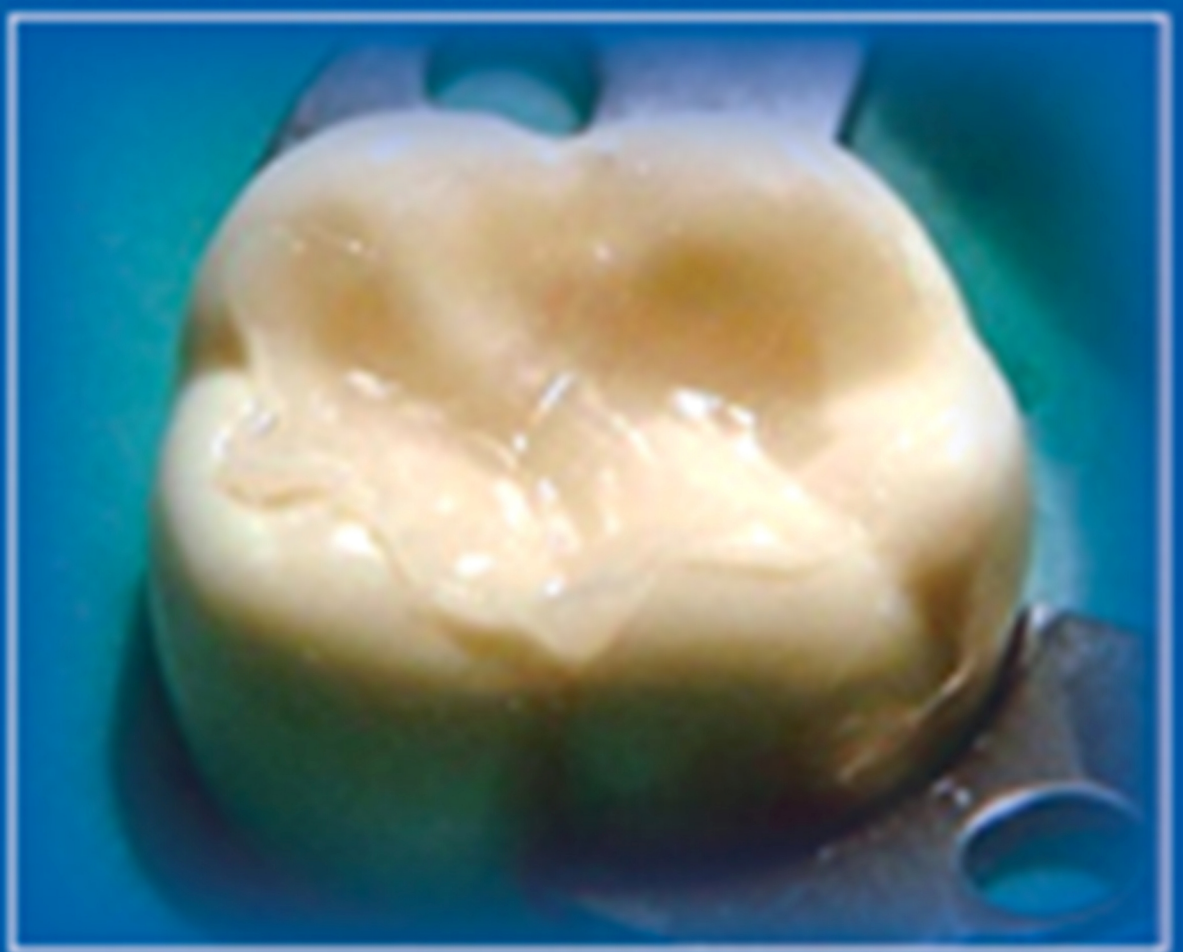
Essentials of Dentistry

Quick Review and Examination Preparation



Rushik Dhaduk

Forewords
Mahesh Verma
Bimal S Jathal
NJ Nirmal
Amish Mehta
Rahul K Thakkur

JAYPEE

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Rushik Dhaduk BDS
Tutor, Dharmsinh Desai University
Nadiad, Gujarat, India

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Affectionally dedicated to

my parents and my dearest bhai and bhabhi

"When emotions are profound, words sometimes are not sufficient to express our thanks and gratitude"

With these few words, I am trying to express my feelings towards my family members for their dedication for my happiness. No words can ever express what their constant undemanding love, sacrifice and prayers have done to help me achieve whatever I am today.

My father's dedication to his work has stirred my mind all the time to work restlessly. His few enforcing words during my childhood have always enforced me during muddling time in my life. He has always stood next to me with elucidation to all problems.

My mother's soothing voice and caring nature has always been booster in my life. She has put piles of efforts and dedications to mould me. I lay this book at her feet.

My brother Mr Bhavikkumar Dhaduk's words are hard to find, when it comes to highlight his role, in my life. I express my thanks to him for his physical presence and sentimental support at very critical times often encountered in my life. I thank him for always standing by aside. He is a friend and a guide, who stood by me as a pillar of strength, shielding and taking care of all my weaknesses. This book bears an indelible imprint of his meticulous work.

And at last but not the least comes my dearest bhabhi. My day doesn't start without pulling her hair. She is more like a friend to me and takes special care of me. Her gleaming smile relaxes mind after taxing day activity.

Foreword

डॉ. महेश वर्मा
Dr. Mahesh Verma
निर्देशक – प्रधानाचार्य
Director - Principal
सत्यमेव जयते

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M.A.M.C. Complex, New Delhi - 110 002
दूरभाष : + 91-11- 23217080, 23233925, 23221921 एक्स. 326
23233400, एक्स. 4362
फैक्स: +91-11-23217080
Tel.: +91-11-23217080, 23233925, 23221921 Ext. 326,
23233400, Ext. 4362
Fax : +91-11-23217081
Email: principal@madch.ac.in
directorprincipalmaids@hotmail.com
Website: www.madch.ac.in

I am very delighted to write the foreword for the book titled *Essentials of Dentistry—Quick Review and Examination Preparation* by Dr Rushik Dhaduk.

The book covers varied important topics pertaining to different specialties especially Oral Radiology, Conservative Dentistry, Periodontics, Oral Pathology, Pediatric Dentistry and Oral Surgery. The book is targeted at young graduates and students preparing for examinations and clinical practice. The book carries basic and clinical sciences topics with illustrations, tables and charts in order to make it userfriendly and attractive. Extensive coverage of important subject matter has been done so as to reduce the task of searching and referring multiple books by the reader. The material has been presented in a very precise and lucid manner so that it could be easily memorized and recollected during the examinations. The undergraduate students would surely find the contents very easy to assimilate and reproduce. The point-wise articulation and self-explanatory diagrams would surely help students cracking the examinations easily.

I congratulate Dr Rushik Dhaduk who is one of the youngest authors for this novel and creative endeavor. I wish him a great future and many editions of the publication.

Prof Mahesh Verma
Director–Principal
Maulana Azad Institute of Dental Sciences
New Delhi–110002, India
Vice President, Dental Council of India

Foreword

I am very delighted to write foreword for the book titled *Essentials of Dentistry—Quick Review and Examination Preparation* by Dr Rushik Dhaduk. The book is most comprehensive text of its kind. Carefully designed diagrams are easy to follow. The book is targeted to undergraduate dental students and it comprises of some of the essential topics from different dental subjects. I wholeheartedly admire Dr Rushik Dhaduk for his hard work and creativity. Today, as he paints his masterpiece in this book, he puts his years of hard work, learning and dedication into it. I wish him a great future ahead.

Dr Bimal S Jathal MDS (Periodontics)
Dean, Faculty of Dental Sciences
Dharmsinh Desai University
Nadiad, Gujarat, India

Foreword

It gives me immense pleasure to know that one of my students Dr Rushik Dhaduk has written a book titled as *Essentials of Dentistry—Quick Review and Examination Preparation* for undergraduate dental students. This book can be helpful to them for examination purpose. Various topics are selected and detailed by him after referring various journals and articles. I wish him all the best for his future.

Dr NJ Nirmal MDS (Prosthodontics)
Dean, Manubhai Patel Dental College
Baroda, Gujarat, India

Foreword

Rushik, as I have always known him, came across my attention not only as a student but also as an innovator and orator in extracurricular activities. While in our department, he was a master juggler of wire bending. His constant perseverance towards academic excellence has always impressed me. Spending even a few minutes with him one cannot avoid being motivated even as a teacher. That he has put on paper his knowledge, to share with peers, and juniors is a very commendable task. While proofing the book, I realized the simplicity in the approach to thoroughly prepare students for the examination. There is no ambiguity that this compilation will enable the students to take up the challenges of a drilling examination with confidence. Dear students, Godspeed.

Dr Amish Mehta MDS (Orthodontics)
Professor and Head
Department of Orthodontics and Dentofacial Orthopedics
Faculty of Dental Sciences, Dharmsinh University
Nadiad, Gujarat, India

Foreword

This new book by Rushik Dhaduk is a well-organized, well-written and up-to-date treatise. This book is unique as it has a specific intention of helping the hugely burdened final year dental students to finish the last lap with ease. The lucid language and illustrations certify this fact and the page on "Instructions to the reader" would be useful to students. This huge volume was created by Rushik's untiring efforts. The ultimate beneficiaries of the ideas expressed in the book, of course, are the exam-going students. I have had the pleasure of knowing Rushik, right from his undergraduate days and now as a consultant in implantology. His approach to the art and science of dentistry is very unique and I am sure that it will rub upon the readers as they go across this book.

Dr Rahul K Thakkur MDS (OMFS)
Facial Plastic (KEM), Head Neck Surgery (TMH)
Professor, Oral and Maxillofacial Surgery
Manubhai Patel Dental College and Hospital, Baroda
Director, Shubhechha Hospital, Baroda, Gujarat, India

Preface

Essentials of Dentistry—Quick Review and Examination Preparation is a unique blend of essences from numerous publications and from various standard sources. In a novel approach, special emphasis has been laid down to arrange reading material in a precise manner that can easily be memorized and recollected during the stressful exam hours. Illustrations have been enriched and arranged in the best possible manner. The layout is made more userfriendly and attractive.

My personal experience during exam preparation and studies helped me a lot to develop the book in a very friendly manner. I was preparing for this book since my college days and it carries my efforts in a very simple language with useful contents. It took years to shape this book in the best possible manner. The book is my little effort to lend a hand to the undergraduate students and it could be a real comrade to the students for exam preparation.

When I look at this publication, I see many hearts and hands to mould this into shape. I hope that the book will help the students to cope up the tight exam schedule by quick review of important topics. Basic topics and frequently asked questions are prepared in a more simplified and comprehensive manner. Various mnemonics will be much useful to memorize and reproduce the details. I am sure that the book will find its own place among undergraduate dental student community.

As no one is perfect in absolute sense, I also humbly accept my shortcomings while writing this book and therefore I sincerely welcome the valuable suggestions from my senior colleagues, students and other readers. As you walk through the pages of this book, you might come across certain errors and mistakes or in the treatment of a subject as a whole. Your suggestions are always welcomed at *feedback@drrushik.com* for improving the next edition.

Rushik Dhaduk

Acknowledgments

First of all, I would like to convey my sincere thanks to all my teachers, my college; Manubhai Patel Dental College and Oral Research Centre, Baroda, Gujarat, India and the Chairman of the college Dr Rajendrasinh Rathore; Dean of the College Dr NJ Nirmal, and the Ex Dean Dr Ramesh Suchde, for taking me to this stand. It is a well-known fact that God comes to us in various forms, as parents and as teachers. Some teachers treat us as if we were their own children and they do take personal care of us. I would always remain grateful and obliged to my teachers for their sincere efforts and keen interest since budding stage of my undergraduate studies. It is my honor to thank every teacher of this institute.

The word TEACHER refers not only to the person who teaches you in the school or college but also to the person who teaches you in one or another way at any point of life. In this context, I would like to express my sincere thanks to Dr Bimal S Jathal for giving me the opportunity to be a part of the teaching faculty of well reputed institution and to all the members of the Faculty of Dental Sciences, Dharmsinh Desai University, Nadiad, Gujarat, India for corroborating me and improving my knowledge and skill.

I strongly believe in saying, "Tell me your friends and I will tell you your future". My special thanks to all my dearest friends Dr Vipul Munia, Dr Megha Patel, Dr Brijesh Patel, Dr Khushal Dodiya, Dr Dilesh Bagadiya, Dr Jignesh Patel, Dr Dhaval Patel, Dr Bhargav Patel and Dr Deval Patel, without their wholehearted support and encouragement this would not have been possible. The erudition of my friends and colleagues has been a constant source of inspiration to me. They worked with me night and day throughout the making of this book.

Dr Brijesh Patel (Ahmedabad), a leading Dental Implant Surgeon and guide to my practice deserves special thanks for his continuous support and encouragement extended as ever during the course of this work, which has converted this “idea” of my mind into a “book”.

My regards to Mr Tarun Duneja (Director-Publishing), Mrs Samina Khan (PA to Director), Mr KK Raman (Production Manager), Mr Akhilesh Kumar Dubey, Hemant Kumar and production staff of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, for their support, devotion and keen interest in shaping the book.

I am also much obliged to Mr Abhijit Bose, Branch Manager and other staff, Jaypee Brothers Medical Publishers (P) Ltd, Ahmedabad, Gujarat, India for their support in making this book published to you.

Contents

CHAPTER

1 Rubber Dam Isolation

INTRODUCTION

The rubber dam is the most effective way for controlling the field of operation. The goals achieved by rubber dam application in isolating the field of operation are a combination of most of the goals achieved by all other isolation methods. Rubber dam ensures appropriate dryness of teeth and improves the quality of restorative material (Fig. 1.1).

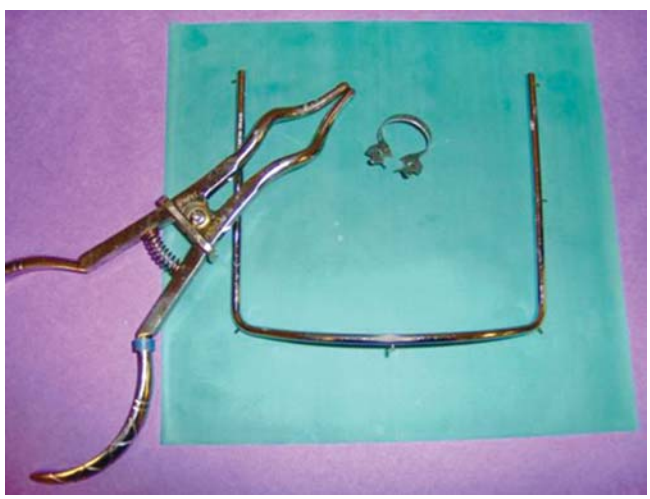


Fig. 1.1: Armamentarium

GOALS OF ISOLATION

Moisture Control

- Rubber dam excludes the sulcular fluid, saliva and gingival bleeding from the operating field.
- It prevents the swallowing of handpiece spray and restorative debris.
- It helps in maintaining the operative field dry, and prevents the mirror fogging due to breathing.
- Rubber dam produces a water tight seal that allows the safe use of sodium hypochlorite, other disinfectants and irrigants.

Retraction and Access

- Soft tissues retraction by rubber dam protects them from injuries during treatment. The inquisitive tongue is kept out of the operating field.

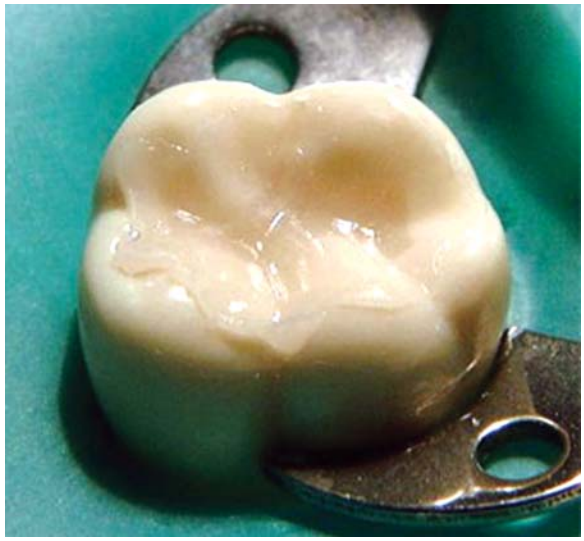


Fig. 1.2: Retraction and access

- It provides maximum exposure of operating site by maintaining open mouth with retraction of gingival tissue, tongue, lips, and cheeks (Fig. 1.2).
- It allows unimpeded vision of the tooth during treatment.

Harm Prevention

- It prevents aspiration of small instruments and restorative debris and also the soft tissue injuries.
- It prevents the aspiration of the aerosol of microbes and saliva produced by turbine handpiece. In addition, the patient does not have a mouth full of water.

Local Anesthesia

- It eliminates discomfort of patient during treatment and controls moisture.
- It reduces salivation, blood flow and gives more comfort to patient.
- Treatment is quicker and more pleasant for both the patient and the clinician.

ADVANTAGES

- Dry, clean operative field.
- Improves access and visibility.
- Potentially improved properties of dental materials.
- Protection of patient and operator.
- Operating efficiency.

DISADVANTAGES

- It is time consuming procedure (for tyro clinicians).
- Some patients may have objection for the rubber dam application.
- Certain oral conditions precluding the use of rubber dam are;
  1. Teeth that aren't sufficiently erupted
  2. Some third molars
  3. Extremely malpositioned teeth
  4. Patient suffering from asthma.

MATERIALS

Rubber dam material is usually latex rubber. For patients who have an allergy to latex, a silicone (nonlatex) rubber dam is available. They are available in variety of thicknesses, colors, sizes and materials (Figs 1.3A to C).

Sizes

5 × 5 inch	For children
6 × 6 inch	For adult

Thickness

The medium thickness is commonly used for general purpose. It nicely adapts to the cervical area of the tooth, providing fluid tight seal without use of floss or ligature ties. It doesn't tear easily and provides improved visibility. However, thin materials are recommended for isolation of mandibular anterior teeth and partially erupted teeth. As being less bulky, they exert less dislodging forces on the clamp.

Thin	0.006 inch/0.15 mm
Medium	0.008 inch/0.20 mm
Heavy	0.010 inch/0.25 mm
Extra heavy	0.012 inch/0.30 mm
Special heavy	0.014 inch/0.35 mm

Color

- Dark brown
- Green
- Black
- Blue.

They are available in dark and light shades with different flavors and aromas. Dark-colored material provides a contrasting color as a background. It has shiny and dull side. Dull side is less light reflective so it is placed facing the occlusal side.

Uses

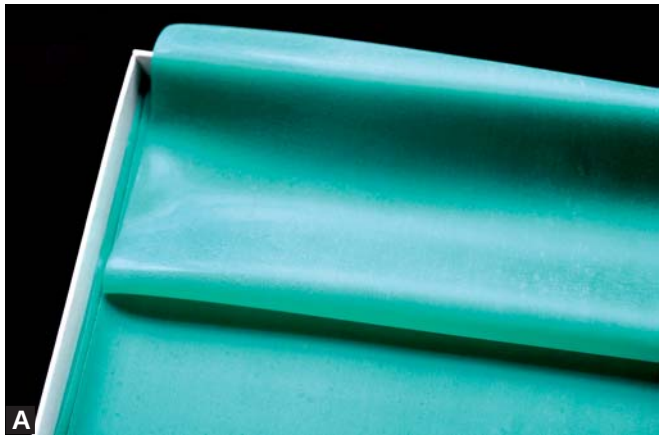
Thicker dam is more effective in retracting tissue and more resistant to tearing so it is recommended for class V lesions.

