) (Fig. 32.6)

In this, bacterial antigens are incubated in a well, on a plastic plate to allow coating by the material. After washing to remove the free antigen, the plates are ready for tests. Samples containing suspected antibodies and controls are

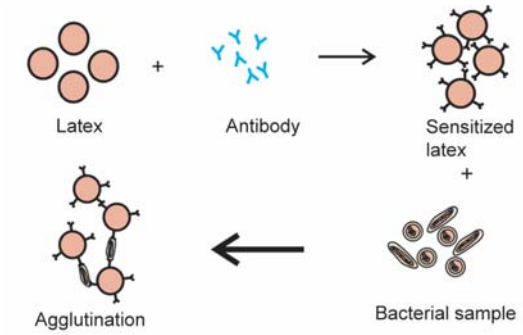

Fig. 32.5: Principle of latex agglutination test

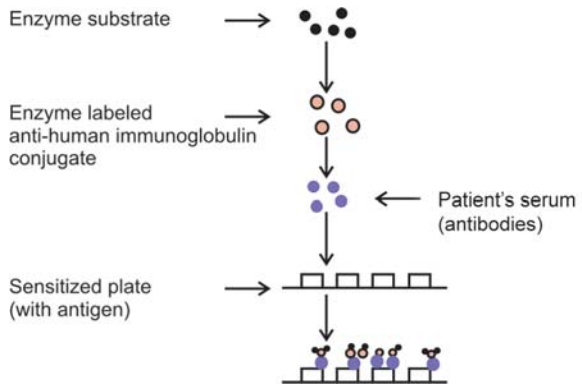

Fig. 32.6: Principle of ELISA assay

then incubated in separate wells to allow antibodies bind the antigen on the surface of the wells. After washing to remove unbound serum components, antisera to the antibody is conjugated to either alkaline phosphatase or horseradish peroxidase then incubated in the wells.

A positive reaction is visualized by addition of a chromogen which changes from a colorless to colored solution.

Flow Cytometry

This is for rapid identification of oral bacteria. This involves labeling bacterial cells from a patient plaque sample with both species specific antibody and a second fluorescein conjugated antibody. The suspension is then introduced into the flow cytometer, which separates the bacterial cells into an almost single cell suspension by means of a laminar flow through a narrow tube. After incubation, the cells are passed through a focussed laser beam. The cells then scatter the light at low and wide angles, and the fluorescent emission can be measured by appropriate detectors.

BIOCHEMICAL DIAGNOSIS

By-products of the cells (PMNLs), complement cleavage, e.g. C3, C4 in gingival crevicular fluid are studied.

Prostaglandins

Levels of prostaglandin E_2 are studied which can differentiate between gingivitis and periodontitis, e.g. aggressive forms showed higher levels than chronic periodontitis. Active sites exhibited five fold increase in PGE_2 levels than inactive sites.

PGE_2 levels are studied by RIA (Radioisotope assay).

Collagenase

It showed positive correlation with disease activity. It is studied by sodium dodecylsulphate polyacrylamide gel electrophoresis (PAGE). In this they studied breakdown products resulting from incubation of collagen with gingival crevicular fluid.

OTHER DIAGNOSTIC AIDS

a. BANA test
b. FSEIA
c. PCR (Polymerase chain reaction)

N-benzoyl-DL-arginine 2-naphthylamide (BANA)

Can identify:

- *B. forsythus*
- *P. gingivalis*
- *Treponema denticola*
- *Capnocytophaga*

Have a common trypsin-like enzyme, which hydrolyzes the colorless substrate.

N-benzoyl-DL-arginine-2-naphthylamide—when hydrolysis takes place, it releases the chromophore beta-naphthylamide, which turns orange-red when a drop of fast garnet is added to the solution.

Filter Separation Enzyme Immunoassay (FSEIA)

It can identify *A. actinomycetemcomitans*, *P. intermedia*, *P. gingivalis*.

The clinician mixes the plaque sample taken with a paper point with this reagent to produce a colored reaction which may be positive or negative. It requires 10 to 15 min of the office time.

Polymerase Chain Reaction (PCR)

History

Kary Mullis had just conceived a simple method of producing virtually unlimited copies of a specific DNA sequence in a test tube and introduced to the scientific community at a conference in Oct, 1985.

DNA Hybridization

The chemistry of PCR, as with much of molecular biology depends on the complementary's of DNA bases.

Mechanism of Action of PCR

1. The PCR is a test tube system for DNA replication that allows a "target" DNA sequence to be selectively amplified, or enriched, several million fold in just a few hours.
2. It involves a series of enzyme-mediated reactions whose end result is a copy of the entire genome.
3. PCR uses just one indispensable enzyme DNA polymerase to amplify a specific fraction of a genome.
4. DNA acts as "Priming site" for the attachment of DNA polymerase, two different primer sequences are used to bracket the target region to be amplified, one primer is complementary to one DNA strand at the beginning of the target region and second primer is complementary to a sequence on the opposite DNA strand at the target region.

Advantages

a. It is a quick, reliable method for detecting all manner of mutations associated with genetic diseases from insertions to deletions and to point mutations.
b. Used for detection of tiny amounts of human immuno-deficiency virus and numerous genetic anomalies.

KEYPOINTS

1. Probes are commonly used to detect and measure the pockets.
2. Periodontal probes can be divided into first generation, second generation and third generation probes.
3. First generation probes are conventional probes which are calibrated with or without color coding. Whereas second and third generation probes are pressure-sensitive, and also third generation probes can automatically detect the CEJ with computerized data capture.
4. Radiographs are used to obtain a visual image of the bone support around the teeth or implants. The most commonly used radiographs are, intraoral periapical and bite wing X-rays.
5. Various advanced radiographic techniques include, photodensitometric analysis, digital radiography, computerized tomography and nuclear medicine bone scan.
6. Identification of bacteria can be done by direct examination using light microscopy, phase contrast and dark field microscopy.
7. Methods used in immunological diagnosis include, immunofluorescence, latex agglutination, ELISA and flow cytometry.

KNOW MORE ...

Second Generation Probes

Some of the examples of second generation probes:

- Pressure probe (Vander Volden)
- Pressure sensitive probe (PSP)
- Borodontic probe
- Hunton probe (disposable probe)
- Yeaphe probe (to assess dentinal hypersensitivity).

Newer Generation Probes

Watts in the year 2000 has added two more generations to it:

- Generation IV and V

Fourth generation probes

These are three-dimensional probes, currently under development, these are aimed at sequential probe positions along the gingival sulcus.

Fifth generation probes

This is the only noninvasive three-dimensional probe. These will have an ultrasound attached to the fourth generation probe.

Some of the commercially available kits for identification of bacteria and its products are:

Assay	*Kit*	*Manufacturer/supplier*	*Comments*
Culture and biochemical identification (GOLD STANDARD)	Laboral Prognostic	Laboral, France Dentsply	Quantification/identification after bacterial culture of A. a,B. f, C. r, F. n, P. i, P. g, P. m Aids in detection of proteinase, elastase
Immunological detection (ELISA)	Evalusite test	Kodak Eastman company (Switzerland)	Detects bacterial antigens of A.a, P. i, P. g. can be used at chairside.
Bacterial enzymes	Perioscan BANA periodontal test	Oral B laboratories OraTec Corporation Manassas (USA)	Detects enzymatic activity of A.a, B.f, P.g Detects enzymatic activity of B. f, P. g, T. d
Bacterial toxins	TOPAS	Affinity Labeling Technologies (USA)	Detects toxins derived from anaerobic metabolism and measures GCF protein level.
Host enzymes	Periocheck	Collagenex pharmaceuticals	Detects enzymatic activity derived from GCF (Matrix metalloproteinases and neutral protease enzymes)
Nucleic acid technology	Affirm DP BTD test OMTL test, Omnigene	Microprobe (USA) BioTechnica Diagnostic (USA) USC (USA) Omnigene (USA)	DNA probes for A. a, B. f, P. i, P. g, T. d DNA probes for A.a, C.r, E.c, F.n, P.i, P.g. DNA probes for B. f, P. g DNA probes for A. a, P.i, P. g, E. c, F. n, T. d, C. r, B.f

A. a: *Aggregatibacter actinomycetemcomitans*, B. f: *Bacteroides forsythus*, C. r: *Campylobacter rectus*, E. c: *Eikenella corrodens*, P.m: *Peptostreptococcus micros*, P. g: *Porphyromonas gingivalis*, P. i: *Prevotella intermedia*, T. d: *Treponema denticola*.

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33 Chapter

Treatment Plan

- SEQUENCE OF THERAPEUTIC PROCEDURES
- PREFERRED SEQUENCE OF PERIODONTAL THERAPY

INTRODUCTION

The aim of the treatment plan is total treatment, i.e. co-ordination of all treatment procedures for the purpose of creating a well-functioning dentition in a healthy periodontal environment. Treatment plan is the blueprint for the management of a case and establishment of periodontal health. Treatment procedures should be performed in a systematic sequence and should be planned well in advance.

SEQUENCE OF THERAPEUTIC PROCEDURES

Preliminary Phase or Emergency Phase

Treatment of emergencies.

- Dental or periapical abscess.
- Periodontal abscess.

Extraction of hopeless teeth and provisional replacement if needed.

Phase I Therapy (Etiotropic Phase)

- Plaque control.
- Diet control.
- Removal of calculus and root planing.
- Correction of restorative and prosthetic irritational factors.
- Excavation of caries and restorations (Temporary or final).
- Antimicrobial therapy.
- Occlusal therapy.
- Minor orthodontic movement.
- Provisional splinting.

Evaluation of Response to Phase I

Rechecking:

- Pocket depth and gingival inflammation.
- Plaque and calculus, caries.

Phase II Therapy (Surgical Phase)

- Periodontal surgery including placement of implants
- Root canal treatment.

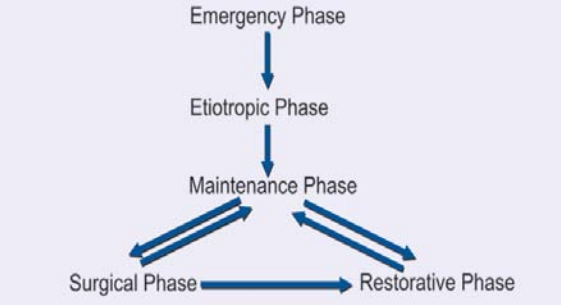
Phase III Therapy (Restorative Phase)

- Final restorations.
- Fixed and removable prosthesis.
- Evaluation of response to restorative procedures.
- Periodontal examination.

Phase IV Therapy (Maintenance Phase)

- Periodic recall visits.
- Checking for plaque and calculus.
- Gingival condition (Pockets, inflammation).
- Occlusion, tooth mobility and other pathologic changes.

PREFERRED SEQUENCE OF PERIODONTAL THERAPY



34 Chapter

Rationale for Periodontal Treatment

- OBJECTIVES OF PERIODONTAL THERAPY
- FACTORS WHICH AFFECT HEALING
  - Local Factors
  - Systemic Factors
- HEALING AFTER PERIODONTAL THERAPY
  - Regeneration
  - Repair
  - Reattachment

OBJECTIVES OF PERIODONTAL THERAPY

If properly performed, periodontal treatment can accomplish the following:

- Eliminate pain.
- Eliminate gingival inflammation.
- Eliminate gingival bleeding.
- Eliminate infection.
- Reduces periodontal pockets and mobility of the teeth.
- Stops pus formation.
- Arrests the destruction of soft tissue and bone.
- Establishes optimal occlusal function.
- Restores tissue destroyed by disease.
- Re-establishes the physiologic gingival contour.
- Prevent the recurrence of disease.
- Reduces tooth loss.

The treatment of periodontal disease is based on the fact that it is caused by bacterial plaque. Hence, the removal of plaque and all factors that favor its accumulation is therefore of primary consideration in local therapy. Systemic therapy may be used as an adjunct to local measures especially if it is indicated in localized juvenile periodontitis and rapidly progressing periodontitis cases. Here the systemic antibiotics are used to completely eliminate the bacteria that invade gingival tissues. The accumulation of plaque can be favored by a variety of local factors such as calculus, overhanging margins of restorations, food impaction. Hence the primary consideration in local therapy should be removal of plaque and all the factors that favor its accumulation. Systemic therapy may be employed as an adjunct to local measures and for specific purposes such as systemic complications from acute infections, post-treatment bacteremia, control of systemic diseases that aggravate the patient's general periodontal condition. Evidence has shown that some nonsteroidal anti-inflammatory drugs such as flurbiprofen and ibuprofen can slow down the development of experimental gingivitis and the studies have shown that it can also inhibit alveolar bone loss in periodontitis.

Another drug that has been shown to reduce bone loss associated with periodontitis, in experimental animals is alendronate, a biphosphonate which is also used to treat Paget's disease and other metabolic diseases. Although some of the systemic conditions associated with periodontal diseases are treated primarily by other than local measures, local therapy is indicated to reduce or prevent the progression of periodontal disease.

FACTORS WHICH AFFECT HEALING

As elsewhere in the body, healing is affected by local and systemic factors.

Local Factors

Healing is delayed due to contamination of micro-organisms; irritation from plaque, food debris, necrotic tissue remnants and trauma from occlusion. Excessive tissue manipulation during treatment, trauma to the tissues can delay healing. In addition, repetitive treatment procedures which affect the orderly cellular activity in the healing process, topically applied cortisone and ionizing radiation can retard healing. Healing is improved by a local increase in temperature, debridement, immobilization of the healing area and pressure on the wound.

Systemic Factors

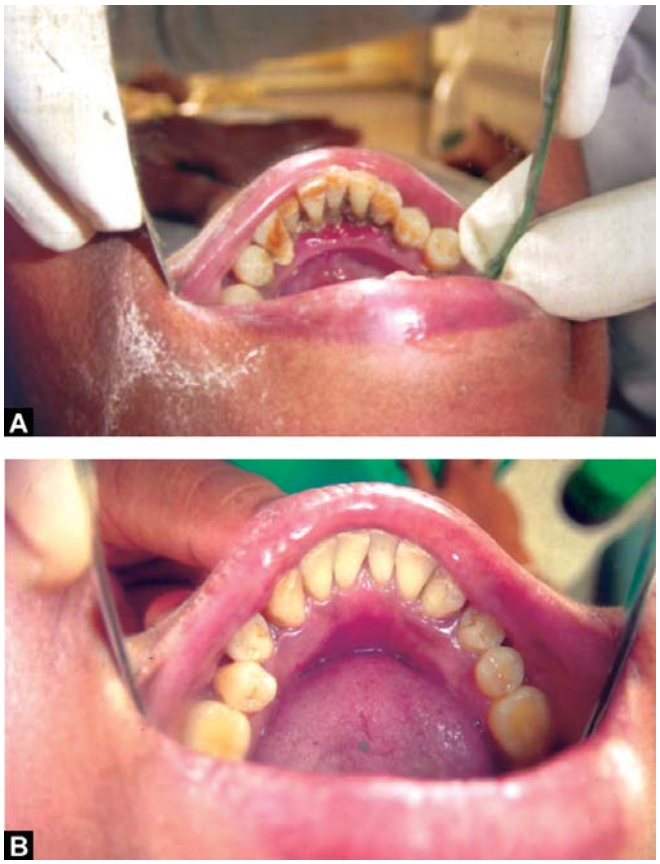
Healing is delayed in:

- Older patients (Because of atherosclerotic vascular changes which results in reduced blood circulation).
- Generalized infections especially in patients with diabetes and other debilitating diseases.
- By insufficient food intake, vitamin C deficiency, deficiency of proteins and other nutrients.
- Increased levels of hormones such as cortisone hinder repair by depressing the inflammatory reaction or inhibiting the growth of fibroblasts, the production of collagen and the formation of endothelial cells.
- Systemic stress, thyroidectomy, testosterone, adrenocorticotropic hormone and large doses of estrogen suppresses the formation of granulation tissue and retard healing.

HEALING AFTER PERIODONTAL THERAPY (FIGS 34.1 AND 34.2)

Regeneration, repair and new attachment are the aspects of healing that have a special bearing on the outcome of periodontal treatment.

a. *Regeneration*: It is the biologic process by which the architecture and function of lost tissues are completely restored by formation of new periodontal ligament, alveolar bone and cementum.
b. *Repair*: It is the healing of tissues without completely restoring the lost tissues.
c. *New attachment*: This is the reunion of connective tissue with a root surface that has been pathologically exposed.
d. *Reattachment*: This is the reunion of connective tissue and a root surface that have been separated by incision or injury.



Figs 34.1A and B: Healing after periodontal therapy

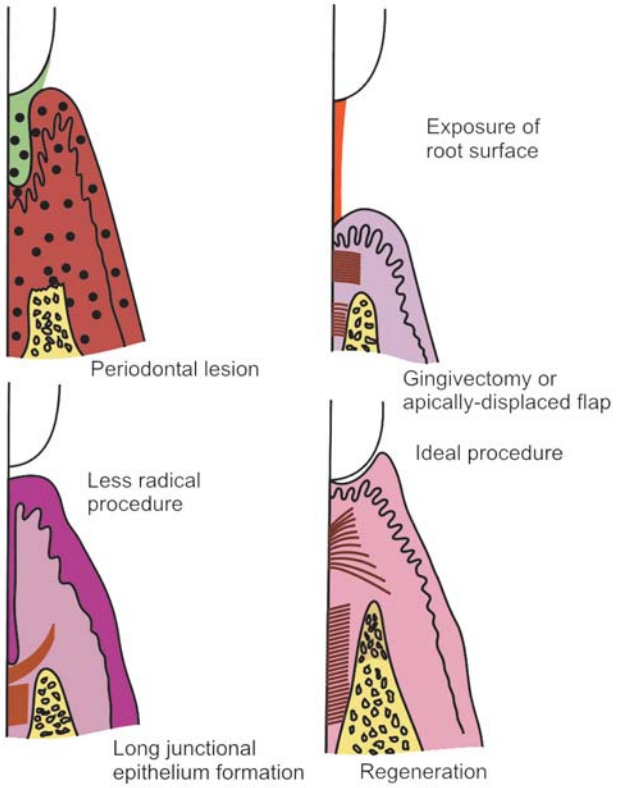


Fig. 34.2: Outcome of periodontal therapy

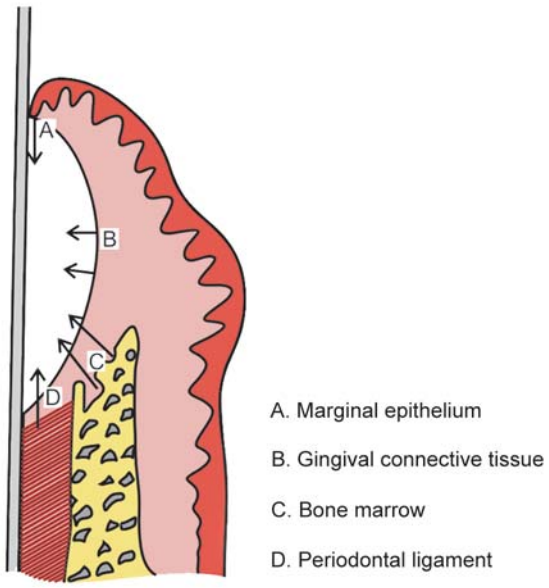


Fig. 34.3: Sources of cells that populate the pocket area

During the healing stages of periodontal pockets, the area is invaded by cells from four different sources: oral epithelium, gingival connective tissue, bone and periodontal ligament (Fig. 34.3).

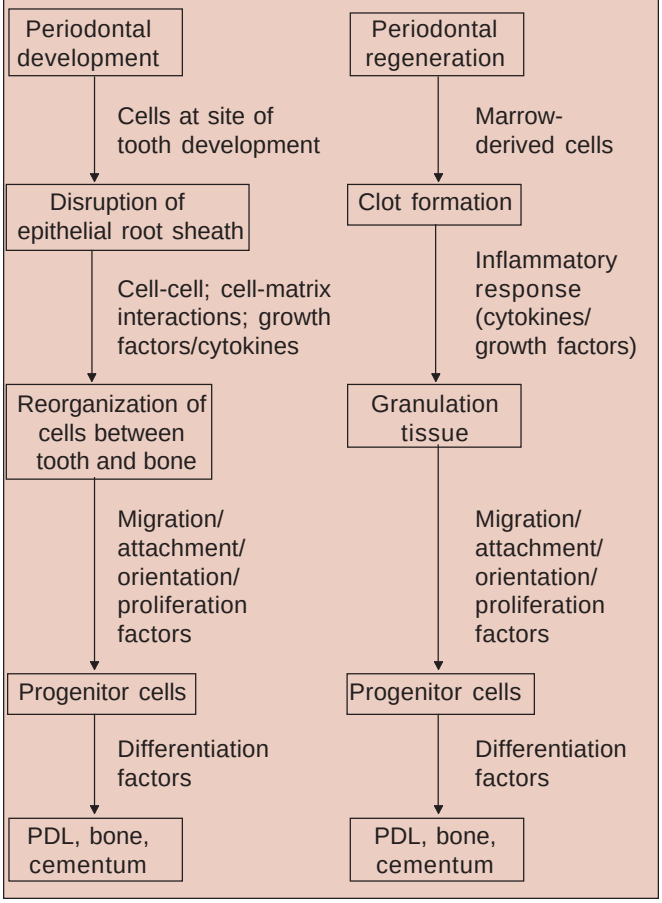
The final outcome of periodontal pocket healing depends on the sequence of events during the healing stages. If the epithelium proliferates along the tooth surface before the cells from other tissues reach the area, the result will be a long junctional epithelium. If the cells from connective tissue populate, the result will be fibers parallel to the tooth surface and remodelling of bone and no attachment to the cementum. If bone cells arrive first, root resorption and ankylosis may occur. Finally, when only cells from the periodontal ligament proliferate coronally, there is new formation of cementum and periodontal ligament. Hence, Melcher pointed out that the regeneration of the periodontal ligament is the key to new attachment because it provides continuity between the alveolar bone and the cementum and also because it contains cells that can synthesize and remodel the three connective tissues of the supporting structures of periodontium.

KEYPOINTS

1. If periodontal treatment is properly performed it can restore the normal health of the periodontal tissues.
2. The periodontal treatment consists of both local and systemic therapy.
3. The primary objective of local therapy is removal of plaque and all those factors that may favour its accumulation.
4. Systemic therapy is used as an adjunct to local therapy and is mainly indicated in localized and generalized aggressive periodontitis.
5. Healing is affected by local and systemic factors. Under local factors, those factors that can delay the healing are excessive tissue manipulation, unnecessary trauma to the tissue, presence of foreign bodies etc. Healing is improved mainly by good debridement and proper immobilization of the wound.
6. Systemic conditions that may have an effect on healing are infections like diabetes, and other debilitating diseases, malnutrition, increased levels of hormones, systemic stress.
7. During healing, the area may be invaded by cells from four different sources:
   a. *Oral epithelium*—results in long junctional epithelium.
   b. Gingival connective tissue results in fibres parallel to root surface.
   c. Bone cells—root resorption and ankylosis.
   d. Only cells from periodontal ligament—results in new attachment.

KNOW MORE ...

Events Occurring during the Development and during the Regeneration of Periodontal Tissues



REVIEW QUESTIONS

1. Describe factors that can affect the healing following periodontal treatment.
2. Define regeneration, repair, new attachment and reattachment.

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35 Chapter

Periodontal Instrumentarium

- PERIODONTAL INSTRUMENTS
  - Diagnostic
  - Surgical
  - Scaling and Curettage
  - Ultrasonic and Sonic
  - Cleansing and Polishing Instruments

PERIODONTAL INSTRUMENTS

Periodontal instruments are designed for specific purposes, such as removing calculus, planing root surfaces, curetting the gingival wall or removing diseased tissue.

Periodontal instrument (Fig. 35.1) is composed of:

a. Blade
b. Shank
c. Handle

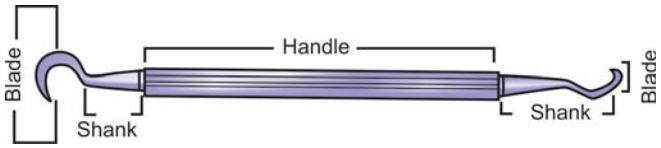


Fig. 35.1: Parts of an instrument

Classification of Periodontal Instruments

Diagnostic Instruments

- Periodontal probes are used to locate, measure and mark pockets.
- Explorers are used to locate calculus deposits and caries.

Scaling, Root Planing, and Curettage Instruments

Scaling and root planing instruments are classified as follows:

a. *For supragingival scaling*:
   - Sickle scalers, cumine universal scaler, posterior Jacquette scaler, Morse scaler, surface scaler, cingulum scaler.

b. *For subgingival scaling:*
   - Hoe scaler, chisel and file scalers are used to remove tenacious subgingival deposits.
   - Curettes are used to plane the root surfaces by removing altered cementum and also, for scraping the soft tissue wall of the pocket.

c. *Sonic and ultrasonic instruments.*

The Periodontal Endoscope

Used to visualize deep pockets and furcations during scaling and root planing.

Cleansing and Polishing Instruments

- Rubber cups, brushes, dental tapes
- Air-powder abrasive system.

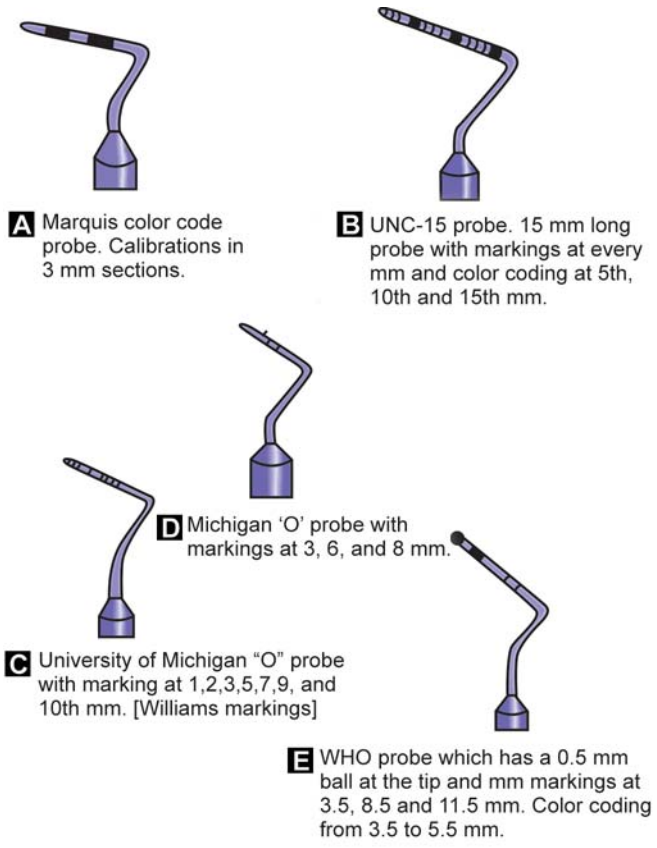
Surgical Instruments

Excisional and incisional instruments, surgical curettes and sickles, periosteal elevators, surgical chisels, Hoes files, scissors and nippers.

Periodontal Probes

A typical probe is a tapered rod-like instrument calibrated in millimeters with a blunt, rounded tip. Periodontal probes are used to measure the depth of the pocket and to determine their configuration.

When measuring a pocket, the probe is inserted with a firm gentle pressure to the base of the pocket. The shank should be aligned with the long axis of the tooth surface to be probed. Furcation areas can be best evaluated with the curved, blunt *Naber's probe* (Figs 35.2 to 35.4).



Figs 35.2A to E: Different types of probes

Types of Periodontal Probes

- Color-coded
- Noncolor-coded

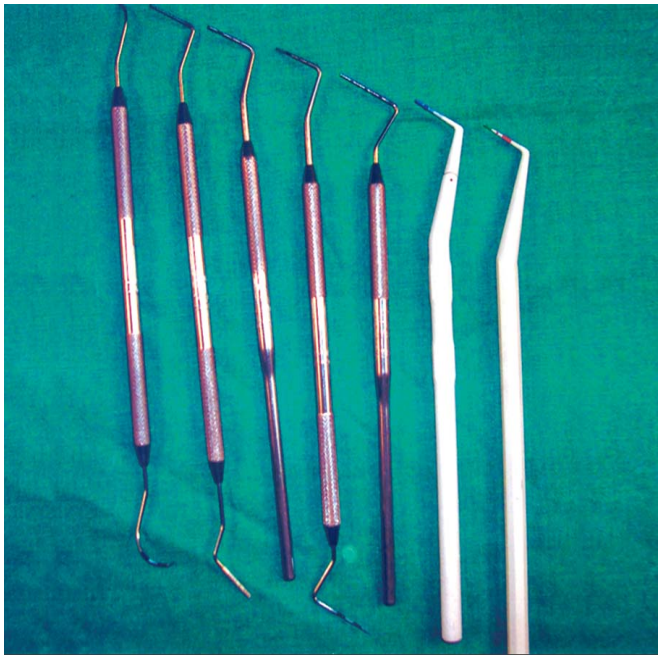


Fig. 35.3: Types of periodontal probes

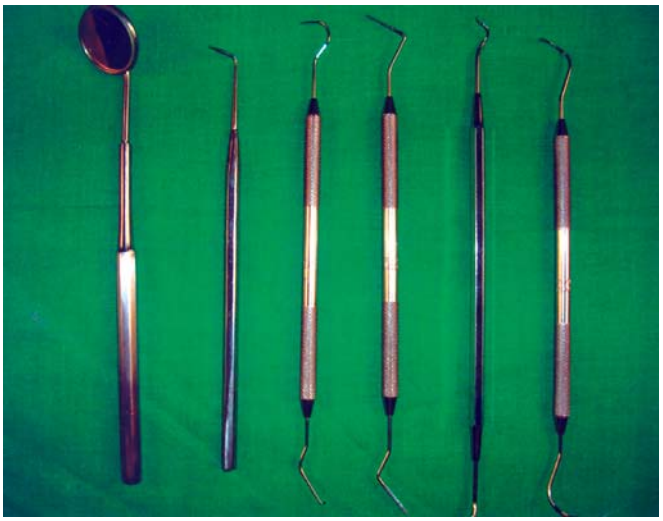


Fig. 35.4: Diagnostic instruments

a. *The Marquis color-coded probe*: The calibrations are in 3 millimeter sections.
b. *The University of North Carolina-15 probe (UNC-15)*: It is a 15 mm long probe with millimeter markings at each millimeter and color coding at the 5th, 10th, and 15th mm.
c. *Williams probe*: Has both color and non-color coding with markings at 1,2,3,5,7,8,9 and 10 mm.

d. The Michigan 'O' probe with Williams marking: At 1, 2, 3, 5, 7, 8, 9, 10 mm (4 and 6 are missing).
e. The Michigan 'O' probe with markings: At 3, 6, and 8 mm.
f. *The WHO probe*: It has a 0.5 mm ball at the tip and millimeter marking at 3.5, 8.5 and 11.5 mm and color coding from 3.5 to 5.5 mm

Explorers

They are used to locate subgingival deposits in various areas, and to check the smoothness of the root surfaces after root planing. Explorers are designed with different shapes and angles for a variety of use.

Scaling and Curettage Instruments (Figs 35.5 to 35.8)

Sickle scalers: Sickle scalers have a flat surface and two cutting edges that converge in a sharply-pointed tip. The arch-shape of the instrument makes the tip so strong that it will not break off during use. They appear triangular in cross-section. The sickle scaler is inserted under ledges of calculus no more than 1 mm below the gingival sulcus. It is used with a pull stroke.

The Morse sickle has a very small, miniature blade; it is useful in the mandibular, anterior area where there is narrow, interproximal space. Sickles with straight shanks are designed for use on anterior teeth and premolars. Sickle scalers with contra-angled shanks adapt to posterior teeth.

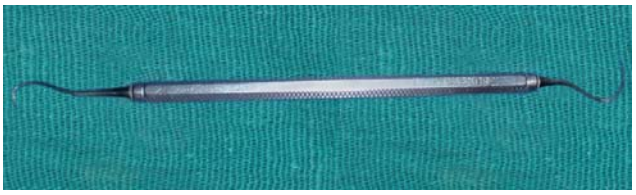
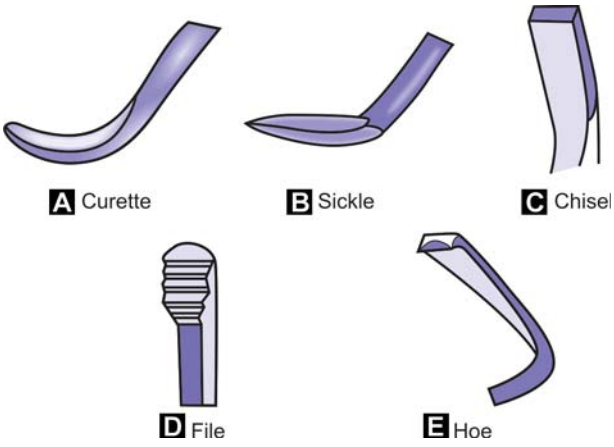


Fig. 35.6: Sickle scaler



Figs 35.7A to E: Basic scaling instruments

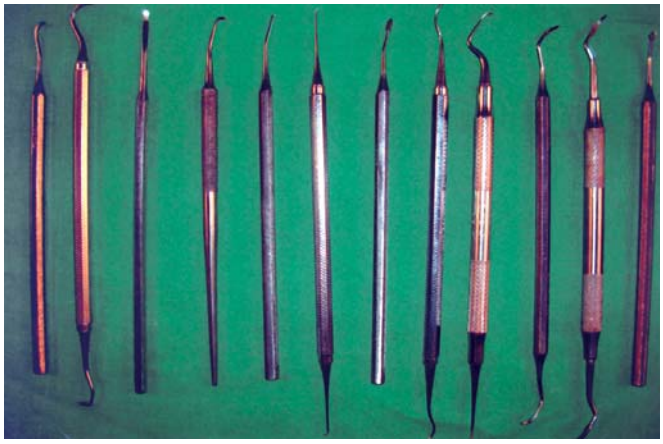
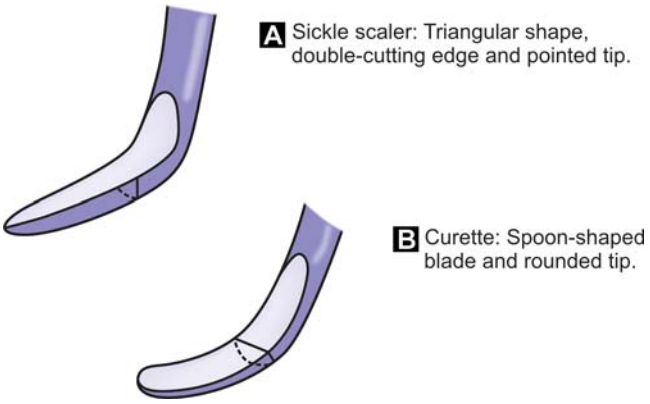


Fig. 35.5: Scaling instruments



Figs 35.8A and B: Basic characteristics of scalers and curettes

Curettes (Figs 35.9 to 35.11): The curette is the instrument of choice for removing deep subgingival calculus, altered cementum, for root planing and for removing the soft tissue lining the periodontal pocket.

Curette can be adapted to provide good access to deep pockets, with minimal soft tissue trauma. There are cutting edges on both sides of the blade. Both single and double-ended curette may be obtained depending upon the preference of the operator.

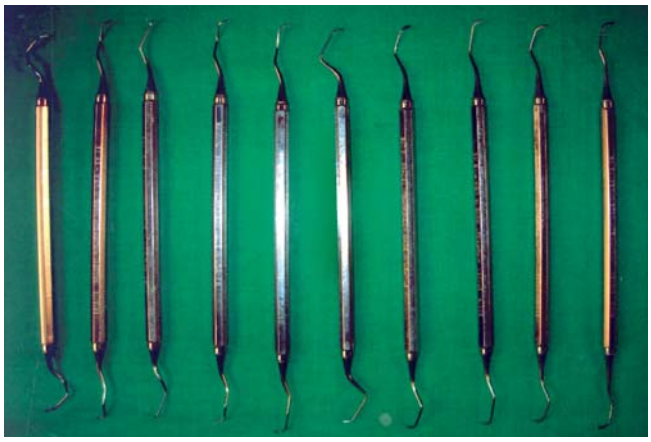
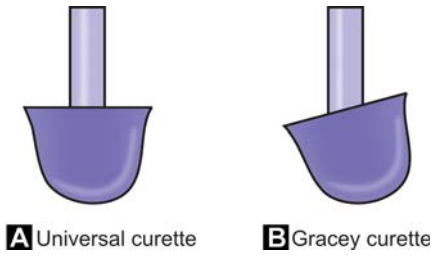


Fig. 35.9: Types of curettes



Figs 35.10A and B: Universal and Gracey curette. Note: offset blade angulation of Gracey curette

Fig. 35.11: Gracey curette blade. Note that the Gracey curette is 50 percent shorter and the tip of the blade is turned upwards as compared to a standard Gracey curette blade

The curved blade and rounded toe of the curette allows the blade to adapt better to the root surface. In cross-section, the blade appears to be semicircular with a convex base.

There are two basic types of curettes.

A. Universal
B. Area-specific

Universal curettes: Universal curettes have cutting edges that may be inserted in most areas of the dentition by altering and adapting the finger rest, fulcrum and hand position of the operator.

The face of the blade of every universal curette is at a 90 degree angle to the lower shank, when seen in cross section from the tip.

Examples of universal curettes: Barnhart curettes # 1-2 and 5-6 and Columbia curettes # 13-14, 2R-2L and 4R-4L. Other universal curettes include Younger-Good # 7-8, the McCalls # 17-18, and Indiana University # 17-18.

Area-specific curettes:

Gracey curettes: They are area-specific curettes, designed and angled to adapt to specific anatomic areas of the dentition. These curettes and their modifications are probably the best instruments for subgingival scaling and root planing because they provide the best adaptation to complex root anatomy.

The term offset blade is used to describe Gracey curettes, because they are angled approximately 60–70 degrees from the lower shank. This unique angulation allows the blade to be inserted in a precise position, necessary for subgingival scaling and root planing, provided that the lower shank is parallel to the long axis of the tooth surface being scaled.

Double-ended Gracey curettes are paired in the following manner:

Gracey # 1-2 and 3-4	:	for anterior teeth
Gracey # 5-6	:	for anterior teeth and premolars
Gracey # 7-8 and 9-10	:	posterior teeth; facial and lingual
Gracey # 11-12	:	posterior teeth; mesial
Gracey # 13-14	:	posterior teeth; distal

Recent additions to Gracey set are:

Gracey # 15-16 and 17-18	:	#15-16 is a modification of #11-12; # 17-18 is a modification of # 13-14. It has a shank elongated by 3 mm.

Distinction between Gracey and universal curettes is given in Table 35.1.

Table 35.1: Distinction between Gracey and universal curettes

	Gracey curette	*Universal curette*
Area of use	Set of many curettes designed for specific areas and surfaces.	One curette designed for all areas and surfaces.
Cutting edge	One cutting edge used, work is done with the outer edge only.	Both cutting edges used, work is done with outer or inner edge.
Curvature	Curved in 2 planes, blade curves up and to the side.	Curved in one plane, blades curves up and not to the side.
Blade angle	Offset blade, face of blade beveled at 60 degrees to the shank.	Not offset, face of blade beveled at 90 degrees to the shank.

Extended Shank Curettes or After Five Curettes

Hu Friedy After Five Curettes are modifications of the standard Gracey curette design. The shank is 3 mm longer, allowing extension into deeper periodontal pockets of 5 mm or more, other features include a thinned-blade for smoother subgingival insertion or reduced tissue distention with a large diameter, tapered shank.

All the standard Gracey numbers except # 9-10 are available in the After Five series.

Mini-bladed curettes: They are modifications of After Five Curettes. The shorter blade allows easier insertion and adaptation in deep, narrow pockets, furcations, developmental grooves, line angles, and deep, tight, facial, lingual, or palatal pockets. As with the After Fives, the Mini Fives are available in all standard Gracey number except for the # 9-10.

Gracey curvettes: They are another set of four mini-bladed curettes.

Sub-0 and # 1-2—anterior and premolars
11-12—posterior mesial surfaces.
13-14—posterior distal surfaces.

The blade length of these instruments is 50 percent, shorter than that of conventional Gracey curette and the blade is curved slightly upward. This curvature allows the curettes to adapt, more closely to the tooth surfaces.

Langer and mini Langer curettes: This set of 3 curettes combines the shank design of the standard Gracey # 5-6, 11-12, and 13-14 curettes with a universal blade honed at 90 degrees rather than the offset blade of the Gracey curette. Hence, these curettes offer a blend of both Gracey and universal curette and can be adapted both on the mesial and distal surfaces without changing instruments.

Schwartz periotrievers: They are a set of two double-ended, highly-magnetized instruments designed for the retrieval of broken instrument tips from the periodontal pocket.

Plastic instruments for implants: It is imperative that plastic rather than metal instruments be used, to avoid scarring and permanent damage to the implants.

Hoe scalers: They are used for scaling ledges or rings of calculus. The blade is bent at a 99 degree angle; the cutting edge is beveled at 45 degrees. The Hoe scalers are used in the following manner:

a. The blade is inserted to the base of the periodontal pocket, so that it makes a two point contact with the tooth. This stabilizes the instrument and prevents nicking of the tooth.
b. The instrument is activated with a firm pull stroke towards the crown, with every effort being made to preserve the two point contact with the tooth.

McCalls Hoe scalers # 3, 4, 5, 6, 7 and 8 are a set of six Hoe scalers designed to provide access to all the tooth surfaces.

Files: They have a series of blades on a base. Their primary function is to fracture or crush tenacious calculus. Files can easily gouge and roughen root surfaces when used improperly. Therefore they are not suitable for fine scaling and root planing. They are sometimes used for removing overhanging margins of dental restorations.

Chisel scalers: Usually used in the proximal surfaces of anterior teeth (too closely spaced). It is a double-ended instrument with a curved shank at one end and a straight shank at the other. The instrument is activated with a push motion.

Quétin Furcation Curettes: These are the curettes specifically designed to fit into the roof or floor of the furcation. These curettes are nothing but hoe scalers with a shallow, half moon radius and the tip is curved in such a way that it also fits into the developmental depressions. They are available in two widths, namely the B-L (buccolingual) and M-D (mesiodistal). These instruments can remove burnished calculus from furcation areas where even the mini-bladed Gracey curettes are often too large to gain access.

Ultrasonic and Sonic Instruments (Fig. 35.12)

Used for removing plaque, scaling, curetting and removing stains

Two types of ultrasonic units are:

- *Magnetostrictive*: Vibration of the tip is elliptical; hence all the sides can be used.
- *Piezoelectric*: Pattern of vibration of the tip is linear; only two sides of the tip are active.

Ultrasonic vibrations range from 20,000 to 45,000 cycles/second. They operate in a wet field and have attached water outlets.

Dental Endoscope

It is introduced for use subgingivally, in the diagnosis, treatment of periodontal diseases. Produced by Dentalview Inc. and called as the perioscopy system.

Fig. 35.12: Ultrasonic scaler (EMS scaler)

- It consists of reusable fiberoptic endoscope, over which there is a sterile sheath. The fiber optic endoscope fits onto the periodontal probes and ultrasonic instruments that have been designed to accept it.
- The sheath delivers water for irrigation that flushes the pocket while the endoscope is in use, and it keeps the field clear.
- This device allows clear visualization, subgingivally, in deep pockets and in furcations. It enables the operator to detect the presence and location of subgingival deposits and guides the operator in their thorough removal.
- Using this device it is possible to achieve levels of root debridement and cleanliness that are much more difficult to produce without it.

The EVA system: They are most efficient and least traumatic instruments, for correcting overhanging or overcontoured proximal alloy and resin restorations.

Cleansing and Polishing Instruments

Rubber cups: They consist of a rubber shell with or without configuration in the hollow interiors.

They are used in the hand piece with special prophylaxis angle. A good cleansing and polishing paste that contains fluoride should be used.

Bristle brushes: Available in wheel and cup shapes, used in hand piece with a polishing paste

Dental tape: It is used with a polishing paste and is used for polishing proximal surfaces that are inaccessible to other polishing instruments.

Air powder polishing: A specially designed hand piece that delivers air powdered slurry of warm water and sodium bicarbonate, this instrument is called prophy-jet. Effective for the removal of extrinsic stains and soft deposits.

Disadvantages: Tooth substance can be lost, damage to gingival tissue is transient and insignificant clinically, but amalgam restorations, composite resins and cements can be roughened.

Contraindications: Patients with medical histories of respiratory illness, hypertension, and patients on

medications affecting the electrolyte balance are contraindicated.

Surgical Instruments (Fig. 35.13)

1. *Excisional and incisional instruments*:
   - Periodontal knives (Gingivectomy knives): Example Kirkland knife (Fig. 35.14).
   - Interdental knives (Fig. 35.15): Example, Orban knife # 1-2, Merrifield knife # 1, 2, 3 and 4.
   - Surgical blades: Example, # 12D, 15 and 15C.
   - Electrosurgery techniques and instrumentation (Fig. 35.16):
     - Electrosection used for incisions, excisions and tissue planing.
     - Electrocoagulation, coagulation or hemorrhage control.
     - Electrofulguration not in general use in dentistry.
     - Electrodessication not in general use in dentistry.
2. *Surgical curettes and sickles*: Required for the removal of granulation tissue, fibrous interdental tissue, and tenacious subgingival deposits.
   Examples:
   - Kramer curettes # 1, 2, 3 and Langer curettes.
   - Kirkland surgical instruments.
   - Ball scaler # B_2-B_3.
3. *Periosteal elevators*: Necessary to reflect and move the flap after the incision has been made for flap surgery.
   Example: Goldman Fox #14.
4. *Surgical chisels and hoes*: They are used during periodontal surgery for removing and reshaping bone. Chisels are used with a push stroke whereas surgical hoes are used with a pull stroke.
   Example:
   - Ochsenbein #1-2, chisel.
   - Rhodes chisel.
5. *Surgical files*: They are used primarily to smoothen rough, bony, ledges and to remove all areas of necrotic bone.
   Example: Schluger and Sugarman files.

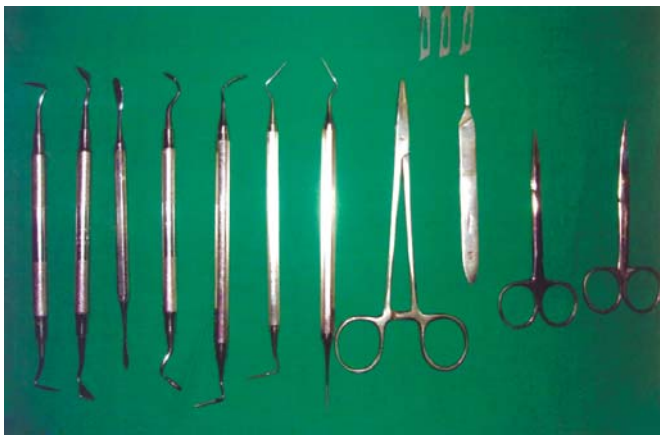


Fig. 35.13: Surgical instruments

Fig. 35.14: Kirkland knife

Fig. 35.15: Orban Interdental knife

Fig. 35.16: Electrosurgery unit

6. *Scissors and nippers*: Used for removing tabs of tissue during gingivectomy, trimming the margins of flaps, enlarging incisions in periodontal abscesses and removing muscle attachments in mucogingival surgery.
   Example: Goldman – Fox # 16 scissors.
7. *Needle holders*: They are used to suture the flap at the desired position.
   Example: Castroviejo needle holder.

KNOW MORE ...

Advantages and Disadvantages of Hand and Ultrasonic Instruments

	Advantages	*Disadvantages*
Hand instruments	• Superior tactile sensation • Good access • Good adaptation • No aerosol production • No heat development	Correct angulation is mandatory Frequent sharpening required Considerable working force is required Tiring for operator Negative time factor
Ultrasonic instruments	• Instrumentation without pressure • Highly accessible to difficult to reach areas • Disruption of biofilm by cavitation • Minimal soft tissue damage • Requires less time • Pocket irrigation is possible • No sharpening of tips • Better patient acceptance • Less tiring for the operator	Poorer tactile sensation Aerosols are highly contaminated Not all hand pieces can be autoclaved Possible risk for patients with pace makers Contraindicated in infectious patients

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36 Chapter

Principles of Periodontal Instrumentation including Scaling and Root Planing

- POSITIONING OF PATIENT AND OPERATOR
- VISIBILITY, ILLUMINATION AND RETRACTION
- CONDITION OF INSTRUMENTS
- MAINTAINING A CLEAN FIELD
- INSTRUMENT STABILIZATION
- INSTRUMENT ACTIVATION
- PRINCIPLES OF SCALING AND ROOT PLANING
- ULTRASONIC INSTRUMENTS
- AEROSOL PRODUCTION

INTRODUCTION

Effective instrumentation is governed by a number of general principles that are common to all periodontal instruments. Proper position of the patient and the operator, illumination and retraction for optimal visibility and sharp instruments are the fundamental prerequisites.

ACCESSIBILITY (POSITIONING OF PATIENT AND OPERATOR)

It facilitates thoroughness of instrumentation. The position of the patient and operator should provide maximal accessibility to the area of operation. Inadequate accessibility impedes thorough instrumentation, prematurely tires the operator, and diminishes his or her effectiveness.

Neutral Seated Position for the Clinician

- Forearm parallel to the floor.
- Weight evenly balanced.
- Thighs parallel to the floor.
- Hip angle of 90 degrees.
- Seat height positioned low enough so that the heels of your feet touch the floor.
- When working from clock positions 9-12:00, spread feet apart so that your legs and the chair base form a tripod which creates a stable position.
- Avoid positioning your legs under the back of the patient's chair.
- Back straight and the head erect.

Patient's Position

The patient should be in a supine position and placed in such a way that the mouth is close to the resting elbow of the clinician.

Body: The patient's heels should be slightly higher than the tip of his or her nose. The back of the chair should be nearly parallel to the floor for maxillary treatment areas.

The chair back may be raised slightly for mandibular treatment areas.

Head: The foremost of the patient's head should be even with the upper edge of the head rest.

- For mandibular areas—chin down position.
- Maxillary areas—chin up position.

Head rest: If the head rest is adjustable, it should be raised or lowered, so that the patient's neck and head are aligned with the torso.

There are four basic clinical positions for the right-handed and left-handed clinician (Table 36.1).

VISIBILITY, ILLUMINATION AND RETRACTION (FIGS 36.1A AND B)

Whenever possible, direct vision with direct illumination from the dental light is most desirable. If this is not possible, indirect vision may be obtained by using a mouth mirror to reflect light where it is needed. Indirect vision and indirect illumination are often used simultaneously.

Dental Mirror

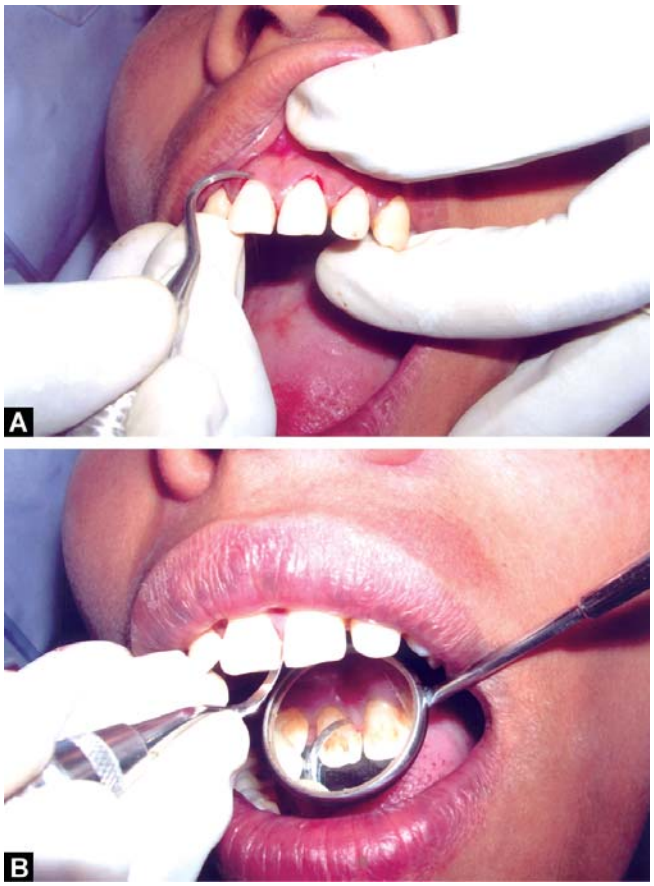
It is a hand instrument which has a reflecting mirrored surface used to view tooth surfaces that cannot be seen by direct vision.

Various Types of Mirror Surfaces

- *Front surface*
  - Produces clean clear image with no distortion
  - Good image quality
  - Easily scratchable
- *Concave surface*
  - Image is magnified
  - Distortion of the image.
- *Plane (Flat surface)*
  - Produces double image
  - Double image is distracting.

Table 36.1: Clinical positions for right and left-handed clinicians

Right-handed clinician	*Left-handed clinician*
• 7 o' clock position to the front of the patient's head. • 9 o' clock position to the side of the patient's head. • 10 to 11 o' clock, to the back of the patient's head. • 12 o' clock position, directly behind the patient's head.	• 5 o' clock position, to the front of the patient's head. • 3 o' clock position, to the side of the patient's head. • 2 to 10 o' clock position, to the back of the patient's head. • 12 o' clock position, directly behind the patient's head.



Figs 36.1A and B: Direct and indirect vision

Various Uses of a Dental Mirror

1. Indirect vision
2. Retraction
3. Indirect illumination
4. Transillumination.

Transillumination: When transilluminating a tooth, the mirror is used to reflect light through the tooth surface.

The transilluminated-tooth almost will appear to glow. It is effective only with anterior teeth because they are thin enough to allow the light to pass through them.

Procedure:

Step 1 : Position yourself in 12 o' clock position.

Step 2 : Using a modified pen grasp, hold the mirror in the non-dominant hand. Bring the arm up and over the patient's face. Gently rest your ring finger on the side of the patient's lip or cheek.

Step 3 : Hold the dental mirror behind the central incisors so that the reflecting surface is parallel to the lingual surface. Position the unit light so that the light beam shines on the dental mirror at a 90 degree angle to the mirrors reflecting surfaces.

Step 4 : Properly-positioned light and the mirror will result in glow.

Retraction: It provides visibility, accessibility and illumination. The following methods are effective for retraction:

1. Use of the mirror to deflect the cheek while the fingers of the non-operating hand retract the lips and protect the angle of the mouth from irritation by the mirror handle.
2. Use of the mirror alone to retract the lips and cheek.
3. Use of fingers of the non-operating hand to retract the lips.
4. Use of the mirror to retract the tongue.
5. Combination of the preceding methods.

While retracting, care should be taken to avoid irritation to the angles of the mouth.

CONDITION OF INSTRUMENTS (SHARPNESS)

Prior to any instrumentation, all instruments should be inspected to make sure that they are clean, sterile and in good condition. The working ends of pointed or bladed instruments must be sharp to be effective.

Advantages of Sharpness

1. Easier calculus removal.
2. Improved stroke control.
3. Reduced number of strokes.
4. Increased patient comfort.
5. Reduced clinician fatigue.

Ideally, it is best to sharpen your instruments after autoclaving and then reautoclave them prior to patient treatment. Dull instruments may lead to incomplete calculus removal and unnecessary trauma because of excess force applied.

MAINTAINING A CLEAN FIELD

Despite good visibility, illumination and retraction, instrumentation can be hampered if the operative field is obscured by saliva, blood and debris. Adequate suction is essential and can be achieved with a saliva ejector or, an aspirator.

Blood and debris can be removed from the operative field with suction and by wiping or blotting with gauze squares. The operative field should also be flushed occasionally with water. Compressed air and gauze square can be used to facilitate visual inspection of tooth surfaces just below the gingival margin during instrumentation. Retractable tissue can also be deflected away from the tooth by gently packing the edge of gauze square into the pocket with the back of a curette.

INSTRUMENT STABILIZATION

Stability of the instrument and the hand is the primary requisite for controlled-instrumentation. Stability and control is essential for effective instrumentation and to avoid injury to the patient or clinician. The two factors that provide stability are, instrument grasp and finger rest (Table 36.2).

Instrument Grasp (Figs 36.2 and 36.3)

A proper grasp is essential for precise control of movements made during periodontal instrumentation. The most effective and stable grasp for all periodontal instruments is the modified pen grasp. This grasp allows precise control of the working end, permits a wide range of movements and facilitates good tactile conduction.

The palm and thumb grasp is useful for stabilizing instruments during sharpening and for manipulating air and water syringes (Fig. 36.4).

Table 36.2: Correct finger placement

Digit	*Recommended position and function*
Thumb and index	The finger pads rest opposite to each other at or near the junction of the handle and the shank. They do not overlap and a tiny space exists between them. The instrument is held in a relaxed manner. The index finger and thumb curve outward from the handle in a C-shape. The main function of these digits is to hold the instrument.
Middle	One side of the finger pad rests lightly on the instrument shank. The other side of the finger pad rests against the ring finger. It helps to guide the working end and also feel the vibration.
Ring	Finger tip balances firmly on the tooth to support the weight of the hand and instrument. The finger is held straight and upright to act as a strong support beam for the hand.
Little	It should be held in a relaxed manner with no function.

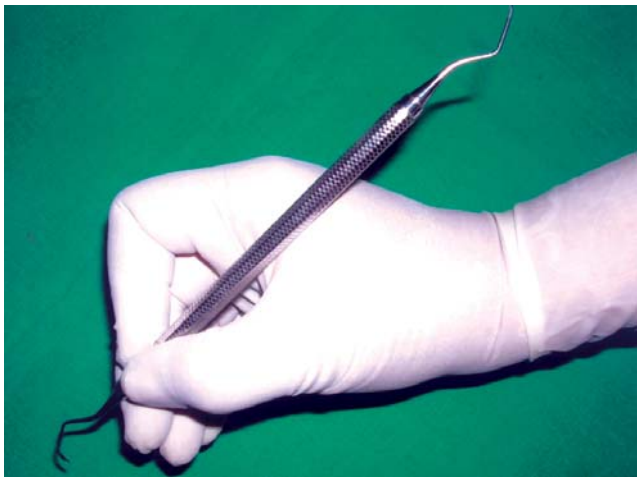


Fig. 36.2: Pen grasp

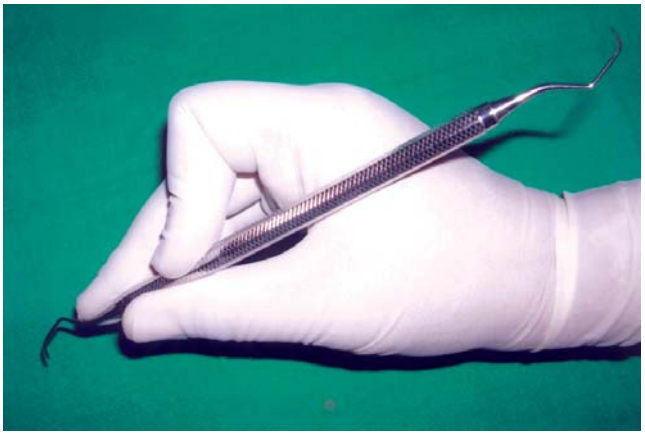


Fig. 36.3: Modified pen grasp

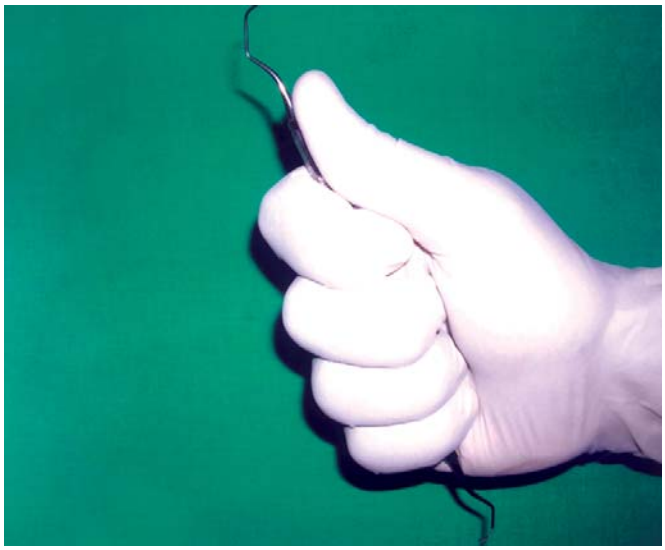


Fig. 36.4: Palm and thumb grasp

Finger Rest

The finger rest serves to stabilize the hand and the instrument by providing a firm fulcrum, as movements are made to activate the instrument. A good finger rest prevents injury and laceration of the gingival and surrounding tissues. The ring finger is preferred by most clinicians for the finger rest. Maximal control is achieved when the middle finger is kept between the instrument shank and the fourth finger. This built-up fulcrum is an integral part of the wrist-forearm action that activates the powerful working stroke for calculus removal.

Finger rests may be generally classified as intraoral finger rests or extraoral fulcrums.

Standard Intraoral Finger Rest

The finger rests on a stable tooth surface immediately adjacent to the working area.

Advantages

- Provides the most stable, secured support for the hand.
- Provides leverage and power for instrumentation.
- Provides excellent tactile transfer to the fingers.
- Permits precise stroke control.
- Allows forceful stroke pressure with the least amount of stress to the hand and fingers.
- Decreases the likelihood of injury to the patient.

Disadvantages

- May not be practical for use in edentulous areas.
- May be difficult to obtain parallelism of the lower shank to the tooth surface for accessing deep pockets.

Advanced Intraoral Finger Rests

a. *Modified intraoral fulcrum*: It is achieved by combining an altered modified pen grasp with a standard intraoral fulcrum. It is useful while instrumenting the maxillary teeth. It alters the point of contact between the middle and ring fingers in the grasp.

Advantages

- Provides good stable support for the clinician's hand.
- Provides leverage, strength and good stroke control.
- Provides good tactile sensitivity to clinician's finger.
- Improves access to deep pockets on maxillary teeth and facilitates parallelism of lower shank to proximal root surfaces.

Disadvantage

Requires more muscle control.

b. *Piggy-backed fulcrum*: The middle finger rests on top of the ring finger.

Advantages

- Improved access to mandibular posterior aspects away from the clinician.
- Enhances the whole hand working together as a unit.

Disadvantage

In patients with limited opening it cannot be used.

c. *Cross-arch fulcrum*: It is accomplished by resting the ring finger on a tooth on the opposite side of the arch from the teeth being instrumented.

Advantage

Allows improved access to the lingual aspect of mandibular posterior teeth.

Disadvantage

Decreases tactile sensitivity and makes strokes difficult.

d. *Opposite arch fulcrum*: It is accomplished by resting the ring finger on the opposite arch.

Advantage

Facilitates access to deep pockets.

Disadvantages

- Decreases tactile information
- Uncomfortable for patients with TMJ problems.

e. *Finger-on-finger fulcrum*: It is accomplished by resting the ring finger on the index finger.

Advantages

- Provides stable rest to fulcrum finger.
- Improves access to deep pockets.

Disadvantage

Non-dominant hand cannot be used for retraction or to hold the mirror.

f. *Basic extraoral fulcrums*: They are essential for effective instrumentation of some aspects of maxillary posterior teeth.

I. *Knuckle-rest technique or palm up technique*: The clinician rests the Knuckle against the patients chin or cheek.

II. *Chin-cup technique or palm down technique*: The clinician cups the patients chin with the palm of the hand.

Advantage

Facilitates instrumentation of the proximal root surfaces of maxillary molars.

Disadvantages

- Least effective of all fulcrum techniques.
- Stroke control is more difficult and decreases tactile information.

INSTRUMENT ACTIVATION

- Adaptation
- Angulation
- Lateral pressure
- Strokes

Adaptation

It refers to the manner in which the working end of a periodontal instrument is placed against the surface of a tooth. The object of adaptation is to make the working end of the instrument conform to the contour of the tooth surface.

The cutting edge has three imaginary sections:

1. Leading third—used more often during instrumentation.
2. Middle third.
3. Heel third.

Precise adaptation must be maintained with all instruments to avoid trauma to the soft tissues and root surfaces and to ensure maximum effectiveness of instrumentation. Bladed instruments such as curette and sharp pointed instruments such as explorers are more difficult to adapt.

Angulation (Fig. 36.5)

It refers to the angle between the face of a bladed instrument and the tooth surface.

1. For insertion beneath the gingival margin, the face to tooth surface angulation should be an angle between 0 to 40 degrees.
2. For calculus removal, angulation should be between 45 to 90 degrees. The exact blade angulation depends on the amount and nature of calculus, the procedure being performed and condition of tissue during scaling or root planing, with angulation of less than 45 degrees, the cutting edge will slide over the calculus smoothening or burnishing it. When gingival curettage is indicated, angulation greater than 90 degrees is deliberately established.

Lateral Pressure

It refers to the pressure created when force is applied against the surface of a tooth with the cutting edge of a bladed instrument. Exact amount of pressure depends upon the procedure performed. It may be firm, moderate or light when insufficient lateral pressure is applied rough ledges or lumps may be shaved to thin, smooth sheets of burnished calculus.

Repeated application of excessively heavy strokes will nick or gouge the root surface. The careful application of varied and controlled amounts of lateral pressure during instrumentation is an integral part of effective scaling and root planing techniques.

Strokes (Fig. 36.6)

There are four types of strokes:

1. Placement stroke.
2. Exploratory stroke or assessment stroke.
3. Scaling stroke.
4. Root planing stroke.

The placement stroke is used to position the working end of an instrument apical to a calculus deposit or at the base of a sulcus or pocket. Characteristics of strokes are described in Table 36.3.

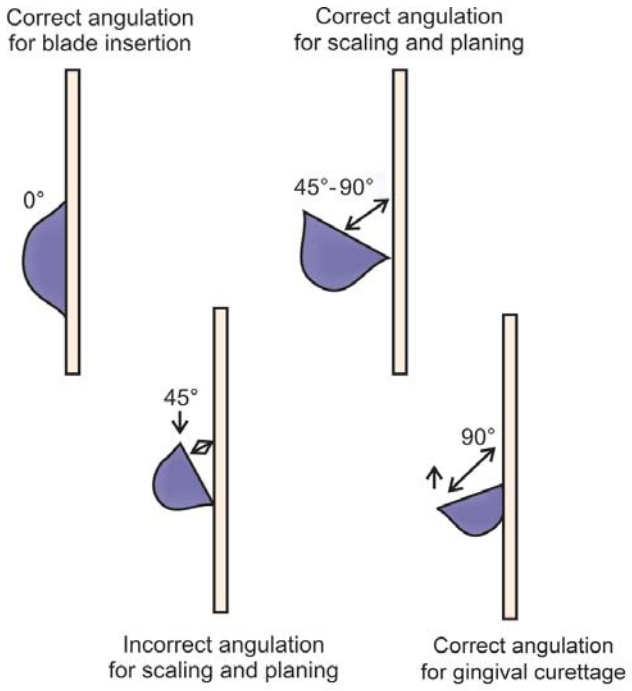


Fig. 36.5: Blade angulations

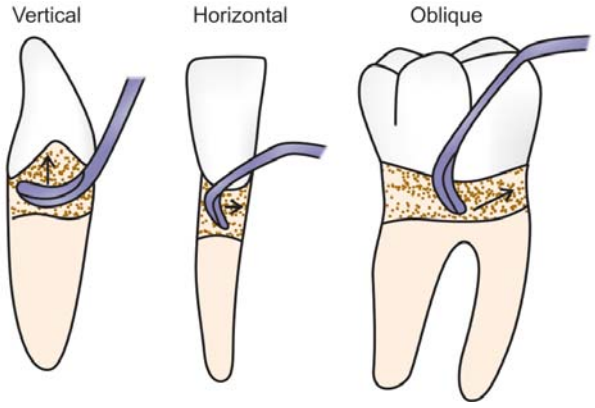


Fig. 36.6: Basic stroke directions

Table 36.3: Characteristics of strokes

	Assessment stroke	*Calculus removal/ scaling stroke*	*Root planing stroke*
Purpose	Assess tooth–anatomy. Level of attachment. Detect calculus and other plaque retentive factors.	Remove calculus deposits.	Remove residual calculus, bacterial plaque and by-products.
Used with	Probes/explorers, curettes.	Sickle scalers, curettes, files.	Curettes
Insertion	0 to 40 degrees	0 to 40 degrees	0 to 40 degrees
Working angulation	50 to 70 degrees	70 to 80 degrees	60 to 70 degrees
Lateral pressure	Contacts tooth surface, but no pressure applied.	Moderate to firm scraping.	Light to moderate.
Character	Fluid stroke of moderate length.	Powerful strokes short in length.	Lighter strokes of moderate length.
Direction	Vertical, oblique, horizontal.	Vertical, oblique, horizontal.	Vertical, oblique, horizontal.
Number	Many, covering entire root surface.	Limited, to area where needed.	Many, covering entire root surface.

Stroke Direction

Instrument strokes are initiated using a pull stroke in a coronal direction away from the junctional epithelium.

Pull strokes may be made in vertical, oblique or horizontal directions.

Vertical strokes : Facial, lingual, proximal surfaces of anterior teeth, mesial and distal surfaces of posterior teeth.

Oblique strokes : Facial and lingual surfaces of anterior and posterior teeth.

Horizontal strokes or circumferential strokes : Line angles of posterior teeth, furcation areas.

PRINCIPLES OF SCALING AND ROOT PLANING

Scaling

This is the process by which plaque and calculus are removed from both supragingival and subgingival tooth surfaces.

Root Planing

This is the process by which residual embedded calculus and portions of cementum are removed from the roots to produce a smooth, hard, clean surface.

The prime objective of scaling and root planing is to restore gingival health by completely removing the tooth surface elements that provoke gingival inflammation.

Principles of Curettes

- Universal
- Gracey

Universal curettes: The working ends of the universal curettes are designed in pairs so that all surfaces of the teeth can be treated with one-double ended instrument or a matched pairs of single-ended instruments.

In any given quadrant, one end of universal curette will adapt to the mesial surface and the other end to the distal surface. The end that adapts to the mesial surface of the facial aspects also adapts to the distal surface on the lingual aspect and vice versa. When adapting the universal curette blade, as much of the cutting edge should be in contact with the tooth surface. Although the entire cutting edge should contact the tooth, pressure should be concentrated on the lower-third of blade during scaling stroke. During root planing stroke, however, lateral pressure should be distributed evenly along the cutting edge.

In the posterior teeth, a single working end can be used to treat both mesial and distal surfaces by using both of its cutting edges.

Gracey curettes

1. Determine the cutting edge by visually inspecting the blade and confirmed by lightly adapting the chosen

cutting edge to the tooth with the lower shank parallel to the surface of the tooth.
2. Make sure the lower shank is parallel to the surface to be instrumented.
3. When using intraoral finger rests, keep the fourth and middle fingers together in a built-up fulcrum for maximum control and wrist-arm action.
4. Use extraoral fulcrum or mandibular finger rests for optimal angulation when working on the maxillary posterior teeth.
5. Concentrate on using the cutting edge (lower) for calculus removal.
6. Allow the wrist and forearm to carry the burden of the stroke, rather than flexing the fingers.
7. Roll the handle slightly between the thumb and fingers to keep the blade adapted as the working end is advanced around line angles and into concavities.
8. Module lateral pressure from firm to moderate to light depending on the nature of calculus.

Supragingival Scaling Technique

Supragingival calculus is less tenacious and less calcified than subgingival calculus. Scaling strokes are not confirmed by surrounding tissues.

Sickles, curettes and ultrasonic and sonic instruments are most commonly used for the removal of supragingival calculus. Hoes and chisels are less frequently used. To perform supragingival scaling, the sickle is held with modified pen grasp and a firm finger rest is established on the teeth adjacent to the working area. The blade is adapted at an angulation of slightly less than 90 degrees to the surface being scaled. The cutting edge should engage the apical margin of supragingival calculus while short, powerful, overlapping scaling strokes are activated coronally in a vertical or oblique direction.

Subgingival Scaling and Root Planing Technique

Subgingival calculus is usually harder than supragingival calculus and is often locked into root irregularities, making it more tenacious.

The direction and length of the strokes are limited by adjacent pocket wall. The curette is preferred by most clinicians for subgingival scaling and root planing because of the advantages afforded by its design. Hoes, files and ultrasonic instruments are also used for subgingival scaling of heavy calculus, but are more hazardous than the curette in terms of trauma to the root surface and the surrounding tissues.

The curette is held with a modified pen grasp and a stable finger rest is established. The correct cutting edge is slightly adapted to the tooth, with the lower shank kept parallel to the tooth surface. The working angulation is established and calculus is removed by a series of controlled overlapping, short powerful strokes primarily utilizing wrist arm motion. Longer, lighter root planing strokes are then activated with less lateral pressure until the root surface is completely smooth and hard. Scaling and root planing strokes should be confirmed to the position of the tooth where calculus or altered cementum is found. This zone is known as *instrumentation zone*.

Evaluation of scaling and root planing: Although smoothness is the criteria by which scaling and root planing are evaluated, the ultimate evaluation is based on tissue response. Clinical evaluation should not be conducted earlier than 2 weeks postoperatively. Re-epithelialization of wound created during instrumentation takes 1 to 2 weeks. Any gingival bleeding on probing noted after this interval is due to persistent inflammation produced by residual deposits. Positive clinical changes after instrumentation often continues for weeks or months. So longer period of evaluation is indicated deciding whether to intervene to further instrumentation or surgery.

ULTRASONIC INSTRUMENTS

They use a water-cooled instrument tip, vibrating at high frequency, to remove supragingival and subgingival calculus deposits from the tooth and bacterial plaque from periodontal pocket. The two categories of mechanized instruments are ultrasonic and sonic hand piece. Ultrasonic units are comprised of electric generator, a handpiece and

interchangeable instrument tip. Ultrasonic devices work by converting electrical current to mechanical energy in the form of high frequency vibration of instrument tip. They operate at frequencies 18,000 to 50,000 cycles/sec. Two types of ultrasonic units are magnetostrictive and piezoelectric units.

Ultrasonic instrument tip must be cooled by fluid to prevent overheating of the vibrating instrument tip. They have been shown to be as effective as hand instruments in subgingival calculus removal, removal of attached and unattached subgingival plaque, removal of toxins from root surfaces, and in reduction and maintenance of pocket depth.

The water lavage has three benefits on the treatment site.

- *Flushing action*–flushes calculus, blood, bacteria, plaque from treatment site.
- Cavitation.
- Acoustic streaming.

As the water exits from instrument tip, it forms a spray of tiny bubbles that collapses and releases shock waves in a process known as *cavitation*. It causes lysis of bacterial cell wall.

The continuous stream of water produces tremendous pressure within the confined space of periodontal pocket. This effect is called *acoustic streaming*. Bacteria, Gram-negative rods are sensitive to acoustic streaming.

Equipment and Armamentarium for Ultrasonic and Sonic Instrumentation

Unit selection:	When purchasing equipment, look for a unit with adjustable power and a selection of varied instrument tip designs.
Instrument selection:	Larger, stronger tip should be used for heavy calculus removal, thin tip should be used on light deposits, de-plaquing, and endotoxin removal.
Infection control:	Ultrasonic and sonic instruments produce a high level of aerosol contamination in dental operatory. Use of barriers, surface disinfectants, protective clothing and laminar airflow system are recommended. Aerosol production is reduced by proper patient position (Supine, head turned), pretreatment rinse with antimicrobial solution, cupping of cheeks or lips for water containment, and use of high volume suction tip.
Personal protection:	Clinician should always use recommended personal protection equipment, like gown is high neck and long sleeves, hair covering, mask, protective eyewear, face shield, and gloves. Patients should rinse with antimicrobial solution prior to start of ultrasonic or sonic procedures.
Patient protection:	Personal protection gear for the patient includes; plastic drape, towel or bib, protective eye wear, and hair covering cap.
Water control:	Use a disposable high-volume evacuation tip for suction, water control reduces aerosol production, improves the visibility of the treatment area, and increases patient comfort.
Power level:	Thin tips may damage the root surface if used on high power setting. All tip designs should be used at lowest frequency power setting, the high setting should be avoided.
Water:	The water spray around the instrument tip should create a light mist or halo effect with no excess dripping of water. Insufficient water can result in trauma to pulp.

Patient chair position:	Position the patient in normal supine position. This position reduces aerosol production and gagging, facilitates proper instrumentation.
Patient head position:	The head should be turned to a side. This causes the water to pool in the cheek, minimizing aerosol production.
Adaptation:	Adapt the side of last several millimeters of the tip to tooth surface. Length of the tip should be parallel to the long axis of tooth. Direct contact of tip to tooth surface should be avoided.
Stroke pressure:	Light strokes
Stroke pattern:	Strokes overlap one another in a sweeping or erasing type motion. Multidirectional strokes are used.
Stroke technique:	Keep the tip moving at all times. To remove heavier deposits, keep the tip in constant motion while making light strokes in sweeping motion, back and forth, over the teeth.

Advantages of Ultrasonic and Sonic Instruments

1. *Design of modified tip*: Modified tips are significantly smaller than hand-activated curettes. Thin tips are easier to insert and easier to adapt to root surface concavities and furcation.
   - Entire length of tip is active.
   - Tips have no cutting edge to cut or tear the tissue, so less tissue damage.
   - Removes less cementum so more conservative approach to subgingival debridement.
2. *Water lavage*:
   - Flushes calculus, debris and plaque.
   - Removes blood and debris allowing better vision.
   - Endotoxin removal.
   - Antimicrobial effect.
3. *Technique for use*: These are used with a light grasp and pressure which is less fatiguing to the clinician.
4. Overhangs can be removed easily.

AEROSOL PRODUCTION

Dental procedures produce airborne particles called *aerosols*, into surrounding environment. These aerosols contain microorganisms, blood, saliva, and oral debris. Microorganisms can survive up to 24 hours. Hence, patient should rinse with antimicrobial solution prior to treatment.

Laminar airflow system, which filters the organisms in air, is recommended.

Contraindications

Use of ultrasonic instruments is contraindicated in patients with:

1. Cardiac pacemaker.
2. Communicable disease.
3. Respiratory disease or difficulty in breathing.
4. With compromised gag reflex, or difficulty in swallowing.
5. With porcelain crown, titanium dental implants, composite resin-restoration, demineralized enamel surface, or dentinal hypersensitivity.
6. In children: Vibrations may damage growing tissue. Newly-erupted and primary teeth have large pulp chamber that are more susceptible to heat generated by instruments.

KEYPOINTS

1. Periodontal instruments are classified as:
   a. Diagnostic instruments–Probes, explorers.
   b. Scaling instruments.
      - Supragingival scalers.
      - Subgingival scalers.
      - Ultrasonic scalers.
      - Supragingival scalers:
        – Sickle scalers.
        – Surface scalers.
        – Jacquette scalers.
      - Subgingival scalers:
        – Hoe scalers.
        – Chisel scalers.
        – Root scalers.
      - Ultrasonic scalers.

c. Root planing and curetting instruments
   - Gracey curettes.
   - Universal curettes.

d. Cleansing and polishing instruments
   - Rubber cups, bristle brushes.
   - Dental tape, air powder polishing.

e. Surgical instruments

2. Fundamental prerequisites for effective instrumentation are governed by a number of general principles, like, proper position of the patient and the operator, illumination and retraction for optimal visibility and sharp instruments.
3. Stabilization of the instrument is very essential to avoid any injury to the patient or clinician.
4. There are four types of strokes used in periodontal instrumentation, placement stroke, exploratory stroke or assessment stroke, scaling stroke and root planing stroke.
5. Scaling is the process by which plaque and calculus are removed from both supragingival and subgingival tooth surfaces where as root planing is the process by which residual embedded calculus and portions of cementum are removed from the roots to produce a smooth, hard, clean surface.
6. The objective of scaling and root planing is to restore gingival health by completely removing all the tooth surface elements that can provoke gingival inflammation.

REVIEW QUESTIONS

1. How do you classify periodontal instrumentarium?
2. What are the various types of probes?
3. Describe the characteristics of a sickle scaler.
4. How are curettes different from scalers?
5. What are the modifications in curettes?
6. Name some differences in Universal and Gracey curettes.
7. What are the principles of periodontal instrumentation?
8. Define scaling and root planing.
9. What are the different types of instrument grasps and finger rests?
10. What are the different types of strokes used in periodontal instrumentation?

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37 Chapter

Plaque Control

DEFINITION OF PLAQUE CONTROL

It is the removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival surfaces.

GOALS OF PLAQUE CONTROL MEASURES

1. Plaque control retards the formation of calculus.
2. Removal of plaque leads to resolution of gingival inflammation.
3. Good plaque control facilitates return to and preservation of oral health.

RATIONALE

Controlling periodontal disease by regular plaque removal is based on the fact that if supragingival plaque is left undisturbed, it will become subgingival with the potential to become colonized by pathogenic bacteria.

BASIC APPROACHES FOR PLAQUE CONTROL

There are two basic approaches for plaque control:

1. *Mechanical:*
   - Individual
   - Professional—for subgingival plaque control, e.g. scaling and root planing.
2. *Chemical:*
   - Individual
   - Professional.

MECHANICAL PLAQUE CONTROL

Individual mechanical plaque control is achieved by:

I. Toothbrush: Manual or powered

II. Interdental aids:
   - Dental floss:

  - Unwaxed
  - Waxed
- Triangular toothpicks:
  - Hand-held
  - Proxa-pic
- Brushes: Proxa brush, bottle brushes
- Yarns, gauze strips, pipe cleaners.

III. Others:

- Rubber tip stimulator
- Water irrigators.

Toothbrushes (Fig. 37.1)

The following aspects are discussed:

a. Historical background.
b. ADA specification of a toothbrush.
c. Design of the toothbrush.
d. Brushing techniques.
e. Instruction in toothbrushing.

a. *Historical background*
   i. Initially aromatic plants like chewing twigs were used and Miswak from arrack trees were used.
   ii. Then Chinese invented the modern toothbrush in the year 1600.
   iii. In 1,780, William Addis designed a toothbrush with bone handle and hog bristles.
   iv. In 1,900 celluloid (plastic) brushes with bone handle were introduced.
   v. World War II—Nylon bristles were introduced.

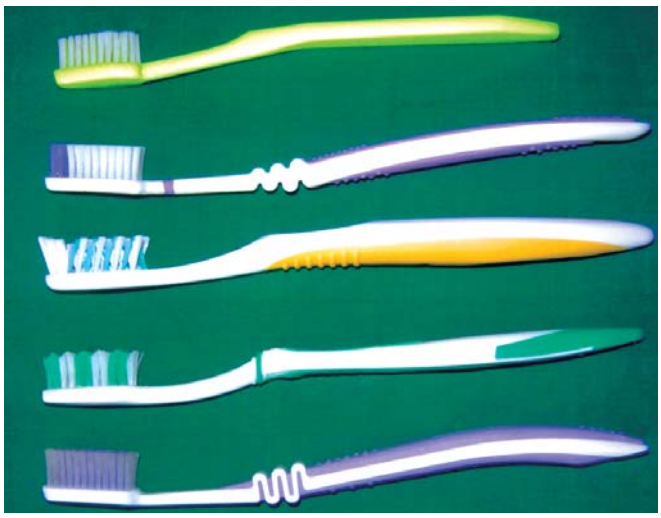


Fig. 37.1: Different types of toothbrushes

b. *ADA specifications of a toothbrush:* The head of the brush should be:
   i. 1 inch to 1¼ inches long.
   ii. 2-4 rows of bristles.
   iii. 5/16 inch to 3/8 inches wide.
   iv. 5-12 tufts per row.
   v. 80-86 bristles per tuft.

c. *Design of the toothbrush:* A toothbrush consists of handle, shank and head. It has bristles which when bunched together are called tufts. The extreme end of the head is toe and that, close to the handle is the heel.

- Size—Large, medium and small.
- Lateral profile—Flat, convex, concave and scalloped.

Bristles: Two types of bristles are available, nylon (synthetic) and natural (hog). Nylon bristles are preferred. Natural bristles are more susceptible to breakage and fraying, contamination with bacteria is high.

Hardness: Depends on material, diameter and length.

- Nylon bristles are more flexible.
- *Soft:* 0.007 inches to 0.009 inches (No. 7, 8, 9)
- *Medium:* 0.010 inches to 0.012 inches (No. 10, 11, 12)
- *Hard:* 0.013 inches to 0.014 inches (No. 13, 14)
- *Extra hard:* 0.015 inches (No. 15)

If the bristles are soft, they should be set close. If they are hard they should be more widely spaced.

Handle design:

   i. Straight
   ii. Angulation in the shank.
   iii. Indentation of handle for a better grip.
      - Frequency of brushing—Every 12 hours.
      - Frequency of change of brush—Every 3 months.
      - Length of brushing time—Initially 10-20 minutes is required until the patient becomes more precise. Later 3-5 minutes may suffice.

d. *Brushing motions in brushing techniques:*
   i. Horizontal:
      - Reciprocating (Scrub)
   ii. Vibratory:
      - Bass method

- Stillman's method
- Charter's method

iii. Vertical sweeping:
- Rolling stroke
- Modified bass
- Modified Stillman's
- Leonard
- Smithbell (Physiological technique)

iv. Rotary–Fones.

BRUSHING TECHNIQUES

The Bass method: Also called as intrasulcular method.

Technique:

1. Place the head of a soft brush parallel to the occlusal plane, with the brush head covering three teeth, beginning at the most distal tooth in the arch.
2. Place the bristles at the gingival margin, establishing an angle of 45 degrees to the long axis of the teeth.
3. Exert gentle vibratory pressure, using short back and forth motions without dislodging the tips of the bristles. This forces the bristle ends into the sulci, as well as into interproximal embrassures and should produce perceptible blanching of the gingiva.
4. Complete 20 strokes in the same position.
5. Lift the brush, move it anteriorly and repeat the process for next three teeth. To help reach the lingual surfaces of the anterior teeth, if the brush seems too large, insert the brush vertically. Press the heel of the brush into the gingival sulci and proximal surfaces at a 45° angle to the long axis of the teeth. Activate the brush with 20 short vibratory strokes.