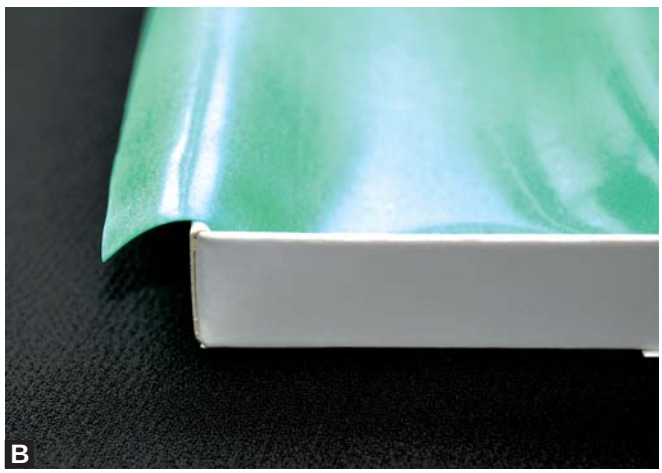
Thinner dam easily passes through the contacts so it is helpful in tight interdental contacts.

Holder

Variety of holders is available to support the dam. They keep the peripheries of the dam out of the mouth. They are of mainly two types:

1. *Strap type*: It is anchored on back of the patient's head and attached to the corners and sides of the dam. The dam is stretched and pulled toward the occipital parts of the head, e.g. Woodburry holder, Wizard holder.
2. *Hanging frame holder*: There is variety of dental dam frames to meet the requirement of clinician. Young's frame is a "U" shaped, elliptical or rectangular metal/plastic frames with multiple prongs at periphery securing the borders of the rubber dam (Fig. 1.4).





Figs 1.3A to C: Rubber dam sheets

Frames were originally constructed from metal and were radiopaque, so that they needed to be removed to avoid obscuring important details on radiographs. However, they are most popular because of ease of application and minimal contact of dam

Fig. 1.4: Hanging frame holder

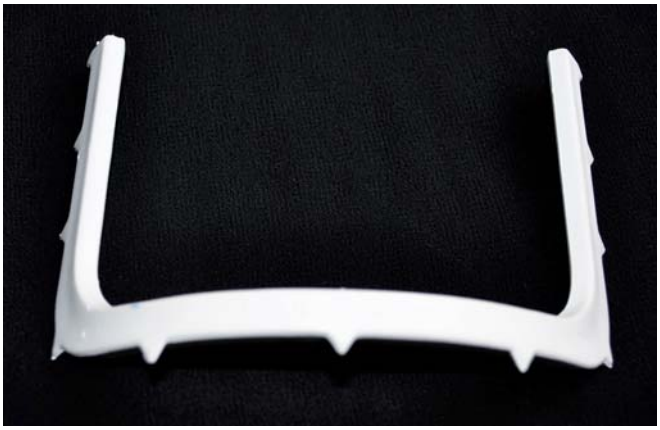


Fig. 1.5: Plastic rubber dam holder

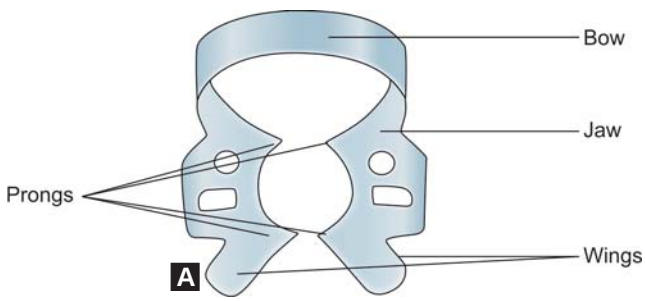
with skin. Other is Nygaard-Østby dental dam frame which is shield-shaped to fit the face. It is made up of radiolucent nylon material (Fig. 1.5).
Various articulated hinged frames and frames made up of soft metal are also available.

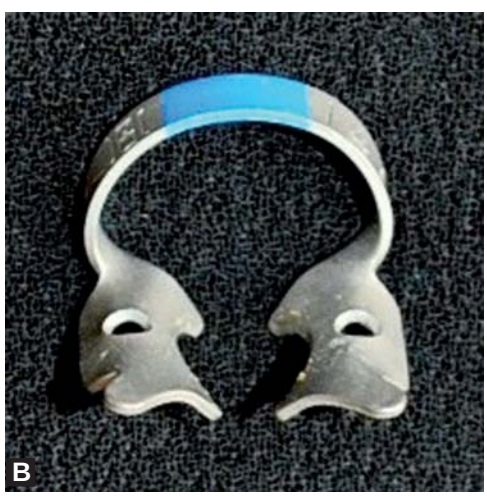
Retainer/Clamp

- The retainer secures the dam to the tooth and helps in soft tissue retraction.
- The rubber dam retainer/clamp has four prongs and two jaws connected by a bow. Most rubber dam clamps are now manufactured from stainless steel as this resists corrosion much better than plated steel (Figs 1.6A to C).
- Specific retainers are designed for certain teeth. However, teeth that are rotated, partially erupted, fractured, unusual crown form or with severe carious involvement require special clamps (Fig. 1.7).
- Properly selected retainer should contact the tooth in four areas. Thus, it prevents rocking/tilting of retainer.
- It shouldn't extend beyond the mesial and distal line angles of the tooth because:
  - It may interfere with matrix and wedge placement.
  - Gingival trauma may occur.
  - Complete seal around the anchored tooth becomes difficult.

- Some retainers have processes which are gingivally directed. They help to anchor the partially erupted tooth and provide additional soft tissue retraction.
- Retainers are available in two forms; *wingless* and *winged*.
- The advantage of a winged clamp on a molar tooth is that both clamp and dam can be placed on the tooth simultaneously. Wings allow a more rapid, efficient means of applying dam. In addition, it gives broader buccolingual deflection of the dam. When a wingless clamp is used on a molar tooth, it is normal to place the clamp first, and place the dam over it.
- Most of the anterior teeth can be isolated with a small winged clamp but if stability is a problem, a wingless clamp is almost always successful.
- Winged retainer has 2 wings—*Anterior* and *lateral.* They provide extra retention and allow the attachment of the dam to the retainer before its placement. Wing may interfere with the placement of the matrix band, band retainers and wedges. Sometimes, anterior wing can be cut away, if not required. However, generally in such condition wingless retainers are preferred (Figs 1.6A to C).
- *Bow* is tied with dental floss of approximately 12 inches in length before the retainer is placed in the mouth.

  For maximum protection floss is tied with both holes. *Floss* allows retrieval of the retainer/its broken parts, if it is accidentally swallowed or aspirated.
- Jaw is sometimes re-contoured according to the shape of the tooth by grinding with mounted stone.
- In case of treatment of anterior teeth except class-V restoration, a retainer is usually not required.





Figs 1.6A to C: Rubber dam clamp

Fig. 1.7: Rubber dam clamps

- Wedges or strips of rubber dam can be used to retain the dam instead of a clamp in the front of the mouth. This is especially useful in the anterior region, when it is necessary to use a split-dam technique.

Punch

This instrument is used to cut the holes in the rubber. It has a *rotating disc/table* with six holes of varying sizes and a tapered, sharp pointed *plunger*. Edges of the holes are angular to provide a sharp clean hole (Figs 1.8A and B).

Holes	*Teeth*
Larger	Molars
Medium	Premolars Upper canines Sometimes for upper incisors
Small	Lower incisors

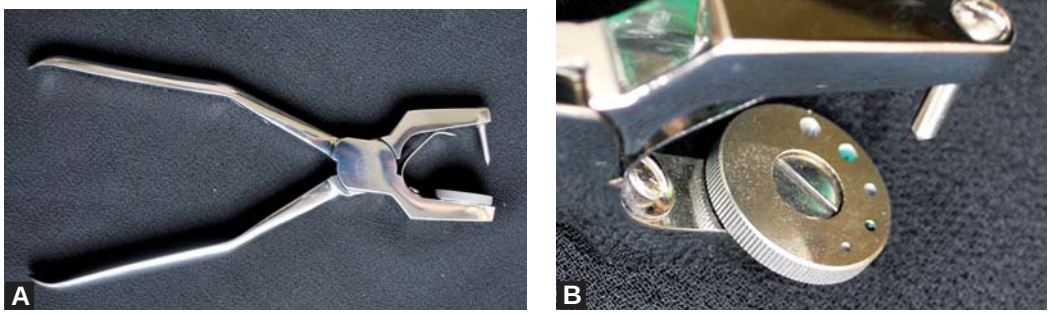
Plunger should be centered in the cutting holes so that edges of the hole don't get chipped by the plunger tip otherwise cutting quality of the punch will be ruined. This results in poor seal at the time of placement and may make the dam susceptible to tearing.

Retainer Forcep

- It is a modified forcep which retracts the jaws of a clamp away from each other allowing the clamp to overcome the occlusal diameter of the tooth. Two commonly used designs are Ash- or Ivory-style clamp forceps (Figs 1.9A to C).
- Each clamp has holes/grooves in each of its jaw to accommodate this forceps.
- It helps in placement and removal of clamp from the tooth.

Napkin

The rubber dam napkin is placed between rubber dam and patient's skin. They are absorbent papers or cloth towels (disposable) (Fig. 1.10).



Figs 1.8A and B: Ainsworth rubber dam punch

Figs 1.9A to C: Rubber dam forceps

Fig. 1.10: Napkin

Advantages

- It prevents skin contact with rubber to reduce the possibility of allergic reactions in sensitive patients.
- It absorbs any saliva seeping at the corners of the mouth.
- It acts as a cushion.
- It provides a convenient method of wiping the patient's lip on removal of the dam.
- It adds to the comfort of the patient.

Lubricant

It is a water-soluble solution which is applied in the area of punched holes to facilitate the passing of the dam septa through the proximal contact, e.g. Commercially available solutions:

- Soap slurry
- Shaving cream
- Silicone lubricant
- *Cocoa butter*/*petroleum jelly* is applied at the corner of patient's mouth to prevent irritation. Dental tape can be used to take rubber dam through a contact point and can then act as a retainer.

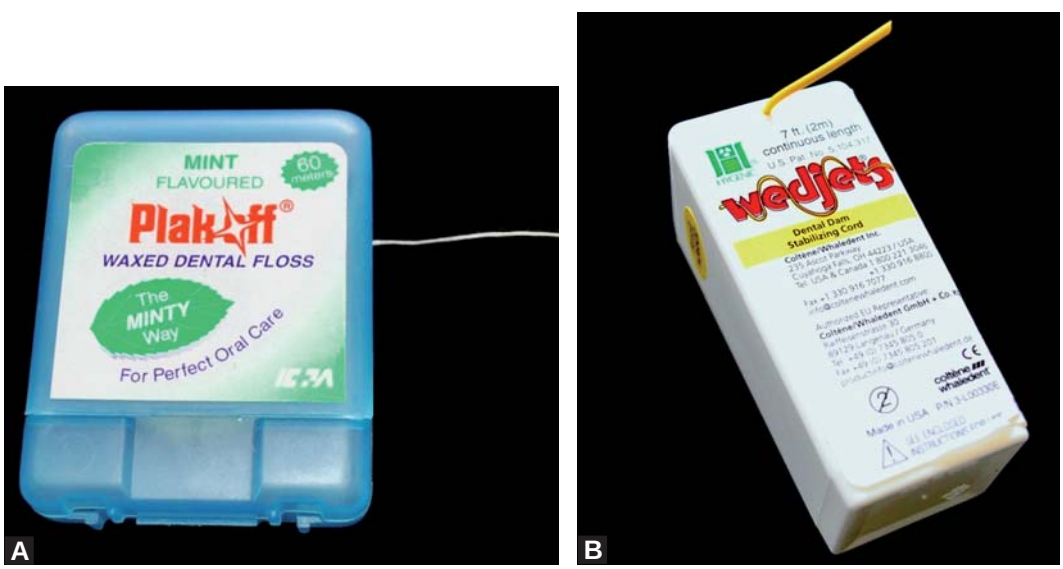
Sealants

Modeling Compound

Low fusing modeling compound is used to secure the retainer to the tooth to prevent its movement during operative procedure. Compound must not cover the holes in the retainer to have ready access to the retainer for rapid removal with forceps.

Anchors other than retainer are:

- Waxed dental tape (floss) (Figs 1.11A and B)
- Small piece of rubber dam.



Figs 1.11A and B: Waxed dental floss

Others

Various caulking agents have been marketed to help seal around rubber dam. For example, OraSeal caulking (Ultradent, South Jordan, UT, USA), rubber base adhesive, cavit and periodontal pack. For larger deficiencies or in the absence of a commercial caulking agent Cavit (ESPE, Seefeld, Oberlay, Germany) or Kalzinol (Dentsply) are useful. On occasions, some elastomeric impression material can be used.

RUBBER DAM TEMPLATE

Rubber dam template is a square sheet with four quadrants. It is of two different sizes- 5 × 5 inch, 6 × 6 inch (Figs 1.12 and 1.13).

Placement of Rubber Dam

General rule for limited isolation is to include one tooth posterior and two teeth anterior to the tooth being operated.

Steps for placement of rubber dam are:

1. Testing and lubricating the proximal contacts.
2. Punching the holes.
3. Lubricating the dam.
4. Selecting the retainer.
5. Testing the retainers.
6. Positioning the dam over the retainer.
7. Applying the napkin.
8. Positioning the napkin.
9. Attaching the frame.
10. Attaching the neck strap.
11. Passing the dam through posterior contact.
12. Applying compound.
13. Applying the anterior anchor.
14. Passing the septa through the contacts.
15. Confirming a properly applied rubber dam.
16. Checking access and visibility.
17. Inserting the wedges.

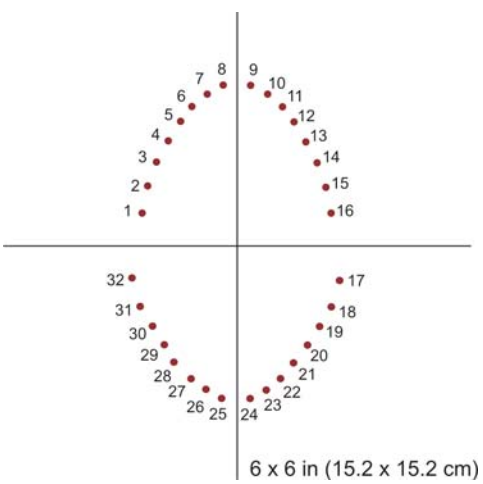


Fig. 1.12: Universal rubber dam template

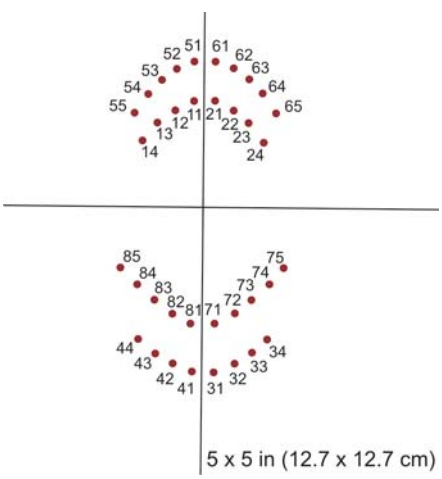


Fig. 1.13: FDI rubber dam template

Removal of Rubber Dam

- Cutting the septa.
- Removing the retainer.
- Removing the dam.
- Wiping the lips.
- Rinsing the mouth and massaging the tissue.

Contraindications

- A child with upper respiratory infections and nasal obstruction.
- Presence of some fixed orthodontic appliances.
- Recently erupted tooth.
- Patient with allergy to latex.

CHAPTER

2 Retention Form of Amalgam Preparation

TOOTH PREPARATION

"Tooth preparation is defined as the mechanical alteration of a defective, injured or diseased tooth to best receive a restorative materials that will reestablish a healthy state for tooth, including esthetic correction where indicate along with normal form and function."

STAGES AND STEPS FOR TOOTH PREPARATION

Initial Tooth Preparation Stage

1. Outline form and initial depth
2. Primary resistance form
3. Primary retention form
4. Convenience form.

Final Tooth Preparation Stage

1. Removal of any remaining infected dentin and/or old restorative material, if indicated.
2. Pulp protection, if indicated.
3. Secondary resistance form and retention form.
4. Procedure for finishing external walls.
5. Final procedures:
   - Cleaning
   - Inspection
   - Sealing.

Retention Form for Amalgam

Primary Retention Form

Design of the cavity preparation must provide retention of the restorative material especially in case of nonbonded restorations. Features of the retention form also enhance the resistance form.

Definition

"Primary retention form is that shape/form of the conventional preparation that resist displacement or removal of the restoration from tipping/lifting forces."

Principles

For class-I and class-II preparations:

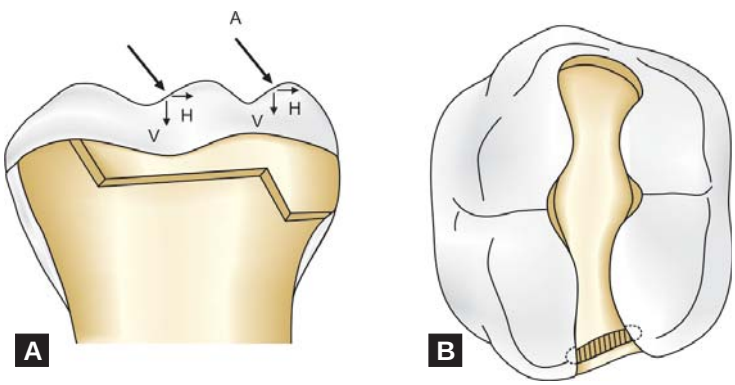
1. External wall should be converging occlusally.
   - Facial and lingual walls of occlusal portion as well as that of proximal portion should converse occlusally.
   - In addition to retention, proximal convergence conserves the marginal ridge and reduces forces of mastication on the restoration.
2. Adhesive systems provide some retention by micromechanically bonding amalgam to tooth structure. It also reduces microleakage.

Additional Features for Class-II Preparation

CL-II tooth restoration may displace four ways.

1. **Proximal displacement of entire restoration**
   When force is applied obliquely to the tooth, it has two components.
   i. Horizontal component (H)
   ii. Vertical component (V)
   Vertical component will seat restoration into the tooth but horizontal component will tend to rotate the restoration proximally at gingival cavosurface margin. This is prevented by facial and lingual retention grooves and occlusal dovetail (Figs 2.1A and B).
2. **Proximal displacement of proximal portion**
   If restoration is considered as being L-shaped, long arm will remain occlusally and short arm remains proximally.
   When the long arm is loaded by vertical force (v), it will seat the restoration more into the pulpal floor will change the location as indicated by dotted line in Figure 2.2. The short arm of L will move proximally which is prevented by facial, lingual and/or gingival grooves.
3. **Lateral rotation around hemispherical floor**
   Lateral rotation is prevented by definite line angles, point angles and ledges.
4. **Occlusal displacement**
   It is prevented by directing occlusal force in such a way that it will seat the restoration (Fig. 2.3).

These four displacements are repeated thousands of times per day and to counteract these movements all parts of the cavity preparation should be self-retaining.