   To reach occlusal surfaces, press the bristles firmly into the pits and fissures. Activate the brush into 20 short back and forth strokes, advancing section by section until all posterior teeth in all quadrants are cleaned.

   To reach distal surfaces of the last tooth in the arch, open the mouth wide and vibrate the tip of the brush against that surface, 20 times for each tooth.

Advantages

1. Short back and forth motion is easy to master.
2. Cleaning action is concentrated on the cervical and interproximal portions of the teeth, where most of the dental plaque detrimental to the gingivae is located.

Errors: As described by Perry and Schmid

1. Placement of bristles on the attached gingiva, rather than into the gingival sulcus. When the brush is activated, the gingival margin and tooth surfaces are neglected; whereas the attached gingiva and alveolar mucosa are traumatized.
2. Bristles are pressed against the teeth rather than directed into the gingival sulci. Activating the brush cleans the facial surfaces but misses the interproximal surfaces and surfaces along the gingival margin.
3. Relaxing the arm owing to tiredness so that the brush is allowed to slide down, creating an angle between the occlusal plane and the long axis of the brush. This prevents the main bulk of bristles from adequately penetrating interproximally and into the gingival sulci.

Modified Bass method (Fig. 37.2): The first part of the modified Bass method is identical to the Bass method. The modification consists of sweeping the bristles downwards over the tooth surface occlusally after completing the vibratory motion in the gingival sulci.

Stillman's method (Fig. 37.3): With Stillman's technique the bristle ends are placed at a 45° angle, with the bristles placed partly on the gingiva and partly on the cervical portion of the teeth.

Once the bristles are in place, pressure is applied to blanch the gingiva and a gentle but firm vibratory rotary motion is applied to the brush with the bristles remaining in the same position.

Advantage: It removes soft deposits from cervical areas.

Modified Stillman's technique:

1. The soft or medium, multitufted brush should be placed with the bristle ends resting partly on the cervical portion of the teeth and partly on the adjacent gingiva, pointing in an apical position, directed at an oblique angle to the long axis of the teeth.

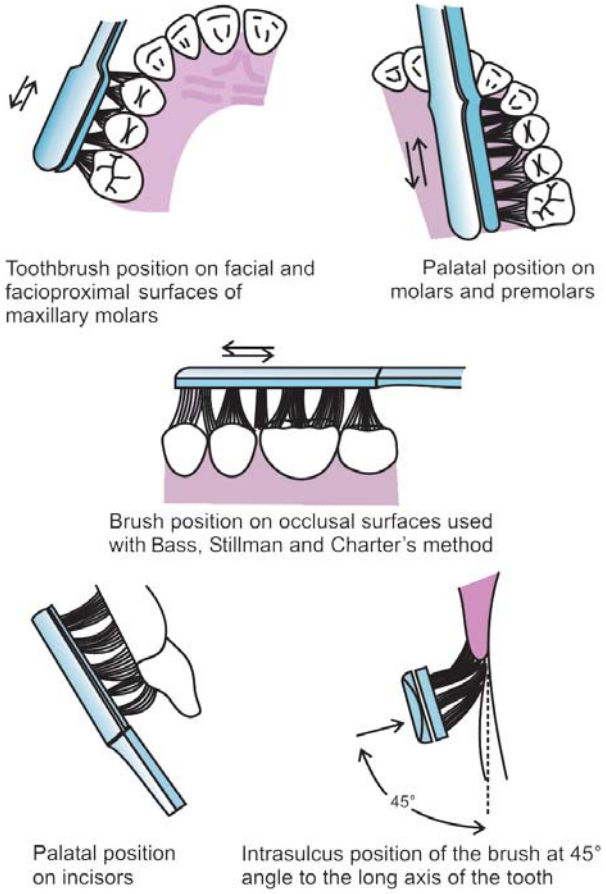


Fig. 37.2: Modified Bass method

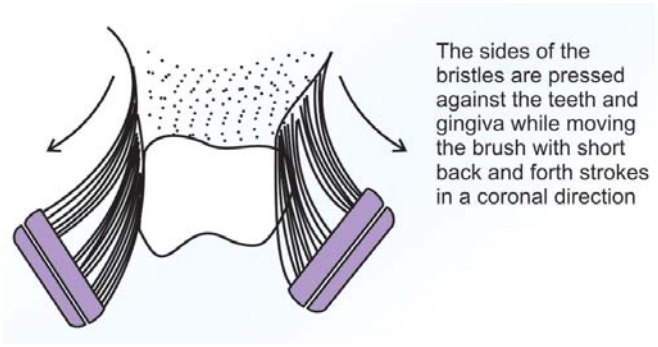


Fig. 37.3: Modified Stillman method

2. Pressure is applied laterally against the gingival margin to produce a perceptible blanching.
3. 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 tooth surface. This process is repeated on all the teeth.

To reach lingual surfaces of the maxillary and mandibular incisors, the handle of the brush is held in a vertical position, engaging heel of the brush. With this technique the sides rather than the ends of the bristles are used, penetration of the bristles into gingival sulci is avoided.

Advantages: The modified Stillman method may be recommended for cleaning areas with progressing gingival recession and root exposure to prevent abrasive tissue destruction.

The Charter's method (Fig. 37.4)

1. A soft or medium, multitufted brush is placed on the tooth with bristles pointing towards the crown at a 45° angle to long axis of the teeth.
2. The sides of bristles are flexed against the gingiva, and the back and forth vibratory motion is used to massage the gingiva.

Advantages: This method is effective particularly in cases with receded interdental papillae, it is suitable for gentle plaque removal and gingival massage, when using soft brush, and this technique can be recommended for temporary cleaning in areas of healing wounds after periodontal surgery.

Roll method/Fones technique:

Technique: The bristles are first directed apically and then swept in an occlusal direction with a rolling motion.

Advantages: It is popular because it is very easy to learn.

Disadvantage: Rolled-gingival margins prevent removal of plaque from sulcus area.

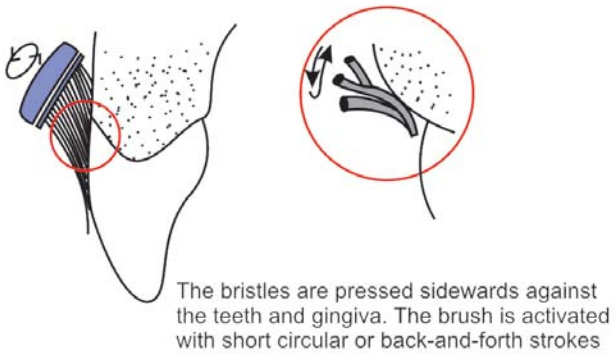


Fig. 37.4: Charter's method

Scrub technique:

- It is the simplest method.
- Consists of merely placing the bristles next to the teeth and moving them back and forth or scrubbing.

Advantage: It is easy to master.

Disadvantage: Poor plaque removal causes cervical abrasion and gingival recession.

e. *Instructions in brushing:*
   i. It should be started on the last/terminal teeth.
   ii. Three teeth are covered at a time.
   iii. 20 strokes are used in each area.
   iv. Overzealous brushing can lead to gingival recession, wedge-shaped defects in the cervical areas of root surfaces and painful ulceration of the gingiva. This type of brushing technique should be identified and discouraged.
   v. To maintain cleaning efficacy toothbrushes must be replaced as soon as the bristles begin to fray.

Powered Toothbrushes (Figs 37.5A and B)

They were introduced in 1939. Various types of motions used in powered toothbrushes are:

i. Reciprocal or back and forth.
ii. Circular.
iii. Elliptical or combination.

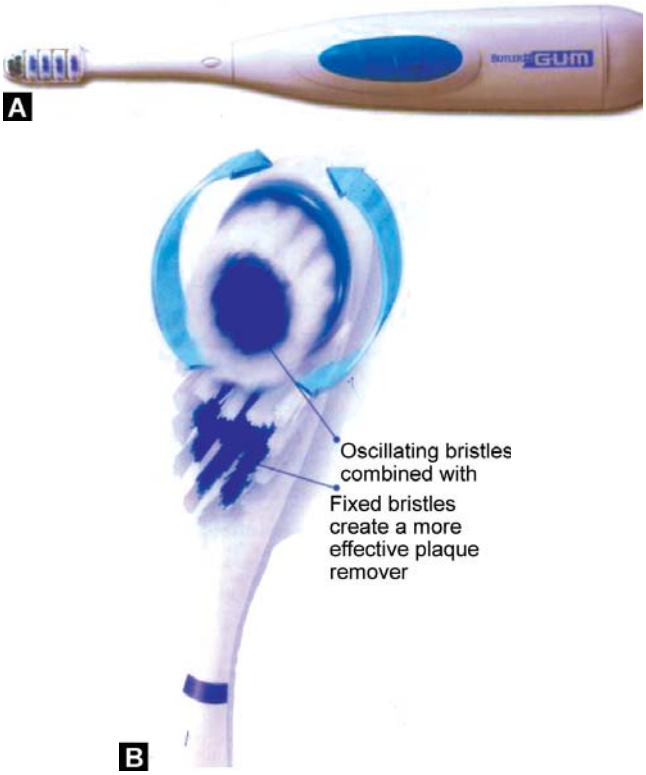
Powered toothbrushes are recommended for:

i. Individuals lacking fine motor skills.
ii. Small children or handicapped or hospitalized patients who need to have their teeth cleaned by someone else.
iii. Patients with orthodontic appliances.
iv. Patients who prefer them.

Conclusion: In conclusion, no specific toothbrush can be singled out as clearly superior for the routine removal of microbial deposits from the teeth. It differs greatly among individuals and should be recommended after considering factors such as the morphology of the dentition, periodontal health and manual dexterity.

Dentifrices

- These are the aids for cleaning and polishing of teeth surfaces.
- They are used in the form of powders, pastes and gels.



Figs 37.5A and B: Powered toothbrush

Composition of toothpaste:

1. *Abrasives* such as 20 to 40 percent $CaCO_3$, $Ca_3(PO_4)_2$ both of which react with fluoride. Now, silicon oxides, Al_2O_3, granular polyvinylchloride are used.
2. *Humectants:* 20 to 40 percent; maintains moisture, e.g. glycerine, sorbitol, mannitol, propylene glycol.
3. *Preservatives:* Such as benzoic acid.
4. *Thickening agents:* Synthetic sodium carboxy-methyl cellulose is used.
5. *Water:* 20 to 40 percent.
6. *Foaming agents:* 1 to 2 percent soap/detergent, e.g. sodium lauryl sulphate.
7. *Flavoring and sweetening agents:*
   - Two percent essential oils and synthetic flavors, e.g. mint and others.
   - Sweetening agents such as saccharine, sorbitol, mannitol.
8. *Desensitizing agents:* Up to 2 percent strontium salts, sodium fluoride, formalin, potassium nitrate and others.
9. *Coloring and preservatives:* Less than 1 percent.

10. *Anticaries agents:* Like sodium monofluorophosphate, sodium fluoride, formalin. Dentifrices containing pyrophosphates or zinc compounds have reportedly shown 10 to 50 percent reduction in calculus. They are thought to produce this effect by the fact that they are absorbed on to hydroxyapatite crystals thus inhibiting the growth of larger organized crystals.

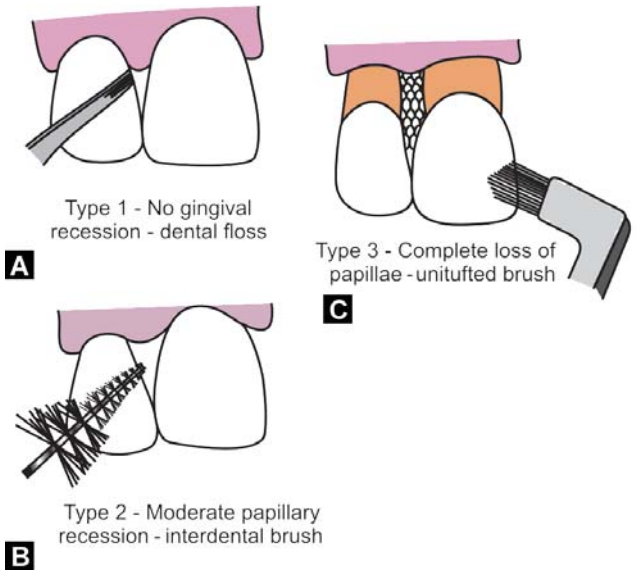
Interdental Cleaning Aids (Fig. 37.6)

Toothbrush regardless of the brushing method used does not completely remove interdental plaque either in individuals with healthy periodontal support or in periodontally-involved patients, with open embrasures. Interdental plaque removal is of importance because most of the pathogenic organisms originate in the interproximal areas. Among the numerous aids available, dental floss and interdental cleansers such as wooden or plastic tips and interdental brushes are most commonly recommended.

Factors determining the selection of interdental aids are the type of embrasures (Figs 37.7A to C)

- Type 1: The interdental papilla fills up the embrasure. Dental floss is advised
- Type 2: Moderate papillary recession is seen in such situations, miniature interdental brushes and wood tips are recommended.
- Type 3: Where there is complete loss of papilla and interdental gingiva is tightly bound to underlying bone (seen in diastema). Unitufted brushes are recommended.

Fig. 37.6: Interdental aids



Figs 37.7A to C: Types of embrasures with interdental cleansing aids

Dental floss: It is the most commonly recommended method of removing plaque from interdental areas.

Types of dental floss:

1. Twisted or non-twisted
2. Bonded or non-bonded
3. Waxed or unwaxed
4. Thick or thin.

Factors determining the choice of dental floss:

a. Tightness of tooth contacts.
b. Roughness of proximal surfaces.
c. The patient's manual dexterity.

The floss must contact the proximal surface from line angle to line angle to clean effectively. It should also cover the entire proximal surface, not just slipped apical to the contact area.

Technique: The floss should be at least 12 to 18 inches long. It is wrapped around the fingers or the ends may be tied together in a loop.

After stretching the floss between thumb and forefinger, pass it gently through each contact area in a back and forth motion.

Once the floss is apical to the contact area, move it up along the tooth till the contact area and down into the sulcus

again, this is repeated several times and the same is repeated on the proximal surfaces of other teeth.

Using floss holder: Indicated in patients with low manual dexterity and for handicapped and hospitalized patients in cleaning their teeth. Ideally, floss holder should possess forks that are rigid enough to hold the floss firmly and simple mounting mechanism, but the disadvantages of floss holders are more time consuming and they must be rethreaded whenever the floss becomes soiled or frayed.

Interdental brushes: They are cone-shaped or cylindrical brushes made of bristles mounted on a handle. Two types are available:

a. *Single* tufted brushes.
b. *Small conical brushes*—They are mainly useful to clean large, irregular concave tooth surfaces adjacent to wide interdental spaces.

Technique: They are inserted interproximally and are activated with short back and forth strokes in between the teeth. They are most useful in furcation areas, isolated gingival recession and on the lingual surfaces of mandibular molars and premolars.

Wooden tips: Wooden tips are either with or without handle. Soft triangular wooden toothpicks such as Stim-U-Dent are placed in the interdental space in such a way that the base of the triangle rests on the gingiva and the sides are in contact with the proximal tooth surfaces and it is moved in and out of the embrasure removing soft deposits from the teeth and also mechanically stimulating the papillary gingiva but its usefulness is limited to the facial surfaces in the anterior region of the mouth.

Wooden toothpicks can be attached to a handle. Example Perio-Aid and can be used on the facial and lingual surfaces throughout the mouth.

Other Aids

Gingival massage: Can be performed with a toothbrush, rubber tip stimulator or interdental cleaning devices. It produces epithelial thickening, increased keratinization and increased mitotic activity in the epithelium and connective tissue. It is questionable whether the above mentioned factors can provide substantial protection against microorganisms or not. But these methods also provide plaque control hence the plaque removal effect is far more important to periodontal health.

Oral Irrigation devices: They are of several types, one can use water faucet to irrigate between and around the teeth. The water pressure is steady and is controlled by turning the faucet handle. Others use an intermittent water jet. Oral irrigators clean non-adherent bacteria and debris from the oral cavity. It has been shown to disrupt and detoxify subgingival plaque and can be useful in delivering antimicrobial agents into periodontal pockets (subgingival irrigation). Currently, there are two types of irrigator tips useful for subgingival irrigation. One is the cannula type tip recommended for office use and other is a soft rubber tip for patient's use at home.

CHEMICAL PLAQUE CONTROL

Mechanical plaque removal remains to be a primary preventive method to control dental diseases and it should not be replaced by chemical plaque control. However, chemical plaque control can be used as an adjunct to effectively control gingival inflammation and prevent the recurrence or progression of periodontal disease. Chemical methods are very effective during phase I therapy, for patients with recurrent problems, ineffective plaque control for any reason and for use after periodontal or oral surgery.

The ADA Council on Dental Therapeutics has adopted a program for acceptance of plaque control agents. The agents must be evaluated in placebo-controlled clinical trials of 6 months or longer and demonstrate significantly improved gingival health compared with controls. To date, only two agents have been accepted by ADA for treatment of gingivitis: chlorhexidine digluconate mouth wash and essential oil mouth rinse.

Ideal Properties of a Mouthwash

It should

1. Eliminate pathogenic microorganisms only.
2. Prevent development of resistant bacteria.
3. Exhibit substantivity.

4. Be safe to oral tissues at the recommended concentration.
5. Significantly reduce plaque formation and gingivitis.
6. Inhibit calcification of plaque to calculus.
7. Not stain and alter taste.
8. Not have adverse effects on teeth or dental materials.
9. Be easy to use.
10. Be inexpensive.

Classification of Antimicrobial Agents

Depending on antimicrobial efficacy and substantivity.

First Generation Agents: Reduces plaque scores by 20 to 50 percent, efficacy is limited by their poor retention in the oral cavity. Hence, used 4 to 6 times daily (poor substantivity)

Examples: Antibiotics, quaternary ammonium compounds, phenols and sanguinarine.

Second Generation Agents: These are retained longer in the oral cavity or tissues and slow release property provides overall reduction in plaque score by 70 to 90 percent; used 1 to 2 times daily (higher substantivity).

Example: Bisbiguanides.

Third Generation Agents: It should be effective against specific periodontopathic organisms. Yet to be developed clinically.

Chemicals Used for Supragingival Plaque Control [Addy's classification]

A. *Antibiotics*
- Penicillin
- Vancomycin
- Kanamycin
- Erythromycin
- Spiramycin
- Metronidazole

B. *Enzymes*
- Mucinase
- Protease
- Lipase
- Amylase
- Elastase
- Lactoperoxidase
- Hypothiocynase
- Mutanase

C. *Quaternary ammonium compounds*
- Cetylpyridinium chloride
- Benzethonium chloride
- Benzalkonium chloride
- Domiphen bromide

D. *Bisbiguanides*
- Chlorhexidine
- Alexidine
- Octenidine/Bispyridines

E. *Metallic salts*
- Copper
- Tin
- Zinc

F. *Herbal extracts*
Sanguinarine

G. *Fluorides*
Strontium Fluoride

H. *Oxygenating agents*
Hydrogen peroxide

I. *Phenolic compounds*
- Thymol
- Menthol
- Eucalyptol

J. *Other antiseptics*
- Iodine
- Povidone iodine
- Sodium hypochlorite
- Hexetidine
- Triclosan

A chemical approach to therapy can be used for either of the following purposes:

1. Prevention or chemoprophylaxis
2. Treatment or chemotherapy

Based on these purposes, antimicrobials are divided into two groups:

1. Preventive agents—which affect development of supragingival plaque.
2. Therapeutic agents—which are directed against subgingival plaque.

The chemotherapeutic agents would be either:

1. *Non-specific:* Affecting all plaque bacteria uniformly and leading to a quantitative reduction in plaque.
2. *Specific:* Acting as a 'Silver bullet' to quantitatively reduce only the periodontopathic plaque bacteria.

Bisbiguanides (Chlorhexidine)

Indications and uses

1. Used as an adjunct to mechanical oral hygiene in initial periodontal therapy.
2. During postsurgical period—immediately after pack. removal, complete plaque control can be achieved without extensive use of proximal cleaning aids (which are painful to use and may delay healing).
3. Improves healing after routine oral surgical procedures and in the postoperative management of immediate denture construction.
4. Patients wearing fixed orthodontic appliances or intermaxillary fixation devices will benefit from a daily rinse with a 0.2 percent chlorhexidine solution (also available in 0.12% concentration).
5. For handicapped patients whose plaque control and gingival status are often very poor.
6. Chlorhexidine will help to control plaque accumulation in patients with drug-induced gingival over-growth.
7. In medically-compromised patients who suffer from recurrent generalized oral infections.
8. It can also be advised in patients with local, oral infections such as denture induced stomatitis, aphthous ulceration, dry socket and acute ulcerative gingivitis.
9. Finally, it can be used as a prophylactic rinse in the prevention of post-extraction bacteremia, dry sockets and to reduce the bacterial content of the aerosol sprays during the ultrasonic scaling.

Disadvantages/unwanted effects

1. Extrinsic staining of teeth, i.e. Brown stains.
2. Painful, desquamative lesions on the oral mucosa may be associated with burning sensation.
3. Impaired taste sensation.
4. Parotid swelling is rare—due to mechanical obstruction of parotid duct.
5. Dryness and soreness of mucosa.

Mechanism of action: It has a broad spectrum of antibacterial activity. In general, gram-positive bacteria are more susceptible as compared to gram-negative bacteria. In relatively high concentrations, chlorhexidine is bactericidal but low concentrations may be bacteriostatic to susceptible bacteria.

The cationic molecules of chlorhexidine bind readily to the oppositely charged cell wall and interfere with the membrane transport initiating a leakage of low molecular weight substances. In high concentrations chlorhexidine penetrates the cell and causes precipitation of the cytoplasm. This explains the bactericidal action of Bisbiguanides in general.

When 10 ml of 0.2 percent chlorhexidine solution is used as a mouthwash for one minute, 30 percent of the drug is retained in the mouth due to its property of substantivity (i.e. the bound drug is released slowly over a period of 8 to 12 hours). No ill effects have been reported following small quantities of chlorhexidine being swallowed since it is poorly absorbed from gastrointestinal tract and is excreted in feces. Long-term human trials have shown that, 0.12 percent chlorhexidine is equally effective in plaque inhibition when used as a mouth rinse (twice daily) however; chemical plaque control cannot be used as a supplement to scaling and root planing (monotherapy) as it is not so effective in controlling total plaque inhibition. Commercially, available products in the concentration of 0.2 percent are Rexidine, Hexidine, Clohex; in 0.12 percent, as Periogard.

Essential oil rinse: Contains thymol, eucalyptol, menthol and methylsalicylate. It has been proved to be effective in reducing plaque and gingival scores. Commercially available as Listerine® (Discussed in detail elsewhere in the book).

Disclosing agents (Fig. 37.8): Offers following advantages:

- Discloses plaque.
- Permits patients to evaluate their performance at home.

Various disclosing agents are:

1. *Erythrosine dye:*
   FDC No.3
2. *Two-tone dye:*
   - FDC No.3—Red
   - No.3—Green

Fig. 37.8: Disclosing solution

It can differentiate older to newer plaque; old plaque stains deep violet and new plaque pale violet.

3. *Plaque-lite system:* Available as wafers/tablets or solutions which are swished around the mouth and excess dye is removed by rinsing the mouth.

KEYPOINTS

1. Plaque control is defined as the removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival surfaces.
2. There are two basic approaches for plaque control,
   i. Mechanical by means of tooth brush, and interdental aids,
   ii. Chemical by means of various chemical agents like chlorhexidine, phenols, quaternary ammonium compounds.
3. The chemical agents are very effective when used along with the mechanical means. However, the plaque inhibiting effect is not as striking when used as a monotherapy.
4. To date, mechanical plaque control seems to be the gold standard in preventing dental diseases. However, chemicals can be used as an adjunct to mechanical therapy.
5. For the treatment of gingivitis only two agents have been accepted by ADA, chlorhexidine mouthwash and essential oil mouthwash.

KNOW MORE ...

Types of Toothbrushes

Other than manual and powered toothbrushes, sonic and ultrasonic and ionic toothbrushes are introduced.

Toothbrush bristles could be, hard or soft, natural or synthetic, multitufted or space tufted.

The hardness of the bristles depend upon:

a. *Diameter of bristles* — wider the diameter, stiffer are the bristles.
b. *Length of bristles* — shorter bristles are stiffer.
c. *Number of filaments in a tuft* — more tufts provide more support.
d. *Curvature of filament* — curved filaments may be more flexible and less stiff than straight filaments.

Various Advantages of Powered Tooth Brushes

- Increases patient motivation thereby resulting in better patient compliance.
- Increased accessibility.
- No specific brushing technique required.
- Uses less brushing force.
- If brushing timer is incorporated (as with some brushes) the patient will brush for the required duration.

Recent Development in Dentifrices

- Toothpastes for children
- Herbal (natural) toothpastes.
- Whitening toothpastes (long-term use is not recommended as it contains highly abrasive silica particles).

Factors Determining the Selection of Interdental Aids

Type of gingival embrasures and others include:

- Alignment of teeth.
- Fixed prosthesis/orthodontic appliance.
- Open furcation area.
- Contact areas.

Powered flossing devices are also available.

Mechanism of Action of Chlorhexidine Gluconate (0.2%)

"Pin cushion effect"

It could be explained as follows: One charged end of chlorhexidine (dicationic) molecule binds to the tooth surface where as the other remains available to initiate the interaction with the bacterial membrane as the microorganism approaches the tooth surface — called as "pin-cushion effect…"

REVIEW QUESTIONS

1. What are the ideal requirements of a toothbrush?
2. Describe the various brushing techniques.
3. What is the composition of a toothpaste?

4. What are interdental aids? What are its selection criteria?
5. Classify the chemicals used for plaque control.
6. What is the composition of disclosing agent and add a note on its uses?
7. What are the ideal properties of a mouthwash?
8. Write in detail on chlorhexidine gluconate.

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38 Chapter

Principles of Periodontal Surgery

INDICATIONS OF PERIODONTAL SURGERY

1. Areas with irregular bony contours, deep craters and others requiring a surgical approach.
2. Deep pockets where complete removal of root irritants is not possible, especially in inaccessible areas like molars and premolar areas.
3. In cases of Grade II and III furcation involvement, where apart from removing local irritants, necessary root resection or hemisection can be considered.
4. Infrabony pockets in non-accessible areas which are non-responsive towards nonsurgical methods.
5. Persistent inflammation in areas with moderate and deep pockets.
6. Correction of mucogingival problems.

CONTRAINDICATIONS OF PERIODONTAL SURGERY

These may be oral or systemic.

1. In patients of advanced age where teeth may last for life without resorting to radical treatment (Procedures indicated in a person of 60 years of age may not be justified in someone of 70 years of age).
2. Patients with systemic diseases such as cardiovascular disease, malignancy, liver diseases, blood disorders, uncontrolled-diabetes, consultation with the patient's physician is essential.
3. Where thorough subgingival scaling and good home care will resolve or control the lesion.
4. Where patient motivation is inadequate.
5. In the presence of infection.
6. Where the prognosis is so poor that tooth loss is inevitable.

GENERAL PRINCIPLES OF SURGERY

a. Preparation of the patient
b. The general conditions that are common to all periodontal surgical techniques, and
c. Complications that may occur during or after surgery.

PREPARATION OF THE PATIENT

Almost every patient has to undergo the initial or preparatory phase of therapy. (scaling + root planing and removal of etiotropic elements) because it:

I. Eliminates some lesions completely
II. Renders the tissues more firm and consistent, thus facilitating more accurate and delicate surgery
III. Acquaints the patients with the office and with the operator and assistants, thereby reducing the patient's apprehension and fear. The re-evaluation phase consists of reprobing and re-examining all the findings that previously indicated the need for the surgical procedure. Persistence of these findings will confirm the indication for surgery. The number and dates of the surgical procedures, the outcome and the postoperative care that is needed are all decided before hand. Informed consent should be taken from the patient after explaining the details of surgical procedures, both verbally and in writing.

General Conditions that are Common to all Procedures

Premedication

For normal patients their use is not clearly demonstrated. The prophylactic use of antibiotics has been advocated for both medically-compromised patients as well as patients undergoing bone-grafting procedures. Emergency equipment should be readily available at all the times.

All the measures should be taken to prevent the transmission of infections. These include the use of disposable gloves, surgical masks and protective eye wear. All surfaces that may be contaminated with blood or saliva and cannot be sterilized, must be covered with aluminium foil/plastic wrap. Ultrasonic scaling is contraindicated in patients with infectious diseases, as it generates aerosols and special care should be taken while using it (Pre-procedural mouth rinsing).

Sedation and Anesthesia

In order to prevent pain during the surgery, the entire area to be treated should be thoroughly anesthetized by means of a regional block and local infiltration. Patients who are apprehensive and neurotic may require special management with agents like sedatives and anti-anxiety drugs.

Tissue Management

1. *Operate gently and carefully:* In addition to being most considerate to the patient, tissue manipulation should be gentle because it produces excessive tissue injury; causes postoperative discomfort and delays healing.
2. Observe the patient at all times.
3. *Be certain the instruments are sharp:* Dull instruments will cause unnecessary trauma because of excess force usually applied to compensate for their ineffectiveness.

Suturing

Suturing materials are classified as either nonabsorbable or absorbable.

Nonabsorbable

- Natural: example—braided-silk
- Synthetic: example—Dacron-coated and impregnated with teflon.

Absorbable

- Natural: example—Surgical gut.
- Synthetic: example—Polyglycolic acid derivatives like vicryl.

Goals of suturing

1. Maintains hemostasis
2. Permits healing by primary intention
3. Reduces postoperative pain
4. Permits proper flap position
5. Prevents bone exposure resulting in delayed healing and unnecessary resorption.

Parts of surgical needles

1. Eye.
2. Body: Widest point of the needle and is referred to as the "grasping area".
3. Point: Point the tip can be conventional or reverse cutting.

Knots and knot typing: Have three components:

1. The loop created by the knot.
2. The knot itself.
3. Ears—cut ends of the suture.

Suturing techniques and materials: One of the cardinal rules in suturing is to avoid placing excessive tension on

the tissues being sutured to the extent of inducing blanching. Such tension will result in necrosis of sutured area and subsequent loss of suture entirely.

Suturing Techniques (Figs 38.1 to 38.8)

Interrupted Suture

a. Direct or loop suture
b. Figure eight
c. Horizontal mattress
d. Vertical mattress
e. Distal wedge or Anchor suture
f. Periosteal suturing (Fig. 38.6)

Continuous Suture

a. Papillary sling
b. Horizontal mattress
c. Vertical mattress

Periodontal Dressing

Various commercially-available periodontal dressings are:

a. Coe pak
b. Kirkland periopak
c. Peridress
d. Periocare
e. Periodontal pack
f. Perio-putty
g. Zone periodontal pak.

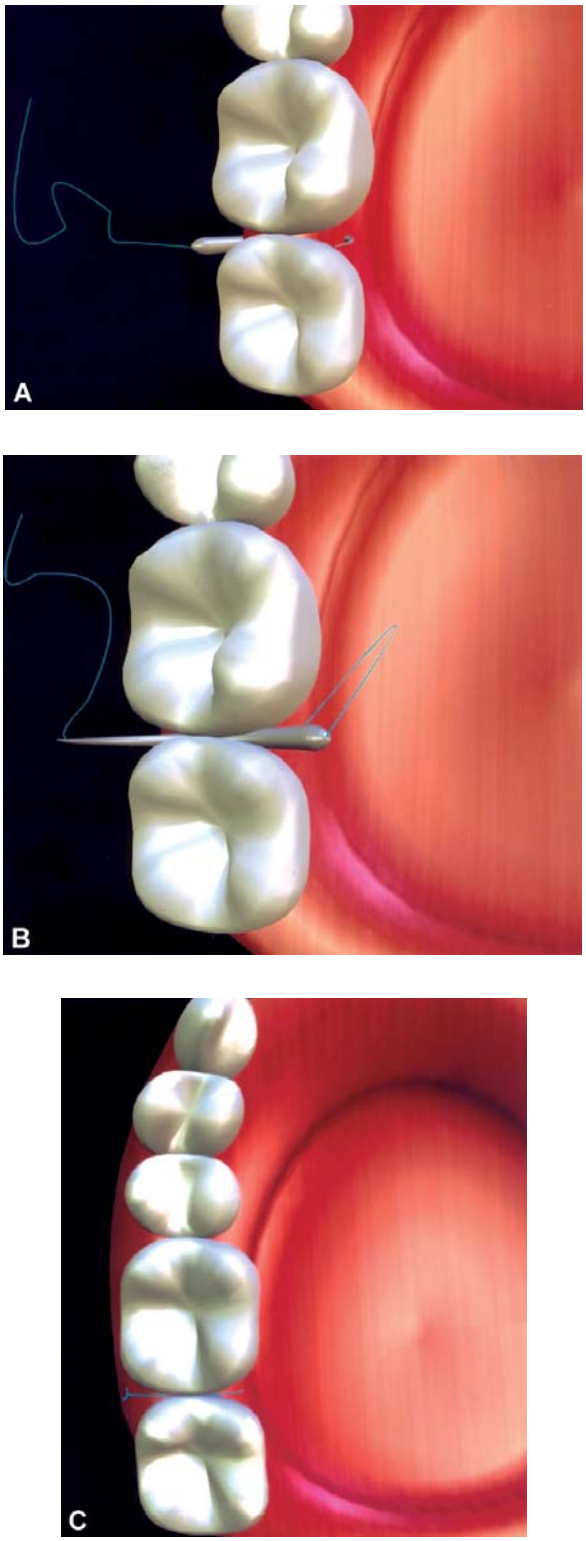
Advantages of Periodontal Packs/Dressings

1. It minimizes the likelihood of postoperative infection and hemorrhage.
2. Facilitates healing by preventing surface trauma during mastication.
3. Protects against pain induced by contact of the wound with food or with tongue during mastication.

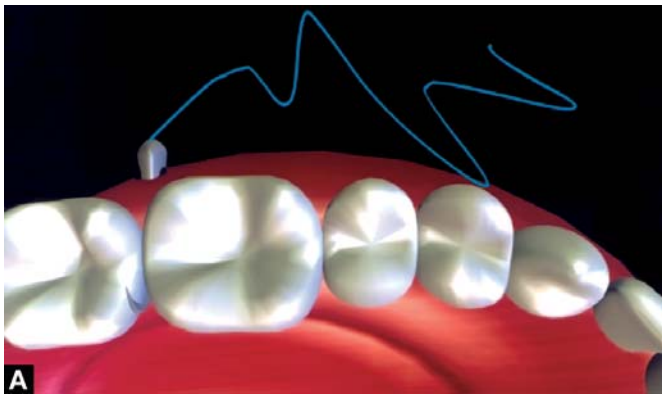
Types of Packs

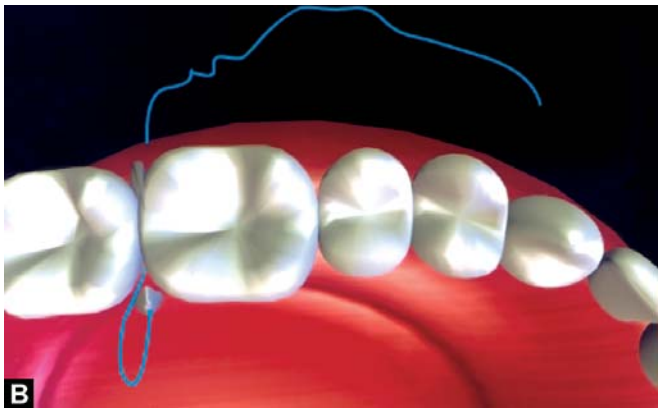
- Zinc oxide eugenol packs.
- Non-eugenol packs.

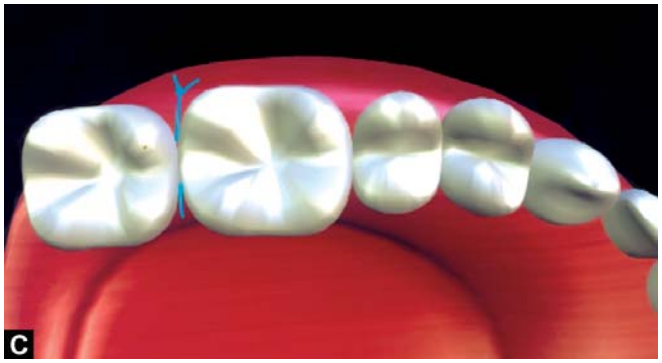
Zinc oxide eugenol packs: Example—Wondre-pak developed by Ward. It is based on the reaction of zinc oxide and eugenol.



Figs 38.1A to C: Direct or loop technique: (A) The needle penetrates from the outer surface of the first flap and engages the inner surface of the opposite flap. (B) The suture is brought back to the initial side. (C) Where the knot is tied

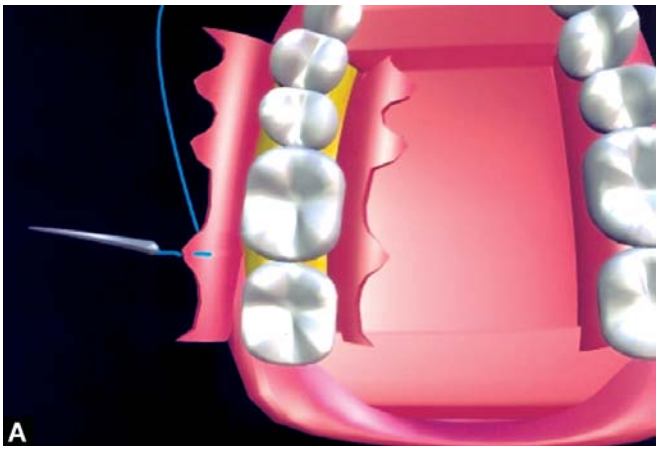


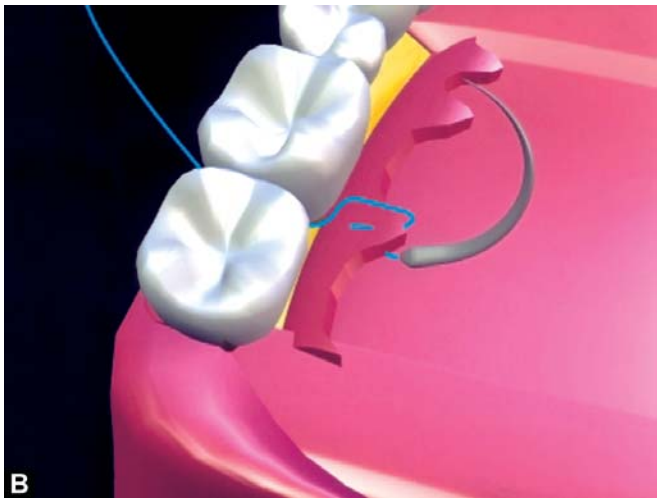




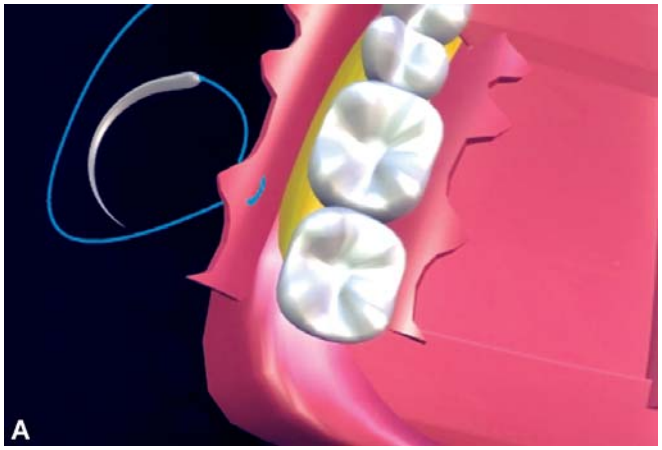
Figs 38.2A to C: Interrupted figure—eight suture: (A) The needle penetrates the outer surface of the buccal flap. (B) And then the outer surface of the opposite flap. (C) The suture is brought to the first flap and the knot is tied

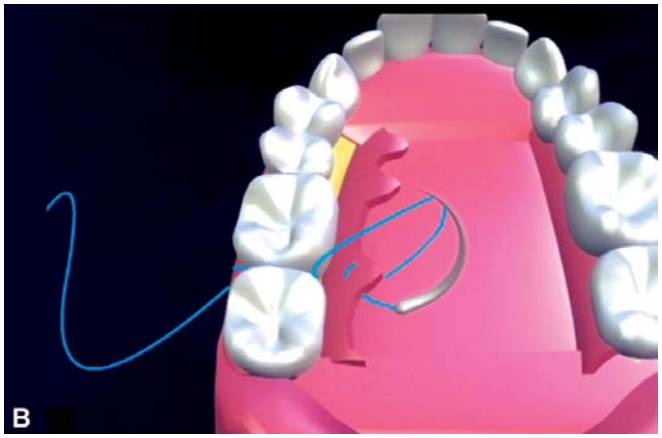
Composition: Apart from zinc oxide and eugenol, it has zinc acetate as an accelerator, asbestos used as a binder and filler, and tannic acid. However, asbestos can induce lung disease and tannic acid may lead to liver damage. Hence, both the substances have been eliminated. They are supplied in a liquid and powder form that is mixed prior to use. Eugenol

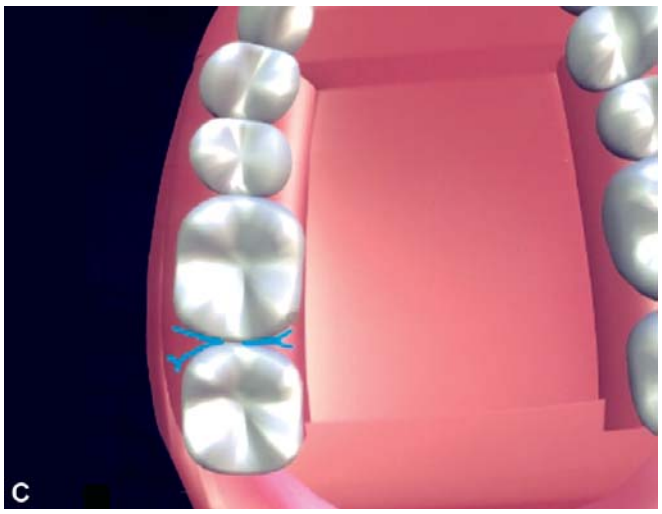




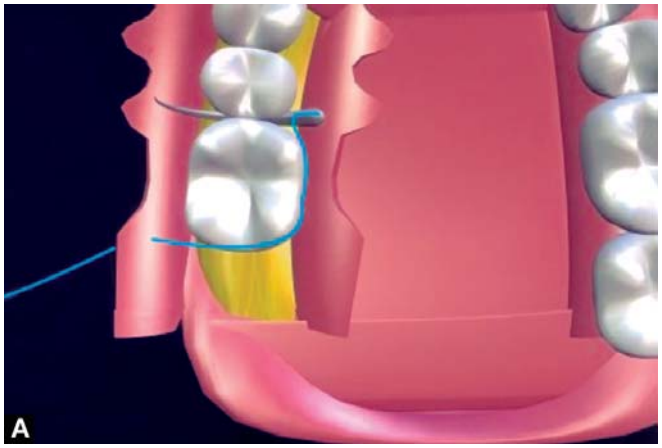
Figs 38.3A to C: Vertical mattress: Interrupted. (A) The needle is inserted approximately 7 mm apical to the tip of the papilla, emerging again from the epithelialized surface of the flap 2 to 3 mm from the tip of the papilla. (B) The needle is brought back through the embrassure, where the technique is again repeated lingually or palatally. (C) The knot is tied buccally

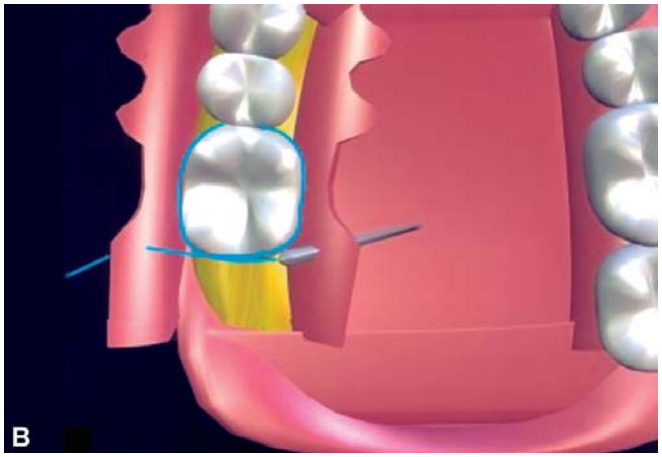


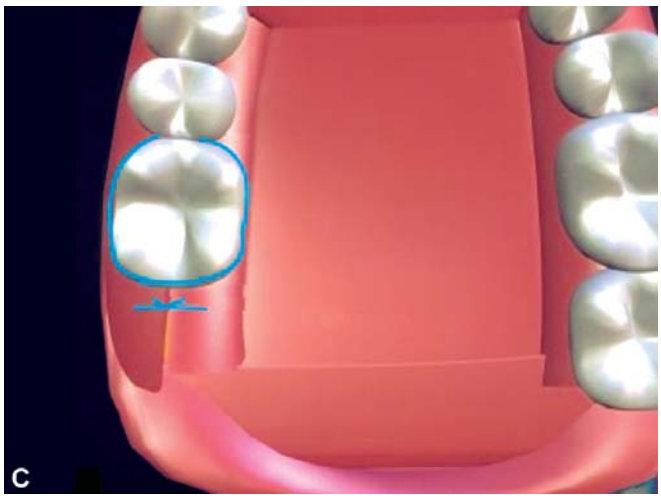




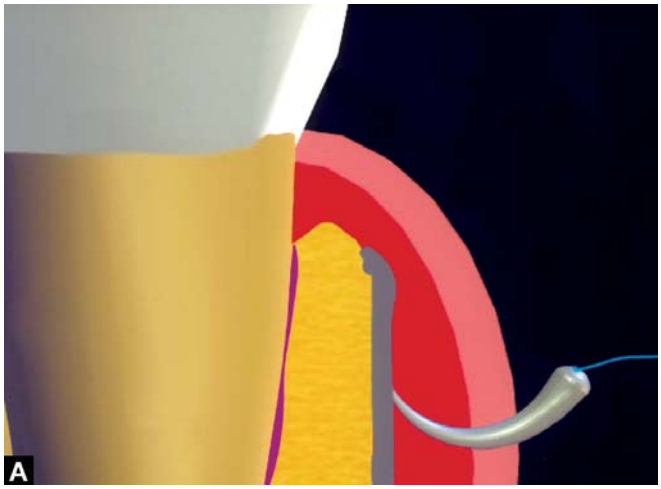
Figs 38.4A to C: Horizontal mattress—interrupted: (A) The needle is inserted 7 to 8 mm apical to one side of the midline of the papilla, that is at the mesiobuccal line angle emerging again 4 to 5 mm through the epithelialized surface on the distobuccal line angle. (B) The needle is passed through the embrasure and the same maneuver is duplicated again on the lingual or palatal surface. (C) Then the knot is tied buccally

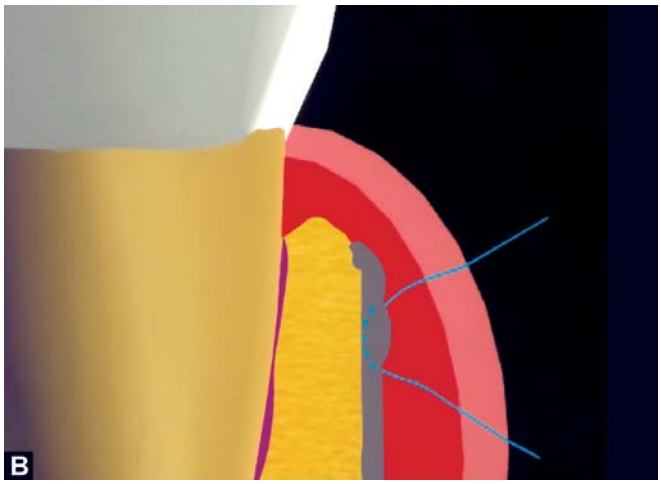






Figs 38.5A to C: Distal wedge technique or anchor suture: (A) The needle is placed at the line angle area of the facial surface, suture is anchored around the tooth. (B) Passed through the inner surface of the opposite flap. (C) The knot is tied





Figs 38.6A and B: Periosteal suturing involves five steps: (1) Needle penetration where needle point is perpendicular to bone. (2) Rotation of the needle point, (3) Glide, where needle point glides against the bone, (4) Rotation, (5) Exits out of the periosteum and bone

may produce allergic reaction that may render the area erythematous combined with a burning sensation in some patients.

Non-eugenol packs: Example-Coe Pak. It is based on the reaction between a metallic oxide and fatty acids. It is supplied in two tubes.

Composition: One of the tubes contains zinc oxide, oil for plasticity, a gum for cohesiveness and lorothiodol—a fungicide. The other tube contains liquid coconut fatty acids thickened with rosin and chlorothymol—a bacteriostatic agent. Other noneugenol packs include cyanoacrylates and methacrylic gels. Retention of packs is obtained by mechanical interlocking in the interdental spaces and by joining lingual and facial portions of the pack.

Preparation and Application of Periodontal Dressing (Figs 38.9 to 38.11)

Coe pak (Non-eugenol) is prepared by mixing equal lengths of pastes from the tubes supplied, i.e. accelerator and base until it has a uniform color. The paste is then placed in a cup of water at room temperature for 2 to 3 minutes, when the pack loses its tackiness, it is ready to be placed on the surgical site.

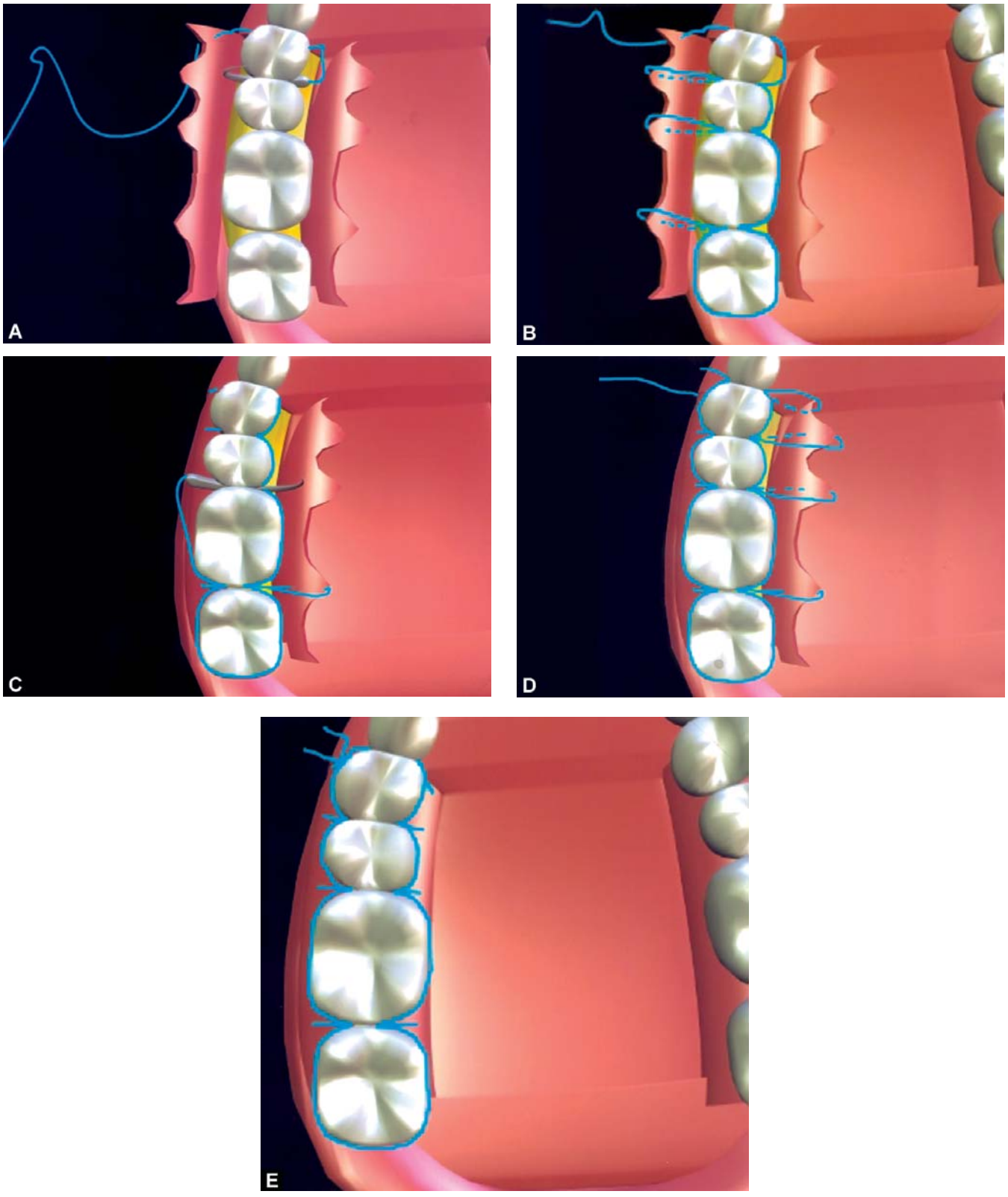
The pack is then rolled into two strips approximately to the length of the treated area and placed on buccal surface from mesial-distal end and the remainder can be placed the same way on the lingual/palatal surfaces. The strips are joined at the distal end by hooking it around the distal most tooth as well as interproximally by applying gentle pressure (with the help of a probe) to join facial and lingual surfaces of the pack. Any overextension onto uninvolved area should be avoided. It is usually kept on for 1 week after surgery.

Findings at pack removal

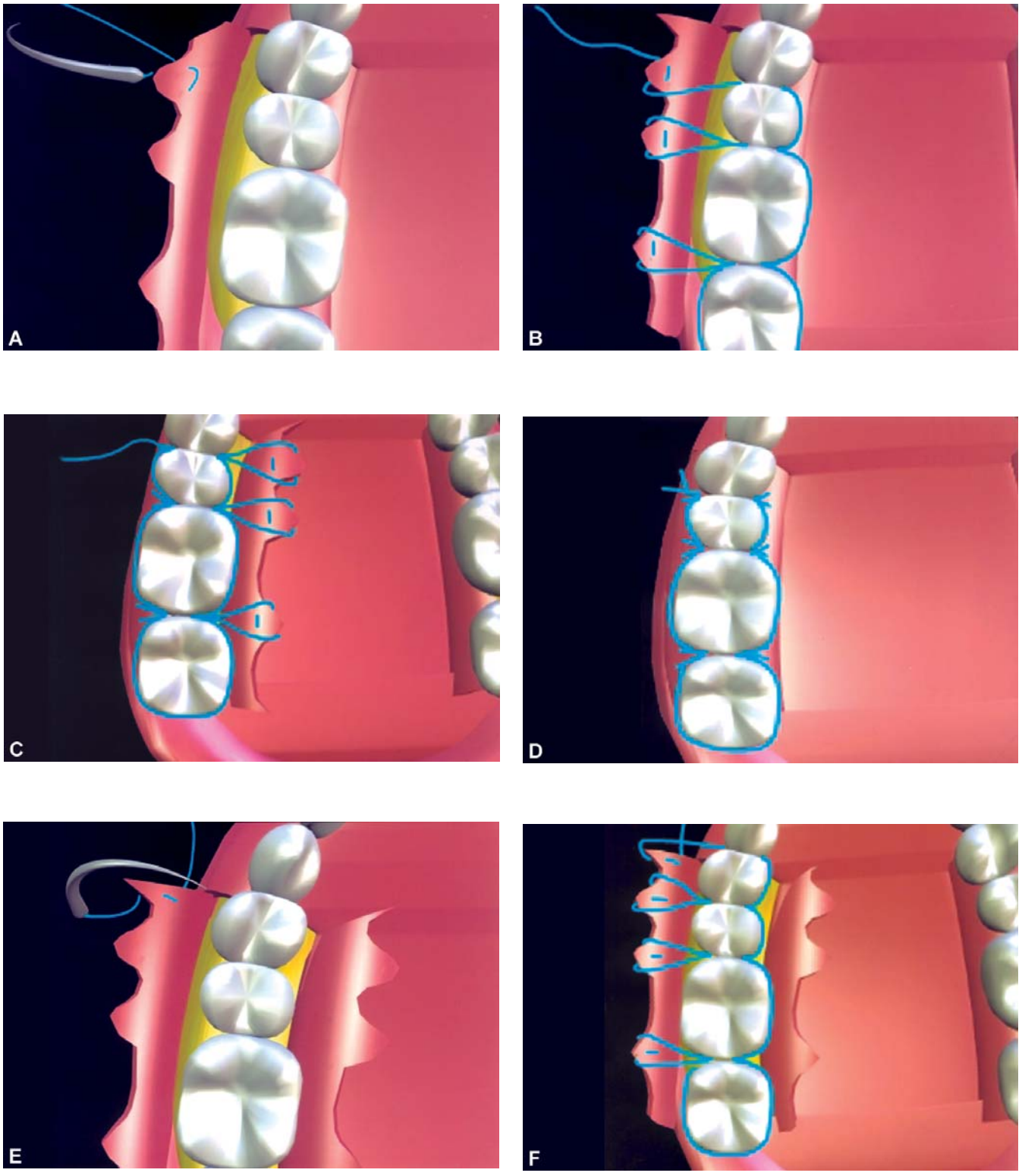
a. If gingivectomy has been performed, the cut surface is covered with a meshwork of new epithelium. If calculus has not been removed completely, red bead-like protuberances of granulation tissue will persist, which should be removed with a curette.
b. After a flap operation, the incision areas are epithelialized but may bleed readily when probed, hence pockets should not be probed.
c. The facial and lingual mucosa may be covered with a grayish-yellow or white granular layer of food debris that has seeped under the pack and can be easily removed with a moist cotton pellet.

Instructions for the Patient after Surgery

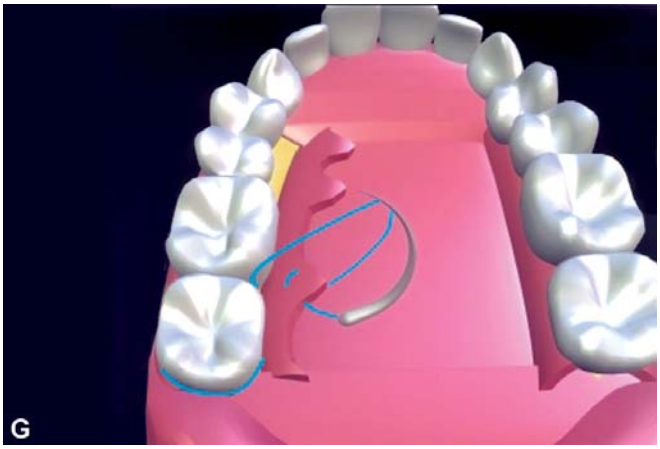
1. Patients should take the advised medication.
2. The pack should remain in place until it is removed after one-week, by the clinician.

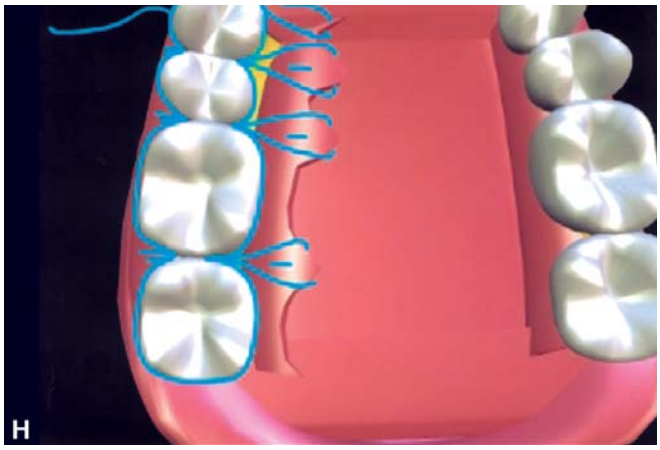


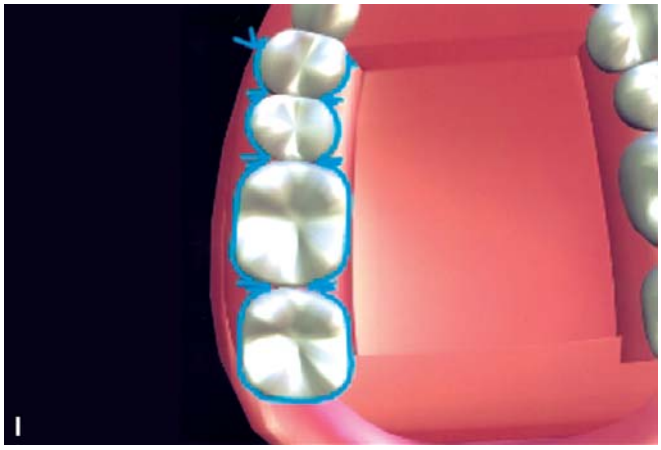
Figs 38.7A to E: Independent sling suture: (A) The needle is inserted on the facial papilla, a sling is placed on the palatal surface. (B) The facial papillae were sutured together. (C) When the last tooth is reached, the suture is anchored around it. (D) The palatal flaps are sutured together in a similar fashion. (E) Final knot is placed



Figs 38.8A to F







Figs 38.8G to I

Figs 38.8A to I: Continuous vertical/horizontal mattress sutures: Technique is identical to independent papillary sling except that vertical or horizontal mattress sutures are substituted for the simple papillary sling

Fig. 38.9: Periodontal pack

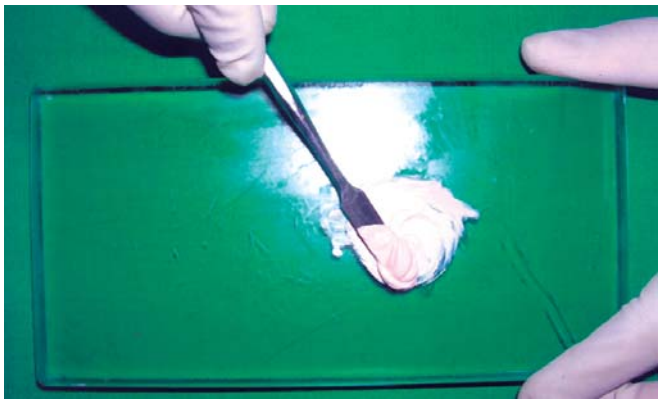


Fig. 38.10: Mixing

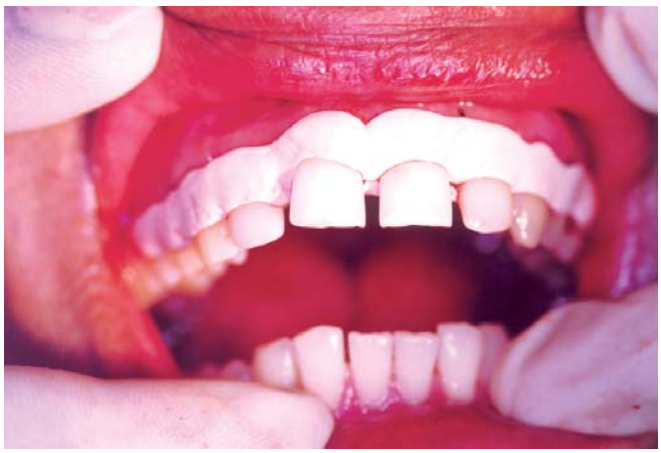


Fig. 38.11: Application of Coe Pak

3. For the first three hours after the operation, avoid hot foods to permit the pack to harden, try to chew on the non-operated side of the mouth. Avoid citrus juices and spiced-food because it causes pain and burning.
4. Do not smoke.
5. Do not brush over the pack.
6. During the first day apply ice.
7. Follow your daily activities, but avoid excessive exertion of any type.
8. Swelling is not unusual.
9. There may be an occasional tinge of blood in the saliva for the first 4 to 5 hours after the operation, beyond this if there is bleeding, report to the doctor immediately.

COMPLICATIONS DURING SURGERY

1. *Syncope* or transient loss of consciousness owing to a reduction in cerebral blood flow. The most common cause is fear and anxiety. It is usually preceded by a feeling of weakness which is followed by pallor, sweating, coldness of the extremities, dizziness and slowing of the pulse.

   Management: (a) Patient should be placed in (Trendelenburg position) a supine position with legs elevated. (b) Tight clothes should be loosened and an open airway ensured. (c) Administration of oxygen is also useful. (d) Glucose administration also helps.

   History of previous syncopal attacks during dental appointments should be explored before proceeding with the treatment.
2. *Hemorrhage:* Periodontal surgery produces profuse bleeding in its initial incisional steps. However, after the granulation tissue is removed, bleeding will disappear or reduce considerably. Excessive hemorrhage after the initial steps may be due to lacerated capillaries and arterioles or damage to larger vessels due to surgical invasion of anatomic areas.

   Treatment: Pressure pack, cotton pellet dipped in ferric subsulphate powder. Thrombin, hastens the process of blood clotting, oxidized cellulose and Gel foam are most commonly used to control the hemorrhage.

Complications during the First Postoperative Week

1. *Persistent bleeding after surgery:* The pack is removed, the bleeding points are located and the bleeding is stopped with pressure, sutures, electrosurgery or electrocautery. After the bleeding is stopped, the pack is replaced by a fresh one.
2. *Sensitivity to percussion* may be due to extension of inflammation into periodontal ligament. Gradual diminution of severity is a favorable sign. The pack should be removed and the gingiva should be checked for localized areas of infection or irritation. The particles of calculus that may have been overlooked should be removed. Relieving the occlusion is usually helpful. Sensitivity to percussion may also be caused by excess pack which interferes with occlusion. Removal of excess should correct the condition.
3. *Swelling:* Within the first two postoperative days patient reports a soft, painless swelling of the cheek in the area of operation. There may be lymph node enlargement and temperature may be slightly elevated. This type of involvement is due to localized inflammatory reaction to operative procedures. If the swelling persists and associated with increased pain. Antibiotics like amoxicillin, 500 mg every 8 hours for 1 week should be prescribed.
4. *Feeling of weakness:* Patients may experience a "washed out", weakened feeling for about 24 hours after the surgery. This represents a systemic reaction to a transient bacteremia-induced by operative procedure. It can be prevented by prescribing prophylactic antibiotics.
5. *Postoperative pain:* Periodontal surgery performed following basic principles should produce only minor pain and discomfort. In a study the results revealed mucogingival procedures results in maximum discomfort and followed by osseous surgery than any other plastic gingival surgeries.

   Common sources of postoperative pain are:

   a. Overextension of pack beyond mucogingival junction.

b. Extensive and excessively prolonged exposure and dryness of bone can also induce severe pain.
c. When severe postoperative pain is present, the patient should be treated on an emergency basis. The wound should be examined (under local anesthesia). This type of pain is related to infection accompanied by localized lymphadenopathy and a slight elevation in temperature.

Treatment: Antibiotics and analgesics should be prescribed.

6. *Sensitive Roots/Root hypersensitivity:* May occur spontaneously when the root becomes exposed as a result of gingival recession or pocket formation or it may appear after scaling and root planing and surgical procedures because the cementum at cementoenamel junction is extremely thin and is removed during the above procedures.

Mechanism: Transmission of stimuli from the surface of dentin to the nerve endings is located in the dental pulp, which may occur through the odontoblastic process or owing to a hydrodynamic mechanism. Example: By displacement of fluid. Treatment for root sensitivity include, use of various desensitizing agents like, strontium chloride, potassium nitrate and sodium citrate available in the form of pastes and are used by the patient.

Agents used in Dental Office are:

- Cavity varnishes
- Anti-inflammatory agents
- Various agents are used to partially-obturate dentinal tubules.

Examples:

- Silver nitrate
- Zinc chloride
- Formalin
- Calcium compounds
- Sodium fluoride
- Stannous fluoride
- Strontium chloride, and
- Potassium oxalate

Other procedures like iontophoresis, restorative resins and dentin bonding agents have been used.

HOSPITAL PERIODONTAL SURGERY

Usually, periodontal surgery is performed in dental clinics, either sextant or quadrant wise at weekly or longer intervals. Under certain circumstances, the full mouth periodontal surgery may have to be done in a hospital operating room under general anesthesia. Indications for this, include:

a. To control and manage the apprehensive patient.
b. Convenience for individuals who cannot endure multiple visits to complete surgical treatment.
c. Patient protection—some patients who are suffering from systemic conditions that are not severe enough to contraindicate surgery but at the same time require special precautions that best provided in a hospital setting. Example: Patients with cardiovascular disease, abnormal bleeding tendencies, prolonged steroid therapy and others.

One must clearly understand that the purpose of hospitalization is to protect patients by anticipating their special needs, but not to perform surgery when it is contraindicated by the patient's general condition.

KEYPOINTS

1. Prior to any surgical procedure, every patient must undergo the initial phase of therapy including scaling and root planing and re-evaluation phase.
2. Periodontal surgery must be performed painlessly, to achieve this; effective local anesthesia should be administered.
3. Proper tissue handling is most important, as it can cause postoperative patient discomfort and delayed wound healing.
4. Excessive hemorrhage following surgery should be of great concern to the operator and should be handled carefully. Hemostasis may be achieved with various hemostatic agents such as gelatin sponge, oxidized cellulose and others.
5. The periodontal dressings that are placed on the surgical area have no curative properties; but they help in protecting the tissue and thereby assist in healing.
6. After the pack placement, written and verbal instructions should be given to the patient before he or she leaves the operatory.

KNOW MORE ...

Types of Periodontal Dressings/Packs

- A light curing dressing (e.g. Barricaid)
- Cyanoacrylate dressing.

Advantages of Noneugenol Packs

- Since it does not contain asbestos or eugenol they are safe to use.
- It has better fracture strength than eugenol packs.

REVIEW QUESTIONS

1. Indications and contraindications of periodontal surgery.
2. Describe various suturing techniques in periodontics.
3. Advantages, disadvantages and types of periodontal packs.
4. Treatment for dentinal hypersensitivity.

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39 Chapter

Gingival Curettage

- DEFINITION AND TYPES
- RATIONALE
- INDICATIONS
- PROCEDURE
- HEALING AFTER SCALING AND CURETTAGE
- CLINICAL APPEARANCE AFTER SCALING AND CURETTAGE

DEFINITION

The term curettage is used in periodontics to mean the scraping of gingival wall of a periodontal pocket to separate diseased soft tissue.

Whereas scaling refers to removal of deposits from tooth/root surface and root planing means smoothening the root to remove infected and necrotic tooth surface.

TYPES

I. *Gingival curettage:* Consists of removal of inflamed soft tissue lateral to pocket wall (Fig. 39.1).
   a. *Subgingival curettage:* It is a procedure that is performed apical to epithelial attachment (Fig. 39.2).
   b. *Inadvertent curettage:* Curettage that is done unintentionally during scaling and root planing.

II. Surgical curettage, chemical curettage, ultrasonic curettage.

RATIONALE

The main accomplishment of curettage is the removal of chronically-inflamed granulation tissue that forms in the lateral wall of the periodontal pocket. This tissue apart from having its usual components like fibroblastic and angioblastic proliferations, also contains areas of chronic inflammation, pieces of dislodged calculus and bacterial colonies (Justification to curettage is more so from the fact that this granulation tissue which is lined by epithelium may hamper or act as a barrier for the attachment of new fibers).

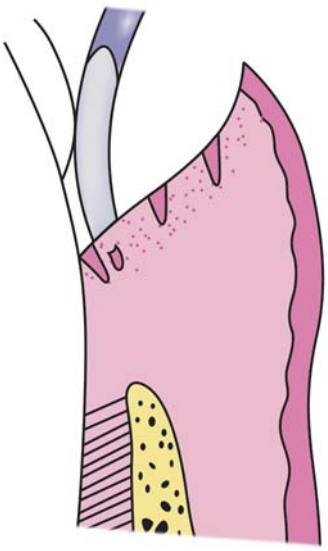


Fig. 39.1: Gingival curettage with a curette

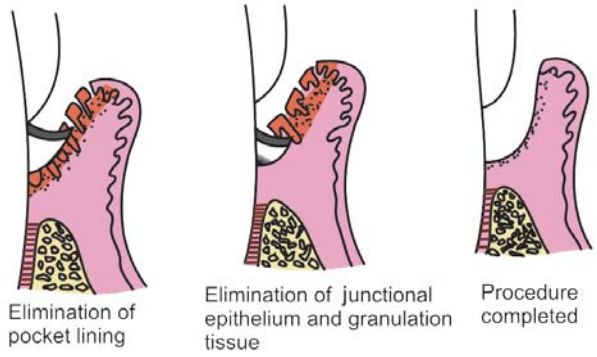


Fig. 39.2: Subgingival curettage

The dilemma now is that, is it justified to do curettage, just to eliminate the inflamed granulation tissue? Because when the root is thoroughly planed, the major source of bacteria disappears and the pathologic changes in the periodontal pocket disappears without any need for curettage. Due to this existing granulation tissue also disappears, if any bacteria present, are destroyed by defence mechanism due to their less number.

On the other hand, curettage may also eliminate all or most of epithelium that lines the pocket wall and underlying junctional epithelium, though there are differing opinions regarding this, the purpose of curettage is still valid particularly in presurgical phase where there is persistent gingival inflammation even after repeated scaling and root planing.

INDICATIONS

1. Can be performed as a part of new attachment in moderately deep infrabony pockets located in accessible areas where a type of "closed surgery" is advised.
2. Can be done as a non-definite procedure to reduce inflammation prior to pocket elimination procedures like flap surgeries.
3. It can also be performed in patients where extensive surgical procedures are contraindicated like aging, systemic complications, etc. where the treatment is compromised and prognosis is impaired.
4. Curettage is frequently performed on recall visits as a method of maintenance treatment for areas of recurrent inflammation and pocket depth, particularly where pocket reduction surgery has previously been performed.

PROCEDURE

- Basic technique.
- Other techniques.

Curettage as such does not eliminate local factors like plaque and calculus, therefore it should always be followed by scaling and root planing procedures.

Basic Technique

After adequate local anesthesia, the correct curette is selected and adapted in such a way that the cutting edge is against the tissues. The instrument is inserted so as to engage the inner lining of the pocket wall and is carried along the soft tissue wall usually in a horizontal stroke. The pocket wall may be supported by gentle finger pressure on external surface.

In subgingival curettage, the tissue attached between the bottom of pocket and the alveolar crest is removed with a scooping motion of the curette to the tooth surface. The area is flushed to remove debris. If necessary sometimes sutures and a pack may be indicated.

Other Techniques

Excisional New Attachment Procedure (ENAP) (Fig. 39.3)

It was developed by United States Naval Corps. It is a definitive subgingival curettage procedure performed with a knife.

The technique is:

1. After adequate local anesthesia, an internal bevel incision is made from margin of free gingiva apically below the base of pocket, it is carried all around the tooth surface, attempting to retain as much interdental tissue as possible.
2. The excised tissue is then removed with a curette and the root surface is planed to a smooth hard consistency.

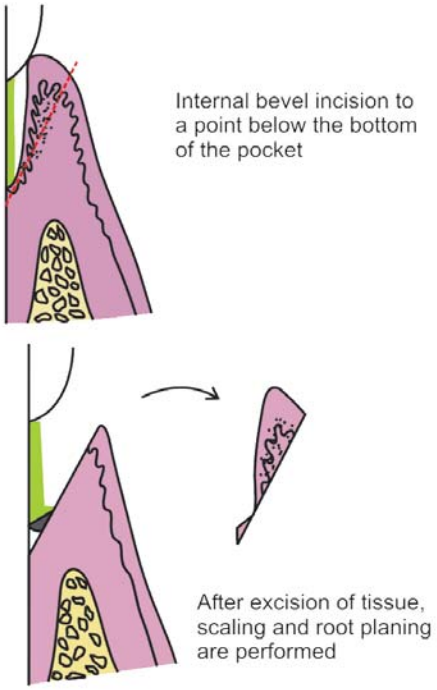


Fig. 39.3: Excisional new attachment procedure (ENAP)

3. Approximate wound edges if necessary, place sutures and a periodontal dressing.

Ultrasonic Curettage

Ultrasonic scalers are used for ultrasonic curettage, here the ultrasonic vibrations disrupt tissue continuity, and the epithelium is lifted off. It also alters the morphologic features of fibroblast nuclei. This method has proved to be as effective as the manual method but results in decreased inflammation and less removal of connective tissue.

Caustic Drugs

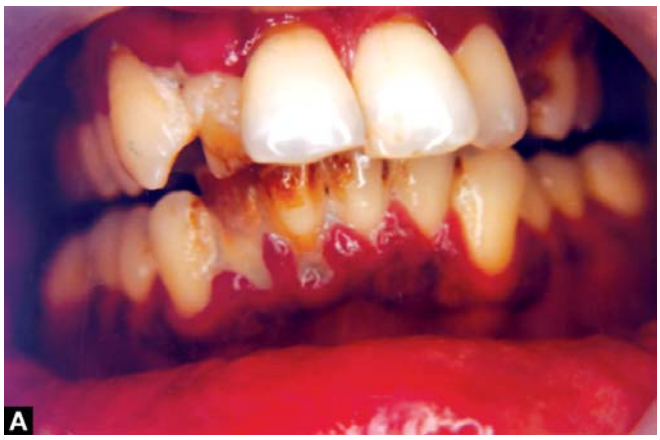
Drugs such as sodium sulfide, antiformin and phenol have been used to induce chemical curettage of the lateral wall of the pocket. Disadvantage is the extent of tissue destruction with these drugs cannot be controlled.

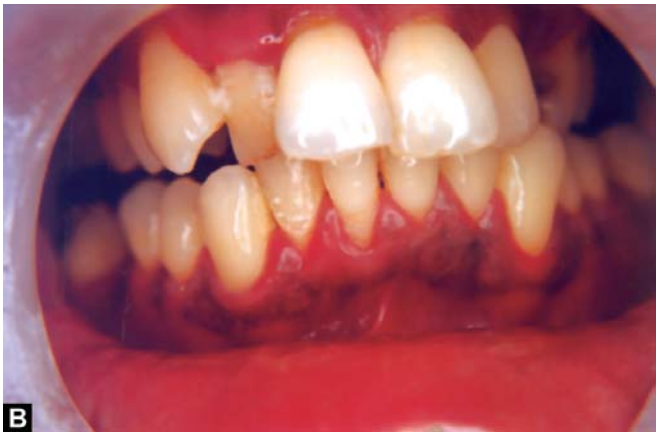
HEALING AFTER SCALING AND CURETTAGE

Immediately after curettage, a blood clot fills the pocket area, hemorrhage is also present in tissues with dilated capillaries and increase in polymorphonuclear leukocytes appear on wound surface. Rapid proliferation of granulation tissue occurs shortly thereafter with a decrease in the number of blood vessel. Restoration and epithelialization of sulcus takes place in 2 to 7 days.

CLINICAL APPEARANCE AFTER SCALING AND CURETTAGE (FIGS 39.4A AND B)

Immediately after curettage, the gingiva appears hemorrhagic and bright-red. After one week, the gingiva





Figs 39.4A and B: Clinical appearance before and after curettage

appears reduced in height with apical shift. The redness is slightly reduced. After two weeks, with proper oral hygiene the gingiva comes back to normal.

KEYPOINTS

1. Curettage is the scraping of the gingival wall of a periodontal pocket to separate the diseased soft tissue.
2. There are gingival, subgingival and inadvertent curettage which should be differentiated.
3. Indications for curettage are very limited and can be used after scaling and root planing.
4. Curettage can also be performed with the help of chemicals and ultrasonic scalers.

REVIEW QUESTIONS

1. What are the indications of gingival curettage?
2. What is ENAP?

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40 Chapter

Gingivectomy

- DEFINITIONS
- PREREQUISITES
- INDICATIONS
- CONTRAINDICATIONS
- TYPES AND TECHNIQUES
- ADVANTAGES AND DISADVANTAGES

INTRODUCTION

The term gingivectomy was coined by Pickerill in 1912 where in all the periodontal tissues including the bone should be removed to achieve healing. It was later modified by Kronfeld (1935) and Orban in about 1940 who introduced modern uses of gingivectomy.

DEFINITIONS

- Gingivectomy is the excision of the soft tissue wall of the pocket (Its objective is the elimination of pockets).
- Gingivoplasty is the recontouring of gingiva that has lost its physiologic form rather than elimination of pockets.

These two procedures are performed together although they may be considered separately for teaching purposes.

PREREQUISITES

The basic prerequisites for gingivectomy are as follows:

1. There should be adequate zone of attached gingiva so that excision of part of it will still leave a functionally adequate zone.
2. The underlying alveolar bone must be in normal or nearly normal form. If there is bone loss it should be of horizontal in nature.
3. There should be no infrabony defects or pockets.

INDICATIONS

If the above mentioned prerequisites are met, gingivectomy may be used to do the following:

1. Eliminate supra-alveolar pockets and abscesses.
2. Remove fibrous or edematous enlargements of the gingiva.
3. Transform rolled or blunted margins to physiologic form.
4. Create more esthetic form in cases in which exposure of the anatomic crown has not fully occurred.
5. Create bilateral symmetry (where the gingival margin of one incisor has receded somewhat more than that of the adjacent incisor).
6. Expose additional clinical crown to gain added retention for restorative procedures (access to subgingival areas, etc.).
7. Correct gingival craters.

CONTRAINDICATIONS

Gingivectomy and gingivoplasty are not indicated in the following situations:

1. In the presence of thick alveolar edges, interdental craters or bizarre crestal bone form.
2. When infrabony pockets are present.
3. If pockets extends till/below the mucogingival junction.
4. Inadequate oral hygiene maintenance by the patients
5. Uncooperative patients.
6. Medically-compromised patients.
7. Dentinal hypersensitivity before the surgical procedure (requires considerable preparation of the patient mentally and is not exactly a contraindication).
8. Esthetically challenging areas, especially in the maxillary anterior region.

TYPES OF GINGIVECTOMY

1. Surgical gingivectomy.
2. Gingivectomy by electrosurgery.
3. Laser gingivectomy.
4. Gingivectomy by chemosurgery.

Surgical Gingivectomy (Figs 40.1 to 40.4)

Technique

Armamentarium:

1. Mouth mirror, probe.
2. Pocket markers, Kirkland and Orban interdental gingivectomy knives.
3. Surgical blade, Bard Parker handle.
4. Surgical curettes, Gracey curettes, tissue forceps, scissors.
5. Periodontal dressing.

Surgical Procedure

Step 1: The pockets on each surface are explored with a periodontal probe and marked with the pocket marker. Insert the probing beak to the bottom of the pocket and mark the depth with the puncturing beak. Mark the bleeding points in all the areas with probing depth.

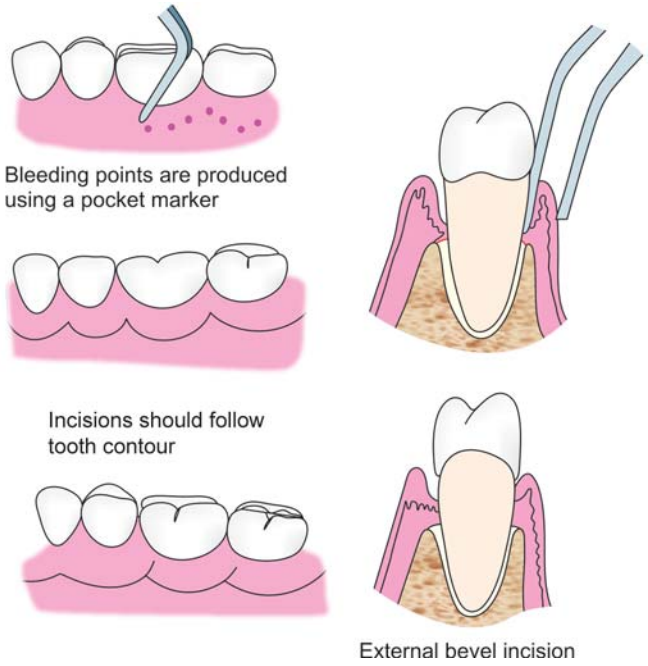


Fig. 40.1: Steps in gingivectomy

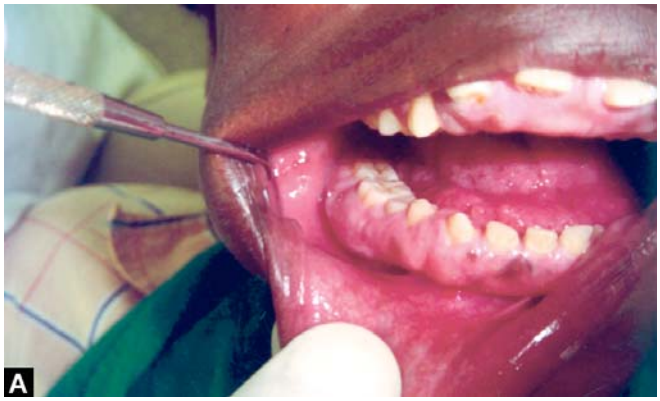
Step 2: Periodontal knives are used for incisions on the facial and lingual surfaces as auxiliary instruments, Bard Parker blades No. 11 and 12 and scissors are then used. The incision is started apical to the points marking the course of the pockets and is directed coronally to a point between the base of the pocket and the crest of the bone. Exposure of bone is undesirable but if it occurs healing may not present a problem if the area is adequately covered by the periodontal pack.

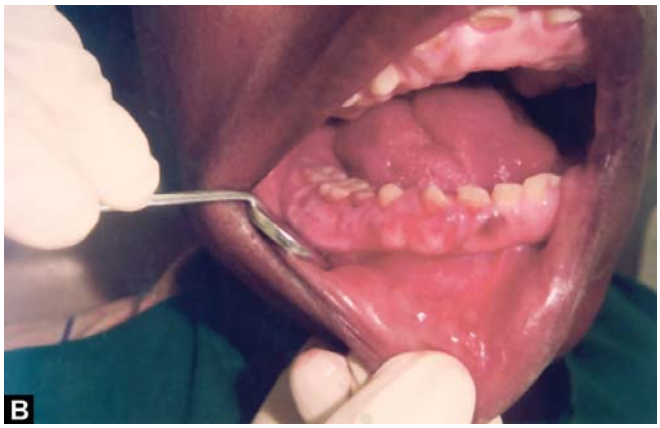
Discontinuous or continuous incisions may be used. The incision should be beveled at approximately 45 degrees to the tooth surface and should recreate as far as possible the normal festooned pattern of gingiva, failure to do this may lead to the formation of fibrous plateau that takes more time to heal and develop a physiologic contour than is ordinarily required.

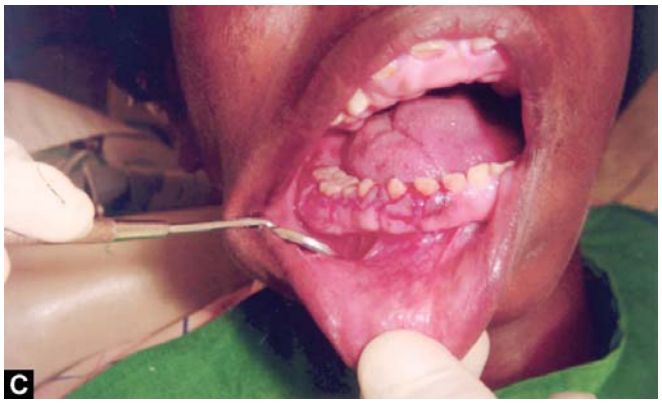
Step 3: Remove the excised pocket wall. Clean the area and closely examine the root surface for any deposits.

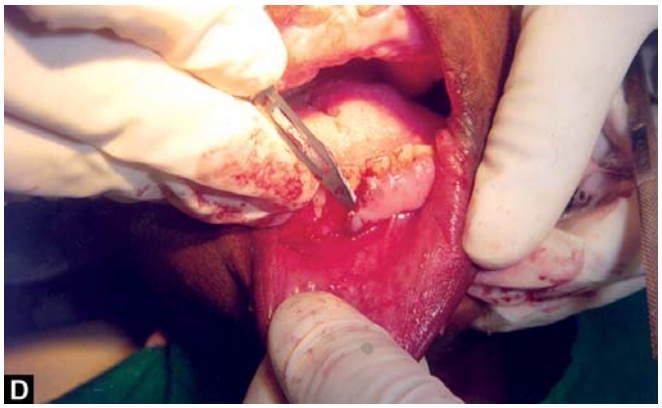
Step 4: Carefully curette out the granulation tissue and remove any calculus and necrotic cementum so as to leave a clean and smooth root surface.

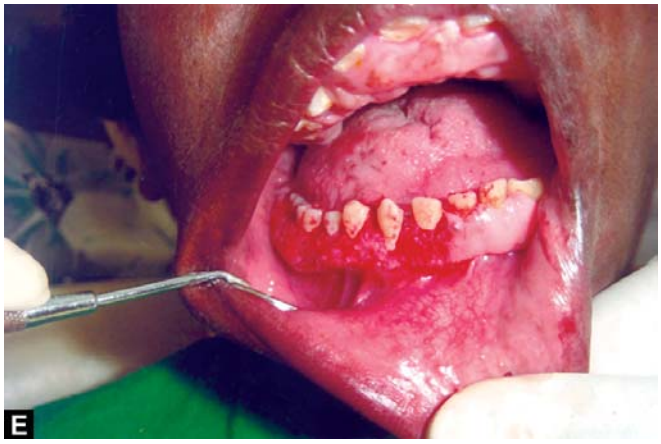
Step 5: Cover the area with surgical pack.



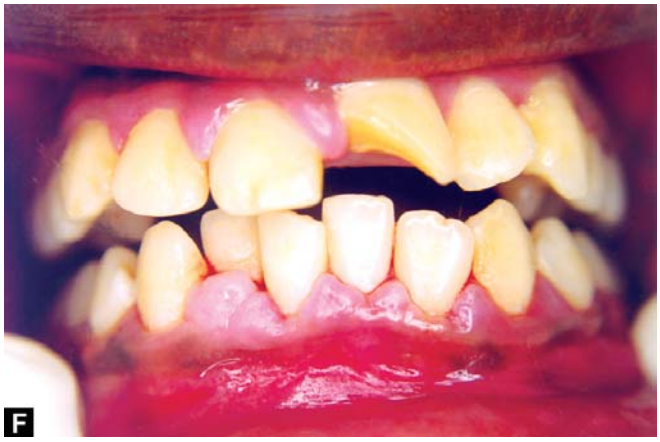


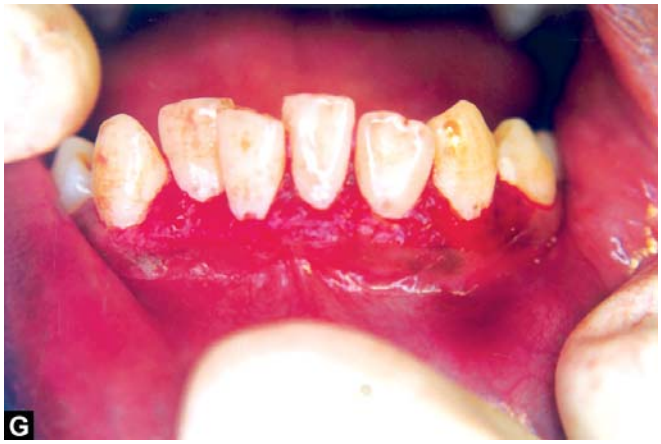




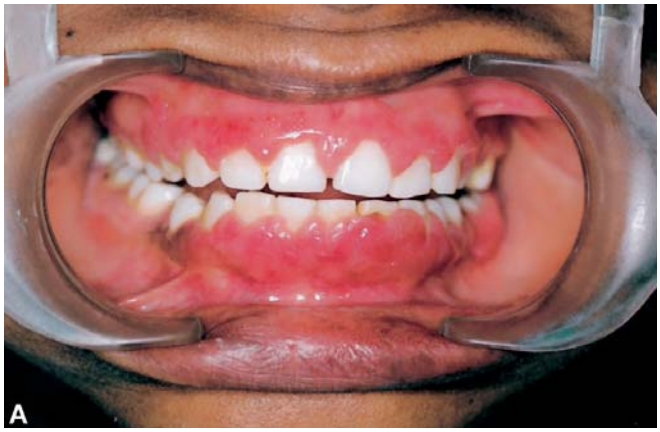


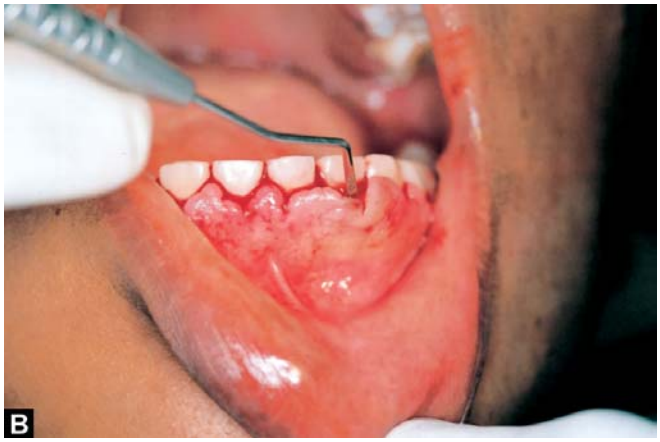
Figs 40.2A to E: Case I clinical steps in gingivectomy. (A) Preoperative view, (B) Bleeding points, (C) External bevel incision, (D) Excision using blade, (E) Immediately after gingivectomy.

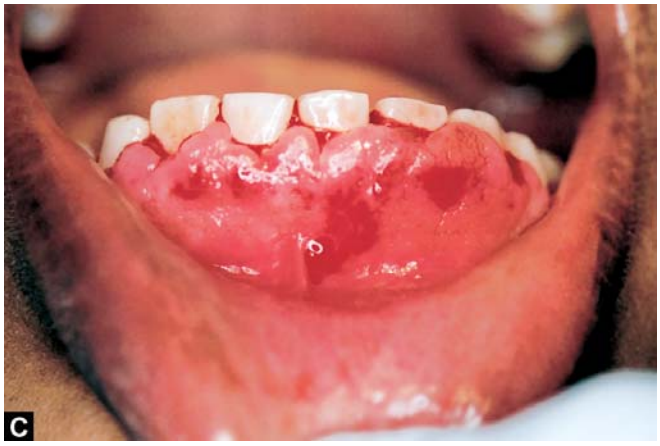


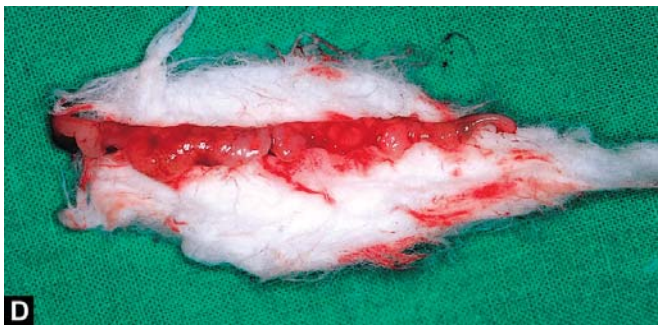


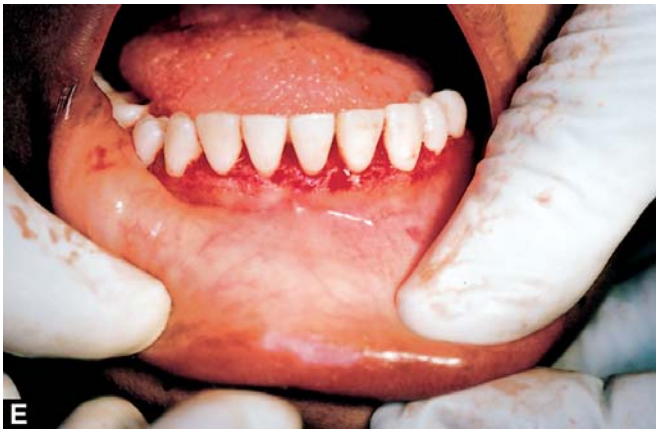
Figs 40.2F and G: Clinical appearance before and after gingivectomy.

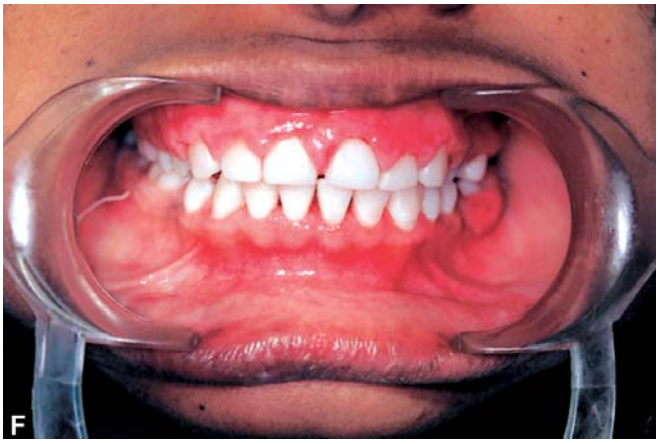


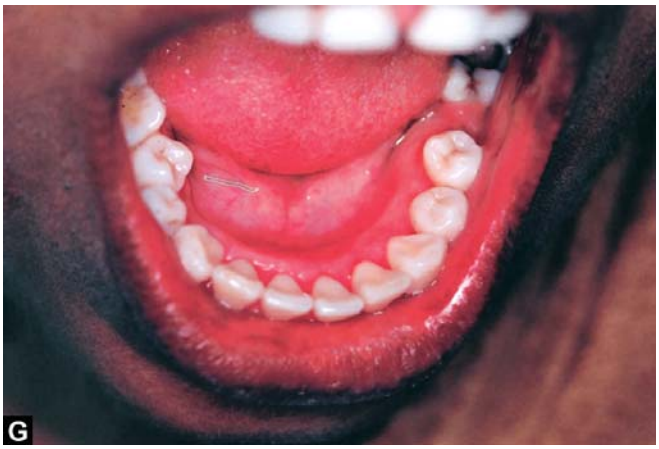








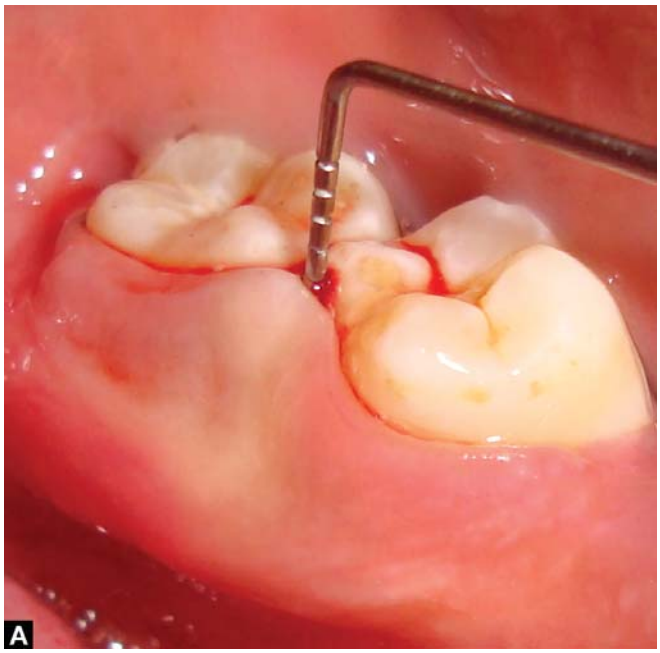


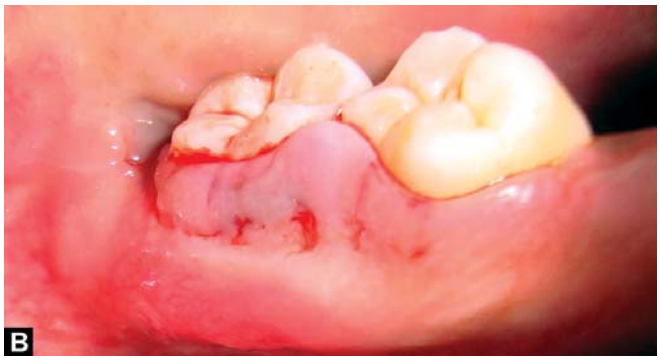


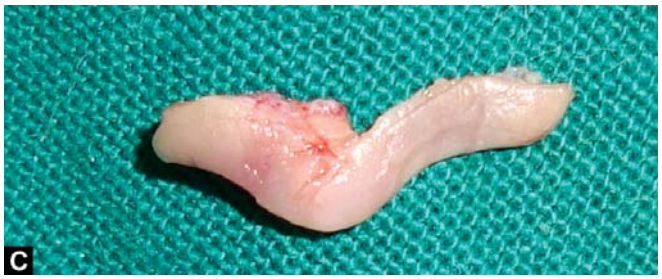
Figs 40.3A to G: Case II gingivectomy. (A) Preoperative view. (B) Probing of pocket depth. (C) Pocket marking. (D) Strip of gingival tissue after gingivectomy. (E) Gingival contouring (immediately after gingivectomy). (F) After healing—facial view. (G) Lingual view

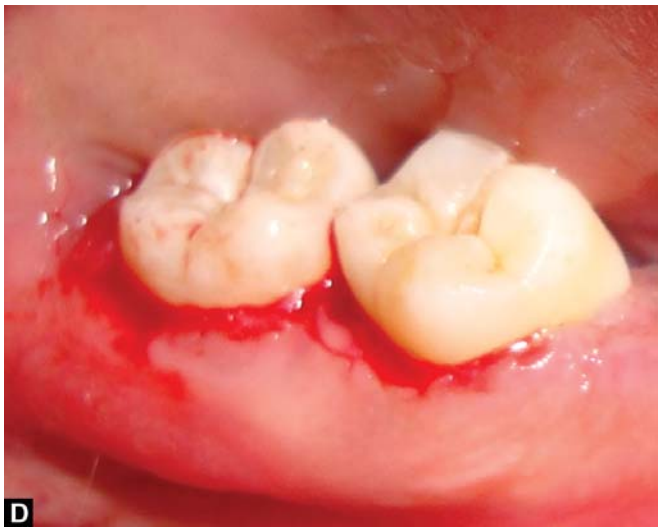
Procedure for Gingivoplasty

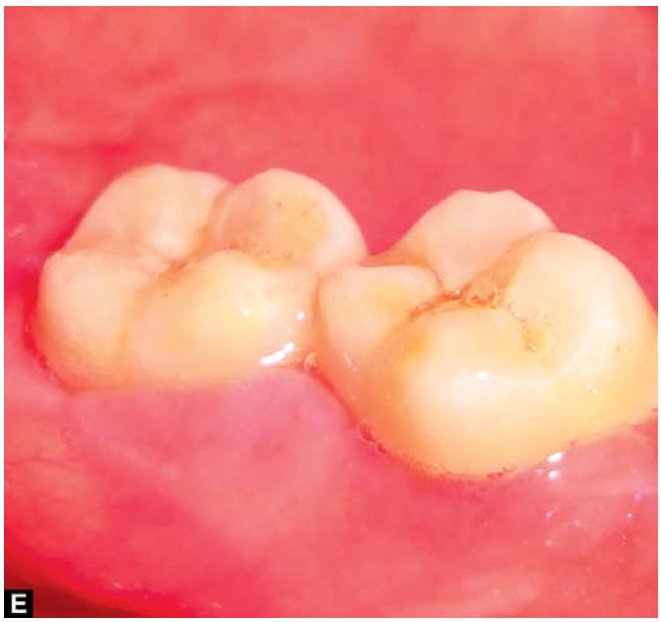
Instruments used are periodontal knife, scalpel, diamond stones or electrodes. It consists of following steps:











Figs 40.4A to E: Case III gingivectomy. (A) Preoperative view with probing of pocket depth. (B) Pocket marking (C) Strip of gingival tissue after gingivectomy. (D) Gingival contouring (immediately after gingivectomy) (E) After healing—Lingual view

a. Tapering the gingival margins.
b. Scalloped marginal outline.
c. Thinning of the attached gingiva and creating vertical interdental grooves.
d. Shaping the interdental papillae to provide sluice ways for the passage of food.

Healing after Surgical Gingivectomy

Basically healing is by secondary intention:

a. The initial response is the formation of a protective surface clot.
b. The clot is then replaced by granulation tissue.

c. Within 24 hours, there is an increase in new connective tissue cells mainly angioblasts and by third day numerous fibroblasts are located in this area.

The highly vascular granulation tissues grow coronally, creating a new free gingival margin and sulcus.

d. Capillaries derived from blood vessels of periodontal ligament migrate into the granulation tissue, and with in two weeks they connect with gingival vessels.

Surface epithelialization is generally complete after 5 to 14 days. Initially, keratinization is less than what it was prior to surgery. Complete epithelialization takes about 1 month.

Electrosurgery (Surgical Diathermy)

Uses high frequency current of 1.5 to 7.5 million cycles per second.

There are three classes of electrodes used:

1. Single wire electrodes for incising and excising.
2. Loop electrodes for planing tissues.
3. Heavy bulkier electrodes for coagulation procedures.

Four types of electrosurgical techniques are available:

a. *Electrosection:* Three procedures are performed incising, excising and planing.
b. *Electrocoagulation:* Used to prevent hemorrhage.
c. *Electrofulguration:* Uses high voltage current. It has limited application in dentistry.
d. *Electrodesiccation:* Uses dehydrating current and least used, as it is a dangerous technique.

Here the active electrode is inserted into the tissue and the tissue surrounding the electrode is mass coagulated in situ. This procedure is useful only in dermatological and cancer surgeries.

Gingivectomy by Electrosurgery

Advantage

Permits adequate contouring of the tissues and controls hemorrhage.

Disadvantages

1. Cannot be used in patients with poorly shielded cardiac pacemaker.
2. Causes unpleasant odor.
3. If it touches the bone irreparable damage may result.
4. Heat generated by this may cause tissue damage and areas of cemental necrosis.

Indications

1. Removal of gingival enlargements.
2. Gingivoplasty.
3. Relocation of frenum and muscle attachments.
4. Incision of periodontal abscesses and pericoronal abscess.

Technique

a. *For gingivoplasty:* Needle electrodes and diamond-shaped electrodes are used for festooning. In all reshaping procedures electrodes are activated and moved in a concise "Shaving" motion (Cutting and coagulating current is used).
b. *For abscess drainage:* Incisions can be made with the needle electrode.
c. *For hemostasis:* Ball electrode is used.
d. *For relocation of frenum and muscle attachment:* Loop electrode is used.

Healing after Electrosurgery

Some investigators report no significant differences but others however have reported delayed healing, greater reduction in gingival height and more bone injury after electrosurgery.

Laser Gingivectomy

Most commonly used lasers are carbon dioxide and Nd:YAG lasers. They are used for excision of gingival over growth. Their use in periodontal surgery is not supported by research.

Gingivectomy by Chemosurgery

Chemicals used are 5 percent paraformaldehyde or potassium hydroxide to remove gingiva.

Disadvantages

1. Their depth of action cannot be controlled hence it may also injure normal tissues.
2. Gingival remodeling is not possible.
3. Healing is delayed.

KEYPOINTS

1. Excision of soft tissue wall of the pocket is called gingivectomy.
2. Gingivectomy may be performed by means of scalpels, electrodes, laser beams or chemicals.
3. In surgical gingivectomy, the incision should be beveled at approximately 45 degrees to the tooth surface and should recreate normal festooned pattern of the gingiva.
4. Gingivectomy should be differentiated from gingivoplasty, in that, it is performed mainly to eliminate the pocket and also includes reshaping whereas the latter is performed for reshaping the gingiva to create normal physiological contour.
5. Basic healing after gingivectomy is by secondary intention.
6. The main advantage of laser and gingivectomy by electrosurgery is contouring of the tissue.

REVIEW QUESTIONS

1. Define gingivectomy and describe the step by step procedure of surgical gingivectomy.
2. Describe various gingivectomy techniques.
3. Describe the indications and contraindications of gingivectomy.
4. Describe healing after gingivectomy.

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42 Chapter

Osseous Surgery

DEFINITION

Osseous surgery may be defined as the procedure by which changes in the alveolar bone can be accomplished to rid it of deformities induced by the periodontal disease or other related factors, such as exostosis and tooth supraeruption.

RATIONALE

It is based on the fact that the discrepancies in levels and shapes of the bone and gingiva predisposes patients to the recurrence of pocket depth post-surgically. Hence, the goal of osseous resective therapy is reshaping the marginal bone to resemble the alveolar process undamaged by periodontal disease. The technique usually involves apically-displaced flaps hence the procedure not only eliminates periodontal pockets but also improves tissue contour to provide a more easily maintainable environment.

TERMINOLOGY

1. *Osteoplasty*—It refers to reshaping the bone without removing the bone supporting the tooth.
2. *Ostectomy*—It refers to removal of bone supporting the tooth.
3. Osseous surgery—It is a periodontal surgery involving modification of the bony support of the teeth.

   According to the American Academy of Periodontology; it is defined as "Procedures to modify bone support altered by periodontal disease, either by reshaping the alveolar process to achieve physiologic form, without the removal of the alveolar supporting bone, or by the removal of some alveolar bone, thus changing the position of crestal bone relative to the tooth root."
4. Osteoplasty—It is defined as reshaping of the alveolar process to achieve a more physiologic form without removal of supporting bones.
5. Ostectomy—It is defined as the excision of bone or portion of a bone in periodontics, ostectomy is done to correct or reduce deformities caused by periodontitis and includes removal of the supporting bone.

TYPES OF OSSEOUS SURGERY

Depending on the relative position of the interdental bone to radicular bone, osseous surgery is of following types (Fig. 42.1):

1. *Positive architecture*—When the radicular bone is apical to the interdental bone.
2. *Negative architecture*—If the interdental bone is more apical than the radicular bone.
3. *Flat architecture*—It is the reduction of interdental bone to the same height as radicular bone.
4. *Ideal*—When the bone is consistently more coronal on the interproximal surface than on the facial and lingual surfaces.

Depending on the thoroughness of the osseous reshaping techniques, osseous surgery is of following types:

1. *Definitive osseous reshaping*—Implies that further reshaping would not improve the overall result.
2. *Compromise osseous reshaping*—It indicates a bone pattern that cannot be improved without significant osseous removal, that would be detrimental to the overall result.

Osseous surgery (Fig. 42.2) can also be:

1. *Additive*—Directed towards restoring the bone to original levels.
2. *Subtractive*—It is designed to restore the form of the pre-existing alveolar bone to the level existing at the time of surgery or slightly apical to this level.

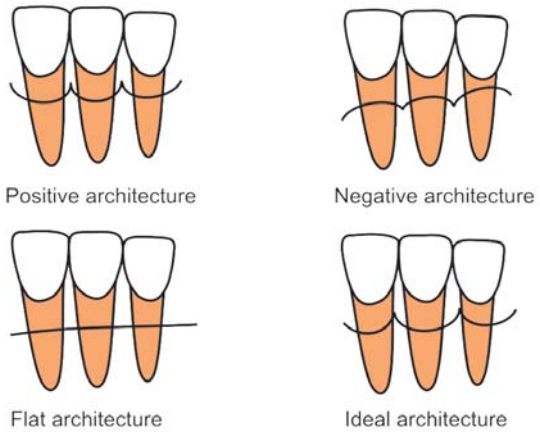


Fig. 42.1: Relative position of interdental bone to radicular bone

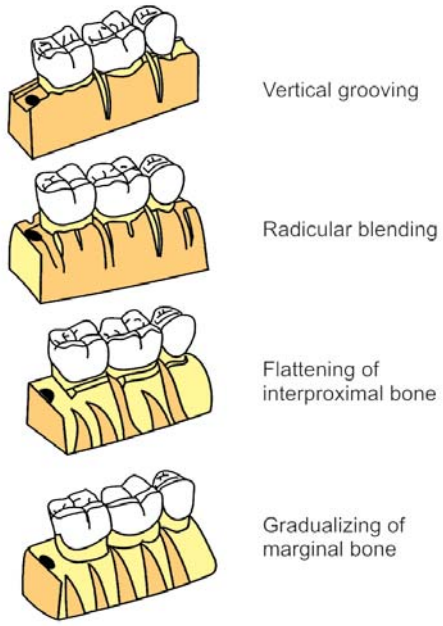


Fig. 42.2: Steps in osseous resective surgery

RESECTIVE OSSEOUS SURGERY

Indications

1. One-walled angular defects.
2. Thick, bony margins.
3. Shallow crater formations.

Contraindications

1. Anatomic factors such as close proximity of the roots to the maxillary antrum or the ramus.
2. Age.
3. Systemic health.
4. Improper oral hygiene.
5. High caries index.
6. Extreme root sensitivity.
7. Advanced periodontitis.
8. Unacceptable esthetic result.

Examination Prior to Surgery

Clinical examination and probing determines the presence and the depth of periodontal pockets and also gives a general sense of the bony topography. Transgingival probing or

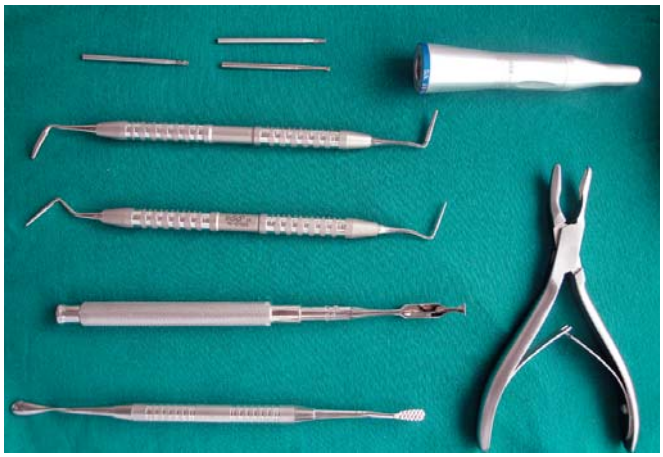


Fig. 42.3: Resective osseous surgery instruments

sounding under local anesthesia confirms the extent and configuration of the infrabony component or furcation defects.

Methods

Instruments used —Combination of hand and rotary instruments are used (Fig. 42.3).

Hand instruments include:

1. Rongeurs-Friedman and Blumenthal.
2. Interproximal files—Schluger and Sugarman.
3. Back action chisels.
4. Oschsenbein chisels.

Rotary instruments include:

1. Carbide round burs.
2. Slow-speed handpiece.
3. Diamond burs.

TECHNIQUE

The following steps are suggested:

1. Vertical grooving.
2. Radicular blending.
3. Flattening of interproximal bone.
4. Gradualizing marginal bone.

Not all the steps are necessary in each case.

Vertical Grooving

It is indicated to reduce the thickness of alveolar housing and it provides continuity from the interproximal surface into the radicular surface. It is the first step of the resective process and is usually performed with rotary instruments such as, round, carbide or diamond burs.

It is indicated in thick bony margins, shallow crater formation and is contraindicated in areas with close root proximity or thin alveolar housing.

Radicular Blending

It is the second step of the osseous reshaping technique. It is the continuation of the first step and it attempts to gradualize the bone over the entire radicular surface and thereby provides a smooth, blended surface for good flap adaptation.

The indications are the same as in step one. Both step one and step two are purely osteoplastic procedures. Apart from thick bony margins and craters, grade-I and grade II furcation involvements are treated with these two steps.

Flattening of the Interproximal Bone

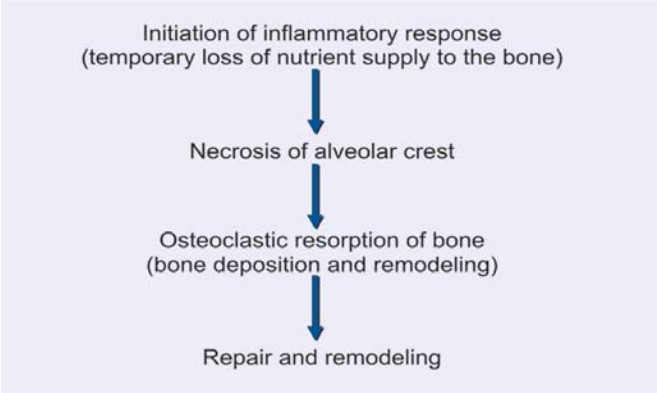
It requires removal of very small amount of supporting bone. It is indicated when interproximal bone levels vary horizontally, e.g. one wall defects or hemiseptal defects.

This step is also best-utilized in areas where there are combined defects, i.e. coronally one-walled defect and apically three-walled defect, so that it is helpful in obtaining good flap closure and hence improved healing. But the main limitation is that, it cannot be utilized in advanced defects where removal of inordinate amounts of bone may be required.

Gradualizing Marginal Bone

The final step in the osseous resective technique, is also an ostectomy procedure. Bone removal is minimal but necessary to provide a sound regular base for the gingival tissue to follow. Failure to do so may result in 'widow's peaks', allows the tissue to rise to a higher level than the base of the bone loss in the interdental area. This may result in selective recession and incomplete pocket reduction. This ostectomy procedure should be performed with great care so as to not damage the roots. Following the osseous resection, the flaps are positioned back to its original position or apically and sutured with minimal tension.

Healing after Surgery



The osteoblastic activity has been observed even after 1 year postoperatively in humans, hence the initial loss in bone height gets compensated to some extent by repair and remodeling.

RECONSTRUCTIVE OSSEOUS SURGERY

Periodontal therapy for treatment of periodontitis involves the elimination of bacterial plaque. When periodontitis is resolved, an anatomic defect remains in the periodontium. This anatomic defect is characterized by reformation of gingival fibers, substantial reduction in inflammation, persistent loss of bone and ligament and the formation of long junctional epithelium.

Thus, periodontal therapy involves two primary components: Elimination of bacterial plaque and elimination of the anatomic defects produced by periodontitis. There are two primary approaches to eliminating these anatomic defects—resective and regenerative, both being surgical.

Evaluation of New Attachment and Bone Regeneration

Clinical Methods

Consist of:

1. Probing depth measurements (pre- and post-treatment).
2. Clinical gingival indices.
3. Determination of attachment level, care should be taken to measure the defect by placing the probe at the exact same point before and after treatment and also with the same angulation. Pre- and postoperative probing measurements without standardized method may not be reliable.

Radiographic Methods

They also have the following drawbacks. Standardized techniques for reproducible positioning of the film and tubing is very difficult. Even with standardization, the radiograph may not show the entire topography of the area before or after the treatment. Furthermore, thin bony trabeculae which was present before treatment may go undetected because minimal amount of mineralized tissue must be present to be seen or registered on the radiograph. Only future studies with subtraction radiography will enhance the usefulness of radiographic evaluation.

Surgical Re-entry

This can give the best view of the state of the bone crest that can be compared with the one taken during the initial surgical intervention and can also be subjected to measurements. Even models can be used to appreciate the results of the therapy (pretreatment and post-treatment). This method is very useful but has two shortcomings:

a. It requires a frequently unnecessary second operation.
b. It does not show the type of attachment that exists (epithelial or connective tissue attachments).

Histological Methods

The type of attachment can only be determined by the histologic analysis of the tissue block taken from the healed area. Although this can give us the clear picture of regeneration of the attachment apparatus, it is not without problems. They are:

a. The need to remove a tooth with its periodontium treated successfully limits the volunteers.
b. Animal models (monkey, dog, pigs) can be used to clarify some aspects and but one must always remember the differences that exists when extrapolations to humans are attempted. Mainly the exact nature of the periodontal disease cannot be reproduced in animals and also bone defects have to be experimentally-induced which may lack their chronicity and self-sustaining features. Even if these defects are allowed to become chronically-

infected, they are never identical because all these types of lesions have different healing sequences and thereby provides different types of information.

In addition, the exact location of epithelial attachment will be lost once you reflect the flap because it will be reflected beyond the normal periodontium. Hence, a notch should be placed on the root surface either apical to the calculus (slightly coronal to the attachment) or crest of the alveolar bone (slightly apical to the attachment). Base of the calculus is a better landmark (for which presence of calculus is required).

The following reconstructive surgical techniques have been proposed.

1. Nongraft-associated new attachment.
2. Graft-associated new attachment .
3. Combination of both.

Nongraft-associated New Attachment

New attachment can be achieved without the use of grafts in:

a. Meticulously treated three-walled defects (Infrabony defect).
b. Perioendodontal abscesses.
c. When the destructive procedure has occurred very rapidly, for example, after treatment of pockets which had acute periodontal abscess.

Various techniques of nongraft-associated new attachment are:

Removal of Junctional and Pocket Epithelium

The methods used to do so include:

i. *Curettage*—Only 50 percent of junctional epithelium and pocket epithelium can be removed.
ii. *Chemical agents*—Mostly used in conjunction with curettage. The most commonly used drugs are sodium sulfide, phenol, camphor, sodium hypochlorite and antiformin. The main disadvantage is that the depth of action cannot be controlled.
iii. *Ultrasonic methods*—It is again not very useful, because of lack of clinicians tactile sense while using these methods.
iv. *Surgical methods*—
- Excisional new attachment procedure with internal bevel incision (ENAP).
- Gingivectomy procedure.
- Modified Widman flap.
- Coronal displacement of the flap.

All these procedures are discussed elsewhere in this book.

Prevention of Epithelial Migration

Eliminating junctional and pocket epithelium may not be sufficient because the epithelium from the excised margin may rapidly proliferate apically to become interposed between the healing connective tissue and cementum.

Guided Tissue Regeneration (GTR)

This concept is based on the assumption that periodontal ligament cells have the potential for regeneration of the attachment apparatus of the tooth.

Two types of membranes have been used:

a. *Degradable*—Collagen, Polylactic acid, Vicryl (polyglactin 910) and Guidor membrane.
b. *Nondegradable*—They must be removed in three to six weeks time, e.g. Millipore, Teflon membrane, Goretex periodontal material.

Surgical procedure (Figs 42.4 to 42.7)

Step 1: Raise a full thickness flap utilizing vertical incisions, extending a minimum of two teeth anteriorly and one tooth distally, to the tooth being treated.

Step 2: Debride the osseous defect and plane the root surfaces.

Step 3: Trim the membrane according to the size of the area being treated. The membrane should extend approximately beyond 2 to 3 mm on all the sides.

Step 4: Suture the membrane around the tooth with a sling suture.

Step 5: The flap is positioned back to its original position or slightly coronal to it and is sutured using interrupted sutures. Make sure the membrane is covered completely. In case of non-resorbable membrane, after 5 weeks of the operation, it must be removed with a gentle tug.

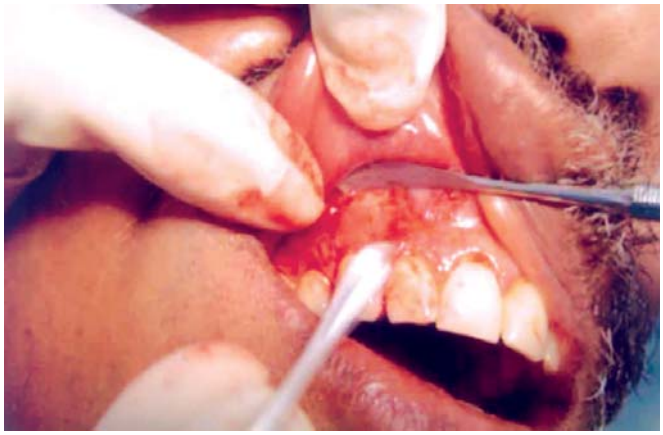


Fig. 42.4: Guided tissue regeneration—adaptation of barrier membrane on the defect

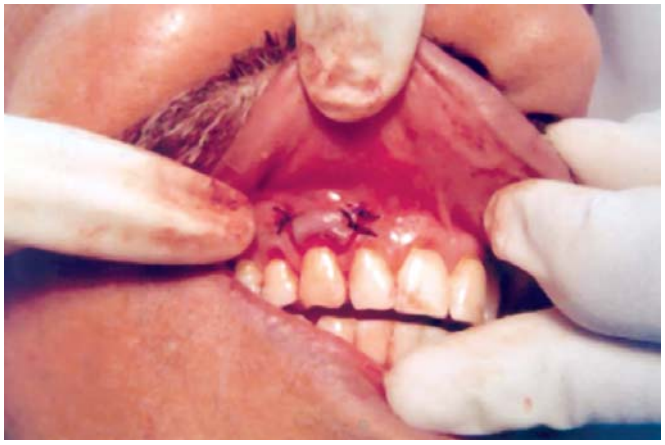


Fig. 42.5: Flap is positioned coronally and sutured

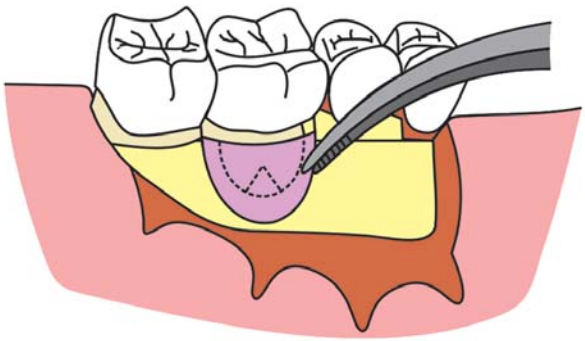


Fig. 42.6: Membrane adaptation

Clot Stabilization, Wound Protection and Space Creation

The successful results obtained with graft materials, barrier membranes and coronally-displaced flaps have been attributed to the fact that all of these protect the wound and

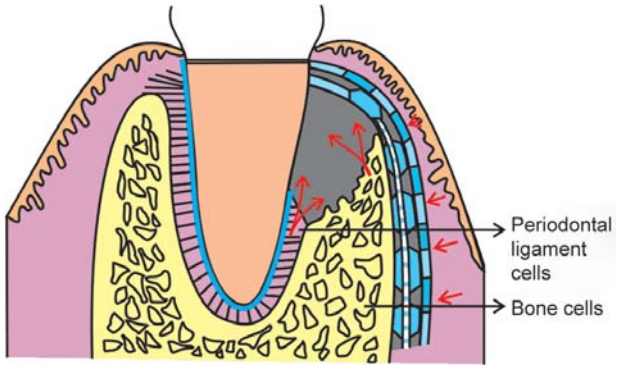


Fig. 42.7: Membrane barrier in place

create a space for undisturbed and stable maturation of the clot. Hence, this hypothesis suggests that preservation of the root surface that is, a fibrin clot interface prevents apical migration of the gingival epithelium and allows for connective tissue attachment during the early wound healing period. A lot of research is required to explore this possibility, for example, it requires more postoperative care, root conditioning to enhance fibrin clot and connective tissue attachment.

Preparation of the Root Surface (Root Biomodification) (Fig. 42.8)

Several substances have been used to condition the root surface, for attachment of new connective, tissue fibers. These include citric acid, fibronectin and tetracycline.

Citric acid: When used with pH1 for two to three minutes on root surface, after surgical debridement, it produces a surface demineralization, which inturn induces cementogenesis and attachment of collagen fibers. The following actions of citric acid have been reported by Register and Burdick in 1975.

1. It removes the smear layer and may open dentinal tubules, thus allowing cementum to form within these tubules creating the blunderbuss effect and produce cementum pins. This could be associated with accelerated cementogenesis.
2. It has also been shown to expose collagen fibers on the root surface, which may splice with the collagen fibers of a soft tissue graft or flap (called as collagen splicing) resulting in collagen adhesion without cementum formation and accelerated healing.

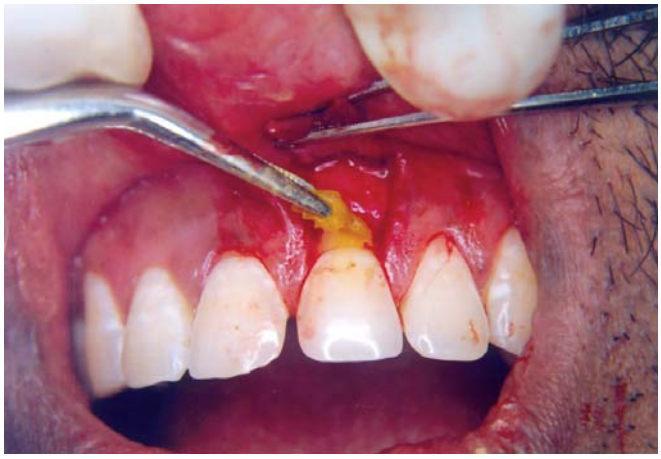


Fig. 42.8: Root biomodification using tetracycline solution

3. Epithelium does not migrate apically because of the accelerated healing either by connective tissue attachment or a collagen adhesion may occur before epithelium migrates.
4. Finally, citric acid, may demineralize small bits of residual calculus, disinfect the root surface and aid in removing endotoxins.

Steps involved:

a. Raise full thickness flap.
b. Perform thorough root planing.
c. Apply cotton pellets soaked in citric acid pH1 for two to three minutes.
d. Remove and irrigate root surface profusely with water.
e. Replace the flap and suture.

Growth factors: They are polypeptide molecules released by the cells in the inflamed area, that regulates events in wound healing. These factors are primarily secreted by macrophages, endothelial cells, fibroblasts and platelets. They include platelet derived growth factor (PDGF), insulin-like growth factor (IGF), fibroblast growth factor (FGF) and TGF (transforming growth factor alpha and beta). These can all be used to control events during periodontal wound healing, e.g. promoting proliferation of fibroblasts from periodontal ligament thereby favoring bone formation.

Enamel matrix proteins: This is based on the observations that amelogenin secreted by Hertwig's epithelial root sheath during tooth development can induce acellular cementum formation which is believed to favor periodontal regeneration, e.g. Emdogain approved by FDA.

Graft-associated New Attachment

Terminology

1. *Graft:* It is a viable tissue/organ that after removal from donor site is implanted/transplanted within the host tissue, which is then repaired, restored and remodelled.
2. *Xenograft or heterograft:* The donor of the graft is from a species different from the host.
3. *Allograft or homograft:* A tissue transfer between individuals of the same species but with non-identical genes.
4. *Autograft:* A tissue transfer from one position to a new position in the same individual.
5. *Alloplastic graft:* A graft of inert synthetic material which is sometimes called implant material.
6. *Osteoinduction:* A process by which the graft material is capable of promoting cementogenesis, osteogenesis and new periodontal ligament.
7. *Osteoconduction:* The graft material acts as a passive matrix, like a trellis or scaffolding for new bone to cover.
8. *Contact inhibition:* The process by which the graft material prevents apical proliferation of the epithelium.
9. *Guided tissue regeneration:* An epithelial exclusionary technique that promotes new connective tissue attachment without the use of any implant material.

Ideal Requirements of a Bone Graft Material

An ideal bone graft material should have biologic acceptability, predictability, clinical feasibility, minimal postoperative hazards, minimal postoperative sequelae and good patient acceptance.

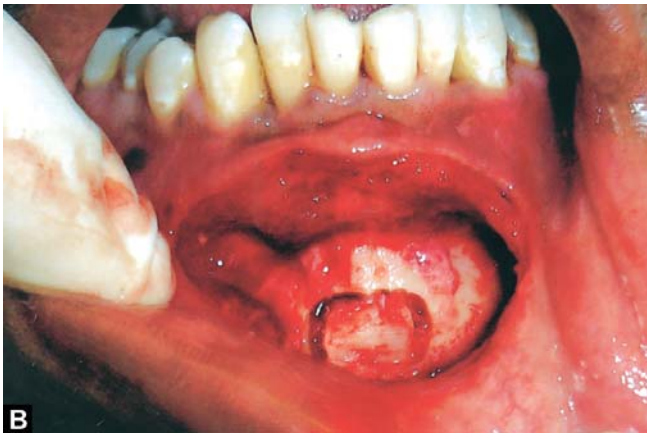
To date there is a no single material that fulfills all the above criteria. Once the material is placed in the defect it may act in a number of ways. It may have no effect, act only as a scaffolding material for the host to lay down new bone, it may actively induce bone formation or through its own validity it may deposit new bone in the defect.

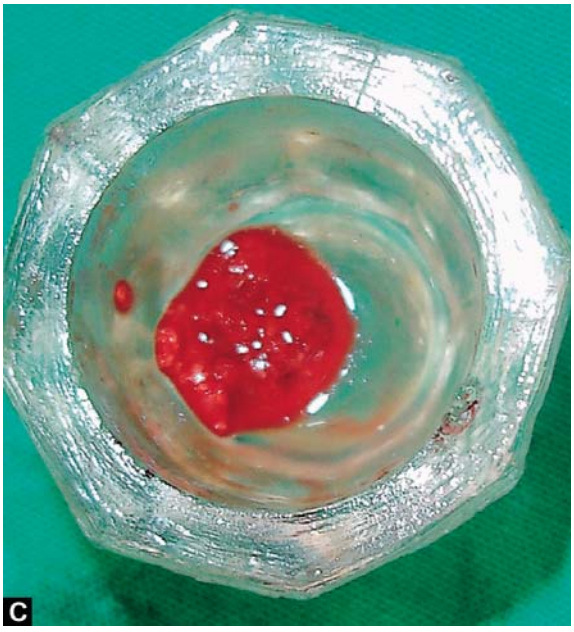
All grafting techniques require presurgical scaling, occlusal adjustment as needed and exposure of defect with full thickness flap, best-suited is papilla preservation flap because it provides complete coverage of the interdental area after suturing. The use of antibiotics after the procedure is generally recommended.

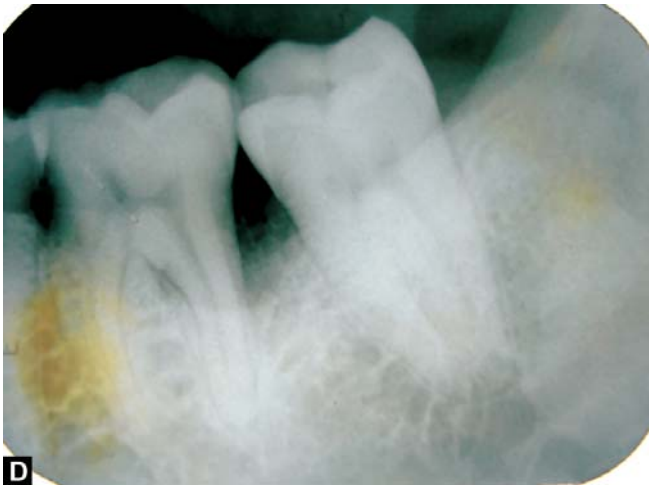
Intraoral autograft: It is the tissue transfer from one area to another in the same individual, sources of intraoral autografts include healing extraction wound, bone from edentulous ridges, immature bone removed during osteoplasty and ostectomy, bone removed from a predetermined site like tori and symphysis (Figs 42.9A to D).

Osseous coagulum: Rationale of this technique is that small particles of donor bone are better resorbed and replaced than the larger particles. This technique uses small particles of donor bone and hence it provides additional surface area for the interaction of cellular and vascular elements. Sources of the implant material include the lingual ridge on the mandible, exostosis, tori, edentulous ridges, the bone distal to the terminal tooth.

In this technique, a bur is used in the donor site to reduce it to small particles which when coated with blood becomes coagulum and is placed in the defect until there is considerable excess and the flap is replaced.







Figs 42.9A to D: Autogenous bone grafting. (A) Preoperative radiograph. (B) Harvesting of bone graft from symphysis region. (C) Harvested autogenous cancellous bone graft. (D) Postoperative radiographic view (after 3 months)

Disadvantages:

1. Low predictability.
2. Inability to procure adequate material.
3. Inability to use aspiration for large defects which leads to poor surgical visibility.

Bone blend: To overcome the above-mentioned problems, bone blend technique has been proposed. It uses an autoclaved plastic capsule and pestle. Bone is removed from the predetermined site with chisels or rongeur forceps, placed in the capsule with a few drops of saline, and triturated for sixty seconds to a workable plastic-like mass and is packed into the bony defect.

Intraoral cancellous bone marrow chips: It can be obtained from:

a. *Maxillary tuberosity*—It contains good amount of cancellous bone with foci of red marrow and the bone is removed with a cutting rongeur.
b. *Edentulous areas*—The bone is removed with curette.
c. *Healing sockets*—They are allowed to heal for eight to twelve weeks and the apical portion is utilized as donor material.

Bone swaging: This technique requires presence of an endentulous area adjacent to the defect from which the bone is pushed into contact with root surface without fracturing the bone at its base.

Disadvantages:

i. It is technically difficult.
ii. Its usefulness is limited.

Bone from extraoral sites:

Iliac autografts/extraoral hip marrow: The use of iliac cancellous bone marrow has shown good results in bony defects with varying number of walls and furcation defects. However, there are many disadvantages associated with it.

i. Additional surgical trauma.
ii. Postoperative morbidity infection, exfoliation, sequestration.
iii. Root resorption.
iv. Rapid recurrence of the defect.

Hence, this technique is no longer used.

Allografts: Allograft or homograft is the tissue transfer between individuals of the same species, but of non-identical genetic composition. Since autografts induce surgical trauma in another part of the patient's body, it would be advantageous if a suitable substitute can be obtained commercially. Bone grafts are commercially available in tissue banks.

They can be:

1. FDBA (Freeze dried bone allograft)—It is an osteoconductive material, varying results have been observed using this material. Average bone fill of 50 percent has been reported.
2. DFDBA (Demineralized freeze dried bone allograft)—Demineralization process exposes the components of bone matrix termed as bone morphogenic protein, e.g. Dembone™, which is a bone inductive protein isolated from the extracellular matrix of human bones. Hence, this is an osteoinductive material (Fig. 42.10).

Xenografts: They have been shown to cause severe immunologic reactions because of molecular divergencies, therefore it is not used any longer. Examples of xenograft are calf bone, kiel bone, anorganic bone.

Alloplasts/non-bone graft material: Non-bone graft materials have also been used for restoration of the periodontium. Some of them are sclera, dura, cartilage, plaster of Paris, ceramics, and coral derived materials.

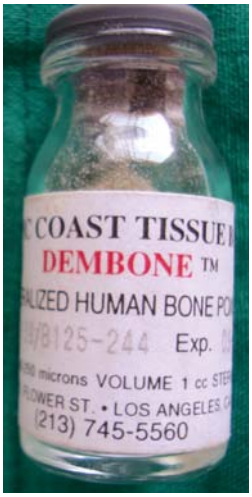
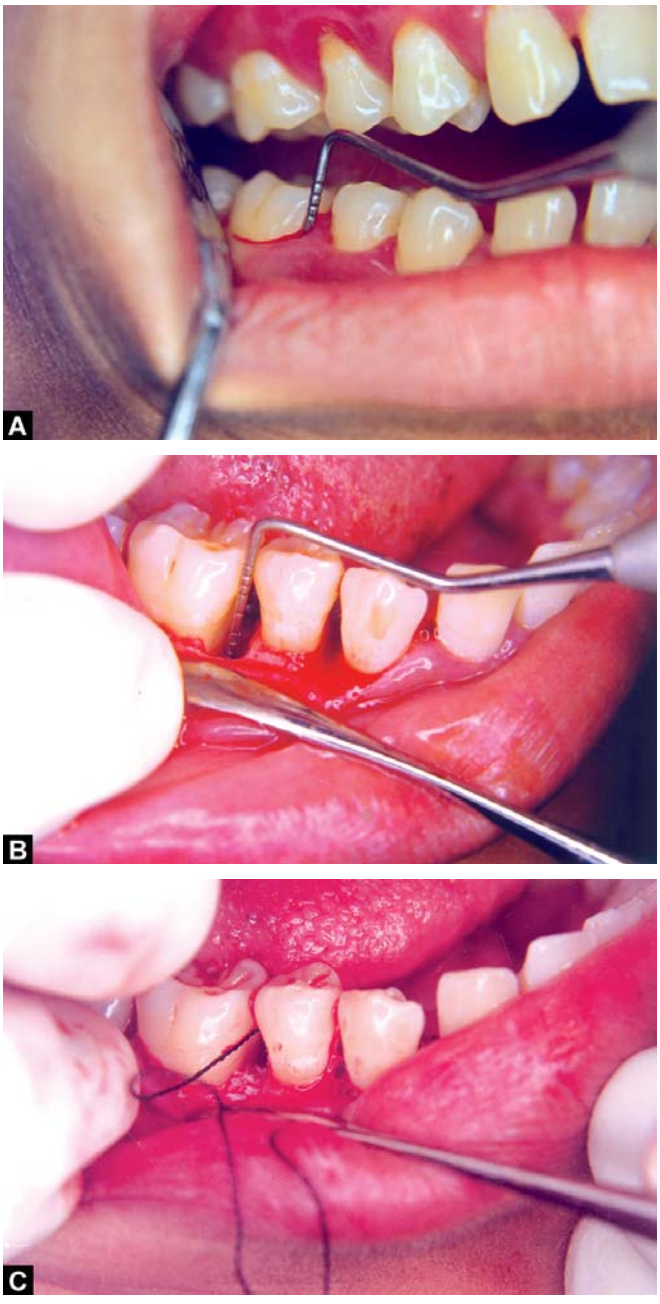


Fig. 42.10: Dembone™ (Demineralized freeze dried bonegraft)

All alloplastic materials have shown more or less similar results because all of them are osteoconductive in nature. Lot of attention has been given to calcium phosphate ceramics which are of two types:

a. Hydroxyapatite—nonresorbable (Figs 42.11A to C).
b. Tricalcium phosphate—partially-bioresorbable.



Figs 42.11A to C: Reconstructive periodontics. (A) Preoperative view. (B) Defect is exposed after flap reflection and debridement. (C) The defect is filled with hydroxyapatite crystal

Bioactive glass: Consists of sodium and calcium salts, phosphates and silicon dioxide with particle size ranging from 90 to 170 um (perioglas®) or 300 to 355 um (biogran®).

Coral-derived materials: Two types of materials are available, natural coral and coral-derived porous hydroxyapatite (both are proven to be biocompatible).

Combined techniques: A combination of both graft and nongraft associated methods have been proposed, e.g. combination of barrier techniques with bone grafts have been suggested by many authors.

KEYPOINTS

1. Osseous surgery can be of resective type and regenerative types.
2. Under resective surgery the following techniques exist:
   a. Vertical grooving.
   b. Radicular blending.
   c. Flattening of interproximal bone and,
   d. Gradualizing marginal bone.
3. Reconstructive surgical techniques are subdivided into—Nongraft-associated and Graft-associated new attachment techniques.
4. Nongraft-associated new attachment procedures are:
   a. Removal of junctional and pocket epithelium.
   b. Prevention of epithelial migration.
   c. Clot stabilization, wound protection and space creation.
   d. Preparation of the root surface.
5. Graft-associated techniques include grafts like autogenous bone grafts, allografts, xenografts and alloplasts which have been suggested and used successfully.

KNOW MORE ...

Specific Osseous Reshaping Procedures

- Intrabony and hemiseptal osseous lesions.
- Reverse osseous topography.

Osteoplasty is Indicated in the Treatment of:

a. Buccal and lingual bony ledges or tori.
b. Shallow intrabony defects.
c. Thick interproximal areas.
d. Incipient furcation involvements.

Current Research is Exploring Various Possibilities of Promoting Periodontal Regeneration

a. Biologic mediators.
b. Enamel matrix proteins.
c. Platelet-rich plasma (PRP) in regeneration.
d. Tissue engineering in regeneration.

Role of Biologic Mediators

These mediators are mostly physiologic molecules released by cells that can regulate events during wound healing. These molecules can function in either autocrine or paracrine mechanisms.

These growth factors, mainly secreted by macrophages, fibroblasts and platelets include:

- Platelet derived growth factor (PDGF).
- Insulin like growth factor (IGF).
- Fibroblast growth factor (FGF).
- Bone morphogenetic proteins (BMP).
- Transforming growth factor (TGF).

Their primary function is to stimulate periodontal wound healing thereby promoting migration and proliferation of fibroblasts → formation of bone.

b. *Enamel Matrix Proteins:* These proteins mainly amelogenin, are secreted by HRS (Hertwigs epithelial root sheath) during the development of tooth, which can induce formation of acellular cementum. Based on this, proteins are developed to promote periodontal regeneration, e.g. Emdogain (approved by US Food and Drug Administration (FDA).
c. *Platelet-rich Plasma (PRP):* Is an autologous concentrate of platelets. Platelets release various growth factors to initiate wound healing, e.g. PDGF, TGF, vascular endothelial growth factors.
d. *Tissue Engineering in Regeneration:* Langer in 1993, proposed tissue engineering as a possible technique for regeneration. Various approaches for tissue engineering could be protein based, cell based and gene based approaches.

These approaches aims at reconstructing the lost tissue by combining three elements, i.e. scaffold, signaling, cells (Tissue engineering triad).

Forum et al (2001) have proposed various factors that can influence the outcome of clinical results:

a. The dimension and morphology of the defect (deeper lesions result in greater bone fill than shallower defects).
b. The number of bony walls (three-walled results in better bone fill than, two-walled and one-walled defects).
c. Amount of root exposure.
d. The angle of the defect to the long axis of the tooth (the smaller the angle, the better is the chance of success).

REVIEW QUESTIONS

1. Define osseous surgery, describe the steps in resective osseous surgery.
2. Define regeneration, repair, new attachment and reattachment.
3. What is GTR?
4. What is root biomodification?
5. What is osteoconduction and osteoinduction?
6. Classify graft-associated new attachment procedures.

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41 Chapter

Periodontal Flap

- INDICATIONS
- DEFINITION
- CLASSIFICATION
- INCISIONS
- FLAP TECHNIQUES FOR POCKET THERAPY
  - Modified Widman Flap
  - Undisplaced Flap
  - Palatal Flap
  - Apically-displaced Flap
  - Distal Molar Surgery
- HEALING AFTER FLAP SURGERY

INTRODUCTION

Although in a strict sense all the instrumental therapy can be considered surgical (as by Webster's definition of surgery), those techniques mentioned here are considered as surgical procedures because it involves intentional severing of tissues.

Hence, the definition of surgery is, "the art, practice or work of treating diseases, injuries or deformities by manual operation or instrumental application".

Periodontal surgery (open) is defined as intentional severing or incising of gingival tissue with the purpose of controlling or eliminating periodontal disease. Therefore, scaling and root planing (blind or closed) are not included.

When should one consider for surgical pocket therapy?

1. For opening up the pocket area in order to remove all the irritants from the tooth surface.
2. For elimination or reduction of the periodontal pocket. The successful periodontal therapy is based on total elimination of plaque, calculus and diseased cementum from the tooth surface. Whenever there is a pocket present, the plaque control becomes difficult and also deeper the pocket the more difficult is the access because, the surface area to be scaled is increased, presence of root surface irregularities and also furcation involvement also creates various problems.

All the above-mentioned problems can be reduced by either resecting or displacing the soft tissue wall of the pocket. Gingivectomy and the flap surgery are the treatment procedures to attain this result (Fig. 41.1).

INDICATIONS/OBJECTIVES OF FLAP SURGERY

1. Gain access for root debridement.
2. Reduction or elimination of pocket depth, so that patient can maintain the root surfaces free of plaque.
3. Reshaping soft and hard tissues to attain a harmonious topography (physiologic architecture).
4. Regeneration of alveolar bone, periodontal ligament and cementum.

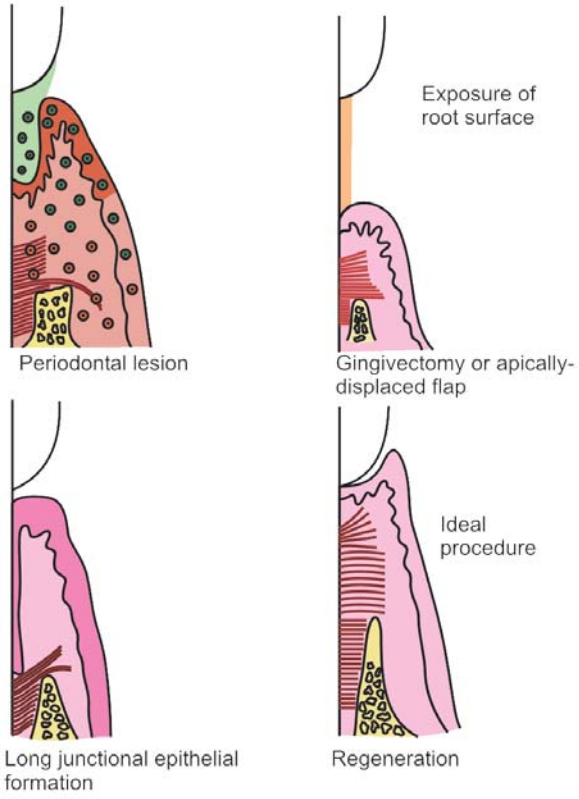


Fig. 41.1: Outcome of periodontal therapy

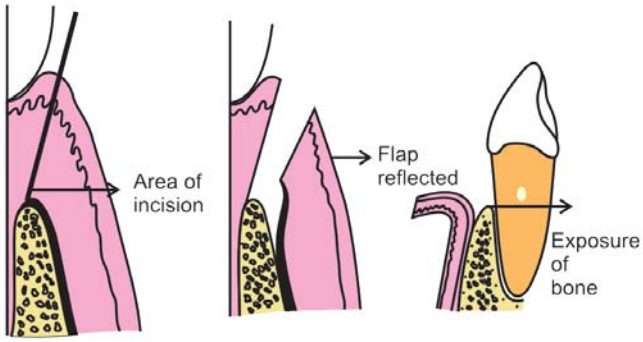


Fig. 41.2: Mucoperiosteal/full thickness flap

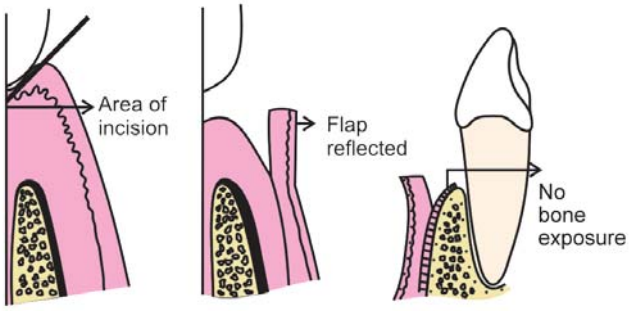


Fig. 41.3: Partial/split thickness flap

Today the flap has become the basic surgical procedure of periodontal therapy. It not only provides access to underlying tissues, but also permits the surgeon to perform a variety of regenerative procedures.

DEFINITION

A periodontal flap is a section of gingiva and/or mucosa surgically-elevated from the underlying tissues to provide visibility of and access to the bone and root surface.

CLASSIFICATION

According to the Thickness of the Flap/Bone Exposure after Flap Reflection

The flaps are classified as:

a. *Full thickness/mucoperiosteal flap*: All the soft tissues including the periosteum is elevated (Fig. 41.2).
b. *Partial thickness/mucosal flap/split thickness flap*: Reflection of only the epithelium and a layer of underlying connective tissue, the bone is covered by a layer of connective tissue including periosteum (Fig. 41.3).

Indications

I. Full thickness : If osseous surgery is contemplated.
II. Partial thickness : For displacing flaps in the presence of dehiscence and fenestrations.

According to the Placement of Flap after Surgery

a. *Nondisplaced flap*: The flap is returned and sutured back in its original position.
b. *Displaced flaps*: The flap is repositioned coronal, apical or lateral to its original position. However, palatal flaps cannot be displaced due to the absence of unattached gingiva.

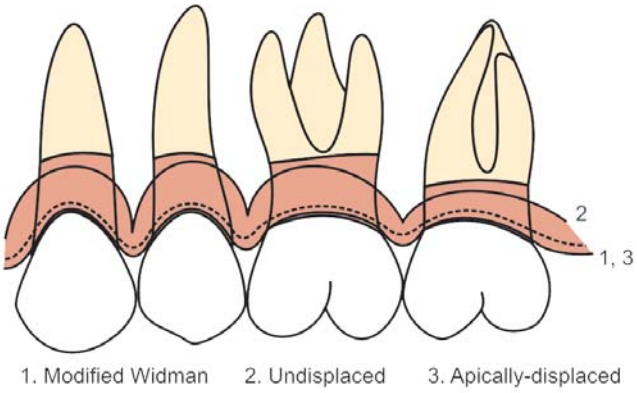


Fig. 41.4: Scallopings required for the different types of flaps

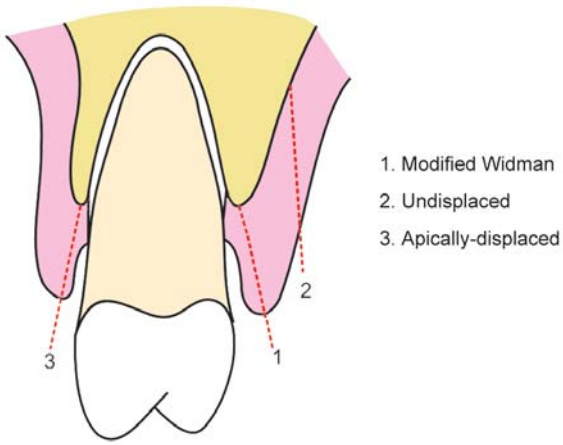
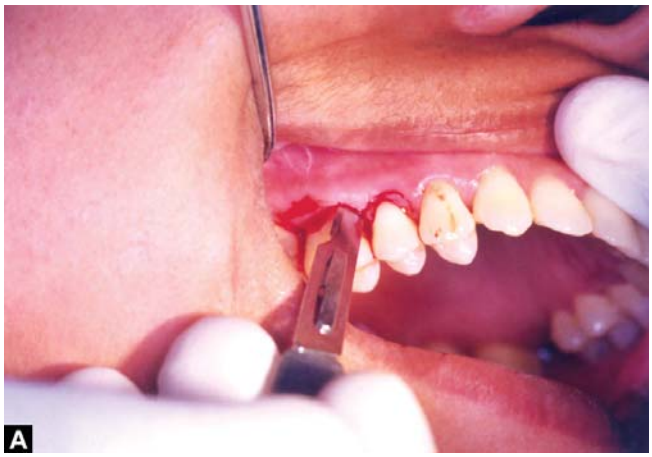
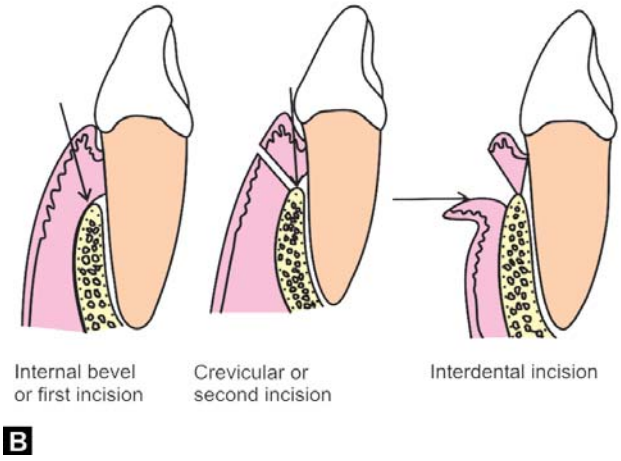


Fig. 41.5: Location of the internal bevel incisions for the different types of flaps





Figs 41.6A and B: Horizontal incisions

According to Design of the Flap/Management of the Papilla

The flaps can be:

a. *Conventional flaps*: Splitting the papilla into a facial half and lingual/palatal half. For example, modified Widman flap, undisplaced flap, apically-displaced flap (Fig. 41.4).

   Indications:

   i. When the interdental areas are too narrow to permit the preservation of flap.

   ii. When there is a need for displacing flaps.

b. *Papilla preservation flaps*: Entire papilla is incorporated into one of the flaps.

   Indications:

   i. Where esthetics is of concern.

   ii. Where bone regeneration techniques are attempted.

INCISIONS

For Conventional Flap

1. Horizontal Incision:
   - Internal bevel incision (Fig. 41.5)
   - Crevicular incision
   - Interdental incision
2. Vertical incision
   - Oblique releasing incision

For Papilla Preservation Flap

Crevicular incision with no incisions across the interdental papilla is given.

Horizontal Incisions (Figs 41.6A and B)

Internal bevel incision: It starts at a distance (1 to 2 mm) from the gingival margin and is aimed at the bone crest. It is a basic incision to most of the flap procedures.

It accomplishes three important objectives:

1. Removes pocket lining.
2. Conserves relatively uninvolved outer surface of the gingiva.
3. It produces a sharp, thin flap margin for adaptation to tooth—bone junction.

It is also known as first incision, reverse bevel incision. No. 15 and No. 11 surgical scalpels are most commonly used.

Crevicular Incision

It is also termed as the second incision, is made from the base of the pocket to the crest of the bone. The incision is carried around the entire tooth. No. 12B blade is used. This results in V-shaped wedge of tissue containing inflamed granulomatous tissue consisting of lateral wall of the pocket, junctional epithelium and connective tissue fibers between the base of the pocket and crest of the bone.

After this, a periosteal elevator is used to separate the flap from the bone. With this access, the surgeon is able to make the *third or interdental incision*, to separate the collar of the gingiva that is left around the tooth. The orban knife is usually utilized for this incision. A curette or a large scaler (U15/30) can be used to remove the gingiva around the tooth. If no vertical incisions are made, the flap is called an *envelope flap.*

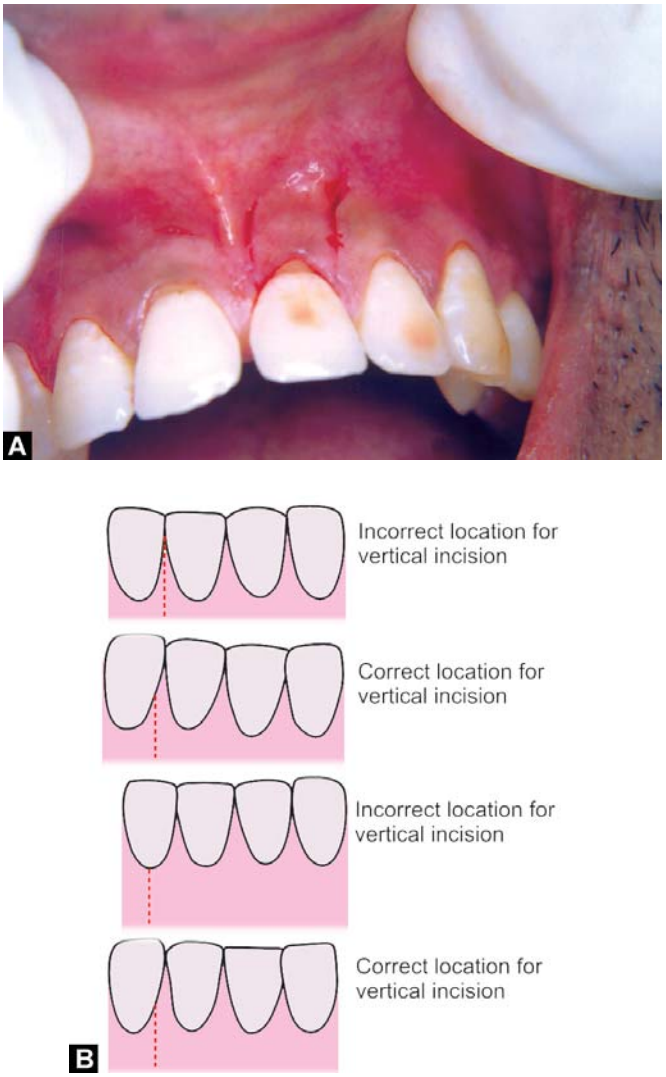
Vertical Incision (Figs 41.7A and B)

It can be done on one or both the ends of the horizontal incision. If the flap has to be displaced the incisions at both ends should be made. Another indication for vertical incision is in the presence of isolated deep pockets. Vertical incisions should be made extending beyond mucogingival junction for easy displacement of flap.

In general, vertical incisions are avoided:

a. In lingual or palatal areas.
b. At the center of the interdental papilla or over the radicular surface.

They are made along the line angles of the tooth, avoid short flaps with long, apically directed incisions as it compromises blood supply to the flap.



Figs 41.7A and B: (A) Vertical incisions without involving interdental papilla. (B) Location of vertical incision

Papilla Preservation Flap (Figs 41.8 to 41.10)

Step 1: Crevicular incision is made around each tooth. No incisions through the interdental papilla.

Step 2: Papilla is usually incorporated facially, hence a semilunar incision across the interdental papilla in the palatal or lingual surface is made, which is at least 5 mm from the crest of the papilla (the deepest curve).

Step 3: The papilla is dissected from the lingual or palatal aspect using Orban knife and elevated intact with the facial flap.

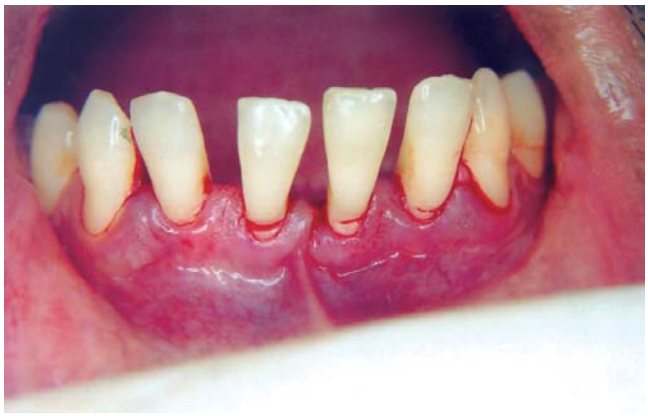


Fig. 41.8A: Facial view after sulcular incisions have been made

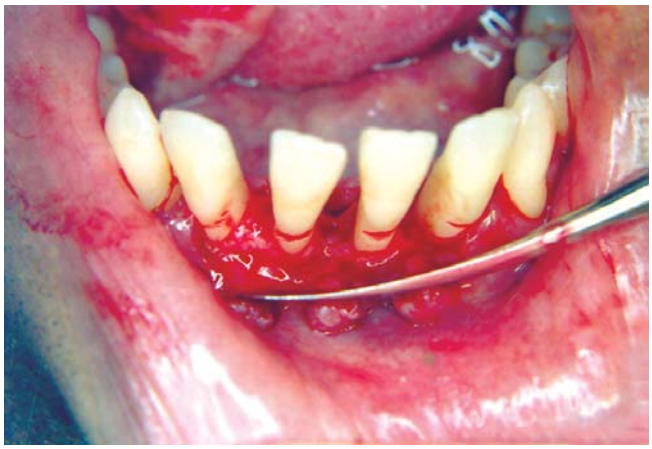


Fig. 41.8B: Flap reflection

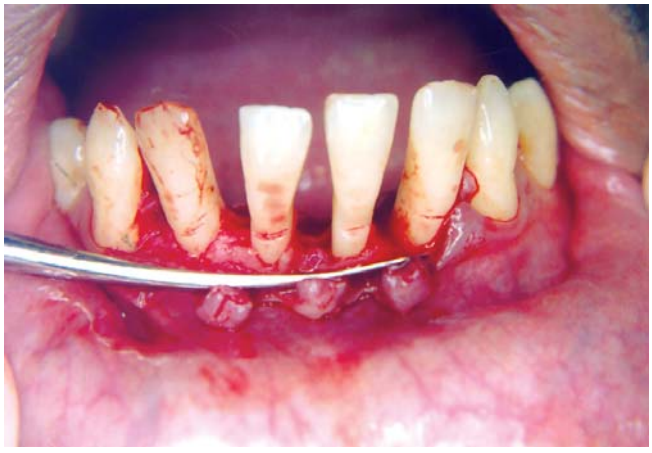


Fig. 41.8C: After curettage and root planing

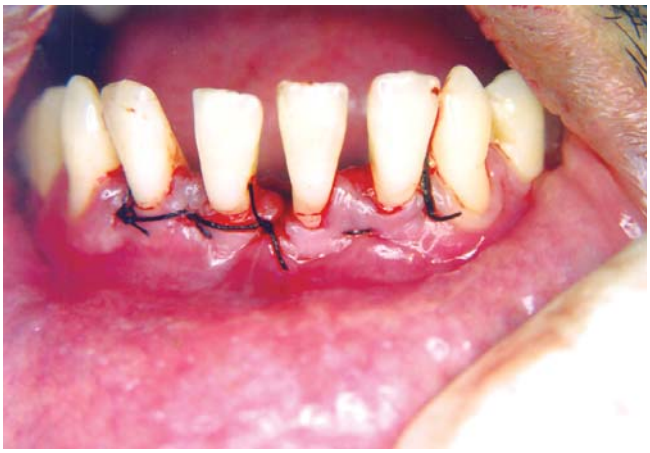


Fig. 41.8D: Flap is secured with sutures

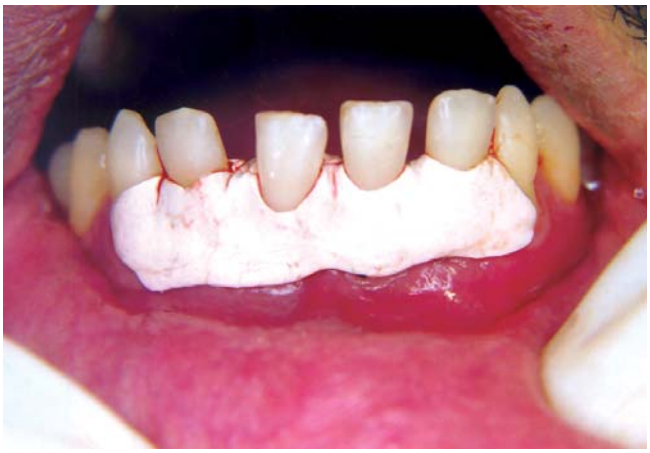


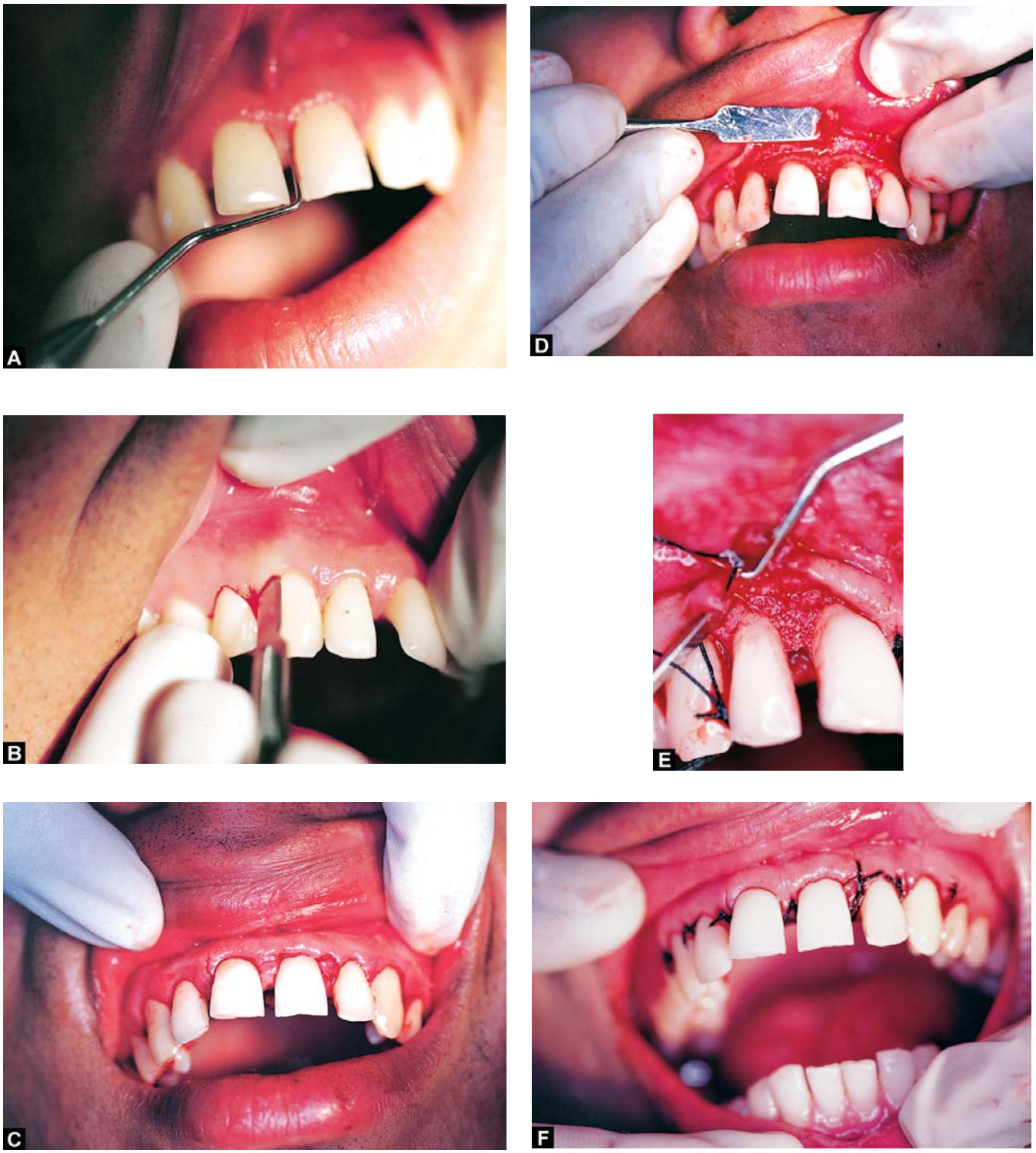
Fig. 41.8E: The surgical site is covered with periodontal dressing

Figs 41.8A to E: Papilla preservation flap

After the desired incisions are made, the flap is elevated either by blunt dissection for a full thickness flap or sharp dissection for partial thickness flap or combination of both for various surgical procedures.

FLAP TECHNIQUES FOR POCKET THERAPY

The periodontal flap is one of the most frequently employed procedure particularly for eliminating/reducing moderate to deep pockets in posterior areas. Considering the objectives it offers, there are three flap techniques available in current use (Table 41.1).



Figs 41.9A to F: Case-2: Papilla preservation flap. (A) Probing depth. (B) Crevicular incision placed. (C) Intrasulcular and semilunar incisions. (D) Reflection and degranulation. (E) Bone graft placement, (F) Suturing of palatal and buccal flaps

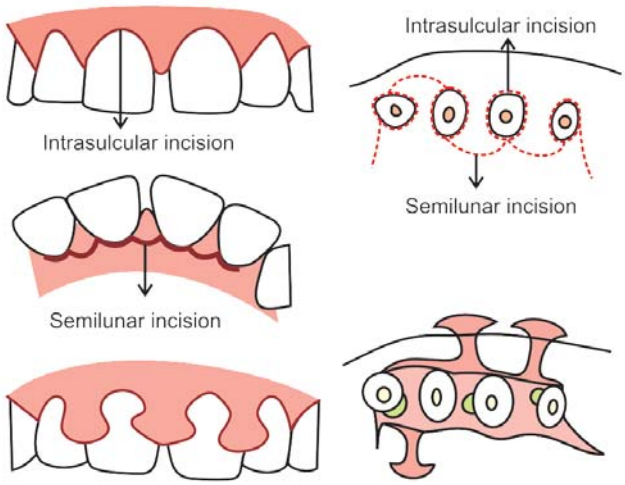


Fig. 41.10: Papilla preservation flap-incisions

Table 41.1: Differences between various flap techniques

Modified Widman flap	*Undisplaced flap*	*Apically-displaced flap*
I. Purpose		
1. To expose root surfaces for instrumentation.	1. Accessibility for instrumentation.	1. Improves accessibility.
2. For removal of pocket lining. (It is not indicated to eliminate/ reduce pocket depth, except for the reduction that occurs in healing by shrinkage).	2. To remove the pocket wall to reduce or eliminate the pocket. (An excisional procedure of the gingiva).	2. It also eliminates the pocket by transforming the previously unattached keratinized pocket wall into attached tissues. (offers dual function).
II. Variations in the design		
It does not intend to remove the pocket wall but eliminate pocket lining. Therefore, internal bevel incision starts close (no more than 1 to 2 mm apical) to the gingival margin.	Internal bevel incision is started at or near a point just coronal to the projection of the bottom of the pocket on the outer surface of the gingiva. (Only performed when sufficient attached gingiva is to be left behind). The incision should be scalloped to preserve as much as interdental papilla.	The internal bevel incision should be placed as close to the tooth as possible (0.5 to 1 mm) because the purpose of this technique is to preserve maximum amount of keratinized tissue, displace it apically and transform it into attached gingiva. The flap is positioned approximately at the tooth bone junction.