Figs 2.1A and B: (A) Components of forces, (B) Retention grooves and occlusal dovetail

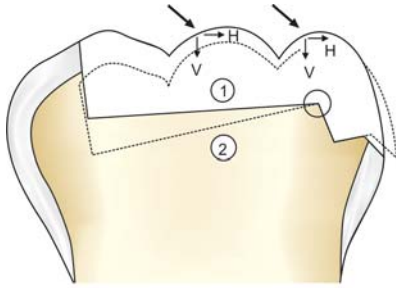


Fig. 2.2: Proximal displacement

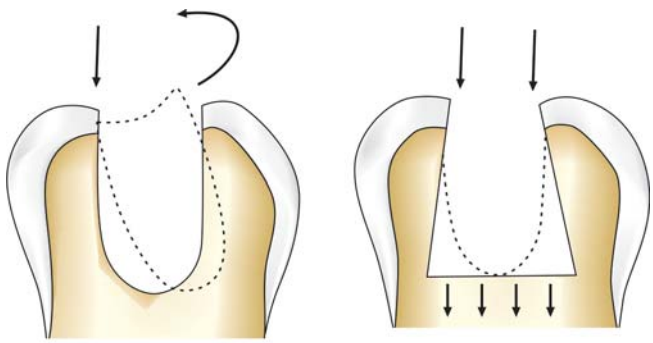


Fig. 2.3: Occlusal displacement

Secondary Retention Form

Secondary retention form is of two types:
1. Mechanical features.
2. Placement of etchant, primer or adhesive on prepared walls.

Mechanical features

Retention locks, grooves and coves

- Various type of retention grooves and retention locks provide retention for proximal portion of preparation.
- Horizontal retention grooves are useful in class III and IV preparation.
- Retention coves are appropriately placed undercuts for the incisal retention of class III amalgams.

Groove extensions

- Extension of the preparation onto facial/lingual fissures provides additional retention.
- Skirts, beveled enamel margins.
- Pins, slots, steps.
  When there is great need of increased retention form, pins and slots are incorporated into the preparation.

Placement of etchant, primer or adhesive on prepared walls

In addition to mechanical features, certain alterations in the preparation walls increase retention.

Enamel wall etching

Enamel walls are etched for bonded restorations. Etching of enamel by appropriate acid increases the total surface area and improves the bonding of material to the tooth structure.

Dentin treatment

Dentinal surfaces may require etching and priming when using bonded amalgam restorations. Sometimes, a glass ionomer is used as a base before the restoration of tooth with amalgam.

CHAPTER

3 Wedges

Wedges are third component of matrix system. The invention of dental wedges (Fig. 3.1) was carried out primarily for achieving anatomical contours of restorations in hollow spaces between neighboring teeth associated with caries. They can be triangular or trapezoidal in cross-section.

PURPOSE

Purpose of wedge is to support the filling material during entering the hollow space and also during subsequent hardening of material in such a way that the filling material can adapt exactly to the healthy part of the tooth without undesirable overhang.

REQUIREMENT

- For the matrix band to fulfill its function, the matrix band has to be approached as far possible to the shape of the tooth and has to be fixed in the position such that the filling material corresponds to the outer face of the healthy tooth and does not protrude into the embrasures. Over extension of filling material favors the formation of new carious lesions and periodontal pathologies.
- The dental wedge has to be absolutely immovable in its position between two teeth during treatment and therefore it has to be pressed with a force, usually generated with the aid of a special instrument, until the dental wedge is maintained in position between the teeth based on friction.

PARTS OF WEDGES

It is comprised of two major parts:

1. A *rigid body* configured to be inserted within an interproximal space between two adjacent teeth, which tapers from a proximal end to a distal end.
2. *Flexible members* protruding away from the rigid body.

TYPES

Wedges are made up of different materials.

Wood or plastic

Advantages of wooden wedges are:

- They are easy to trim with a scalpel and they adapt well to the tooth surface.
- Wooden wedges are more stable compared to plastic wedges.
- On contact with saliva, they swell and firmly wedge between the teeth.

Advantage of plastic wedge is:

- Transparent plastic wedge transmit the light and useful for composite fillings.

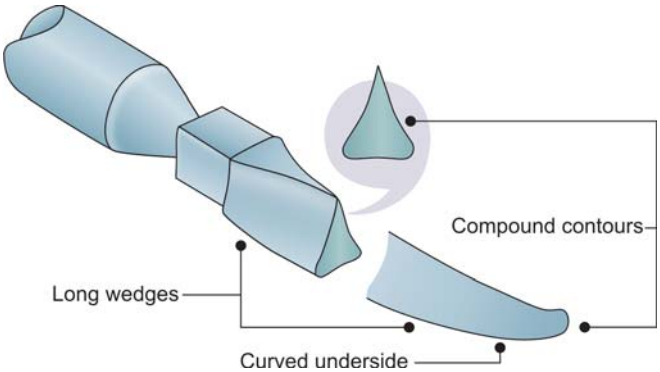


Fig. 3.1: Dental wedge

SIZES

The dental wedges are offered also in different sizes such that the selection can be made corresponding to the respective anatomy.

SHAPES

Wedges can be triangular or trapezoidal in cross section. The side view of the dental wedge is comparable to a long extended, pointed *converging triangle* where the cross-section of the dental wedge resembles a turned over V, which is downwardly open. The open side of V (dental wedge) comes in contact with the interdental papilla and other side walls with greater elasticity aids in easy insertion, firm support and good adaptation to the tooth surfaces.

Dental wedges may exhibit a *dice-(cube) shaped prolongation* at their broad end, inclined remote from the tip. The base of the wedge should be slightly larger than the space between the adjacent teeth in order to separate them apart for easy insertion of matrix.

The two lower longitudinal edges are obtusely angled off for the better adaptation to the anatomy of interdental surfaces of tooth and also to prevent the injury to the interdental papilla.

Some additional features:

- The edge is bent upward like a saber which takes care of gingival papilla at the exit point of the dental wedge. It prevents the wedge from piercing the papilla or rubber dam during placement.
- The base face and the side faces of the edge are arched concavely inwardly. This curved underside leaves room for the papilla, causing less compression.
- The impact edges of the base face and of the side faces of the dental wedge are rounded. This soft radius of the edge prevents trauma to the papilla during insertion and removal of wedge.

The dental wedge with these characteristic features is ideal for proper adaptation to the proximal surfaces of teeth and helps is restoring tooth with anatomically best adapted shape.

FUNCTION

When dental wedge is pressed between the neighboring teeth and the gums, it pushes the matrix band against the tooth to be treated and simultaneously presses the teeth somewhat apart. It creates the cervical constriction to prevent the overhang of restoration. After hardening of restorative material, when the matrix band is removed, the teeth can support each other mutually by returning into their starting position and the space previously claimed by the matrix band does not remain as a gap or as a slot opening.

METHODS OF INSERTION (FIGS 3.2 TO 3.4)

- Correct wedge selection will supply proper seal and separation while helping to prevent the wedge from loosening or backing-out during the procedure. Select the largest wedge that will fit the interproximal space. The flexible, compressible material will allow a larger wedge to adapt the tooth surface during placement. It is not necessary to bend the wedge for placements at anterior teeth.
- Continue to hold the wedge and simultaneously place your index finger or the back, flat end of metallic instrument close to the wedge head.
- Use your index finger to apply force to the wedge head, inserting it into the interproximal space to the desired position.
- In case of wedge with wand (handle), once the proper placement has been achieved, twist the handle with your fingers and separate it from the wedge head without damaging surrounding tissues (Fig. 3.5)

  Removal of the handle from the wedge is optional. Depending on the procedure, you may wish to leave the wand attached to the wedge head for easy removal of the wedge, when the procedure is complete.
- If you wish to drive the wedge further, after removing the handle, use a hemostat or sturdy cotton plier to correct the placement.

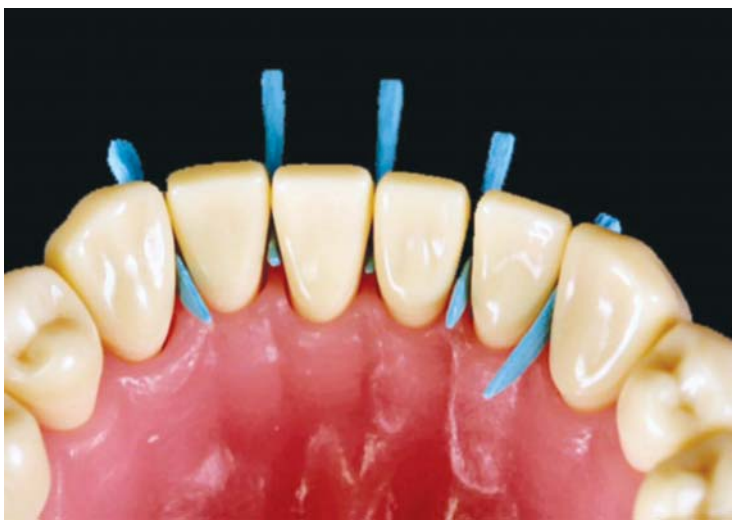


Fig. 3.2: Wedge insertion

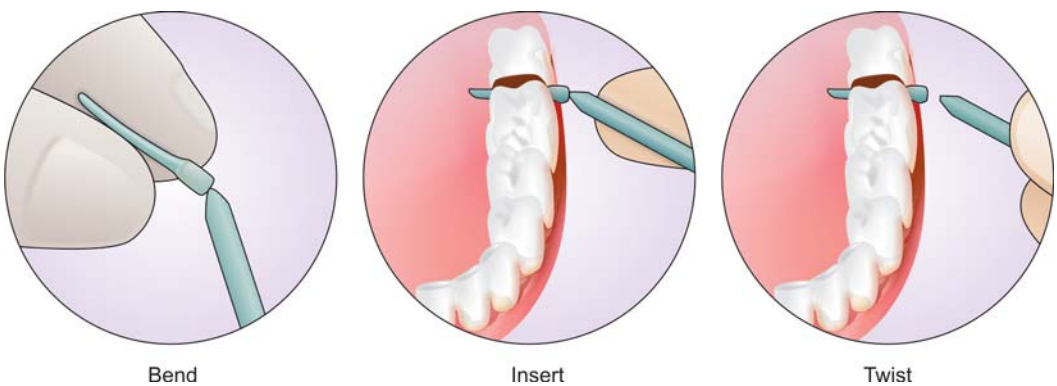


Fig. 3.3: Insertion of wedge

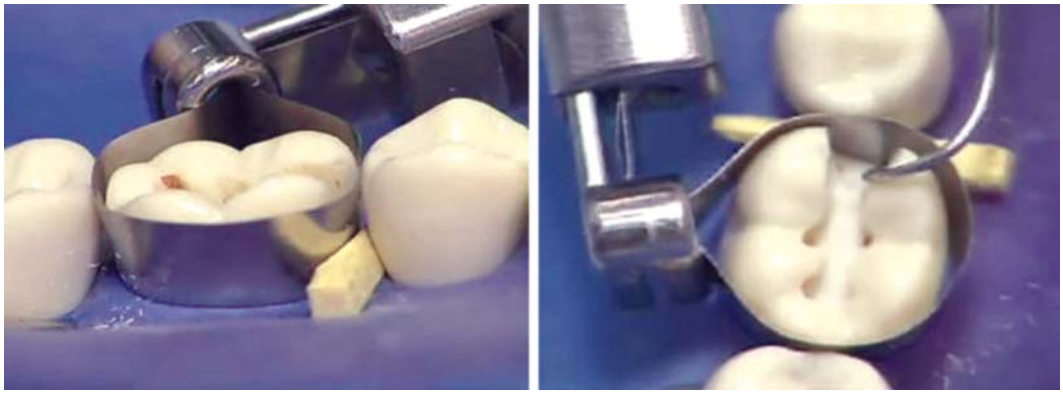


Fig. 3.4: Insertion of wedge

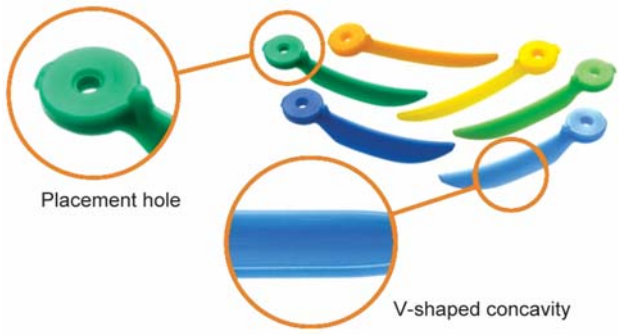


Fig. 3.5: Wedge design

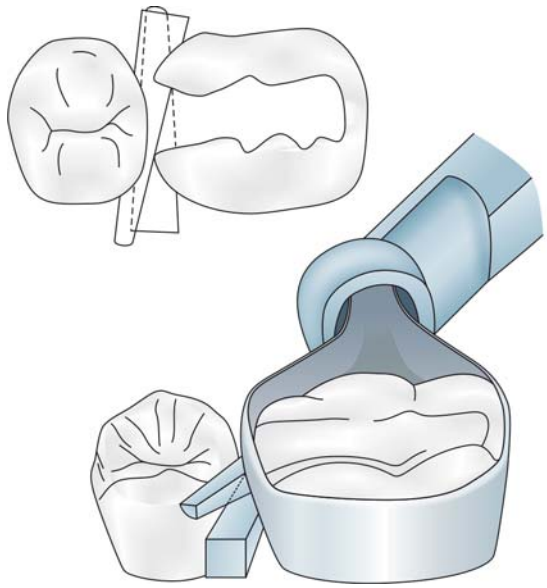


Fig. 3.6: Piggy back

(The decision whether the wedge should be inserted from buccally or lingually depends upon the two main factors;
1. Location of retainer
2. Design of cavity preparation.
Generally, wedge is inserted from the lingual or palatal side as the lingual embrasure is larger than the buccal. If it interferes with the tongue, it is preferred to insert from the buccal side).

Different Wedging Methods

Piggy back (Fig. 3.6)	A second smaller wedge is placed on the first wedge to prevent gingival overhanging. It is useful in patients with gingival recession.
Double wedging	Wedges are placed from both lingual and facial surfaces. It is useful in case of wide proximal box.
Wedge wedging	It is used in cases of maxillary 1st premolar due to presence of mesial concavity; a second wedge is inserted between the first wedge and band.

CHAPTER

4 Gates Gliddens and Peeso Reamers

Gates Glidden and peeso reamer drills are one piece rotary cutting instruments used in root canal preparations.

Structural features that make gates and peeso unique are:

- Bud-(flame) shaped cutting point mounted on a fine shaft attached to a latch type shank.
- Non-end-cutting: Fine blunt nipple which acts as a pathfinder in canal without damaging the canal walls by creating ledge and apical perforation.
- Reliable and relatively inexpensive.
- They break so high up on the shaft next to the part that fits into the slow handpiece so usually very easy to remove from the tooth.

  (They are designed such that the amount of torque required to break the shaft of the instrument is greater than the amount of torque required to separate the handle from the shaft. This means the handle will virtually always separate from the shaft before the shaft itself breaks).
- Non-end-cutting makes them by definition reamers not drills (Fig. 4.1).

SIZES OF GATES GLIDDEN DRILL

- They are available in two lengths; 38 mm for anterior teeth and 28 mm for posterior teeth (Fig. 4.4).
- They are available in six different sizes from 1 to 6 depending on tip diameter (Fig. 4.2).
- Tip diameter ranges from 0.5–1.5 mm.

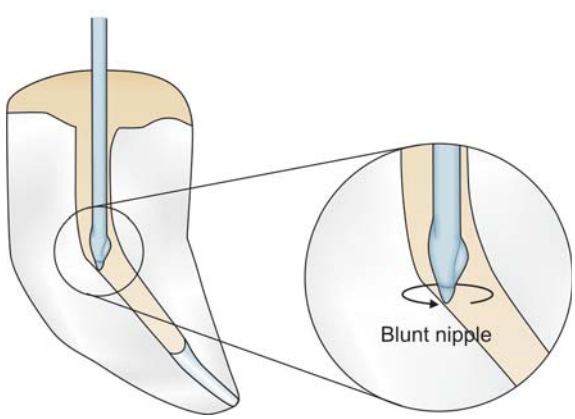


Fig. 4.1: Noncutting end

SIZES OF PEESO REAMER

- They are also available in size 1–6 (Figs 4.3 to 4.5).
  ISO sizes: 50–170
- Tip diameter ranges from 0.7–1.7 mm.

For example, the numbers on both the Gates and the Peeso are denoted by the number of circumferential grooves located on the shaft just below the cutout for the latch.

Fig. 4.2: Gates glidden drill

Fig. 4.3: Peeso reamer

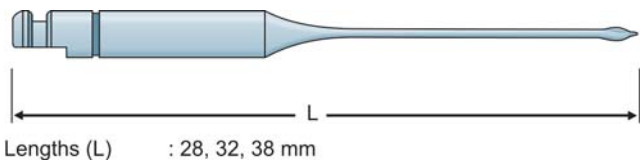


Fig. 4.4: Gates drills

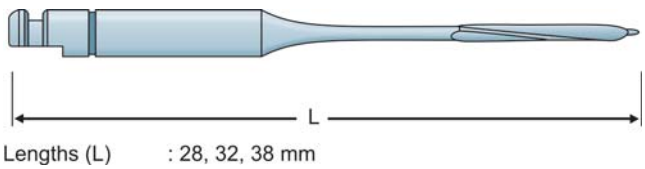


Fig. 4.5: Peeso reamers

PARTS

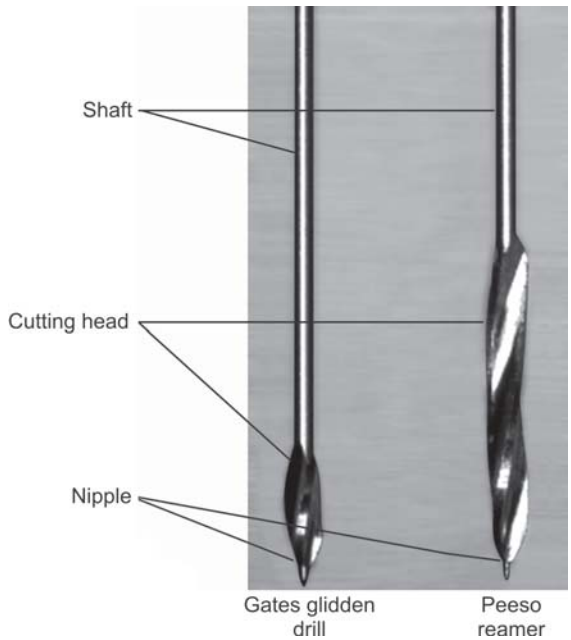


Fig. 4.6: Structural comparison of Gates Glidden drill and peeso reamer

Figure 4.6 shows the main parts of each instrument. The differences are:

1. The cutting head is much smaller on the Gates compared to that of Peeso.
2. The shaft is thinner on the Gates compared to that of the Peeso.
3. Peeso reamer have parallel cutting sides rather than elliptical shape of Gates.
4. The diameters of the heads are different for the same number instrument.

USEFULNESS IN PREPARING ROOT CANALS

- Gates-Glidden drills are side cutting instruments with safety tips. They should be used at 750-1500 rpm (Fig. 4.7).
- They can be used with both crown down as well as step back fashion.
- It is used to remove the lingual shoulder during access cavity preparation and to enlarge the canal orifice.

Not only are these instruments good for making post-holes but they are exceptionally good for preparing root canals in an easy and reliable manner. They are especially good to use in a modified crown-down technique.

In essence, the Peeso and Gates Glidden drills represent the rotary instrumentation sequence in the Simplified Endodontic Technique (SET). In the SET sequence, we first clean and shape the apex to a size number 20 (yellow) stainless steel instrument. If we would continue instrumentation without altering the canal at this stage, it would become more and more difficult to manipulate the larger number files or reamers in the canal. This would occur because the coronal end of the canal would still be narrow and the larger files would bind and work along their entire length, both at the apical and coronal ends.