They are:

i. Modified Widman flap.
ii. Undisplaced flap.
iii. Apically-displaced flap.

Modified Widman Flap (Figs 41.11 to 41.13)

Presented in 1974 by Ramfjord and Nissle.

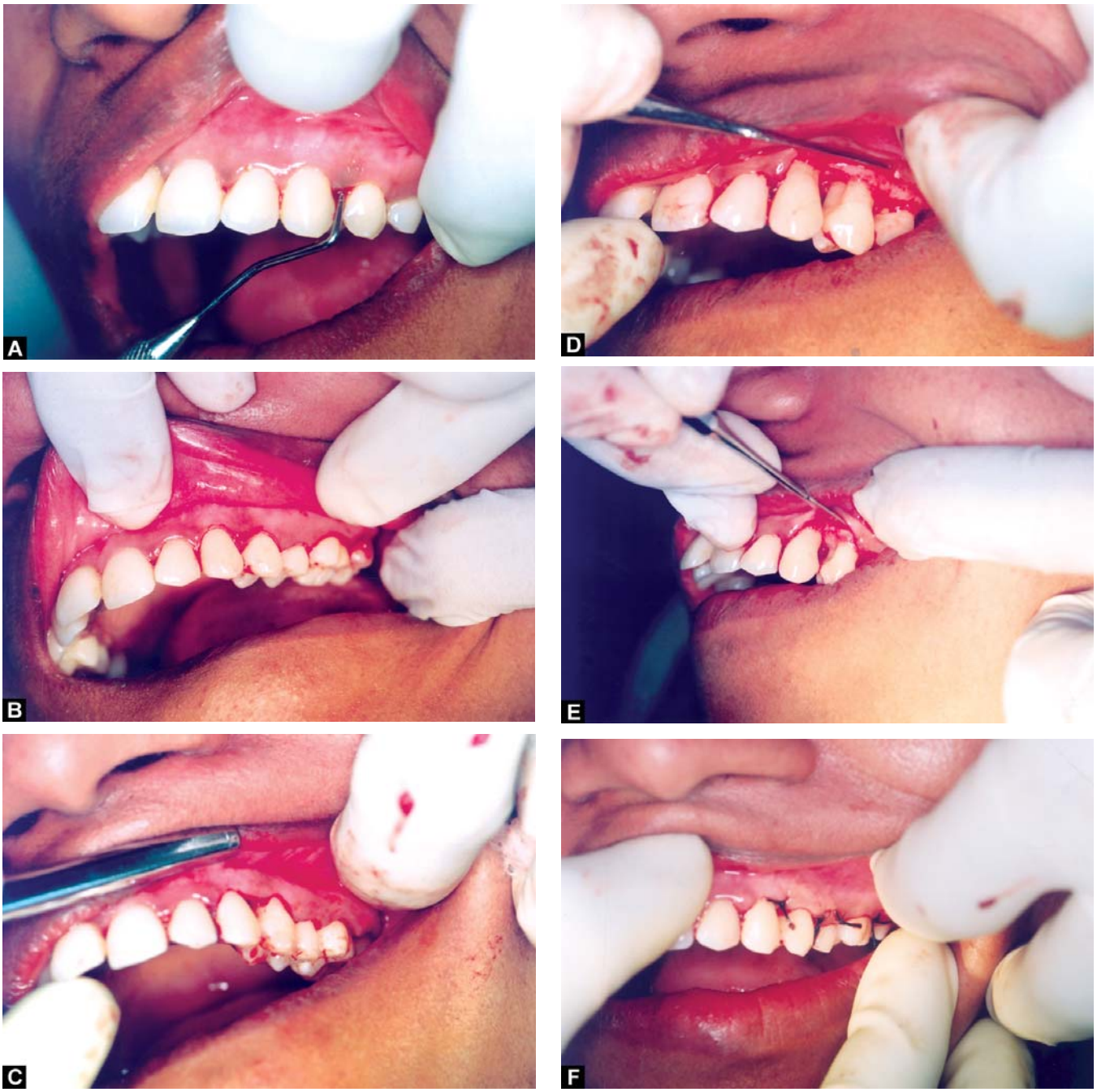
Step 1: It is an initial, internal bevel incision 0.5 to 1 mm away from the gingival margin, directed to the alveolar crest. Vertical releasing incisions are not required (different from Widman flap).

Step 2: Gingiva is reflected with a periosteal elevator

Step 3: A crevicular incision is made.

Step 4: After the flap is reflected, third incision is made in the interdental spaces with Orban's knife and the gingival collar is removed.

Step 5: Tissue tags and granulation tissue are removed with a curette. The root surfaces are examined and scaled.



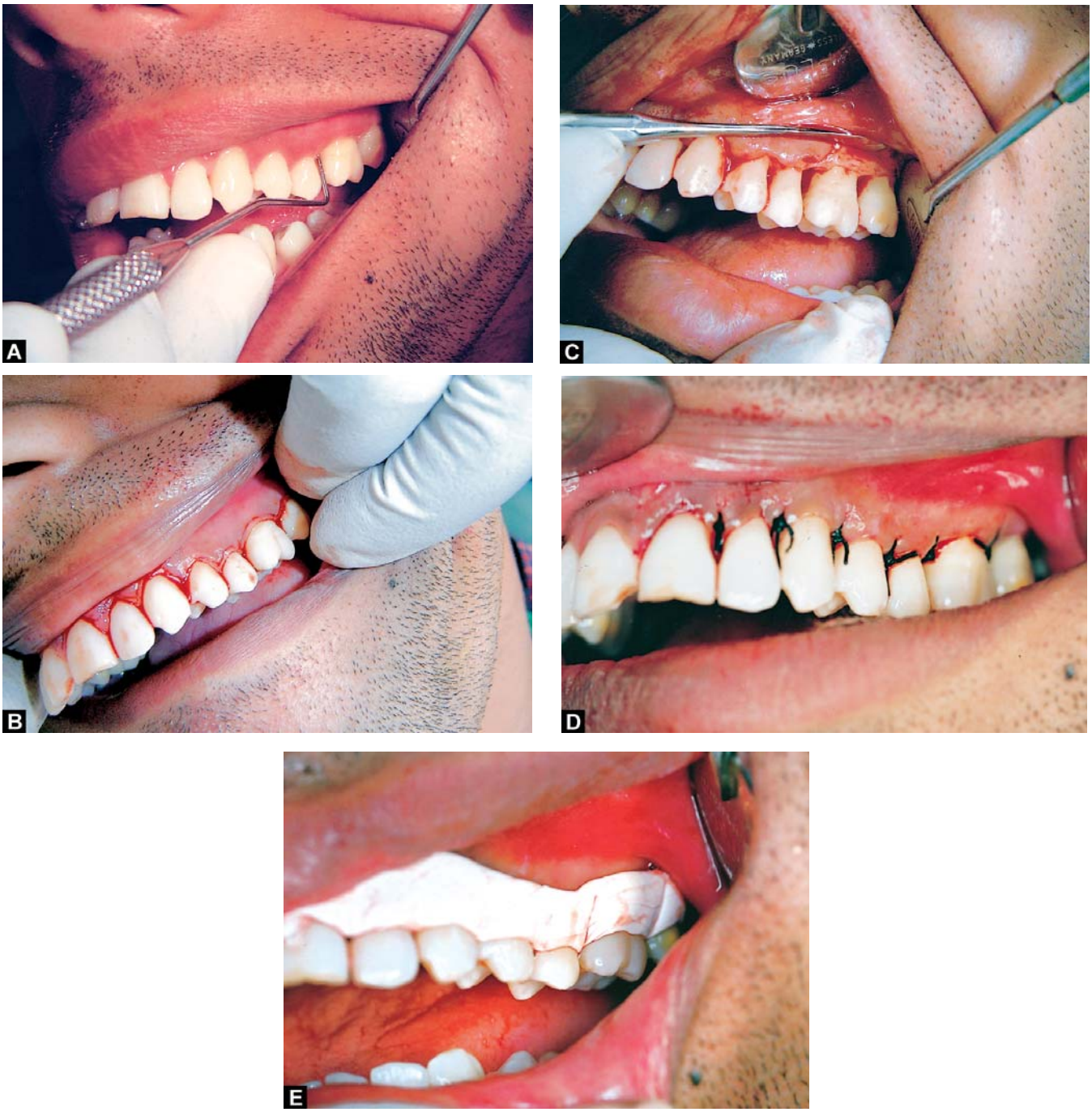
Figs 41.11A to F: Periodontal flap technique (Modified Widman flap). (A) Persistent pockets 2-3 weeks after initial therapy, (B) Facial incisions (internal bevel, crevicular), (C) Interdental incisions performed, (D) Facial flaps are reflected, (E) Curettage and root planing performed, (F) Interrupted interdental sutures are placed

Step 6: Bone architecture is not corrected, good approximation of flaps is necessary, hence sometimes flaps may have to be thinned.

Step 7: Interrupted direct sutures are placed.

Undisplaced Flap (Figs 41.14 to 41.16)

In this procedure the entire soft tissue pocket wall is removed with the initial incision. Thus, it may be considered as internal bevel gingivectomy. To perform this enough attached gingiva should remain after removal of the pocket wall.



Figs 41.12A to E: Case-2: Modified Widman flap procedure. (A) Preoperative view with probing. (B) Internal bevel and crevicular incisions are placed. (C) Flap reflection and degranulation. (D) Suturing with direct loop sutures. (E) Pack placed

Step 1: The pockets are measured with the periodontal probe and a bleeding point is produced on the outer surface of the gingiva to mark the base of the pocket. In this procedure, the final placement of the flap is determined by first incision.

Step 2: The initial, internal bevel incision is made following the scalloping bleeding points made on the gingiva. This incision is usually carried to a point apical to the alveolar crest depending on the thickness of the tissue. The thicker the tissue, the more apical will

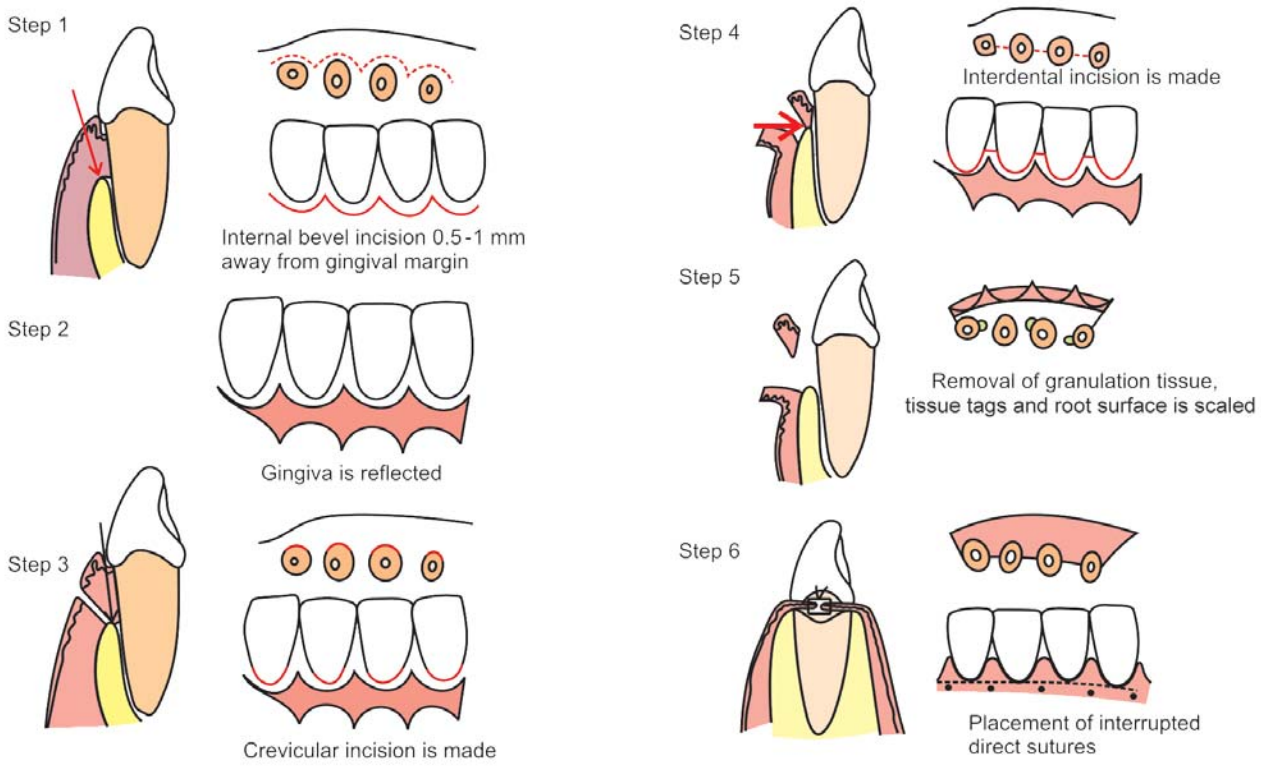


Fig. 41.13: Modified Widman flap—various steps

be the end point. The flap should be thinned with the initial incision only.

Step 3: The second or crevicular incision is made from the bottom of the pocket to the bone.

Step 4: The flap is then reflected with a periosteal elevator (blunt dissection).

Step 5: Interdental incision is made with an Orban's interdental knife.

Step 6: Triangular wedge of tissue is removed with a curette.

Step 7: The area is debrided, removing tissue tags and granulation tissue with sharp curettes. The roots are scaled.

Step 8: The flap is then placed back to end at the root bone junction.

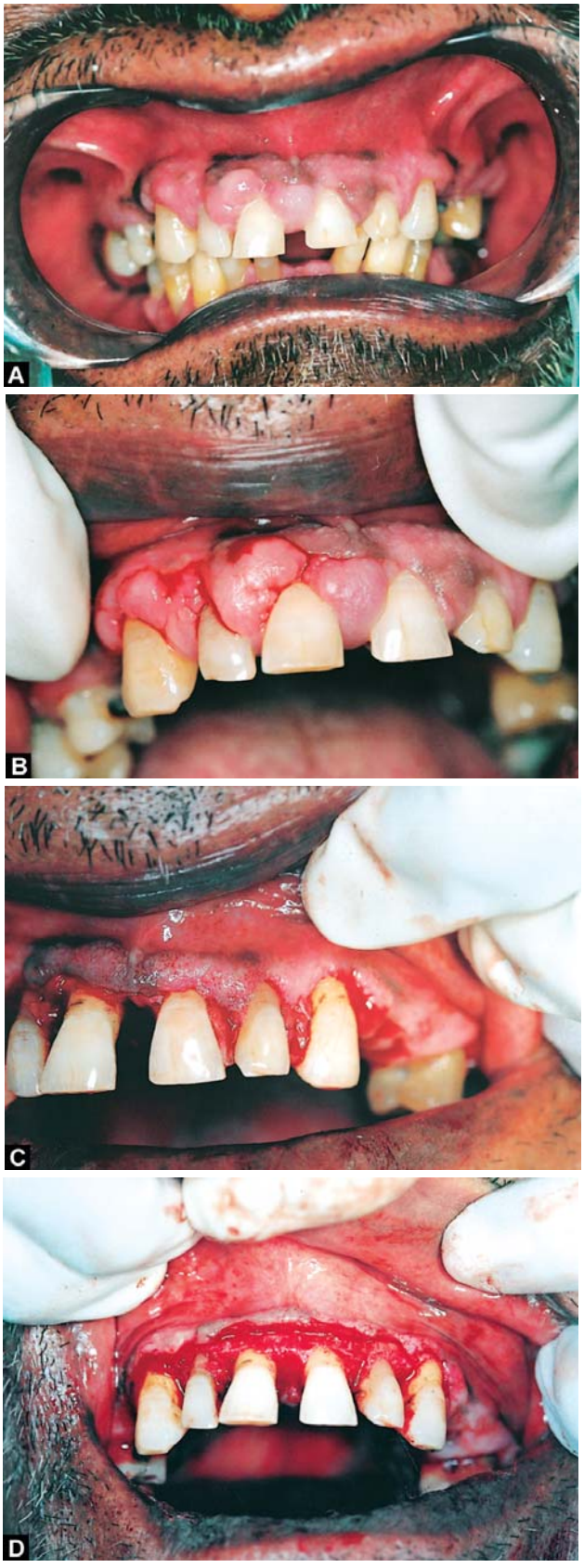
Step 9: The flaps are sutured together with continuous sling suture or interrupted sutures.

Palatal Flap (Figs 41.17 and 41.18)

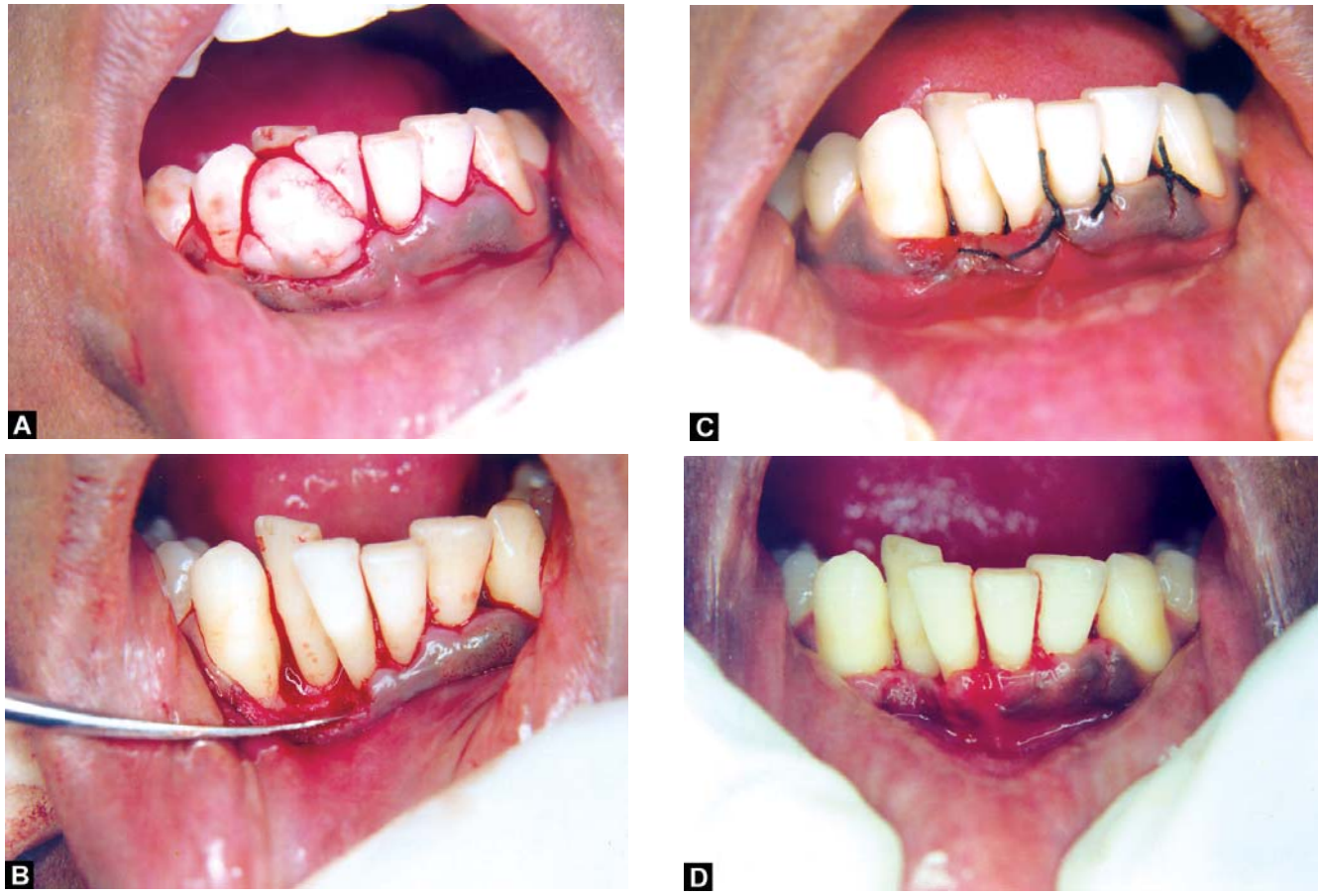
The surgical approach is different here because of the nature of the palatal tissue which is attached, keratinized tissue and has no elastic properties associated with other gingival tissues, hence no displacement and no partial thickness flaps.

Variations in the Techniques

1. Usual internal bevel incision, followed by crevicular and interdental incisions, but if the tissue is thick horizontal gingivectomy incision is made, followed by an internal bevel incision. Thinning of the flap should be done prior to reflection of the flap.
2. The apical portion of the scalloping incision should be narrower than the line angle area, because the palatal root tapers apically, rounded scallop will result in improper adaptation of the flap around root.



Figs 41.14A to G: Undisplaced flap. (A) Preoperative view. (B) Internal bevel incision. (C and D) Reflection and degranulation. (E) Suturing buccal and palatal flaps. (F) Palatal view after suturing. (G) Facial view after healing



Figs 41.15A to D: Case-2: Undisplaced flap—step-by-step procedure. Clinical presentation. (A) Incisions placed at the base of the pocket. (B) Flap reflection. (C) After thorough debridement, sutures are placed. (D) Facial view after healing

Basically, one should make sure the flap fits around the tooth snugly without exposing the bone, to achieve this proper placement of the incision is very important.

Apically-displaced Flap

Used for both pocket eradication and/widening the zone of attached gingiva.

Step 1 : Internal bevel incision is made, 1 mm from the crest of the gingiva and directed towards the crest of the bone.

Step 2 : Crevicular incisions are made followed by initial elevation of flap and then interdental incision is performed, the wedge of tissue containing the pocket wall is removed.

Step 3: Vertical releasing incisions are made extending beyond the mucogingival junction and flap is elevated with a periosteal elevator (either split thickness or full thickness).

Step 4: Remove all the granulation tissue, root planing is done and flap is positioned apically at the tooth bone junction.

Step 5: Flaps are sutured together.

Distal Molar Surgery

- For maxillary molars
- For mandibular molars.

Technique for maxillary molars (Fig. 41.19): Two parallel incisions at the distal surface of terminal tooth are made. The deeper the pocket, the greater will be the distance between the two parallel incisions. Followed by this a transversal incision is made so that a long rectangular piece of tissue is removed. This can be confirmed with the regular

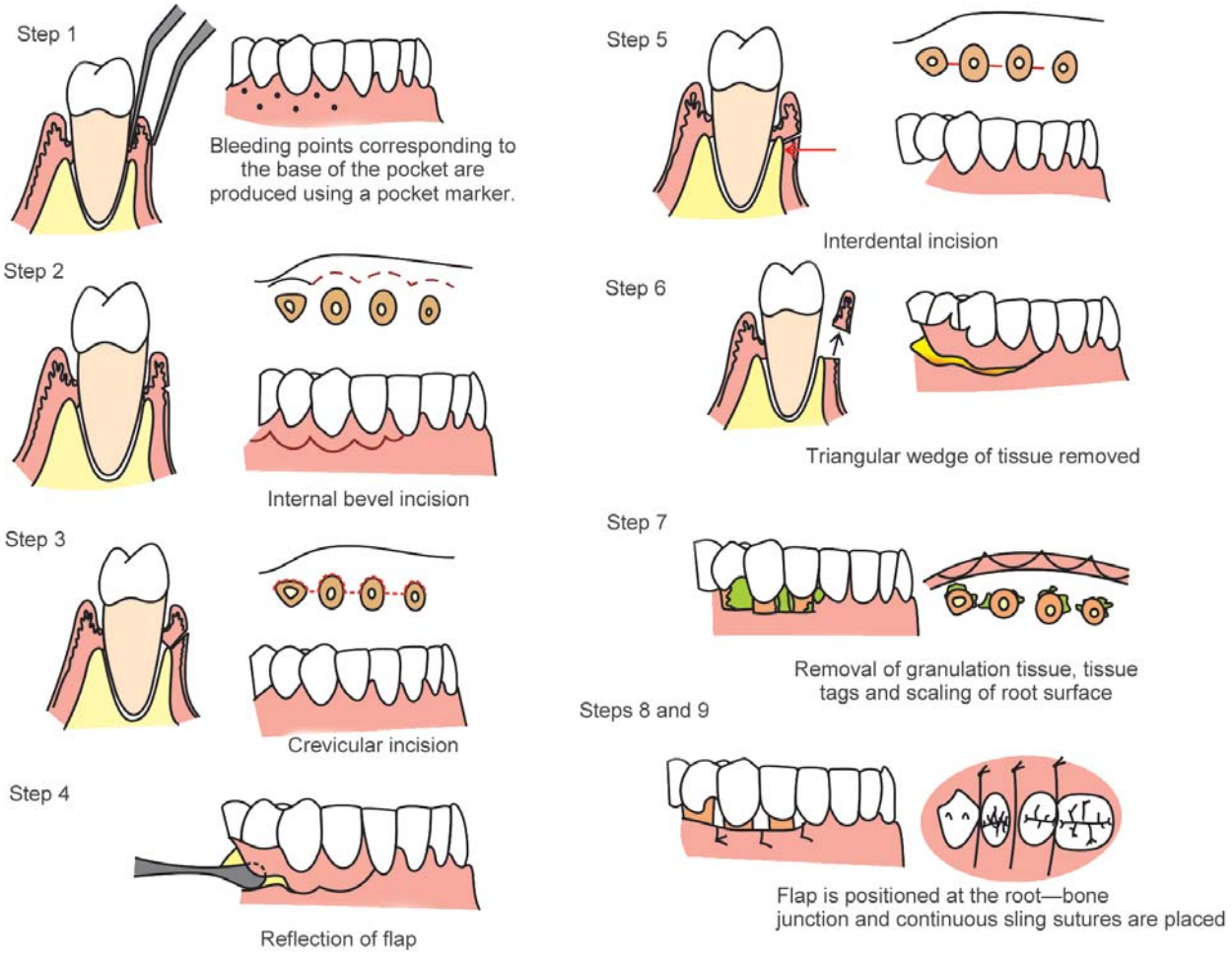


Fig. 41.16: Undisplaced flap technique—various steps

flap in the quadrant being treated. These incisions can be placed using No. 12 blade, after flap reflection and curetting the bone surface, the flaps are sutured together.

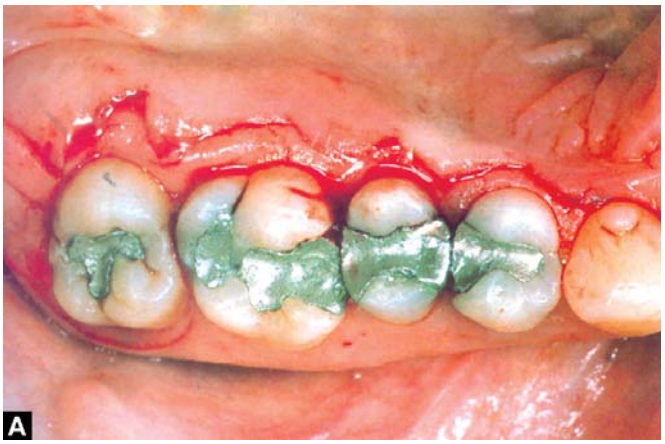
Technique for mandibular molars (Figs 41.20A and B): Two parallel incisions at the retromolar pad area are made. The incisions should follow the areas of greatest attached gingiva and underlying bone. After the reflection of the flap and removal of tissue, osseous surgery may be performed (if necessary) and flaps are sutured so as to approximate the flap margins closely to each other.

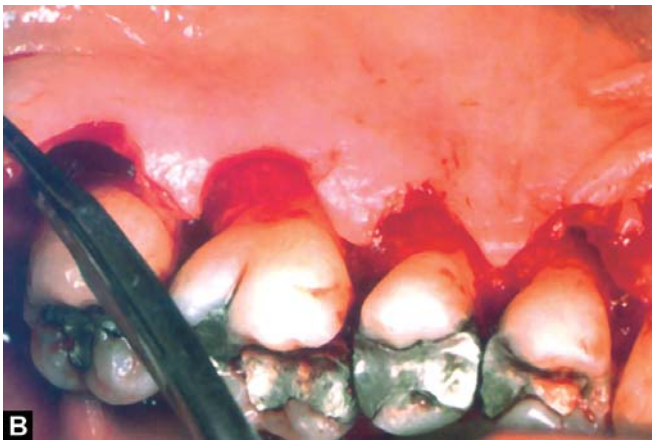
HEALING AFTER FLAP SURGERY

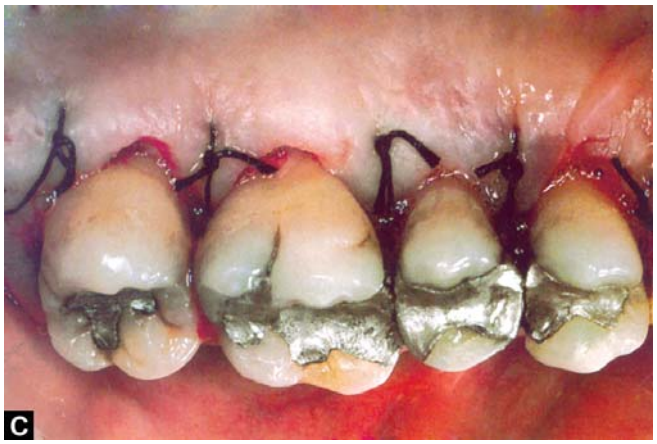
Immediately after suturing (0 to 24 hours), connection between the flap and the tooth or bone surface is established by the blood clot, which consists of a fibrin reticulum with many polymorphonuclear leukocytes, erythrocytes, debris from injured cells and capillaries at the edge of the wound. There are also bacteria and an exudate or transudate as a result of tissue injury.

One to three days after flap surgery—space between the flap and the tooth or the bone is thinner and epithelial cells migrate over the border of the flap, when the flap is closely-adapted to the alveolar process there is only a minimal inflammatory response.

One week after flap surgery—an epithelial attachment to the root has been established by means of hemidesmosomes and a basal lamina. The blood clot is replaced by granulation tissue derived from gingival connective tissue, bone marrow and the periodontal ligament.







Figs 41.17A to C: Palatal flap

Two weeks after surgery—collagen fibers begin to appear parallel to the tooth surface. Union of the flap and the tooth is still weak (due to immature collagen fibers) but clinically it appears almost normal.

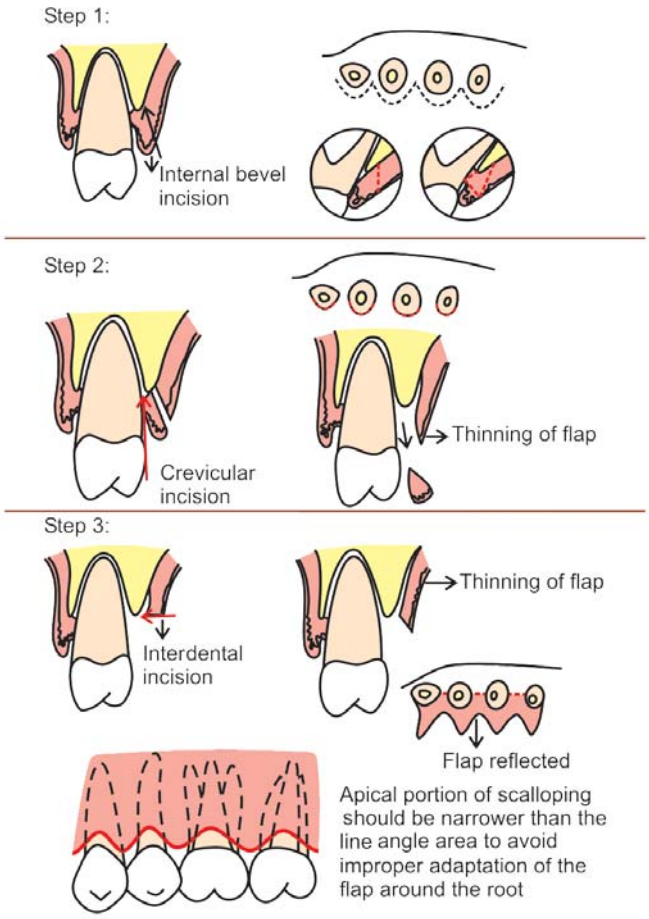


Fig. 41.18: Palatal flap—step-by-step procedure

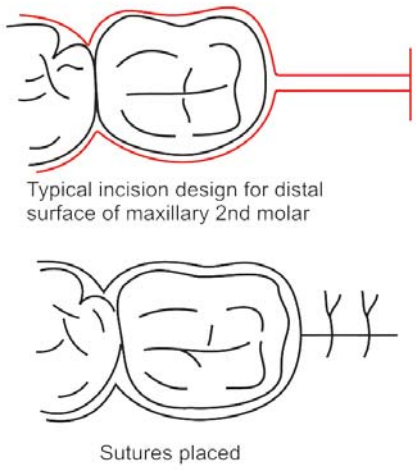
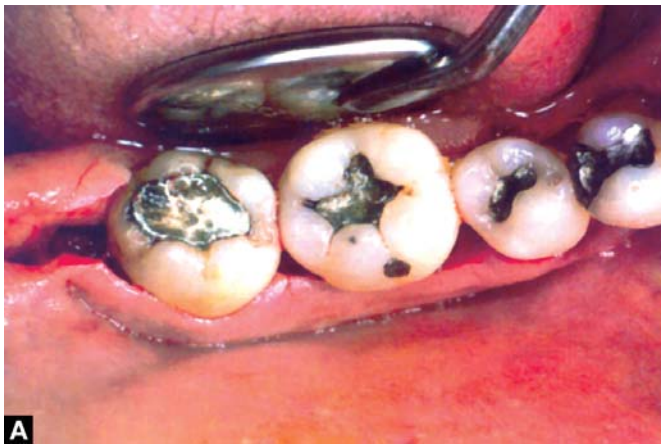
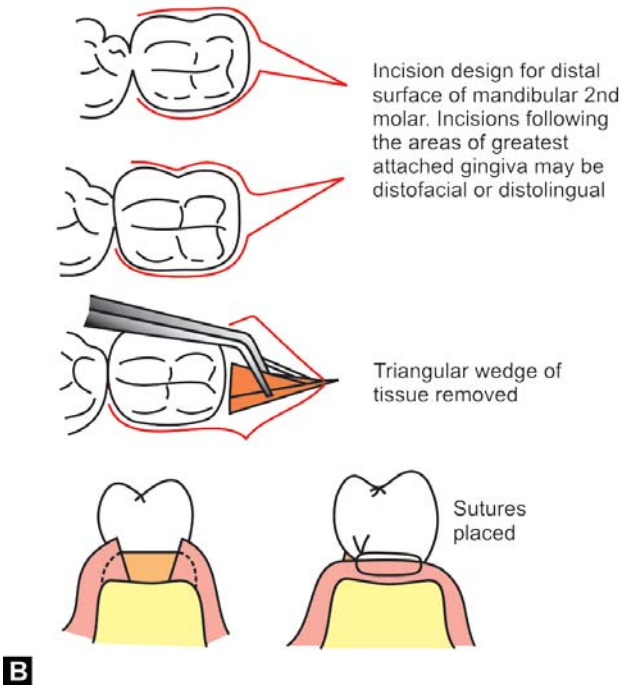


Fig. 41.19: Distal molar surgery for maxillary molars

One month after surgery—a fully-epithelialized gingival crevice with a well-defined epithelial attachment is present. Supracrestal fibers begin to adapt a functional arrangement.





Figs 41.20A and B: Distal molar surgery for mandibular molars

HEALING AFTER FULL THICKNESS FLAP

Same as above but superficial bone necrosis takes place in one to three days followed by osteoclastic resorption at 4 to 6 days and declines thereafter. This results in bone loss of about 1 mm and greater, if the bone is thin.

KEYPOINTS

1. Periodontal flap is a section of gingiva and or mucosa surgically-elevated from the underlying tissues to provide visibility of and access to the bone and root surface.
2. Periodontal flaps are classified according to the thickness as, full thickness and partial thickness flap; and according to the placement of flap as displaced and undisplaced flap; according to the design of the flap as conventional flap and papilla preservation flap.
3. In conventional flap, horizontal incisions and vertical incisions are given.
4. There are three types of horizontal incisions, internal bevel, crevicular and interdental incision.
5. Currently there are three types of periodontal flaps available, Modified Widman flap, Undisplaced flap and Apically-displaced flap.
6. Main objectives of periodontal flap surgery are gain access to root surface, reduction or elimination of pocket depth and attempt regeneration of periodontal ligament, alveolar bone and cementum.

REVIEW QUESTIONS

1. Define and classify the periodontal flaps. Describe the step-by-step procedure of modified Widman flap.
2. What are the indications of flap surgery?
3. Describe healing following flap surgery.

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43 Chapter

Mucogingival Surgery

- DEFINITION
- MUCOGINGIVAL PROBLEMS
- OBJECTIVES, INDICATIONS AND CONTRAINDICATIONS
- TECHNIQUES TO INCREASE WIDTH OF ATTACHED GINGIVA
  - Gingival Extension Operation
  - Apical Displacement
  - Other Techniques
- TECHNIQUES FOR ROOT COVERAGE
  - Indications
  - Modifications
- OPERATIONS FOR REMOVAL OF FRENA

DEFINITION

Mucogingival surgery was first introduced in the 1950s. According to the glossary of periodontal terms mucogingival surgery refers to *"Periodontal surgical procedures designed to correct defects in the morphology, position and/or amount of gingiva surrounding the teeth*. Recently, it has been suggested that *"periodontal plastic surgery"* may be more appropriate and would be defined as *"Surgical procedures performed to correct or eliminate anatomic, developmental or traumatic deformities of the gingiva or alveolar mucosa"*.

Mucogingival surgery consists of "plastic surgical procedures for the correction of gingiva, mucous membrane relationships that complicate periodontal diseases and may interfere with the success of periodontal treatment" (According to Glickman).

MUCOGINGIVAL PROBLEMS

These are:

i. Pockets extending up to or beyond mucogingival junction
ii. Recession causing denudation of root surfaces
iii. High frenum and muscle attachments
iv. Inadequate width of attached gingiva.

OBJECTIVES, INDICATIONS AND CONTRAINDICATIONS OF MUCOGINGIVAL SURGERY

Objectives

1. Widening the zone of attached gingiva
2. Coverage of denuded roots

3. Removal of aberrant frenum.
4. Creation of some vestibular depth when it is lacking.
5. As an adjunct to routine pocket elimination procedures.

Indications

1. Augmentation of the edentulous ridge.
2. Prevention of ridge collapse associated with tooth extraction.
3. Crown-lengthening.
4. Loss of interdental papilla which presents as esthetic/or phonetic defect.

Contraindications

They are the same as in any other periodontal surgery.

The treatment procedures that may fall within the definition of periodontal plastic surgery are:

- Gingival augmentation procedures for correction of mucosal defects (around implants)
- Root coverage procedures
- Gingival preservation at ectopic tooth eruption
- Removal of aberrant frenulum
- Ridge augmentation
- Crown-lengthening procedures.

The most commonly performed mucogingival surgical procedures are discussed in this chapter.

TECHNIQUES TO INCREASE THE WIDTH OF ATTACHED GINGIVA

Gingival Extension Operation

The width of attached gingiva varies in different individuals and on different teeth in the same individual. Attached gingiva is different from the keratinized gingiva. The width of the attached gingiva is determined by subtracting the depth of the sulcus or pocket from the total distance between the crest of the margin to the mucogingival junction. Originally, it was thought that minimal width of attached gingiva is required for optimal gingival health to be maintained. However, several studies have challenged this and concluded that even in the presence of minimal amounts of attached gingiva the tissues can be maintained in a normal state.

Various gingival extension procedures are:
Gingival augmentation apical to the area of recession.

Free Soft Tissue Autograft

Advantages

- High degree of predictability—only with increasing width of keratinized gingiva
- Simplicity
- Ability to treat multiple teeth at the same time
- This procedure can be performed where there is inadequate keratinized gingiva adjacent to the involved area.

Disadvantages

- Two operative sites
- Compromised blood supply
- Greater discomfort
- Lack of predictability in attempting root coverage.

Free soft tissue autografts are indicated in the presence of:

- An inadequate zone of attached gingiva
- Abnormal muscle attachment
- Shallow vestibular depth
- Gingival recession
- Deep pockets to prevent rapid initial down growth of epithelium.

Procedure

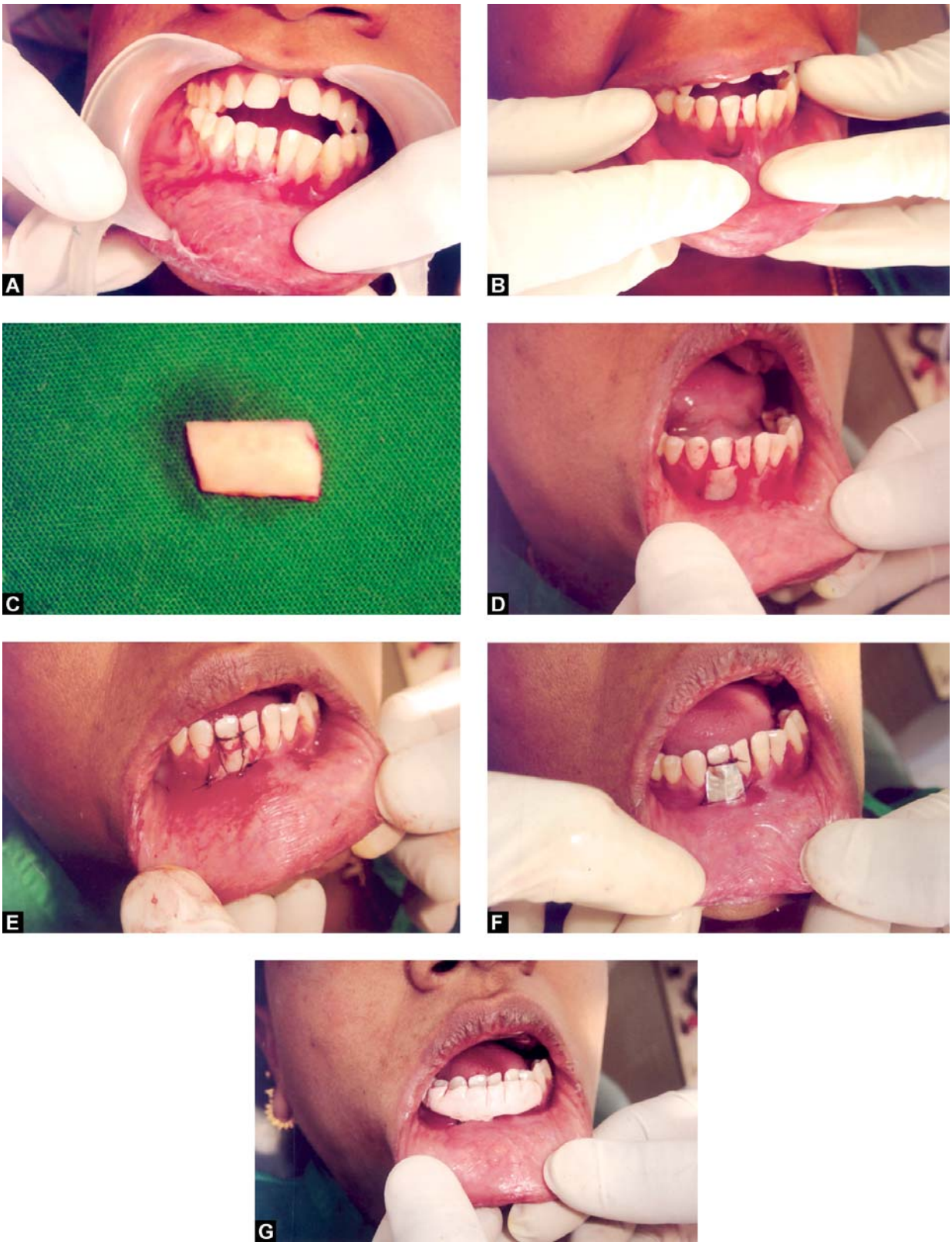
- Classic technique
- Variant techniques.

The Classic Technique (Figs 43.1A to H and 43.2A to H)

Step 1: Eliminate the pockets—If pockets are present resect them with a gingivectomy incision and scale and plane the root surfaces. If there are no pockets present in the area, the gingival margin is left intact.

Step 2: Preparation of the recipient site—It can be done by two techniques.

1. *First technique:* By incising at the existing mucogingival junction with a #15 BP blade to a little more than the desired depth and then blending the incision on both



Figs 43.1A to G: Free soft tissue autograft. (A) Lack of attached gingiva in relation to lower right lateral incisor, (B) Classic technique—Surgical bed preparation, (C) Free gingival graft procured from the palate, (D) Donor tissue placed on the surgical bed, (E) The graft is sutured with silk sutures, (F) Tinfoil covering the graft site, (G) Periodontal dressing is placed

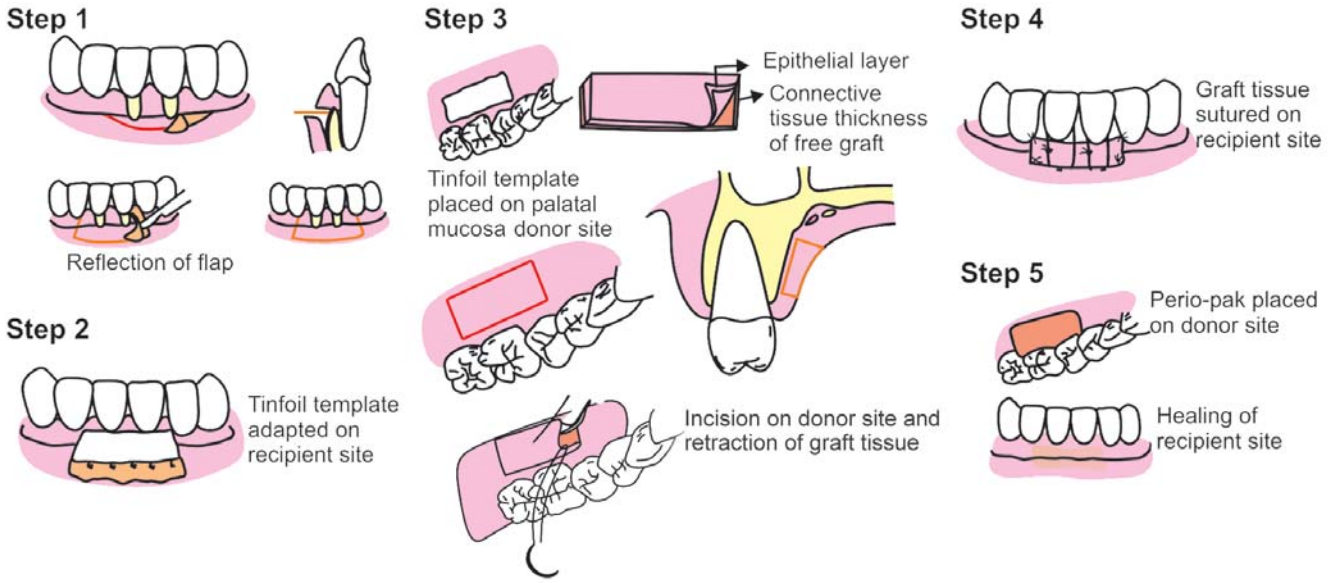


Fig. 43.1H: Various steps in classic technique

ends with the existing mucogingival line. Periosteum should be left covering the bone.

2. *Another technique:* Consists of outlining the recipient site with two vertical incisions from the cut gingival margin into the alveolar mucosa. Extend the incisions to approximately twice the desired width of the attached gingiva, allowing for 50 percent contraction of the graft when healing is complete. A # 15 blade is inserted along the cut gingival margin and is used to separate a flap consisting of epithelium and connective tissue without disturbing the periosteum. Suture the flap where the apical portion of the free gingival graft will be located.

Grafts can also be placed directly on the bone tissue. The advantages of this variant includes, less postoperative mobility of the graft, less swelling, better hemostasis—1½ to 2 times less shrinkage, however a healing lag is observed for the first two weeks. Next make a tinfoil template of the recipient site, to be used as a pattern for the graft.

Step 3: Obtaining the graft from the donor site.

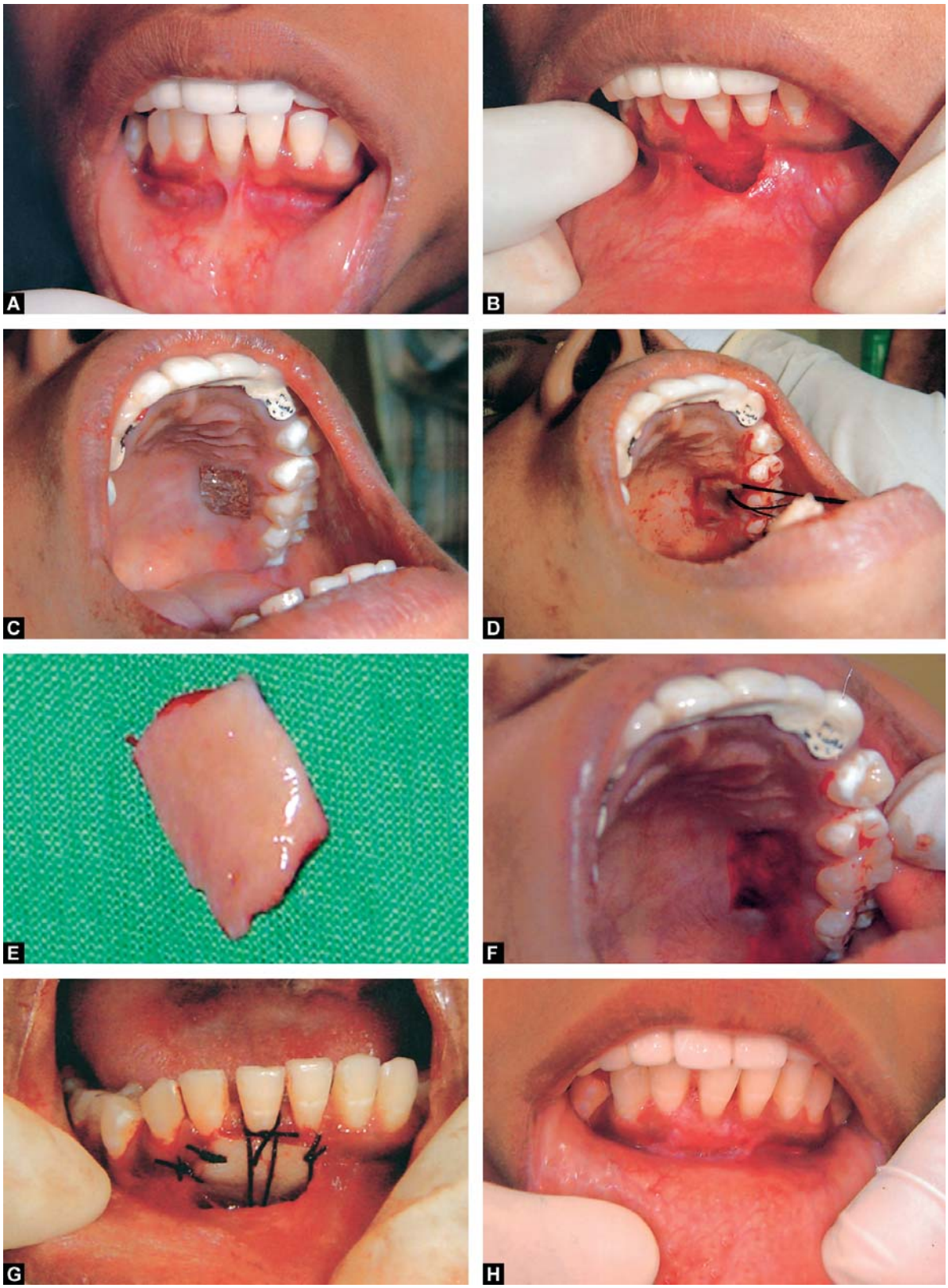
For classic technique: A partial thickness graft is used, the sites from which it can be obtained are in order of preference:

- Attached gingiva
- Masticatory mucosa from an edentulous ridge
- Palatal mucosa.

The graft should consist of epithelium and a thin layer of underlying connective tissue. Place the template over the donor site and make a shallow incision around it with a # 15 blade. Insert the blade to the desired thickness at one edge of the graft and elevate it by holding with tissue forceps or placing sutures at the margins of the graft.

Proper thickness is important for the survival of the graft. It should be thin enough to allow the ready diffusion of nutritive fluid from the recipient site. If it is too thin the graft may shrivel and expose the recipient site. If the graft is too thick, its peripheral layer is jeopardized because of the excessive tissue that separates it from new circulation and nutrients. Thick grafts may also leave a deeper wound at the recipient site and may cause damage to the palatal arteries. The ideal thickness of a graft is between 1 to 1.5 mm. After the graft is separated, remove loose tissue tags from the under surface.

Step 4: Transfer and immobilization of the graft—Remove the excess clot from the recipient site because thick clot interferes with vascularization of the graft. Position the graft and adapt it firmly to the recipient site. Dead space will retard the vascularization and jeopardize the graft. Suture the graft at the lateral borders and to the periosteum to secure it in position. Be sure that the graft is immobilized because movement interferes with healing.



Figs 43.2A to H: Case-2: Free soft tissue autograft. (A) Gingival recession with lack of attached gingiva. (B) Surgical bed preparation. (C) Tinfoil template placed on palatal donor site. (D) Harvesting of graft tissue. (E) Harvested donor tissue. (F) Donor site after harvesting. (G) Graft tissue sutured with graft stretching and vertical sutures. (H) Facial view after healing

Step 5: Protection of the donor site—Cover the donor site with a periodontal pack for one week and repeat if necessary. A modified Hawley retainer is useful to cover the pack on the palate and over the edentulous ridges.

Variant techniques

Four variants to the classic technique are described:

a. Accordion technique
b. Strip technique
c. The connective tissue technique
d. Combination of strip and connective tissue techniques.

Accordion technique *(Figs 43.3A to F)*

Accordian technique has been described by Rateitschak and colleagues. Expansion can be achieved by giving alternate incisions on the opposite sides of the graft.

Strip technique *(Fig. 43.4)*

The strip technique by Han and associates consists of 2 or 3 strips of tissue about 1 mm wide and long enough to cover the entire length of the recipient site. These strips are placed at the center and base of the recipient site and sutured from the oral mucosa. The area is then covered with tinfoil and a surgical pack.

Connective tissue technique

The connective tissue technique was originally described by Edel. The advantages of this technique include:

a. Donor site—Healing by primary intention is achieved because the donor material can be obtained from connective tissue that is present beneath the palatal flap.
b. Color matching is better.

Combination techniques

It can be performed as follows:

Remove a strip of tissue about 3 to 4 mm thick from the palate, place it between two wet tongue depressors, and slice it longitudinally with a sharp BP blade. Use a superficial portion that contains epithelium and connective tissue, and the deeper portion that only consists of connective tissue.

Healing of the graft *(Figs 43.5A and B)*

The full thickness graft consists of fat, glandular tissue as well as the epithelium. It is not easily accepted and not required in periodontal surgery.

The process of accepting a graft:

I. *Plasmatic circulation stage (2-3 Days):* Direct joining of capillaries between the graft and the recipient bed is seen and capillaries of the recipient bed invade the graft.
II. *Capillary circulation stage (4-5 Days):* Capillaries start functional circulation again. The epithelium at this stage is almost desquamated and sparse.
III. *Organization stage (10-14 Days):* Organization is completed by growth of fibroblasts in the graft and in the recipient bed. The network of capillaries become dense and regeneration of epithelium becomes active to complete the epithelialization (wound healing is by secondary intention).

Apically-Displaced Flap (Fig. 43.6)

This technique can be used for the combined purposes of eliminating pockets and widening the zone of attached gingiva. Depending on the purpose it can be a full thickness (or) split thickness flap. The partial (split) thickness flap is generally used to avoid exposure of bone and the accompanying risks of bone resorption and aggravation of bone dehiscences and fenestrations. The full thickness flap is indicated when access to bone is desired for recontouring purposes.

Procedure

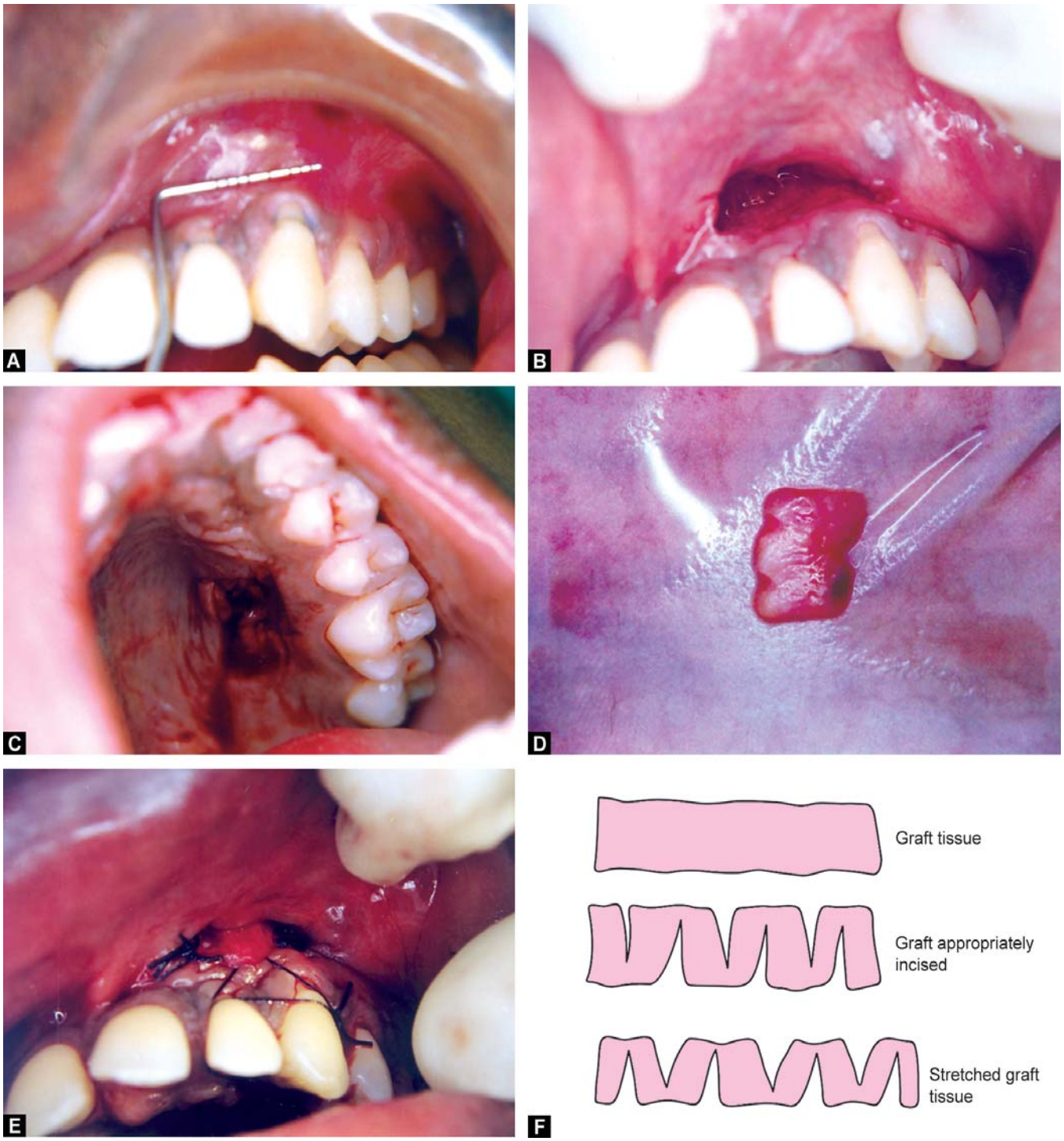
Step I : Internal bevel incision 0.5-1 mm from the crest of the marginal gingiva is given.

Step II : Crevicular incision and interdental incisions are placed.

Step III : Vertical incisions extending beyond the mucogingival junction are made so that the flap can be displaced easily. Reflect the flap depending on the purpose (split thickness or full thickness).

Step IV : Debride the area and place the flap apical to its original position and suture.

The edge of the flap can be located in any of the three following positions:



Figs 43.3A to F: Free soft tissue autograft (Accordion technique): (A) Lack of attached gingiva, (B) Recipient bed preparation, (C) Donor site immediately after the removal of tissue for grafting, (D) Donor tissue (with alternate incisions), (E) Graft transferred to recipient site and sutured in place, (F) Various steps in Accordion technique.

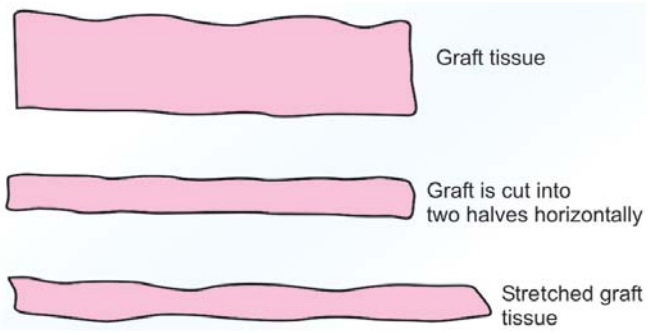


Fig. 43.4: Various steps in strip technique

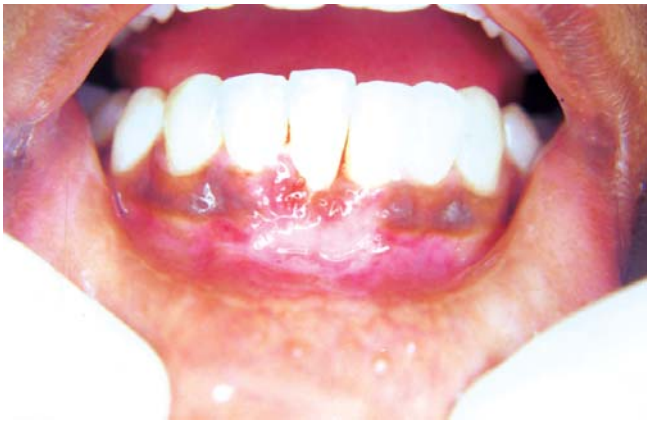


Fig. 43.5A: Healing of a grafted site

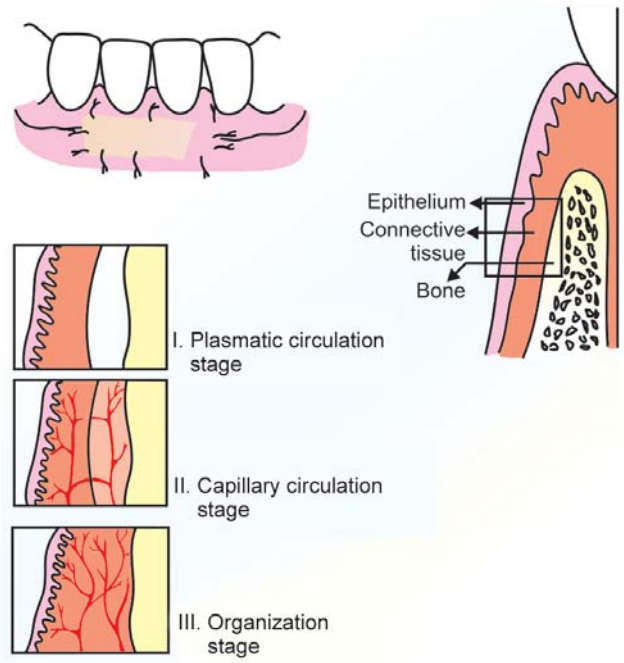


Fig. 43.5B: Healing phases of the graft

1. *Slightly coronal to the crest of the bone*—This may create the risk of recurrent pockets with thick gingival margins.
2. *At the level of the crest*—This results in satisfactory gingival contour.
3. *2 mm short of the crest*—This produces the most desired gingival contour because new tissue will cover the crest of the bone to produce a firm, tapered gingival margin. But it also increases the risk of a slight reduction in bone height.

Other Techniques for Widening the Zone of Attached Gingiva

Fenestration Operation/Periosteal Separation

It utilizes a partial thickness flap, except in a rectangular area at the base of the operative field where the periosteum is removed, exposing the bone. This is the area of fenestration and its purpose is to create a scar that is firmly bound to the bone.

The results obtained are not as predictable as those obtained with the free gingival graft or apically-displaced flap (Fig. 43.7).

Vestibular Extension Operation

Originally described by Edlan and Mejchar, it produces statistically significant widening of attached non-keratinizing tissue. Currently this technique is of historical interest only.

Procedure

The operative field is outlined by two vertical incisions from the junction of the marginal and attached gingiva to approximately 12 mm from the alveolar margin into the vestibule. The vertical incisions are joined by horizontal incision. A mucosal flap is elevated exposing the periosteum on the bone. The periosteum is separated from the bone, starting from the line of attachment of the mucosal flap. The periosteum including muscle attachments is transported to bone and is sutured to the inner surface of the periosteum. The periosteum is then transported to the lip and is, sutured where the initial horizontal incision was made.

Gingival Augmentation coronal to Recession(root coverage)

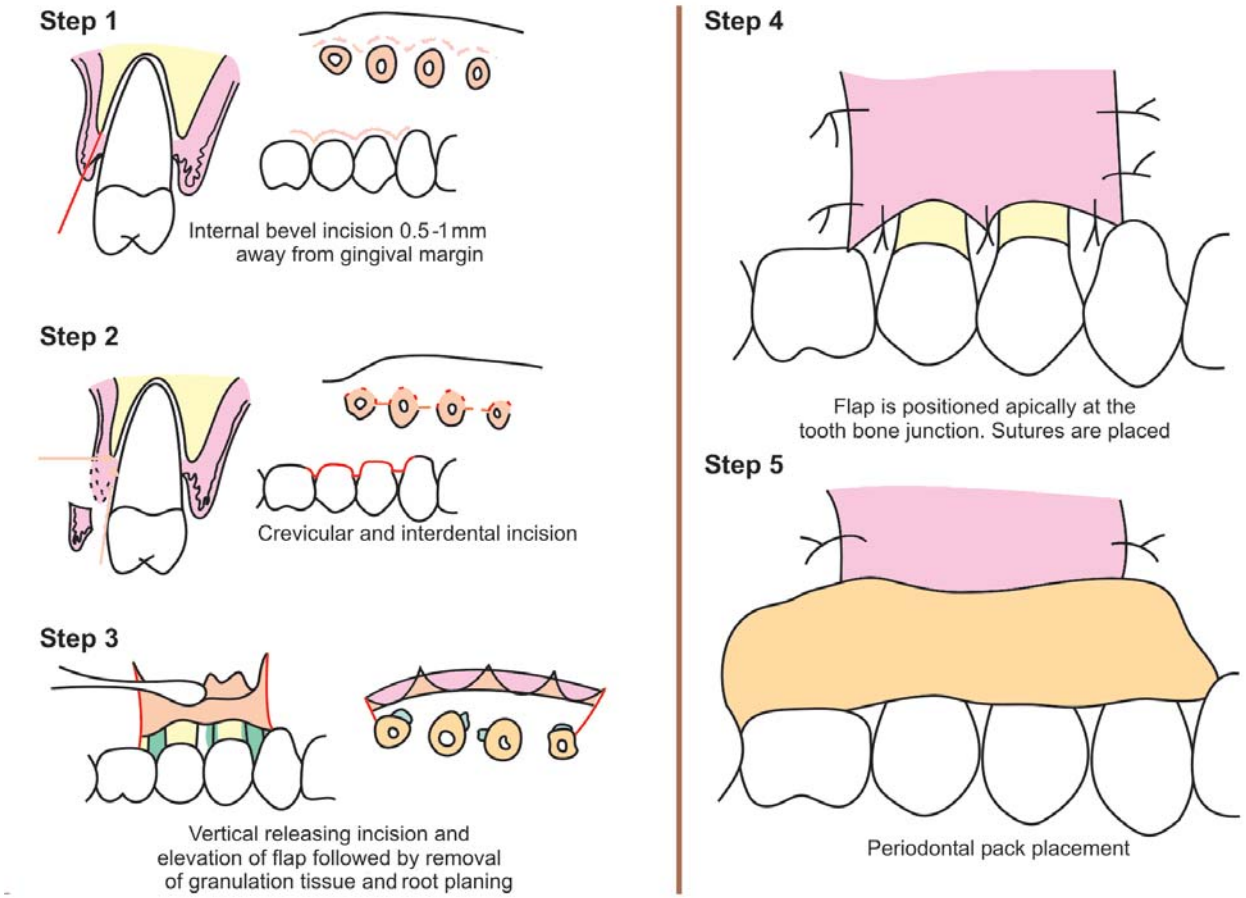


Fig. 43.6: Various steps in apically-displaced flap

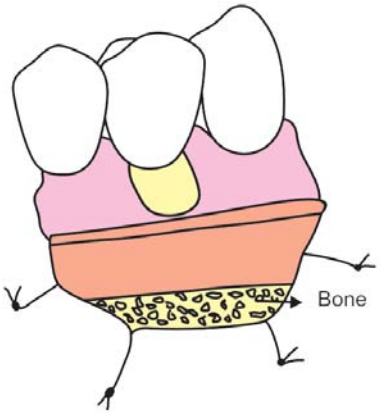


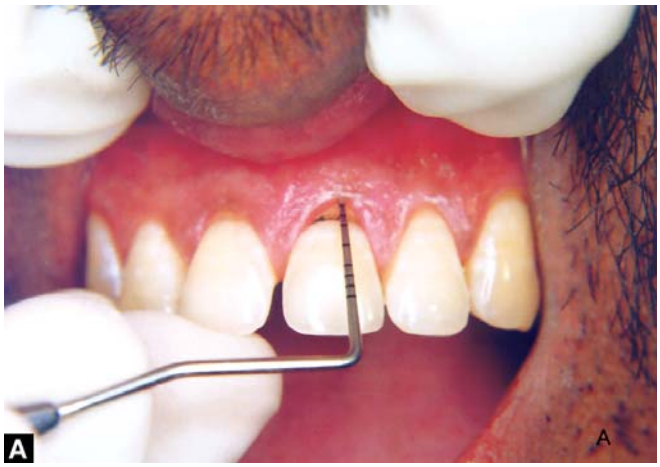
Fig. 43.7: Periosteal separation/fenestration

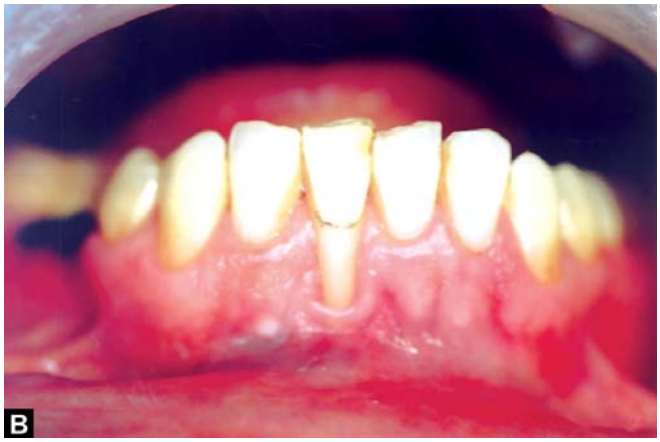
Procedures for Root Coverage

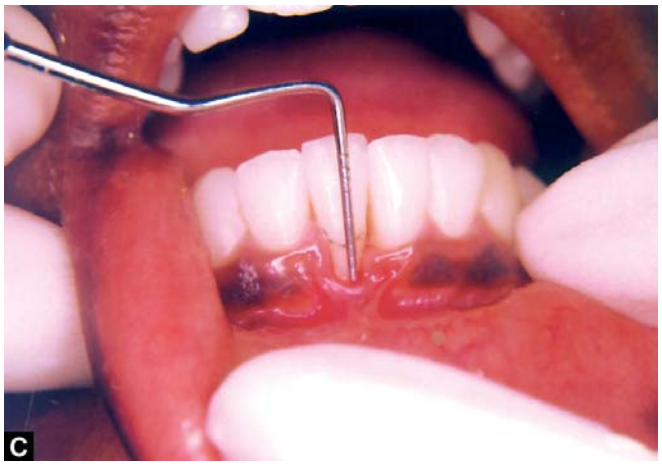
a. *Advantages for root coverage procedures are:*
   i. Reduces root sensitivity.
   ii. Improves esthetics.
   iii. Manage the defects resulting from root caries removal and/or cervical abrasions.
   iv. Manage mucogingival defect which fail to respond to altering abusive toothbrushing techniques and/or plaque removal.

b. *Classification*—Several classifications have been proposed in 1960s, *Sullivan and Atkins* classified isolated gingival recession into four types:
   i. Shallow-narrow.
   ii. Shallow-wide.
   iii. Deep-narrow.
   iv. Deep-wide.

Miller (1985) expanded this classification so as to help the clinician to predict the outcome of the therapy. The following four classes of recession have been proposed by Miller (Figs 43.8A to D). Recession can also be generalized and localized (Figs 43.9A and B).







Figs 43.8A to C: Miller's classification of gingival recession (Class I, II and III)

Class I: Marginal tissue recession that does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This type of recession can be narrow or wide.

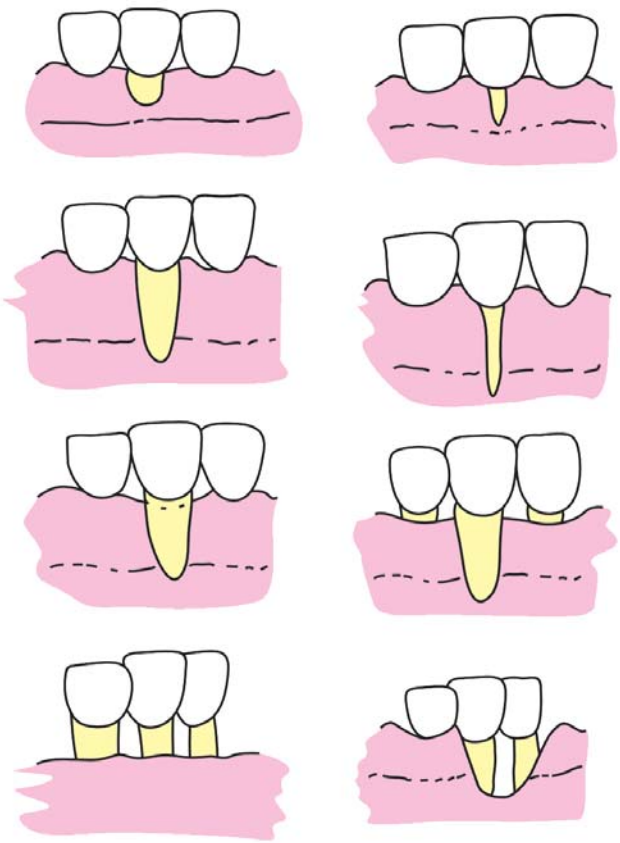


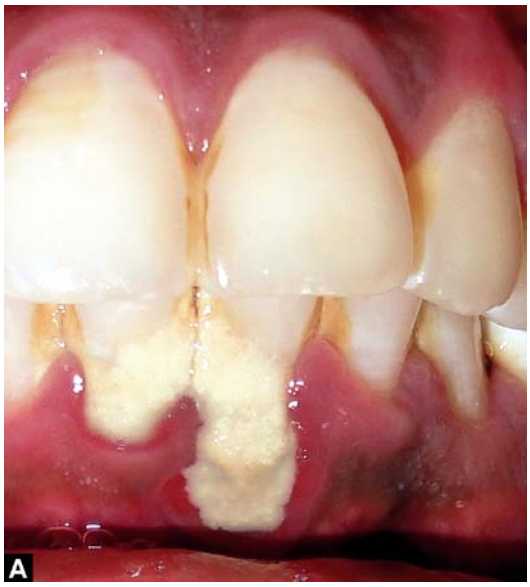
Fig. 43.8D: Class I, II, III and IV recession

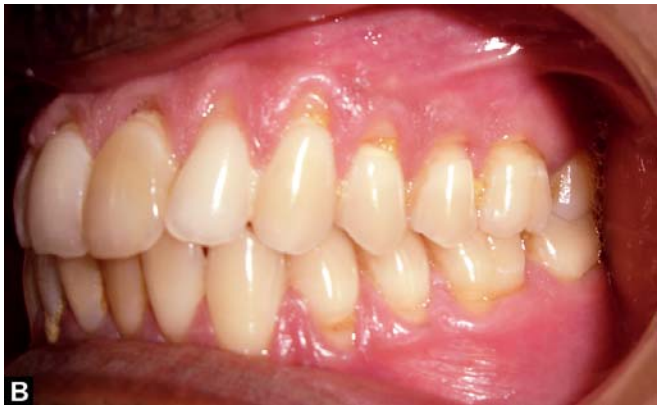
Class II : Marginal tissue recession that extends to or beyond the mucogingival junction. There is no loss of bone and soft tissue in the interdental area. This type of recession may be wide or narrow.

Class III : Marginal tissue recession that extends to or beyond the mucogingival junction. In addition, there is bone and/or soft tissue loss interdentally or tooth may be malposed.

Class IV : Marginal tissue recession extends to or beyond the mucogingival junction. With severe bone and soft tissue loss interdentally and/or severe tooth malposition.

Type of recession		*Prognosis*
Class I and II	—	good to excellent
Class III	—	only partial coverage can be expected
Class IV	—	very poor.





Figs 43.9A and B: (A) Localized recession, (B) Generalized recession

Procedures for root coverage can be divided into:

1. Conventional procedures.
2. Regenerative procedures.

Conventional procedures

Depending on the width of the attached gingiva, i.e. if adequate width is present at the donor site the following procedures can be selected:

a. Laterally (horizontally) displaced flap.
b. Double-papilla flap.
c. Coronally-positioned flap

If the donor site is associated with inadequate width:

i. Free soft tissue autograft.
ii. Subepithelial connective tissue grafts are available.

Regenerative procedures:

Guided tissue regeneration (GTR) has been proposed.

Pedicle autografts:

a. Laterally (Horizontally) displaced flap *(Figs 43.10A to C)*

In 1956, Grupe and Warren developed an original and unique procedure called the sliding flap operation.

Advantages of laterally-displaced flap:

a. One surgical site
b. Good vascularity of the pedicle flap.
c. Ability to cover isolated, denuded roots that have adequate donor tissue laterally.

Disadvantages of laterally-displaced flap:

a. Limited by the amount of adjacent keratinized attached gingiva.
b. Possibility of recession at the donor site.
c. Dehiscence or fenestration at the donor site.
d. Limited to one or two teeth with gingival recession.

Indications:

a. For covering the isolated denuded root.
b. When there is sufficient width of interdental papilla in the adjacent teeth.
c. Sufficient vestibular depth.

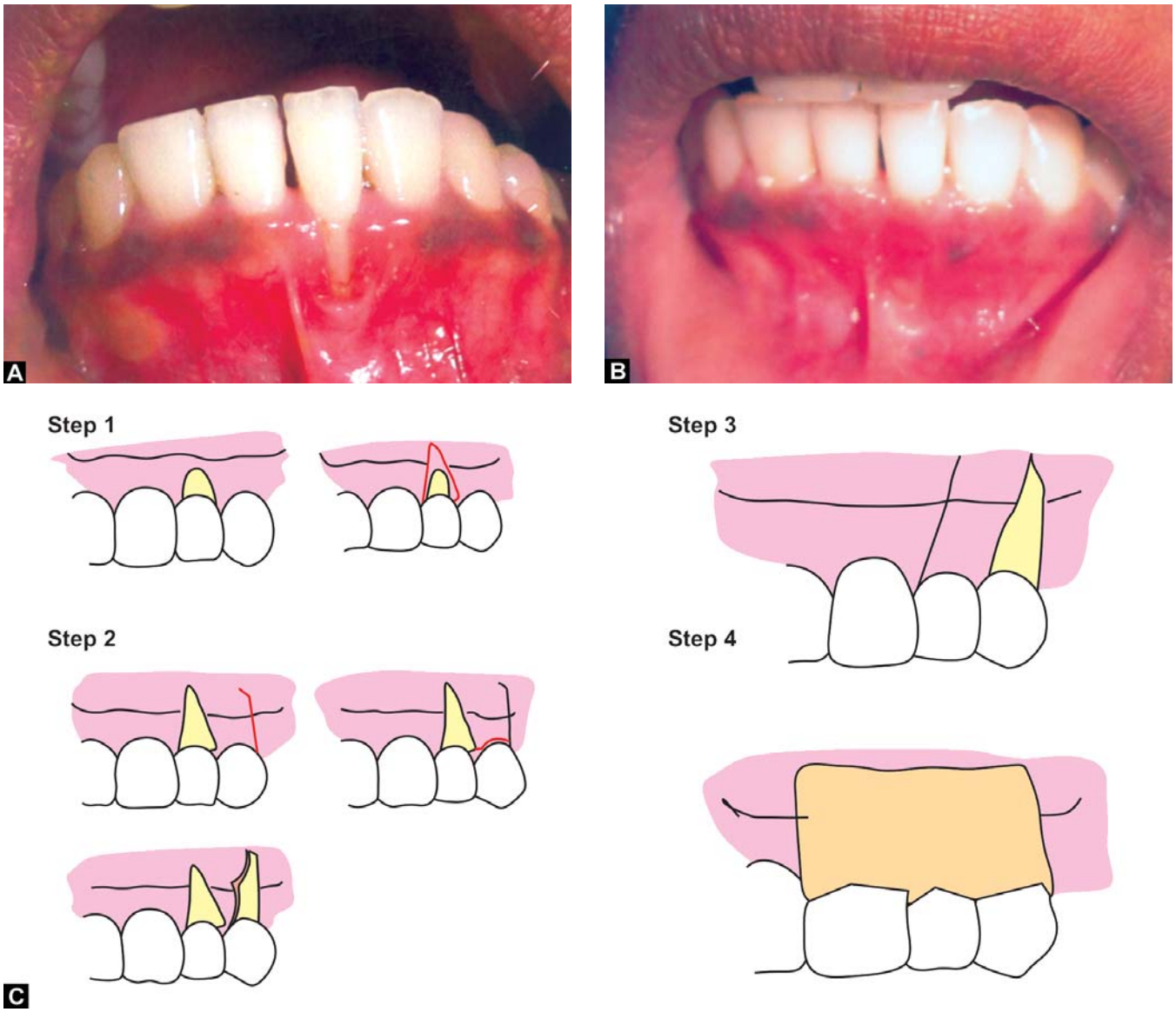
Contraindications:

a. Presence of deep interproximal pockets.
b. Excessive root prominence.
c. Deep or extensive root abrasion or erosion.
d. Significant loss of interproximal bone height.

The following is the step-by-step procedure for laterally-displaced flap:

Step I : *Preparation of the recipient site*
Make an incision, resecting gingival margin around the exposed roots. This band of marginal gingiva is removed with a scaler or curette. The exposed root surface is planed well. If granulation tissue is present along the incised edge of the gingiva, it should be removed carefully with curettes.

Step II : With a # 15 blade a vertical incision is made extending from marginal gingiva into the



Figs 43.10A to C: Laterally-displaced flap. (A) Preoperative view, (B) Postoperative view (after 2 month), (C) Various steps in laterally position flap

mucogingival junction. A crevicular incision is then made from the vertical incision to the defect. A flap is then raised utilizing either partial thickness or full thickness reflection. Each of these has its own advantages and limitations, e.g. partial thickness flap offers advantages like rapid healing at the donor site and reduced risk of facial bone loss. However, if the gingiva is thin, flap survival becomes difficult. It may sometimes be necessary to give a short oblique incision into the alveolar mucosa at the distal corner of the flap, pointing more towards the recipient site. This will enable us to slide the flap laterally without excess tension at the base.

Step III : Transfer the flap

After the flap is transferred onto the adjacent root, the flap is sutured to the adjacent gingiva and alveolar mucosa with interrupted sutures.

Step IV : Protect the flap and donor site

Cover the surgical site with a periodontal pack and after one week the pack and sutures can be removed. Postoperatively, antibiotics are not always necessary in the normal course of treatment, but analgesics are prescribed to

control pain. The flap may heal by connective tissue adhesion, or connective tissue attachment or long junctional epithelium. Some of the studies have shown better results with citric acid root conditioning.

Modifications:

Considering the drawbacks of the procedure many modifications of lateral sliding flap have been proposed.

a. Converging oblique incisions over the recipient site and a vertical or oblique incision at the distal end of the donor site so that the flap which is being transposed is wider at its base.
b. In the donor site, the marginal periodontium is left undisturbed in order to reduce the likelihood of recession and bone resorption.

b. Double papilla flap (Figs 43.11A to G)

First described by Wainberg as the Double Lateral Repositioned Flap and was refined by Cohen and Ross as the Double Papilla Flap.

Indications:

1. When the interproximal papillae adjacent to the mucogingival problem are sufficiently wide.
2. When the attached gingiva on an approximating tooth is insufficient to allow for a Lateral Pedicle Flap.
3. When periodontal pockets are not present.