The file binds over almost the entire length of the canal and it is bent or curved in that canal (Fig. 4.9). Binding and bending are two situations that lead to instrumentation breakage. To lessen the chance of breakage, the file should only “work” or bind in the apical 2-3 mm of its flutes at any one time.

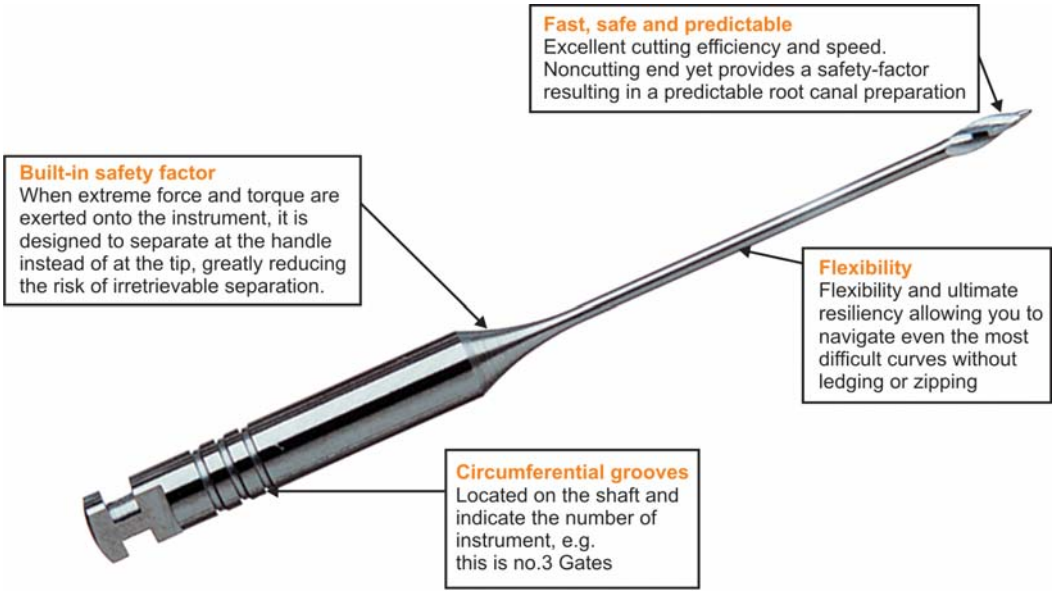


Fig. 4.7: Features of Gates Glidden drill

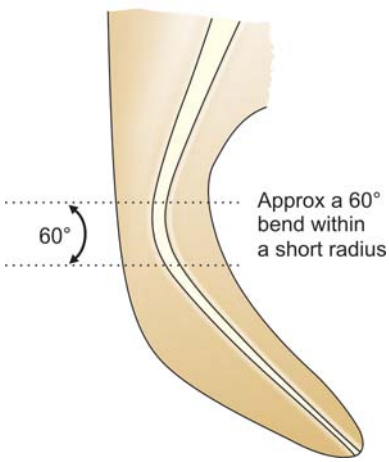


Fig. 4.8: Curvature of canal

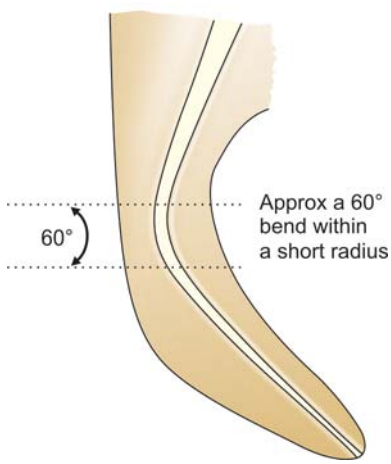


Fig. 4.9: Bending of file

This can easily be accomplished by using the Peeso or Gates to open up the coronal end of the canal to let the subsequent endodontic instruments work properly. In essence, we are doing a modified crown-down technique, using the Peeso or Gates.

Figures 4.8 to 4.10 show the typical mesial canal of a mandibular molar.

STRAIGHTENING THE CANAL

- Once the apex has been instrumented to a size — 20 stainless steel instrument, a number 2 Peeso or number 3 Gates is introduced into the canal. These instruments cut much better and more easily in wet environment. If you do not have a slow speed with water spray, use xylocaine or water from the triple syringe or even irrigating solution.

- Cut only 2-3 mm in depth, and then remove the drill from the canal. Clean the flute and now go back into the canal and cut another 2-3 mm deeper (Fig. 4.11).
- Continue in this manner till one-third to one-half of the way down the canal wall. The result will be that you have done two very good things:

1. You have lessened the curvature of the canal; usually from a 60° curve to roughly a 45° curve (Fig. 4.10).
2. You have straightened out the canal, so there is less of a radius and the endodontic instruments do not have to bend as much. Figures 4.12A and 4.13A show the typical mesial canal of a mandibular molar. Direction of file is shown by black lines. Figures 4.12B and 4.13B show the canal after preparation.

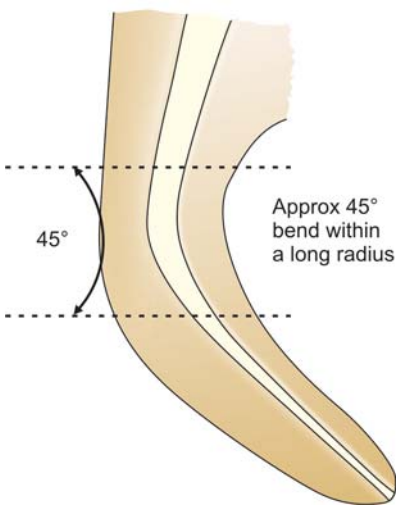


Fig. 4.10: Reduction of canal curvature

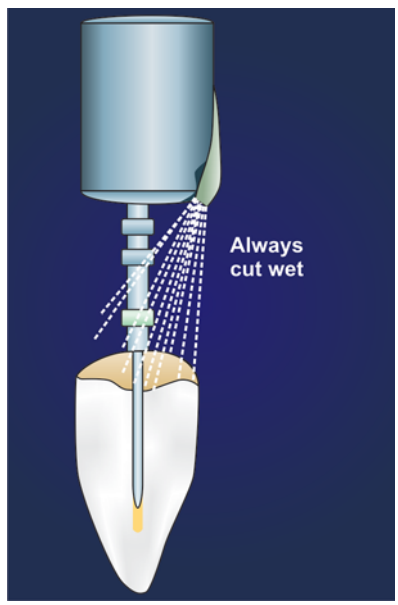
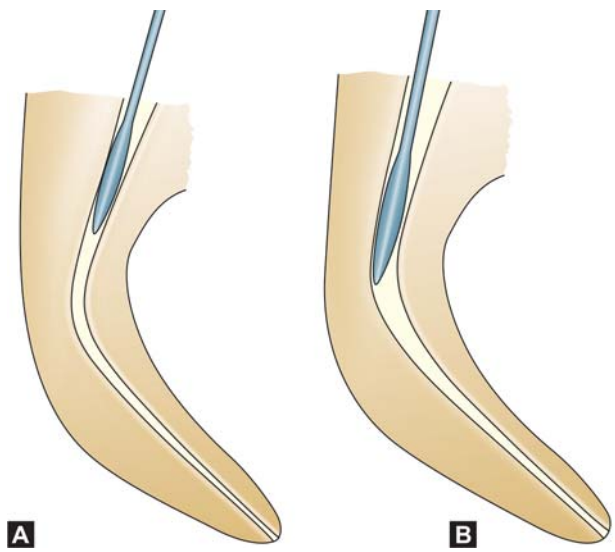


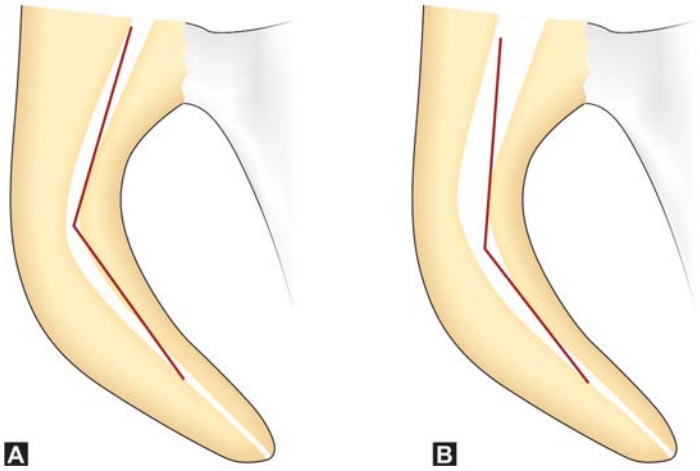
Fig. 4.11: Always cut wet



Figs 4.12A and B: The canal (A) before (B) after preparation

MAIN USES

- To widen the coronal 2/3rd of the canal quickly and safely.
- Peeso reamer is used mainly in postpreparation to remove the gutta-percha.
- To widen the canal during retrieval of the broken instrument.



Figs 4.13A and B: The canal (A) before and (B) after preparation

FLEXOGATES

- They are derivatives of Gates glidden drill.
- Used to enlarge the apical region of the canal.
- Less likely to cause apical transportation.
- Less fracture resistant but has breakage point approximately 16 mm form the tip making its retrieval easier when broken.
- Have noncutting guiding tip.

CHAPTER

5 Dental Caries Classifications

BASED ON ANATOMIC SITE

Occlusal/Pit and Fissure Caries

It is the caries that results in pit and fissures which are developed by the imperfect coalescence of the enamel during developmental stage of the tooth. This caries can be represented as cone with base towards dentinoenamel junction and apex towards enamel surface.

Smooth Surface Caries

The carious lesion on the smooth surface of tooth is called as smooth surface caries. It doesn't begin in the clean enamel but rather in an area that is habitually unclean. The base of the cone is towards enamel surface and apex is towards dentinoenamel junction.

Root/Senile Caries

Dental caries occurring on tooth that has been exposed to oral environment and it is called as root or senile caries. This is usually more rapid than other forms of caries especially in older patients.

	Surface texture	*Surface defect*	*Pigmentation*
Grade 1 (Initial)	Soft and can be penetrated with the explorer	–	Variable, Light tan to brown
Grade 2 (Shallow)	Soft, irregular, rough. Can be penetrated with explorer	< 0.5 mm in depth	Variable, Tan to dark brown
Grade 3 (Cavitation)	Soft and can be penetrated with the explorer	> 0.5 mm in depth	Variable, Light brown to dark brown
Grade 4 (Pulpal)	Deeply penetrating lesion with pulpal involvement	–	

Site and Size (Latest Classification)

Site	
Site 1	Lesions originating in the pit and fissure and smooth surface of enamel of tooth crown. It includes the buccal pits on mandibular molars and lingual pits of maxillary molars, erosion lesions on incisal edges and occlusal surfaces.
Site 2	All lesions involving contact areas, i.e. proximal surfaces of anterior and posterior teeth.
Site 3	All lesions at gingival areas in either enamel or dentin around the whole circumference of tooth.

Size	
Size 1	Localized to the point where it is just beyond demineralization.
Size 2	Larger lesions where there is sufficient sound tooth structure remaining to support the restoration.
Size 3	Remaining tooth structure is weak to support the restoration. Cavity design has to be modified.

BASED ON SEVERITY

Incipient Caries

Incipient caries is an early caries lesion which is seen on the smooth surfaces of the teeth as a "White spot". It occurs due to subsurface demineralization below the intact surface layer and can undergo remineralization. It may be confused with developmental defects of enamel. *The incipient caries disappear on wetting, while the developmental defect persists.*

Occult (Hidden) Caries

Occult caries is not clinically diagnosed but can be seen on the radiographs. Caries usually increase with time, if untreated. Increase in fluoride exposure inhibits the superficial enamel caries but the caries underneath the enamel spreads in dentin. The lesion is covered by relatively intact layer of enamel. Therefore, such hidden lesions are called as "Fluoride bombs or Fluoride syndrome".

Cavitation

Once the caries reaches to the dentinoenamel junction, it spreads rapidly to the pulp along the tubules and is seen as a definite cavitation on the clinical examination.

BASED ON PROGRESS

Arrested Caries

With change in the oral echo system, the carious lesion may become arrested. The arrested carious lesion of dentin appears as a dark brown pigmentation.

Recurrent/Secondary Caries

The caries occurring at the borders or underneath the restoration is called as secondary caries. It occurs due to the poor marginal adaptation of the restoration and subsequent marginal leakage.

BASED ON CHRONOLOGY

Acute/Rampant Caries

Acute carious lesion is infectious, light-colored lesion which spreads rapidly.

Chronic/Slow Caries

Chronic carious lesion is discolored and fairly hard. Slow destruction rate allows for extrinsic pigmentation.

BASED ON DIRECTION OF SPREAD

Forward Caries

Caries cone in the enamel is larger or same than that of dentin. It spreads in forward direction from enamel to dentin.

Backward Caries

Once enters the enamel, caries spread along the dentinoenamel junction rapidly and again extends towards the enamel in backward direction.

Residual Caries

The caries that remains in a completed cavity preparation whether by operational intention or by accident is called as residual caries.

CHAPTER

6 Differences between the Inlay and Amalgam Restorations

Cast inlay	*Amalgam restoration*
Cast inlay is an indirect procedure requiring two appointments. One for tooth preparation and the other for delivering restoration.	Amalgam restoration is direct procedure that can be finished in one appointment.
Minimum thickness of restorative material required to resist deformation is 1-2 mm.	Requires at least 1.5 mm thickness of amalgam to resist deformation.
Instruments used for cavity preparation	
No. 271 bur is mostly used.	No. 245 bur is mostly used.
Cavity preparation (Figs 6.2, 6.3, 6.4, 6.6 and 6.7)	
Axial walls should be parallel or slightly divergent occlusally (2-5° taper per wall) for achieving primary retention	Retention form is achieved through axial walls that converge occlusally.
An occlusal bevel of 30-40° is placed for proper adaptation of the inlay	Bevels are not indicated as amalgam has low tensile strength
Gingival bevel of 30° is placed in the gingival wall to 1. Remove the weak enamel to improve resistance form. 2. To provide a lap sliding fit of the casting. 3. To allow burnishing of the metal margin to improve the adaptation (Fig. 6.1).	Gingival bevel of 15-20° is placed in the gingival wall to remove the unsupported enamel rods thereby improving the resistance form. Gingival bevel is not required in deciduous teeth as there are no unsupported enamel rods in this region.
Proximal portion is box-shaped	Proximal box has an inverted trunk shape.
The isthmus width should not exceed 1/3rd of intercuspal distance.	The isthmus width should not exceed 1/4th of intercuspal distance (Figs 6.5A and B).
Flares (Primary + Secondary) prepared on the facial and lingual proximal walls carries the same function as that of bevels on occlusal and gingival walls.	Flares are not indicated
Cavosurface angle is 130-140° (Lap joint) Marginal metal is 30-40°	Cavosurface angle is 90-100° (Butt joint) Metal margin is 90°

(*Contd.*)

Contd....

Cast inlay	*Amalgam restoration*
Retention grooves are placed on the axiofacial and axiolingual line angles to provide secondary resistance form	Retention locks are prepared on the axiofacial and axiolingual angles for secondary retention
Indications	
It is appropriate for moderate to extensive cavity preparations.	It is appropriate for conservative preparations.
Contraindications	
In young patients, due to presence of large pulp chambers and in old patients, due to lengthy and multistep procedures.	Contraindicated in extensive carious lesions and indicated in young patients
Advantages	
Superior control of contours and contacts can be achieved.	There is less control on the placement of proper contours and contacts.
Disadvantages	
More cost to the patient	Less cost to the patient
Requires at least two appointments	Treatment can be completed in one appointment most of the time.

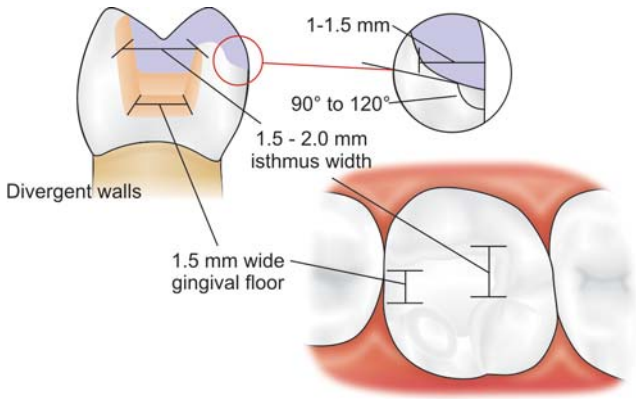


Fig. 6.1: Preparation for cast restoration

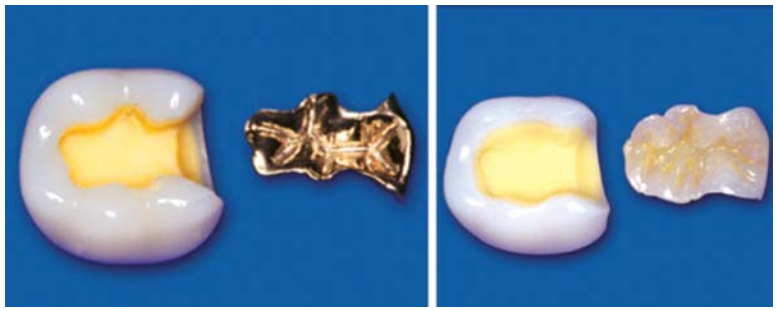


Fig. 6.2: Inlay

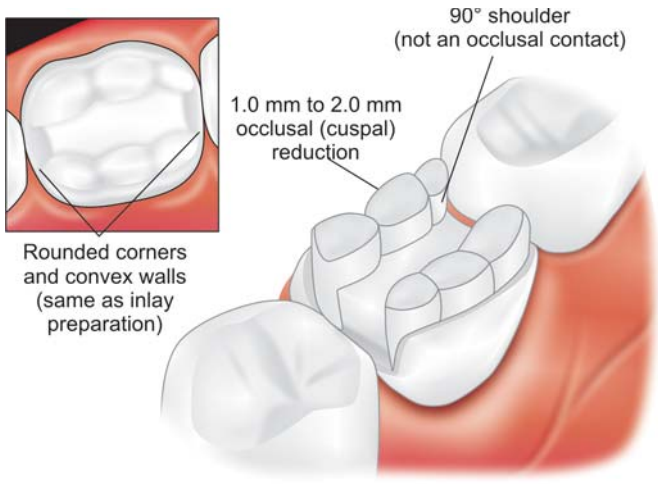


Fig. 6.3: Onlay preparation

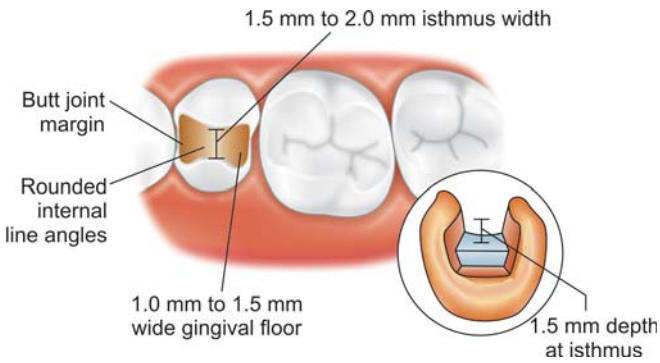
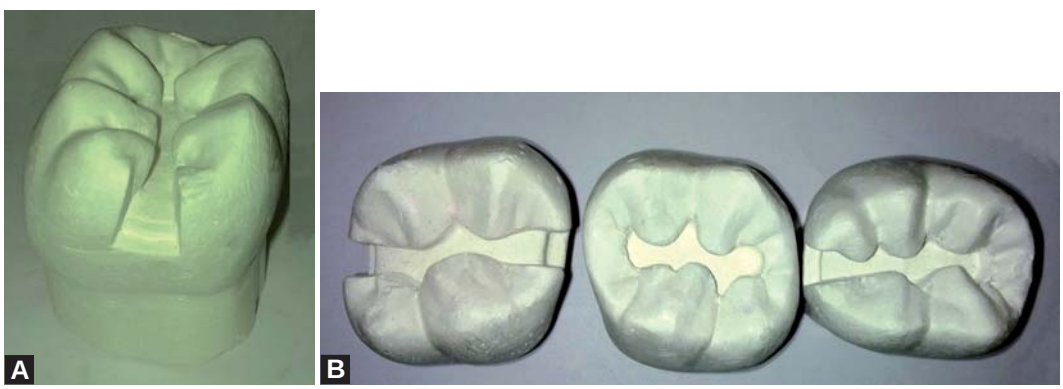


Fig. 6.4: Inlay preparation



Figs 6.5A and B: Amalgam cavity preparation

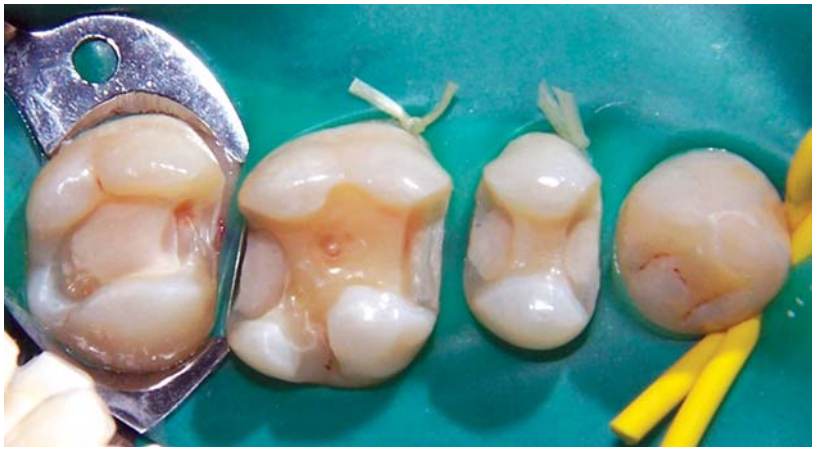
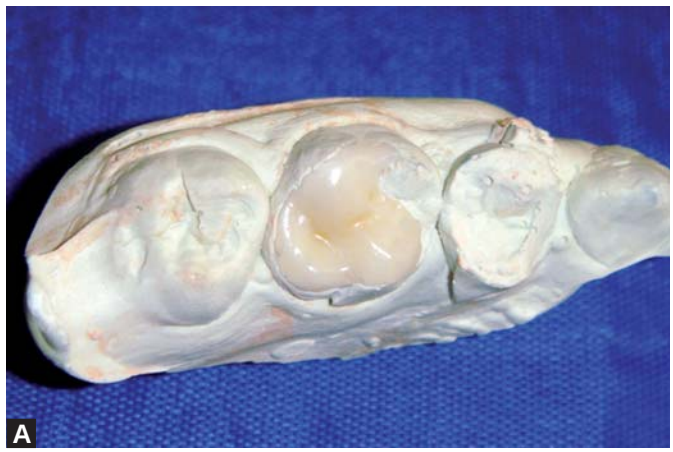
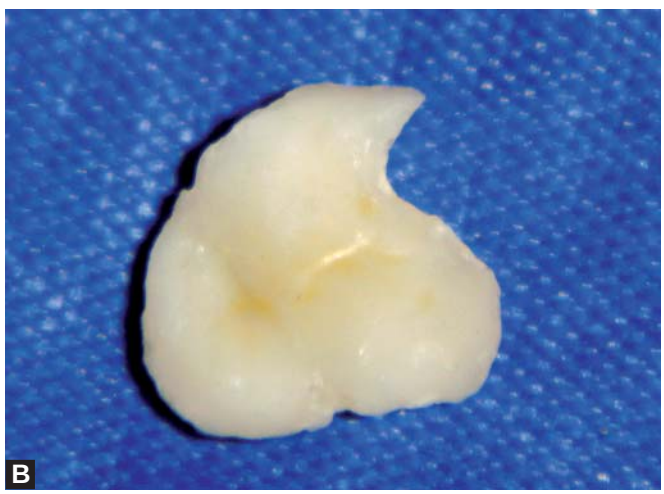


Fig. 6.6: Cavity preparation for cast restorations





Figs 6.7A and B: Cast restorations

CHAPTER

7 Electric Pulp Testing

Electric pulp testing (EPT) is based on relative conductivity of dental hard tissues. The objective of electric testing is to determine the sensibility of each tooth at the lowest sensory response threshold. It doesn't provide information regarding the health of pulp, status of inflammation or integrity of a vital pulp but it suggests whether pulp is vital or nonvital. Electric pulp testers use an electric current (AC or DC) to stimulate a response from the nerve tissue in the pulp.

PRINCIPLE

Electric pulp testing depends on the nerve supply of the pulp whereas the pulp vitality depends on blood supply of tooth. Thus, it is not the actual test for tooth vitality. Electric pulp tester produces a response by electric excitation of neural elements within the pulp by subjecting tooth to increasing degree of electric current.

ELECTROLYTE

The electrolytes applied in EPT procedures are Nicholas-colloidal graphite or Grossman toothpaste. KY jelly is also commonly used as an electrolyte.

TYPES OF PULP TESTERS

Electric pulp testers (EPTs) are widely used diagnostic tools in endodontics. They deliver a current sufficient to overcome enamel and dentin resistance to stimulate the myelinated sensory fibers (Ad fibers) at the pulpal-dentinal junction. The unmyelinated C-fibers of the pulp do not respond as they require greater current for stimulation.

The two main varieties of pulp testers are bipolar and monopolar. Monopolar pulp testers are commonly used for vitality tests (Fig. 7.1). Electric pulp tester is called as "*Vitalometer*".

SITE

- Testing with an EPT requires electrode contact onto tooth surface.
- The best site to apply the probe of tester and electrolyte during EPT is incisal 2/3rd of labial surfaces and the occlusal 2/3rd of the buccal surface of tooth (Fig. 7.2).
- Placing the electrode at the incisal edge of anterior teeth evokes a response with the least amount of electrical current.
- There are several considerations regarding optimal placement of the electrode in assessing the vitality of teeth. The response threshold is reached when an adequate number of nerve terminals are activated to attain a so-called *summation effect.*
- The response to a given stimulus will be greatest where the neural density is the highest. Therefore, an area of high neural density will have a relatively fast and strong response and requires the least electric current.

Fig. 7.1: Electric pulp tester

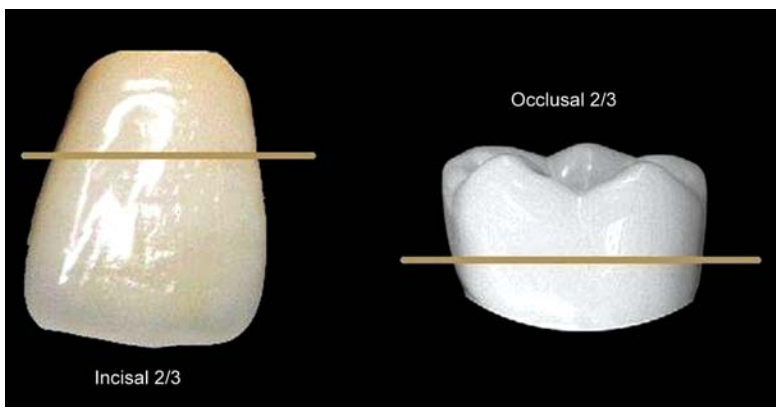


Fig. 7.2: Sites for placement of electrode

- In permanent teeth, the highest concentration of neural elements is in the pulp horns with progressively fewer in the cervical and radicular regions of the pulp.
- The response threshold increases as the electrode is moved apically from the cuspal tip to the cervical region of the tooth.

PROCEDURES

- Describe the test to the patient to reduce anxiety and to eliminate biased response.
- Isolate the tooth to be tested with cotton rolls and saliva ejector.
- Put small amount of electrolyte (toothpaste is commonly used, other is KY jelly) on the tip of the electrode of the pulp tester.
- Dry the tooth surface before the procedure to avoid short circuiting through saliva to the periodontium. Tooth can be isolated with rubber dam. A tooth may also need to be isolated with strips of rubber dam between the contact points to prevent conduction through metallic restorations into adjacent teeth.
- Ask the patient to give signal when he/she feels tingling or hot sensation.
- Place the tip of instrument on the facial surface of the tooth with lowest power and gradually increase the power (Figs 7.3A and B) . (Some newer instruments apply the

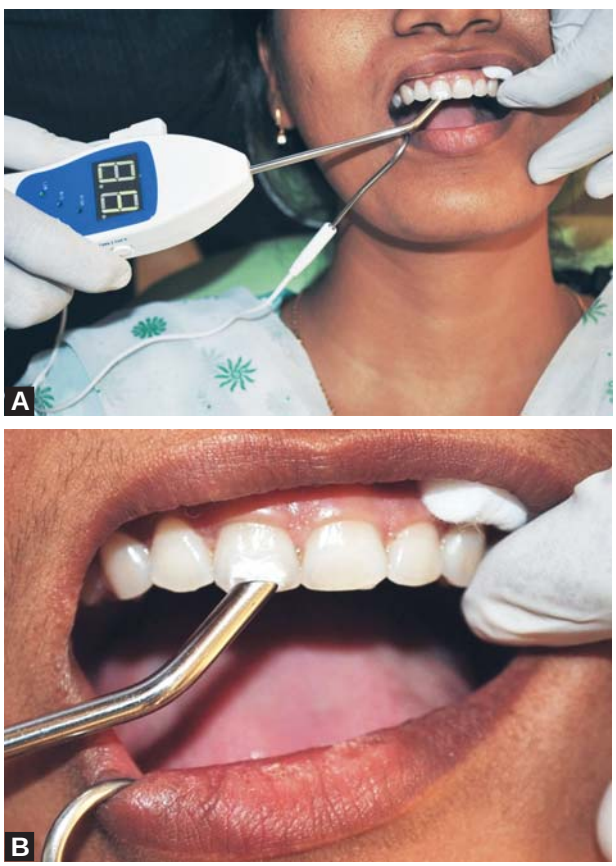
power logarithmically and automatically to the tooth being tested and preclude the chances of patient receiving full charge on the first application. Such pulp testers turn on automatically when the probe contacts the tooth and turn off automatically when the probe is removed from the tooth surface).

- If patient feels some sensation, it means tooth is vital and note the power at the same time.
- Monopolar testers such as the Analytic Technology tester require the circuit to be completed by the operator or patient. If operator is wearing the rubber gloves, patient is asked to hold the metal handle to complete the circuit.
- Compare this reading with the normal adjacent and contralateral teeth. If patient doesn't feel any sensation, it may indicate necrotic pulp.

PRECAUTIONS

- EPT should first be performed on control teeth favorably an adjacent or contralateral tooth prior to being performed on the affected tooth to eliminate the bias.
- Gloves should not be worn during EPT as they interfere with completion of circuit. While using the rubber gloves, one of the two methods must be followed for circuit completion:
  i. Use of lip clip (Fig. 7.4)
  ii. Contacting the patient's finger to the metal electrode.

  (Later method is more preferred as patient can lift the finger from electrode when a sensation is felt. This will break the circuit and terminate the stimulus).



Figs 7.3A and B: Placement of electrode

- Multirooted tooth should be tested at more than one location. It may show negative response at one location and positive response at other location. It indicates that one canal is necrotic while other contains vital pulp tissues.
- In case of tooth with full coverage restorations, a cavity is prepared through the restoration and dentin is exposed and then the tip of pulp tester is applied on to the dentin.
- Once the assembly is adjusted, current is increased gradually till the patient feels tingling sensation or pain. Each tooth should be tested two to three times and average reading is noted.

CONTRAINDICATIONS

- EPT should not be used in patients having cardiac pacemaker as it may interfere with electric activity of pacemaker.
- It should not be used on recently erupted tooth with immature apex because the relationship between the odontoblasts and the nerve fibers has not yet developed fully. It means tooth doesn't have fully developed sensitivity and may lead to false-negative reading.
- It should not be used on recently traumatized tooth.

RESPONSES

The ability of electric test to indicate pulp vitality is based on sensitivity of neural transmission, which can lead to false-positive and false-negative values, but a positive response usually indicates that there are vital sensory fibers present. Generally, thicker enamel gives delayed the response.

False-negative

1. Presence of pulp stone.
2. Extensive caries.
3. High amount of reparative dentin.
4. Patients heavily medicated with analgesics, sedatives, narcotics or alcohol.
5. Teeth with large restoration and pulp protective base.
6. Recently erupted tooth with immature root. (It has underdeveloped plexus of Rashkow as well as greater unmyelinated nerve fibers. Pulp tester is based on excitation of Aδ

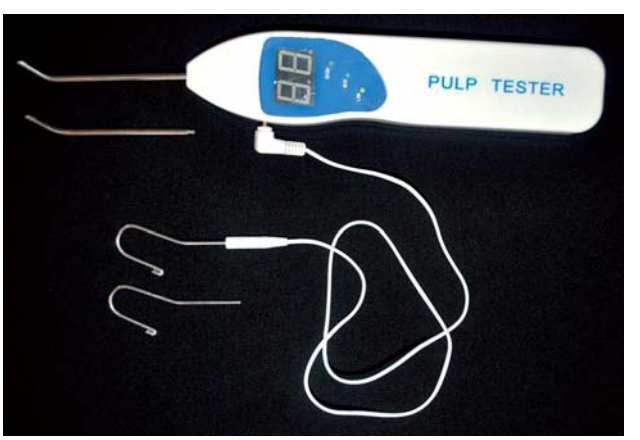


Fig. 7.4: Pulp tester with lip clips

fibers which are not fully developed till the age of adolescent and hence may not respond in recently erupted tooth).
7. Recently traumatized tooth as nerves fibers are in state of shock.
8. Inadequate contact of electrode with tooth structure due to insufficient conductor or due to improper placement of tip.
9. Low battery of tester. (With battery operated device, if battery is running low, it may not deliver full current).

False-positive

1. Presence of partially necrotic pulp in one canal of multirooted tooth because partial necrosis is indicated as total necrosis by pulp tester.
2. Moist gangrenous pulp which requires more current to elicit the response.
3. Electrode contacting any part of oral mucosa allowing current to reach the attachment apparatus directly.
4. Anxious patient may become hyperactive before perception of the actual sensation.
5. Improper isolation of tooth.

False vitality responses are common in posterior teeth due to presence of vital pulp tissue in one of the canals.

CHAPTER

8 Dental Adhesion

ADHESION

Adhesion is a process of solid and/or liquid interaction of one material (adhesive or adherent) with other (adherend) at a single interface. For example, Pit and fissure sealant.

The material or film added to produce the adhesion is known as the "adhesive or adherent" and the material to which it is applied is called the "adherend".

ADHESIVE JOINT

It is the result of interaction of a layer of intermediate material (adhesive or adherent) with two surfaces (adherends) producing two adhesive surfaces.

Adhesion is classified as:

Physical bonding	It involves Van der Waals or electrostatic interactions that are relatively weak. It occurs when the surfaces are smooth and chemically dissimilar.
Chemical bonding	It involves bonding between atoms formed across the interface from the adhesive to the adherent. The extent of this bonding is limited and overall contribution to the strength is quite low.
Mechanical bonding	It is the result of interface that involves undercuts and rough surface to produce interlocking of materials. Almost every case of dental adhesion is based primarily on mechanical bonding

MICROMECHANICAL BONDING

It is the microscopically interlocked adhesive and adherent with dimensions of less than 10 m produced by mechanical roughness.

Requirements for adhesion are:

1. To produce good bonding there must be good wetting. 0° contact of adhesive to the adherent ensures complete wetting.
2. Surfaces being bonded should be clean.

BONDING SYSTEMS

These are classified on the basis of the primary adherent. They are:

- Enamel bonding
- Dentin bonding
- Amalgam bonding.

Dentin Bonding Systems (DBS)

- DBS consists of unfilled, liquid acrylic monomer mixture placed onto an acid conditioned and primed surface.
- Difficulties in dentin bonding are:
  1. Dentin is living heterogeneous tissue and contains plenty of fluids.
  2. Presence of smear layer on the cut dentin surface.
  3. The potential biologic side effects that different chemicals can cause within the pulp.
- As most composites are hydrophobic in nature, dentin bonding agent should be hydrophobic to bond with composite and hydrophilic to interact with dentin.
- Steps of dentin bonding are:
  1. Etching/conditioning
  2. Application of primer
  3. Application of bonding agent

Conditioners

These are the agents which aids in removal or modification of smear layer.

For example, EDTA, 10 percent phosphoric acid, citric acid, maleic acid.

Primers

The primer is used to displace residual moisture, thus creating a surface upon which the hydrophobic bonding resin can adhere. The primer assists the adhesive to flow into and penetrate the etched tooth surface. The primer often contains a hydrophilic portion that interacts with the moisture present in the tooth structure, as well as a hydrophobic end that provides bonding sites for the methacrylate monomers in the bonding resin.

For example, 2-HEMA and HEMA dissolved in acetone or alcohol.

Dentin bonding agents are unfilled resins which have a role in formation and stabilization of hybrid layer (micromechanical attachment between resin and conditioned primed dentin). Even though tags are formed into tubules, bond strength is mainly due to micro mechanical bonding of intertubular dentin. Clinical longevity of dentin bonding system is not as long as enamel bonding due to high water content.

1st generation	Uses glycerol-phosphoric acid dimethacrylate
2nd generation	Uses chloro substituted phosphate esters of various monomers
3rd generation	Three step procedure of conditioning, priming, bonding
4th generation	Relies on formation of hybrid layer. Called as self-etching primers as conditioning and priming are combined in one step.
5th generation	Steps two and three are combined.
6th generation	One step procedure as all three solutions are in one bottle.
7th generation	Fluoride releasing bonding agents.

ADHESIVE CONSIDERATIONS FOR DIRECT COMPOSITE RESTORATIONS

Direct composite resins are the predictable alternative to amalgam or other metal-based restorations. The clinical success of the direct composite materials is most liked-related to the improved clinical skills and techniques and dramatic advances in adhesion technology.

For this, the dentist is required to understand the rationale for specific clinical techniques, as well as material idiosyncrasies, in order to optimize the adhesive interface between the composite restoration and tooth surface.

The actual linkage between composites and the tooth structure is usually mediated through the use of dentin bonding agent. Long-term bonding to phosphoric acid-etched enamel surfaces has proven to be highly reliable and predictable; long-term bonding to the dentin is not predictable, regardless of dentin bonding agent. Clinicians can generally bond predictably to enamel, but not as predictably to dentin because of the morphologic, histologic and compositional differences between the two substrates. Dentin is highly variable substrate. Superficial, middle and deep dentin can vary significantly in their structural and chemical composition. Enamel, on other hand, is quite consistent throughout and is also considerably more mineralized than dentin.

Factors that further complicate the bonding of dentin are dentin depth, age of the teeth and history of tooth trauma and/or pathology. This, coupled with very high water content of dentin, presents a significant challenge for consistent and reliable long-term bonding.