Advantages:

1. The risk of loss of alveolar bone is minimized because the interdental bone is more resistant to loss than is radicular bone.
2. The papillae usually supply a greater width of attached gingiva than from the radicular surface of a tooth.
3. The clinical predictability of this procedure is fairly good.

Disadvantage:

Technique sensitive—Having to join together the small flap in such a way so that they act as a single flap.

c. Coronally-repositioned flap or coronally-positioned flap (Figs 43.12 and 43.13)

Indications:

- Esthetic coverage of exposed roots.
- For tooth sensitivity owing to gingival recession.

Advantages:

- Treatment of multiple areas of root exposure.
- No need for involvement of adjacent teeth.
- High degree of success.
- Even if the procedure does not work, it does not increase the existing problem.

Disadvantage:

There is a need for two surgical procedures if the zone of keratinized gingiva is inadequate.

This procedure can be performed utilizing two techniques.

First Technique

Step 1 : Make two apically-divergent vertical releasing incisions, extending from a point coronal to the cementoenamel junction at the mesial and distal line angles of the tooth and apically into the lining mucosa.

Step 2 : A split thickness flap is prepared by sharp dissection at the mesial and distal ends and is connected with an intracrevicular incision. Facially, apical to the recession, a full thickness flap is raised.

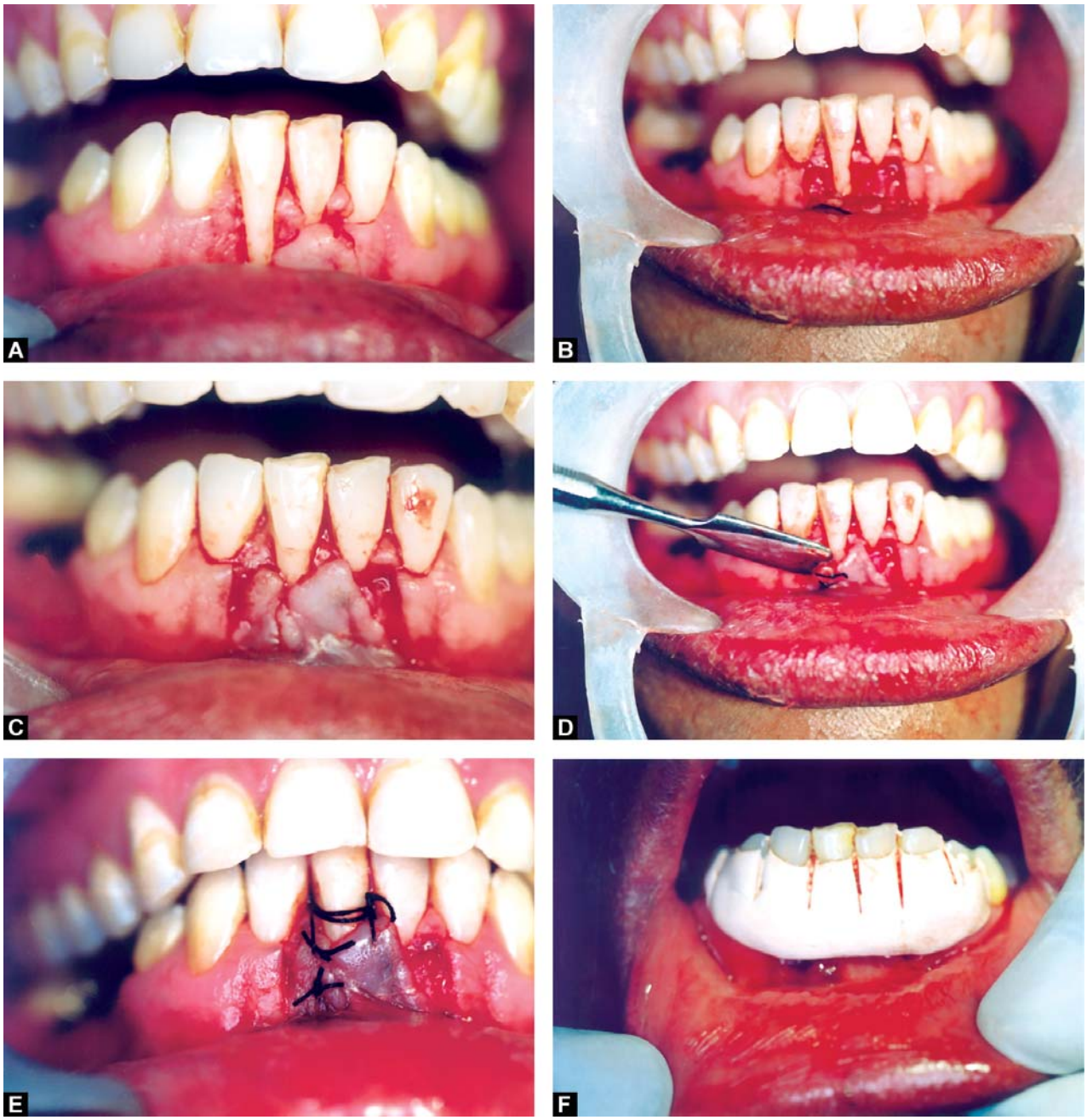
Step 3 : Once the flap is reflected the root surfaces are debrided thoroughly. Some authors have suggested the use of citric acid with a pH 1.0 for conditioning the root surface.

Step 4 : At the base of the inner surface of the flap, approximately 3 mm apical to the bone dehiscense, a horizontal incision is made through the periosteum, followed by a blunt dissection into the lining mucosa to release muscle tension. Now the mucosal graft can be easily positioned coronally at the level of cementoenamel junction.

Step 5 : The flap is secured firmly with the help of interrupted sutures and additional sling sutures can be placed to maintain the flap in place. Periodontal dressing is placed to protect the wound during initial healing.

Modifications:

One of the limitations of this procedure is, it cannot be performed when there is insufficient width of attached



Figs 43.11A to F: Double papilla flap. (A) Recipient site preparation, (B) Two vertical or oblique incisions at mesial and distal end of the defect, (C) Flaps are reflected (partial thickness), (D) The transposed flaps are sutured to obtain a single flap, (E) Final suturing, (F) The area covered with periodontal dressing

gingiva. To solve this problem, a two stage surgical procedure has been proposed. First, a gingival extension operation with a free gingival graft is performed. Two months later a second stage surgery is performed by coronally repositioning the flap which may include free soft tissue graft.

Second Technique

Semilunar flap *(Figs 43.14A to C)*

Indication:

- Areas where gingival recession is only 2 to 3 mm.

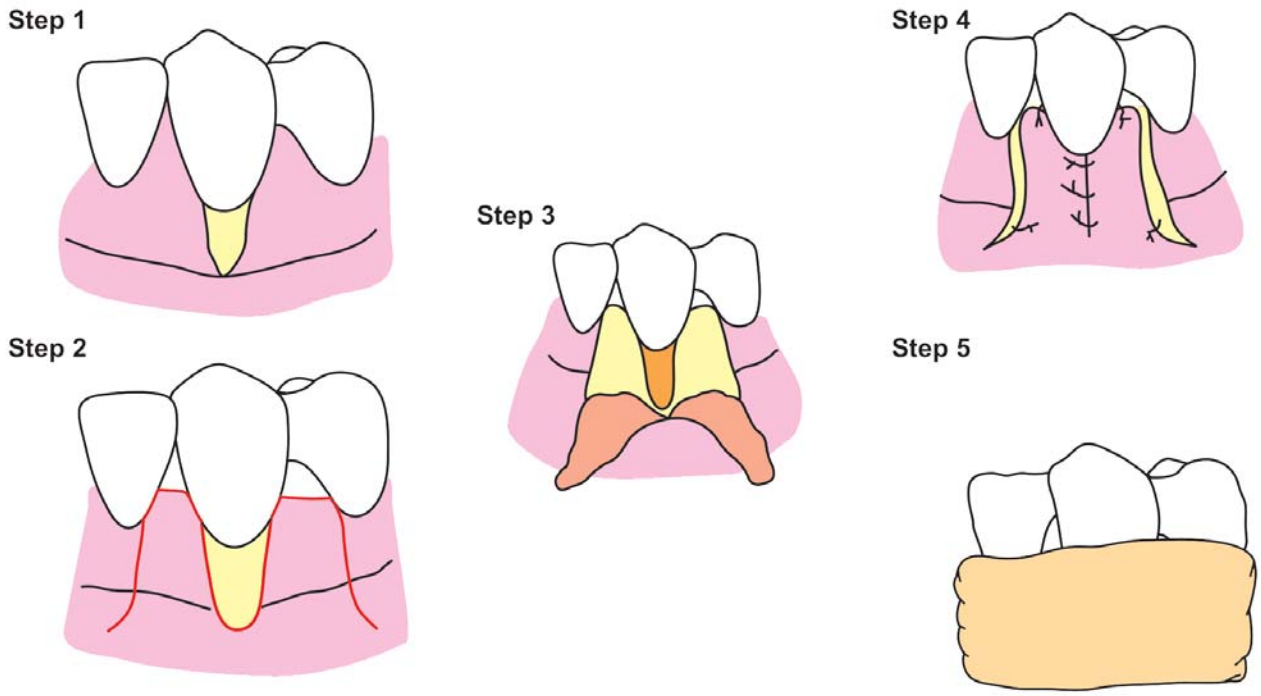
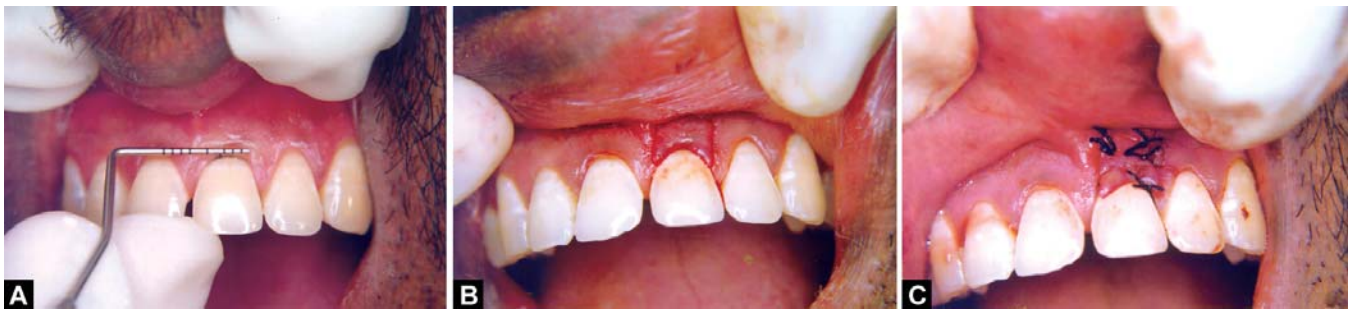


Fig. 43.11G: Various steps in double papilla flap (Refer the text for explanation)



Figs 43.12A to C: Coronally-positioned flap. (A) Measure the width and length of gingival recession (class I), (B) Two vertical releasing incisions are placed, (C) Flap positioned coronally and sutured

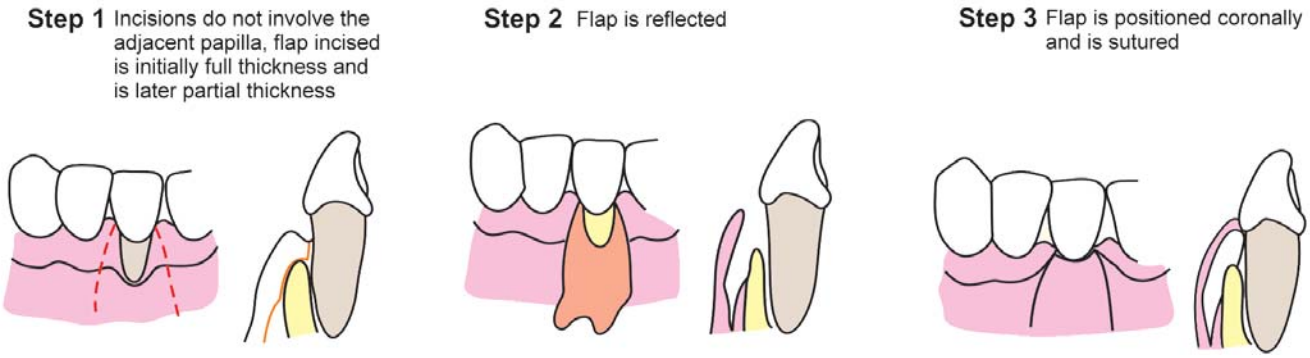
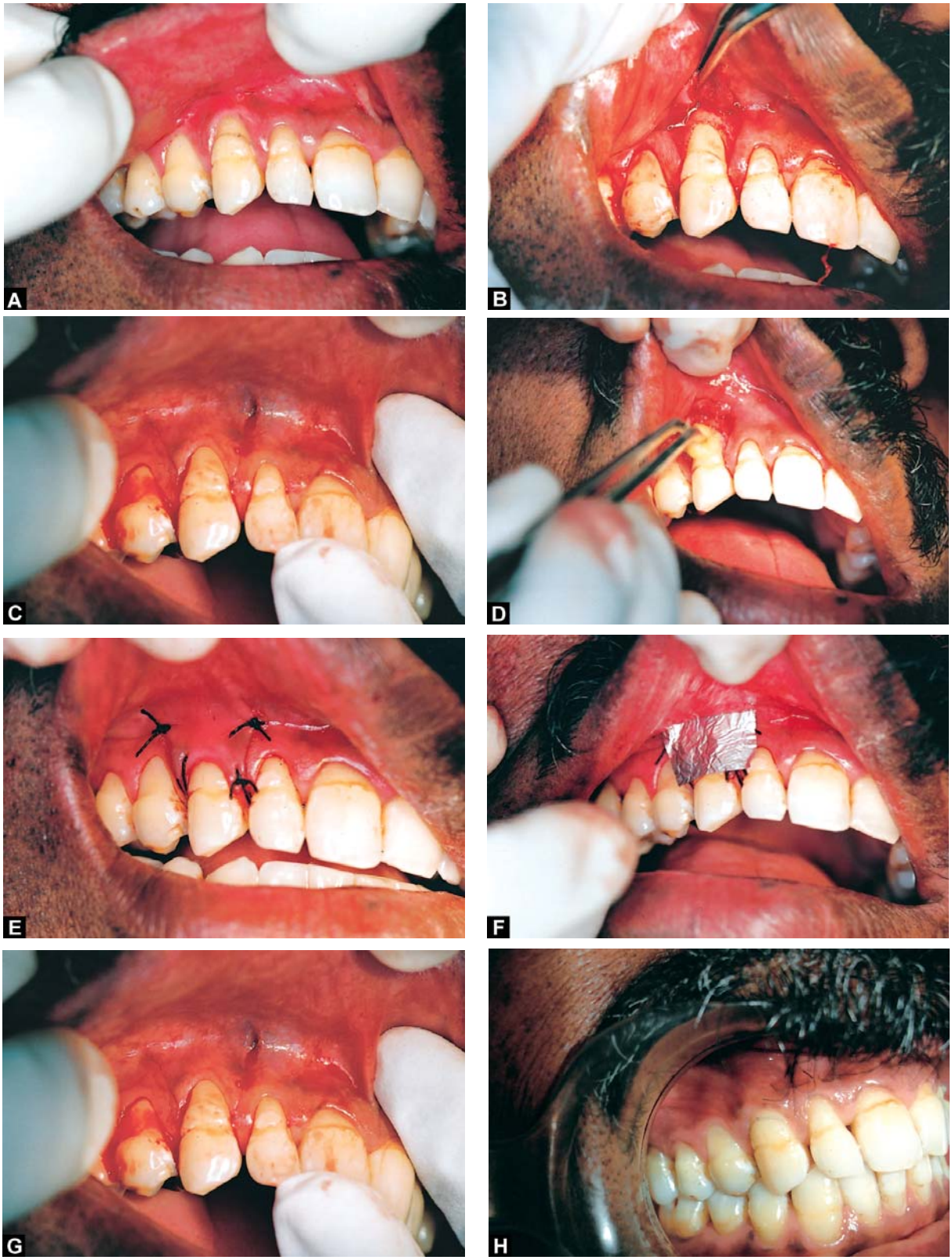
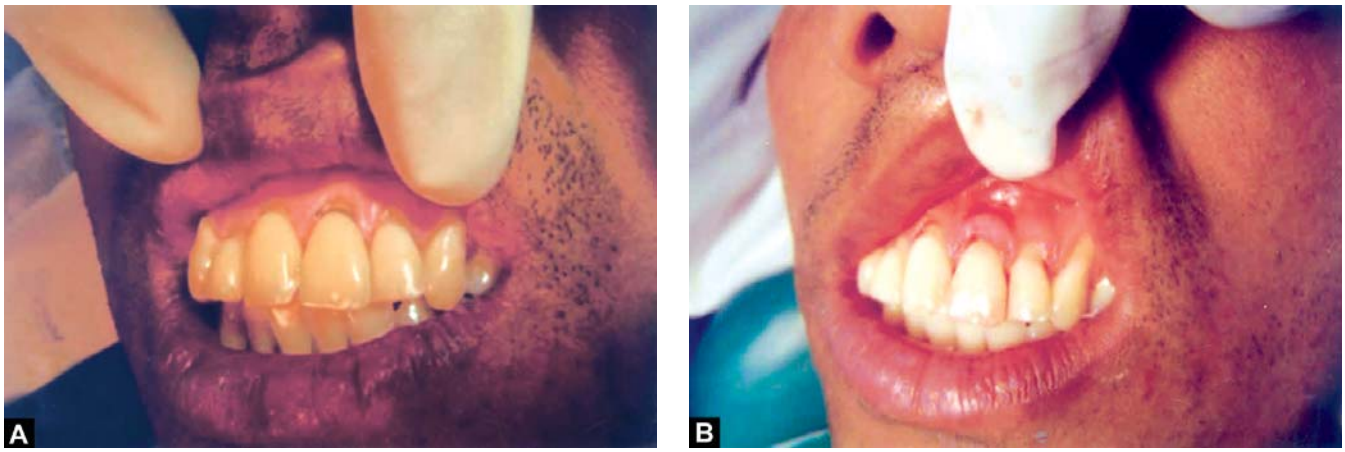


Fig. 43.12D: Various steps in coronally-positioned flap (Refer the text for explanation)



Figs 43.13A to H: Case 2: Coronally repositioned flap procedure. (A) Preoperative view showing class I Miller's recession. (B) Incision, (C) Reflection of flap, (D) Coronally displaced flap, (E) Root conditioning with tetracycline soaked cotton pellet, (F) Suturing of coronally displaced flap, (G) Tin foil placed prior to pack placement, (H) Facial view after healing



Figs 43.14A and B: Semilunar flap: (A) Class I gingival recession in relation to 21-preoperative view, (B) Semilunar incision and coronal displacement of flap

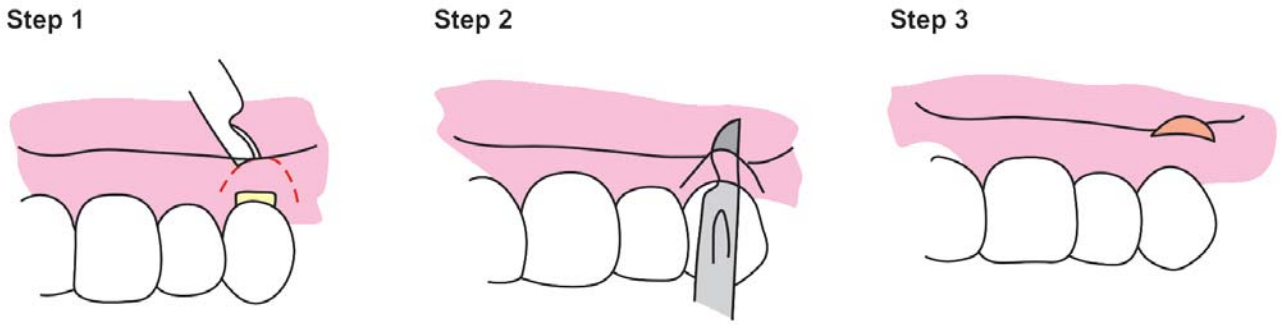


Fig. 43.14C: Various steps in semilunar flap (Refer the text for explanation)

Advantages:

- No vestibular shortening, as occurs with the coronally-positioned flap.
- No esthetic compromise of interproximal papillae.
- No need for sutures.

Disadvantages:

- Inability to treat large areas of gingival recession.
- The need for a free gingival graft if there is an underlying dehiscence or fenestration.

Step 1 : A semilunar incision is placed, following the curvature of the gingival recession, make sure that the incision ends about 2 to 3 mm short of the tip of the papillae. This is very important, as the flap derives all of its blood supply from the adjacent papillary areas.

Step 2 : Split thickness dissection is performed coronally and this is connected to an intracrevicular incision.

Step 3 : The tissue will now collapse freely, covering the denuded root. The flap is then held in place with a moist gauze for a few minutes. Suturing is not generally required. Periodontal dressing may be placed.

Free Gingival Autograft

The classic technique proposed for creating a widened zone of attached gingiva was described in the earlier section of this chapter. Miller in 1985, applied this with few modifications to cover the denuded roots. The procedure is as follows:

Step 1 : A horizontal right angled incision is made in the interdental papillae, followed by, vertical incision at the proximal line angles of the adjacent teeth. The retracted tissue is excised. The periosteum should be left intact in the apical areas. A graft is obtained from the donor site, transferred to the recipient site and immobilized. Donor site is protected as described earlier.

Subepithelial Connective Tissue Graft (Figs 43.15A to F)

Proposed by Langer and Langer in 1985. In 1994, Bruno described modification of the original Langer and Langer technique.

Indications:

- Where esthetics is of prime concern
- For covering multiple denuded roots
- In the absence of sufficient width of attached gingiva in the adjacent areas.

Advantages:

- High degree of cosmetic enhancement
- Incurs no additional cost for autogenous donor tissue
- One step procedure
- Minimal palatal trauma
- Increased graft vascularity.

Disadvantages:

- High degree of technical skills required.
- Complicated suturing.

The technique is as follows:

I. *Preparation of recipient site:* The initial horizontal right angle incision is made into the adjacent interdental papillae at, or slightly coronal to the cementoenamel junction of the tooth with an exposed root surface. A butt joint is provided. It is made sure that the papillary incisions are not more than 1 mm deep, this is done to preserve the papillary blood supply. A partial thickness flap is raised without vertical incisions. The exposed root is meticulously planed with curettes. At times, it may be desirable to use finishing burs to reduce the root convexity.

   Following root planing, the root is treated with either citric acid pH 1.0 or tetracycline HCl in a concentration of 250 mg mixed in 5 ml of sterile water. The solution is applied to the root surface for 2 to 3 minutes with cotton pellets. Once the root is treated, the approximate mesiodistal width necessary for the graft is measured with a periodontal probe.

II. *Excision of the donor tissue (Fig. 43.16):* The first incision is made approximately 2 to 3 mm apical to the gingival margin, perpendicular to the long axis of the teeth. The second incision is made parallel to the long axis of the teeth, 1 to 2 mm apical to the first incision. A small periosteal elevator is used to raise a full thickness periosteal connective tissue graft. Vertical incisions may be necessary at the mesial and distal extent of the graft to facilitate easy removal of the connective tissue.

   Once the graft is removed, the area is sutured with 4-0 silk suture material.

III. *Grafting to the recipient site:* The donor connective tissue is secured to the papillae with interrupted sutures and the overlying partial thickness flap is then replaced over the donor tissue and interrupted sutures are placed in the mesial and distal papillae. No attempt is made to cover the donor tissue completely. A periodontal dressing is placed to cover the surgical site. The routine postoperative care is followed. The dressing and sutures are removed after seven days postoperatively.

Modifications:

In the recipient site preparation:

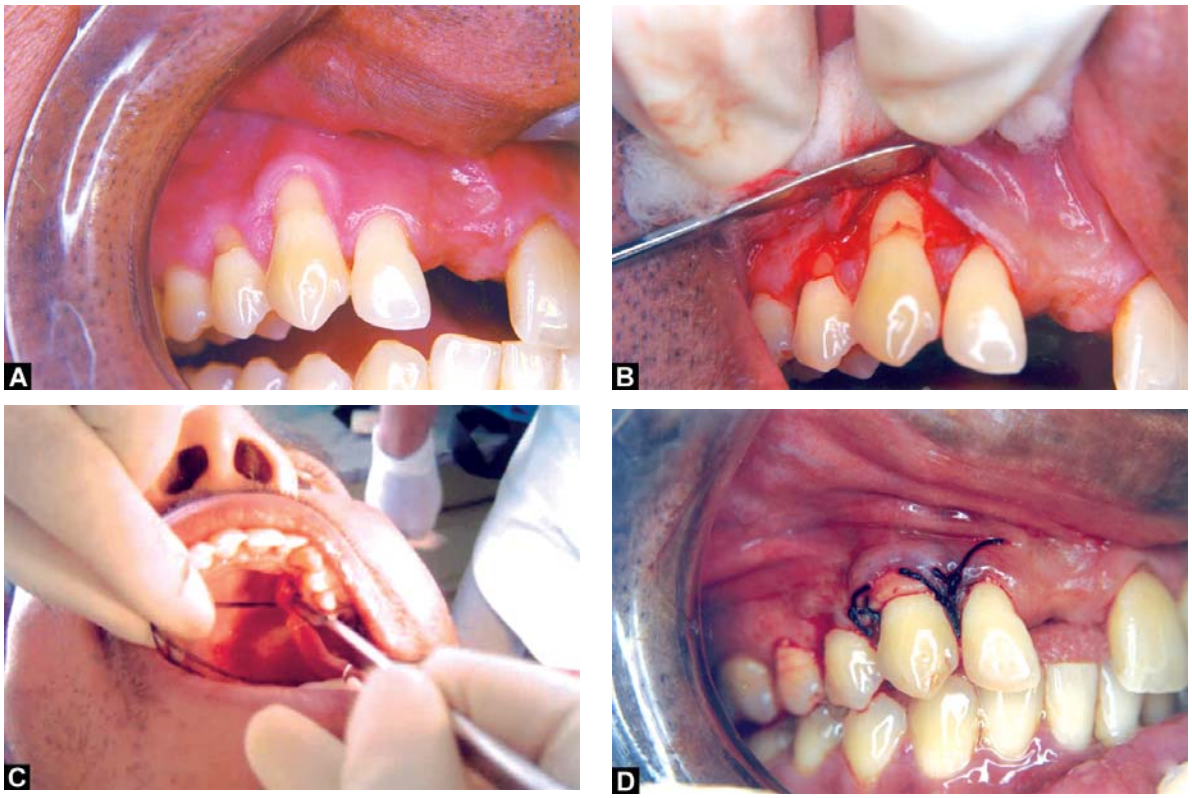
a. ***Envelope technique* (Fig. 43.17):** In this technique, first eliminate the sulcular epithelium by an internal bevel incision. Secondly, an 'envelope' is prepared apically and laterally to the recession by split incision.

b. ***Langer's technique* (Figs 43.18A to D):** In addition to the horizontal incision, two vertical releasing incisions 1 to 2 mm away from the gingival margin, extending one-half to one tooth wider mesiodistally than the area of gingival recession, is given.

c. ***Pouch and tunnel technique* (Figs 43.19 and 43.20):** In case of multiple adjacent recessions "envelopes" are prepared on each tooth for receiving connective tissue graft.

Procuring the Graft from the Donor Site

In the palate, a partial thickness flap is raised with two vertical releasing incisions, followed by this, the connective tissue is harvested by placing two horizontal and two vertical incisions to the bone. After the graft removal, the flap is positioned back and sutured.



Figs 43.15 A to E: Subepithelial connective tissue graft. (A) A recession defect at a maxillary right canine, (B) Recipient site preparation, (C) Procuring connective tissue graft from the palate, (D) The graft is placed in the recipient site and secured in position with interrupted sutures, (E) Shows 3 months healing result

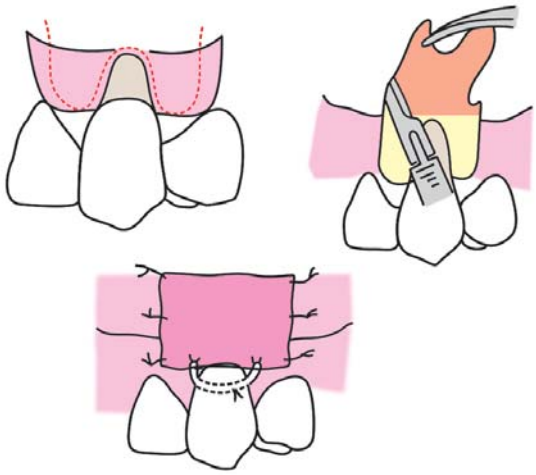


Fig. 43.15F: Various steps in free connective tissue graft—Recipient site preparation (Refer the text for explanation)

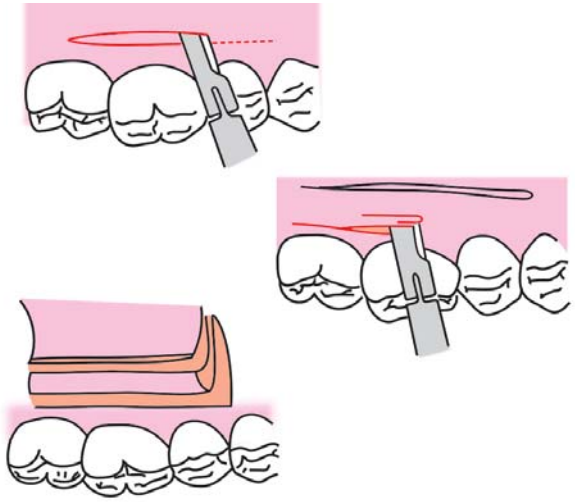


Fig. 43.16: Various steps in free connective tissue graft—Donor site preparation

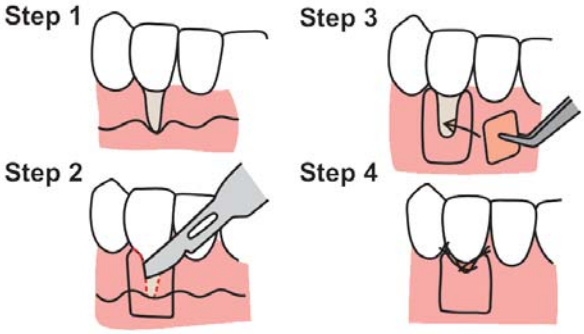
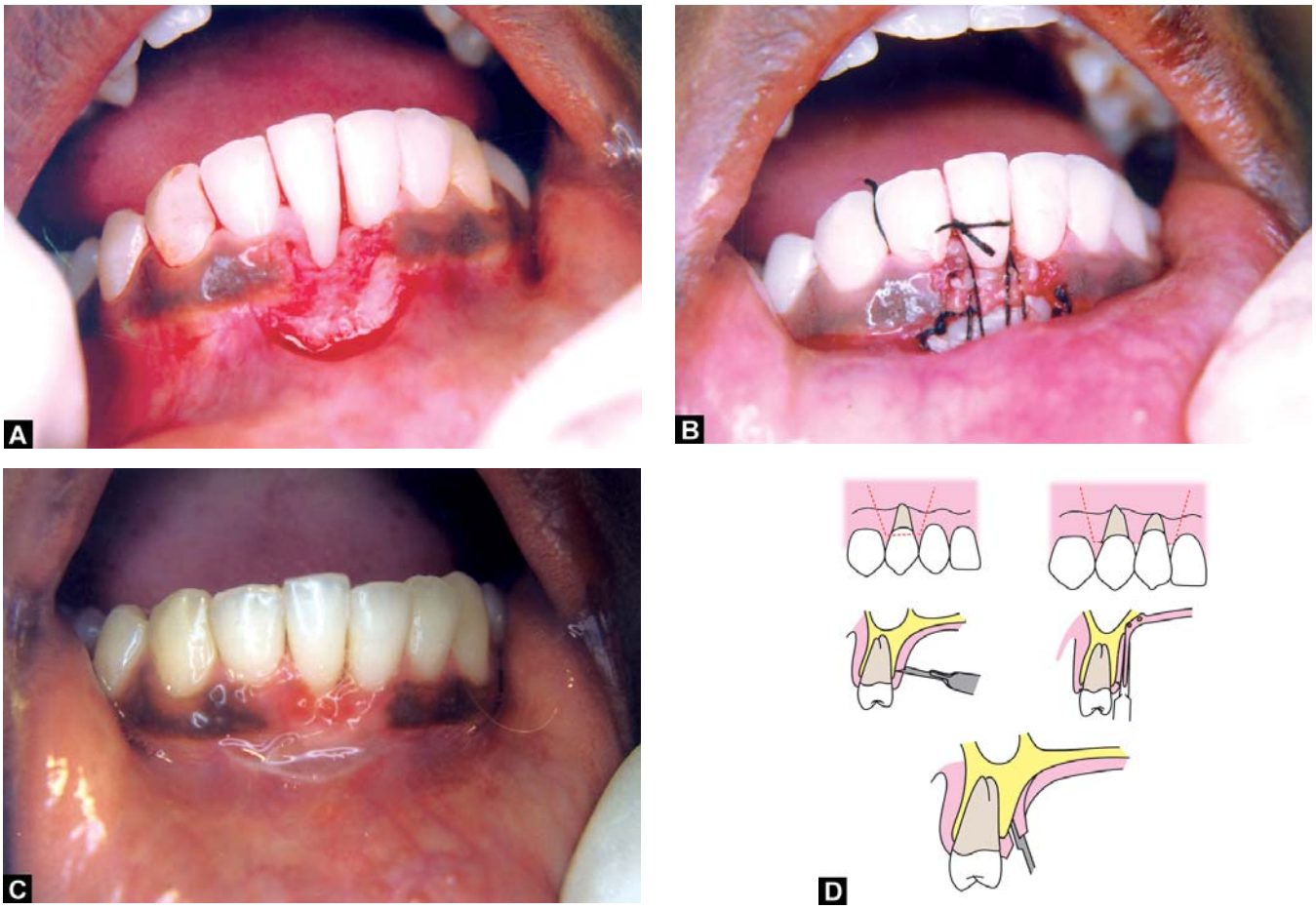


Fig. 43.17: Various steps in free connective tissue graft—Envelope technique



Figs 43.18A to D: Subepithelial connective tissue graft (Langer's technique). (A) A gingival flap is reflected with two vertical incisions, (B) A large connective tissue graft is sutured in place, (C) Two weeks postoperative view, (D) Various steps in Langer's technique

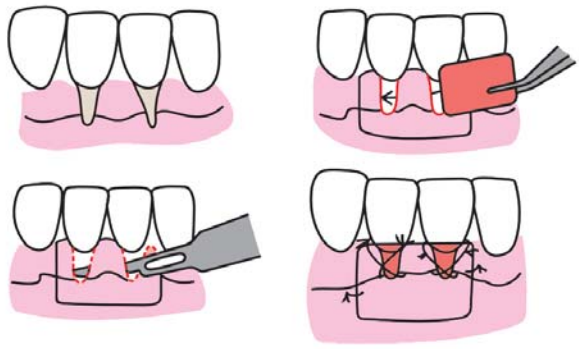
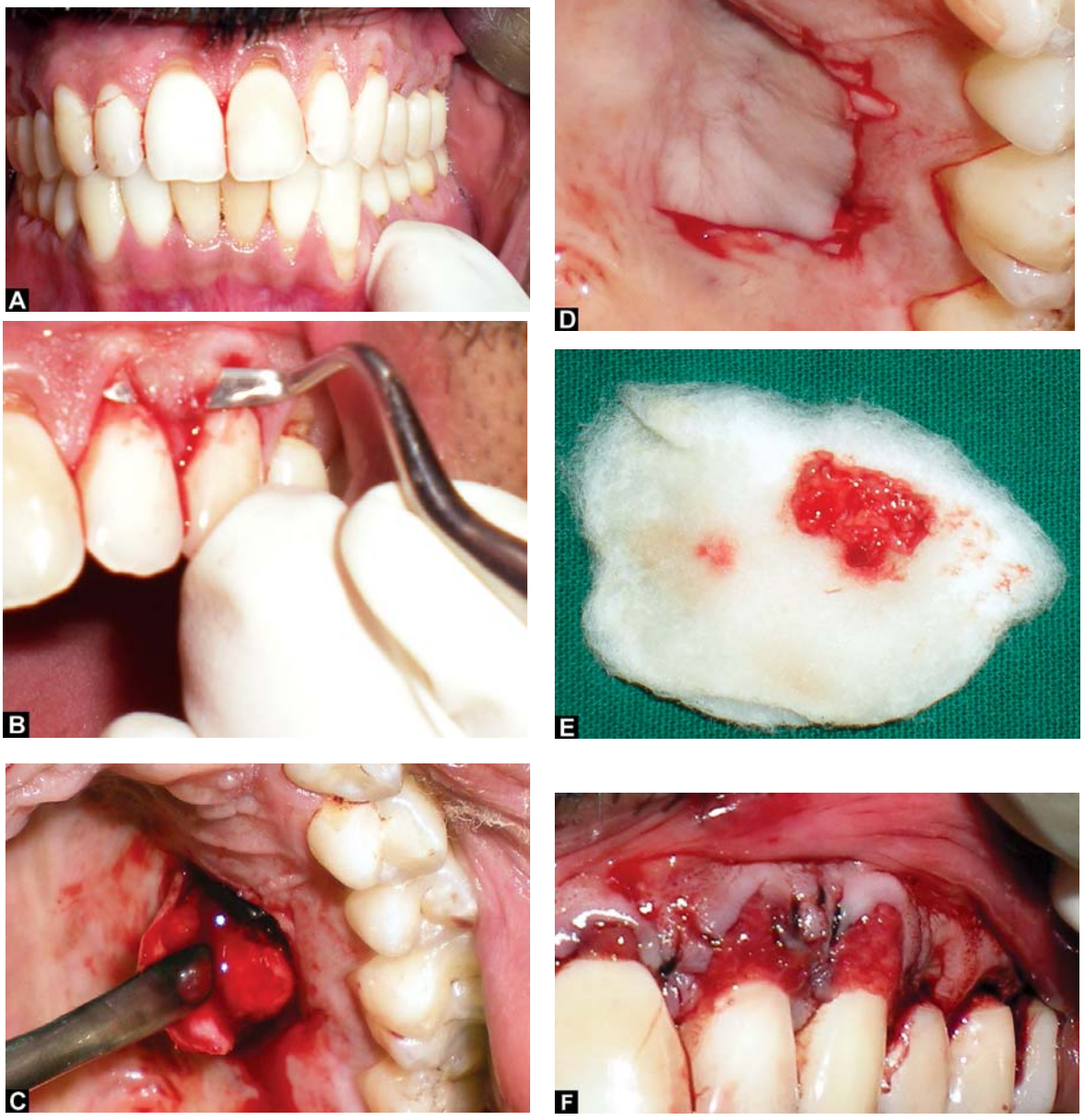
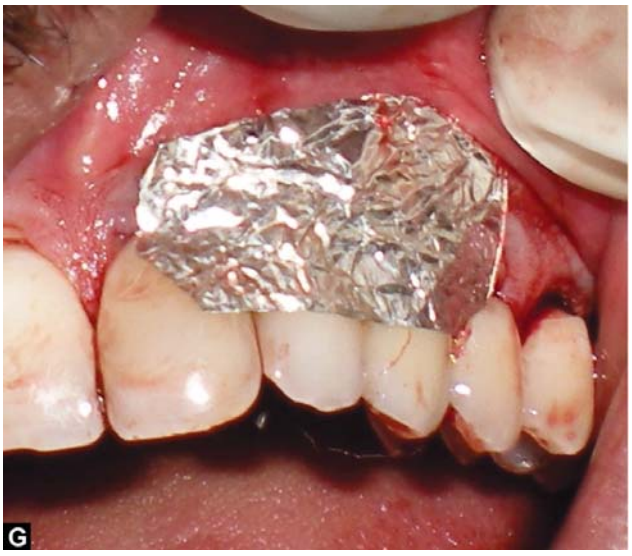
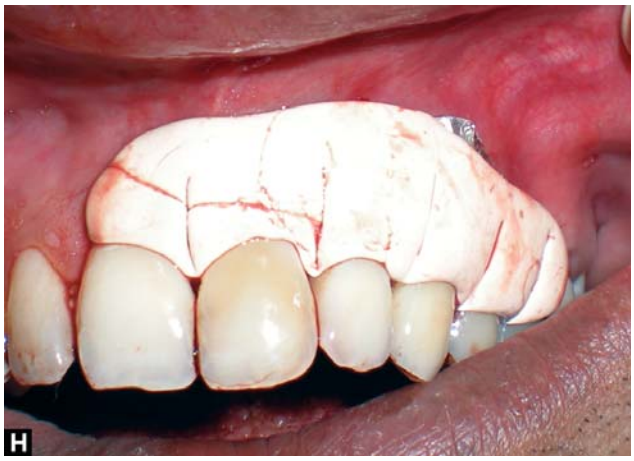


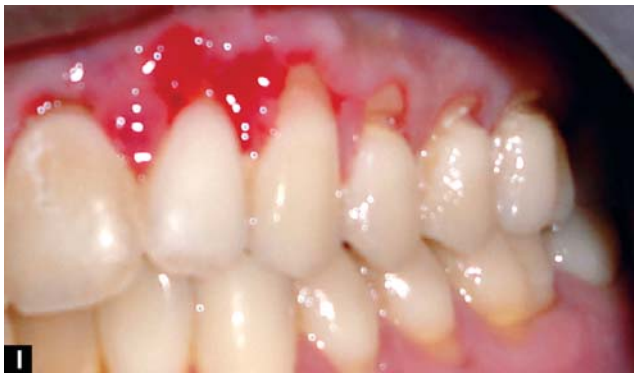
Fig. 43.19: Various steps in pouch and tunnel technique



Figs 43.20A to F: Various steps in tunnel procedure







Figs 43.20G to I: Various steps in tunnel procedure

GUIDED TISSUE REGENERATION TECHNIQUE FOR ROOT COVERAGE

Indications

- Esthetic demand.
- Indicated for single tooth with wide, deep localized recessions.
- For areas of root sensitivity where oral hygiene is impaired.
- For repair of recessions associated with failing or unesthetic class V restorations.

Advantages:

- Techniques does not require a secondary donor surgical site reducing postoperative discomfort.
- New tissue blends evenly with the adjacent tissue, providing highly esthetic results.

Disadvantages:

- It is sensitive technique.
- Insurance of additional cost of barrier membrane.

The technique consists of the following steps:

Step 1 : A full thickness flap is raised up to the mucogingival junction, beyond it, atleast 8 mm apical to the mucogingival junction a partial thickness flap is raised.

Step 2 : The root surface is meticulously planed, and a Goretex membrane is trimmed according to the size of the defect and tied to the tooth. Make sure that it covers at least 2 mm of marginal periosteum. In order to prevent the membrane from collapsing in the defect, some authors have suggested to pass a suture through the membrane, that covers the bone and this suture is knotted on the exterior and tied to bend the membrane, so that a space is created between the root and the membrane.

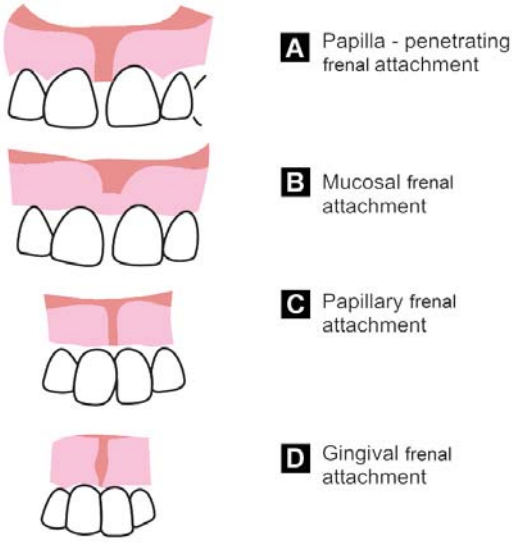
Step 3 : The flap is then positioned coronally and sutured. Four weeks later, the membrane is carefully removed without disturbing the growing tissue.

Modifications

a. *Titanium–reinforced membranes* are used to create the space below the membrane.
b. *Resorbable membranes* have been used to prevent a second surgery.

OPERATIONS FOR REMOVAL OF FRENA

A frenum is a fold of mucous membrane usually with enclosed muscle fibers, that attaches the lips and cheeks to the alveolar mucosa and/or gingiva and underlying periosteum.



Figs 43.21A to D: Types of frenal attachments

Indications

To prevent:

1. The accumulation of irritants.
2. The deflection of the wall of periodontal pocket which may aggravate its severity.
3. Its interference with post-treatment healing.
4. Pocket formation.
5. Injury while brushing.

Frenectomy: It is the complete removal of frenum including its attachment to the bone. It is indicated for, correction of abnormal diastema.

Frenotomy: It is the incision and relocation of the frenum to create a zone of attached gingiva between the gingival margin and the frenum (Suffices for periodontal problems).

Frenal attachment may be of four types (Figs 43.21A to D).

a. *Papillary:* Where the frenum is inserted into the interdental papilla.
b. *Mucosal type:* Where the frenum is attached in the alveolar mucosa.
c. *Papillary penetrating types:* Where the frenum is inserted from the facial to palatal papilla.
d. *Gingival:* Where the frenum is in the attached gingiva.

Techniques for the Removal of the Frenum (Figs 43.22A to I)

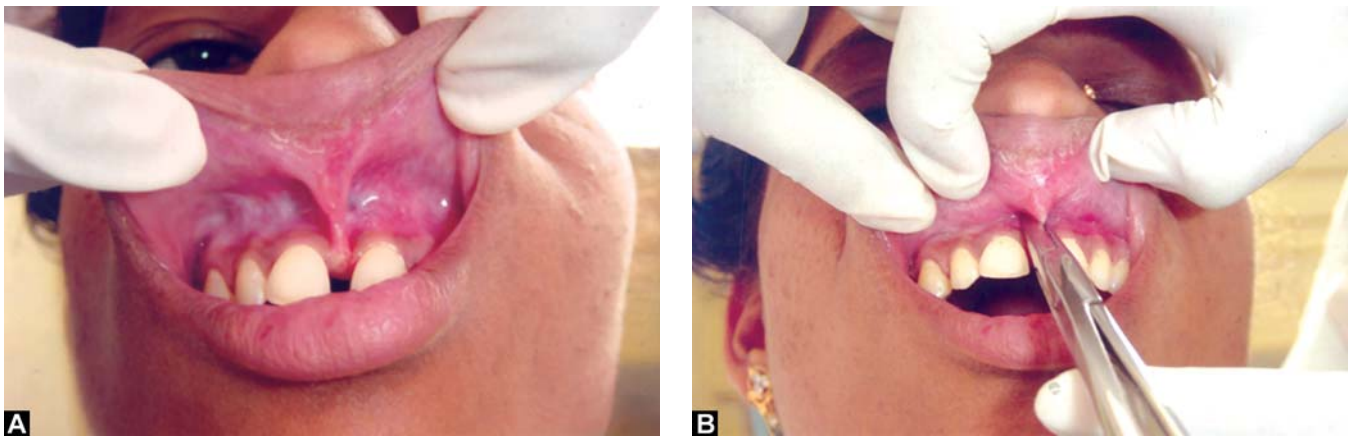
Step 1 : After anesthetizing the area, engage the frenum with a hemostat.

Step 2 : Incise along the upper surface of the hemostat, simultaneously make a similar incision along the under surface of the hemostat.

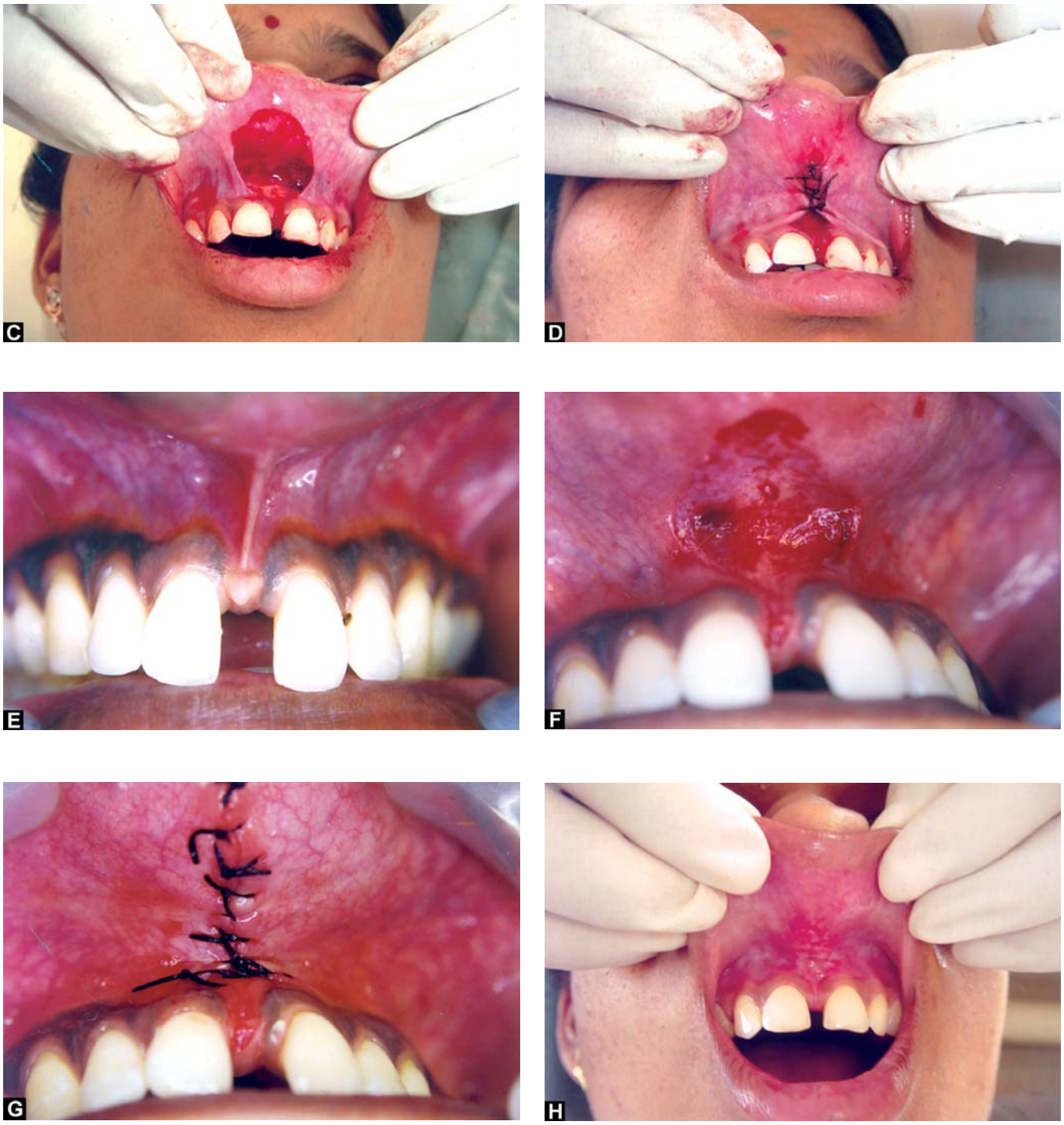
Step 3 : Remove the triangular resected portion of the frenum along with hemostat. This exposes the fibrous connective tissue attachment to the bone.

Step 4 : Make a horizontal incision to dissect and separate the fibers attached to the bone.

Step 5 : Close the wound by placing interrupted sutures.



Figs 43.22A and B: Frenectomy procedure. (A) A case of high frenal attachment with tension test being positive, (B) Engage the frenum with a hemostat



Figs 43.22C to H: (C) After removal of triangular resected portion of the frenum, (D) The wound is closed with interrupted sutures, (E) A case of frenectomy, (F) Frenum relieved, (G) Sutures placed, (H) Clinical appearance parmediately after suture removal

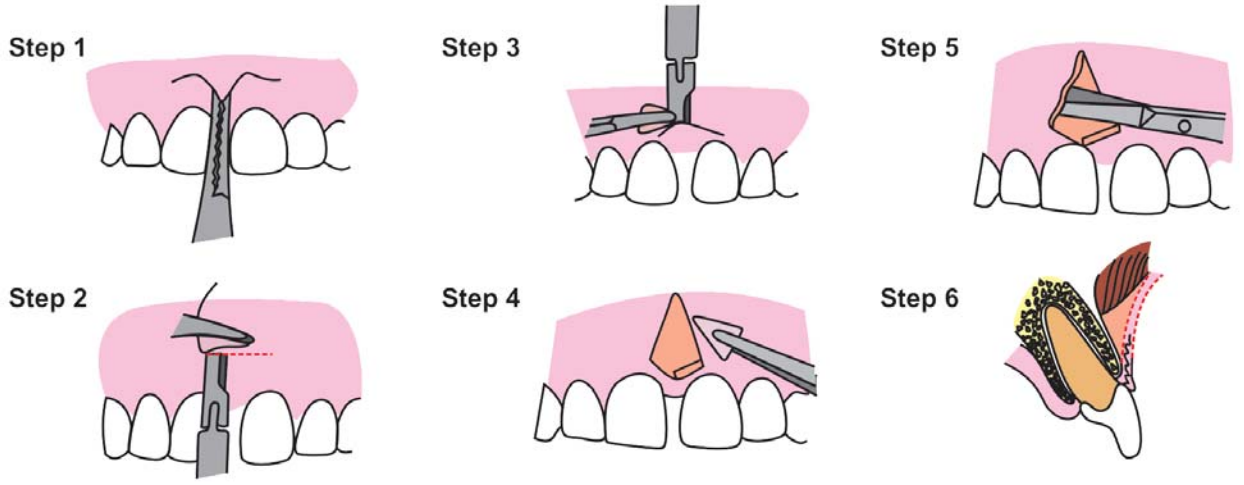


Fig. 43.22I: Various steps in frenectomy (Refer the text for explanation)

KEYPOINTS

1. Mucogingival surgery consists of plastic surgical procedures for the correction of gingiva mucous membrane relationships that complicate periodontal diseases and may interfere with the success of periodontal treatment.
2. Recently, it has been renamed as "Periodontal plastic surgery".
3. Most commonly performed mucogingival surgical procedures are:
   - Techniques to increase width of attached gingiva
   - Root coverage procedures
   - Operations for removal of frena.
4. Techniques for increasing width of attached gingiva are:
   - Gingival extension operation by free soft tissue autograft.
   - Apical displacement of the pocket wall and other techniques like fenestration operation and vestibular extension operation.
5. Sullivan and Atkins have classified gingival recession into, shallow-narrow, shallow-wide, deep-narrow and deep-wide, PD Miller has modified this into Class-I, Class-II, Class-III and Class-IV gingival recession.
6. Procedures for root coverage are divided into:
   - Conventional, e.g. pedicle autografts.
   - Regenerative procedures.
7. If the width of the attached gingiva is adequate in the donor site the following conventional procedures can be selected:
   - Laterally displaced flap
   - Double papilla flap
   - Coronally-positioned flap and semilunar flap.
8. If the donor site is associated with the inadequate width:
   - Free soft tissue autograft, and
   - Subepithelial connective tissue grafts are available.
9. Regenerative procedures like guided tissue regeneration (GTR) using various barrier membranes have been used successfully to cover the denuded roots.
10. Frenectomy is the complete removal of frenum including its attachment to the bone. Whereas frenotomy is the incision and relocation of the frenum to create a zone of attached gingiva between the gingival margin and the frenum.

KNOW MORE ...

1996 World Workshop in Clinical Periodontics Renamed Mucogingival Surgery as Periodontal Plastic Surgery

The following problems are included:

1. Lack of sufficient vestibular depth.
2. Inadequate crown length for restorative procedures.
3. Denuded root surface.
4. Alveolar defects.
5. Open gingival embrasures.
6. Excessive gingival pigmentation.
7. Aberrant frenum.
8. Inadequate attached gingiva.
9. Impacted/unerupted teeth requiring orthodontic treatment.
10. Excessive gingival display and gingival asymmetry.

Gingival depigmentation → physiologic hyperpigmentation can be managed by mucosal excision, cryosurgery or laser surgery.

Surgical crown lengthening → ideally, crown lengthening procedure should endure ostectomy along with apically repositioned flap. Hence excising the gingiva alone to increase the crown length would be inadequate.

Interdental papilla reconstruction → the most common cause of "black triangles" between the teeth is loss of papilla height as a result of loss of periodontal support due to plaque-associated lesions.

Nordland and Tarnow (1998) proposed a classification system regarding papillary height based on three anatomical landmarks (Fig. 43.23):

a. The interdental contact point.
b. The apical extent of CEJ (facial cementoenamel junction).
c. The coronal extent of the proximal CEJ.

Normal: The interdental papilla occupies the entire space apical to the interdental contact point/area.
Class I: The tip of the interdental papilla is located between the interdental contact point and the level of the proximal CEJ.
Class II: The tip of the interdental papilla is located at or apical to the level of the proximal CEJ but coronal to the facial CEJ.
Class III: The tip of the interdental papilla is located at or apical to the level of facial CEJ.
Treatment of "Black Triangles" include:

- Orthodontic treatment.
- Modification of crowns.
- Esthetic restoration.
- Combination therapy.

Surgical Techniques for Reconstruction of Missing Papilla

Beagle (1992) proposed a pedicle graft procedure utilizing a split thickness flap which is dissected from the palatal aspect of the interdental area. The flap is elevated labially, folded and sutured to create the new facial papilla.

Han and Takei (1996) proposed a surgical approach semilunar coronally repositioned flap based on the use of a free connective tissue graft.

Many other techniques have also been proposed, some of them utilizes microsurgery but none of them have exhibited any predictable results.

REVIEW QUESTIONS

1. Enumerate various mucogingival problems.
2. Describe the techniques to increase width of attached gingiva.
3. Define gingival recession. Write about the etiology, classification and procedures for covering the denuded root surfaces.
4. Differences between frenectomy and frenotomy.

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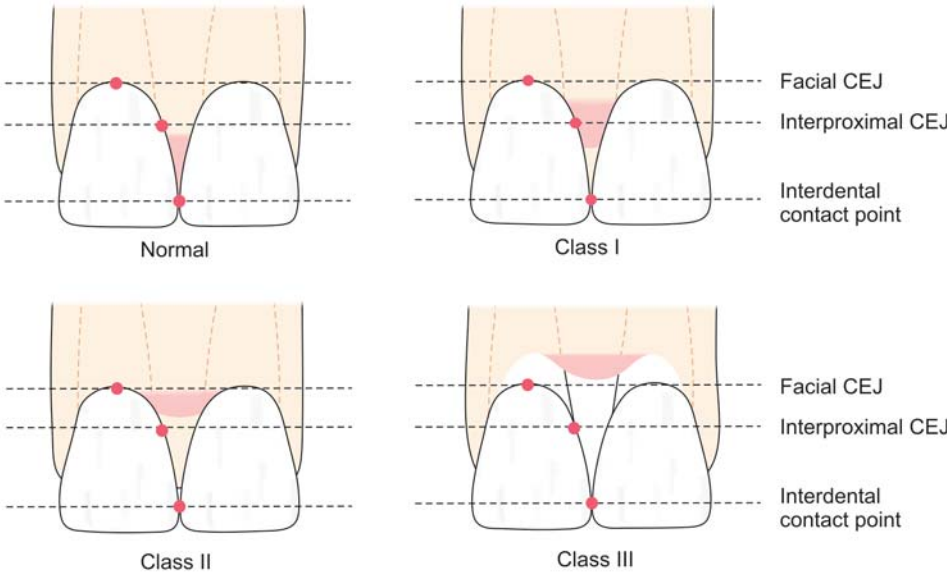

Fig. 43.23: Illustration of classification system for papilla height

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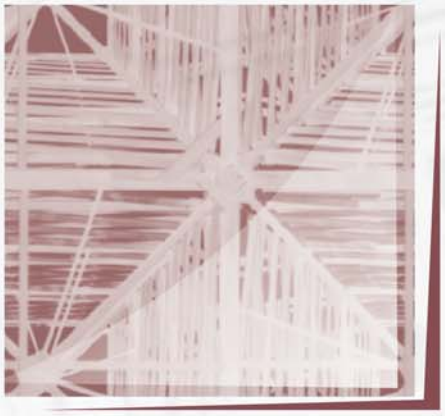


Part V

Treatment of Periodontal Diseases

44 Chapter

Furcation Involvement and its Management

- DEFINITION
- ETIOLOGY
- CLASSIFICATION
- CLINICAL FEATURES
- PROGNOSIS
- TREATMENT
- INDICATIONS AND CONTRAINDICATIONS ROOT RESECTION AND SEPARATION

DEFINITION

Furcation involvement refers to commonly occurring conditions in which the bifurcations and trifurcations of multi-rooted teeth are invaded by the disease process.

According to American Academy of Periodontology (2001), "It is defined as pathologic resorption of bone within the furcation".

ETIOLOGY

Primary Etiologic Factor

It is bacterial plaque and long-standing inflammation of periodontal tissues.

The extent of attachment loss in the furcation defect is related to:

Anatomic Considerations (Fig. 44.1)

1. *Root trunk length*—It is the portion of the root between cementoenamel junction and the separation of the roots (Fig. 44.2).
2. *Root separation*—It is the portion of the tooth where adjacent roots forming the furcation are not in contact with each other and are separated by alveolar bone.
3. The surface of the tooth just coronal to the root separation. This area is usually concave, grooved or fluted.
4. The roof of furcation which contains furcation ridges. These ridges run mesiodistally in lower and buccolingually in upper molars.

The roof of the furcation is concave in about 50 percent of maxillary first molars and is located 4.5 mm apical to the cementoenamel junction. The roof is 0.5 to 1 mm coronal to the root separation. Access to this area is extremely difficult for instrumentation of deep pockets.

Anatomic and clinical characteristics of *tooth, bone and gingiva* are of importance for the clinical management of furcation lesions.

Tooth

The following features should be considered:

a. *Root trunk length*—When the root trunk is short, the furcation will become involved early in the disease

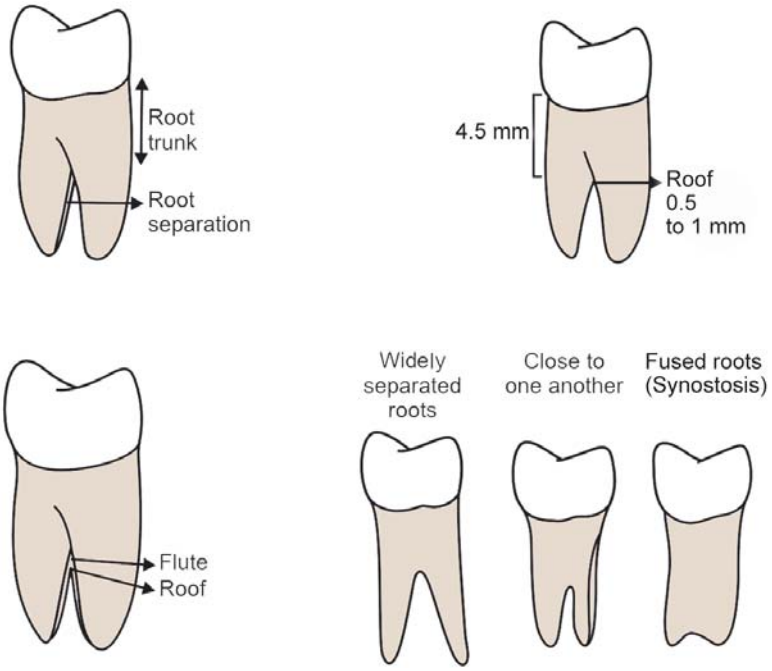


Fig. 44.1: Anatomic considerations

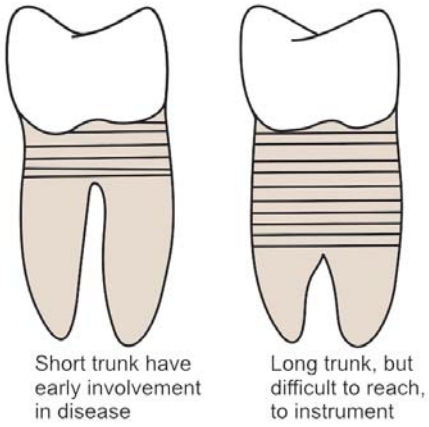


Fig. 44.2: Relationship between disease involvement and trunk length

process. When the root trunk is long the furcation will be invaded later, but will be more difficult for instrumentation.

b. *Concavity of the inner surface of exposed roots*—All the root surfaces facing the furcation exhibit some degree of concavity or depression in an occlusoapical direction. This may make instrumentation for plaque removal and root planing almost impossible. But these concavities increases the attachment area of a tooth and produce a root shape that is resistant to torque. It is common in mesiobuccal root of maxillary first molar and mesial root of mandibular first molar.

c. *Degree of separation of roots/inter-radicular dimension*—Wide separation of roots improves access, thereby facilitating instrumentation.

d. *Cervical enamel projections*—They occur approximately in 15 percent of molars. They favor plaque accumulation and must be removed to facilitate scaling and root planing.

It was classified by Masters and Hoskins in 1964 as:

Grade I: The enamel projection extends from the cementoenamel junction of the tooth toward the furcation entrance.

Grade II: The enamel projection approaches the entrance to the furcation but does not enter the furcation and hence has no horizontal component.

Grade III: The enamel projection extends horizontally into the furcation.

e. *Anatomy of the furcation*—Presence of bifurcation ridges, presence of accessory canals can complicate the furcation treatment.

f. *Presence of accessory pulpal canals*—It is believed that once the pulp is infected through the accessory canal,

endoperiocommunication may result, which in turn can cause either destruction of interradicular periodontium or interfere with the healing response of either periodontal or endodontic procedures.

Bone

Bone shape in the exposed furcation area has a horizontal component that determines the grades (I, II and III) of the involvement and a vertical component that most often creates a depression in the center of the remaining bone similar to a crater in an interdental area. The vertical component can also appear as a vertical or angular loss toward one of the roots. The latter defect can have one, two or three osseous walls or can be funnel-shaped around one root.

Gingiva

The presence of sufficient attached keratinized gingival tissue and adequate vestibular depth will facilitate the gingival management of the furcation area.

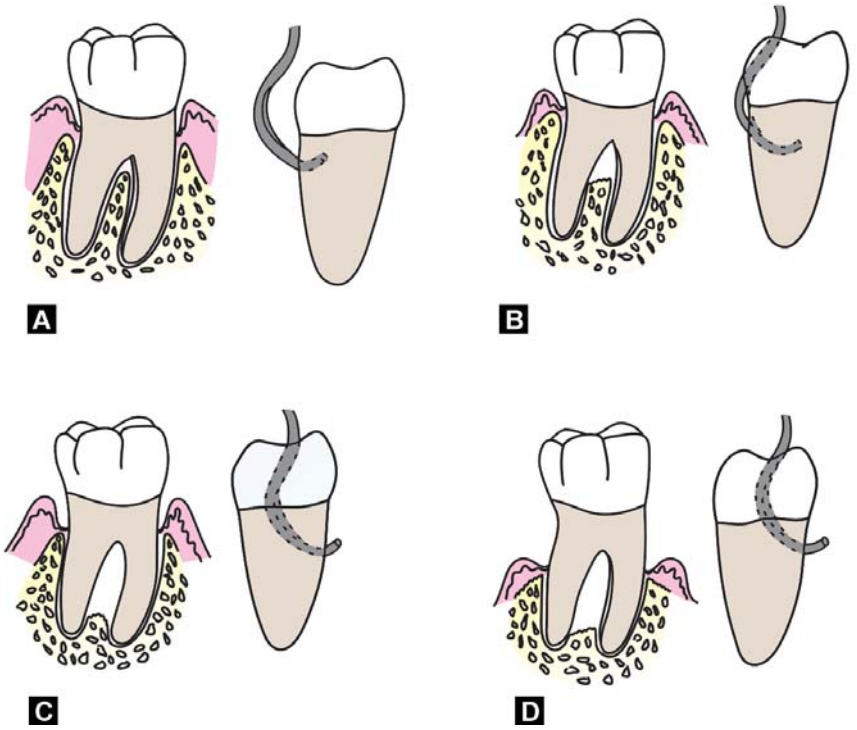
CLASSIFICATION

Glickman in 1953 (Figs 44.3A to D) had classified furcation involvement into:

Grade I: It is the incipient or early lesion. The pocket is suprabony, involving soft tissue. There is slight bone loss in the furcation area. No radiographic changes.

Grade II: In grade II cases bone is destroyed on one or more aspects of the furcation, but a portion of alveolar bone and periodontal ligament remains intact, permitting only partial penetration of the probe into the furcation. The lesion is essentially a cul-de-sac. The radiograph may or may not reveal the grade II involvement.

Grade III: In this type of furcation involvement, the interradicular bone is completely lost but the facial or lingual surfaces are occluded by gingival tissues. Therefore, the furcation opening cannot be seen clinically, but it is essentially a through and through tunnel. If the radiographs



Figs 44.3A to D: Glickman's classification of furcation involvement: (A) Grade I, (B) Grade II, (C) Grade III, (D) Grade IV

are taken with proper angulation and the roots are divergent, the lesion will appear as a radiolucent area between the roots.

Grade IV: As in grade III lesions, the inter-radicular bone is completely lost but in grade IV involvement the gingival tissues recede apically so that the furcation opening is seen clinically. The radiographic changes are essentially the same as that of grade III lesion.

Based on vertical component (Tarnow and Fletcher in 1984) (Fig. 44.4)

Depending on the distance from the base of the defect to the roof of the furcation, furcations can be classified as:

Subgroup A: Vertical destruction of bone upto one-third of the inter-radicular height (0-3 mm).

Subgroup B: Vertical destruction of bone upto two-third of the inter-radicular height (4-7 mm).

Subgroup C: Vertical destruction beyond the apical-third (7 mm or more).

Based on horizontal component (Hamp and Coworkers in 1975) (Fig. 44.5)

Furcations can be classified as:

Degree I : Horizontal bone loss of less than 3 mm.
Degree II : Horizontal bone loss of more than 3 mm.
Degree III : Through and through horizontal lesion.

Furcation can be clinically-detected by using *Naber's probe* along with a simultaneous blast of warm air to facilitate visualization and *radiographs* also help detect the furcation invasions.

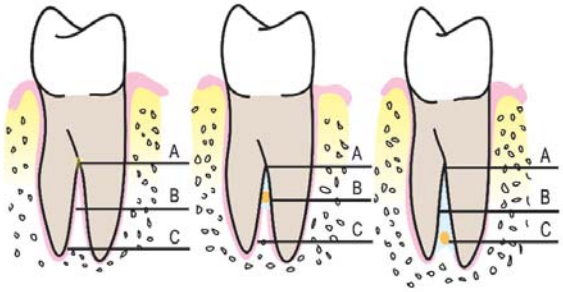
Other classifications include:

According to Goldman (1958):

Grade I : Incipient.
Grade II : Cul-de-sac.
Grade III : Through and through.

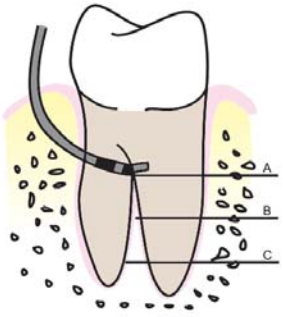
According to Ramfjord and Ash (1979):

Class I : Beginning involvement. Tissue destruction less than 2 mm (i.e less than 1/3rd tooth width)
Class II : Cul-de-sac. Tissue destruction more than 2 mm (i.e. more than 1/3rd tooth width) but not through and through.
Class III : Through and through involvement.

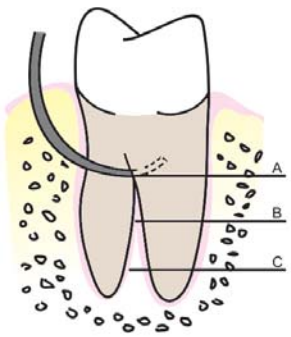


Subclass A : Vertical bone loss of 3 mm or less
Subclass B : Vertical bone loss of 4-6 mm
Subclass C : Vertical bone loss of 7 mm or more from the fornix

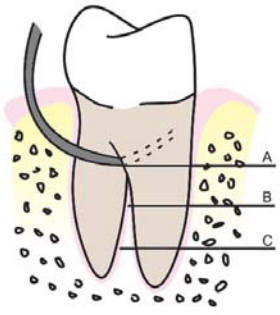
Fig. 44.4: Vertical classification of furcation involvement



Class I : Less than 3 mm of horizontal attachment loss



Class II : More than 3 mm of horizontal attachment loss. But not through and through. Furcation involvement



Class III : Through and through furcation involvement

Fig. 44.5: Horizontal classification of furcation involvement (Hamp and coworkers)

According to Fedi (1985):

Its a combination of Glickman's and Hamp's classification.

Grade I : It is the incipient or early lesion. The pocket is suprabony, involving soft tissue. There is slight bone loss in the furcation area. No radiographic changes.

Grade II : In grade II cases bone is destroyed on one or more aspects of the furcation, but a portion of alveolar bone and periodontal ligament remains intact, permitting only partial penetration of the probe into the furcation. The lesion is essentially a cul-de-sac. The radiograph may or may not reveal the grade II involvement.

Grade II : Can be further divided into:

Degree I : Interradicular bone loss less than 3 mm.

Degree II : Interradicular bone loss more than 3 mm.

Grade III and Grade IV are same as Glickman's classifications.

CLINICAL FEATURES

Clinically

1. The mandibular first molars are the most common sites and maxillary premolars are the least common.
2. The denuded furcation may be visible clinically or covered by the wall of the pocket.
3. Associated with suprabony and infrabony pockets.
4. Periodontal abscess.
5. Root caries and tooth mobility are common.

Microscopically

It is simply a phase in the rootward extension of the periodontal pocket. In its early stages, there is a widening of the periodontal space with cellular and inflammatory fluid exudation, followed by epithelial proliferation into the furcation area from an adjoining periodontal pocket. Extension of the inflammation into the bone leads to resorption and reduction in bone height. The bone destructive pattern may produce horizontal loss, or there may be angular osseous defects associated with infrabony pockets. Plaque, calculus and bacterial debris occupy the denuded furcation space.

PROGNOSIS

Maxillary first premolars and maxillary as well as mandibular molars are the teeth with multiple roots. Furcation involvement will be likely to occur in these multirooted teeth. Maxillary first premolar often shows fusion of the roots and the furcation area may be located very much apically and also the roots of the maxillary first premolars are placed buccally and palatally with furcation opening in a mesiodistal direction. For these reasons, furcation involvement in maxillary first premolar has poor prognosis.

In the case of maxillary molars furcations may open bucally, mesially and distally because of the presence of the three roots. Since access from proximal areas is difficult for plaque control, prognosis of furcation involvement in maxillary molars is not good.

Mandibular molars have two roots, placed mesially and distally and the furcation opens buccolingually. The roots are usually divergent especially in mandibular first molars. As a result prognosis of furcation involvement in mandibular molar (especially the first molar) is considered good.

TREATMENT

It is aimed to prevent further attachment loss and improve the maintenance of furcation area. Two treatment modalities have been proposed:

1. Traditional treatment procedures.
2. Reconstructive or regenerative treatment.

Factors to be considered when deciding on a mode of therapy are as follows:

1. Degree of involvement.
2. Crown root ratio.
3. Length of roots.
4. Degree of root separation.
5. Strategic value of the tooth or teeth in question.
6. Root anatomy of the involved tooth.
7. Residual tooth mobility.
8. Endodontic therapy and complications.

9. Ability to eliminate the defect.
10. Periodontal condition of the adjacent teeth.

Traditional Treatment Procedures

They are those which are directed to maintain the state of the health but do not attempt to regenerate the lost periodontium. The goal is to prevent the further progression of the disease and provide an environment which will help in adequate plaque control.

The procedures are:

Grade I: They are usually associated with suprabony pockets, hence,

a. Initial preparation or scaling and root planing.
b. Curettage or gingivectomy to expose the furcation area.
c. Odontoplasty—to reshape the facial groove in order to prevent plaque accumulation.

Grade II: In shallow grade II invasions,

a. Osteoplasty with limited ostectomy may be helpful.
b. Odontoplasty can be performed.

In severe grades II to IV invasions elimination of furcation by:

a. *Root resection or amputation:* After periodontal flap reflection, surgical removal of the root portion of the affected tooth is most commonly performed in maxillary first molars.
b. *Hemisection or root separation:* It is the surgical removal of the root along with the crown. Most commonly done in mandibular molars.
c. *Bicuspidization/root separation:* Splitting of a two-rooted tooth into two separate portions. Frequently performed in mandibular molars.
d. *Tunnel preparation:* It is by transforming the grade II lesion to grades III and IV for better access, but it is not performed anymore because of increased incidence of root caries.

Grade III and grade IV can be treated with root resection and root separation.

Reconstructive and Regenerative Treatment Procedures

Grade I: Traditional treatment will do.

Grade II: Various regenerative techniques include:

a. Autogenous bone grafting, e.g. osseous coagulum, bone blend.
b. Allografts, e.g. freeze, dried bone allografts, demine-ralized freeze-dried bone allografts (FDBA, DFDBA).
c. Alloplasts—hydroxyapatite, tricalcium phosphate.
d. Citric acid root conditioning with coronally positioned flap.
e. Guided tissue regeneration and combination techniques.

For grade III and grade IV furcation involvements the success rate is limited.

Definition of Root Resection, Hemisection and Root Separation (Figs 44.6 to 44.8)

(According to 1986 glossary of periodontal terms).

Root resection: It is the surgical removal of all or a portion of the root before or after endodontic treatment.

Hemisection: It is the surgical removal of a root with the associated part of the crown. It is frequently used with reference to lower molars.

Root separation/bicuspidization: It is the sectioning of the root complex and the maintenance of all roots.

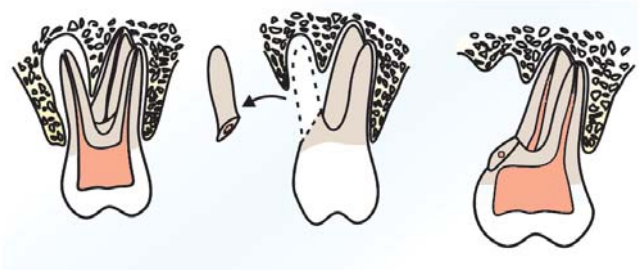


Fig. 44.6: Root separation/resection

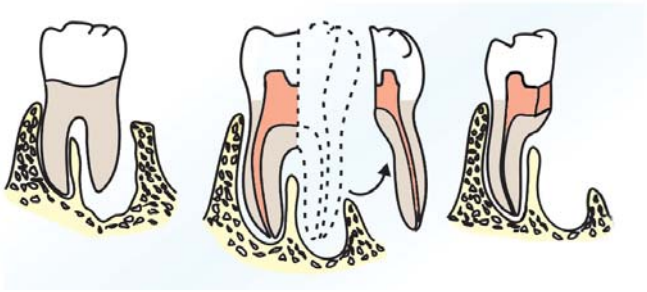
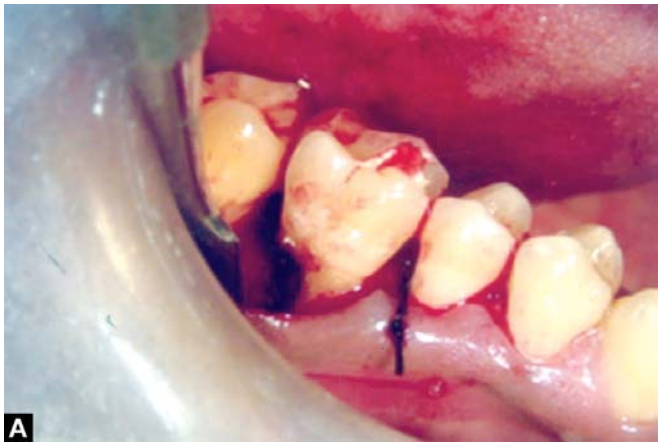
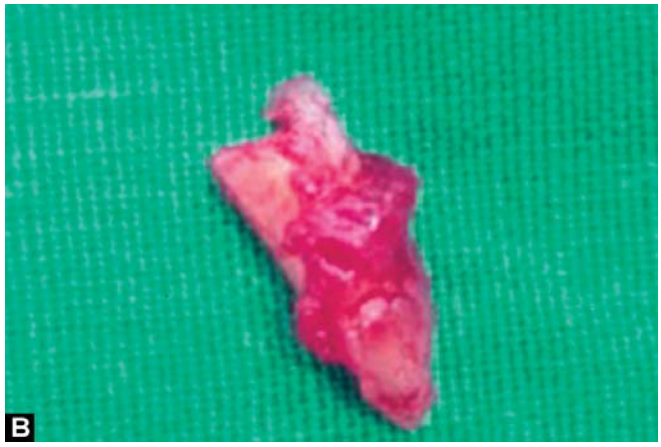


Fig. 44.7: Hemisection





Figs 44.8A and B: Root separation and resection

INDICATIONS AND CONTRAINDICATIONS FOR ROOT RESECTION AND SEPARATION

Indications

a. Severe bone loss affecting one or more roots untreatable with regenerative procedures.
b. Class II or III furcation invasions or involvement.
c. Severe recession or dehiscence of a root.

Contraindications

a. General contraindications like systemic diseases and poor oral hygiene.
b. Fused roots, unfavorable tissue architecture.
c. Roots that are endodontically-untreatable.

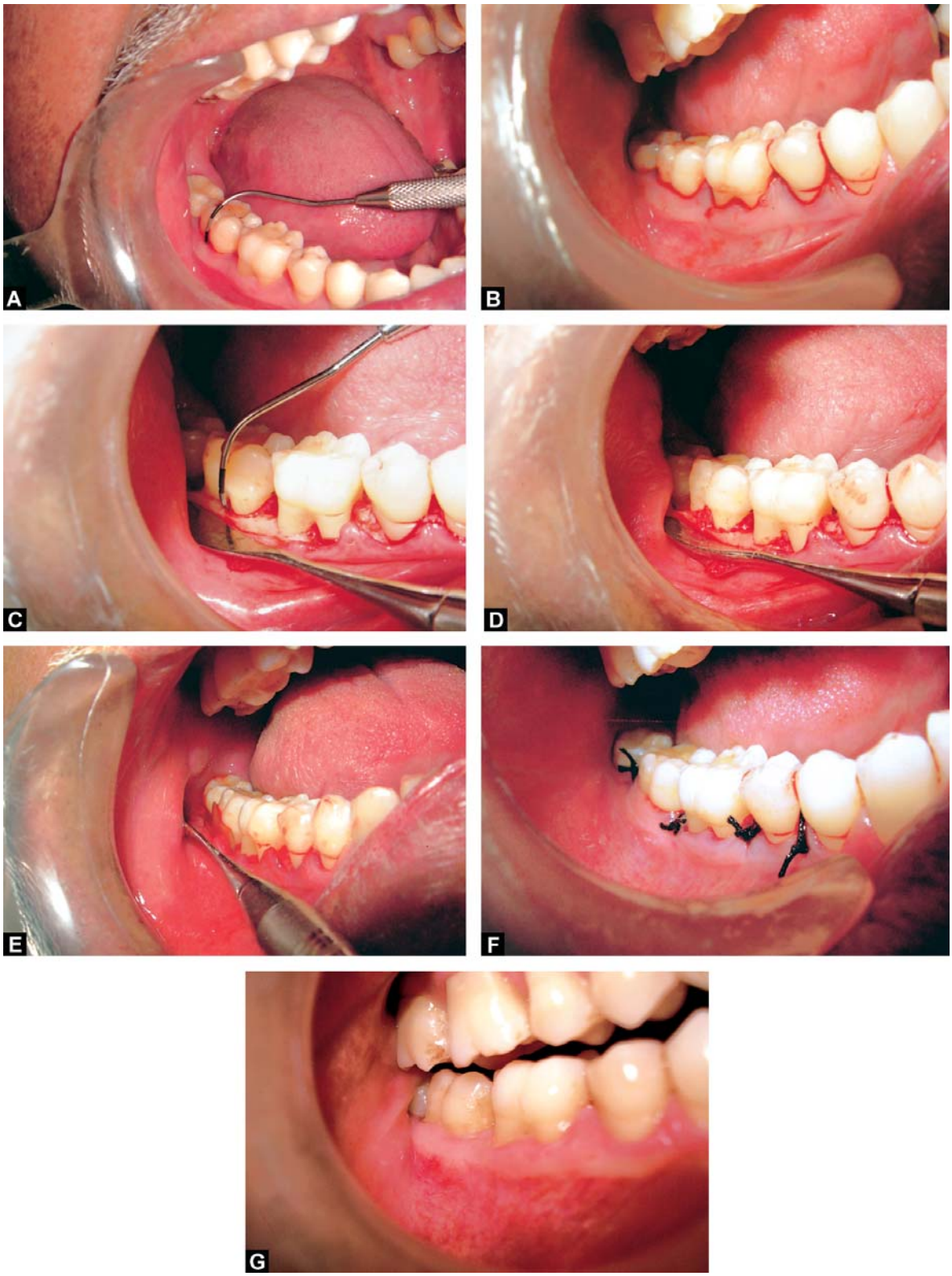
Three phases of treatment is suggested for root separation and resection.

a. *Endodontic phase*—If possible it should be performed prior to surgery. It offers the following advantages:
   - Better bone recontouring during surgery.
   - Allows precise flap closure.
   - Easy adaptation for temporary prosthesis.

b. *Restorative phase*—Construction of a provisional restoration.
c. *Surgical phase*—Most commonly distobuccal root of the maxillary first molar is resected.

The procedure is as follows: (Figs 44.9A to G)

1. After appropriate local anesthesia a full thickness mucoperiosteal flap is raised.
2. After debridement, resection of the root with advanced bone loss is carried out. First an oblique cut/incision is made directed from apical to the contact point through the tooth to the facial and distal orifices of the tooth. In case of vital resection, it is advised to perform a horizontal incision through the root because an oblique incision can expose a large surface area of the radicular pulp or pulp chamber which inturn can lead to more postoperative pain.