However, it doesn't mean that stable and strong initial bonds to dentin can't be attained. Recent studies have shown that many current adhesive systems are capable of producing bond strengths to dentin equal or surpass those to acid-etched enamel controls. Microleakage, nanoleakage, hydrolysis, dentin permeability, pulpal pressure, shrinkage stress, "water free" formation, phase separation, dentin tubule orientation, occlusion, enzymes released by bacteria, and operator error have all been implicated as potential causes of deterioration of the dentin/adhesion interface over time.

The principle mean by which adhesion takes place is by an exchange of calcium and phosphates (inorganic minerals) from enamel and dentin replacing the same with resins and nanofillers. The resin-based adhesive systems developed to date can be categorized as one of two types:

1. Total-etch system
   (Etch-and-rinse adhesive systems)
2. Self-etch adhesive systems.

Both systems result in a unique interface at the junction of tooth surface and adhesive. The difference in the interface that exists for enamel, as compared to dentin, is an important consideration in the selection of either total etch or a self-etching adhesive system (see Fig. 8.5). In the last decade, most new developments in dental adhesives have been focused on simplifying the components required for the bonding systems and improving performance of the resulting self-etching adhesives.

Careful consideration of several factors is essential in selecting an adhesive system.
Enamel bonding is superior with etch-and-rinse adhesive systems, while self-etch systems may be more suitable for restorations with large areas of dentin.

DENTAL ADHESIVE SYSTEMS (FIG. 8.1)

Typically, total-etch adhesives produce high and substantive adhesion values for both enamel and dentin. In comparison, self-etch adhesives generally demonstrate better adhesion to dentin than to enamel. These are key considerations for anterior restorations. If the surface to which the adhesive will be applied consists of significantly more enamel than dentin—and particularly if the surface consists of intact enamel, such as with an anterior diastema, an unprepared veneer, or a minimally prepared adhesive bridge, an etch-and-rinse system is preferred.

The main disadvantage of self-etch adhesive systems is that the enamel must be instrumented to provide an effective etch. Self-etch adhesives typically do not provide as high an enamel bond compared to etch-and-rinse adhesives.

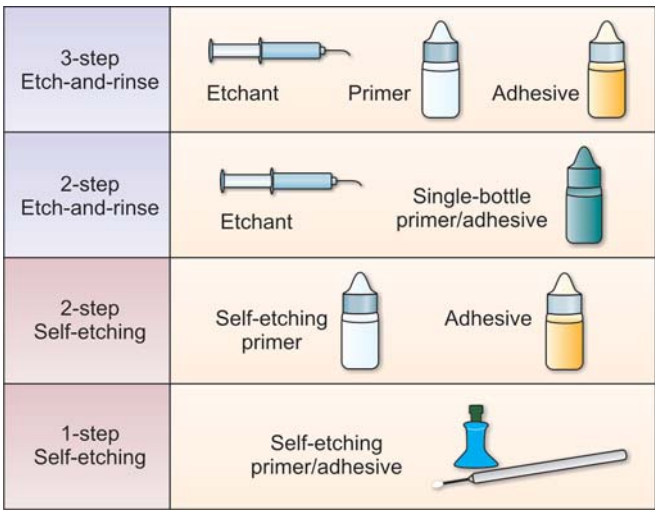


Fig. 8.1: Dental adhesive systems

In some cases, self-etch systems demonstrated a higher incidence of marginal staining on enamel margins, while the dentin margins were acceptable (e.g. posterior/ more extensive preparations). Self-etch adhesives thus may be preferred over the etch-and-rinse adhesives if the remaining enamel has been instrumented and if the surface area to be bonded consists mainly of dentin. They may also be a better choice if sustained isolation from oral fluids is likely to be a problem.

Mechanisms of Action

The basic mechanism for enamel and dentin bonding using etch-and-rinse systems consists of the following steps (Fig. 8.2):

- Demineralization of the surface by the acid (etchant)
- Penetration of the adhesive monomers into the microscopic spaces created by the etchant
- Curing of the adhesive monomers to form resin tags that microscopically provide a mechanical bond and seal to dentin and enamel.

The etchant creates a demineralized surface 3 mm to 5 mm in depth that is available for resin penetration while also removing the smear layer debris created during instrumentation. Failure to remove the smear layer reduces the dentin permeability, as that layer acts as a barrier and prevents adhesion to the underlying intact tooth structure. Demineralization results in the exposure of the collagen contained in the dentin. Macroresin tags are created peripherally around the demineralized surface of the hydroxyapatite crystals, and microtags are formed by resin penetration into demineralized crypts within the crystals.

The primer and adhesive (or combined primer-adhesive, in the case of two-step systems) then interpenetrates the exposed collagen and remaining mineral, penetrating to the residual intact mineralized dentin within this microscopic etched surface and providing retention upon curing of the adhesive. This newly created interface is referred to as the "hybrid layer" and is observed in three distinct areas (Fig. 8.3):

- Within the dentinal tubules
- In the microscopic branches lateral to the tubules
- In the intertubular dentin.

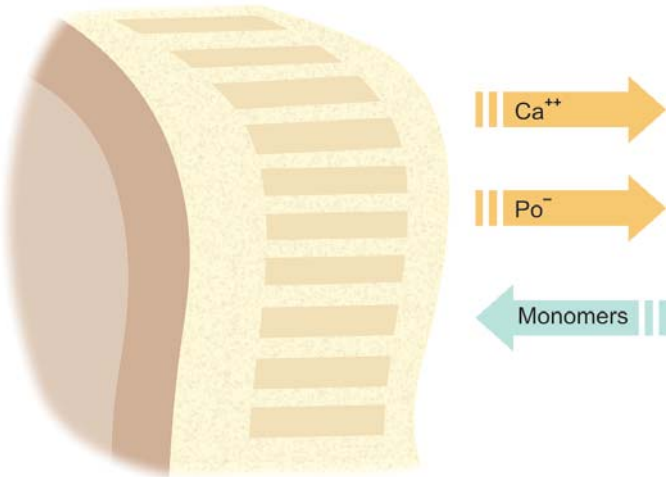


Fig. 8.2: Mechanism of action

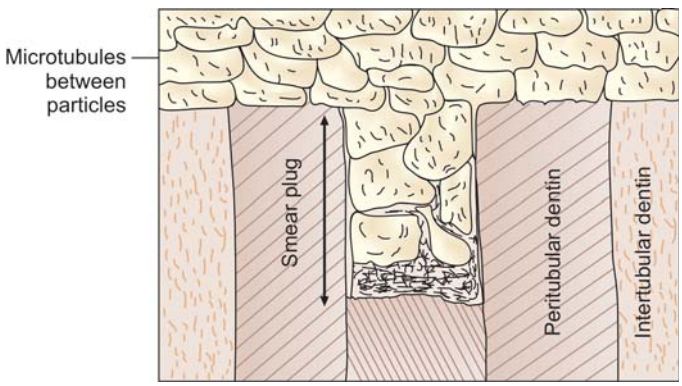


Fig. 8.3: Formation of hybrid layer

Accordingly, they treat cut dentin surface in two fundamentally different ways. The clinical picture stands in stark contrast to the dematerialized, highly permeable condition of the dentin that exists after the removal of smear layer by phosphoric acid needed for fourth-generation bonding systems. One could conclude that the significantly reduced outcome than what is generally produced by the more technique sensitive total etch adhesives.

One key performance factor that influences the success of etch-and-rinse systems includes the thorough removal of the etchant prior to bonding.

An important consideration when selecting between etch-and-rinse and self-etch systems is the condition of the enamel that will be bonded. If the enamel comprises mostly intact or uninstrumented enamel, the choice of an etch-and-rinse system is preferred. If the tooth surface to be etched consists mainly of dentin, self-etching systems may be a better choice in order to avoid the need to reinfiltrate the dentin after exposing the dentinal tubules with the etchant of the etch-and-rinse system.

Self-etch adhesives modify the layer and then penetrate through it to further demineralize the superficial layer of the dentin and combine with collagen and the remaining hydroxyapatite to form a hybrid layer of not more than 0.5–1 micron. This depth of 1 micron is much lesser than 4–5 micron depths of total etch (Fig. 8.4).

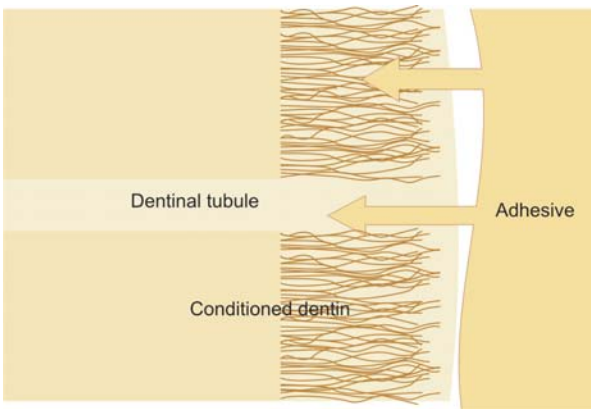


Fig. 8.4: Penetration of adhesive

Three-step etch-and-rinse systems contain an etchant, a primer and a bonding resin (adhesive). The primer is used to displace residual moisture, thus creating a surface upon which the hydrophobic bonding resin can adhere. The primer assists the adhesive to flow into and penetrate the etched tooth surface. The primer often contains hydrophilic portion that interacts with the moisture present in the tooth structure, as well as a hydrophobic end that provides bonding sites for the methacrylate monomers in the bonding resin. The subsequently applied bonding resin (adhesive) fills the residual space and seals the dentinal tubules. Polymerization of the bonding resin stabilizes the hybrid layer and also provides a polymerized surface layer upon which the final composite resin is bonded. Two-step systems combine the primer and adhesive materials into one component, thus condensing that application process into only one step. Several authors have suggested that the two-step systems may introduce greater variability into bonding results. However, these systems have exhibited excellent clinical performance over the years.

While it has been suggested that self-etch adhesive systems result in less postoperative sensitivity, a recent study shows that there is no differences in postoperative sensitivity between patients treated with self-etch adhesives and those treated with etch-and-rinse adhesives nor have any differences been discovered in marginal integrity. It was concluded that restorative technique influences postoperative sensitivity.

RATIONALE OF ADHESIVE SYSTEMS

Primers of total etch system are far less acidic, and consequently require a preliminary treatment with 32 percent phosphoric acid to change the dentin surface in a way that facilitates a strong dentin bond. This phosphoric-acid treatment completely removes the smear layer formed during cavity preparation, a layer that otherwise would block the dentin tubules, thus resulting in increased dentin permeability and, within potential for postoperative sensitivity. This smear layer removal is necessary for the fourth generation primer and the bond to penetrate the tubules and form the hybrid zone. The self-etch technique, on the other hand leaves smear layer in place. The self-etch system is based on infiltration and modification of the smear layer by an acidic monomer. Intact smear layer impedes the fluid movement within the dentinal tubules and thus reducing postoperative sensitivity.

All dentin bonding systems employ acids of one type or another type to facilitate adhesion to the tooth tissues. Acidic treatment of enamel and/or dentin creates a zone of demineralization, which is subsequently (i.e. total-etch) or concurrently (i.e. self-etch) infiltrated with various bi-functional primers and resins. While many adhesive systems are capable of providing acceptable clinical results if used in a knowledgeable fashion with attention to detail, all have their particular idiosyncrasies.

The fourth generation, or three step total-etch systems generally have good long-term clinical track records and perhaps the most versatile of all the adhesive categories because they can be employed for virtually any bonding protocol (i.e. direct, indirect, self cure, dual cure). These systems are still the *"gold standard"* by which the newer systems are judged. Indeed, none of the newer systems in the marketplace today perform any better, and often perform worse, than the original multiple component total etch systems of 15 years ago, if bond strength to dentin/enamel, microleakage and long-term durability are used as the evaluation criteria.

The fifth generation, or two step total-etch system, evolved from the desire to simplify the three step total-etch system protocol. As a group, these are among the most popular systems presently being utilized in dentistry. They have generally proven to be highly effective, simpler and faster than the multiple component predecessors. On the down side, many in this category, with some exception, are not as predictable as the three step total-etch systems when it comes to bonding to self- and dual-cure composites. It has been suggested that a small amount of residual acidic resin monomer can deactivate the tertiary amine that promotes the dark-cure polymerization of the resin cement or restorative. It is for this reason that it is suggested that, unless specifically designed for use with dual- and/or self-cure resin cement or restorative, self-etching adhesives should be avoided. In fact, even with etch-and-rinse adhesive systems, mixing different manufacturers' materials should be avoided to prevent incompatibility issues between the interface of the adhesive and the dual- and/or self-cure restorative or cement material.

In addition, the two-step total-etch systems may be more susceptible to water degradation over time than three-step total-etch systems. This is because the polymerized primer of the two-step systems tends to be hydrophilic in nature. When using a three-step system, the hydrophilic primer is covered by a more hydrophobic resin, making it less susceptible to water sorption.

Majority of studies show that fourth generation and fifth generation total-etch system in the placement of a direct composite perform best when placed on moist dentin. This has been termed as "wet" bonding, although the moist bonding may be a more accurate description of the phenomena. Dentin exposed to phosphoric acid results in dissolution of the inorganic hydroxyapatite matrix. As the matrix dissolves, the collagen fibrils, which are inherent in dentin, become exposed as they are no longer supported and surrounded by their inorganic scaffolding. The phosphoric acid treatment leaves the protein component of dentin completely unsupported, literally floating in the rinse water. If the surface is too much dried, collapsed collagen fiber network creates an impermeable organic barrier for primer penetration. This leads to gaps formation and bond strength decreases. On another side, if excessive water left on the substrate creates a different problem in that the primers do not remain dissolved in their solvents. Consequently, resin globules and water trees can form, interfering with bond strength and creating fluid movement within the tubules, promoting postoperative sensitivity.

It is this friable "collagen network" that must be infiltrated by subsequently placed primers and resins to ensure good bonding. Air-drying of acid-etched dentin causes collapse of collagen network and interferes with subsequent primer/resin infiltration. In dentin that is left moist (after acid etching) the collagen fibrils remain in a relatively "open" state and appear to be more permeable to subsequently placed primers and resins. The recommended technique when utilizing a total-etch protocol on unlined dentin is not air dry the dentin once the phosphoric acid conditioner is washed off. The excess water is simply blotted out with moist cotton pellets prior to placing the primer. This leaves a visibly moist dentin surface. It warrants noting that some total-etch systems, usually those that are acetone-based, appear to be more sensitive to this wet/dry dentin issue than others (e.g. alcohol/water-based systems). It is mandatory to follow moist bonding with acetone

containing adhesives. This highlights an inherent ambiguity many have with the concept of wet bonding, namely exactly how wet is?

Perhaps the biggest advantage of the sixth generation or two component, self-etching systems is that their efficacy appears to be less dependent on the hydration state of dentin that total etch systems. Since dentin is not pretreated with phosphoric acid as in the case with total-etch systems- no exposed collagen layer is present to collapse on air drying prior to placement of self etching primers. Clinically, this means that "wet" bonding is not a concern and the tooth surface can be briefly air-dried prior to placing self-etching primer. This is not to say that self-etch systems perform any better than total etch system, but they need to be less technique sensitive in this regard. One could also argue that a possible advantage of self-etching system is that demineralization of the dentin occurs concurrently with primer infiltration. If the tooth surface to be etched consists mainly of dentin, self-etching systems may be a better choice in order to avoid the need to reinfiltrate the dentin after exposing the dentinal tubules with the etchant of the etch-and-rinse system thus eliminating the potential for over-etching when treating dentin.

In principle, this helps ensure that the entire zone of demineralization is saturated with primer where it can then be polymerized *in situ*. On the down side, many products in this category do not etch enamel as well as their total-etch cousins and many are not compatible with self- and dual-cure composites. A common clinical technique that can be used is to first etch the enamel with traditional phosphoric acid prior to using self-etching system. This ensures good bond strength to enamel but it does require an additional step in the bonding protocol. Those utilizing this technique should take care to confine the phosphoric acid solely to enamel. Additional etching of the dentin with phosphoric acid could, in principle create an "over etch" situation where the demineralization zone is too deep for subsequently placed primers to completely penetrate.

The seventh generation or one bottle self-etching systems represents the latest simplification of adhesive systems. With these systems all the ingredients required for bonding are placed in and delivered from, a single bottle. This greatly simplifies the bonding protocol. Incorporating and placing all of the chemistry required for a viable adhesive system into a single bottle and having it remain stable over a reasonable period of time, poses a significant challenge. These inherently acidic systems tend to have a significant amount of water in their formulations and may be prone to hydrolysis and chemical breakdown. In addition, once placed and polymerized, they are generally more hydrophilic than two-step self-etching systems, which makes them more prone to water sorption. This could contribute to hydrolysis and degradation of the adhesive interface, as well as a reduction in mechanical properties of the composite restoration. The acidic nature of the polymerized primers in seventh generation adhesives generally makes them unsuitable for use with self-cure composites since their acidic nature degrades the tertiary aromatic amines required for chemical polymerization of self-cure composites. According to some author's opinion, while offering ease and simplicity, seventh generation adhesive systems should be used cautiously until more independent research clearly demonstrate their short- and long-term effectiveness.

In principle, the "ideal" adhesive system would be one that is hydrophilic when first placed in order to interact with dentin, which inherently has high water content, but then becomes completely hydrophobic once polymerized in order to discourage water sorption and hydrolysis. Unfortunately, no such chemistry currently exists. A new recently introduced total-etch system among the first to address this issue by utilizing chemistries that are less hydrophilic in nature.

NEWER ADHESIVE DEVELOPMENTS

In the last decade, most new developments in dental adhesives have been focused on simplifying the components required for the bonding systems and improving performance

of the resulting self-etching adhesives. The true one-bottle self-etch systems have now evolved to only one material. This is applied to the tooth with no mixing required. Some of the newer systems have modified the self-etch ingredients so as to result in improvements in the product's shelf lives, with diminished hydrolysis in the package. As noted above, while the quality of dentin adhesion with these self-etch systems is comparable to the etch-and-rinse adhesives, the enamel bonding of these systems still falls short of values for bond strengths achieved in the laboratory using a phosphoric acid etch system.

The self-etch systems remain popular due to the lack of concern regarding the technique used with moist dentin. The moist bonding technique has been repeatedly shown to augment bond strengths of ethanol- and acetone-based etch-and-rinse adhesives, while low bond strengths have been associated with excessively air-dried dentin in the etch-and-rinse materials. The major reason for this is presumed to be the effect of collapsing the collagen network at the bonding interface. The collapsed collagen prevents complete infiltration of the resin monomers into the demineralized dentin, leading to gaps and voids within the adhesive interface.

KEY POINTS TO ENSURE EFFECTIVE BONDING WITH TOTAL ETCH SYSTEM

1. Etch the enamel first for at least 15 seconds, followed by etching the dentin for a maximum of 15 seconds. This will ensure that the demineralized areas created during etching will be filled through capillary action, subsequently forming resin tags.

   Enamel should not be under-etched (less than 15 seconds) and dentin should not be over-etched (more than 15 seconds) — either of which could result in a reduced bond strength because deeper demineralization of dentin makes it difficult to infiltrate it completely with the adhesive. Also, the presence of some hydroxyapatite within the exposed collagen fibrils will provide some protection from hydrolysis. Ensure that the dentin is slightly moist after rinsing-off the etchant and 'drying' the dentin. This "moist bonding" helps prevent exposed collagen from collapsing and forming coagulate, which could prevent adhesive penetration into the dentin. Bonding is impaired, if the dentin is either too dry or too moist.

Different methods recommended in order of preference are:

- Take a small piece of cotton, roll it into a small pellet, dip it in sterile water, squeeze out the excess water and wipe of the cavity in such a way that no excess water remains on enamel or dentin.
- Take a small piece of dry cotton and remove the excess water from cavity. Care should be taken not desiccate the preparation.
- Use air syringe taking care to see that no oil or water is spurting out and gently dry for not more than 3 seconds. (Least preferred)

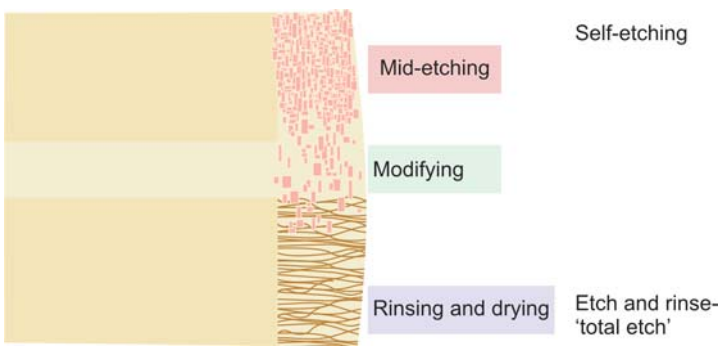


Fig. 8.5: Self-etching vs etch and rine

2. Dispense the etch-and-rinse primer or primer-adhesive combination immediately prior to placement, so as to avoid evaporation of the solvents contained therein. Use of delivery system that results in simultaneous dispensing is preferable.
3. Apply an amount of adhesive sufficient to thoroughly coat and penetrate the entire etched surface. If using a two-step system, several layers of the primer-adhesive should be applied to ensure an adequately thick layer of adhesive—solvent present in the primer-adhesive can otherwise result in a layer that is too thin. Because etching dentin removes the smear layer, a larger surface area for adhesion is exposed, thus requiring more adhesive than is necessary for the etched enamel surface.
4. Thoroughly air-dry the adhesive to remove the ethanol, acetone, or hydrophilic solvent carrier, reducing the potential for interference with the polymerization process and to spread the adhesive evenly on prepared surface. Solvent chase out the water from the dentinal tubules so that resins can penetrate deeper inside so after this work is over its presence dilute the bonding agent and weakens the bond. Air drying removes the solvent and concentrates the resin in hybrid zone and strengthens it.
5. Ensure that the adhesive is thoroughly light-cured prior to composite restoration placement to avoid displacement of uncured adhesive during placement.

   Flowable resins have low physical properties and exhibits high polymerization shrinkage. They are used in thin layers and should be cured for 10–15 seconds more because of increased presence of polymerizable resins.

KEY POINTS TO ENSURE EFFECTIVE BONDING WITH SELF-ETCH SYSTEM

1. Enamel, if present, should be beveled to improve retention. Some authors prefer to place bevel as long as the cavity for better esthetics and bonding especially in class V.
2. The surfaces to be bonded should be clean and debris-free to prevent contamination of the adhesive, since there is no rinsing step that would remove any contaminants.
3. If using a multidose, single-bottle adhesive, the amount required should be dispensed immediately prior to use in order to reduce the risk of solvent loss, which could potentially result in phase separation of monomer and water. Using a unit-dose package helps to avoid this problem and also provides an appropriate amount of adhesive for use.
4. Apply the self-etch adhesive to the enamel first, followed by application to the dentin. Some products recommend agitation of the adhesive to enhance its penetration.
5. Do not use a separate etchant on the dentin prior to using self-etch adhesives. While it may be believed that this would improve the bond to dentin, self-etch adhesives are not designed to work on etched dentin. The result would be clinically undesirable and could potentially result in catastrophic failure of the restoration. If using a separate etchant on enamel, great care must be taken to avoid its contact with dentin and to thoroughly rinse it off prior to the use of self-etch adhesives.
6. If using a one-step self-etch adhesive, apply multiple applications (as recommended by manufacturer) to help ensure adequate coating and penetration.
7. Thoroughly dry the adhesive (with a gentle stream of air) once it has been placed, to ensure removal of any volatile solvent and prevent interference with polymerization.
8. Light-cure the adhesive prior to placing the composite.

CHAPTER

9 Bleaching

The lightening of color of tooth through application of chemical agent to oxidize/reduce the organic pigmentation in the tooth is known as *bleaching*.

INTRODUCTION

It was 1st advocated by Dr JA Chapple (1877). He used oxalic acid. Harlan used hydrogen peroxide for bleaching first time in 1884, which he called hydrogen dioxide.

Although many mechanisms by which bleaching removes discoloration are not fully understood, the basic process involves oxidation, in which the bleaching agent enters the enamel/dentin of discolored tooth and releases the molecules that removes the stains. The effectiveness of bleaching procedure broadly depends upon the cause of the stain, location and depth of the stain, length of period the stain permitted to retain on the structure of tooth and depth of penetration and period of retention of bleaching agent inside the tooth to release the deep stains.

The process of bleaching for superficial stains is fairly simple. However, deep stains may require mild etching to remove the superficial organic materials and to expose the deeper areas of enamel. Once hydrogen peroxide was established as a most effective bleaching agent, attempts were put to facilitate its absorption and penetration to speed the procedure of oxidation. In 1918, Abbot discovered what the basic combination today remains: A high intensity light that produces the rapid rise in the temperature of hydrogen peroxide to accelerate the procedure of bleaching. Since then there is continuous improvement in the effectiveness and the ease of use of bleaching agents, heat and light catalyst devices, and the alternative methods.

Three most prominent bleaching processes are peroxide, chlorine and chloride. Peroxide requires least time and it is most commonly used.

CLASSIFICATION OF BLEACHING AGENTS

a. Oxidizing agents:

Sodium peroxide	
30-35% H_2O_2	Toxic free radicals, perhydroxyl anions or both E.g. Pyrozone, Superoxol, Perhydral
95% sodium perborate	Sodium metaborate, H_2O_2, Nascent O_2
10% Carbamide peroxide (Urea hydrogen peroxide)	Urea, ammonia, CO_2, 3.5% H_2O_2

b. Reducing agents:

- Sulphur dioxide
- Sulphurous acid
- Sodium thiosulphate

(c) Chlorine bleaching agent:
Chlorinated lime + Acetic acid
(d) Decalcifying agent:
- HCl
- H_2PO_4

Products Based on H_2O_2

Superoxol	30% watery solution
Pyrozone	25% H_2O_2 in ether
Perhydral	30% H_2O_2 in water
Starbrite	35% H_2O_2 gel
Dentalite plus	25% solution

Recently 1-10% H_2O_2 Solutions Marketed are:

Peroxyl—1-5% gel (Colgate)
Brite smile—1-10% solution (Brite smile)
Natural white—6% gel

HYDROGEN PEROXIDE

Hydrogen peroxide bleaching requires least time and it is most commonly used. The low molecular weight hydrogen peroxide and its capability to denature the proteins probably enhance its ability to penetrate teeth. Volume of the oxygen released determines the strength of the solution rather than the concentration. Volume indicates the amount of oxygen released by one volume of designated hydrogen peroxide.

Concentration	*Volume*
27.5%	100
35%	130
50%	200

Mechanism of Action of H_2O_2

- The exact mechanism is not known. It includes oxygen releasing, mechanical cleansing and oxidation/reduction *(redox)* reactions (Fig. 9.1).
- It differs according to type of discoloration and physical and chemical environment present at the time of reaction.
- In redox reaction, the oxidizing agent (H_2O_2) has free radicals with unpaired electrons. On giving them, it becomes reduced and reducing agent (tooth) accepts the electrons and becomes oxidized.
- Depending upon condition, H_2O_2 is capable of releasing *free radicals and*
  $H_2O_2 \longrightarrow H^\circ + {}^\circ OOH / H_2O_2 \longrightarrow HO^\circ + {}^\circ OH$
  Perhydroxyl anions
  $H_2O_2 \longrightarrow H^+ + :OOH^-$ or
  A *combination* of both free radicals and anions
  $HOO^\circ + OH^- \longrightarrow O_2^\circ + H_2O$ in a basic solution
  $HOO^\circ \longrightarrow O_2^\circ + H^+$ in an acidic solution

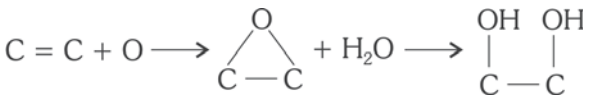
These compounds are attracted towards the electron rich alkenes bonds

↓

Form unstable epoxides

↓

Form alcohol



Double bond creates discoloration thus breaking them often eliminates discoloration.

- Moreover, H_2O_2 increases permeability of tooth and movement of ions through it. Initially, highly pigmented carbon ring compounds are opened and converted into lighter colored

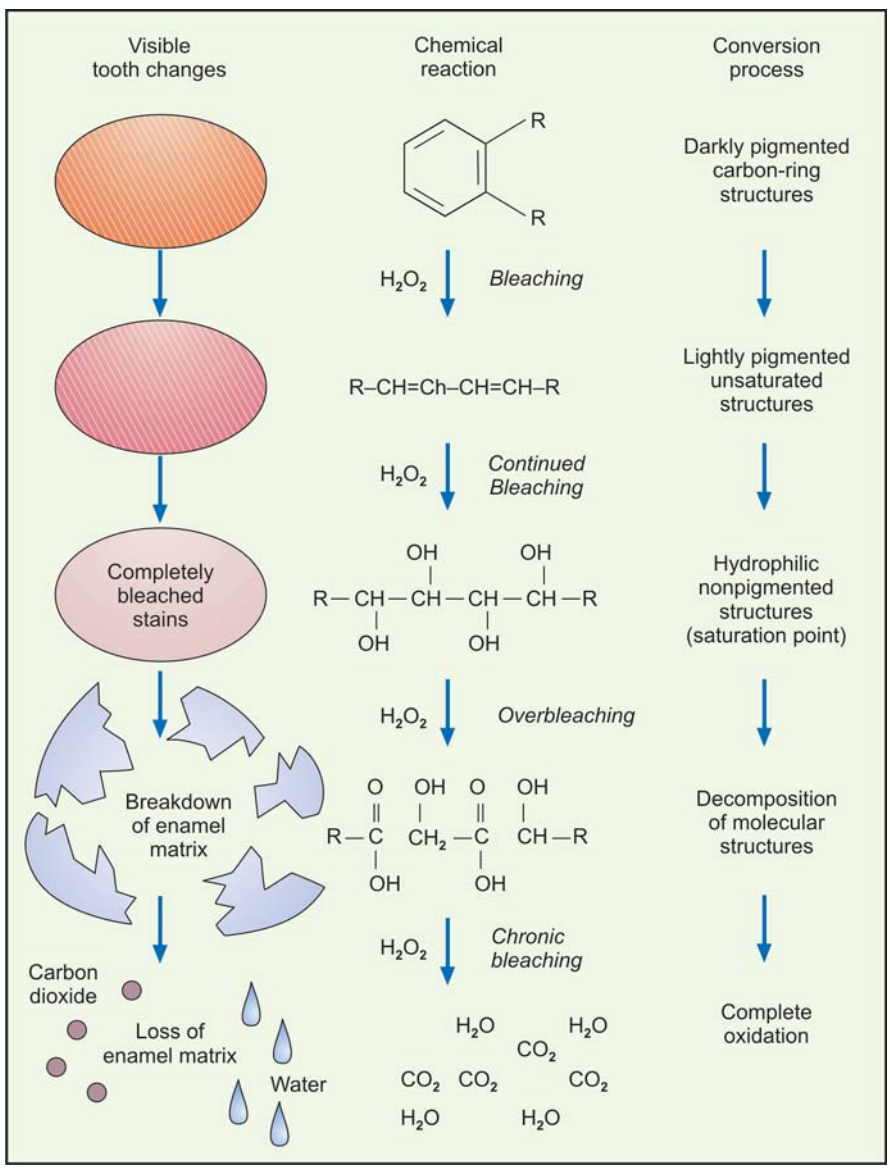


Fig. 9.1: Hydrogen peroxide bleaching

chains. These carbon double bonds, usually pigmented yellow are converted into hydroxyl group which are colorless. "As these processes continue tooth continually lightens".

- The point at which only the hydrophilic colorless structure exists is called as *"Saturation point"*. At this point optimal amount of bleaching has occurred.
- Bleaching process must be stopped at this point as further bleaching leads to tooth structure loss making tooth more porous and brittle.

Carbamide Peroxide

- Carbamide peroxide is available at concentration 3 to 15 percent but usually used at a concentration of 10 to 15 percent.
- It breaks down into hydrogen peroxide. 10 percent Carbamide peroxide produces 3.6 percent hydrogen peroxide.
- Carbamide peroxide product contains either carbopol or glycerin base.

BLEACHING TECHNIQUES

Broadly divided into:

I. *Nonvital bleaching:*
   Out office — Walking bleach using sodium perborate with either 30 percent H_2O_2 or distilled water
   In office — Thermocatalytic bleaching

II. *Vital bleaching:*
   Out office — Night guard vital bleaching (10-15% Carbamide peroxide)
   In office — Using Superoxol
   - Modified mc Inne's solution
   - 35 percent Carbamide peroxide gel

In Office Bleaching

Vital Bleaching

Step 1:

- Teeth must be cleaned of all surface stains and plaque. Isolate and protect the teeth and mouth.
- Soft tissues can be protected by applying Vaseline or Oraseal (ultradent).

Step 2:

- Teeth to be bleached should be isolated with rubber dam. If floss is to be used as ligature around the teeth, waxed floss is preferred as unwaxed floss may absorb the hydrogen peroxide and burn the tissue.
- Prepare the tooth by cleaning with pumice.

Step 3:

- Some clinicians prefer to etch the severely stained tooth with 35 percent of phosphoric acid for 5-7 seconds as it enhances the penetrability of bleaching solution and greatest amount of immediate stain reduction can be made possible.

Step 4:

- Dry the teeth thoroughly and apply the 35 percent of hydrogen peroxide bleaching solution such as superoxol with help of gauze piece.

Step 5:

- Bleaching can be done with heat, without heat or by dual application.

- In case of with heat, place the bleaching light at a distance of approximately 30cm from the teeth to be bleached. Direct the light to the labial surface of teeth.
- Gradually increase the temperature by adjusting rheostat from 46°C (115°F) as long as patient feels no sensitivity.
- Temperature recommended for vital teeth bleaching varies from 46°C to 60°C.

  Care must be taken to prevent the irreversible thermal damage to the teeth. Patient's comfort is used as a guide for adjusting the temperature. The thumb rule is adjusting temperature at least 10 degree below the temperature at which patient feels discomfort.

Step 6:
- Keep the teeth saturated with bleaching solution for 20 to 30 minutes.

Step 7:
- Flush the teeth with copious amount of warm water before removing the dam.

Step 8:
- In case of uneven discoloration, apply 35 percent hydrogen peroxide to the severely stained areas of tooth and allow it to remain on tooth for 5–10 minutes. Enamel surface can be disked with abrasive in case of severe staining.

Step 9:
- Polish the bleached teeth using the aluminum oxide abrasive disks and wheels (3M soflex system, Shofu cosmetic contouring kit). If patient feels sensitivity than 1.1 percent neutral sodium fluoride or desensitizing paste can be applied in between the appointments.

Out Office Bleaching

Nonvital Bleaching

Step 1:
- Check the vitality of tooth with thermal or electric pulp testing. Sometimes the access cavity preparation without anesthesia is used to determine the vital status of tooth.

  If the tooth is previously endodontically treated, check the adequacy of obturation and health of surrounding bone.

Step 2:
- Through prophylaxis of nonvital tooth and the adjacent healthy tooth helps in revealing the true color of the tooth. Shade is recorded with the help of shade guide for future reference.

Step 3:
- Isolate only the tooth to be bleached or the two adjacent teeth as well for comparison of shade.
- Ligate the cervical region of tooth with floss to protect the gingival region and it also permits the accurate observation of the critical cervical region of tooth during bleaching process.

Step 4:
- Endodontic access cavity is prepared according to normal guidelines. And after the obturation, the access cavity is thoroughly cleaned to remove the any remaining root canal filling material and endodontic sealer.
- Place the barrier material either GI or Cavit against the gutta percha and condense it thoroughly so that no voids remain in between them.

Step 5:

- Place the bleaching solution inside the prepared cavity. A cotton pellet saturated with Superoxol or a syringe of 35 endodontic hydrogen peroxide is placed on to the labial wall or in the pulp chamber.
- Thermocatalytically activate the bleaching solution with heated instrument. If free bleach liquid is present inside the prepared cavity, don't use the heated pluggger as excess temperature may make the solution to boil out of the access cavity (Fig. 9.2).
- Cotton saturated with the solution is pressed against the cavity wall with the help of heated pluggger.

Step 6:

- Thermocatalytic and walking bleach may be used separately or in combination.
- At the end of heating, rinse the cavity thoroughly and place the walking bleach inside the cavity.
- Solution used is a thick mix of sodium perborate and superoxol or sodium perborate and water inside the cavity and press against the wall (Fig. 9.3).
- Evaluate after 3-7 days and repeat the procedure if necessary.

Step 7:

- Seal the access cavity with temporary and leave it for 2-7 days.

Step 8:

- When the desired results are achieved, remove the material from the access cavity and restore the tooth with permanent filling material.

FACTORS AFFECTING BLEACHING

1. *Surface debridement*: Thorough scaling and polishing removes the superficial debris and enhance the effectiveness.
2. *Hydrogen peroxide concentration*: The higher the concentration, greater the effect of oxidation process. The highest concentration used is 35 percent. (Addition of gelling agent reduces the concentration to 25 percent).

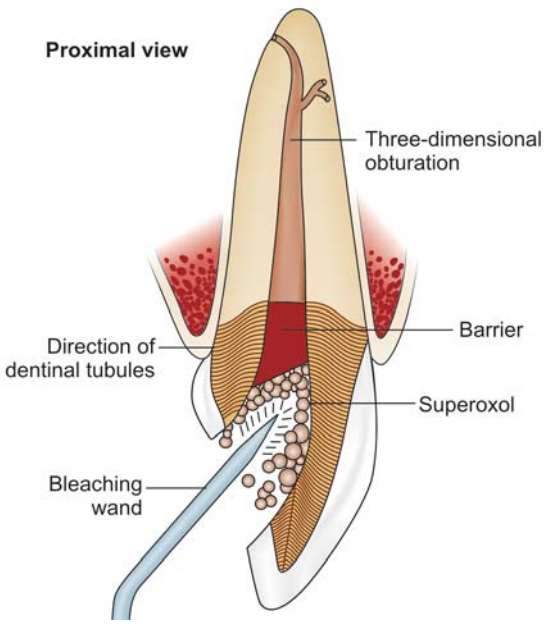


Fig. 9.2: Thermocatalytic bleach

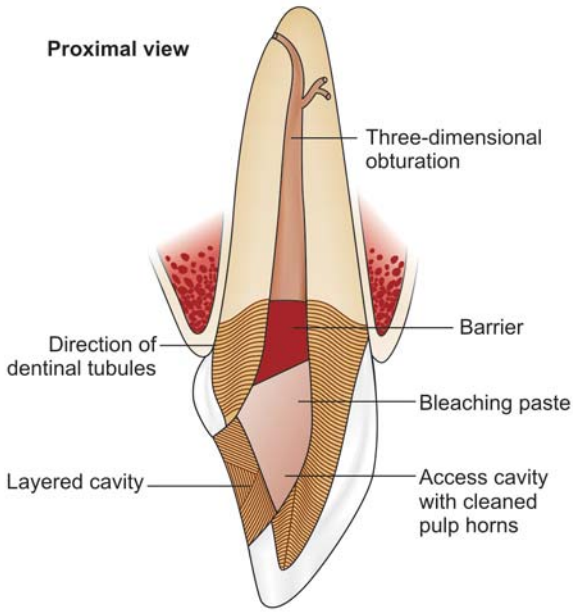


Fig. 9.3: Walking bleach

3. *Temperature*: Increase in temperature by 10°C doubles the rate of chemical reaction. Generally, if the temperature is elevated to a point at which the patient doesn't feel discomfort, then the procedure is taking place at a safer temperature range.
4. *pH*: During the storage of hydrogen peroxide, the acidic pH must be maintained to extend shelf life. The optimum pH of hydrogen peroxide to have its oxidation effect is 9.5 to 10.8. This gives 50 percent greater results in the same amount of time compared to lower pH.
5. *Time*: The effect of bleaching is directly related to time of exposure. Longer the exposure, greater the color change.
6. *Sealed environment*: Placing the hydrogen peroxide into sealed environment increases the bleaching efficacy.