Figs 44.9A to G: Furcation involvement. (A) Preoperative, (B) Incision placement, (C) Reflection of flap, (D) Bone graft placement, (E) Membrane placement, (F) Suturing, (G) Facial view after healing

3. After sectioning, the root is elevated from its socket and is removed. If necessary odontoplasty is performed to remove any furcation deformities .If any bony defects are present in the adjacent teeth, then resective or regenerative procedures are performed.
4. The flaps are then approximated and sutured to maintain the position.

KEYPOINTS

1. Furcation involvement refers to commonly occurring conditions in which the bifurcation and trifurcation of multirooted teeth are invaded by the disease process.
2. Primary etiologic factor is bacterial plaque, predisposing or anatomic factors include, aberrant root morphology, cervical enamel projections or pearls and presence of accessory canals.
3. Furcation involvement can be classified according to Glickman as Grade I, Grade II, Grade III and Grade IV.
4. Depending on the distance from the base of the defect to the roof of the furcation (vertical component). Tarnow and Fletcher classified furcation involvement into— subgroup A, subgroup B, subgroup C.
5. Furcation involvement can be diagnosed by Naber's probe and radiographs.
6. Treatment of furcation involvement is divided into:
   a. Traditional treatment procedures.
   b. Reconstructive or regenerative treatment.

KNOW MORE ...

Furcation Anatomy

Terminology: (Root complex): It is the portion of a tooth that is located apical to the CEJ (cementoenamel junction).

The root complex may be divided into two parts:

- The root trunk.
- The root cones.

Root trunk: Represents the undivided region of the root, which is the distance between the CEJ and the separation line between the roots.

Root cone: Present within the divided region of the root complex. Two or more root cones make up the furcation region.

Furcation: It is the area located between individual root cones.

Furcation fornix: It is the roof of the furcation.

Divergence: It is the distance between two roots which normally increases in apical direction.

Coefficient of separation: The length of the root cones in relation to the length of the root complex.

Diagnosis of Furcation Defects

a. Clinical probing — using a curved graduated periodontal probe (Naber's probe), an explorer or a small curette, furcations can be identified.
   Maxillary molars — mesial furcation entrance is located closer to palatal than to the buccal surface, hence, the mesial furcation should be probed from the palatal aspect of the tooth. Whereas the distal furcation is in the midway between buccal and palatal surfaces, hence, this furcation can be probed either from the buccal or palatal surface. The distal maxillary furcation is most frequently involved.
b. Radiographs — can only be used to confirm the findings made during probing of a furcation in individual tooth.

REVIEW QUESTIONS

1. Define and classify furcation involvement. Describe the treatment for Grade II furcation involvement.
2. What are the indications and contraindications for root resection/separation?

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45 Chapter

Pulpoperiodontal Problems

- PATHWAYS OF COMMUNICATION BETWEEN PULP AND PERIODONTIUM
- EFFECTS OF PULPAL DISEASE ON PERIODONTIUM
- EFFECTS OF PERIODONTITIS ON THE DENTAL PULP
- ENDODONTIC-PERIODONTAL LESIONS
  - Classification
  - Microbiological Findings
  - Diagnosis and Treatments

INTRODUCTION

Normally, periodontitis is caused by extension of inflammation from the gingiva into deeper periodontal tissues. However, periodontitis can also be caused by pulpal infections that have entered the periodontal ligament either through the apical foramen or through the lateral canals. Such a periodontal lesion is termed as "*retrograde periodontitis.*" Similarly, "*retrograde pulpitis*" can also occur as a result of periodontal disease and periodontal treatment.

PATHWAYS OF COMMUNICATION BETWEEN PULP AND PERIODONTIUM (FIG. 45.1)

It can be classified into three categories:

1. *Pathways of developmental origin.*
   - Apical foramen
   - Accessory canals and lateral canals.
   - Developmental grooves.
   - Enamel projections and pearls at the cervical portion.
2. *Pathways of pathologic origin*
   - Tooth fracture (vertical).
   - Idiopathic resorption can be:
     a. *Internal*: From the pulp to the surface of the tooth.
     b. *External*: From the external surface of the root to the pulp.
     Both internal and external resorption produces communication.
   - Loss of cementum due to external irritants.
3. *Pathways of iatrogenic origin*
   - Exposure of dentinal tubules following root planing.
   - Accidental lateral perforation during endodontic procedure.
   - Root fracture due to endodontic procedure.

EFFECTS OF PULPAL DISEASE ON THE PERIODONTIUM

The three major causes of pulpal inflammation are:

1. Instrumentation during periodontal, restorative or prosthetic procedures.
2. Progression of dental caries.
3. Tooth fractures.

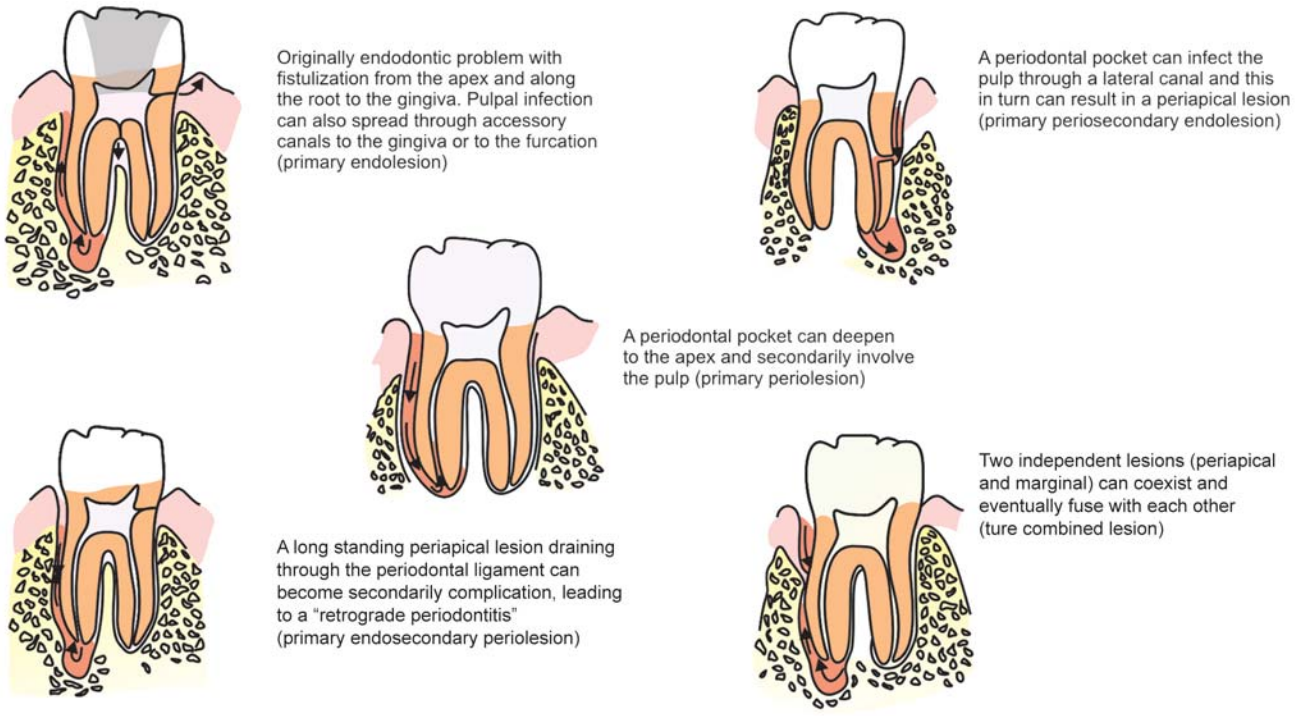

Fig. 45.1: Possible pathways for spread of infection between pulp and periodontal tissues

Of these dental caries is the most common cause of pulpal disease. As long as the pulp remains vital, the inflamed pulpal tissue is unlikely to cause any change in the periodontium. But acute inflammation of the pulp increases the intrapulpal pressure and the inflammatory fluid may be pushed out through the apical foramen or accessory canals. This results in periapical abscess which may drain through the periodontal ligament into the gingival sulcus and a periodontal pocket may form in due course of the time. Similar lesions may develop adjacent to accessory or lateral canals.

Accessory canals branch off and run parallel to the main root canal and are seen in the apical-third of the roots and furcation areas of multirooted teeth, whereas lateral canals run perpendicular to the root canal and are seen in greater number in the middle-third of the root. Passage of infection from the pulp into the furcation area through the accessory canals has been reported by many authors.

EFFECTS OF PERIODONTITIS ON THE DENTAL PULP

Bender and Seltzer (1972) in their studies have reported that teeth with caries or restorations also suffering from periodontal disease have more atrophic pulps than teeth with caries or restorations, but no periodontal disease. The cause of these atrophic changes (which is observed radiographically as narrowed canal space) is the disruption of blood flow through the lateral canals, which leads to localized areas of coagulation necrosis in the pulp. Subgingival scaling and root planing may also produce changes in the pulp, one possible explanation for this is that blood vessels leading into lateral canals are severed causing localized areas of pulpal necrosis (Fig. 45.2).

ENDOPERIOLESIONS

Classification

Pulpoperiodontal lesions have been classified by many authors based on different criteria. Classification proposed by Simon et al (1972) is most commonly employed.

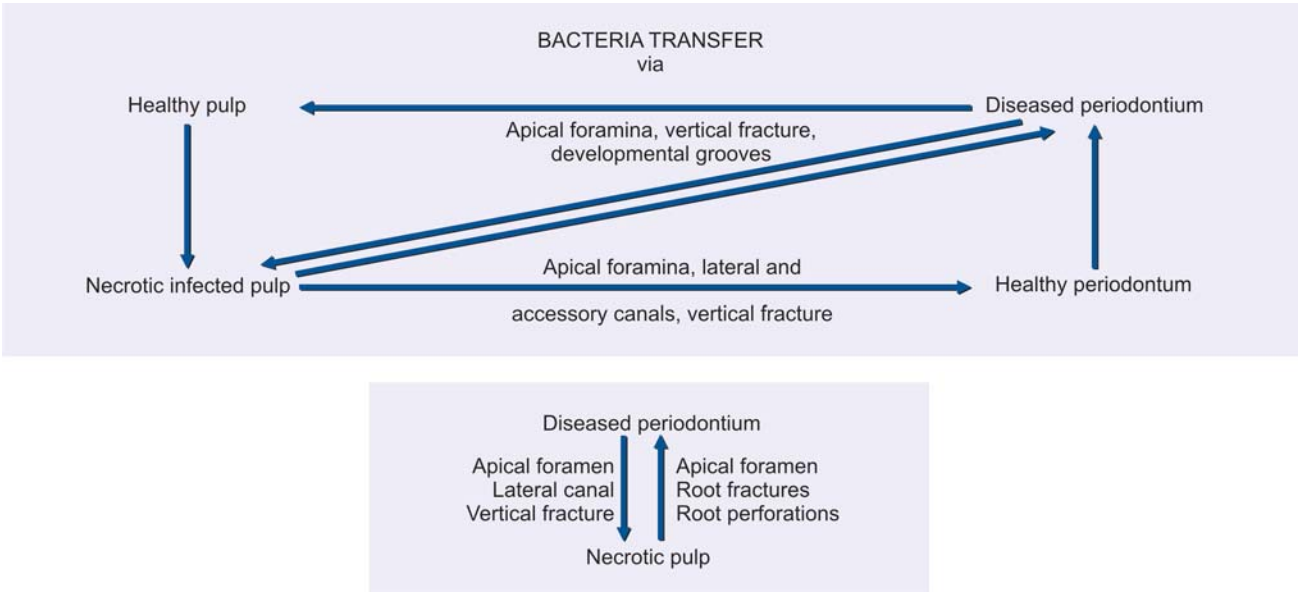

Fig. 45.2: Schematic diagram showing possible pathways for spread of infection between pulp and periodontal tissue

Based on the etiology, diagnosis, prognosis and treatment, endoperiolesions can be classified into five groups (Table 45.1).

1. Primary endodontic lesion.
2. Primary endodontic with secondary periodontal lesion (Fig. 45.3).
3. Primary periodontal lesion.
4. Primary periodontal lesion with secondary endodontic involvement.
5. True combined lesions.

Microbiological Findings

Bacteria play an important role in the pathogenesis of both pulpal and periodontal disease. There seems to be significant similarity between the microbiological findings of root canals and pockets with advanced periodontitis. *B. forsythus*, *P. gingivalis* and *T. denticola*, *Fusobacteria*, *Spirochetes*, *Wolinella* and *Peptostreptococcus* have been found in endo-perio lesions.

Diagnosis and Treatment

When one suspects the possible endodontic periodontal lesion, the first task is to assess endodontic status of the tooth in question. Traditional diagnostic aids including, radiographic analysis with gutta percha tracing, periodontal probing, fiberoptic illumination to rule out whether a fracture exists, more importantly vitality test, percussion tests should be carried out. Numerous studies have demonstrated the inaccuracy of vitality testing. Probably because pulp testing only indicates the neural response and gives little information about the vascularity or true vitality of the pulp. Research is currently under way to improve diagnostic testing through the use of Doppler devices, pulse oximetry and even magnetic resonance imaging.

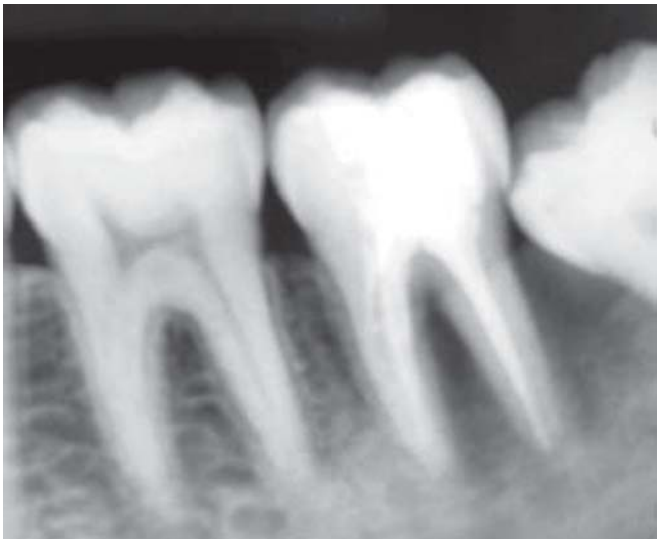

Fig. 45.3: Radiograph of endoperiolession

Table 45.1: Classification of endoperiolesions

Type of lesion	*Sequelae*	*Diagnosis*	*Treatment*	*Prognosis*
Primary endodontic lesion	It is characterized by necrotic pulp with a chronic apical periodontitis and a draining sinus tract through the periodontal ligament and gingival sulcus. There may not be any periodontal involvement in the other areas. The sinus tract can be probed, with gutta percha and silver points. Radiographically, no crestal bone loss.	Pulp vitality tests are negative.	Conventional root canal therapy should be performed with multiple appointments. No root planing should be done when the sinus tract is along the periodontal ligament, because these fibers are important for re-attachment to occur.	It is excellent. Healing is usually complete within 3 to 6 months.
Primary endodontic lesion with secondary periodontal involvement	If primary endodontic lesion is not diagnosed and treated early, secondary periodontal problem may result due to accumulation of plaque and calculus in the drianage fistulous tract. Radiographically, bone loss may be evident.	Pulp vitality tests are negative. Periodontal probing will reveal deep periodontal pocket in this area.	First endodontic therapy including root canal therapy. Periodontal therapy in the form of root planing.	Endodontic component is excellent but regeneration of attachment apparatus is limited by the periodontal prognosis.
Primary periodontal lesion	Periodontal pocket which extends upto the apex of the tooth. Spread of infection from the pocket into the pulp may occur in these cases. In the pulp fibrosis and calcification is noticed but not necrosis. Minimal/no pain is experienced by the patient.	Pulp is vital, periodontal probing, may reach the apex of the involved teeth. The periodontal problems are seen on the other teeth also.	Periodontal therapy to eliminate the pocket is indicated. Root canal therapy is not usually indicated unless pulp vitality test results change. Periodic reevalution is necessary.	It is entirely dependent on the periodontal therapy.
Primary periodontal lesion with endodontic involvement	Primary periodontal lesion may involve necrosis and patient may experience severe pain. Pulpal necrosis could be as a result of periodontal therapy where the blood vessels to the pulp are severed during periodontal instrumentation.	Similar to those used in primary endodontic and secondary periodontal lesions. Generalized perioodontal problem. Pulp vitality test results can be mixed.	Both endodontic and periodontal therapy.	It is dependent on the periodontal therapy, healing response of periapical lesion is not predictable.
True combined lesion	These are those lesions that are formed when pulpal and periodontal pathoses develop independently and unite. These lesions are usually of periodontal origin. Teeth with vertical root fractures also belong to this category.	Pulp testing is negative. The tooth in question will have probing depths upto the apex of the tooth.	Both endodontic and periodontal therapy including root resection and hemisection is proposed.	Questionable prognosis

Treatment

Some controversy seems to exist, as to whether endodontic therapy or periodontal therapy has to be performed first, in the management of endoperiolesions. Considering many facts it was advised to perform endodontic therapy prior to periodontal therapy.

KEYPOINTS

1. When inflammation from pulp extends into the periodontium either through the apical foramen or through the lateral canals destruction of periodontal tissues may occur. This is termed as 'retrograde periodontitis'.
2. Pathways of communication between pulp and periodontium could be of :
   - Developmental origin.
   - Pathologic origin
   - Iatrogenic origin.
3. Endoperiolesions are classified into:
   - Primary endodontic lesions
   - Primary endodontic with secondary periodontal lesions
   - Primary periodontal lesions
   - Primary periodontal lesion with secondary endodontic involvement.
   - True combined lesions.

KNOW MORE ...

Other Classifications of Endodontic-Periodontal Lesions

- According to Oliet and Pallock (1968): Based on treatment procedure:
  1. Lesion that require endodontic treatment procedures only.
  2. Lesion that require periodontal treatment procedures only.
  3. Lesions that require combined endodontic-periodontic treatment procedures.
- According to Weine (1972):
  1. Class I: Tooth in which symptoms clinically and radiographically simulate periodontal disease but are infact due to pulpal inflammation and/or necrosis.
  2. Class II: Tooth that has both pulpal or periapical disease and periodontal disease concomitantly.
  3. Class III: Tooth that has no pulpal problem but requires endodontic therapy plus root amputation to gain periodontal healing.
  4. Class IV: Tooth that clinically and radiographically simulates pulpal or periapical disease but infact has periodontal disease.

REVIEW QUESTIONS

1. Enumerate various pulpoperiodontal problems.
2. What is retrograde periodontitis?
3. What are the effects of periodontitis on the dental pulps?

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46 Chapter

Splints in Periodontal Therapy

- DEFINITION
- OBJECTIVES
- CLASSIFICATION AND TYPES
- PRINCIPLES
- INDICATIONS AND CONTRAINDICATIONS
- ADVANTAGES AND DISADVANTAGES

DEFINITION

Dental splinting: It is defined as the joining of two or more teeth into a rigid unit by means of a fixed or removable restorations/devices. Splint by definition is an appliance used for immobilization of injured or diseased parts.

A periodontal splint: It is an appliance used for maintaining or stabilizing mobile teeth in their functional position.

The main objective of splinting is to promote healing and to increase the patients comfort and function. There are two schools of thought regarding the use of splinting.

Harmful Aspects

- It creates an environment for plaque accumulation.
- Since functional movement of the tooth within the socket is not possible it may lead to ankylosis.

Beneficial Aspects

- Since they are splinted to the neighboring healthy teeth, mobility during mastication is prevented.
- Non-mobile teeth heal faster than mobile teeth.

OBJECTIVES OF SPLINTING

1. Provides rest.
2. For redirection of forces—the forces of occlusion are redirected in a more axial direction over all the teeth included in the splint.
3. For redistribution of forces—redistribution ensures that forces do not exceed the adaptive capacity of the periodontium.
4. To preserve arch integrity—splinting restores proximal contacts, reducing food impaction and consequent breakdown of the periodontium.
5. Restoration of functional stability—restores a functional occlusion, stabilizes mobile abutment teeth and increases masticatory efficiency.
6. Psychologic well-being—gives the patient freedom from mobile teeth thereby giving him a sense of wellbeing.
7. To stabilize mobile teeth during surgical, especially regenerative therapy.
8. To prevent the eruption of teeth without an antagonist.

CLASSIFICATION OF SPLINTS

According to the Period of Stabilization

a. *Temporary stabilization*—to be worn for less than 6 months, e.g. removable/fixed
b. *Provisional stabilization*—to be worn for months or several years, e.g. acrylic splints, metal bands.
c. *Permanent splints*—used indefinitely, e.g. removal/fixed, intracoronal/extracoronal.

According to the Type of Material

i. Bonded, composite resin button splint.
ii. Braided wire splint.
iii. A-splints.

According to the Location on the Tooth

Intracoronal

- Composite resin with wire
- Inlays
- Nylon wire

Extracoronal

- Tooth-bonded plastic
- Night guard
- Welded-bands

VARIOUS COMMONLY USED SPLINTS

1. **Splints for Anterior Teeth**
   a. *Direct bonding system* using acid etch techniques and a light cured resin.
   b. *Intracoronal wire and acrylic wire resin splint*—It involves the teeth with stainless steel wire placed in the slots thus stabilizing the teeth.
2. **Splints for Posterior Teeth**
   a. Intracoronal amalgam wire splints—It uses resin restoration with wire on the proximal amalgam restored areas of the tooth.
   b. Bite-guard.
   c. Rigid occlusal splint.
   d. Composite splint.

PRINCIPLES OF SPLINTING

1. *Inclusion of sufficient number of healthy teeth:* It is suggested that the healthy teeth included in the splint should have double the root surface area of the mobile teeth to be splinted. Since the posterior teeth are multi-rooted the number of healthy teeth to be included in the splint in the posterior segment will be less as compared to the anterior.
2. *Splint around the arch*: Muscles of the lips, cheek and tongue exert some forces on the teeth. Based on the direction of such forces applied on the teeth, the dental arch can be divided into two posterior sextants and an anterior sextant. In the posterior sextant the tongue pushes the teeth buccally and the muscles of the cheek counter act it by pushing them lingually. When the splint is confined to any one sextant, the splinted teeth tend to tilt lingually or outwards depending on the muscular forces. Such a collapse of the splinted sextant can be prevented by including few teeth from the adjacent sextant. This is termed *splinting around the arch*.
3. *Coronoplasty may be performed* to relieve traumatic occlusion.
4. The splint should be fabricated in such a way as to facilitate *proper plaque control*.
5. Splint should be *aesthetically-acceptable* and should not interfere with occlusion.

INDICATIONS AND CONTRAINDICATIONS OF SPLINTING

Indications

1. It stabilizes moderate to advanced tooth mobility that can not be reduced by other means and which has not responded to occlusal adjustment and periodontal therapy.
2. When it interferes with normal masticatory function.
3. Facilitates scaling and surgical procedures.
4. Stabilizes teeth after orthodontic movement.
5. Stabilizes teeth after acute dental trauma, e.g. subluxation, avulsion, etc.

6. In order to prevent tipping and drifting of teeth.
7. Prevent extrusion of unopposed teeth.

Contraindications

1. Moderate to severe tooth mobility in the presence of periodontal inflammation and/ primary occlusal trauma.
2. Insufficient number of firm/sufficiently firm teeth to stabilize mobile teeth.
3. Prior occlusal adjustment has not been done on teeth with occlusal trauma or occlusal interference.
4. Patient not maintaining oral hygiene.

ADVANTAGES

1. May establish final stability and comfort for patient with occlusal trauma.
2. Helpful to decrease mobility and accelerate healing following acute trauma to the teeth.
3. Allows remodelling of alveolar bone and periodontal ligament for orthodontically, splinted teeth.
4. Helpful in decreasing mobility thereby favoring regenerative therapy.
5. Distributes occlusal forces over a wider area.

DISADVANTAGES

1. All the splints hamper patient's self care. Accumulation of plaque at the splinted margins can lead to further periodontal breakdown in a patient with already compromised periodontal support.
2. Number of studies have shown that splinting does not actually reduce tooth mobility (once the splint is removed).
3. The splint being rigid acts as a lever with uneven distribution of forces, even if one tooth of the splint is in traumatic occlusion, it can injure the periodontium of all the teeth within the splint.
4. Development of caries is an unavoidable risk. Thus, it obviates the need of excellent oral hygiene in the patient.

In conclusion splinting decreases mobility thereby improving the health of the tooth. It is important to note that if used incorrectly or not managed properly it may fail to achieve the desired results.

KNOW MORE ...

Characteristics of an Ideal Splint

- Should be easily available.
- Economical and stable.
- Easily maintainable.
- Rigid and durable.
- Compatible with the adjacent tissues.

REVIEW QUESTIONS

1. Define and classify periodontal splints.
2. What are the advantages and disadvantages of periodontal splints?

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47 Chapter

Dental Implants: Periodontal Considerations

- TERMINOLOGY
- HISTORICAL BACKGROUND
- BIOLOGICAL CONSIDERATIONS
  - Soft Tissue Implant Interface
  - Bone Implant Interface
- BIOMATERIALS USED FOR IMPLANTS
- CLASSIFICATION OF IMPLANTS
- CLASSIFICATION OF IMPLANT SYSTEMS
- TREATMENT PLANNING
- HEALING FOLLOWING IMPLANT SURGERY
- PERI-IMPLANT COMPLICATIONS AND DISEASES

INTRODUCTION

The concept of replacing missing teeth for esthetics and function has been an elusive goal for more than 1500 years. This has led to the evolution of many materials and techniques including complete dentures, removable and fixed partial dentures. To overcome the limitations of these materials and techniques, dentistry has long sought a superior method of artificial tooth replacement through dental implants with a goal of restoring the normal contour, comfort, esthetics, health and the most traditional dental disciplines, which include the bone and soft tissue reconstruction.

TERMINOLOGY

Dental implant is an integral component of the oral implant complex, which also consists of supportive bone, interposed keratinized and mucosal oral soft tissues and prosthetic suprastructure.

A dental implant is a permucosal device that is biocompatible and biofunctional and is placed on or within the bone associated with the oral cavity to provide support for fixed or removable prosthesis.

Oral implantology is the science and discipline concerned with the diagnosis, design, insertion, restoration and for management of alloplastic or autogenous oral structures to restore the loss of contour, comfort, function, esthetics, speech and/or health of the partially or completely edentulous patient.

Implant surgery is that part of reconstructive surgery that is concerned with the placement of endosseous, subperiosteal and transosseous implants for the restoration and maintenance of mastication and speech. Such surgery

may also eliminate chronic pain related to nerve dehiscence, preserving remaining bone structure and prevent the possibility of a pathologic fracture.

Osseointegration: Direct structural and functional connection between ordered living bone and the surface of the load carrying implant.

HISTORICAL BACKGROUND

The need to replace missing teeth has haunted for time immemorial.

According to Marshall (1987) there are five distinct eras of implant dentistry.

The Ancient Era (Before 1000 AD)

The discovery of 2,000 years old cranium with a cast iron dental implant in France demonstrated that dental implantology was one of the oldest existing forms of dentistry. Ancient Egyptians attempted intraosseous transplantation of animal teeth. A fine dark flint stone-shaped like a tooth implanted in a Mayan Skull in Central America during back to 600 AD.

The Medieval Era (1000 to 1799 AD)

The medieval period of implant dentistry was primarily concerned with transplantation of teeth with implants made up of ivory, shells and bone or transplantation of teeth from one human to other or removing the teeth from the impoverished people and transplanting them into the mouths of the affluent.

The Foundation Era (1800 to 1910 AD)

During this period newer materials and methods were developed like fabricating and inserting gold roots into freshly extracted socket which were soldered together, use of gold plate in the treatment of cleft palate, using teeth made up of porcelain which had lead coated platinum post fixed as an implant. Payne (1898) gave the first clinical demonstration on implants.

The Modern Era (1910 to 1978 AD)

Implant designs were carried out with the aim of finding a superior metal which was biocompatible to the periodontal structures. By developing screw type implants using vitallium, subperiosteal implants and extending their framework to external oblique region and introduction of the use of an endosseous implant with a central post and circumferential extensions, a step was taken in that direction.

The Contemporary Era (1978 Till Date)

Implants developed during this era are core-vent implant, screw-vent implant, swede-vent implant, a hydroxyapatite-coated bio-vent implant; microvent implant, starvent implant; sterioss, root form endoosseous implant, step root form design.

Constant research is being conducted in the development of newer implant systems with the aim of developing a perfect implant which will function well without any failure.

BIOLOGICAL CONSIDERATIONS OF IMPLANTS

I. Soft tissue implant interface.
II. Bone implant interface.

Soft Tissue Implant Interface

The mucosal tissues around intraosseous implants form a tightly-adherent band consisting of a dense collagenous lamina propria covered by keratinized stratified squamous epithelia.

The implant epithelium junction is analogous to the junctional epithelium around natural teeth; in that, the epithelial cells attach to the titanium implant by means of hemidesmosomes and basal lamina.

This evidence supports the concept that a viable biologic seal can exist between the epithelial cells and the implants.

A sulcus forms around the implant lined with a sulcular epithelium that is continuous apically with the junctional epithelium.

Collagen fibers are nonattached and run parallel to the implant surface, owing to the lack of cementum. Since endosseous implants are permucosal, the soft tissue-implant interface should be considered in their placement and maintenance. This suggests that epithelium adheres to implant surfaces and has similar biological features of the epithelium tooth interface.

Bone Implant Interface

The relationship between endosseous implants and bone involves mechanisms like:

- Fibro-osseous integration
- Osseointegration and
- Bioactive integration.

Fibro-osseous Integration

It is defined as "tissue to implant contact by interposition of a healthy dense collagenous tissue between the implant and the bone interface". Normally, fibro-osseous union between the implant surface and adjoining alveolar bone is not desirable because union formed is a weak union. The formation of fibro-osseous integration is attributed to proliferation of connective tissue into the interface, which hampers the osseous integration process.

Osseointegration

It is defined as a direct structural and functional connection between ordered living bone and the surface of the load carrying implant.

Bioactive Integration

It is defined as the integration which results by a physiochemical interaction between collagen of bone and hydroxyapatite crystals of the implants.

BIOMATERIALS USED FOR IMPLANTS

Metals and Alloys

a.	Titanium	100 percent pure Titanium
b.	Titanium-Aluminum Vanadium	Titanium 90 percent Aluminum 6 percent, Vanadium 4 percent
c.	Cobalt-Chromium	Cobalt 66 percent + Chromium 27 percent + Molybdenum 7 percent
d.	Stainless steel	Iron 70 percent + Chromium 18 percent + Nickel 12 percent
e.	Tantalum	100 percent pure
f.	Zirconium	100 percent pure
g.	Gold	100 percent pure
h.	Platinum	100 percent pure

Inert Ceramics

a. Aluminum oxide (Al_2O_3)
   - Polycrystalline
   - Single crystal.

b. Zirconium oxide zircona.
c. Titanium oxide.

Calcium Phosphate Ceramics

Calcium phosphate.

Bioactive and Biodegradable Ceramics

a. Hydroxyapatite.
b. Tricalcium phosphate.
c. Bioglass.
d. Ceramic.
e. Calcium aluminates.
f. Carbon.
g. Carbon silicon.
h. Polycrystalline glassy carbon.

Polymers

a. Polymethyl methacrylate.
b. Polytetrafluoroethylene.
c. Polyethylene.
d. Polyethylene tetraphthalate.
e. Polypropylene.
f. Polyoxymethylene.

g. Silicone rubber.
h. Polysulfone.

CLASSIFICATION OF IMPLANTS

Implants are classified based on:
1. Shape and form and
2. Surface characteristics.

Based on the Shape and Form

1. Endosteal.
2. Subperiosteal.
3. Transosteal.
4. Intramucosal inserts/submucosal implants/subdermal implants.
5. Endodontic stabilizer.

With regard to shape, it is possible to distinguish between:
a. Post or root form implants—Exhibiting rotation symmetry.
b. Blade implants—Extension implants.

The post or root implant designs can be of the following types:
1. Solid tapering types.
2. Solid cylinder type.
3. Pin type.
4. Screw-shaped implant type.
5. Basket design.
6. Hollow cylinder design.

The blade implant designs can be of following types:
1. Conventional blade design.
2. Vented blade design.

Based on Surface Characteristics

1. Titanium plasma—sprayed coating.
2. Sand blasting—surface etching.
3. Laser induced surface roughening.
4. Hydroxyapatite coating.

CLASSIFICATION OF IMPLANT SYSTEMS

1. Branemark implant system (Nobel Biocare System).
2. International team for implantology (ITI) system.
3. Implant innovations systems.
4. Astra-dental implant system.
5. IMZ implant system (Interpore IMZ).
6. Corevent system.
7. Sterioss system.
8. Stryker implant system.
9. Endosteal hollow basket system.

TREATMENT PLANNING

Clinical Assessment

Selection of cases for implants is based on the:
I. Age limitations for case selection.
II. Anatomic prerequisites:
   1. Resorptive process.
   2. Soft tissue situation.
   3. Available bone.
   4. Mandibular canal.
   5. Height of bone.
   6. Width of bone.
   7. Bone shape (contour).
   8. Length of bone.
   9. Implant crown relationship.
   10. Maxillary sinuses.

The Absolute Requirements for Treating Implant Patients

1. Have an acceptable patient.
2. Implant made of biocompatible material.
3. Be durable.
4. Have proper surface quality.
5. Have acceptable socket created in bone.
6. Have surgical procedure properly done.
7. Have healing completed with acceptable bone interface.
8. Have healing period without pathological stress.
9. Have normal implant function without pathological stress.

Indications for Implant Therapy

A. *The edentulous patient:*
   - Edentulous mandible
   - Edentulous maxilla.

B. *The partially-edentulous patient:*
   - Free end edentulous situation
   - Multiple missing teeth.

C. *Single tooth loss.*

Absolute Contraindications for Implant Treatment

1. Uncontrolled-diabetes mellitus.
2. Long-term immunosuppressant drug therapy.
3. Diseases of connective tissue.
4. Blood dyscrasias and coagulopathies.
5. Regional malignancy.
6. Metastatic disease.
7. Previous radiation to the jaws that might lead to postsurgical osteoradionecrosis.
8. Alcohol or drug addiction.
9. Severe psychologic disorders.

Intraoral Contraindications

This includes:

1. Unfavorable interarch relationships.
2. Problematic occlusal and functional relationships.
3. Pathologic considerations in alveolar bone, example, fibro-osseous disease.
4. Pathologic alteration of the oral mucosa, example, cysts, infections.
5. Xerostomia.
6. Macroglossia.
7. Unrestored teeth—poor oral hygiene.

Radiographs

Radiographs used in dental implants are panoramic radiographs. However, this technique has certain inherent problems that have to be taken into consideration like distortion of spatial relationships. In order to eliminate the distortion problems panoramic radiographs and their use of templates with incorporated metal spheres have been demonstrated.

Other Radiographic Procedures Employed

These are:

1. Periapical dental radiographs.
2. Rast-O-Pan bite blocks.
3. Lateral cephalometric radiograph.
4. Occlusal radiograph.
5. Tomography
6. Computed tomography

Surgical Procedures

Most threaded endosseous implants can be placed either in one stage (or) two stages.

One-stage: Endosseous Implant Surgery

In this procedure the coronal portion stays exposed through gingiva during the healing period. For example, ITI system, TG Implant of 3i system and Life core single– stage system.

One stage endosseous implant surgery: In this implant surgery, the implant (or) healing abutment protrudes about 2 to 3 mm from the bone crest and the flaps are adapted around the implant. In posterior areas of the mouth the flap is thinned and sometimes placed apically to increase the zone of keratinized attached gingiva.

Surgical Technique (Figs 47.1A to F)

Flap design and incisons: The flap design is always a crestal incision bisecting the existing keratinized tissue. The soft tissue is not thinned in anterior or other esthetic areas of the mouth to prevent the metal collar from showing, full-thickness flaps are elevated buccally and lingually.

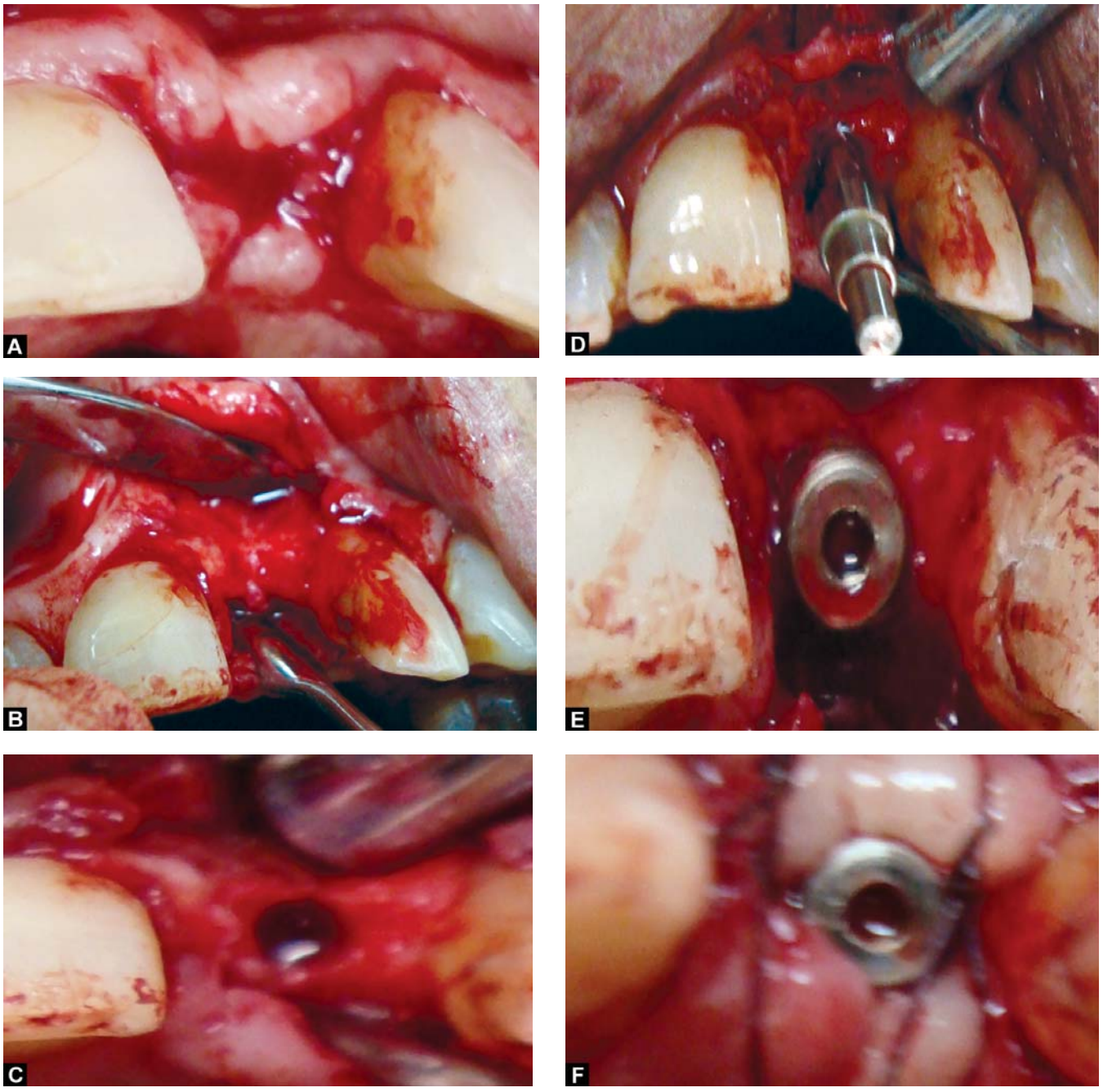
Placement of the implant: The implant site preparation to place implants in one stage surgery is identical to principles of two-stage except, implants or healing abutment is placed in such a way that head of implant protrudes about 2 to 3 mm from the bone crest.

Closure of the flap: The keratinized edges of the flap are tied with independent sutures around the implant, when keratinized tissue is abundant, scalloping around the implant provides better flap adaptation.

Advantages and disadvantages of one stage implant surgery.

Advantages:

a. Mucogingival management around the implant is easier.
b. Patient comfort increases because less surgeries are involved.
c. Esthetic management is easier in many cases.



Figs 47.1(A to F): Surgical steps in implant placement. (A) Initial incision placed (B) Reflection of flap (C) Osteotomy procedure at implant site (D) Checking for parallelism of implant (E) Placement of implant (F) Repositioning of flap and placement of sutures

Disadvantage:

If extensive bone loss occurs at the implant site. Vertical bone augmentation is necessary, and or bone quality is poor then two-stage surgical approach is recommended.

Two-stage: Endosseous Implant Surgery

In the two-stage implant surgical approach, the first stage ends by suturing the soft tissues over the implant so that it remains excluded from the oral cavity.

In the mandible, the implants are left undisturbed for 2 to 3 months, whereas in the maxilla, they remain covered for approximately 4 to 6 months because of slower healing due to less dense bone. During this period, the healing bone makes direct contact with the implant surface (osseointegration) and sometimes grows to its occlusal surface, even covering it.

In second-stage surgery, the buried implant is uncovered and a titanium abutment is connected to allow access to the implant from the oral cavity. The restorative dentist then proceeds with the prosthodontic aspects of the implant therapy.

HEALING FOLLOWING IMPLANT SURGERY

If the space between an implant and its osseous bed is narrow, bone formation is comparable to primary healing after a bone fracture, because no callus is formed. Direct bridging via lamellar bone occurs, at a rate of about 1 m/day. Healing of implants with a wide space around them is comparable to secondary healing of a bone fracture, as bone formation occurs via formation of a fibrous and bony callus, at about 50 to 100 µm/day. The temporal sequence is woven bone with subsequent remodeling into lamellar bone.

During preparation of the implant bed, periosteal intracortical and endosteal blood vessels are damaged. As a result blood accumulates in peri-implant space, with a loose attachment of fibrin on the surfaces of both bone and implant. This hematoma will be remodeled by proliferating tissue with new capillaries and fibrous collagen connective tissue in 7 to 14 days. New bone formation can occur directly in the vicinity of the implant depending upon the degree of its stability. Implant instability influences cell differentiation and therefore also bone formation. So the implant stability is an absolute requirement for all types of implants with adequate blood supply.

Bony remodeling of the callus is completed after 4 to 6 weeks, thorough activation of the Haversian system, numerous resorption canals are formed, and the remodeling process into lamellar bone begins. These mineralization processes, which transforms the osteoid into calcified osseous substance, proceed at about 1 micrometer per day.

Different Phases of Healing

Osseous Healing—Early Phase

Preceded by hemorrhage and formation of a blood clot, this coagulum consists of fibrin and embedded blood cells and represents the scaffold for reparative (granulation) tissue, the coagulum begins to organize with ingrowth of capillaries and pre-osteoblasts (centripetal bone growth).

During this early stage, in addition to new bone formation, the macrophages as well as multinucleated giant cells appear and recognizes the implant as foreign body. As bone formation is initiated at the implant surface, the number of multinucleated giant cells are reduced.

Osseous Healing—Late Stage

Depending upon the width of the gap between the implant surface and the osseous bed, direct filling of the space can occur about 0.2 mm by means of concentric bony apposition. Wider spaces will usually be filled within 14 days by a network of new woven bone, which will be remodeled in about 2 months into lamellar bone: remnants of the early woven bone may persist centrally. Direct bony contact with implant surface ranges from 56 to 85 percent with screw-type implants and 46 to 82 percent with linkow blade implants. Areas of the implant surface not covered with bone will manifest adipose cells without an intervening fibrous layer.

PERI-IMPLANT COMPLICATIONS

Despite the long-term predictability of osseointegrated implants, biologic, biomechanical, and esthetic complications can occur in a small percentage of cases.

PERI-IMPLANT DISEASES

Pathologic alterations in the tissues that contact a dental implant can be placed in the above category.

Types of Peri-implant Diseases

1. *Peri-implant mucositis*: Inflammatory changes, which are confined to soft tissue surrounding an implant is termed as peri-implant mucositis.

2. *Peri-implantitis*: It is a progressive peri-implant bone loss in conjunction with soft tissue inflammatory lesion. Peri-implantitis begins at the coronal portion of the implant, while the more apical portion of implant remains osseointegrated. This means that the implant is not clinically-mobile until late stages when bone loss had progressed to involve the complete implant surface.

Clinical Features

- Color changes, bleeding upon gentle probing.
- Pocket formation and radiographic bone destruction.
- Suppuration, calculus build-up and swelling.
- Mobility has been extensively described to detect early and late failures.

Diagnosis

A number of clinical parameters used to evaluate periodontal conditions have also been used to assess peri-implant conditions. These parameters include evaluation of oral hygiene, peri-implant marginal tissues, and bone implant interface.

Probing

A successful implant generally allows probe penetration of approximately 3 to 4 mm and the location of peri-implant bone level can be expected to be about 1 mm apical to the position of the probe tip.

Radiographs

Reveal the peri-implant bone status as well as the marginal bone level. Periapical intraoral radiographs are obtained instead of OPG (which have lower discrimination power).

Direct imaging may have the potential to replace conventional radiology.

To diagnose a compromised implant site, soft tissue measurements using manual or automated probes have been suggested; careful monitoring of probing depth and clinical attachment level seems useful in detecting changes of the peri-implant tissue.

Microbial Monitoring

It is useful in evaluating the peri-implant health condition and microbial composition of a peri-implantitis site.

Management and Maintenance

Management

Occlusal therapy: When excessive forces are considered the main etiologic factor for peri-implant bone loss treatment involves an analysis of fit of the prosthesis

- The number and position of implants.
- Occlusal evaluation.

Change in prosthesis design, improvement of implant number, position and occlusal equilibration can contribute to arrest the progression of peri-implant tissue breakdown.

Anti-infective therapy: The nonsurgical treatment of peri-implantitis involves:

- Local removal of plaque deposits with plastic instruments and polishing of all accessible surface with pumice.
- Subgingival irrigation of all peri-implant pockets with 0.12 percent chlorhexidine
- Systemic antimicrobial therapy for 10 consecutive days
- Improved patient compliances with oral hygiene until a healthy peri-implant site is established.
- Conventional hand and ultrasonic instruments are not suitable for the preparation and detoxification of the implant surface.
- Irradiation with soft lasers for elimination of bacteria associated with peri-implantitis has also shown promising results in the destruction of bacterial cells.

Surgical techniques for treatment of peri-implantitis: Once the inflammatory process in the peri-implant tissue is under control, an attempt may be made to improve or re-establish osseointegration.

The surgical procedures are modified from techniques used to treat bone defects around the teeth.

Re-osseointegration: It can be defined as the growth of new bone in direct contact to the previously contaminated

implant surface without an intervening band of organized connective tissue.

Maintenance

After surgical intervention, all patients are placed on a close recall schedule. It is advised to schedule maintenance visits at least every 3 months. This allows for monitoring of plaque levels, soft tissue inflammation, and changes in the levels of bone.

Oral Hygiene Aids

- Toothbrushes with soft, rounded bristles should be used because the surfaces of the implants are easily damaged.
- Toothpaste should be only minimally-abrasive; the tooth-cleaning procedures should be conducted by rinsing or brushing with chlorhexidine.
- Gauze strips or superfloss are effective for cleaning interproximally.
- Irrigators can also be used as adjunctive aids.

KEYPOINTS

1. A dental implant is a biologic or alloplastic biomaterial inserted into soft or hard tissues of the mouth for functional or cosmetic purposes.
2. Osseointegration is a direct structural and functional connection between ordered living bone and the surface of the load carrying implant.
3. Soft tissue interface of the implant consists of mucosal tissues around intraosseous implants which form a tightly adherent band consisting of dense collagenous lamina propria covered by stratified squamous keratinizing epithelium.
4. Bone implant interface is the relationship between endosseous implants and bone which involves mechanisms like fibro-osseous integration, osseo-integration and bioactive integration.
5. Based on shape and form implants are classified into—Endosteal, subperiosteal, transosteal, submucosal implants and endodontic stabilizer.
6. Based on surface characteristics implants are classified into titanium plasma sprayed coating, sandblasting-surface etching, laser-induced surface roughening and hydroxyapatite coating.
7. Surgical procedure involves:
   a. One-stage endosseous implant surgery.
   b. Two-stage endosseous implant surgery.
8. Healing involves two phases:
   a. Osseous healing—Early phase.
   b. Late stage.
9. Pathologic alterations in the tissues that contact a dental implant are peri-implant diseases, which are peri-implant mucositis and peri-implantitis

KNOW MORE ...

Implant Failure

Failures in implant therapy can happen sometimes and this could be due to:

a. Complications that arise during the early phase following implant insertion—Early implant failures.
b. Complications that arise after the reconstruction of the implant—late implant failures.

Causes for Early Implant Failures

1. Improper preparation of the implant site.
2. Bacterial contamination.
3. Improper mechanical stability following implant insertion.
4. Premature loading of the implant.

Causes for Late Implant Failures

According to proceedings of the 3rd European Workshop on Periodontology the late implant failures could be as a result of:

1. Excessive load.
2. Infection, e.g. periimplant mucositis, periimplantitis.

REVIEW QUESTIONS

1. Describe the biological considerations of implant therapy.
2. Enumerate various types of implants and implant systems.
3. What is peri-implantitis?

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48 Chapter

Maintenance Phase (Supportive Periodontal Treatment)

- IMPORTANCE OF MAINTENANCE PHASE
- RATIONALE FOR SUPPORTIVE PERIODONTAL TREATMENT
- CAUSES FOR RECURRENCE OF PERIODONTAL DISEASE
- OBJECTIVES OF MAINTENANCE PHASE
- PARTS OF MAINTENANCE PHASE
- DETERMINATION OF MAINTENANCE RECALL INTERVALS

IMPORTANCE OF MAINTENANCE PHASE

Chronic periodontitis, like most other chronic infections require supervision and maintenance overtime. Maintenance therapy after active treatment includes not only the care that patients receive through personal oral hygiene but also by the recall visits and re-evaluations done by the dental team. Maintenance therapy is often supportive in nature hence, it is also known as *supportive periodontal treatment (SPT)*. In this phase, patients must be made to understand the purpose of a maintenance program, and the dentist must emphasize on the fact that the preservation of the teeth in question are dependent on it.

RATIONALE FOR SUPPORTIVE PERIODONTAL THERAPY

Rationale for maintenance phase is to prevent or minimize the recurrence of periodontal diseases by controlling factors known to contribute to the disease process.

The main aim of long-term therapy is to provide supervised control for the patient in order to maintain a healthy and functional, natural dentition for life. It is only with proper maintenance, including early detection and treatment of recurrent periodontal diseases that such an objective can be achieved.

CAUSES FOR RECURRENCE OF PERIODONTAL DISEASE

1. Incomplete subgingival plaque removal.
2. Nature of dentogingival unit.
3. Improper restorations placed after the periodontal treatment was completed.
4. Failure of the patient to return for periodic recall visits.
5. Presence of some systemic diseases that may affect host resistance to previously acceptable levels of plaque.

GOALS OF SUPPORTIVE PERIODONTAL TREATMENT

1. To prevent or minimize the recurrence and progression of periodontal disease in patients who have been

Symptoms and causes of recurrent diseases	
Symptoms	*Probable cause*
Increase in mobility	Poor oral hygiene causing increase in inflammation and calculus formation, faulty restoration, deteriorating or poorly designed prosthesis, systemic diseases modifying host response to plaque.
Recession	Toothbrush abrasion, inadequate keratinized gingiva, frenum pull, orthodontic therapy.
Increased mobility with no change in pocket depth and no radiographic change.	Trauma due to lateral occlusal interference, Bruxism, high restoration, poor crown root ratio, poorly designed or worn out prosthesis.
Increased pocket depth with no radiographic change.	Poor oral hygiene, subgingival calculus, ill fitting partial dentures, failure of new attachment procedures, cracked teeth, new periodontal diseases.
Increased pocket depth with increased radiographic bone loss.	Poor oral hygiene, subgingival calculus, deteriorating restoration, inadequate surgical treatment, cracked teeth, systemic diseases modifying host response.

previously treated for gingivitis, periodontitis and for peri-implantitis.

2. To prevent or reduce the incidence of tooth loss by monitoring the dentition and by any prosthetic replacement of the natural teeth.
3. To increase the probability and treating in a timely manner, other diseases or conditions found in the oral cavity.

OBJECTIVES OF MAINTENANCE PHASE

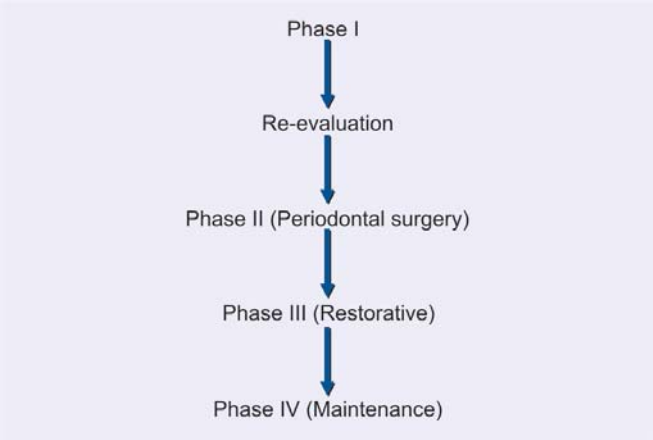
1. Preservation of alveolar bone support (radiographically).
2. Maintenance of stable, clinical attachment level.
3. Reinforcement and re-evaluation of proper home care.
4. Maintenance of a healthy and functional oral environment.

PARTS OF MAINTENANCE PHASE

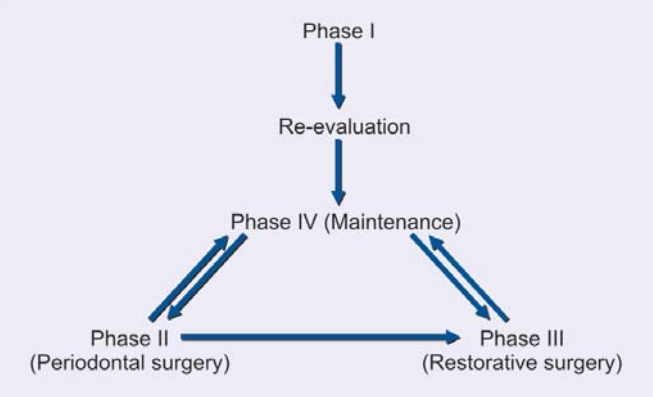
Part–I: Examination (Approximate Time –7 Min)

- Medical history changes
- Oral pathological examination
- Oral hygiene status
- Gingival changes

Flow chart 48.1: Incorrect sequence of periodontal treatment phases



Flow chart 48.2: Correct sequence of periodontal treatment phases



- Pocket depth changes
- Mobility changes
- Occlusal changes
- Restorative and prosthetic changes.

Part–II: Treatment (Approximate Time – 35 Min)

- Oral hygiene reinforcement
- Scaling and polishing
- Chemical irrigation.

Part–III: Schedule Next Procedure

- Schedule next recall visit
- Schedule further periodontal treatment
- Schedule or refer to restorative or prosthetic treatment.

Sequence of Maintenance Visits

1. Incorrect sequence of periodontal treatment phases (Flow chart 48.1).
2. Correct sequence of periodontal treatment phases (Flow chart 48.2).

Schallhorn and Snider (1981) proposed four separate categories of periodontal maintenance therapy. They are:

1. *Preventive maintenance therapy*—periodontally-healthy individuals.
2. *Trial maintenance therapy*—mild-to-moderate periodontitis.
3. *Compromised maintenance therapy*—medically-compromised patients where active therapy is not possible.
4. *Post-maintenance treatment therapy*—maintenance for prevention of recurrence of disease.

DETERMINATION OF MAINTENANCE RECALL INTERVALS

Based on studies of human periodontal treatment, it is recommended that the patient be seen initially for recall treatment 2 to 4 weeks following treatment (for transitional procedures). After 3 or 4 such sessions the interval can be extended to 3 months, but may be varied according to the patient's needs.

Following factors may be considered in determining the recall intervals:

1. *Severity of disease*: The more severe the disease, the more frequently the patient is recalled.
2. *Effectiveness of home care*: Good home care decreases the frequency of recall.
3. *Degree of control of inflammation achieved*: As the tissue regain the total health, the frequency decreases.

Procedures to be Performed at Recall

	Procedure	*Evaluation*
1.	At clinical examination	All findings recorded at the base line are compared. Evaluations of complete oral and periodontal status, occlusal and prosthetic appliances, etc.
2.	Radiographically	Assessment of bone levels. Any additional findings are recorded.
3.	Assessment of disease	By comparing the findings obtained at base line.
4.	Assessment of patients oral hygiene	Comparison with baseline data. Behavioral modification if necessary.
5.	Treatment	Removal of any fresh deposits, occlusal therapy, application of antimicrobial agents if indicated. Appointments for future periodontal therapy.

Management of Particular Type of Recall Patients

1. The patients who will not cooperate in oral hygiene but will pay for office care: In this case patient management includes frequent, thorough root instrumentation.
2. The patient who has refused surgical treatment: The recall must be shorter for this patient than for patients who have received surgical therapy. The root instrumentation phase involves more time and is difficult to perform properly.
3. The patient who has been hospitalized for several weeks: If the patients condition permits, periodic scaling and review of oral hygiene by the hygienist should be performed in hospital.
4. The patients who has been fully and successfully treated yet now shows distinct breakdown in localized areas: A general health review, if this is favorable, retreatment

of involved areas is done. Flap curettage and antibiotic therapy should be given. Recall appointments are scheduled for at least once in every 3 months.

KEYPOINTS

1. Maintenance therapy is often supportive in nature hence it is also called supportive periodontal treatment (SPT).
2. The main objective of supportive periodontal therapy is to prevent or minimize the recurrence of periodontal diseases by controlling factors known to contribute to the disease process.
3. The interval of recall visits is determined by severity of disease, effectiveness of patients home care and degree of control of inflammation achieved.
4. Based on many long-term studies, the interval for maintenance visits has been recommended. The patient should be seen initially for recall treatment at 2 to 4 weeks following treatment. After 3 or 4 such sessions the interval can be extended to 3 months (can be varied according to patient needs).

KNOW MORE ...

SPT in Daily Practice

The SPT recall hour is generally composed of:

1. Examination, re-evaluation and diagnosis (ERD) for 10-15 minutes.
2. Motivation, reinstruction and instrumentation (MRI) for 30-40 minutes.
3. Treatment of reinfected sites (TRS).
   - Includes small surgical procedures.
   - Local drug delivery.
   - Local intensive debridement.

   This section may require additional appointments.
4. Polishing of the entire dentition, application of fluorides and determination of future SPT visits — approximately 5 to 10 minutes may be reserved.

REVIEW QUESTIONS

1. What are the objectives of supportive periodontal therapy?
2. What is the sequence of maintenance visits?
3. Describe the importance of maintenance therapy in periodontics.

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49 Chapter

Occlusal Evaluation and Therapy in the Management of Periodontal Disease

TERMINOLOGY

Occlusion: It is defined as the functional relationship between the components of the masticatory system including the teeth, supporting tissues, neuromuscular system, temporomandibular joints and craniofacial skeleton.

Intercuspal position (ICP): The position of the mandible when there is maximal intercuspation between the maxillary and mandibular teeth.

Excursive movement: Any movement of the mandible away from ICP.

Protrusion: Movement of the mandible anteriorly from ICP.

Retrusion: Movement of the mandible posteriorly from ICP.

A physiologic occlusion: It is when no signs of dysfunction or disease are present and no treatment is indicated.

A non-physiologic (or traumatic) occlusion: It is associated with dysfunction or disease due to tissue injury, and treatment may be indicated.

A therapeutic occlusion: It is the result of specific interventions designed to treat dysfunction or disease.

CLINICAL EVALUATION OF OCCLUSION

Temporomandibular Disorders (TMDs)

Screening Examination

Screening for temporomandibular disorders (TMDs) should be included in all routine dental examinations.

The accepted components of this examination are:

1. *Maximal intercuspal opening*—recorded in millimeters.
2. *Opening—closing pathway*—deviations if any are recorded.
3. *Auscultation for TMJ sounds*—joint sounds can be, discrete clicks or diffuse grating sounds (crepitus).
4. *Palpation for TMJ tenderness*—could be mild, moderate or severe tenderness, recorded with light bilateral palpation along the lateral aspects of condyles.
5. *Palpation for muscle tenderness*—the masseter and temporalis muscles are examined bilaterally using

moderate finger pressure. The muscle pain may be mild, moderate or severe type.

Intraoral Evaluation of Occlusion

1. Identification of intercuspal position (ICP) zones of contact by placing mylar strips between the teeth and asking the patient to close and open.
2. Guidance in excursive movements.
3. Tooth mobility.
4. Attrition—excessive attrition should be noted.

MANAGEMENT OF TRAUMA FROM OCCLUSION

When an adequate quantity of periodontal support is present to withstand the normal forces of occlusion, yet excessive parafunctional forces exceed the adaptive capacity of the attachment apparatus, the disease process is referred to as *primary occlusal trauma*. When the quantity of the remaining normal attachment apparatus has been compromised by periodontal disease and cannot withstand the normal forces of occlusion, the disease process is referred to as *secondary occlusal trauma*.

a. In cases of primary occlusal trauma with gingivitis or periodontitis. The treatment is simple and conservative. First, periodontal therapy is done which include plaque control, scaling and root planing. If there is progressive mobility then occlusal therapy in the form of selective grinding and the use of night guard may be justified.
b. In cases of secondary occlusal trauma and advanced periodontitis, the treatment is often complicated. It often requires advanced periodontal therapy, including root resection, antimicrobial therapy and regenerative procedures along with adjunctive orthodontics, occlusal adjustment by selective grinding and splinting for periodontal stabilization is advocated.

POSSIBLE REQUIREMENTS FOR OCCLUSAL STABILITY

1. Intercuspal position
   a. Light or absent anterior contacts
   b. Well-distributed posterior contacts
   c. Coupled contacts between posterior teeth
   d. Cross tooth stabilization
   e. Forces directed along long axis of each tooth.
2. Smooth excessive movement without interference
3. No trauma from occlusion
4. Favorable subjective response to occlusal form and function.

OCCLUSAL THERAPY

Occlusal therapy is performed to establish a stable functional relationship, favorable to the oral health of the patient, including the periodontium. Various procedures to achieve this objective are:

a. Interocclusal appliance therapy.
b. Occlusal adjustment.
c. Restorative procedures.
d. Orthodontic movement and orthognathic surgery.

Restorative procedures and orthodontic tooth movements in the management of periodontal patient is covered elsewhere, in this book.

Occlusal adjustment or coronoplasty is the selective reshaping of occlusal surface with the goal of establishing a stable, non-traumatic occlusion. This is achieved by reshaping the crown surfaces and eliminating undesirable occlusal supracontacts and the creation of a stable mandibular position.

Occlusal adjustment had once been the most commonly employed procedure for treating occlusal trauma, temporomandibular joint problems and other associated problems. Since occlusal adjustment is an invasive, irreversible intervention, it should rarely be considered. It should never be undertaken as a preventive measure.

Coronoplasty is generally performed after gingival inflammation and periodontal pockets have been eliminated. The author recommends a reference book for a detailed description on the procedure of occlusal adjustment.

KNOW MORE ...

Occlusal Therapy

Guidelines to occlusal therapy in general are:

a. A sound biologic rationale should exist.
b. Should be considered an adjunct to periodontal therapy.
c. Be prepared for irreversible occlusal changes in the context of restorative care.
d. Thorough informed consent must be provided to the patient.

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50 Chapter

The Role of Orthodontics as an Adjunct to Periodontal Therapy

- RATIONALE FOR ORTHODONTIC TREATMENT IN PERIODONTAL THERAPY
- USE OF ORTHODONTICS AS AN ADJUNCT TO OVERALL TREATMENT
- INDICATIONS AND CONTRAINDICATIONS OF ORTHODONTIC THERAPY
- TIMING OF ORTHODONTIC PROCEDURES IN PERIODONTAL TREATMENT
- IATROGENIC EFFECTS ASSOCIATED WITH ORTHODONTIC TREATMENT
- MICROBIOLOGY AROUND ORTHODONTIC BANDS

INTRODUCTION

The primary objective of periodontal therapy is to restore and maintain the health and integrity of the attachment apparatus of teeth. In adults, the loss of teeth or periodontal support can result in pathologic tooth migration involving either a single tooth or a group of teeth. This may result in the development of a median diastema or general spacing of the teeth, rotation or tipping of premolars and molars with the collapse of the posterior occlusion and decreasing vertical dimension. Adjunctive orthodontic therapy is necessary to resolve these problems.

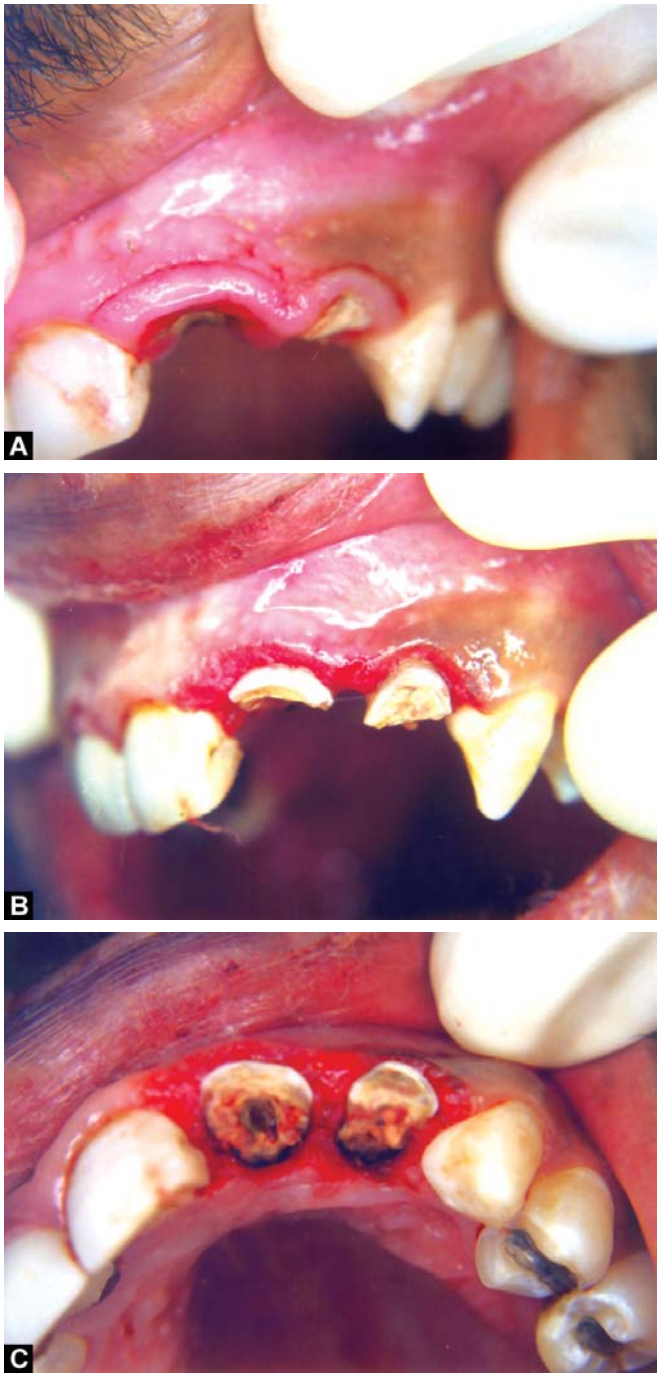
RATIONALE FOR ORTHODONTIC TREATMENT IN PERIODONTAL THERAPY

Reducing Plaque Retention

Example: Crowded teeth (arch length deficiency), mesially-tipped teeth, usually into an edentulous area creates plaque accumulation sites that are difficult to clean. In addition, they open the distal contact creating an area of food impaction. Crowding also creates enlarged contact surfaces and altered embrasure spaces that are displaced apically, thereby becoming less accessible to use floss and other plaque removing devices. In these situations orthodontic treatment can improve the health of the tissues.

Improving Gingival and Osseous Form

There is an interrelation between the position of the tooth, the shape of the gingiva and bone that surrounds it. For example, lower first or second molar tilted into an edentulous mesial space. In these cases there is a narrow space between its crown and the bone that easily becomes inflamed and in which case a pocket may develop. Orthodontic treatment may improve the shape of the periodontium and reduces the indications for bone surgery (Figs 50.1A to C).



Figs 50.1A to C: Crown lengthening with internal bevel gingivectomy

Facilitating Prosthetic Replacements

The uprighting of tilted abutment teeth may be important for a better contoured crown and this will benefit the periodontal condition.

Improving Esthetics

Correction of pathologic tooth migration and diastema between anterior teeth. Correction of tongue-thrusting and other habits

USE OF ORTHODONTICS AS AN ADJUNCT TO OVERALL TREATMENT

1. Uprighting or repositioning of teeth to improve parallelism of abutment teeth (e.g. tipped abutment teeth)
2. Improving future pontic spaces (e.g. inadequate spaces)
3. Correcting cross bites
4. Extruding teeth/Intruding teeth
5. Correcting crowding of teeth
6. Achieving adequate embrasure space and proper root positioning
7. Repositioning teeth for implant placement
8. Restoring lost vertical dimension
9. Increasing/decreasing overjet /overbite
10. Closure of diastema.

INDICATIONS AND CONTRAINDICATIONS OF ORTHODONTIC THERAPY

Indications

This includes common problems that can be solved by minor orthodontic therapy such as crowded teeth, closure of anterior diastema, mesial tilting of molars and open contacts.

Contraindications

The only contraindication is the persistence of active disease in spite of phase-I therapy procedures. The superimposition of tooth movement on inflamed gingiva may exacerbate the periodontal problem. This can occur by shifting the position of plaque subgingivally, increasing the rate of periodontal attachment loss and altering the morphology of the bone.

TIMING OF ORTHODONTIC PROCEDURES IN PERIODONTAL TREATMENT

It is generally recommended that orthodontics be preceded by periodontal therapy based on the belief that orthodontics

in the presence of inflammation can lead to rapid and irreversible breakdown of the periodontium. But any elimination procedures like pocket or osseous reduction procedures may be postponed until the end of orthodontic therapy because tooth movement may modify gingival and osseous morphology.

IATROGENIC EFFECTS ASSOCIATED WITH ORTHODONTIC TREATMENT

Orthodontic treatment may cause injuries to the teeth and periodontium but in most of the cases the changes are reversible and regeneration and repair of the tooth structures and periodontal tissues can occur. In some cases the changes may get out of control resulting in irreparable damage. All the precautions should be taken to avoid this and radiography should be performed at regular intervals in order to disclose any iatrogenic effects during the orthodontic treatment. They are as follows:

Root Resorption

Some amount of root resorption is unavoidable especially if it is seen at the marginal and middle thirds of the root, which can be repaired by apposition of cellular cementum.

Effects of Orthodontic Bands on the Periodontium

Short-term effects are—Gingivitis and gingival hyperplasia, mostly not associated with loss of attachment.
Long-term effects are—Loss of attachment, root resorption or no effects. Any of these three possibilities may be seen in adult patients.

MICROBIOLOGY AROUND ORTHODONTIC BANDS

a. Increased *Lactobacillus* count (Bloom and Brown, 1964)
b. Increased motile organisms (Leggott et al, 1984)
c. Increased anaerobes like *Prevotella intermedia* (Diamanti, Kipioti et al, Huser et al, 1990)
d. Decreased count of facultative microorganisms/anaerobes (Diamanti, Kipoti et al, Huser et al 1990).

Effects of Orthodontics on Dentition with Normal Height of Attachment Apparatus

Number of experimental studies have demonstrated that orthodontic forces causes no damage to the supra-alveolar connective tissue and orthodontic treatment will therefore not result in periodontal tissue breakdown and pocket formation.

Dentition with Reduced Height of Attachment Apparatus

The experimental studies have demonstrated that,
a. In the absence of plaque, orthodontic forces and tooth movements failed to induce gingivitis, whereas in the presence of plaque similar forces cause angular bone defects associated with attachment loss.
b. Orthodontic forces if kept within biologic limit failed to cause gingival inflammation even in the regions with reduced periodontal support, but is of the non-inflammatory type.

Response of the Periodontal Ligament to Orthodontic Forces (Fig. 50.2)

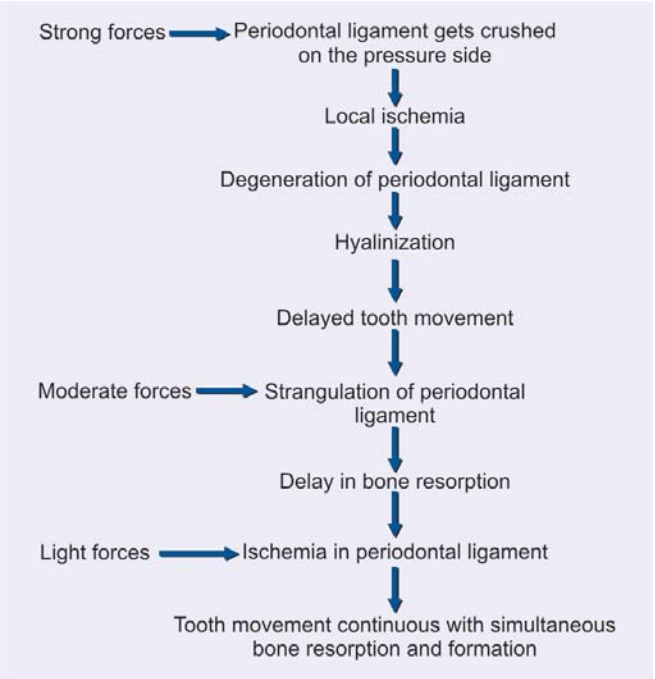


Fig. 50.2: Response of the periodontal ligament to orthodontic forces

When bone surrounding the tooth is subjected to a force it responds in the following manner:

1. Resorption occurs where there is pressure and new bone forms where there is tension.
2. When pressure is applied to the tooth, there is a initial period of movement for 6 to 8 days as the periodontal ligament is compressed. Compression of periodontal ligament results in blood supply being cut off to an area of the periodontal ligament and this produces an avascular, cell-free zone by a process termed "*hyalinization*". When hyalinization occurs the tooth stops moving (depending on the forces). The hyalinized zone is eliminated by periodontal regeneration that occurs from the reorganization of the area through resorption by the marrow spaces (undermining resorption) and adjacent areas of unaffected periodontal ligament and alveolar bone. Once the hyalinized zone is removed, tooth movement can occur again. Regeneration of periodontal ligament does not occur when inflammation is present in periodontal tissue. Hence, the inflammation needs to be controlled by periodontal treatment.

KEYPOINTS

1. Adjunctive orthodontic therapy is necessary to resolve problems like generalized spacing of teeth, rotation or tipping of teeth, collapse of the posterior occlusion and decreasing vertical dimension.
2. Basic rationale for orthodontic intervention in periodontal therapy is to reduce plaque accumulation, improve gingival and osseous form, facilitate prosthetic replacement and improve esthetics.
3. The only contraindication for orthodontic treatment is, persistent inflammation in spite of phase I therapy. In the presence of active infection, the orthodontic tooth movement can exacerbate the periodontal problems.
4. Some of the common iatrogenic effects of orthodontic treatment are, root resorption, gingival inflammation associated with orthodontic bands, changes in periodontal ligament and others.

KNOW MORE ...

Rationale for Orthodontic Treatment

Apart from reducing plaque retention, improving gingival, osseous form and esthetics, it also benefits:

a. The patient with a severe fracture of a maxillary anterior tooth, with forced eruption can permit adequate tooth restoration.
b. The patient with 'dark triangles' due to interdental papillary recession.
c. The implant patient—by repositioning the adjacent teeth.

REVIEW QUESTION

1. What are the effects of orthodontic treatment on periodontium?

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51 Chapter

Periodontal-Restorative Inter-relationship

- MARGINS OF RESTORATIONS
- RULES FOR MARGIN PLACEMENT
- RESTORATIVE MARGINS ENCROACHING ON THE BIOLOGIC WIDTH
- CROWN CONTOUR
- HYPERSENSITIVITY TO DENTAL MATERIALS
- PROXIMAL CONTACT AND EMBRASURE
- PONTIC DESIGN

INTRODUCTION

It is a well-established fact that the periodontal health and the restoration of teeth share an intimate and inseparable interrelationship. One must always remember that for restorations to survive, long-term restorative procedures must be performed on a periodontium free of inflammation and pockets without any mucogingival involvement, and with the contour and shape of the periodontium corrected for a good functional and esthetic restorative result. For the periodontium to remain healthy, restorations must be critically prepared so that, they will remain in harmony with the surrounding periodontal tissues.

MARGINS OF THE RESTORATIONS

A clinician has three options for margin placement:

i. Supragingival
ii. Equigingival (even with the tissue)
iii. Subgingival

Restorations should be chosen not just by keeping esthetics in mind but for their favorable periodontal impact as well.

Supragingival margins have the least impact on the periodontium. The use of equigingival margins was thought to retain more plaque thereby interfering with gingival health, today these concerns are not valid. The greatest biologic risk occurs when margins are placed subgingivally. Hence, from periodontal point of view, both supragingival and equigingival margins are well-tolerated.

In view of the scientific evidence available, restorative margins should be preferably placed supragingivally. However in certain situations, where subgingival margins are unavoidable like carious tooth, tooth fracture or esthetic concern, it should be placed not more than 0.5 mm into the sulcus so that, these margins could be accessible for finishing procedures.

In addition, if the margins are placed too far below the gingival tissue crest, it violates the gingival attachment apparatus.

RULES FOR MARGIN PLACEMENT

Rule 1: If the probing depth is 1.5 mm or less, the restoration margin has to be placed below gingival tissue crest.

Rule 2: If the probing depth is more than 1.5 mm, then the margin of the restoration is placed at one half of the probing depth below the gingival crest.

Rule 3: If the sulcus probing depth is more than 2 mm, then the tooth has to be evaluated for gingivectomy procedure to reduce the sulcus depth to 1.5 mm. Once this is achieved margin placement is done in accordance to Rule 1.

RESTORATIVE MARGINS ENCROACHING ON THE BIOLOGIC WIDTH (FIG. 51.1)

The soft tissue attachment to the tooth between the base of the gingival sulcus and the crest of the alveolar bone is called the *biologic width*.

Invasion into this biologic width should be avoided in order to prevent attachment loss and persistent gingival inflammation.

Biologic width = Junctional epithelium (0.97 mm) + Connective tissue attachment (1.07 mm) = 2.04 mm.

METHODS TO CORRECT BIOLOGICAL WIDTH VIOLATION

Biological width violation can be corrected either surgically (removing bone away from proximity to the restorative margin) or orthodontically (by moving the tooth and thus moving the margin away from the bone).

The orthodontic procedure can be accomplished in two ways:

1. *By slow orthodontic extrusive force*: The tooth is extruded slowly bringing the bone and gingival tissue with it.
2. *By rapid orthodontic extrusive force*: Where the tooth extrusion to the desired amount is carried out over several weeks.

CROWN CONTOUR

Restoration contour plays an important role in the maintenance of periodontal health. An ideal contour must provide:

i. Access for hygiene.
ii. Fullness to create the desired gingival form.
iii. Esthetically-pleasing tooth contour.

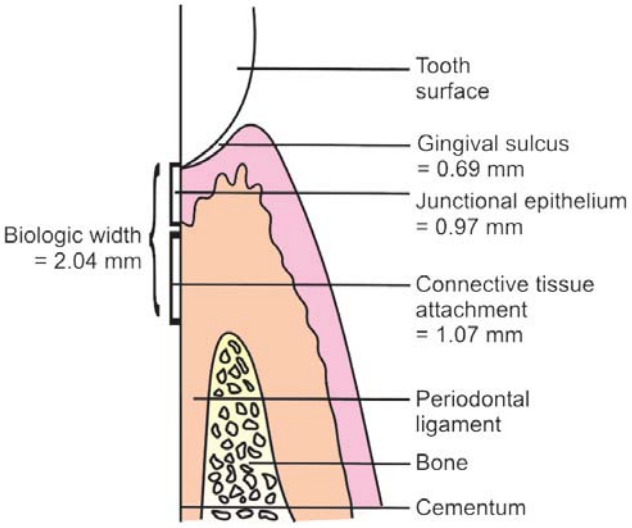

Fig. 51.1: Biologic width

iv. Protection of the marginal gingiva from mechanical injury during mastication—explained in gingival protection theory.

Hence, the crown contour should, therefore help in easy plaque removal, not its retention. Overcontouring leads to more plaque accumulation with subsequent gingival inflammation, under contouring of crowns is therefore considered ideal.

HYPERSENSITIVITY TO DENTAL MATERIALS

Rough dental surfaces favor plaque accumulation and contribute to periodontal disease. Regardless of the material used subgingivally, a smooth surface is essential on all materials. Today, the most commonly used materials are glass ionomer cements and composite resins. Though the composite resins have very smooth surface upon finishing, they do not last very long because, once it is exposed to the oral environment, its surface becomes rough. From periodontal point of view glass ionomer seems to be more acceptable because of its capability to release fluoride that has the potential to interfere with adherence of bacteria on the tooth surface.

PROXIMAL CONTACT AND EMBRASURE

It is believed that, the ideal interproximal embrasure should house the gingival papilla without impinging on it. Proper

proximal contact is essential to prevent food impaction. The contact point should be placed occlusally and facially to facilitate access for interproximal plaque control. The ideal contact should be 2 to 3 mm coronal to the attachment, which coincides with the depth of the average interproximal sulcus.

PONTIC DESIGN

Traditionally, four types of pontic designs have been proposed—sanitary, ridge lap, modified ridge lap and oviate pontic designs.

1. *Sanitary pontic*: Where the tissue surface of the pontic is 3 mm from the underlying ridge (Fig. 51.2).
2. *Ridge lap pontic*: Where the tissue surface of the pontic straddles the ridge much like a saddle. The entire surface is convex and is very difficult to clean (Fig. 51.3).
3. *Modified ridge lap pontic*: The tissue surface on the facial surface is concave, however, the lingual saddle has been removed to allow access for oral hygiene (Fig. 51.4).

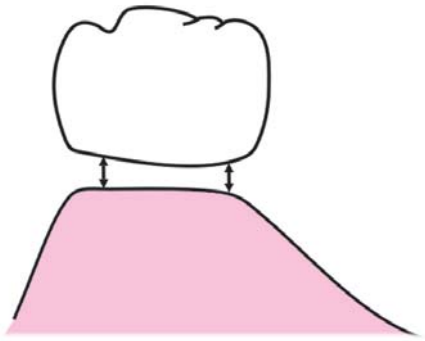

Fig. 51.2: Sanitary pontic

Fig. 51.3: Ridge lap pontic

Fig. 51.4: Modified ridge lap pontic

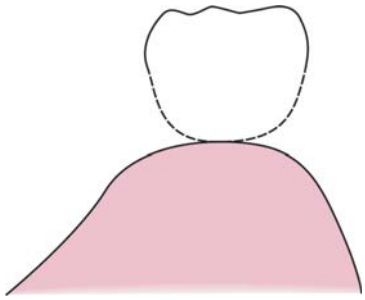

Fig. 51.5: Ovate pontic

4. *The ovate pontic*: This is the ideal pontic design. It is created by forming a receptor site in the edentulous ridge with either a diamond bur or electrosurgery.

Whenever fixed prosthesis is designed to replace missing teeth, contact between the pontic and mucosa should be avoided or kept minimal so that meticulous plaque control can be advocated (Fig. 51.5).

KEYPOINTS

1. Restorative procedures should be performed on a periodontium free of inflammation and other signs of periodontal disease.
2. The restorative margins can be placed at supragingival, equigingival or subgingival locations. Supragingival margin has the least impact on the periodontium.
3. The soft tissue between the base of the gingival sulcus to the crest of the alveolar bone is called the '*biologic width*' which is 2.04 mm.
4. Ideal contour must provide, access for hygiene, fullness to create the desired gingival form and esthetically-pleasing tooth contour.
5. Four types of pontic designs have been proposed: sanitary, ridge lap, modified ridge lap and ovate pontic designs.

KNOW MORE ...

Sequence of Treatment in Preparing Periodontium for Restorative Dentistry

It can be divided into two phases:

1. *Control of active periodontal inflammation with non-surgical and surgical treatment*
   - Extraction of hopeless teeth.
   - Scaling, root planing and oral hygiene instructions.
   - Re-evaluation, anti-infective therapy.
   - Periodontal surgery.
   - Adjunctive orthodontic therapy.
2. *Preprosthetic surgery*
   - Treatment of mucogingival deformities.
   - Ridge reconstruction and preservation.
   - Crown lengthening procedure.

REVIEW QUESTIONS

1. What are the effects of restorative procedure on the periodontium?
2. What is the biologic width?

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52 Chapter

Drugs Used in Periodontal Therapy

- CLASSIFICATION OF VARIOUS DRUGS USED IN PERIODONTAL THERAPY
  - Depending on Antimicrobial Efficacy and Substantivity
  - Chemicals Used for Supragingival Plaque Control
- ANTIBIOTICS USED IN PERIODONTAL THERAPY
- NON-STEROIDAL ANTI-INFLAMMATORY DRUGS
- LOCAL DRUG DELIVERY SYSTEMS
- LOCAL ADMINISTRATION OF ANTIBIOTICS AND ANTIMICROBIAL AGENTS
- CURRENTLY AVAILABLE LOCAL DRUG DELIVERY SYSTEMS IN PERIODONTAL THERAPY

INTRODUCTION

It is a well established fact that mechanical therapy is the foundation for periodontal therapy. In the past only necrotizing ulcerative gingivitis was treated with antibiotic therapy because it was considered as a fusospirochetal infection. With emerging evidence of bacterial specificity in relation to aggressive forms of periodontitis and also the difficulty in suppressing tissue invaded pathogens with conventional therapy in certain cases (like juvenile periodontitis) led to the development of antimicrobial treatment strategies. But total elimination of the periodontal pathogens with antibiotic therapy alone may not be possible, unless it is combined with scaling and root planing.

CLASSIFICATION OF VARIOUS DRUGS USED IN PERIODONTAL THERAPY

Various drugs used in periodontal therapy can be divided into:

1. Antiplaque and anticalculus agents.
2. Antibiotics in the management of periodontal disease.
3. Anti-inflammatory drugs.

Depending on Antimicrobial Efficacy and Substantivity

Depending on antimicrobial efficacy and substantivity drugs can be classified as:

1. *First generation agents*: They reduce plaque score by 20 to 50 percent, efficacy is limited because of poor substantivity.
   Examples: antibiotics, quaternary ammonium compounds and sanguinarine. It should be used 4 to 6 times daily.
2. *Second generation agents*: They are retained longer in the tissues and their slow release property provides overall reduction in plaque score by 70 to 90 percent (should be used twice daily).

Example: chlorhexidine, triclosan with either copolymer or zinc citrate.

3. *Third generation agents*: It should be effective against specific periodontopathic organisms. The most promising agent seems to be Delmopinol which is a surface active agent. Though it is a weak antimicrobial agent, it can exert its effect by binding to salivary proteins and thereby alters the cohesive and adhesive properties of the films formed.

Chemicals used for Supragingival Plaque Control

It is discussed elsewhere in this book.

Phenols

Mechanism of Action: Most phenols exert a nonspecific antibacterial action which is dependent upon the ability of the drug in its nonionized form to penetrate the lipid component of the cell walls of gram-negative organisms. The resulting structural damage will affect the permeability which in turn affects the exchange of substances in addition to several metabolic processes that are dependent upon enzymes contained within the cell membranes. Phenolic compounds have also been shown to exhibit anti-inflammatory properties, which may result from their ability to inhibit neutrophil chemotaxis, the generation of neutrophil superoxide ion and the production of prostaglandin synthetase.

Listerine™ is an over the counter phenol precipitate that contains thymol, eucalyptol, menthol, methyl salicylate, benzoic acid and boric acid.

Quaternary Ammonium Compound

Quaternary ammonium compounds are cationic antiseptics and surface active agents. Quaternary ammonium compounds tend to be more effective against gram-positive than gram-negative organisms. This may suggest that these compounds are most effective as anti plaque agents when used against early developing plaque, which predominantly contains gram-positive bacteria.

Quaternary Ammonium Compounds include:

i. Benzathonium chloride.
ii. Benzalkonium chloride.
iii. Cetyl pyridium chloride—CPC (Reach™).
iv. Domiphen bromide (Cetrimide).

Mechanism of action: They have some similarities in their mechanism of action to chlorhexidine, the molecules possess both hydrophobic, and hydrophilic groups allowing for ionic and hydrophobic interactions. It is assumed that the interaction with bacteria occurs with cationic binding, to the phosphate groups in the cell wall teichoic acid in the gram-positive bacteria and to the phosphate groups of the cell wall and in general to the membrane lipopolysaccharide of gram-negative bacteria. The membrane integrity may subsequently be disrupted by interaction with the lipophilic portion of the molecule, causing disturbance of membrane functions and leakage of cytoplasmic material.

ANTIBIOTICS USED IN PERIODONTAL THERAPY

Chemotherapeutic agents refers to the ability of an active chemical substance to provide a therapeutic clinical benefit.

Antimicrobial agents are chemotherapeutic agents that reduce the amount of bacteria present, either by specifically targeting certain organisms or by nonspecifically reducing all bacteria.

Antibiotics are a form of antimicrobial agents produced by or obtained from microorganisms, that have the capacity to kill other microorganisms or inhibit their growth.

Chemotherapeutic agents may be administered either:

i. Systemically, or
ii. Locally.

Systemic Administration of Antibiotics

It may reduce or eliminate bacteria that cannot be removed by scaling or root planing, e.g. bacteria in the tissues/root surfaces.

Other uses of systemic administration of antibiotics include:

- Decrease in plaque and gingivitis.
- Retards bone loss.

- Use of antibiotics in conjunction with nonsurgical therapy reduces/eliminates the need for periodontal surgery.
- It is also useful in cases of aggressive periodontal diseases that may be resistant to traditional nonsurgical or surgical therapies, e.g. localized juvenile periodontitis, rapidly progressive periodontitis.

Advantages of Systemic Medication

i. It ensures drug penetration till the base of the pocket.
ii. Affects tissue invasive organisms.
iii. Takes less time and is inexpensive.
iv. Treats multiple sites simultaneously.
v. In acute conditions like necrotizing ulcerative gingivitis, etc. it is used to decrease active inflammation.
vi. As a premedication for patients with medical problems requiring prophylactic antibiotic coverage.

Local Administration of Antibiotics

Many chemotherapeutic agents can be delivered locally.

Advantages of Local Drug Administration

i. Greater concentrations are achieved with reduced drug doses.
ii. Systemic side effects are reduced.
iii. Slow releasing devices have the advantage of releasing antibiotics gradually.
iv. Their effect can be directed to specific target area.

Disadvantages

It can induce superinfections or hypersensitivity reactions.

According to Gibson

An ideal antibiotic for use in prevention and treatment of periodontal diseases should be:

- Specific to periodontal pathogens
- Allogenic
- Nontoxic, substantive
- Not in general use for treatment of other diseases
- Inexpensive.

Tetracyclines

Antibacterial Actions

Tetracyclines are a group of antibiotics that are derived naturally from Streptomyces or derived semi-synthetically. These antibiotics are bacteriostatic and are generally more effective against gram-positive bacteria than gram-negative bacteria. Tetracyclines are very effective in treating periodontal diseases mainly because of their concentration in gingival crevicular fluid (GCF) which is 2 to 10 times more than that in serum. In addition, many studies have proved that tetracyclines even at low concentrations are very effective against many periodontal pathogens.

Tetracycline hydrochloride is also a chelating agent and chelates Ca^{2+}, Mg^{2+}, Al^{3+} in the gastrointestinal tract. These ions, especially calcium are present in a variety of food substances. Since the chelate is present poorly absorbed, it is advisable to take tetracycline either half an hour before or after food.

Other properties of tetracyclines which are of value in the management of periodontal diseases are:

a. *Tetracyclines and collagenase inhibition (Host-derived collagenase):* These enzymes are derived from a variety of sources including fibroblasts, epithelial cells, macrophages and neutrophils. Collagenase derived from neutrophils are more susceptible to tetracycline induced inhibition.
b. *Tetracyclines and bone resorption:* The antiproteolytic properties together with anticollagenase activity has resulted in the use of these drugs to stop/inhibit bone resorption. Tetracyclines also inhibit bone resorption induced by parathyroid hormone, prostaglandin E series and bacterial endotoxins.
c. *Anti-inflammatory actions of tetracyclines:* Potential anti-inflammatory properties include the ability of tetracyclines to suppress polymorphonuclear leukocyte activity, in particular, the scavenging action of reactive oxygen metabolites. Alternatively, the drugs may block eicosanoid synthesis (PGE_2) by inhibiting phospholipase A_2 activity.

d. *Tetracycline and fibroblast attachment:*
   - Pretreatment of dentine with tetracyclines enhances fibroblast attachment and colonization. Tetracycline can both condition the root surface and influence the attachment properties of fibroblasts
   - Tetracyclines can bind to demineralize and release from dentine. The substantivity of tetracycline is proportional to the concentration of the drug rather than to the time of application
   - The drug also enhances fibronectin binding. (Hence, their use as an adjunct in treating periodontitis is very well established and their effect is maximum in patients with localized juvenile periodontitis and refractory periodontitis).

Adverse Effects of Tetracyclines

a. *Gastrointestinal disturbances*
   - Diarrhea
   - Nausea and vomiting
   - Severe colitis (rare).

b. *Overgrowth of resistant organisms*
   - Stomatitis
   - Vaginitis
   - Staphylococcal enterocolitis.

c. *Photosensitivity*
   - Skin rashes
   - Hypersensitivity reactions.

Significant Drug Interactions Involving Tetracyclines

Drug	*Possible sequelae*
Penicillin	Antagonism of bactericidal action.
Antacids	Impaired absorption of tetracycline.
Insulin	Enhanced hypoglycemic action of insulin.
Contraceptive pill	Pill failure.
Digoxin	Increased serum digoxin concentration.
Warfarin sodium	Enhanced anticoagulant effect.
Carbamezapine and phenytoin	Decreased serum concentration of doxycycline and minocycline.

Contraindications for the Use of Tetracyclines

a. *Pregnancy*—staining of deciduous teeth, impaired bone growth.
b. *Breastfeeding*—staining of developing teeth and gastrointestinal disturbance in children.
c. *Renal impairment*—aggravates uremia.
d. Hepatic disease.
e. *Systemic lupus erythematosus*—exacerbation of lesions.

Specific Agents and their Dosage

Tetracycline, minocycline and doxycycline are all semi-synthetic members of the tetracycline group that are commonly used in periodontal therapy.

Tetracycline—Requires administration of 250 mg four times a day. It is inexpensive but compliance may be reduced by taking 4 capsules a day.

Minocycline—Exhibits broad spectrum of antibacterial activity especially in patients with adult periodontitis.

Dosage—200 mg twice a day for 1 week, side effects include reversible vertigo.

Doxycycline—Same spectrum of activity as minocycline. Absorption from gastrointestinal tract is not altered by calcium, metal ions or antacids.

Dosage—100 mg twice on the first day followed by 100 mg once a day or 50 mg twice a day for 4 days.

Metronidazole

It is bactericidal to anaerobes and it is believed to disrupt bacterial DNA synthesis. They are not very effective against *A. actinomycetemcomitans* infections but quite effective against obligate anaerobes such as *Porphyromonas gingivalis* and *Prevotella intermedia.*

Clinically—It is used to treat necrotizing ulcerative gingivitis, adult periodontitis and rapidly progressive periodontitis.

Dosage—200 mg four times a day for 1 week/400 mg three times a day for 1 week.

Side effects

- It has antabuse effect when alcohol is ingested
- The symptoms include severe cramps, nausea and vomiting
- It inhibits warfarin metabolism
- Patients on anticoagulant therapy should avoid this drug because it prolongs prothrombin time.

Penicillins

Penicillins are bactericidal but induce allergic reactions. Most widely studied antibiotics are amoxicillin and amoxicillin clavulanate potassium (Augmentin).

Amoxicillin—It exhibits broad spectrum activity and is useful in patients with refractory or juvenile periodontitis.

Dosage—500 mg three times a day for 1 week.

Augmentin—It is a combination of amoxicillin with clavulanate potassium. It is useful in patients with refractory or juvenile periodontitis.

Dosage

- Augmentin 375 mg — amoxicillin 250 mg + clavulanic acid 125 mg three times a day for 1 week.
- Augmentin 625 mg — amoxicillin 500 mg + clavulanic acid 125 mg twice a day for 1 week.

Ciprofloxacin—It is active against gram-negative rods but has minimal effect on *Streptococcus* species. At present, ciprofloxacin is the only antibiotic effective against all the strains of *A. actinomycetemcomitans.*

Clindamycin—It acts against anaerobic bacteria and is shown to be effective in patients with refractory periodontitis. It is associated with pseudomembranous colitis more often than other antibiotics.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs)

Strong evidence suggests that products of cyclooxygenase pathway (e.g. PGE_2) are may be important mediators of pathogenic changes occurring in periodontium. Hence NSAID may be of therapeutic value in treating periodontal disease because they interfere with arachidonic acid metabolism and thereby inhibit inflammatory process. Various drugs that are studied include flurbiprofen, ibuprofen, mefenamic acid and naproxen.

Actions of Flurbiprofen

- Decreased PMNL migration
- Decreased vascular permeability
- Decreased platelet aggregation.

Mechanism of Action of NSAID (Fig. 52.1)

After the activation of inflammatory cells in the periodontium by bacteria, phospholipids in the plasma membranes of cells are activated by the enzyme phospholipase A_2 and this leads to release of free arachidonic acid in the area. Arachidonic acid can then be metabolized into prostaglandins, thromboxanes and prostacyclins by cyclooxygenase or into leukotrienes, HETE and SRS-A by lipoxygenase enzyme.

LOCAL DRUG DELIVERY SYSTEMS

The limited efficacy of mouth rinsing and irrigation in deep periodontal pockets led to the development of alternative delivery systems.

The main aim of the drug delivery system is to direct antimicrobials to the infection sites and maintaining effective

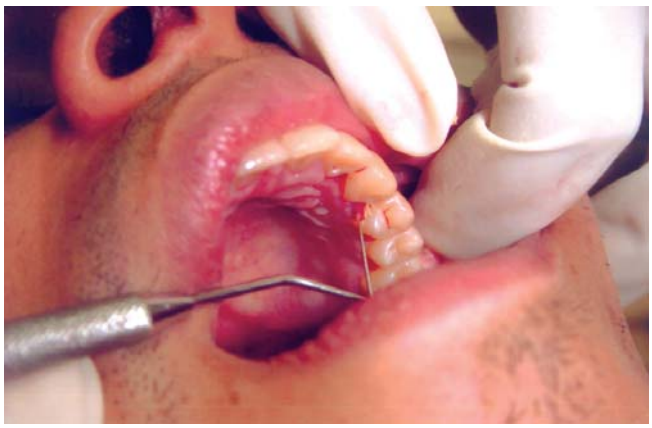

Fig. 52.1: Mechanism of action of NSAIDs

level of drugs for sufficient period of time without eliciting any major side effects.

Indications

1. As an adjunct in the treatment of few localized non-responding sites in an otherwise controlled patient.
2. In ailing and failing implant cases.
3. In medically compromised patients where surgical procedures are not recommended.
4. Periodontal abscess.
5. Periodontal maintenance therapy.
6. Patient with gastrointestinal intolerance to systemic drug medication.

Contraindications

1. Patients with history of allergy to a particular antimicrobial agent.
2. In pregnancy and lactating periods.
3. Children under the age of 12 years.
4. Patients with complete renal failure.
5. Patients susceptible to infective endocarditis.

Advantages

1. High concentration in subgingival sites.
2. Independent of patient compliance.
3. Does not harm the symbiotic useful microflora of gastrointestinal tract.
4. Systemic intolerance is bypassed.

Disadvantages

1. Difficulty in placing of therapeutic concentrations of the antimicrobial agents in deeper sites.
2. Has to be professionally placed or if manually placed, requires manual dexterity and patient compliance.
3. Complete drug penetration is not possible and extra pocket sites are unaffected.

LOCAL ADMINISTRATION OF ANTIBIOTICS AND ANTIMICROBIAL AGENTS (FIG. 52.2)

I. Vehicles for local delivery of chemotherapeutic agents include: Dentifrices, mouth rinses, chewing gum and slow release devices.

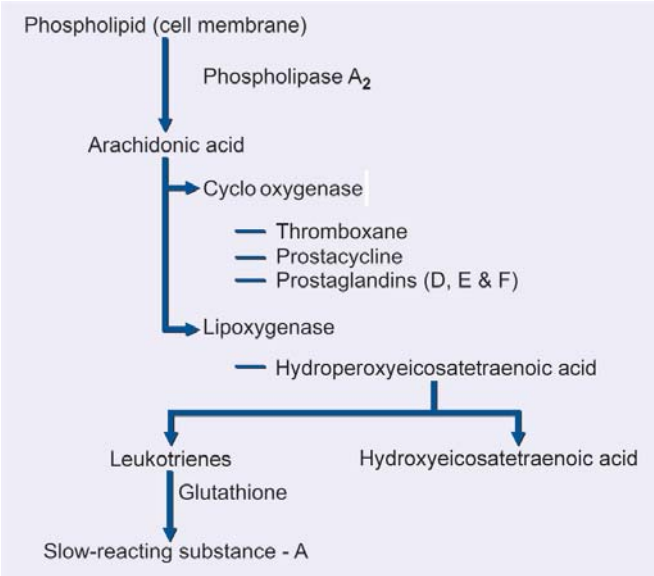

Fig. 52.2: Intrasulcular placement of tetracycline fibers

II. Methods of delivery of chemotherapeutic agents include: Root biomodification (direct professional application to the root surface during surgery), Keyes technique, irrigation methods include home and professional drug delivery.

Vehicles for Local Delivery

Dentifrices, mouth rinses and chewing gum are inefficient delivery systems in periodontics because they fail to direct drugs into the periodontal pocket. They can be useful in reducing inflammation associated with gingivitis but they do not penetrate well into the periodontal pockets.

Slow-release Devices/Controlled-Release Delivery Systems

They are designed to release chemotherapeutic agents into the periodontal pocket over an extended period of time, hence, they can achieve high levels of concentration at sites where they are precisely needed.

Classification of Controlled-Release Local Delivery Systems

i. *Reservoirs without a rate-controlling system*, e.g. hollow fibers, gels and dialysis tubing (effective only for 24 hours).

ii. *Reservoirs with a rate-controlling system*, e.g. polymeric matrices, polymer membranes, monolithic matrices and coated particles (effective for more than 24 hours).

EVA system (Ethylene vinyl acetate) is based on polymer technology with tetracycline dispersed within a solid (monolithic) polymer of EVA. A formulation of 25 percent tetracycline in EVA (Actisite) has been developed as 0.5 mm nonbiodegradable fiber and has been approved by the Food and Drug Administration (FDA).

The use of slow release devices placed in deep periodontal defects, in conjunction with scaling and root planing may offer promising results in the management of such defects.

Methods of Delivery of Chemotherapeutic Agents

a. Keyes technique.
b. Root biomodification.
c. Irrigation devices.
    i. Home irrigation devices
        - Supragingival home irrigation devices
        - Subgingival home irrigation devices
        - Marginal home irrigation devices
    ii. Professional subgingival irrigation.

Keyes Technique

Involves application, by tooth brushing, of a slurry of sodium bicarbonate and hydrogen peroxide for the control of plaque microorganisms. Various studies have proved that minimal clinical benefit can be expected from this technique simply because tooth brushing offers an ineffective means of delivering medicaments into the periodontal pocket.

Root Biomodification

Application of various medicaments to root surfaces during surgical therapy has been evaluated. These agents include tetracycline, doxycycline, citric acid and fibronectin. Application of these agents on diseased root surfaces during surgery may enhance connective tissue attachment to the roots, although available data are inconclusive (discussed elsewhere in this book).

Home Irrigation Devices

The advantage of home irrigation device is, it allows the patient to deliver medicaments into the periodontal pocket at home on a more frequent basis than in practice with professional subgingival irrigation. The limiting factors for these devices are inability to access the depth of periodontal pocket and manual dexterity of the patient.

i. *Supragingival home irrigation devices*: Results in greater access to periodontal pockets than mouth rinsing alone. The depth of penetration of medicament is a maximum penetration of 4 to 5 mm. Therefore, these devices may be useful in delivering medicaments in cases of gingivitis with shallow pocket depths, but they are less useful in delivering medicaments in periodontitis patients with deeper pockets.
ii. *Subgingival home irrigation devices*: Generally includes a blunt ended metal cannula that the patient inserts into the periodontal pocket. It increases the depth of penetration of fluid, but largely depends on the manual dexterity of the patient and has the potential for injury owing to the metal tip.
iii. *Marginal home irrigation devices*: These are a hybrid of both supragingival and subgingival home irrigation. It has a jet tip attachment that has been modified so that it can deliver fluids subgingivally. Home irrigation devices allow penetration of fluid up to 90 percent in pockets of 6 mm depth and lesser penetration in pockets with deeper depths. The irrigation is more effective (supra/subgingival) if it is pulsed rather than constant.

Professional Subgingival Irrigation

It includes the use of a wide array of powered and manually operated irrigators. Irrigation using a syringe with a blunt needle has been used. The most commonly used solutions are chlorhexidine gluconate, stannous fluoride, tetracycline, metronidazole and hydrogen peroxide. Irrigation with medicaments performed in conjunction with root planing provide little, if any, additional benefit over root planing alone.

CURRENTLY AVAILABLE LOCAL DRUG DELIVERY SYSTEMS IN PERIODONTAL THERAPY

Antimicrobial agent	*Product name*	*Nature*	*Dosage form*
Doxycycline	Atridox®	Biodegradable	Mixture in syringe
Tetracycline	Actisite™	Nonresorbable	Fibers
Metronidazole	Elyzol	Biodegradable	Mixture in syringe
Minocycline	Arestin Dentamycin Periocline	Biodegradable	Mixture in syringe
Chlorhexidine	Periochip	Biodegradable	Chip device

Frequently used Local Antimicrobials with Dosage Specification

Drug	*Dosage*	*Release period in days*
Actisite™	12.7 mg/9 inch fiber of diameter .5 mm	10
Atridox®	10%	
Periochip	2.5 mg of chlorhexidine gluconate in (4 × 5 ×.35 mm) chip.	7

CONCLUSION

Considering various factors of cost effectiveness, efficacy, potential advantages and disadvantages it can be concluded that local drug delivery could serve as a potent weapon in periodontal chemotherapy and at the same time a valuable adjunct to mechanotherapy, but its use as a monotherapy agent is still a topic of debate.

REVIEW QUESTIONS

1. Classify antimicrobial agents, write in detail on role of tetracyclines in periodontal therapy.
2. What are the advantages and disadvantages of systemic antibiotics?
3. Classify local drug delivery systems.

KEYPOINTS

1. Mechanical removal of tooth deposits is the foundation of periodontal therapy. Since there is a possibility of bacterial invasion of periodontal tissues in cases of aggressive forms of periodontitis, systemic antibiotic therapy was thought to be a valuable adjunct to mechanical therapy.
2. Best results can be obtained by antibiotic therapy combined with scaling and root planing.
3. Tetracyclines are bacteriostatic antibiotics which provides a broad spectrum of activity against both gram-positive and gram-negative organisms. They are most commonly used in the management of periodontal disease because the GCF (gingival crevicular fluid) concentration can be 5 to 10 times more than the serum.
4. Apart from antimicrobial activity, it also has other important properties like collagenase inhibition, anti-inflammatory action, inhibition of bone resorption, etc. Other drugs that are commonly used in periodontal therapy are metronidazole, penicillins and quinolones.
5. Prolonged antibiotic therapy can cause adverse effects like:
   a. Development of resistant organisms.
   b. Hypersensitivity reaction.
   c. Drug interactions.
   d. Superinfection.
   e. Dependency on patients compliance to take the drug regularly.
   f. Though periodontal disease involves only small area of the body, due to systemic administration of the drug the whole body gets unnecessarily loaded with the drug.
6. In order to overcome all the adverse effects of systemic administration, local drug delivery system was introduced in periodontics.

KNOW MORE ...

Biological Implications of Systemic Antibiotic Therapy in the Treatment of Periodontal Diseases

1. The putative periodontal pathogens tend to reside in the inaccessible areas, which cannot be eliminated by the mechanical treatment.
2. They may also evade mechanical treatment due to their ability to penetrate periodontal tissues or dentinal tubules.
3. Treated sites may be recolonized by periodontal pathogens residing in nondental areas such as the dorsum of tongue or tonsils.

An Ideal Antimicrobial Agent should Fulfill a Number of Criteria/Conditions

a. The drug must demonstrate (*in vitro*) high antibacterial activity against the prominent periodontopathic organisms.

b. It should demonstrate a sufficient drug dose in the sub-gingival environment to kill target organisms.
c. Drug should not elicit any major local or systemic side effects.
d. It should be able to maintain the required concentration of the drug over a long enough period to cause sufficient damage to the microbiota responsible for periodontal infections.
e. Finally, it should have better outcome, less adverse effects, simpler to perform, cheaper and faster.

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53 Chapter

Questionnaire for Clinical Case Discussion

QUESTIONS

1. What is the significance of recording chief complaint, history of present illness, past dental history, family history and medical history?
2. What are the effects of bruxism, mouth-breathing, tongue-thrusting, and cigarette smoking on the periodontium? How do you diagnose the above conditions?
3. What are the various methods of plaque control?
4. What are the various brushing techniques and its advantages and disadvantages?
5. What is the difference between Bass method and modified Bass method?
6. Describe and demonstrate the modified Bass method.
7. What are the various interdental aids? Which is used where? Discuss the criteria for its selection.
8. In which conditions do you see lymph node pathology?
9. What is the normal color of the gingiva and what are the factors responsible for maintaining the normal color of gingiva?
10. What are the conditions and factors responsible for change in gingival color?
11. Describe normal contour and factors responsible for it. What changes in contour are seen in disease?
12. Name some diseases in which gingival contour are changed.
13. Describe size of gingiva in health and disease. Name some factors responsible for normal size and enlarged gingiva.
14. How do you check for normal consistency of the gingiva? In disease condition what are the changes that take place in consistency of the gingiva?
15. What is the significance of stippling? How is normal stippling manifested?
16. What is the normal position of the gingiva?
17. Define gingival recession and describe its etiology, classification, clinical significance and treatment.
18. What is the significance of bleeding on probing? How do you check for gingival bleeding?
19. What are the etiological factors responsible for gingival bleeding?
20. What are the microscopic changes associated with gingival bleeding?
21. What is the width of attached gingiva? How do you measure the width of attached gingiva?
22. What are the tests done to identify whether the width of attached gingiva is adequate or inadequate?
23. What are the mucogingival problems?
24. How do you clinically differentiate between true, pseudo, and combined pocket; supra bony and infra-bony pocket?
25. How do you treat pockets?
26. Describe the sequelae following unreplaced first molars.

27. Describe the sequelae following loss of proximal contact form.
28. How do you diagnose furcation involvement?
29. Describe the etiology, classification and treatment of furcation involvement.
30. What are the causes for tooth mobility? Give the classification of mobility.
31. What is pathological migration? How do you clinically identify it?
32. What are the causes for pathological migration?
33. Define attrition, abrasion and erosion.
34. When is a tooth tender on percussion.
35. What is retrograde periodontitis?
36. How do you diagnose a case of trauma form occlusion?
37. What are the types, signs and symptoms of trauma from occlusion?
38. What is the difference between mobility and fremitus?
39. What are the various indices used for accessing oral hygiene?
40. What are the indices for gingival bleeding?
41. Effects of over-hanging restorations on periodontium.
42. Importance of recording various lab investigations.
43. Effects of improper orthodontic treatment on periodontium.
44. What are the things you look for in a radiograph?
45. What are the advantages and disadvantages of intraoral periapical radiographs?
46. Justify your diagnosis of the case. If diagnosis is gingivitis then why? Or if diagnosis is periodontitis then why?
47. Define prognosis. What are factors responsible for individual and overall tooth prognosis?
48. Discuss the treatment plan in a sequential order.

QUESTIONS RELATED TO PERIODONTAL INSTRUMENTS AND TREATMENT

49. Classify periodontal instruments.
50. Give the characteristics of a scaler and curette with differences.
51. Differences between Universal and Gracey curette.
52. Classify various supragingival and subgingival scalers.
53. What are the instruments used for root planing?
54. Define scaling and root planing.
55. What are the principles of periodontal instrumentation?
56. What are finger rests and grasps?
57. Different angulations needed for scaling procedures.
58. Recent advances in periodontal instrumentation?

QUESTIONS AND ANSWERS

Q.1. What is the significance of recording chief complaint, history of present illness, past dental history, family history and medical history?

Answer

Chief complaint: It is the patient's description of the symptoms related to the disease for which treatment is being sought (to be recorded in patient's own words). It is often recorded as patient's desire and when the patient is questioned about the principal reason for his/her dental visit, his/her desire is frequently expressed in the form of cleaning, filling, etc. This should not be recorded as chief complaint. The dentist has to further derive into for the actual complaint from the patient with questions like why he/she desires that particular treatment, because most of the times in periodontics we deal with chronic problems, hence they do not actually have any complaint.

History of present illness: It is the chronological account of the problem indicated by the chief complaint. It comprises the onset, duration, any relieving or aggravating factors of the illness which best describe the disease present and helps in determining the diagnosis and treatment plan. Taking the detailed history of the illness of the patient enables the operator to be as confident as possible that the patient will not be endangered during treatment.

Past dental history: It consists of patient's previous experience of oral diseases and his treatment for those diseases. It should acquaint the dentist with the patient's previous dental treatment, professional and home care procedures, frequency and nature of periodontal care, reason for prior extraction of teeth, history of prior dental and periodontal disease and bleeding and anesthetic problem, drug allergies during previous dental procedures. All these

data aid in the development of diagnosis and prognosis and may have a significant influence on the plan of treatment.

Family history: Many diseases such as idiopathic gingival fibromatosis, hemophilia are inherited. Thus family history should record details of condition existing in other members of the family.

Medical History

The objectives are:

1. To determine systemic factors or diseases that will require special consideration prior to, during or after periodontal therapy.
2. It aids the clinician in the diagnosis of oral manifestation of systemic disease and in the detection of systemic condition that may affect the periodontal tissue response to local factors or that require special precaution and modification in treatment procedure.
3. To identify the patients who are taking drugs or medication that could adversely interact with drugs prescribed, that would complicate dental therapy or they may serve as a clue to an underlying systemic disease, the patient has failed to mention.
4. To provide information for the dentist to modify the treatment plan for the patient in light of any systemic diseases or potential drug interaction.
5. To help establish a good patient-doctor relationship.

Q.2. What are the effects of bruxism, mouthbreathing, tongue-thrusting, and cigarette-smoking on the periodontium? How do you diagnose the above conditions?

Answer

Mouthbreathing

It leads to localized gingival inflammation that is usually confined to the labial gingiva of the maxillary anterior teeth. The tissue becomes reddened and swollen and it bleeds easily. The surface of the gingiva is shiny. Gingival changes associated with mouthbreathing are:

a. Localized gingival inflammation in the maxillary anterior region.
b. Crowding of teeth with gingivitis.

Diagnosis

It can be made by simple tests:

1. *Mirror test:* A double side mirror is held between the nose and the mouth. Fogging on the nasal side of the mirror indicates nasal-breathing while fogging towards the oral side indicates oral breathing.
2. *Cotton test:* A butterfly-shaped piece of cotton is placed over the upper lip below the nostrils. If the cotton flutters down it indicates nasal breathing.
3. *Water test:* The patient is asked to fill his mouth with water and retain it for a period of time. While nasal breathers accomplish this with ease, mouth-breathers find the task difficult.

Tonguethrusting

It entails persistent, forceful wedging of the tongue against the teeth, particularly in the anterior region. It causes excessive lateral pressure, which maybe traumatic to the periodontium. It also causes spreading and tilting of the anterior teeth with an open bite anteriorly, posteriorly or in the premolar area. It causes labial drifting of teeth, thereby the antagonism between forces that direct the tooth labially and inward pressure from lip may lead to tooth mobility. The altered inclination causes accumulation of food debris. There is loss of proximal contact, which leads to food impaction. It is also an important contributing factor in pathologic tooth migration.

Tongue-thrusting can be diagnosed by its position. Normally, it is placed against the palate with the tip behind the maxillary teeth during swallowing. During tongue-thrusting it is placed behind the mandibular teeth. It can be diagnosed by altered inclination of the teeth, spacing between the teeth and marked open bite.

Cigarette Smoking

Smoking has a detrimental effect on the progression of periodontal disease and healing after periodontal therapy. The following are its effects:

1. Brownish, tar-like deposits and discoloration of tooth surface.
2. Diffuse grayish discoloration and leukoplakia of the gingiva may occur.

3. *Smoker's Palate characterized* by prominent mucous glands due to inflammation of orifices of mucous glands and a wrinkled, cobblestone surface may occur.
4. Postsurgical healing is delayed.
5. Produces an immediate transient but marked increase in gingival fluid flow, as a result of blood changes induced by nicotine.
6. More severe gingivitis and periodontitis. More calculus has been reported in smokers.
7. Factors responsible for increased periodontal problems in smokers:
   a. Increased keratinized cells in gingiva.
   b. Nicotine metabolites have been found.
   c. Oral polymorphonuclear leukocytes have reduced phagocytosis.
   d. Vascular reaction is suppressed in smokers.

Diagnosis can be made by the presence of excessive black or brown stains on the tooth surface, discoloration of the gingiva and heavy amount of calculus is also found.

Bruxism

Bruxism is clenching or grinding of the teeth when the individual is not chewing or swallowing. It has been estimated that during clenching or grinding the individual might impose a load of over 20 kg on a tooth over a period of 2.5 seconds each time. This is far in excess of normal functional stresses and causes increased flow within the viscoelastic periodontal ligament and distortion of alveolar bone. It also causes attrition of teeth.

Clinical Features

1. Advanced attrition, presence of wear facets.
2. Increased tooth mobility patterns which do not commensurate with the amount of attachment loss or degree of gingival inflammation.
3. Widened periodontal ligament spaces are seen in radiographs.
4. Hypertonicity of the muscles of mastication.
5. Temporomandibular joint discomfort.

Diagnosis

History and clinical examination in most cases is sufficient to diagnose bruxism. Occlusal prematurities can be diagnosed by use of articulating papers. Electromyographic examination can be carried out to check for hyperactivity of the muscles of mastication.

Q.3. What are the various methods of plaque control?

Answer

Plaque control is the removal of microbial plaque and prevention of its formation on teeth. Basically there are two types of plaque control.

Mechanical Plaque Control

a. Toothbrushes
b. Powered toothbrushes
c. Interdental cleansing aids
   - Interdental brushes
   - Single tufted brushes
   - Dental floss
d. Wooden tips
e. Gum stimulator
f. Oral irrigation devices
g. Dentifrices

Chemical Plaque Control

The following chemicals are available in varying concentrations:

a. Chlorhexidine gluconate 0.2 percent (Hexidine®, Clohex®) and 0.12 percent (Periogard®)
b. Phenolic compounds (Listerine®) containing,
   - Thymol – 0.06 percent
   - Eucalyptol – 0.09 percent
   - Benzoic acid – 0.15 percent
   - Menthol – 0.04 percent
   - Acetyl salicylate
c. Quaternary ammonium compounds (Cetyl pyridium chloride) commercially available as Reach®.

All the above mentioned chemicals are used only as adjunct to mechanical plaque control.

Q.4. What are the various brushing techniques and its advantages and disadvantages?

Answer

Many methods of toothbrushing have been described, but studies evaluating the effectiveness of the most common brushing techniques have shown that no one method is clearly superior. The best method is the one that suits the individual's needs and abilities and the responsibility of the dental professional is to instruct the patient as to how to perform the task thoroughly.

Various techniques are:

Scrub Technique

Advantage

Very easy to learn and master the technique.

Disadvantages

1. Inefficient plaque removal
2. Causes gingival trauma and recession
3. Causes cervical abrasion.

Roll Technique

Disadvantages

1. Difficult to learn and requires sufficient dexterity.
2. Poor plaque removal from the sulcus area.
3. Poor plaque removal from the gingival one-third of posterior teeth because of the contour of the teeth.

Bass Method

Advantages

1. The short back and forth motion is easy to master because it requires the same simple movement, familiar to most patients who were using scrub technique.
2. It cleans the cervical and interproximal portions of teeth.

Disadvantages

1. It is time consuming because different areas of teeth have to be brushed.
2. Patient might miss the gingival sulcus and may place the brush on the attached gingiva.
3. It only dislodges the plaque in interproximal areas but does not remove them.

Modified Bass Method

Advantages

1. The short back and forth motion is easy to master because it requires the same simple movement familiar to most patients who were using the scrub techniques.
2. It dislodges and removes plaque from cervical and interproximal portions of the teeth.

Disadvantage

It is time consuming because different areas of teeth need to be brushed.

Modified Stillman Method

Advantages

1. The gingiva is mechanically stimulated.
2. The gingival third of the tooth is contacted with a short vibratory motion and plaque is removed between the gingival margin and the exposed area of tooth surface. Hence, advised only in areas of gingival recession.
3. The tips of the bristles tend to reach the interproximal areas to clean and stimulate the interdental papilla without injury.

Disadvantages

1. Patient might miss the gingiva and cervical areas of the teeth, thus, leaving behind plaque.
2. Patient might not apply sufficient pressure to produce blanching of tissue.

Charter's Technique

Advantages

1. In this method plaque is removed gently, without much pressure.
2. It massages the gingiva and encourages healing, hence advised only during postsurgical period.

Disadvantage

Poor plaque removal when indicated in routine patients with or without periodontal involvement.

Q.5. What is the difference between Bass method and modified Bass method?

Answer

Bass method	*Modified Bass method*
Technique: The bristles are directed apically into the gingival sulcus at 45° to the long axis of the tooth and are activated by applying gentle pressure in an apical direction and by making short vibratory strokes.	The modification consists of sweeping the bristles towards the occlusal surface after completing the vibratory motion in gingival sulcus.
Efficiency: It is efficient in removing dental plaque from the gingival third of the tooth and from shallow gingival sulcus.	It is efficient in removing dental plaque from the gingival sulcus and interdental areas.

Q.6. Describe and demonstrate the modified Bass method.

Answer

It is also known as intrasulcus cleansing or intrasulcular method.

Technique: Here the patient is asked to place the head of a soft brush parallel with the occlusal plane, beginning with the most distal tooth in the arch covering 3 to 4 teeth at a time. The patient is then asked to place the bristles at the gingival margin, creating an angle of 45 degree to the long axis of the teeth and exerting gentle vibratory pressure, using short back and forth motion, without dislodging the tips of the bristles. After completing such 20 strokes in the same position, the head of the brush is tilted and moved occlusally. This is repeated for 4-5 times. Patient is instructed to continue brushing around the arch, 3 teeth at a time, both facially and lingually. The brush is inserted vertically to reach the lingual surfaces of the anterior teeth. The pits and fissures of the occlusal surfaces are brushed with 20 short, back and forth movements.

Advantages

1. It removes plaque from the sulcus and other areas of tooth.
2. It aids in gingival massage.

Disadvantage

It is hard to master.

Q.7. What are the various interdental aids? Which is used where? Discuss the criteria for its selection.

Answer

Various interdental aids are:

- Dental floss
- Interdental brushes
- Gauze strips
- Unitufted brushes
- Toothpicks

Criteria for selection of these aids depends upon the type of embrasure:

Type I: Interdental papilla completely fills the embrasure space. Dental floss can be used.

Type II: Mild loss of interdental papilla due to slight spacing between the teeth. Miniature bottlebrushes can be advised, e.g. Proxabrushes.

Type III: No proximal contact between the teeth, like diastema where there is no interdental papilla. Unitufted brushes are advised.

Dental floss: It is used for cleaning in narrow gingival embrasures that are occupied by intact papillae and bordered by tight contact zones. It is the most effective dental hygiene aid.

Interdental brushes: It is used in areas of moderate papillary recession, proximal tooth surfaces adjacent to open embrasures, orthodontic appliances, fixed prosthesis, dental implant and other areas that are hard to reach with regular tooth brush. It is also used in concave proximal areas and exposed class IV furcation.

Gauze strip: It is used for proximal surface of widely spaced teeth, for surface of teeth next to edentulous spaces and fixed appliances.

Unitufted brush: It is used for inaccessible areas such as lingual surface of the mandibular molars, abutment teeth, distal surface of the most posterior teeth, crowded teeth, open interproximal areas and isolated areas of deep recession. It is also indicated in fixed dental prosthesis.

Toothpicks: It is used for plaque removal at or just underneath gingival margin, for concave proximal tooth surface and for exposed furcation areas.

Q.8. In which conditions do you see lymph node pathology?

Answer

Various systemic diseases contribute for lymph node enlargements, which are as follows:

i. Syphilis
ii. Tuberculosis
iii. Acute bacterial infections
iv. Fungal/parasitic infections
v. Malignant tumor metastasis from nasopharynx, oral cavity, pharynx, thyroid
vi. Lymphatic diseases such as Hodgkin's disease and lymphatic lymphoma
vii. Leukemia's such as chronic lymphatic, chronic granulocytic leukemia
viii. Endocrinal disorders such as hyperthyroidism, hypopituitarism.

Common conditions of oral cavity, which cause cervical lymphadenopathy are:

i. Acute inflammatory conditions such as periodontal/periapical abscess, acute necrotizing ulcerative gingivitis, acute herpetic gingivostomatitis, aphthous ulcers and others.
ii. Oral manifestations of systemic diseases.
iii. Malignant conditions: Secondary carcinoma such as epithelioma of tongue, lip, cheek and face.

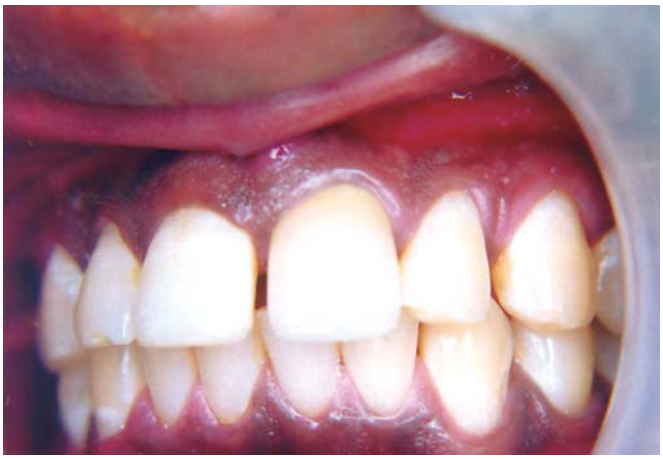


Fig. 53.1: Color of the gingiva in health

Q.9. What is the normal color of the gingiva and what are the factors responsible for maintaining the normal color of gingiva?

Answer

Normal color of gingiva is coral pink (Fig. 53.1)

Factors responsible are:

- Vascular supply
- Thickness and degree of keratinization of epithelium
- Presence of pigment containing cells.

Q.10. What are the conditions and factors responsible for change in gingival color (Fig. 53.2)?

No. Disease condition	*Color change/ clinical change*	*Factors responsible*
1. Chronic gingivitis of red, reddish-blue, deep-blue	Varying shades	1. Vascular proliferation (erythematous) 2. Reduction of keratinization owing to epithelial compression by inflamed tissue 3. Venous stasis 4. Tissue necrosis
2. Acute gingivitis a. NUG/HV b. Herpetic gingivostomatitis c. Chemical irritation	Bright red erythematous Shiny-slate gray Dull-whitish gray	Same as above

3.	Bismuth, arsenic, mercury pigmentation	Black line following the contour of marginal gingival	Perivascular precipitation of metallic sulfides in subepithelial connective tissue seen only in areas of inflammation due to increased permeability of blood vessels
4.	Lead pigmentation	Bluish-red or deep-blue linear pigmentation (Burtonian line)	Perivascular precipitation of metallic sulfides
5.	Silver pigmentation	Violet marginal line	Perivascular precipitation of metallic sulfides.

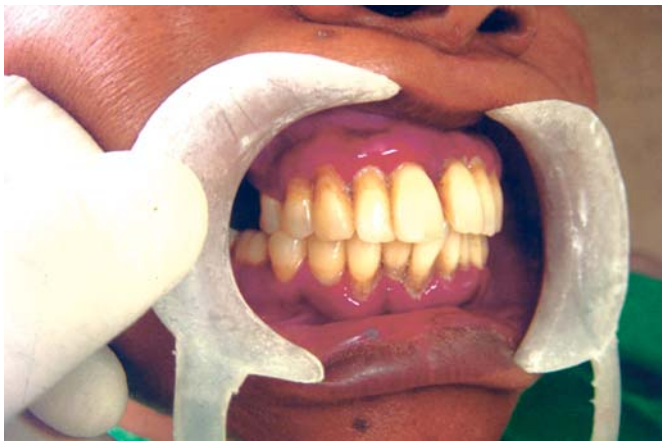


Fig. 53.2: Color of the gingiva in disease. (Note the marginal erythema)

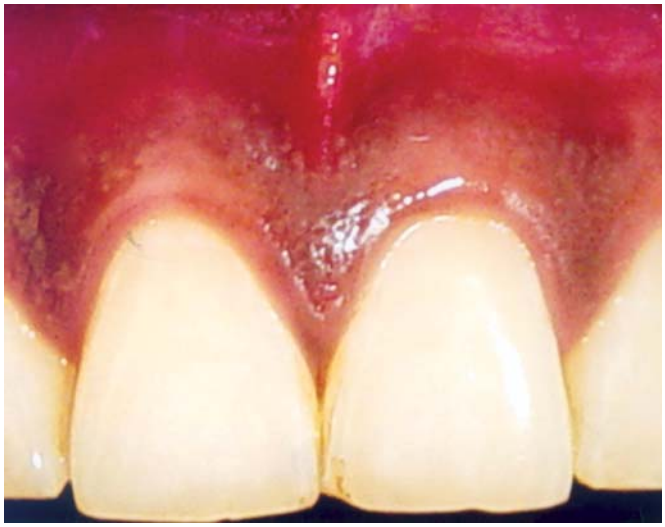


Fig. 53.3: Contour of the gingiva in health

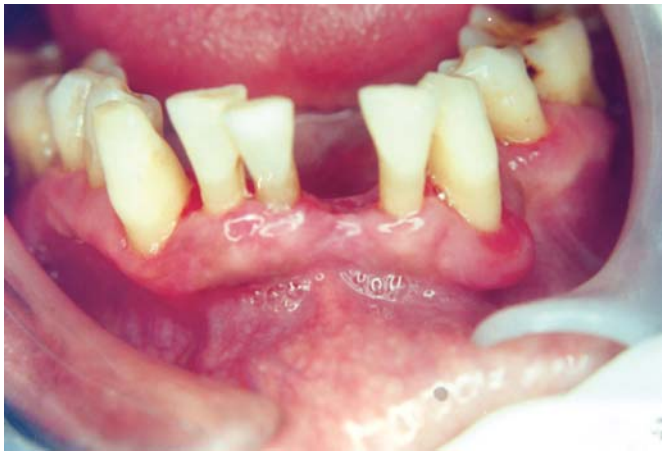


Fig. 53.4: Contour of the gingiva in disease

Q.11. Describe the normal contour and factors responsible for it. What changes in contour are seen in disease?

Answer

The normal contour of the marginal gingiva is scalloped and knife edged, whereas the interdental papilla, in the anterior region is pyramidal in shape and posteriorly it is tent-shaped (Figs 53.3 and 53.4).

The factors responsible for the contour of gingiva are:

- Shape of teeth and their alignment in the arch.
- Location and size of proximal contact.
- Dimensions of facial and lingual gingival embrasures.

During disease process:

1. The marginal gingiva becomes rolled or rounded and interdental papilla becomes blunt and flat, e.g. chronic gingivitis
2. Punched out crater-like depressions at the crest of interdental papilla extending to the marginal gingiva is seen in necrotizing ulcerative gingivitis (NUG).
3. Irregularly-shaped denuded appearance of the gingiva, seen in chronic desquamative gingivitis.
4. Exaggerated scalloping of the gingiva as seen in gingival recession.
5. Apostrophe shaped indentation extending from the gingival margin to varying depths on the facial gingival surfaces are called as Stillman's clefts.

6. Lifesaver like enlargement on the marginal gingival, most commonly on the canine and premolar facial surface called as McCall's festoons.

Q.12. Name some diseases in which gingival contour is changed.

Answer

Gingival contour is changed in conditions like:

- Gingival recession
- Gingival enlargement
- Acute and chronic gingivitis
- Stillman's clefts and McCall's festoons.

Q.13. Describe size of gingiva in health and disease. Name some factors responsible for normal size and enlarged gingiva.

Answer

Normal

The size of gingiva corresponds to sum of total bulk of cellular and intercellular elements and their vascular supply. Alteration in size is a common feature of gingival disease (Figs 53.5 to 53.7).

Change in disease: Size of gingiva is enlarged and is called as *gingival enlargement*. It maybe *inflammatory* or *noninflammatory*. In inflammatory, there is increase in cells and decrease in fibers. In noninflammatory there is increase in fibers and decrease in cells.

Hypertrophy constitutes an increase in size of cells and corresponding increase in the size of the organ.

Hyperplasia constitutes increase in number of cells in an organ or tissue there by contributing to an overall increase in organ size.

Types of gingival enlargement

i. Inflammatory enlargement
   - Chronic
   - Acute

ii. Fibrotic enlargement
   - Drug-induced
   - Idiopathic

iii. Combined enlargement

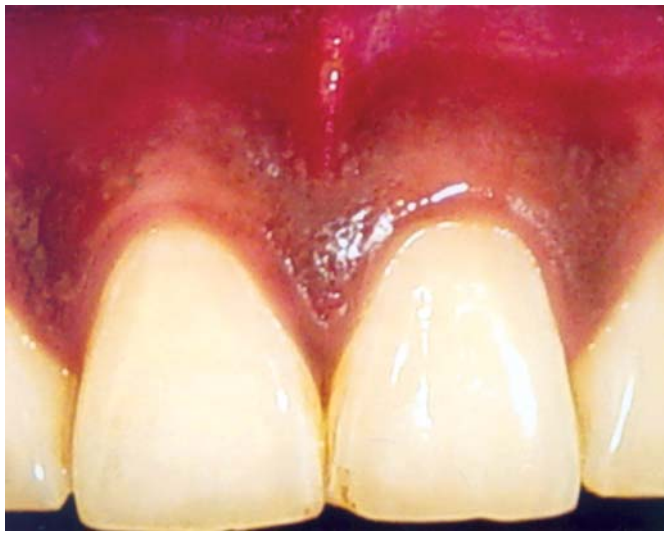


Fig. 53.5: Size of the gingiva in health

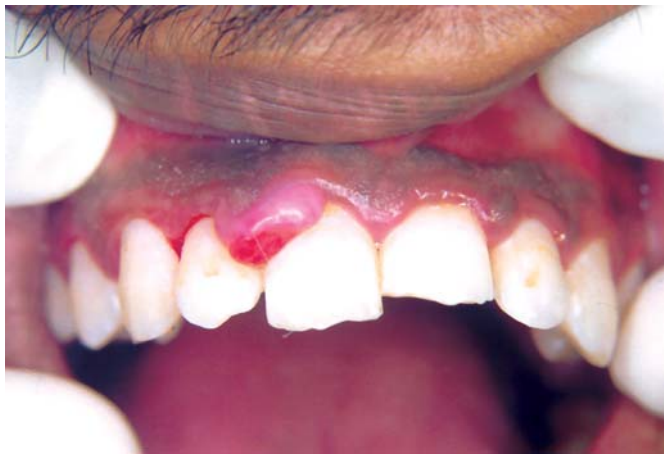


Fig. 53.6: Inflammatory gingival enlargement

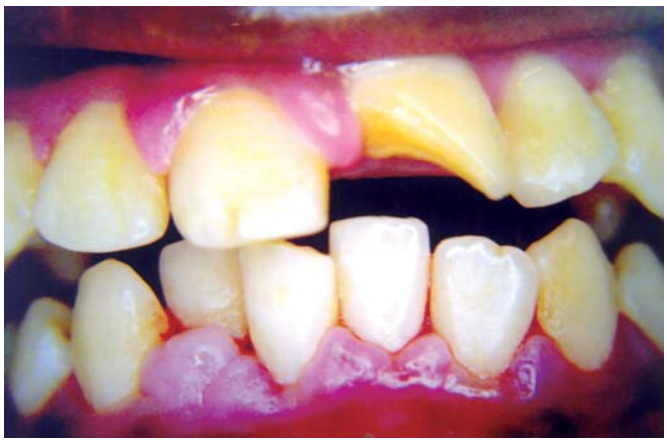


Fig. 53.7: Fibrotic gingival enlargement

iv. Enlargement associated with systemic diseases or conditions
   - Pregnancy
   - Puberty
   - Vitamin C deficiency
   - Plasma cell gingivitis
   - Nonspecific conditioned enlargement
   - Leukemia
   - Granulomatous disease

v. Neoplastic enlargement
   - Benign
   - Malignant

vi. False enlargement

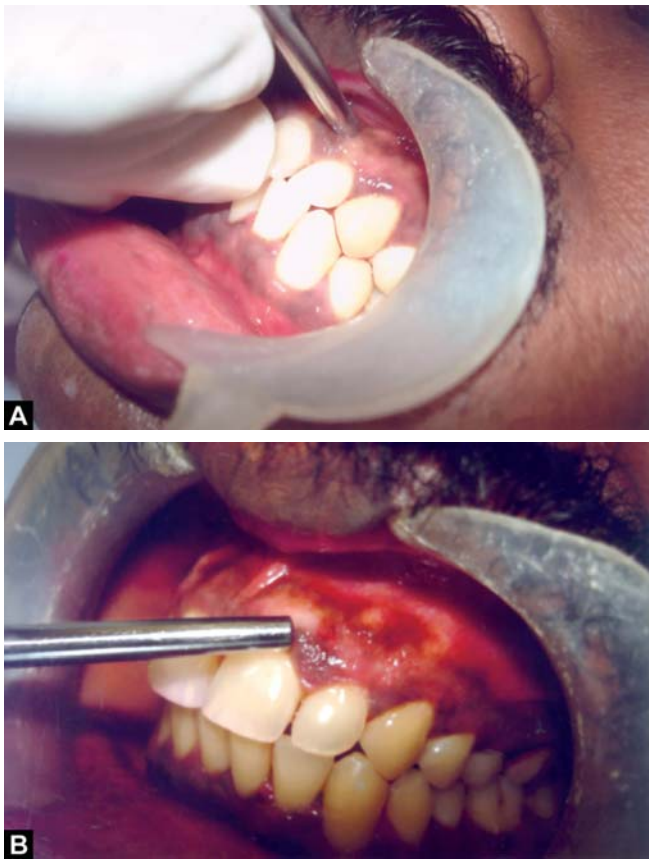
Q.14. How do you check for normal consistency of gingiva? In disease what are the changes that take place in consistency?

Answer

Clinical and histopathologic changes in gingival consistency in disease (Figs 53.8A and B).

Clinical changes	*Microscopic changes*
Chronic gingivitis	
1. Soggy puffiness that pits on pressure	Infiltration by fluid and cells of inflammatory exudates.
2. Marked softness and friability with ready fragmentation and pinpoint surface areas of redness and desquamation	Degeneration of connective tissue and epithelium, thinning of epithelium, edema and leukocytic invasion.
3. Firm, leathery consistency.	Fibrotic and epithelial proliferation associated with longstanding chronic inflammation.
Acute gingivitis	
1. Diffuse puffiness and softening	Diffuse edema, fatty infiltration.
2. Sloughing with grayish flake like particles of debris adhering to eroded surface	Necrosis with formation of a pseudomembrane and degenerated epithelial cells in a fibrous mesh work. Inter and intracellular edema with degeneration of nucleus and cytoplasm and rupture of cell wall.
3. Vesicle formation	

The normal consistency of the gingiva can be checked by palpation either with a blunt instrument or digital pressure. Pitting on palpation indicates soft and edematous gingiva.



Figs 53.8A and B: Clinical demonstration for checking. (A) Normal consistency, (B) Blanched gingiva

Q.15. What is the significance of stippling? How is normal stippling manifested?

Answer

Stippling is a form of adaptive specialization or reinforcement for function. It is a feature of healthy gingiva and reduction and loss of stippling is a common sign of gingival disease (Fig. 53.9).

Stippling is produced by alternate rounded protruberances and depressions in the gingival surface. Papillary layer of the connective tissue projects into the elevations, and the elevated and depressed areas are covered by stratified squamous epithelium. Stippling is best viewed by drying the gingiva.

Q.16. What is the normal position of the gingiva?

Answer

The normal position of gingiva is 1 millimeter above the cementoenamel junction.

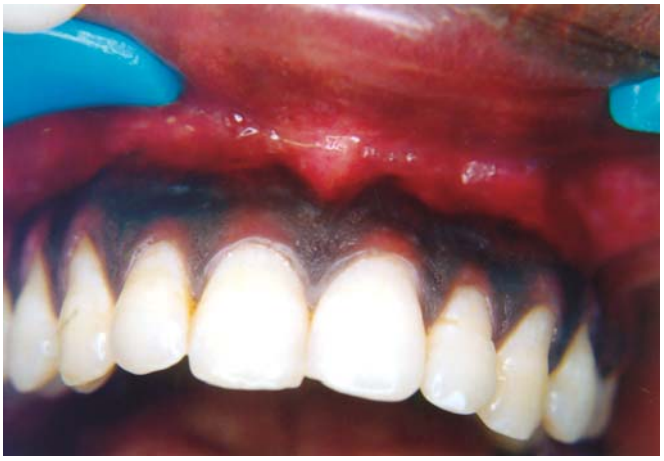


Fig. 53.9: Stippling in normal healthy gingiva

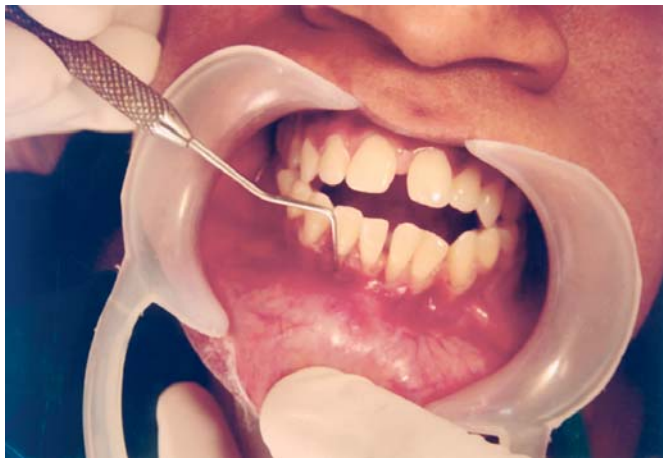


Fig. 53.10: Gingival recession

Physiologic factors responsible for normal position of gingiva are:

1. Position of teeth in the arch.
2. Root bone angle
3. Mesiodistal curvature of tooth surface.

Pathologic factors responsible for change in gingival position are:

1. Toothbrush trauma
2. Gingival inflammation
3. High frenal attachment
4. Tooth malposition
5. Friction from soft tissue

Q.17. Define gingival recession and describe the etiology, classification, clinical significance and its treatment.

Answer

Gingival recession is defined as apical shift of the gingival margin to a position apical to the CEJ, with exposure of root surface to the oral cavity (Fig. 53.10).

Etiology

Mainly three factors are responsible:

a. *Inflammatory*: Plaque induced inflammatory periodontal diseases, toothbrush injury, and surgical treatment of inflammatory periodontal disease.

b. *Anatomic factors*: Developmental anatomic abnormalities, (dehiscences, thin bony plates, high frenum attachments) malocclusion.

c. *Iatrogenic factors*:
   - Deleterious habits (Pressure of foreign objects like finger nails, pencils, hairpins).
   - Clasps and mandibular oral denture bars or aprons in partial dentures.
   - Prolonged orthodontic treatment, improper restorations.

Classification

I. Sullivan and Atkins classified gingival recession as:
   a. Shallow narrow
   b. Shallow wide
   c. Deep narrow
   d. Deep wide

II. Millers classification

Class I: Includes marginal tissue recession that does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This type of recession can be narrow or wide (group a and b in Sullivan and Atkins classification).

Class II: Marginal tissue recession that extends to or beyond the mucogingival junction. There is no loss of bone or soft tissue in the interdental area. This type of recession can be classified into wide and narrow (group c and d of Sullivan and Atkins classification).

Class III: Marginal tissue recession that extends to or beyond the mucogingival junction, in addition there is bone and or soft tissue loss interdentally or malpositioning of the tooth.

Class IV: Marginal tissue recession that extends to or beyond the mucogingival junction, with severe bone loss and soft tissue loss interdentally and or severe tooth-malpositioning.

Clinical Significance

Exposed root surfaces are susceptible to *caries*. Wearing away of the cementum exposed by recession leaves an underlying dentinal surface that is *extremely sensitive*. Hyperemia of the pulp and associated symptoms may also result from exposure of the root surface. Interproximal recession creates spaces in which, plaque and food debris can accumulate.

Treatment

Denuded root surfaces are covered for two purposes:

1. To solve esthetic problem (in anterior teeth)
2. To widen zone of attached gingiva, thereby solving a possible mucogingival problem

Several procedures are as follows:

I. **Treatment based on width of attached gingiva**

Adequate	*Inadequate*
1. Pedicle graft • Double papillae • Laterally-displaced	1. Free soft-tissue autograft.
2. Coronally, repositioned flap with semilunar incision	2. Subepithelial connective tissue graft.

II. **Treatment based on distribution of recession**

Localized	*Generalized (involving few teeth)*
i. Pedicle graft • Double papillae • Laterally-displaced	i. Free soft tissue graft.
ii. Coronally repositioned flap with semilunar incision	ii. Subepithelial connective tissue graft.
iii. Guided tissue regeneration	iii. Coronally-repositioned flap.

Q.18. What is the significance of bleeding on probing? How do you check for gingival bleeding?

Answer

Bleeding on probing is important as it is one of the earliest signs of gingival inflammation and is also a very sensitive, objective clinical indicator. However, its relationship to disease progression is unclear. Histologically, bleeding on probing results from increased vascularity, thinning and degeneration of the epithelium and the proximity of the engorged vessels to the inner surface (Fig. 53.11).

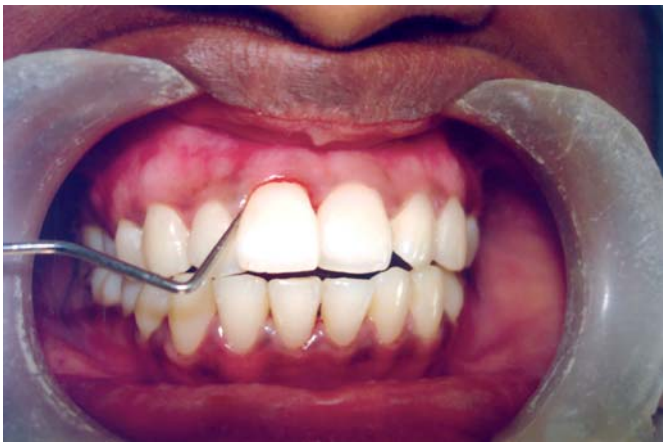


Fig. 53.11: Gingival bleeding on probing

It can be checked by using a blunt periodontal probe, which is carefully introduced to the bottom of pocket using forces up to 0.75N and gently moved laterally along the pocket wall. Bleeding may occur spontaneously or delayed by 30 to 60 seconds.

Q.19. What are the etiologic factors responsible for gingival bleeding?

Local Factors

Chronic bleeding

- Chronic inflammation

Acute bleeding

- Aggressive toothbrushing
- Sharp pieces of hard food
- Gingival burns
- Acute gingival diseases, e.g. NUG

Systemic Factors

- Vascular abnormality
  - Vitamin C deficiency
  - Allergy such as Schönlein-Henoch purpura
- Platelet disorder, e.g. idiopathic thrombocytopenic purpura.

- Hypoprothrombinemia, e.g. vitamin K deficiency from liver disease or sprue.
- Coagulation defects, e.g. Hemophilia, Christmas disease
- Malignancy, e.g. leukemia.
- Deficient platelet thromboplastic factor, e.g. uremia, multiple myeloma, postrubella infection purpura.
- Drugs, e.g. salicylates, dicoumarol, heparin.

Q.20. What are the microscopic changes associated with gingival bleeding?

Answer

Changes in Epithelium

Thinning of epithelium and microulcerations along the surface of the epithelium.

Factors responsible for it are the widening of the intercellular junctions and destruction of epithelium by the bacteria and their byproducts (e.g. collagenase, hyaluronidase, protease, chondroitin sulfate or endotoxin) gaining access into the connective tissue.

Changes in Connective Tissue

Dilation of the capillaries and venules and engorgement of the blood vessels.

Factors responsible for it are that they are the manifestations of the gingival inflammation resulting in diapedesis and emigration of the polymorphonuclear leukocytes. Because the capillaries are engorged and closer to the thinned and degenerated epithelium, which is less protective, any ordinary stimulus causes rupture of the capillaries and gingival bleeding occurs.

Q.21. What is the width of attached gingiva? How do you measure the width of attached gingiva?

Answer

It is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket.

The width of attached gingiva is determined by subtracting the sulcus or pocket depth from the total width of the gingiva (Fig. 53.12).

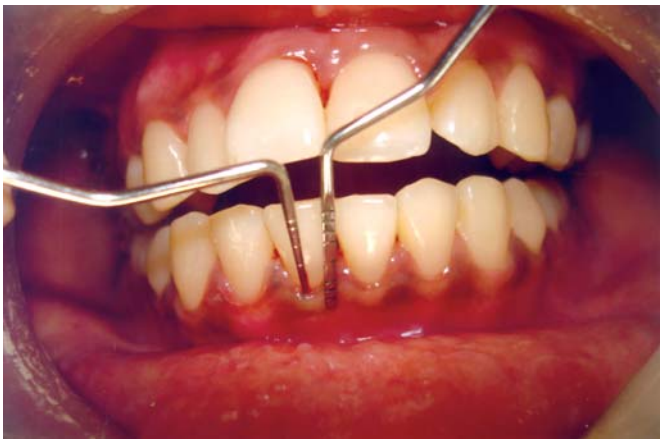


Fig. 53.12: Measurement of width of attached gingiva

Q.22. What are the tests done to identify whether the width of attached gingiva is adequate or inadequate?

Answer

There are four different methods used to find the width of attached gingiva.

1. *Measurement approach*: In this method first the pocket depth or the sulcus depth is measured, and then the total width of gingiva is measured, i.e. from gingival margin to mucogingival line. Thus by subtracting these two measurements we get the width of attached gingiva.
   Total gingival width–pocket depth = width of attached gingiva.
2. *By using Schiller's potassium iodide solution*
   It is similar to measurement approach. Potassium iodide solution only stains the keratinized epithelium, i.e. marginal gingiva, attached gingiva and interdental papilla.
   After application of this solution the total width of gingiva is measured that is from gingival margin to mucogingival line, and later the sulcus depth or pocket depth is measured. Then by subtracting the total gingival width from pocket depth we get the width of attached gingiva.
   Stained total gingival width – pocket depth = width of attached gingiva.
   Here, KI solution is used to know the accurate width of gingiva, i.e. from free marginal gingiva to mucogingival line, which is keratinized.

3. *Tension test*: This is done by stretching the lip or cheek to demarcate the mucogingival line and to see for any movement of the free gingival margin. And if the free gingival margin moves during stretching of lips then the attached gingiva is considered to be inadequate (Figs 53.13 and 53.14).
4. *Roll test*: It is done by pushing the adjacent mucosa coronally with a dull instrument. If the gingiva moves with the instrument then the width of attached gingiva is considered inadequate. In adequate width, the gingiva does not move because the attached gingiva is firmly attached to the underlying bone.

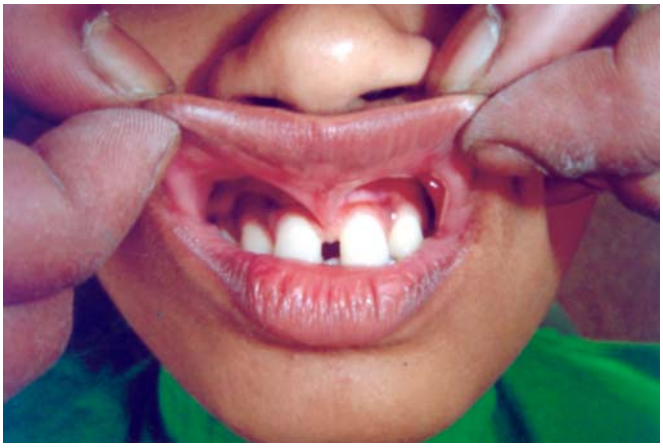


Fig. 53.13: Tension test (for abnormal frenal attachment)

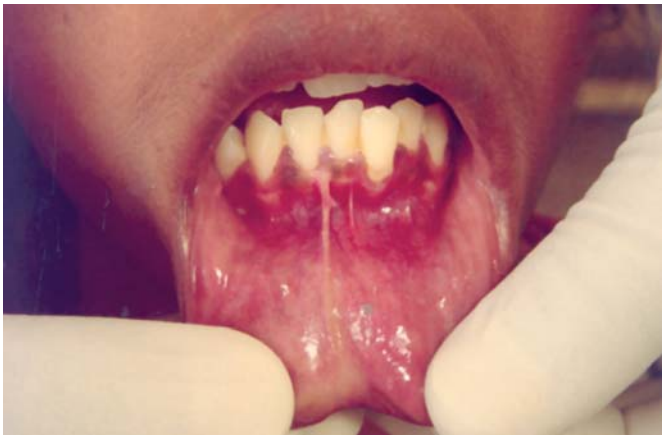


Fig. 53.14: Tension test (for lack of attached gingiva)

Q.23. What are the mucogingival problems?

Answer

Mucogingival problems are the disease conditions, which extend to or beyond the mucogingival junction.

Various mucogingival problems are:

1. Inadequate width of attached gingiva.
2. Gingival recession extending up to mucogingival junction.
3. Decreased vestibular depth.
4. Abnormal frenal attachment.
5. Pockets extending up to the mucogingival junction.

Q.24. How do you clinically differentiate between true, pseudo and combined pockets; suprabony and infrabony pockets?

Answer (Figs 53.15 to 53.17)

Pseudopockets: It is associated with gingival enlargement and also while probing, there is increased sulcus depth, the probe tip does not seem to go beyond the cementoenamel junction because junctional epithelium is still attached at the CEJ.

True pockets: It is associated with apical migration of junctional epithelium (attachment loss). This is clinically assessed by probing measurement.

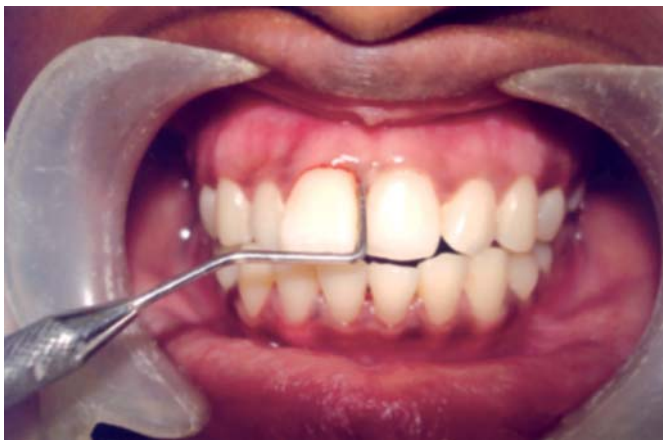
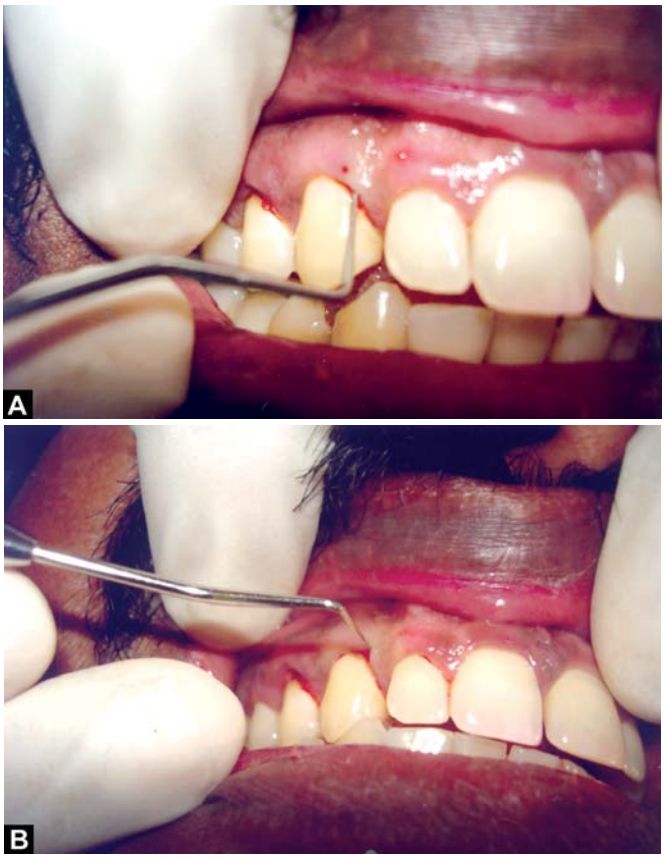


Fig. 53.15: Detection of a pocket (probe placed parallel to the long axis of the tooth)



Figs 53.16A and B: True and Pseudopocket. (A) Clinical detection of true/pseudopocket. (B) Detection of cementoenamel junction

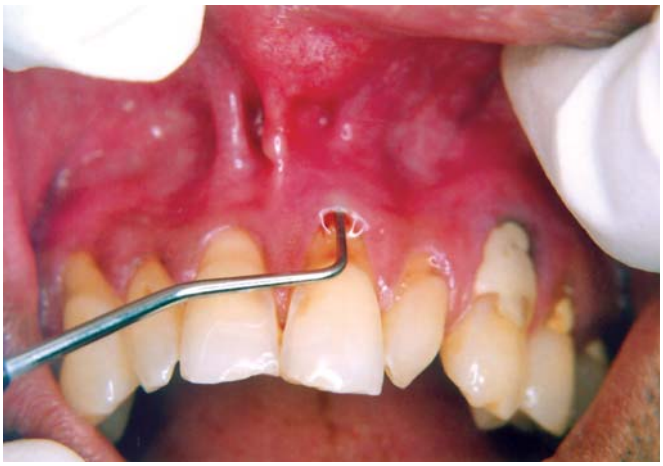


Fig. 53.17: Suprabony/Infrabony pocket (Probe in place with lateral pressure)

The probing method is as follows, since normally junctional epithelium is attached at the cementoenamel junction.

First: Detect the CEJ by running the probe perpendicular to the root surface.

Second: Once the CEJ is detected, bring the probe back to parallel position and observe whether probe can be probed beyond this point. If it can, then it is understood that the junctional epithelium is shifted apically, hence diagnosed as a true pocket.

Combined pocket: It is associated with both gingival enlargement and attachment loss.

Suprabony pocket: It is a true pocket with base of the pocket coronal to underlying alveolar bone crest. Clinically, it can be assessed by probing measurements. After the probe is inserted into the gingival sulcus and when lateral pressure is applied, if soft tissue resistance is felt, then it is diagnosed as suprabony pocket.

Infrabony pocket: It is a true pocket with base of pocket apical to level of adjacent alveolar bone. Clinically, probe is inserted into the gingival sulcus and when lateral pressure is applied, hard tissue resistance is felt.

Q.25. How do you treat pockets?

Answer

The various methods of pocket elimination are classified as follows:

1. *New attachment techniques*: These offer the ideal result, because they eliminate pocket depth by reuniting the gingiva to the tooth at a position coronal to the bottom of the pre-existing pocket.
2. Removal of the pocket wall is the most common method. It can be removed by:
   a. Retraction or shrinkage by scaling and root planing.
   b. Surgical removal by gingivectomy technique or by means of an undisplaced flap.
   c. Apical displacement with an apically displaced flap.

3. Removal of the tooth side of the pocket, which involves either extraction of the tooth or hemisection of the tooth.

Q.26. Describe the sequelae following unreplaced missing first molars.

Answer

It consists of the following:

i. Second and third molars tilt mesially leading to distal cusp elevation thereby plunger cusp effect. In the mesial cusp, due to mesial tilt the distance between the mesial cusp tip and bone is reduced thereby progression of periodontal disease becomes rapid.

ii. Supraeruption of opposing first molar may lead to:

↓

Loss of proximal contact

↓

Food impaction

↓

Plaque accumulation

↓

Gingivitis

↓

Periodontitis

iii. Premolars move distally, mandibular incisors tilt or drift lingually.

Anterior overbite is increased. The mandibular incisors strike the maxillary incisors near the gingiva and traumatizes the gingiva.

iv. The maxillary incisors are pushed labially and laterally.

v. Extrusion of anterior teeth leading to diastema.

Q.27. Describe the sequelae following loss of proximal contact form.

Answer

The tightness of contact should be checked by means of clinical observation and with a dental floss.

Abnormal contact relation or loss of proximal contact can cause plaque retention and food impaction → gingivitis and periodontitis → mobility and loss of tooth.

Q.28. How do you diagnose furcation involvement?

Answer

Furcation involvement can be diagnosed by two methods:

1. Clinical examination
2. Radiographs

A thorough clinical examination is the key to diagnosis and treatment planning. Careful probing is performed by using a Naber's probe. Transgingival probing further helps to define the anatomy of the furcation defect (Fig. 53.18).

Radiographs can also be used as diagnostic aids in diagnosing furcation involvement:

Grade I: Radiographic changes are not usually found

Grade II: Radiographs may or may not depict the furcation involvement

Grade III: Show a radiolucent area in the crotch of the tooth

Grade IV: Seen as a radiolucent area.

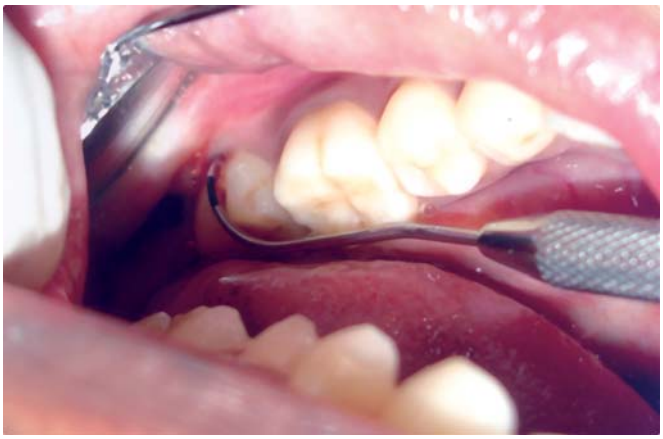


Fig. 53.18: Diagnosis of furcation involvement using Naber's probe

Q.29. Describe the etiology, classification and treatment of furcation involvement.

Answer

Etiology

1. Extension of inflammatory periodontal disease.
2. Trauma from occlusion.

3. Cervical enamel projections
4. Pulpoperiodontal diseases
5. Iatrogenic cofactors
6. Anatomical factors

Classification of Furcation Involvement

According to Glickman (1953):

Grade I: Pocket formation into the flute of the furcation but inter-radicular bone is intact

Grade II: Loss of inter-radicular bone and pocket formation of various depths into the furcation but not completely probable to the opposite side of the tooth.

Grade III: Complete loss of inter-radicular bone with pocket formation that is completely probable to the opposite side of the tooth.

Grade IV: Loss of attachment and recession that has made the entire furcation clinically visible.

According to Goldman and Cohen, furcation involvement can be:

Grade I: Incipient lesion

Grade II: It is a cul de sac lesion

Grade III: Through and through furcation involvement

According to Hamp et al.

Degree I: Horizontal loss of periodontal tissue support less than 3 mm.

Degree II: Horizontal loss of support exceeding 3 mm but not encompassing the total width of the furcation area.

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

According to Tarnow and Fletcher

Subclass A: Vertical destruction to one-third of the total inter-radicular height (1-3 mm).

Subclass B: Vertical destruction reaching two-thirds of the inter-radicular height. (4 to 6 mm).

Subclass C: Inter-radicular osseous destruction into or beyond the apical third (more than 7 mm).

Treatment

	Traditional procedures	*Regenerative procedures*
Grade I	Scaling, root planing, curettage, gingivectomy, odontoplasty.	
Grade II	Scaling, root planing, curettage, odontoplasty, osteoplasty with limited ostectomy, in shallow grade II invasions. In more severe furcation involvements, root resection/ hemisection is done.	Autogenous and autologous osseous grafting, synthetic grafting, GTR, root conditioning, coronally displaced flap and combination procedures.
Grade III	Tunneling, root sectioning, hemisection, extraction	GTR and combination procedures
Grade IV	Maintenance	

Q.30. What are the causes for tooth mobility? Give the classification of mobility.

Answer

Causes for mobility can be divided into local and systemic factors (Fig. 53.19):

Local Factors

a. Bone loss and loss of tooth support—
   1. Bone destruction caused by extension of gingival inflammation, which can be either due to plaque products or pharmacologically active substances.
   2. Trauma from occlusion, either in absence or associated with inflammation.

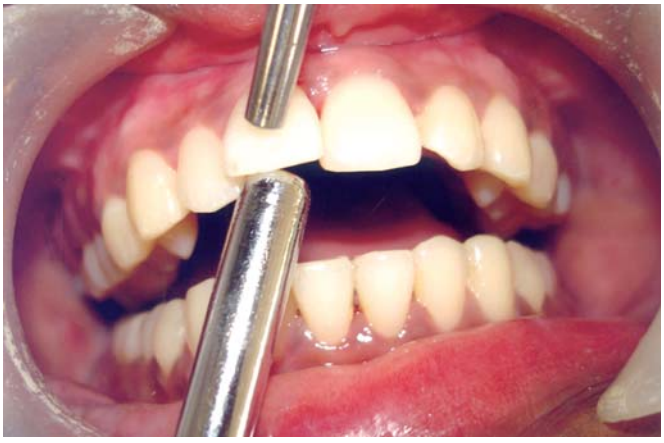


Fig. 53.19: Clinical demonstration of mobility

b. Hypofunction
c. Periapical pathology
d. After periodontal therapy
e. Parafunctional habits like bruxism or clenching
f. Pathology of jaws like tumor, cyst, etc
g. Traumatic injury to dentoalveolar unit
h. Tooth morphology
i. Overjet and overbite
j. Implant mobility

Systemic Factors

a. Age
b. Sex and Race : Slightly higher incidence seen in females and Negros
c. Menstrual cycle
d. Oral contraceptives
e. Pregnancy
f. Systemic diseases such as Papillon-Lefevre syndrome, Down's syndrome, neutropenia, Chediak-Higashi syndrome, etc.

Grading of Mobility

Degree I: Mobility of crown of the tooth 0.2-l mm in horizontal direction

Degree II: Mobility of crown of the tooth exceeding 1 mm in horizontal direction

Degree III: Mobility of crown of the tooth in vertical direction as well.

Q.31. What is pathological migration ? How do you clinically identify it?

Answer

Pathologic migration refers to tooth displacement that results when the balance among the factors that maintain physiological tooth position is disturbed by periodontal disease. It is relatively common and may be an early sign of disease, or it may occur in association with gingival inflammation and pocket formation as the disease progresses.

The characteristic clinical features are as follows:

1. It occurs most frequently in the anterior region.
2. It is usually accompanied by mobility rotation and extrusion of teeth.
3. Drifting of maxillary and mandibular anterior incisors labially creating diastema between the teeth.
4. Should be differentiated from physiological migration.

Q.32. What are the causes for pathologic migration?

1. Weakened periodontal support
2. Changes in the forces exerted on the teeth
   - Unreplaced missing teeth
   - Failure to replace first molars.
3. Other causes like:
   - Pressure from tongue
   - Pressure from granulation tissue of periodontal pocket.

Q.33. Define attrition, abrasion and erosion.

Answer

Attrition : It is the occlusal wear resulting from functional contact with opposing teeth or, it is the term used for dental wear caused by teeth against teeth.

Abrasion : Refers to the loss of tooth substance induced by mechanical wear other than that of mastication.

Erosion: It is a sharply defined wedge-shaped depression in the cervical area of the facial tooth surface or wear to the nonoccluding tooth surface and includes sharply defined, wedge-shaped depressions in the facial cervical areas of the tooth.

Q.34. When is tooth tender on percussion?

Answer

Various causes for tooth to be tender on percussion are:

A. Periodontal diseases (Tooth is tender on lateral percussion)
B. Irreversible pulpitis
C. Root fracture
D. Crown fracture
E. Periapical pathology

Q.35. What is retrograde periodontitis?

Answer

Normally, periodontitis is caused by extension of inflammation from the gingiva into deeper periodontal tissues. However, periodontitis can also be caused by pulpal infections that have entered periodontal ligament either through apical foramen or through the lateral canal. Such a periodontal lesion is termed as retrograde periodontitis.

Q.36. How do you diagnose a case of trauma from occlusion?

Answer

a. *Fremitus test* (Fig. 53.20): It is a measurement of the vibratory pattern of the teeth when the teeth are placed in the contacting positions and movements. To measure fremitus, dampened index finger is placed along the buccal and labial surfaces of the maxillary teeth and the patient is asked to tap the teeth together in the maximum intercuspal position and then grind systematically in the lateral, protrusive movements and positions. The teeth that are displaced by the patient in these positions are identified and graded. The following classification system is used:
Class I fremitus: Mild vibration or movements detected.
Class II fremitus: Easily palpable vibration but no visible movements.
Class III fremitus: Movements visible with naked eye.

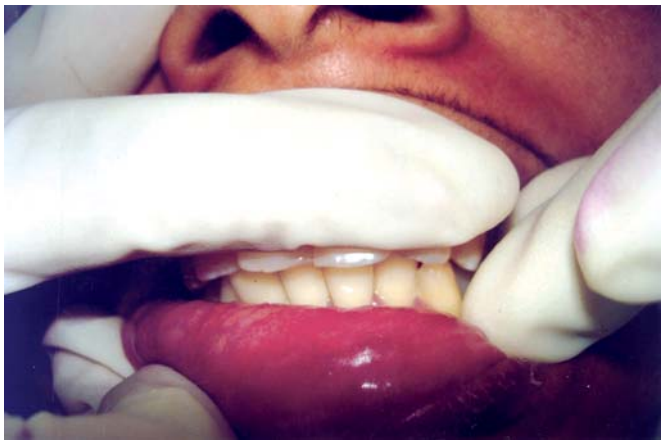


Fig. 53.20: Fremitus test is performed placing wet finger on the labial surface of maxillary anterior teeth

b. *With help of occlusion registration strips/articulating paper*.
c. *Auditory test for trauma from occlusion*: In centric relation, there is a distinct ringing sound during tooth contact. But in the traumatic occlusion with deflection present, sound is dull and imperceptible.

Tactile Method

In centric relation and normal excursive movement, response to finger is smooth but with deflection present as in trauma from occlusion, a roughness can be detected.

Q.37. What are the types, signs and symptoms of trauma from occlusion?

Answer

The various types are:

1. Primary and secondary occlusal trauma.
2. Acute and chronic.

Signs and Symptoms

1. Presence of gingival recession.
2. Presence of Stillman cleft and McCall's festoons.
3. Presence of traumatic crescent.
4. Presence of infrabony pockets.
5. Food impaction.
6. Excessive wearing of tooth cusps and appearance of wear facets.
7. Hypersensitivity of the teeth.
8. Increased tooth mobility.
9. Tenderness of TMJ and muscles of mastication.
10. Widening of periodontal ligament and thickening of lamina dura.
11. Vertical bone loss.
12. Condensation of alveolar bone.
13. Root resorption.
14. Buttressing bone formation seen in an occlusal radiograph.
15. Smudging of articulating paper.
16. Dull percussion note of an affected tooth.
17. Vibrations are felt on performing Fremitus test.

Q.38. What is the difference between mobility and fremitus?

Answer

Mobility: It is a measurement of horizontal and vertical tooth displacement created by the examining force applied that should be roughly 100 g force.

Fremitus: It is a measurement of the vibratory patterns of the teeth when the teeth are placed in contacting positions and movements.

- Fremitus differs from mobility, in that fremitus is tooth displacement created by the patient's own occlusal force
- Therefore, the amount of force varies greatly from patient to patient, unlike mobility, where in the force with which it is measured tends to be the same for each examiner.

Q.39. What are the various indices used for assessing oral hygiene?

Answer

1. Oral hygiene index.
2. Simplified oral hygiene index.
3. Patient Hygiene performance index.
4. Plaque component of periodontal disease index.
5. Shick and Ash modification of plaque criteria.
6. Turesky-Gilmore-Glickman modification of the Quigley Hein plaque index.
7. Plaque index.
8. Calculus index.

Q.40. What are the indices for gingival bleeding?

Answer

1. Sulcular bleeding index.
2. Papillary bleeding index.
3. Bleeding points index.
4. Interdental bleeding index.
5. Gingival bleeding index.

Q.41. Describe the effects of overhanging restorations on periodontium.

Answer

Overhanging restorations causes violation of biologic width, which may lead to:

1. Ideal locations for the accumulation of plaque
2. Changing the ecologic balance of the gingival sulcus, i.e. favor the growth of gram-negative anaerobic species, which altogether leads to the pathogenesis of chronic inflammatory periodontal disease.

Q.42. What is the importance of recording various lab investigations?

Answer

Complete Blood Count

The complete blood count will include:

i. Hemoglobin (Hgb).
ii. Hematocrit (Hct).
iii. Red blood cell count (RBC).
iv. White blood cell count (WBC).

I. Hemoglobin
   i. Hgb is the oxygen carrier of the blood. It is decreased in hemorrhage and anemias. It is increased in hemoconcentration and polycythemia.
   ii. The normal range is 14 to 18 g/dl of blood in men and 12 to 16g/dl of blood in women.

II. *Hematocrit*: It reflects the relative volume of cells to plasma in the blood. In anemias and after blood loss, the Hct is lowered. In polycythemia and dehydration it is raised. The normal Hct range is 40 to 54 percent for men and 37 to 47 percent for women.

III. *RBC count*: RBC contains HgB. An increase in RBCs may indicate hemoconcentration or polycythemia.
Decrease in number of RBCs may be indicative of blood loss or anemia.

IV. *WBC count*: WBCs are important in defence against invading microorganisms. Normal count is 5000 to 10,000/mm^3. Increase in WBC is seen in leukemia, bacterial infection, infectious mononucleosis and certain parasitic infections. Decrease in WBC's seen in aplastic anemia, lupus erythematosus, acute viral infections, and drug and chemical toxicity.
   a. Neutrophils (50 to 70%)
      - Increase in most bacterial infections
      - An increase in number of immature neutrophils is frequently found in acute infections. This is called shift to the left.

b. Eosinophils (1 to 4%): Increase in allergic conditions and parasitic infections.
c. Basophils (0 to 1%): May be increased in blood dyscrasias.
d. Lymphocyte (25 to 40%): It increases in measles and in severe bacterial or chronic infections.
e. Monocyte (4 to 8%): It may be increased during recovery from severe infections and Hodgkin's diseases.

V. *Blood glucose*: Basic tests of blood glucose are:
a. Fasting blood sugar
b. Two hour postprandial blood sugar test
c. Glucose tolerance test
d. Random blood sugar test
The normal range for blood glucose is 70 to 110 mg/dl of serum.

VI. *Blood urea nitrogen*:
- Normal value is 8 to 23 mg /dl of blood.
- Increased value seen in extensive kidney disease, congestive heart failure, dehydration.

VII. Serology—For screening of syphilis.
- All are nonspecific tests for syphilis and may give both false positive and false negative results. Interpretation of these results requires patient history and clinical findings. Confirmation is by Fluorescent treponemal antibody-absorption test (FTA-abs) or the microhemagglutination *treponemal palladium* test (MHA-tp).

VIII. Screening test for hemorrhagic disorders:
a. *Bleeding time*: It is the time required for hemostasis to occur in a standard wound of the capillary bed. It varies with vascular and platelet abnormalities. The normal range is 1 to 7 minutes.
b. *Platelet count*: It is decreased in thrombocytopenic purpura. In myeloproliferative diseases, platelets are increased. Normal platelet count is 150,000 to 400,000/mm^3.
c. *Prothrombin time*: It is an indirect test of the clotting ability of the blood. It gives an indication of prothrombin deficiency arising from liver disease. Normal range is 12 to 17 sec.
d. *Partial thromboplastin time*: It is designed to help the clinician recognize mild to moderate deficiencies of intrinsic clotting factors.

Q.43. Effects of improper orthodontic treatment on periodontium.

Answer

Orthodontic tooth movement is possible because the periodontal tissues are responsive to externally applied forces. Orthodontic forces at optimum rate and direction have no negative impact on the periodontium. Unlikely when these forces are abnormal, it results in deleterious effects such as:

- Moderate orthodontic forces ordinarily result in bone remodelling and repair, whereas excessive forces may produce necrosis of the periodontal ligament and adjacent alveolar bone.
- Excessive orthodontic forces also increase the risk of apical root resorption.
- Use of elastics to close diastema may result in severe attachment loss with possible tooth loss as the elastics migrate apically along the root.
- Surgical exposure of impacted teeth and orthodontic assisted eruption has the potential to compromise the periodontal attachment on adjacent teeth.
- Dentoalveolar gingival fibers are stretched when teeth are rotated during orthodontic therapy.

Q.44. What are the things you look for in a radiograph?

Answer

Radiographs help in diagnosis of periodontal disease, determination of patient's prognosis and the evaluation of the outcome of treatment. But radiograph is an adjunct to the clinical examination, not a substitute for it.

The normal anatomic landmarks to be seen in a radiograph are (First identify the type of X-ray its side, i.e. left or right and which arch it belongs to):

Maxillary arch

1. Nasopalatine foramen.
2. Median palatal sutures.

3. Nasal fossa.
4. Nasal septum.
5. Anterior nasal spine.
6. Maxillary sinus.
7. Maxillary tuberosity.

Mandibular arch

1. Genial tubercles.
2. Lingual foramen.
3. Mental process.
4. Mental foramen.
5. Oblique ridge.
6. Mandibular canal and submandibular fossa.

Lesions to be seen are:

1. Caries
   a. Interproximal caries
   b. Occlusal caries
   c. Root surface caries
2. Periodontal disease, i.e. periodontitis—Bone loss.
   a. Types of bone loss
      i. Vertical or angular
      ii. Horizontal bone loss
      iii. Crater-like loss
      iv. Arc-shaped bone loss.
   b. Contour or Architecture of bone
      i. Positive
      ii. Negative
   c. Continuation of lamina dura
   d. Trabecular pattern of bone
   e. Furcation involvement
   f. Amount of bone loss.
3. Tooth fractures
   a. Crown fracture
   b. Root fracture
4. Resorption
   a. Internal resorption
   b. External resorption

Q.45. What are the advantages and disadvantages of IOPA radiographs and OPG?

Answer

Advantages of OPG

1. For visualizing the maxilla and mandible in one film.
2. For patient education.
3. To evaluate impacted teeth.
4. In the evaluation of multiple unerupted supernumerary teeth.
5. To evaluate eruption sequence and pattern, growth and development.
6. For detecting any pathology involving the jaws.
7. For evaluating traumatic injuries.
8. Radiographic examination of patients with limited mouth opening.
9. To identify diseases of the temporomandibular joint.
10. Reduces the risk of multiple exposures to radiation.

Disadvantages

1. Images are not as sharp as in an intraoral film.
2. It cannot be used in the diagnosis of caries.
3. It cannot be used in the evaluation of bone loss due to periodontal disease.
4. It shows superimposition especially in the premolar region.
5. Structures in the anterior region may not be well-defined.

Advantages of IOPA

1. Better visibility of periapical region.
2. Helps in the diagnosis of periapical pathology.
3. To study crown and root length and their morphology.
4. Helps in evaluating root apex formation.
5. Evaluation of endodontic treatment.
6. Helps in evaluation of fracture of teeth.

Disadvantages

1. Cannot be taken in case of patients with limited mouth opening.
2. It cannot detect any pathology involving the jaws.
3. Multiple radiographs have to be taken for the examination of the entire arch thereby increasing the risk of exposure to radiation.

Q.46. Justify your diagnosis, if diagnosis is gingivitis then why? Or if it is periodontitis then why?

Answer

Gingivitis is detected when there is evidence of gingival inflammation such as swelling, redness, bleeding on probing, often the pocket depth less than or equal to 4 millimeter but by-definition there is no measurable loss of probing periodontal attachment associated with periodontal infection. Gingival recession maybe associated with gingival abrasion or prominent radicular surface. Plaque is usually present; calculus may or may not be present. Tooth mobility is rarely present and radiographic changes do not suggest bone loss.

Periodontitis can be classified as early, moderate, advanced. According to AAP's classification, in early periodontitis there are shallow pockets, minor to moderate bone loss, satisfactory topography and generally no tooth mobility. Moderate periodontitis is characterized by moderate to deep pockets, moderate to severe bone loss, unsatisfactory topography and slight tooth mobility. In advanced periodontitis, there are deep pockets, many areas of severe bone loss, advanced tooth mobility and often a need for prosthesis to replace missing teeth or splint the mobile teeth. Periodontitis is by definition diagnosed when there is measurable attachment loss (e.g. >2 mm) and often associated with probing depth more than 4 mm. Plaque and calculus are usually present. Tooth mobility may also occur. Radiographic changes seen include alveolar crestal resorption of varying degree and in advanced cases, loss of alveolar bone in furcation areas may be seen.

Q.47. Define prognosis. What are the factors responsible for individual and overall tooth prognosis?

Answer

Prognosis is a prediction of the duration, course and termination of a disease and its response to treatment.

Factors for individual tooth prognosis are:

- Percentage of bone loss.
- Probing depth.
- Distribution and type of bone loss.
- Presence and severity of furcation involvements.
- Mobility.
- Crown-root ratio.
- Pulpal involvement.
- Tooth position and occlusal relationship.
- Strategic value.

Factors for overall prognosis are:

- Age
- Medical status
- Individual tooth prognosis (distribution and severity)
- Degree of involvement, duration and history of the disease
- Patient cooperation
- Economic consideration
- Knowledge and ability of the dentist
- Etiologic factors.

Q.48. Discuss the treatment plan in a sequential order.

Answer

The objectives of treatment are:

a. Elimination of disease.
b. Restoration of efficient function.
c. Production of satisfactory appearance.

Sequence of therapeutic procedures:

a. *Preliminary phase/Emergency phase*
   1. Treatment of emergencies like dental or periapical and periodontal problems
   2. Extraction of hopeless teeth and provisional replacement, if needed
b. *Phase I therapy (Etiotrophic phase).*
   1. Plaque control
   2. Diet control
   3. Removal of calculus and root planing
   4. Correction of restorative and prosthetic irritational factors
   5. Excavation of caries and restoration
   6. Antimicrobial therapy (local/systemic)
   7. Occlusal therapy
   8. Minor orthodontic movement
   9. Provisional splinting.

Evaluation of response to Phase I therapy

I. *Rechecking*
   - Pocket depth and gingival inflammation
   - Plaque and calculus, caries.

c. *Phase II therapy (Surgical phase)*
   - Periodontal surgery, including placement of implants
   - Root canal therapy

d. *Phase III therapy (Restorative phase).*
   a. Final restorations
   b. Fixed and removable prosthodontics

   Evaluation of response to restorative procedures, periodontal examination

e. *Phase IV therapy (Maintenance phase)*

   Periodic recall visits, re-checking for:
   - Plaque and calculus.
   - Gingival condition (inflammation, pockets).
   - Occlusion, tooth mobility.
   - Other pathologic changes.

Q.49. Classify periodontal instruments.

Answer

They are classified into surgical and nonsurgical instruments.

Nonsurgical Instruments

1. Hand instruments.
2. Machine driven instruments.

Hand Instruments

1. Diagnostic instruments, examples: explorers and periodontal probes
2. Scaling and root planing and curettage instruments, examples: sickle scaler, curettes, hoe, chisel and file
3. Cleaning and polishing instruments, examples: Dental tapes, rubber cups and bristle brushes.

Machine Driven Instruments

i. Scaling and curettage instruments, examples: ultrasonic, and sonic instruments

ii. Used for other purpose, example: Schwartz periotrievers – used for retrieval of broken instruments. EVA system - correcting overhanging restorations

Surgical Instruments

1. Excisional and Incisional Instruments, examples:
   - Periodontal knives (Gingivectomy knives)
   - Interdental knives No 1-2
   - Surgical blades. (Scalpel No. 11, 12 and 15)
   - Electrosurgery
2. Surgical curettes and sickles.

   Examples:
   - Kramer curettes No. 1, 2 and 3
   - Ball scaler
3. Periosteal elevators, examples: No. 24 G elevator and Goldman fox No: 14
4. Surgical chisels and Hoes, examples: chisel in Oschsenbein No. 1-2, Rhodes chisels
5. Surgical files example: Sugarman files
6. Scissors and Nippers, example: Goldman fox No: 16 scissors
7. Needle holders, example: Castroviejo needle holder.

Q.50. Give the characteristics of a scaler and curette with differences.

Answer

Characteristics of a Scaler

i. Triangular in cross-section
ii. Pointed tip not suited for subgingival use
iii. Two cutting edges per working end.
iv. Pointed tip and straight cutting edges do not adapt well to rounded root surfaces and concavities.
v. Face perpendicular to the lower shank so that cutting edges are in level with one another
vi. Shank
   - Anterior sickles—Simple design
   - Posterior sickles—Complex design

vii. Functions: Removal of medium to large-sized supra-gingival calculus deposits.

Provides good access to the:

1. Proximal surfaces on anterior crowns
2. Enamel surfaces apical to contact areas of posterior teeth

viii. Examples: Anterior sickle scaler—OD-1, Jacquette-30 Jacquette-33, Towner-015, Goldman H6 and H7, Posterior sickle scalers—Jacquette 34/35, Jacquette 14/15, Jacquette 31/32, Ball 2/3.

Characteristics of Curette

	Universal	*Area-specific*
Cross-section	Semicircular	Semicircular
Back	Rounded	Rounded
Cutting edges	Two cutting edges per working end	One cutting edge per-working end
Toe	Rounded	Rounded
Face	Perpendicular to the lower shank	Tilted at a 60°-70° to the lower shank
Application	Used to instrument all Tooth surfaces in the dentition	Limited to use on a specific area of specific tooth surface
Function	Removal of light to medium-sized calculus deposits	Removal of light calculus deposits
Examples	Columbia 2R/2L, Columbia 13/14, Rule 3/4, Barnhart 1/2	Gracey series Kramer-Nevins series, Turgeon series,
	Barnhart 5/6	After five
	Langer 1/2	Mini five
	Langer 3/4	Vision curvette series
	Langer 5/6	
	Langer 17/18	

Q.51. Differences between Universal and Gracey curette.

Answer

	Gracey curette	*Universal curette*
Area of use	Set of many curettes designed for specific areas and surfaces	One curette designed for all areas and surfaces
Cutting edge use	One cutting edge used; work with outer edge only	Both cutting edges used work with either outer or inner edge
Curvature	Curved in two planes; Blade curves up and to the side	Curved in one plane; Blade curves up, not to side
Blade angle	Offset blade: face of blade beveled at 60° to shank	Not offset; face of blade beveled at 90° to shank

Q.52. Classify various supragingival and subgingival scalers.

Answer

Supragingival scalers

1. Sickle scaler
2. Surface scaler
3. Cumine scaler
4. Morse scaler

Subgingival scalers

1. Chisel scaler
2. Hoe scaler
3. Periodontal file
4. Islet scaler

Q.53. What are the instruments used for root planing?

Answer

Curette is the instrument of choice for removing deep subgingival calculus; root planing, altered cementum and removing the soft tissue lining the periodontal pocket.

These include:

a. Universal curettes

b. Area-specific curettes

In Gracey curettes:

- 1-2, 3-4—Anterior teeth
- 5-6—Anterior teeth and premolars
- 7-8, 9-10—Posterior teeth facial and lingual
- 11-12—Posterior teeth (mesial)
- 13-14—Posterior teeth (distal)

Q.54. Define scaling and root planing.

Answer

Scaling is defined as a process by which plaque and calculus are removed from both supragingival and subgingival tooth surfaces.

Root planing: It is a process by which residual embedded calculus and portions of cementum are removed from the roots to produce a smooth hard, and clean surface.

OR

It denotes a technique of instrumentation by which the softened cementum is removed and the root surface is made hard and smooth.

Q.55. What are the principles of instrumentation?

Answer

General principles of instrumentation are:

1. Accessibility.
2. Visibility, illumination and retraction.
3. Condition of instruments.
4. Maintaining a clean field.
5. Instrument stabilization.
6. Instrument activation.

Q.56. What are finger rests and grasps?

Answer

Finger rests serve to stabilize the hand and the instrument by providing a firm fulcrum as movements are made to activate the instrument. A good finger rest prevents injury and laceration of the gingiva and surrounding tissues by poorly controlled instruments. Most clinicians for the finger rest prefer the fourth finger. Maximal control is achieved when the middle finger is kept between the instrument-shank and fourth finger. This "built-up fulcrum" is an integral part of the wrist-forearm action that activates the powerful working stroke for calculus removal (Figs 53.21 to 53.29).

Finger rest

Extraoral finger rests:

a. Palm up.
b. Palm down.

Intraoral finger rests

a. Conventional
b. Cross-arch
c. Opposite arch
d. Finger on finger

Grasps: A proper grasp is essential for precise control of movements made during periodontal instrumentation.

The commonly used grasps are:

a. *Modified pen grasp*: The thumb, index finger, and middle finger are used to hold the instruments as pen is held, but the middle finger is positioned so that the side of the pad next to the finger nail is resting on the instrument shank. The index finger is bent at the second joint from the fingertip and is positioned well above the middle finger

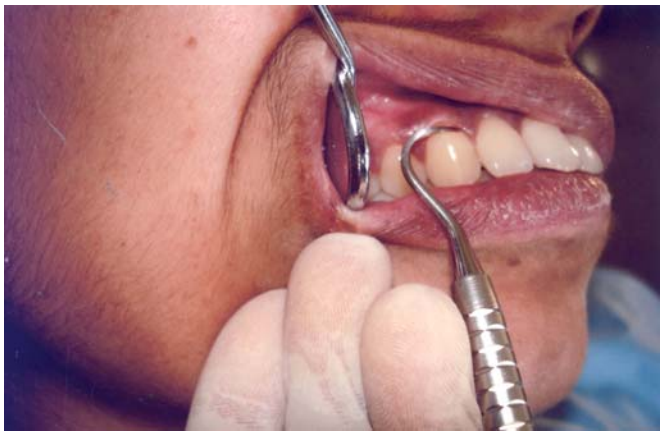


Fig. 53.21: Extraoral fulcrums—Palm up

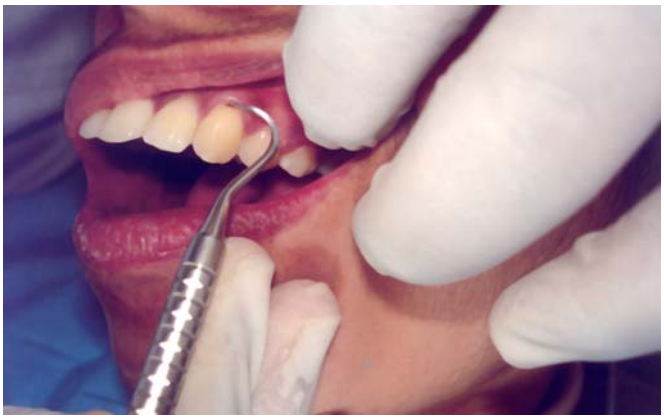


Fig. 53.22: Extraoral fulcrums—Palm down

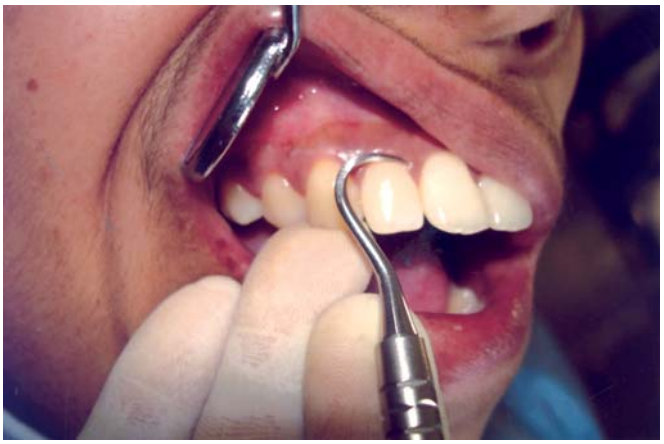


Fig. 53.23: Conventional—Intraoral finger rests

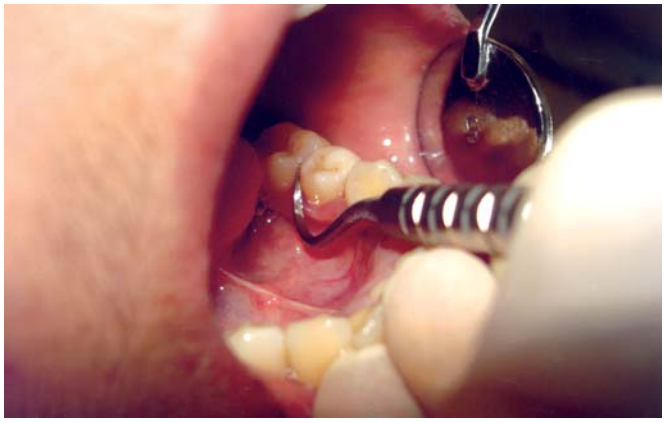


Fig. 53.24: Intraoral finger rests—Cross-arch

Fig. 53.27: Instrument grasps—Pen grasp

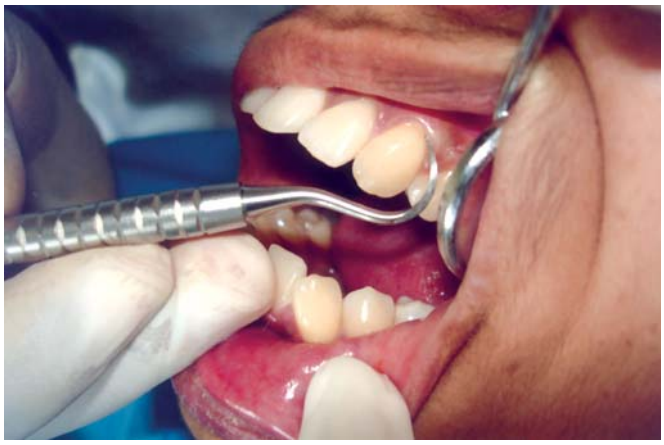


Fig. 53.25: Opposite arch (Intraoral finger rests)

Fig. 53.28: Instrument grasps—Modified pen grasp

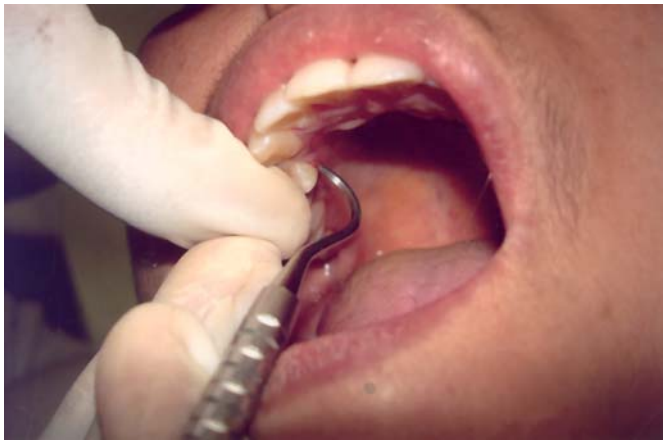


Fig. 53.26: Finger on finger (Intraoral finger rests)

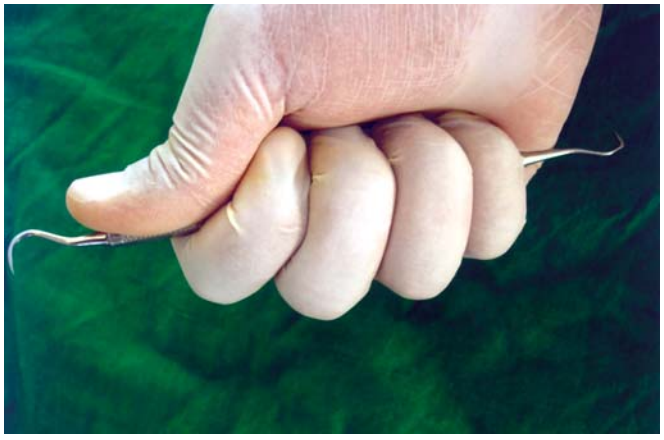


Fig. 53.29: Instrument grasps—Palm and thumb grasp

on the same side of the handle. The pad of the thumb is placed midway between the middle and index fingers on the opposite side of the hand thereby creating tripod effect.

b. *Palm and thumb grasp*: It is used for stabilizing instruments during sharpening and for manipulating air and water syringes, but it is not recommended for periodontal instrumentation.

Q.57. Different angulations needed for scaling procedure.

Answer

Angulation refers to the angle between the face of bladed instrument and tooth surface. It is also called tooth blade relationship.

a. For subgingival insertion of bladed instruments angulations should be close to 0 degrees as possible.
b. For scaling and root planing, angulation should be between 45 to 90°.
c. For gingival curettage angulation>90° is established so that the cutting edge will engage and remove the pocket epithelium.

Q.58. Recent advances in periodontal instrumentation.

Answer

Advances in Diagnostic Instruments

Probes: They are classified, based on the generation of development.

Ist Generation Probes: Conventional probes, e.g. Williams probe

IInd Generation Probes: Pressure sensitive probes, e.g. Borodontic probes

IIIrd Generation Probes: Computer aided probes, e.g. Florida probes

IVth Generation Probes: Records sequential probe positions along gingival sulcus. They are still under research.

Vth Generation Probes: Ultrasound probes. They are still under research.

Dental endoscope : Perioscopy system, which works on fiber optic system. It is used for subgingival calculus detection, caries, defective restoration, and fractures.

ADVANCES IN ROOT PLANING INSTRUMENTS

1. *Gracey no 15-16*: It is a modification of standard 11-12 and has an 11-12 blade with a 13-14 shank. It is useful for mesial surfaces of posterior teeth.
2. *Gracey no 17-18*: It is a modification of standard 13-14. It is a 13-14 Gracey curette with a terminal shank elongated by 3 mm and a more accentuated angulation of shank.
3. *Extended shank curettes (after five curettes)*: Here shank is elongated by 3 mm and is used for deeper periodontal pockets of 5 mm or more.
4. *Mini-bladed curettes (mini five curettes):* They are modifications of after five curettes with their blade reduced by half. It is used in furcation area, developmental grooves, tight pockets, deep narrow pockets.
5. *Gracey curettes*: Here the blade length is half of the conventional gracey curette and blade curves upwards.
6. *Langer and mini-langer curettes*: It is a combination of universal and gracey curettes available in 5-6, 11-12 and 13-14. It has similar shank design to a gracey curette but blade angle is 90 degrees.

Glossary

Abrasion Refers to the loss of tooth substance induced by mechanical wear other than that of mastication. *—Carranza*

Abscess A localized collection of pus.

Acellular Cementum/ Primary Cementum Cementum lacking embedded cells. *—Grant/Stern*

Acellular Cementum/ Primary Cementum Cementum that forms in conjunction with root formation and tooth eruption. *—Lindhe*

Acantholysis Hyperplasia of the prickle cell layer (stratum spinosum) of stratified squamous epithelia resulting in thickened rete redges and widening of this layer.

Acquired Coatings It includes those exogenous origins such as saliva, bacteria, calculus and surface stains.

Acquired Immunodeficiency Syndrome (AIDS) It is characterized by profound impairment of the immune system.

Acquired Pellicle It is a homogeneous, membranous, acellular film that covers the tooth surfaces and frequently forms the interface between the surface of the dental plaque and calculus. *—Saul Schluger*

Active Eruption Active eruption is the movement of the teeth in the direction of the occlusal plane. *—Carranza*

Actual Position It is the level of the epithelial attachment on the tooth. *—Carranza*

Acute Gingivitis Inflammation of gingiva which is of short duration, sudden onset and can be painful.

Acute Trauma from Occlusion It results from abrupt occlusal impact such as that produced by biting on hard object. *—Carranza*

Adaptation Refers to the manner in which the working end of the periodontal instrument is placed against the surface of the tooth. *—Carranza*

Addison's Disease It is caused by adrenal dysfunction and produces isolated patches of discoloration varying from bluish black to brown. *—Carranza*

Additive Osseous Surgery Includes procedures directed at restoring the alveolar bone to its original level.

Adjuvant A nonspecific stimulant of the immune response which enhances the response to antigen. Many bacterial cell wall components have this property.

Adult Periodontitis A form of periodontitis that usually has an onset beyond the age of 35 years. Bone resorption usually progresses slowly and predominantly in the horizontal direction.

Aerobe A microorganism which grows in the presence of oxygen.

Ageing of Periodontium Ageing is a slowing of nature function, a disintegration of the balanced control and organization that characterize the young adult. *—Carranza*

Aggressive Periodontitis It is a periodontal destruction that becomes clinically significant around adolescence or early adulthood. *—Carranza*

Aggressive Periodontitis — It is characterized by the rapid loss of attachment and bone loss occurring in an otherwise clinically healthy patient with the amount of microbial deposits inconsistent with disease severity and familial aggregation of diseased individuals.

Aggressive Periodontitis — Was formerly classified as early onset periodontitis, i.e. localized juvenile periodontitis (LJP). Generalized aggressive periodontitis was previously classified as Generalized Juvenile Periodontitis (GJP) and Rapidly Progressive Periodontitis.

Agranulocytosis — Agranulocytosis is characterized by a reduction in the number of circulating granulocytes and results in severe infection including ulcerative necrotizing lesions of the oral mucosa, skin, gastrointestinal and genitourinary tracts. *—Carranza*

Alleles — Variations in the nucleotide sequence at a locus is termed as alleles.

Allograft or Homograft — A tissue transfer between individuals of the same species but with non-identical genes.

Allograft — A graft between genetically dissimilar members of the same species. *—Periodontal Literature Review*

Alloplast Graft — A graft of inert synthetic material, which is sometimes called, implant material.

Alveolar Bone Proper — It is a plate of compact bone, the radiographic image of which is termed as Lamina dura. *—Genco*

Alveolar Bone Proper — It is the thin lamella of bone surrounding the root, seen as lamina dura in the radiographs (Opaque line). *—Grant*

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

Alveolar Process — Alveolar process is defined as the parts of the maxilla and mandible that form and support the sockets of the teeth. *—Lindhe*

Alveolar Process — It is that part of the jawbone which supports the teeth.

Alveolar Process — It is that portion of the maxilla and the mandible that form and support the sockets of the teeth. *—Carranza*

Alveolar Process — It is the part of the maxilla or mandible that forms and supports the teeth. *—Grant/ Stern /Listgarten*

Anaerobic — Growth in absence of oxygen.

Anemia — It is a deficiency in the quantity or quality of blood as manifested by reduction in the number of erythrocytes and in the amount of hemoglobin. *—Carranza*

Angular Chelitis — The commissure appears erythematous with surface crusting and fissuring.

Angular Defects — They are classified on the basis of the number of the osseous walls it may have one, two or three walls.

Angulation — Refers to the angle between the face of the bladed instrument and the tooth surface. Also known as tooth-blade relationship. *—Carranza*

Ankylosis — Fusion of the cementum and the alveolar bone with the obliteration of the periodontal ligament is termed as ankylosis. *—Carranza*

Antibiotics — Are a naturally occurring, semisynthetic or synthetic type of antimicrobial agent that destroys or inhibits the growth of selective microorganisms, generally at low concentrations.

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

Antibody — An immunoglobulin which binds to a specific antigen. Lymphocytes which have recognized an antigen may differentiate into plasma cells and secrete antibody against that antigen.

Antigen — Any following material that is specifically bound by antibody.

Antigen — A structure recognized as foreign by the immune system. The word is derived from antibody generator.

Antigenicity Capacity of the foreign agent to combine with or be recognized by metabolites or products of immune response.

Antimicrobial Agent It is a chemotherapeutic agent that works by reduction in bacterial number.

Antiseptic Are chemical antimicrobial agents that are applied topically or subgingivally to mucous membrane wounds, intact dermal surfaces to destroy microorganisms and inhibit their metabolism and reproduction. *—Carranza*

Apparent Position It is the level of the crest of the gingival margin.

Attached Gingiva Width of the attached gingiva—it is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket. *—Carranza*

Attached Gingiva The attached gingiva is continuous with the marginal gingiva. It is firm, resilient and tightly bound to the underlying periosteum of the alveolar bone. *—Carranza*

Attrition Is occlusal wear resulting from functional contacts with opposite teeth. *—Carranza*

Autograft Tissue transferred from one position to another within the same individual.

Bacterial Succession The appearance and disappearance of species as mixed flora evolves.

Biofilm It describes the relatively indefinable microbial community associated with a tooth surface or any hard non shedding material. *—Wilderer and Charaklis [1989]*

Biofilm It is defined as matrix enclosed bacterial population adherent to each other and/or to surface or interfaces. *—Costerton*

Biofilms Biofilms are natural communal aggregations of microorganisms that may form on wide range of surfaces. *—Lindhe*

Bio-integration A bonding of the living bone to the surface of an implant which is independent of any mechanical interlocking mechanism. *—Periodontal Literature Review*

Biologic Depth It is the distance between the gingival margin and the base of the pocket. (Coronal end of the junctional epithelium). *—Carranza (it can be measured only in carefully prepared and adequately oriented histological section)*

Biologic Width It is defined as; the dimension of space that the healthy gingival tissues occupy above the alveolar bone. *—Carranza*

Bruxism or occlusal necrosis It is defined as the clenching or grinding of the dentition during nonfunctional movements of the mastication system. *—Periodontal Literature Review*

Bruxism It is the clenching /grinding of teeth when individual is not chewing or swallowing.

Bulbous Bone Contours Bulbous bone contours are bony enlargements caused by exostosis, adaptation to function, or buttressing bone formation. Commonly found in maxilla.

Bullous Pemphigoid It is a chronic autoimmune subepidermal, bullous disease with tense bullae that rupture and become flaccid in the skin. *—Carranza*

Bundle Bone The layer of the alveolar bone into which the principal fibers (Sharpey's fibers) are inserted. *—Lindhe*

Buttressing Bone Formation/Lipping Bone formation occurring in an attempt to buttress bony trabeculae weakened by resorption.

Buttressing Bone Formation If it occurs within jaw, it is termed central buttressing bone formation.

Buttressing Bone Formation When it occurs on external surface, it is referred to as peripheral buttressing bone formation.

Bystander damage Accidental damage to host tissues caused by complements, neutrophils and macrophages during inflammation.

Calculus A hard concretion that forms on teeth or dental prosthesis through calcification of bacterial plaque *—Periodontal Literature Review*

Calculus Calculus is a calcified plaque. *—Manson and Eley*

Calculus Calculus is essentially mineralized plaque covered on its external surface by vital, tightly adherent, nonmineralized plaque. *—Genco*

Calculus Consists of mineralized bacterial plaque that forms on the surface of the natural teeth and dental prosthesis. *—James E Hinrichs (Carranza)*

Calculus Is a calcified mass which forms on and adheres to the surface of teeth and other solid objects in the mouth, e.g. restoration and denture.

Calculus It is defined as a hard deposit that forms by mineralization of dental plaque and is generally covered by a layer of unmineralized plaque. *—Carranza*

Calculus When dental plaque calcifies the resulting deposit is called Dental calculus. These calcified deposits occur as hard, firmly adhering masses on the clinical crowns of the teeth. *—Grant*

Capnophilic Organisms which require greater concentration of carbon dioxide. *—Periodontal Literature Review.*

Carrier Molecule Is a part of the antigen recognized by T-lymphocytes. *—Grant*

Cell-mediated Immunity An immune reaction mediated by T-cells.

Cell-mediated Immunity An immune reaction mediated by T-lymphocytes (activated lymphocytes release biologic response modifiers (lymphokines) on exposure to antigen.

Cellular Cementum/ Secondary Cementum Cementum that contains cells in lacunae located within the mineralized matrix. *—Grant/ Stern/Listgarten*

Cellular Cementum/ Secondary Cementum Cementum that forms after tooth eruption and in response to functional demand. *—Lindhe*

Cementum Cementum is a calcified connective tissue which covers the root dentine and into which periodontal fiber bundles are inserted. *—JD Manson & BM Eley*

Cementum Cementum is a calcified mesenchymal tissue that forms the outer covering of the anatomic root.

Cementum Cementum is a hard bone like tissue covering the anatomic roots of the teeth. *—Genco*

Cementum Cementum is a hard bone like tissue covering the root surfaces and occasionally small portions of the crown of the teeth. *—Lindhe*

Cementum Cementum is mineralized connective tissue that covers the roots of the teeth. *—Grant/ Stern/Listgarten*

Chemotaxis Locomotion of cells which is directed along an increasing chemical gradient of a soluble substance.

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

Chemotherapeutic Agents General term used for a chemical substance that provide chemical therapeutic benefit.

Chronic Gingivitis Is slow in onset and is of long duration, and is painless unless complicated by acute or subacute exacerbations. *—Carranza*

Chronic Periodontitis An infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss. *—Carranza*

Chronic Trauma from Occlusion It results from gradual changes in occlusion produced by tooth wear, drifting movement, extrusion of teeth, etc. *—Carranza*

Clenching It is the closure of the jaws under vertical pressure.

Coatings of Developmental Origin Those normally formed as part of tooth development (reduced enamel epithelium, dental cuticle). *—Carranza*

Coefficient of Separation The length of root cones in relation to the length of root complex. *—Lindhe*

Combined Lesion Combined lesion occurs when pulpal necrosis and periapical lesion occurs on a tooth that also is periodontally involved. *—Carranza*

Combined Osseous Defect The number of walls in the apical portion of the defect may be greater than that in its occlusal portion.

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

Complement It is an interacting network of about 30 membrane associated cell receptors and soluble serum glycoprotein. *—Carranza*

Compliance Action in accordance with recommendations.

Compromise Osseous Reshaping It indicates a bone pattern that cannot be improved without significant osseous removal that would be detrimental to overall result. *—Carranza*

Contact Inhibition Inhibition of movement and division caused by physical contact between cells of the same type.

Contact Inhibition The process by which the graft material prevents apical proliferation of the epithelium.

Coronal Cementum Cementum that is found over enamel. *—Grant/ Stern/Listgarten*

Curettage It is used in Periodontics for scraping of the gingival walls of the periodontal pocket to separate the diseased soft tissue.

Curettes Are manual instruments with cutting edge on both sides of the blade and rounded toe.

Curtain procedure A procedure designed to retain the marginal portion of the facial and interproximal gingiva for esthetic purpose in surgical treatment of periodontal pockets usually in maxillary anterior region by means of a palatal approach. The facial gingiva will be coronal to the palatal margin, thus creating a curtain.

Cuticle Thin, acellular structure with a homogenous matrix, sometimes enclosed within clearly demarcated, linear borders. *—Carranza*

Cyst Cyst is a swelling consisting of fluid in a sac, which is lined by epithelium or endothelium. *—Bailey and Love*

Cytokine A small protein messenger released by cells which affects the division, differentiation and function of other cells, which may be of the same or different types.

Debridement The removal of inflamed, devitalized or contaminated tissue or foreign material from or adjacent to a lesion.

Definitive Osseous Reshaping Implies that further osseous reshaping would not improve the overall result.

Degree of Separation It is the angle of separation between two roots. *—Lindhe*

Dehiscence Alveolar dehiscence is a dipping of the crestal bone margin exposing the root surface. *—Grant/ Stern /Listgarten*

Dehiscence Denuded surfaces involving the marginal bone, the defect is called as dehiscence. *—Carranza*

Dental Calculus It is a tightly adherent plaque that has undergone mineralization.

Dental Calculus It is an adherent, calcified or calcifying mass that forms on the surfaces of teeth and dental appliances.

Dental Epidemiology Is the study of pattern (distribution) and dynamics of dental diseases in a human population. *—Carranza*

Dental Implant Dental implant is perimucosal devices that is biocompatible and bio-functional and is placed on or within the bone associated with the oral cavity to provide support for fixed or removable prosthesis.

Dental Plaque Dental plaque comprises of complex microbial communities that form on virtually all surfaces of the teeth exposed to the bacteria-laden fluids of the mouth. *—Robert Wirthilin and Gary C Armitage*

Dental Plaque Dental plaque is an adherent, intercellular matrix consisting primarily of proliferating microorganisms, along with a scattering of epithelial cells leukocytes and macrophages.

Dental Plaque Dental plaque is defined as a soft deposit that forms the biofilm adherent to the tooth surface or other hard surfaces in the oral cavity including removable and fixed restorations. *—Carranza*

Dental Plaque It is a host associated biofilm.

Dental Splint An appliance designed to immobilize and stabilize loose teeth.

Dental Stain Pigmented deposits on tooth surface. *—Carranza*

Dentifrices Agent that aids in cleaning and polishing tooth surfaces.

Desmosome A specialized attachment between epithelial cells which impart strength to epithelium.

Desquamative Gingivitis Coined in 1932 by Prinz, to describe a peculiar condition characterized by intense erythema, desquamation, and ulceration of the free and attached gingiva. *—Carranza*

Diagnosis It may be defined as identifying disease from an evaluation of the history, signs and symptoms, laboratory tests and procedures.

Differentiation Differentiation involves the process of keratinization, which consists of sequence of biochemical and morphologic events that occur in the cell as it migrates from the basal layer. *—Carranza*

Diffuse Gingivitis It affects gingival margin, the attached gingiva and the interdental papillae. *—Carranza*

Disclosing Agents They are solutions or wafers capable of staining bacterial deposits on the surface of teeth, tongue and gingiva.

Disease Atrophy Nonfunctional and hypofunction may be expressed as disease atrophy. The disease of the periodontal tissue is characterized by a rearrangement of fibers of the periodontal ligament and of the trabeculation of the alveolar bone without gingival recession. *—Grant*

Disinfectant An antimicrobial agent that is generally applied to inanimate surfaces to destroy microorganisms.

Displaced Flap It is placed apically, coronally or laterally to their original position. *—Carranza*

Disuse or Functional Atrophy When occlusal forces are reduced, the number and thickness of the trabeculae are reduced, with atrophy of periodontal ligament appearing thinned, with reduction in number of fibers and density. *—Carranza*

Divergence It is the distance between two roots (cones); this distance normally increases age in apical direction. *—Lindhe*

Down's Syndrome Is a congenital disease caused by a chromosomal abnormality and characterized by mental deficiency and growth retardation.

Ecological Niche The functional position which bacterial species occupies within a complex ecosystem such as plaque.

Electrocoagulation It provides a wide range of coagulation or hemorrhage control obtained by using electrocoagulation current.

Electrosection Electrotomy or acusection is used for incision, excision and tissue planing.

Electrodesiccation Uses dehydrating current and is least used.

Electrofulguration A method of electrosurgery used to produce superficial desiccation of tissue, which employs a highly or moderately damped alternating current that is radiated through a monoterminal active electrode that is held close to the patient so that the sparks spray over the lesion being treated.

Electrosurgery (Radiosurgery) It is currently used to identify surgical techniques performed on soft tissue using controlled high frequency electrical current in the range of 1.5-7.5 million cycles/sec or MHz.

Electrosurgery Division of tissue by high frequency electrical current applied locally with a metal instrument or needle.

Endodontic-periodontal Lesions Pulpal necrosis precedes periodontal changes. A periapical lesion originating in pulpal inflammation and necrosis may drain to the oral cavity through periodontal ligament and adjacent alveolar bone. *—Carranza*

Endotoxin A complex heat stable toxin which is a structural component of gram-negative bacterial cell wall. The active component is a lipopolysaccharide.

Epidemiology The study of the distribution and determinants of health-related states or events in specified populations and the application of this study to control health problems. *—Carranza*

Epithelial Adaptation It is the close apposition of the gingival epithelium to the tooth surface without obliteration of the pocket.

Epulis It is the generic term to designate all discrete tumor and tumor-like mass of gingiva. *—Carranza*

Erosion (Cuneiform Defect) It is a sharply defined wedge shaped depression in the cervical area of the facial tooth surface. *—Carranza*

Etiology The cause of disease, in the case of gingivitis and periodontal disease, microbial dental plaque.

Evidence-based Therapy Current concept of evaluating health care requires a scientific basis for treatment.

Excisional New Attachment Procedure It is a definitive subgingival curettage performed with a knife.

Excursive Movement Any movement of the mandible away from intercuspal position.

Exostosis Exostosis is outgrowths of the bone of varied size and shape.

Exploratory Stroke It is a light "feeling" stroke that is used with probes and explorer to evaluate the dimensions of the pocket and to detect calculus and irregularities of the tooth surface.

Exposure It is defined as a factor that may possibly lead to disease or may be protective against a disease. *—Mauricio Ronderers*

Extrusion Pathologic migration in the occlusal or incisal direction is termed as extrusion. *—Carranza*

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

Facultative Anaerobe Growth in either an aerobic or anaerobic environment.

Fc The part of an immunoglobulin molecule which binds to specific receptors on neutrophils and macrophages, but which does not bind to antigen.

Fenestration It is a circumscribed hole in the cortical plate over the root and does not communicate with the crestal margin. *—Grant/ Stern /Listgarten*

Fenestration An isolated area in which the root is denuded of the bone and the root surface is covered only by the periosteum and the overlying gingiva are termed fenestration. Here the margin is intact. *—Grant/ Stern /Listgarten*

Fibroma Arises from the gingival connective tissue or from the periodontal ligament. *—Carranza*

Fibronectin A connective tissue protein which binds cells to the extracellular matrix.

Fibro-osseous Integration In which soft tissue such as fibers and/ or cells, are interposed between the two surfaces.

Fimbriae A fine filamentous surface appendage on a bacterium. These are important for attachment and adhesion and are important antigens on some species.

Finger Rest The finger rest serves to stabilize the hand instrument by providing a firm fulcrum as movement is made to activate the instrument.

Flap A loosened section of the tissue separated from the surrounding tissue except at its base. *—Periodontal Literature Review*

Flat Architecture It is reduction of the interdental bone to the same height as radicular bone. *—Carranza*

Food Impaction It is the forceful wedging of food into the periodontium by occlusal forces. *—Carranza*

Food Impaction The forceful wedging of the food into the interproximal space by chewing pressure or the forcing of food interproximally by tongue or cheek pressure.

Fremitus A palpable or visible movement of teeth when subjected to occlusal forces.

Fremitus It is the measurement of the vibratory patterns of the teeth when teeth are placed in contacting positions and movements. *—Genco*

Frenectomy It is complete removal of the frenum, including its attachment to underlying bone and may be required in correction of an abnormal diastema between maxillary central incisors.

Frenotomy It is the incision of frenum. *—Carranza*

Frenum It is a fold of mucous membrane usually with enclosed muscle fibers that attaches the lip and cheeks to the alveolar mucosa and / or gingiva and underlying periosteum.

Full Thickness Flap All the soft tissue, including the periosteum is reflected to expose the underlying bone.

Furcational Entrance It is the transitional area between the divided and undivided part of the root. *—Lindhe*

Furcational Fornix The roof of the furcation.

Furcation Invasion Pathologic resorption of bone within the furcation. *—Periodontal Literature Review*

Furcation Involvement Furcation involvement refers to the invasion of the bifurcation and trifurcation of multirooted teeth by periodontal disease. *—Carranza*

Furcation Furcation is defined as the area located between individual root cones. *—Lindhe*

Furcation It is the anatomic area of a multirooted tooth where the roots diverge. *—Periodontal Literature Review*

Generalized Aggressive Periodontitis Clinically characterized by "generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors". *—Carranza*

Generalized Diffuse Gingivitis Involves the entire gingiva of the entire mouth.

Generalized Gingivitis Involves the entire mouth.

Generalized Marginal Gingivitis Involves the gingival margins in relation to all the teeth. *—Carranza*

Generalized Periodontitis It is considered generalized when >30% of the sites assessed in the mouth demonstrate attachment loss and bone loss.

Genetic Marker Genetic marker refers to any gene or nucleotide sequence that can be mapped to a specific location or region on a chromosome. *—Carranza*

Genotype The generic composition of an organism.

Gingiva Gingiva is the part of the oral mucosa that covers the alveolar process of the jaws and surrounds the neck of the teeth. *—Genco*

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. *—Periodontal Literature Review*

Gingiva The gingiva is the part of the masticatory mucosa, which covers the alveolar process and surrounds the cervical portion of the teeth. *—Lindhe*

Gingiva The gingiva is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth. *—Carranza*

Gingiva The gingiva is the part of the oral mucous membrane attached to the teeth and the alveolar processes of the jaws. *—Grant/ Stern/Listgarten*

Gingiva The gingiva is that part of the oral mucosa which surrounds the tooth and covers the alveolar ridge. *—Manson & Eley*

Gingival Abscess Gingival abscess is a localized, painful, rapidly expanding lesion, which is usually of sudden onset. It is generally limited to the marginal gingiva or interdental papilla. *—Carranza*

Gingival Abscesses Gingival abscesses are those that are localized to the marginal gingiva or interdental papilla. *—Gary C Armitage*

Gingival Abscesses Localized in the gingiva, caused by injury to the outer surface of the gingiva and not involving the supporting structures. *—Carranza*

Gingival Crevicular Fluid GCF is an altered serum transudate found in the gingival sulcus. *—Genco*

Gingival Crevicular Fluid Tissue fluid that seeps through the crevicular epithelium. It is increased in the presence of inflammation. *—Periodontal Literature Review*

Gingival Curettage It consists of the removal of inflamed soft tissue lateral to pocket wall.

Gingival Curettage It means the scraping of the gingival wall of the periodontal pocket to separate diseased soft tissue.

Gingival Cyst Found within the gingiva, most commonly in the canine—premolar region. *—Periodontal Literature Review*

Gingival Enlargement Increase in the size of the gingiva.

Gingival Hyperplasia Defined as an enlargement of gingiva due to the increase in the number of cells. *—Periodontal Literature Review*

Gingival Pocket (Pseudo Pocket) This type of pocket is formed by gingival enlargement without destruction of the underlying periodontal tissues.

Gingival Recession Is the exposure of root surface due to apical shift in the marginal gingiva. *—Grant*

Gingival Sulcus The gingival sulcus is the shallow crevice or space around the tooth bounded by the surface of the tooth on one side and the epithelia lining the free margin of the gingiva on the other. *—Carranza*

Gingivectomy Excision of a portion of the gingiva. *—Periodontal Literature Review*

Gingivectomy It means excision of gingiva. *—Carranza*

Gingivitis Inflammation of the gingiva.

Gingivoplasty Gingivoplasty is “a surgical reshaping of the gingiva”.

Gingivoplasty It is the reshaping of the gingiva to create physiologic gingival contour, with the sole purpose of recontouring the gingiva in the absence of periodontal pocket. *—Carranza*

Glycosaminoglycan A molecule of repeating sugar subunits which forms a major component of connective tissue ground substance.

Gnotobiotic Animal A laboratory animal whose microbial flora is known. Specific microorganisms are introduced into germ-free animals to make them gnotobiotic.

Gracey Curettes Set of many curettes designed for specific area and surfaces.

Graft Any tissue or organ used for implantation or transplantation.

Graft It is a viable tissue/ organ that after removal from donor site are implanted within the host tissue, which is then repaired, restored and remodeled.

Guidance Pattern of opposing tooth contact during excursive movements of the mandible. The teeth making such contact cause separation of the other teeth.

Guided Tissue Regeneration An epithelial exclusionary technique that promotes new connective tissue attachment without the use of any implant material.

Guided Tissue Regeneration Procedures attempting to regenerate lost periodontal structures through differential tissue responses.

Habit An act of repeated performance, almost automatic, such as bruxism or tongue thrusting.

Halitosis Also termed as fetor ex ore and oral malodor, is foul or offensive odor, emnating from oral cavity. *—Carranza*

Hapten Is a part of antigen recognized by B-lymphocytes. *—Grant*

Hemidesmosome Structure which attaches epithelial cells to the basement membrane. It resembles half a desmosome, but has different structural components.

Hemisection It is surgical removal of a root with the associated part of crown.

Hemisection It is the splitting of the two-rooted tooth into two separate portions. The process has been called Bicuspidization or Separation.

Hemiseptum The one wall defect is also called as hemiseptum.

Homeostasis The production of a stable equilibrium in a body system by means of feedback mechanisms.

Horizontal Bone Loss Common pattern of bone loss in which bone is reduced in height, but the bone margins roughly perpendicular to the tooth surface.

Hydrolytic Enzymes A class of enzymes which cleaves various types of bond, including peptide, ester and glycosidic bonds, with the addition of water. Many degradative and lysosomal enzymes fall into this category.

Hypercementosis It refers to the prominent thickening of the cementum. It may be localized or generalized. *—Carranza*

Hyperfunction Is a functional adaptation in which additional structural elements are formed on cementum and in bone to withstand the added force expressed clinically and microscopically as a thickened lamina dura, buttressing bone formation, osteosclerosis and cemental spurs. *—Grant*

Hypersensitivity Root surfaces exposed by gingival recession may be hypersensitive to thermal changes or tactile stimulation.

Iatrogenic Factors Inadequate dental procedure that contribute to the deterioration of the periodontal tissues. *—Carranza*

Immune Complex A complex of antigen and antibody molecules bound to one another and together.

Immunogenicity It is the capacity to induce a detectable immune response such as the production of antibody or the stimulation of cellular immunity.

Immunoglobulin A glycoprotein composed of heavy and light peptide chains; functions as antibody in serum and secretions. *—Periodontal Literature Review*

Implant 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 and esthetic purposes.

Implant To insert a graft or alloplastic device into the oral hard or soft tissue for replacement of missing or damaged anatomic parts or stabilization of periodontally compromised tooth or group of teeth. *—Periodontal Literature Review*

Inadvertent Curettage Some degree of curettage is done unintentionally while scaling and root planing is performed. *—Carranza*

Inflammation A cellular and vascular reaction of tissue to injury. *—Periodontal Literature Review*

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 injured tissue. *—Periodontal Literature Review*

Inflammation Is an observable alteration in tissue associated with changes in vascular permeability and dilation, often with the infiltration of leukocytes into affected tissue. *—Periodontal Literature Review*

Intercuspal Position (ICP) The position of the mandible when there is maximum intercuspation between the maxillary and mandibular teeth. *—Synonym-Centric occlusion*

Interdental Gingiva The interdental gingiva occupies the gingival embrasure, which is the interproximal space beneath the area of tooth contact. *—Carranza*

Interference Any contact in ICP or excursion that prevents the remaining occlusal surfaces from achieving stable contact. *—Synonym-Supra contact*

Interleukins Cytokine which mediate communication between leukocytes.

Intermediate Cementum It is an ill-defined zone near cementodentinal junction of certain teeth that appear to contain cellular remnants of Hertwig's sheath embedded in calcified ground substance. *—Carranza*

Intrabone/Intrabony/ Supracrestal Pocket — Bottom of the pocket is apical to the level of the adjacent alveolar bone. *—Carranza*

Intrabony Defects — The three-wall vertical defect was originally called intrabony defect.

Junctional Epithelium — A single or multiple layer of non-keratinizing cells adhering to the tooth surface at the base of the gingival crevice. *—Periodontal Literature Review*

Junctional Epithelium — It denotes the tissue that joins to the tooth on one side and to the oral sulcular epithelium and connective tissue on the other.

Juvenile Periodontitis — A disease of 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.

Lamina Dura — The compact bone (Alveolar bone proper), which lines the tooth socket, and in a radiograph appears as a dense opaque line is called as lamina dura. *—Lindhe*

Lateral Pressure — Refer to the pressure created when force is applied against the surface of a tooth with the cutting edge of a bladed instrument. *—Carranza*

Laterotrusion — Movement of the mandible laterally to the right or left from ICP.

Laterotrusive Movement — The side of either dental arch corresponding to the side of mandible moving away from the midline. *—Synonym-Working side*

Ledges — Ledges are plateau like bone margins caused by resorption of thickened bony plates. *—Carranza*

Leukemia — It is a malignant neoplasia of WBC precursors. *—Carranza*

Leukoplakia — A white patch or plaque that does not rub off and cannot be diagnosed as any other disease. *—World Health Organization*

Lichen Planus — It is a relatively common, chronic dermatosis characterized by the presence of cutaneous, violaceous papules that may coalesce to form plaques.

Lichen Planus — Is defined as unique cutaneous entity consisting of an eruption of papules distinct in color and configuration, in patterns and location of appearance and in microscopic and gross structure. *—Fitz Patrick*

Lipopolysaccharide — The active component of bacterial endotoxins.

Localized Gingivitis — It is confined to the gingiva of a single tooth or group of teeth.

Localized Aggressive Periodontitis — Is characterized as having localized first molar or incisor disease with proximal attachment loss on at least 2 permanent teeth, one of which is a first molar. *—Carranza*

Localized Diffuse Gingivitis — It extends from the gingival margin to the mucobuccal fold but is limited in area.

Localized Marginal Gingivitis — It is confined to one or more areas of the marginal gingiva.

Localized Periodontitis — It is considered localized when <30% of the sites assessed in the mouth demonstrate attachment loss and bone loss.

Loci — Loci are defined as the specific location on the chromosomes.

Lymphokine — Cytokine secreted by lymphocytes. Cytokine is now preferred term for this group of substances because they are secreted by other cell types as well.

Malnutrition — Any disorder of nutrition, it may be due to imbalance or insufficient diet or to defective assimilation or utilization of foods. *—Dorland's*

Marginal Gingiva — The marginal or attached gingiva, is the terminal edge or border of the gingiva surrounding the teeth in a collar-like fashion. *—Carranza*

Marginal Gingivitis — Inflammation of the gingival margin and may include a portion of the contiguous attached gingiva.

Marginal Plaque — Plaque is in direct contact with the gingival margin and also referred to as marginal plaque. *—Carranza*

Marginal Plaque — It is the supragingival plaque that is in direct contact with the gingival margin.

Masticatory Mucosa The gingival and the mucosal covering of the hard palate.

Materia Alba Is a yellowish or whitish, soft loose deposit found in neglected mouths comprising of a mass of microorganisms, desquamated epithelial cells, food debris, leukocytes plus salivary deposits. *—J.D Manson and B.M Eley*

Materia Alba Is a deposit composed of aggregates of microorganisms, leukocytes, dead, exfoliated epithelial cells attached to surfaces of the teeth, plaque and gingiva. *—Saul Schluger*

Materia Alba It refers to the soft accumulation of bacteria and tissue cells that lack the organized structure of dental plaque and are easily displaced with a water spray. *—Carranza*

McCall's Festooning Are life preserver-shaped enlargements of the marginal gingiva that occurs most frequently in the canine and premolar areas on the facial surface.

Mediotrusive Side The side of either dental arch corresponding to the side of mandible moving towards the midline. *—Synonym-Balancing side, non-working side*

Metalloproteinases Enzymes which degrade protein in the presence of metal ions, usually calcium or magnesium.

Micro-aerophilic Organisms which grow best in an atmosphere of reduced oxygen.

Microbial Dental Plaque Is described as aggregations of bacteria that are tenaciously attached to the teeth and other surfaces. *—Genco*

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

Mobility Is a measurement of horizontal and vertical tooth displacement created by examiner force.

Moderate Periodontitis Periodontal destruction is generally considered moderate when 3 to 4 mm of clinical attachment loss has occurred.

Modified Pen Grasp The thumb, index finger, and middle finger are used to hold the instrument as a pen is held, but the middle finger is positioned so that the side of the pad next to the finger nail is resting on the instrument shank. The index finger is bent at the second joint from the finger tip and is positioned well above the middle finger on the same side of the handle. *—Carranza*

Modified Widman Flap A scalloped, replaced, mucoperiosteal flap, accomplished with an internal bevel incision that provides access for root planing. *—Periodontal Literature Review*

Monocyte A large mononuclear phagocytic leukocyte, the circulating precursors of macrophages.

Mucogingival Deformity It is defined as "a significant departure from the normal shape of gingiva and alveolar mucosa" and may involve the underlying alveolar bone.

Mucogingival It is defined as "a generic term used to describe the mucogingival junction and its relationship to the gingiva, alveolar mucosa, frenula, muscle attachments, vestibular fornices and the floor of the mouth".

Mucogingival Surgery Is defined as periodontal surgical procedures designed to correct defects in the morphology, position, and/or amount of gingiva. *—Carranza*

Mucogingival Surgery Periodontal surgical procedures used to correct defect in the morphology, position and or amount of gingiva. *—Periodontal Literature Review*

Mucogingival Surgery Surgical procedure for the correction of relationships between the gingiva and oral mucous membrane with reference to three specific problems. Those associated with attached gingiva, shallow vestibule and a frenum interfering with the marginal gingiva.

Mucosa A mucous membrane.

Mucous Membrane Pemphigoid Also known as Cicatricial Pemphigoid, is a chronic, vesiculobullous autoimmune disorder of unknown cause that predominantly affects women in the fifth decade of life. *—Carranza*

Multifactorial Diseases whose etiologies include both genetic and environmental factors.

Muscular Contact Position (mcp) The position of the mandible when lifted into contact from resting position.

Necrotizing Ulcerative Periodontitis Is an extension of necrotizing ulcerative gingivitis (NUG) into the periodontal structures, leading to attachment and bone loss.

Necrotizing Ulcerative Gingivitis It is an inflammatory destructive disease of gingiva, which presents characteristic signs and symptoms. *—Carranza*

Necrotizing Ulcerative Periodontitis It is defined as 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. *—Periodontal Literature Review*

Negative Architecture Refers to, if interdental bone is more apical than radicular bone.

Neutrophil A phagocytic polymorphonuclear leukocyte.

New Attachment It is the embedding of new periodontal ligament fibers into new cementum and the attachment of the gingival epithelium to the tooth surface previously denuded by disease. *—Carranza*

New Attachment It is the union of the connective tissue or epithelium with a root surface that has been deprived of its original attachment apparatus. *—Periodontal Literature Review*

Non-displaced Flap When the flap is returned and sutured in its original position.

Non-thrombocytopenic Purpura It occurs as a result of either vascular wall fragility or thrombasthenia.

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

Offset Blade To describe gracey curettes, because they are angled approximately 60-70 degree from the lower shank.

Opsonin A substance capable of enhancing phagocytosis. *—Periodontal Literature Review*

Opsonin A substance which facilitates phagocytosis when bound to the surface of a bacterium or other particle.

Oral Candidiasis Infection of oral mucous membrane by a fungus of the genus *Candida.*

Oral Epithelium Oral epithelium covers the crest and outer surface of the marginal gingiva and the surface of the attached gingiva.

Oral Epithelium The oral or outer epithelium covers the crest and outer surface of the marginal gingiva and the surface of the attached gingiva. *—Carranza*

Oral Mucosa The tissue lining the oral cavity.

Oral or Outer Epithelium The outer or oral epithelium covers the crest and outer surface of the marginal gingiva and the surface of the attached gingiva. *—Carranza*

Osseointegration A direct contact on the light microscopic level, between living bone tissue and an implant.

Osseous Craters Osseous craters are concavities in the crest of the interdental bone, confined within the facial and lingual walls.

Osseous Defects Different types of bone deformities can result from periodontal disease.

Osseous Surgery It is defined as a procedure by which changes in the alveolar bone can be accomplished to rid it of deformities induced by the periodontal disease process or other related factors such as exostosis and tooth supraeruption. *—Carranza*

Osseous Surgery Periodontal surgery involving modification of bony support of the teeth. *—Periodontal Literature Review*

Ostectomy Includes removal of tooth supporting bone. *—Carranza*

Ostectomy It is defined as the excision of bone or portion of bone in periodontics; Ostectomy is done to correct or reduce deformities caused by periodontitis and includes removal of the supporting bone.

Osteoclast Activating Factor (OAF) A mixture of cytokines, including IL-1, TNF, TNF-β, and PDGF, which induces bone resorption and was previously thought to be a distinct cytokine.

Osteoconduction It is a physical effect by which the matrix of the graft forms scaffold that favors outside cells to penetrate the graft and form new bone.

Osteoconduction The graft material acts as passive material, like a trellis or scaffolding for new bone to cover.

Osteogenesis Development of the bone; Formation of bone.

Osteogenesis Refers to formation or development of new bone by cells contained in the graft.

Osteoinduction A process by which graft material is capable of promoting cementogenesis, osteogenesis and new periodontal ligament.

Osteoinduction It is a chemical process by which molecules contained in the graft (BMPs) convert the neighboring cells into osteoblast, which in turn form bone.

Osteoplasty Thinning of the buccal bone, using diamond burs, included as part of the surgical technique, results in areas of bone necrosis with reduction in the bone height, which is later remodeled by new bone.

Osteoplasty It is defined as the reshaping of the alveolar process to achieve a more physiologic form without removal of supporting bone.

Osteoplasty Refers to reshaping the bone without removing tooth supporting bone. *—Carranza*

Osteoplasty Reshaping the alveolar process to achieve a more physiologic form without removal of the supporting bone. *—Periodontal Literature Review*

Papillary Gingivitis It involves interdental papillae and often extends into the adjacent portion of the gingival margin.

Papillon-Lefévre Syndrome Is characterized by hyperkeratotic skin lesions, severe destruction of the periodontium and in some cases, calcification of the dura.

Partial Thickness Flap Includes only the epithelium and a layer of the connective tissue. *—Carranza*

Passive Eruption Passive eruption is the exposure of the teeth by apical migration of the gingiva. *—Carranza*

Pathogenesis The mechanism by which a disease develops.

Pathologic Migration Refers to tooth displacement that results when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease. *—Carranza*

Pellicle A thin biofilm. *—Genco*

Pellicle An organic coating formed when the crown of the teeth emerges and is exposed to saliva, which is called dental pellicle. *—Grant/ Listgarten*

Pellicle Pellicle is defined as tooth or mucosal adherent salivary protein. *—Periodontal Literature Review*

Pellicle The earliest layer of plaque composed of salivary glycoprotein, which is later colonized by bacteria.

Pemphigoid Applies to a number of cutaneous, immune-mediated, subepithelial bullous diseases that is characterized by a separation of the basement membrane zone. *—Carranza*

Pemphigus Vulgaris Are a group of autoimmune bullous disorders that produce cutaneous and/ or mucous membrane blisters. *—Carranza*

Peri-implant Mucositis Inflammatory changes confined to the soft tissue surrounding an implant. *—Carranza*

Peri-implantitis A term used to describe inflammation around a dental implant and/ or its abutment. *—Periodontal Literature Review*

Peri-implantitis Progressive peri-implant bone loss in conjunction with a soft tissue inflammatory lesion is termed as peri-implantitis. *—Carranza*

Periodontal Abscess It is a localized accumulation of pus within gingival wall of a periodontal pocket.

Periodontal Abscess Localized purulent inflammation in the periodontal tissues. *—Carranza*

Periodontal Abscess Periodontal abscess are acute lesions that may result in very rapid destruction of the periodontal tissues. *—Carranza*

Periodontal Abscess Periodontal abscess are purulent infections localized to the gingival, periodontal or pericoronal regions. *—Carranza*

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

Periodontal Disease Comprises of a group of inflammatory conditions of the supportive tissues of the teeth that are caused by bacteria. *—Carranza*

Periodontal Dressing/Pack A protective material applied over the wound created by periodontal surgical procedures. *—Periodontal Literature Review*

Periodontal Flap Periodontal flap is a section of the gingiva and/or the mucosa surgically separated from the underlying tissues to provide visibility and access to the bone and the root surface. *—Carranza*

Periodontal Healing/Regeneration Restoration of lost periodontium.

Periodontal Instruments They are designed for specific purposes, such as removing calculus, planing root surfaces, curetting the gingiva, removing diseased tissue.

Periodontal Ligament The periodontal ligament is a dense, fibrous connective tissue attaching the tooth to the alveolar bone. *—Grant/ Stern /Listgarten*

Periodontal Ligament The periodontal ligament is the connective tissue that surrounds the root and connects it to the bone. *—Carranza*

Periodontal Ligament The periodontal ligament is the soft, richly vascular and cellular connective tissue which surrounds the roots of the teeth and joins the root cementum with the lamina dura or the alveolar bone proper, or the socket wall. *—Lindhe*

Periodontal Ligament The periodontal ligament is the tissue that surrounds the roots of the teeth and attaches it to the bony alveolus.

Periodontal Plastic Surgery It is defined as the surgical procedures performed to correct or eliminate anatomic, developmental or traumatic deformities of the gingiva or alveolar mucosa.

Periodontal Pocket Gingival sulcus deepening can occur by coronal movement of gingival margin, apical displacement of gingival attachment or combination of two processes.

Periodontal Pocket Periodontal Pocket is defined as “a pathologically deepened gingival sulcus”. *—Carranza*

Periodontal Pocket Pocket occurred with the destruction of the supporting periodontal tissues.

Periodontal Splint It is an appliance used for maintaining or stabilizing mobile teeth in their functional position.

Periodontal Surgery It is defined as intentional severing or incising of gingival tissue with the purpose of controlling or eliminating periodontal disease.

Periodontal Trauma It is a morbid condition produced by repeated mechanical force exerted on the periodontium exceeding the physiologic limit of tissue tolerance and contributing to a breakdown of supporting tissues of tooth. *—Genco*

Periodontal-Endodontic Lesion The bacterial infection with periodontal pocket associated with loss of attachment and root exposure may spread through accessory canals to the pulp, resulting in the pulpal necrosis. *—Carranza*

Periodontics The clinical science that deals with the periodontium in health and disease, is called periodontology, the practice of which is periodontics. *—Grant/ Stern/ Listgarten*

Periodontitis Inflammation of the supporting tissues of the teeth. An extension of the inflammation from gingiva into the adjacent bone and ligament. *—Periodontal Literature Review*

Periodontitis It is the most common type of periodontal disease and results from extension of the inflammatory process initiated in the gingiva to the supporting periodontal tissues.

Periodontitis Periodontitis is defined as "an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both".
—Carranza

Periodontitis Periodontitis is the inflammation combined with loss of attachment.
—Mark U. Thomas, Brain L. Mealey

Periodontium Periodontium is the functional unit of the tissues supporting the tooth.
—Grant/ Stern/ Listgarten

Periodontium The periodontium consists of the investing and supporting tissues of the tooth (gingiva, periodontal ligament, cementum, alveolar bone). *—Micheal G. Newman*

Periodontium The periodontium consists of those tissues that surround and anchor the tooth in the maxillary and mandibular alveolar process. *—Mark I Ryder*

Periodontology The clinical science that deals with the periodontium in health and disease is called periodontology, the practice of which is periodontics. *—Grant*

Periodontology The various diseases of the periodontium are collectively termed as periodontal diseases. Their treatment is referred to as periodontal therapy.

Periodontology The various diseases of the periodontium are collectively termed as periodontal disease. The branch of dentistry concerned with prevention and treatment of periodontal disease is termed periodontics or periodontia.

Phagocytosis Is the process by which cells ingest particles of a size visible by light microscopy.
—Carranza

Phase I Therapy To alter or eliminate the microbial etiology and contributing factor for gingival and periodontal disease.

Phenotype Collection of traits or characteristics.

Physiologic Mesial Migration With time and wear, the proximal contact areas of the teeth are flattened and the teeth tend to move mesially. This is referred to as physiologic mesial migration.

Physiologic Occlusion It is one that has demonstrated the ability to survive despite anatomic obliterations from the hypothetical normal or preconceived ideal form of occlusion and function.
—Genco

Plaque Control It is the removal of dental plaque on a regular basis and prevention of its accumulation on teeth and adjacent gingival surfaces.

Plaque An organized mass consisting mainly of microorganisms that adheres to teeth, prosthesis and oral surfaces and is found in the gingival crevice and periodontal pockets.
—Periodontal Literature Review

Plasma Cell A mature B lymphocyte which secretes antibody.

Positive Architecture Refers to radicular bone being apical to interdental bone.

Predictive Value Refers to the probability of the test results.

Primary Herpetic Gingivostomatitis It is an infection of the oral cavity caused by the herpes simplex virus type-1.

Primary Trauma from Occlusion It is the injury resulting from excessive occlusal forces applied to a tooth or teeth with normal support. *—Periodontal Literature Review*

Primary Trauma from Occlusion It includes a tissue reaction, which is elicited around a tooth with normal height of the periodontium. *—Lindhe*

Primary Trauma from Occlusion When trauma from occlusion is the result of alteration in the occlusal forces; it is called primary trauma from occlusion. *—Carranza*

Probing Depth It is a distance to which an ad hoc instrument (probe) penetrates into pocket.
—Carranza

Prognosis Prognosis is a prediction of the probable course, duration and outcome of the disease based on a general knowledge of the pathogenesis of the disease and the presence of the risk factors for the disease. *—Carranza*

Proliferation Proliferation of keratinocytes takes place by mitosis in the basal layer and less frequently in the suprabasal layers, where a small proportion of cells remain as a proliferative compartment while a larger number begins to migrate to the surface.

Proteoglycan A protein with numerous glycosaminoglycan side chains. A component of connective tissue ground substance.

Protrusion Movement of the mandible anteriorly from intercuspal position (ICP).

Provisional Prognosis It allows the clinician to initiate treatment of teeth that have a doubtful outlook in the hope that a favorable response may tip the balance and allow teeth to be retained.

Psychosomatic Disorders Harmful effects that result from psychic influences on the organic control of the tissues. *—Carranza*

Puberty Gingivitis A higher prevalence and severity of gingivitis and gingival enlargement is found in the circumpubertal period. *—Carranza*

Puetz-Jegher's Syndrome Produces intestinal polyposis, melanin pigmentation in the oral mucosa & lips. *—Carranza*

Pyogenic Granuloma It is a tumor like gingival enlargement that is considered an exaggerated conditioned response to minor trauma. *—Carranza*

Pyorrhea An archaic term for several periodontal diseases.

Radicular Cementum Cementum that covers the root.

Radius of Action Range of effectiveness of about 1.5-2.5 mm within which bacterial plaque can induce loss of bone. *—Page and Schroeder*

Reattachment It is used to refer the repair in areas of the root not previously exposed to the pocket, such as after surgical detachment of the tissue or the following traumatic tear in the cementum, tooth fracture or treatment of periapical lesions. *—Carranza*

Reattachment To attach again. The reunion of epithelial and connective tissue with root surfaces and bone such as occurs after an incision or injury. *—Periodontal Literature Review*

Recession Refers to the location of gingiva, not its condition.

Recurrent Gingivitis Reappears after having been eliminated by treatment or disappearing spontaneously. *—Carranza*

Redox Potential A measure of the ease with which oxidation and reduction reactions will occur.

Refractory Periodontitis According to *American Academy of Periodontology,* it has been defined as; those cases which do not respond to any treatment provided whatever the thoroughness or frequency.

Refractory Periodontitis It 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. *—Periodontal Literature Review*

Regeneration It is the growth and differentiation of new cells and intercellular substances to form new tissue and parts. *—Carranza*

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 function of the part. *—Periodontal Literature Review*

Repair It simply restores the continuity of the diseased tissue. *—Carranza*

Repair Simply restores the continuity of the diseased marginal gingiva and re-establishes a normal gingival sulcus at the same level on the root as the base of the pre-existing periodontal pocket. Healing by scar formation. *—Carranza*

Reverse Architecture Reversed architecture defects are produced by loss of interdental bone, including the facial plates, lingual plates or both, without concomitant loss of the radicular bone, thereby reversing the normal architecture.

Retrograde Periodontitis Periodontitis caused by pulpal infection that have entered the periodontal ligament either through the apical foramen or through the lateral canals.

Retrusion Movement of the mandible posteriorly from ICP.

Risk Factor Is defined as an aspect of personal behavior or lifestyle, an environmental exposure, or an inborn or inherited characteristic, that, on the basis of epidemiologic evidence, is known to be associated with health conditions considered important to prevent. *—The Dictionary of Epidemiology*

Risk Factor May be environmental, behavioral or biologic factor that when present increases the likelihood that an individual will get the disease. *—Carranza*

Risk Indicator A probable or putative risk factor that has been associated with the disease through cross-sectional studies.

Risk Marker A factor that is associated with increased probability of future disease.

Risk It is the probability that an individual will get a specific disease in a given period.

Root Amputation The removal of a root from mutirooted teeth.

Root Planing Stroke It is a moderate to light pull stroke that is used for final smoothening and planing of the root surface.

Root Planing It is the process by which residual embedded calculus and portion of cementum are removed from the roots to produce a smooth, hard and clean surface. *—Carranza*

Root Resection/Hemisection It is the splitting of two rooted tooth into two separate portions.

Root Resection It is the surgical removal of all or a portion of root before or after endodontic treatment.

Root Resection Surgical removal of all or portion of a tooth root.

Root Separation/Bicuspidisation It is the sectioning of the root complex and maintenance of all roots.

Saccharolytic Bacteria which break down carbohydrates for energy are saccharolytic.

Saliva Defined as the fluid secreted by the salivary glands that begins the digestion of food. *—Genco*

Scaling Stroke It is short, powerful pull stroke that is used with bladed instruments for the removal of supragingival and subgingival calculus.

Scaling Scaling is a process by which plaque and calculus are removed from both supragingival and subgingival tooth surface.

Secondary Trauma from Occlusion Injury resulting from normal occlusal forces applied to tooth or teeth with inadequate support. *—Periodontal Literature Review*

Secondary Trauma from Occlusion Tissue reaction elicited around a tooth by occlusal forces, with reduced height of periodontium. *—Lindhe*

Secondary Trauma from Occlusion When the trauma results from reduced ability of the tissues to resist the occlusal forces, it is known as secondary trauma from occlusion. *—Carranza*

Segregation Analyses Classic twin study—reared together monozygotic and dizygotic twins are compared to estimate the effects of shared genes. *—Carranza*

Segregation Analyses The observed pattern of disease in families is compared with patterns expected under various models of inheritance. *—Carranza*

Sensitivity Refers to ability of a test or observation to detect the disease whenever it is present.

Serumal Calculus Calculus formed apical to gingival margin, often brown or black, hard and tenacious. *—Periodontal Literature Review*

Severe Periodontitis Periodontal destruction is considered severe when 5 mm or more of clinical attachment loss has occurred.

Sickle Scaler Sickle scalers have a flat surface and two cutting edges that converge in a sharply pointed tip.

Slight (mild) Periodontitis Periodontal destruction generally considered slight when no more than 1 to 2 mm of clinical attachment loss has occurred.

Slough Necrotic tissue in the process of separating from the viable portion of the body.

Specific Immune Response The ability of T-cells and B-cells to recognize specific oligomeric structures on a pathogen and generate progeny that also recognize the structure, enables the immune system to respond more rapidly and effectively when exposed to that pathogenic agent.

Specificity Recognition of the structural features of an oligomer by receptors on immune cells. *—Carranza*

Specificity Refers to the ability of a test or observation to clearly differentiate one disease from another.

Splint Any apparatus, appliance, or device employed to prevent motion or displacement of fractured or movable parts.

Splint It is an appliance used for immobilization of injured or diseased parts.

Stillman's Clefts They are apostrophe shaped indentations extending from and into the gingival margin for varying distances on the facial surface.

Stippling Stippling is a form of adaptive specialization or reinforcement to function. *—Carranza*

Subgingival Calculus (Serumal Calculus) It is located below the crest of the marginal gingiva and not visible in routine clinical examination. *—Carranza*

Subgingival Curettage Refers to the procedure that is performed apical to the epithelial attachment severing the connective tissue attachment down to the osseous crest. *—Carranza*

Subgingival Plaque Can be defined as the community of microorganisms that develops on tooth surfaces that are apical to the gingival margin. *—Lindhe*

Subgingival Plaque Subgingival plaque is found below the gingival margin, between the tooth and gingival sulcular tissue. *—Carranza*

Subacute Less severe phase of acute condition.

Subclinical Gingivitis The initial response of the gingiva to bacterial plaque is not apparent. *—Carranza*

Subtraction Radiography It is a technique that facilitates both qualitative and quantitative visualization of even minor density changes in bone by removing the unchanged anatomic structures from the image.

Subtractive Osseous Surgery Designed to restore the form of the pre-existing alveolar bone to the level existing at the time of surgery or slightly more apical to this level.

Sulcular Epithelium The Sulcular epithelium lines the gingival sulcus, it is a thin, non-keratinized squamous epithelium without rete pegs and extends from the coronal limit of the junctional epithelium to the crest of the gingival margin. *—Carranza*

Sulcular Epithelium The soft tissue wall of the gingival sulcus is lined coronally with sulcular epithelium, extending from the gingival margin to the junctional epithelium.

Suprabony Pocket In which the bottom of the pocket is coronal to the underlying alveolar bone.

Supragingival Calculus Calculus formed coronal to the gingival margin, usually formed more recently than subgingival calculus. *—Periodontal Literature Review*

Supragingival Calculus Supragingival calculus is located coronal to the gingival margin and therefore visible in the oral cavity. It is usually white or yellowish in color, hard with clay like consistency and easily detachable from the tooth surface. *—Carranza*

Supragingival Plaque Supragingival plaque is defined as the community of the microorganisms that develop on the tooth surfaces coronal to the gingival margin. *—Lindhe*

Supragingival Plaque Supragingival plaque is formed at or above the gingival margin.

Surgery It is the art practice or work of treating diseases, injuries or deformities by manual operation or instrumental application.

Susceptibility Genes Genes involved in complex multifactorial diseases referred to as susceptibility genes. *—Carranza*

Synergy An interaction which is cooperative.

Tetralogy of Fallot — It is characterized by pulmonary stenosis, right ventricular enlargement, a defect in interventricular septum and the malposition of the aorta to the right. The oral changes include a purple red discoloration of the lip and gingiva and periodontal destruction. *—Carranza*

Thrombocytopenic Purpura — It is defined as a platelet count < 100000/mm^3.

Tissue Inhibitor of Metalloproteinases (TIMP) — An enzyme inhibitor secreted by fibroblasts to control the activity of metalloproteinases in the turn over of connective tissue ground substance.

Tooth Mobility — The degree of looseness of a tooth beyond physiological movements. *—Periodontal Literature Review*

Trauma from Occlusion — A tooth subjected to excessive occlusal forces but otherwise exhibits health with intact periodontal support; no loss of gingival connective tissue attachment and no apical migration of junctional or sulcular epithelium is described as 'trauma from occlusion'. *—Genco*

Trauma from Occlusion — It is defined as "a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position". *—Stillman (1917)*

Trauma from Occlusion — It is defined as "damage in the periodontium caused by stress on the teeth produced directly or indirectly by teeth of the opposing jaws".

Trauma from Occlusion — It is defined as, "a condition occurring on a tooth having a major loss of support, which is displaced in the remaining alveolus by a force applied to it, even the force of the tongue, cheek or chewing soft food". *—Genco*

Trauma from Occlusion — It is the term used to describe pathologic alteration or adaptive changes which develop in periodontium as a result of undue force produced by masticatory muscles. *—Lindhe*

Trauma from Occlusion — When occlusal forces exceed the adaptive capacity of the tissue, tissue injury results. The resultant injury is termed as "Trauma from Occlusion". *—Carranza*

Trauma — Refers to the precise pathologic changes occurring in the tissues.

Traumatic Occlusion — An occlusion which produces such injury is called traumatic occlusion. *—Carranza*

Treatment Plan — It is the blueprint for case management. It includes all procedures required for the establishment and maintenance of oral health. *—Carranza*

Tunnel Preparation — A surgical procedure performed on a multirooted teeth, usually a mandibular molar, resulting in a completely opened furcation to provide access for hygiene.

Ultrasonic Scaler — An instrument vibrating in the ultrasonic range which, accompanied by the stream of water, can be used to remove adherent deposits from teeth.

Universal Curette — One curette designed for all area and surfaces.

Vitalometer — A device that measures the response of a tooth to an electric stimulus to aid in determining pulp vitality.

Vertical or Angular Defects — They occur in an oblique direction, leaving a hollowed-out trough in the bone alongside the root.

Vertical/Angular Defects — Vertical/Angular defects are those that occur in an oblique direction, leaving out a hollowed-out trough in the bone alongside the root; the base of the defect is located apical to the surrounding bone.

Vincent's Angina — It is a fusospirochetal infection of the oropharynx and throat. *—Carranza*

Wasting Diseases of the Teeth — Wasting is defined as any gradual loss of tooth substance characterized by the formation of smooth polished surfaces without regard to the possible mechanism of this loss. *—Carranza*

Wegener's Granulomatous — It is a rare disease characterized by acute granulomatous necrotizing lesions of the respiratory tracts, including nasal and oral defects. *—Carranza*

Xenograft or Heterograft — The donor of the graft is from different species from the host.

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