DISADVANTAGES OF BLEACHING

1. The effect of bleaching on natural tooth is not permanent compared to crowns and veneers which can be restored to their original shade by cleaning and polishing. This is particularly significant when patient is unwilling to give up the stain promoting habits, such as smoking or drinking excessive tea or coffee.
2. Many times, it requires two or three sessions more compared to instant veneering.
3. It is not effective for all forms of discolorations, such as banding seen in severe tetracycline staining. The banding effect will remain, albeit somewhat lighter in color. Furthermore, bleaching cannot totally correct opacity or white spots frequently seen in fluorosis.
4. Bleaching can be inappropriate or dangerous when the surface, thickness and health of enamel has been compromised for any reason like microcracks permeating the deeper penetration of stain or thinned enamel as seen in many systemic disorders and in older age.
5. Bleaching is somewhat unpredictable to speculate the color change.
6. It can sometimes cause some low-grade reversible pulpal inflammation and may lead to hard tooth structure damage.

7. Seepage of bleaching agent into the surrounding periodontal tissues can lead to gingival irritation or severe damage.
8. Greatest potential hazard with nonvital bleaching procedure is cervical resorption of tooth.

ADVANTAGES OF BLEACHING

1. The chief advantage is its minimal invasiveness, which requires no alteration of tooth structure and absolute loss of enamel.
2. Relatively low cost compared to other esthetic treatments.

CHAPTER

10 Endodontic Hand Instruments and Instrumentation

CLASSIFICATIONS OF ENDODONTIC INSTRUMENTS

According to Function (by Grossman)

Instruments are divided into four groups.

1. Exploring Instruments
   To locate the canal orifice or to assist in obtaining patency of the root canal.
   - Smooth barbed broach
   - DG-16 explorer.
2. Debriding Instruments
   To extirpate the pulp and to remove debris and other foreign materials.
   - Barbed broach.
3. Shaping Instruments
   To shape root canal apically and laterally.
   - Reamers
   - Files.
4. Obturating Instruments
   To pack gutta-percha into root canal.
   - Plugger
   - Spreaders
   - Lentulo spirals.

Harty's Classification

1. Instrument for access cavity preparation
   - Basic instrument pack
   - Burs
   - Rubber dam.
2. Instruments for root canal preparation
   - Hand instruments
   - Power assisted root canal instruments
   - Electronic canal measuring device
   - Measuring instruments, gauges and stands
   - Instruments for retrieving broken instruments and posts.
3. Instruments for filling root canals
   - Lateral condensation
   - Vertical condensation
   - Hybrid technique
   - Thermoplasticized gutta-percha.

4. Equipments for storing instruments
5. Sterilization of endodontic instruments
6. Equipments for improving visibility
7. References.

ISO and FDI Classification

ISO and FDI grouped root canal instruments according to their method of use.

Group I	*Group II*	*Group III*	*Group IV*
Hand operated instrument	Same as group I but the handles are replaced by *latch type* of adaptor for the insertion in the slow speed handpiece. These instruments consist of 2 parts: – An operative cutting edge – Latch type of attachment	Similar to the Group II type having a latch type attachment but they are fabricated from a *single piece* of metal so latch, shank and cutting edge are made of one piece.	Endodontic points
E.g. Barbed broaches, K-file, H-file, Reamer, pluggers.	Ni Ti rotary instruments like profile, pro taper	E.g. Gates Glidden drills.	E.g. GP points, silver points, paper points

Other Classification

1. Instruments used for initial examination
2. Instruments used for diagnosis
3. Instruments used for isolation
4. Instruments used for access cavity preparation
5. Instruments used for working length determination
6. Instruments used for canal preparation
7. Instruments used for irrigation
8. Instruments used for obturation
9. Instruments used for postspace preparation
10. Instruments used for retrieval of broken instrument/post/gutta percha
11. Instruments used for endodontic surgery.

STANDARDIZATION

In the past, instruments were not standardized as regards to the diameter, taper and length. Later in 1957, *INGLE and Le VINE* gave the recommendations for having uniformity in instrument diameter and taper, which were further developed by International Standards Organization (ISO) (Fig. 10.1).

1. Instruments shall be numbered from 10-100. The numbers are advanced by 5 units up to 60 and then by 10 up to 100.
2. Each number is the representative of the diameter of the instrument at the tip in the 100th of millimeter.
   E.g. No.10 is 10/100 = 0.1 mm at tip.
3. The working blade (flute) should begin at the tip designated site D_1 shall extend exactly 16 mm up to the shaft terminating at designated site D_2 (D_{16}).

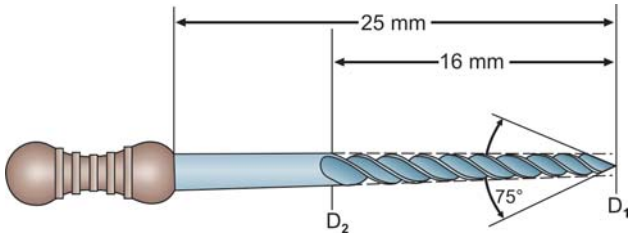


Fig. 10.1: Instrument standardization

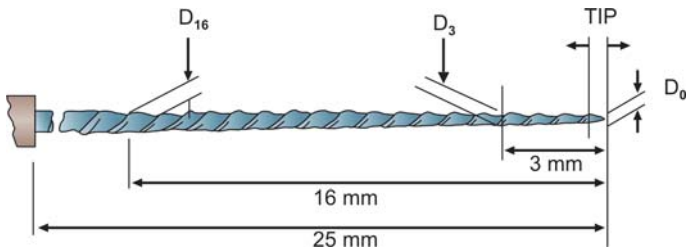


Fig. 10.2: Modifications

There is constant increase in taper of 0.2 mm/mm, so the diameter at D_2 is 0.32 mm greater than that at D_1.

E.g. For No. 20 reamer

D_1 = Diameter at the tip = 0.2 mm

D_2 = Diameter 16 mm away from tip (D_1) = 0.32 + 0.20
= 0.52 mm

(Later on, they are made available in sizes 6,8,10 up to 150)

Other Specifications Added Later

After several years, the ISO committee made another change in standardization nomenclature (Fig. 10.2).

- The name of the original point D_1 (where the cutting flutes begin) is altered and given a new identification, D_0 (Fig. 10.3).
- Point D_2 was given a new identity, D_{16} (where cutting flutes end).
- An additional diameter measurement point D_3 is 3 mm from the tip of the cutting end of instrument D_0 (earlier D_1).
- The tip angle of the instrument should be 75° $\pm$ 15°.
- Size of the instrument should increase by 0.05 mm from No.10 to No. 60 and by 0.1 mm from No. 60 to No. 150.
- Instrument handles have been color coded for easier recognition.
- Color coding facilitates quick picking of instruments during treatments and thereby saves time (Fig. 10.4).
- They are available in different lengths of 21, 25, 28 and 30 mm. Ordinarily 25 mm instruments are used.
- Greater taper (0.04, 0.06, 0.08) are available.

21 mm	For molars especially when patient cannot open wide
28-30 mm	For cuspids and long anteriors
25 mm	For anteriors
40 mm	For endodontic implant

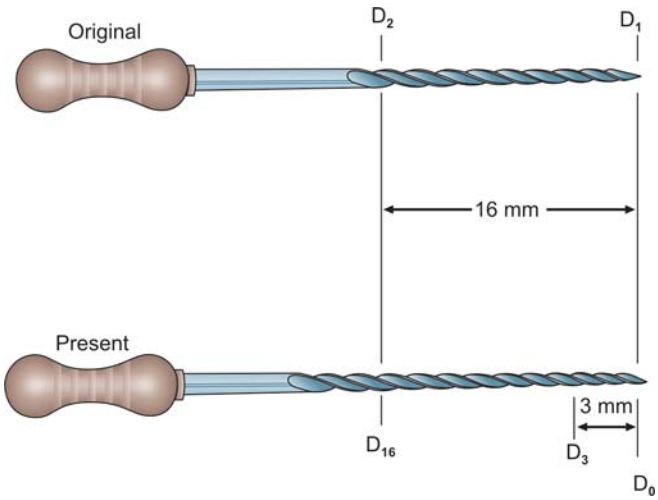


Fig. 10.3: Comparison of original and present specifications

MATERIALS USED FOR MANUFACTURING INSTRUMENTS

Various materials are used for manufacturing the endodontic instruments depending on the particular characteristic of the material.

Carbon steel is more brittle and less resistant to corrosion than stainless steel. Stainless steel is more resistant to corrosion but looses sharpness faster than carbon steel. Nickel-titanium instruments have superflexibility.

The traditional endodontic instruments made up of stainless steel sustain deformation and retain permanent shape change. New generation endodontic instruments, made up of nickel-titanium, have the property called, superelasticity. It allows instruments to return to its original shape following significant deformation. Nickel-titanium files are more flexible and better conform to canal curvature, resist fracture and wear less than stainless steel files. They are biocompatible and extremely resistant to corrosion.

	Carbon steel	*Stainless steel*
Composition	Fe – 98% C – 1-1.2% Mn – 0.2% Si – 0.2%	Fe – 81% Cr – 18% C – 0.6-1%
Corrosion resistance	Very low	High
Cutting efficiency	High	Low, so wears rapidly
Physical properties	Stiffer, stronger, harder but brittle. Less heat generation on cutting	Less hard than carbon steel but less brittle. More heat generation
Efficiency	Superior, at high speed and better at cutting the enamel	Superior, at low speed and better at cutting dentin

MANUFACTURING OF ENDODONTIC INSTRUMENTS

Instruments are prepared from round wire by modifying it to form tapered instruments with cutting edges, of different shapes and sizes. There are two main techniques for manufacturing these instruments.

Color	Number
Pink	6
Grey	8
Purple	10
White	15-45-90-150
Yellow	20-50-100
Red	25-55-100
Blue	30-60-120
Green	35-70-130
Black	40-80-140

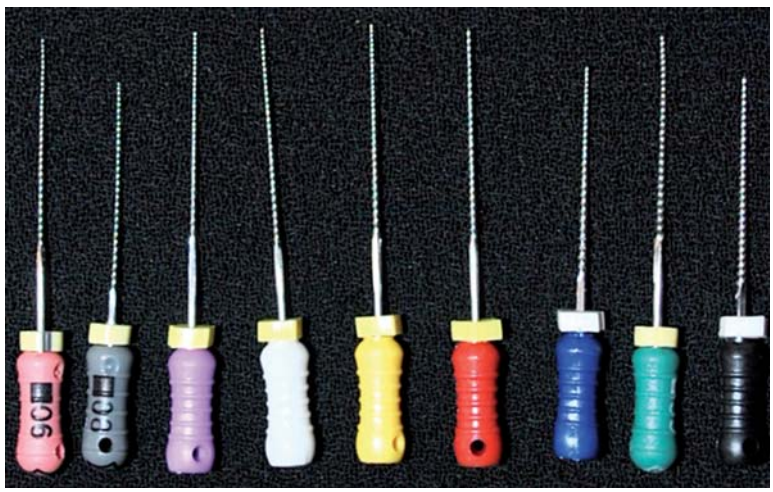


Fig. 10.4: Color coding

- By grinding and twisting the metal shaft's long axis:
  The raw metal is ground into tapered blanks and then it is twisted counterclockwise to form the desired instrument (Fig. 10.5).
  - K-files □
  - Reamers △
  - K-flexo □
  - Flexo □
- By machining the instruments (cutting a solid round metal rod) on lathe machine.
  - H-file
  - Flex-R
  - Uni files

(□—more resistant to fracture, △—increased cutting efficiency)

FEATURES OF ENDODONTIC INSTRUMENTS

Cutting Blade

Portion of the file behind the tip (0.16 mm in length) that removes dentin is called cutting blade.

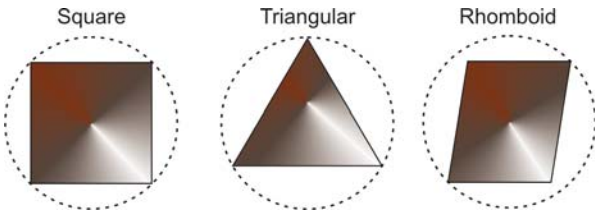


Fig. 10.5: Cross-sections of instruments

Taper

2°	0.02 mm increase in diameter/mm
4°	0.04 mm increase in diameter/mm
6°	0.06 mm increase in diameter/mm

Rake Angle

More acute the rake angle, the sharper the blade.

- Positive rake angle: Blade turns in the same direction of force applied to the surface being cut.
- Neutral: Blade is 90° to the surface.
- Negative rake angle:
  - Blade is turned opposite to the surface being cut.
  - Standard instruments have negative rake angles.
  - Such instruments require more energy to cut and they are less efficient.

Instrument Tips

- Cutting tip
- Modified tip (Safe cutting tip)
- Canal master tip (Noncutting tip or Batt tip)
- Precision cutting tip.

BARBED BROACH

Barbed broach is one of the oldest root canal instruments. However, because of its ease of breakage, it is confined to removal of soft tissues only. It is a short handled instrument meant for single use. It is made from soft steel wire, and barbs are formed by cutting into metal and notching the cut portion away from the shaft (Fig. 10.6). It is used in the straight portion of the canal. Working portion is 10 mm in length.

Methods of Use

- It should be used with caution as it is a fragile instrument.
- It is loose within the canal and engages soft tissues only just short of the apical foramen.
- It is rotated 360° to engage pulp and then withdrawn.
- If the tissue is not removed, the broach is lightly scrubbed along the wall from the apex outwards.
- When the instrument is forced apically within a tightly fitting canal, the barb is bent towards the shaft allowing deeper insertion of the instrument. While removing the instrument from the canal, barb is wedged against the wall of the canal and the sharp barb tips dig into the canal wall and fracture the shaft.

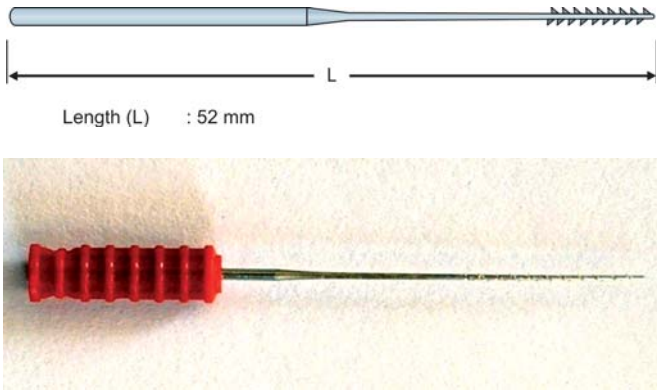


Fig. 10.6: Barbed broach

Use

Broaches should not be used until the canal is wide enough for insertion, and without digging the dentin. Care should be taken to use the barbed broach in the narrow buccal canals of maxillary molars and the mesial canals of maxillary molars.

- Vital pulp extirpation and removal of necrotic debris.
- Retrieval of paper points and cotton dressing from within the root canal.

Broaches are available in different sizes.

0	xxxxf	no. 10	Purple
1	xxxf	no. 15	White
2	xxf	no. 20	Yellow
3	xf	no. 25	Red
4	f	no. 30	Blue
5	m	no. 35	Green
6	st	no. 40	Black

RASP

- It is similar to barbed broach except in taper and barb size.
- It has shallower and more rounded barbs.
- They are used to remove the pulpal tissue from the canal.

	Broaches	*Rasps*
Barb height	• 1/2 tip diameter • Barbs are derived from core, making itweaker instrument	• **1/3** tip diameter· • Not as weak as barbed broach
Taper	0.007 to 0.010 mm per mm of length	0.015/0.020 mm per mm of length
Numbers of barbs	Average 40	Average 50–60

REAMERS

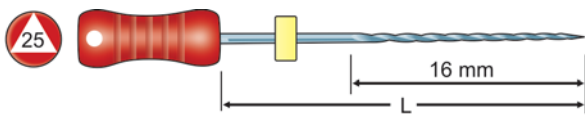
Reamers are K-type instruments which are used to ream the canal. They are symbolized by Δ.

Features

- Reamers have fewer blades than K-files. Cutting blades are 1/2 to 1 per mm while in file, it is 1 1/2 to 2 per mm (Fig. 10.7).
- Angle of blank is 60° creating a sharp knife like edge to shave the canal. Three 60° angles provide superior cutting efficiency.
- Triangular cross section shows less resistance to bending, are more flexible, and there is less risk to torsional fracture than those made from square blank.
- They are manufactured by twisting triangular blanks and are triangular in cross section (Fig. 10.8).
- Basic action: Insert into the canal, turn 1/4 to 1/2 clockwise in order that the blades engage into the dentin and then pull so that the dentin chips from the root canal. (Insertion-rotation-retraction)
- Reaming is the only action to produce a round, tapered preparation, when used in straight canals.
- Hand operated instruments are known as type K reamers.
- It is used with *pushing rotating motion.*
- Engine reamers do not readily follow the course of curved canal and may perforate the root canal. They are likely to break.
- Angle of blade to the long axis of K-reamer is about 10–30°. Hence, these instruments are primarily designed to be used in rotary reaming motion.
- Reamers tend to remain self-centered in the canal resulting in less chances of canal transportation.

Uses

- To enlarge and shape the irregular shapes of root canal into a cavity of rounded cross-section.
- Cleaning and shaping of root canal during Schilder's method.
- Though reamer has fewer numbers of flutes than file; cutting efficiency is less than that of file. More space between flutes provides more room for better removal of debris. They are used to remove the intracanal debris and old gutta-percha fillings from the canal.



Lengths (L) : 21 mm, 25 mm

Fig. 10.7: Reamer

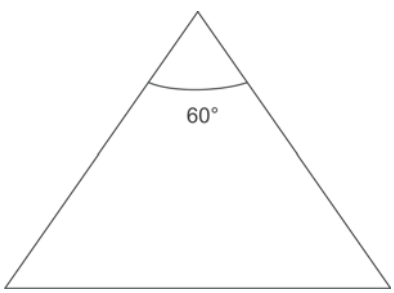


Fig. 10.8: Cross-section of reamer

FILES (FIG. 10.9)

Files are used for cleaning and shaping of the canal by machining the dentin. Various types of root canal files are;

- K-files
- K-flex
- Flexo file
- Flex-R
- Headstrom and safety headstrom
- S-file.

Files are predominantly used with filing or rasping action in which there is little or no rotation in the canals. After insertion into the canal, it is pressed against the canal wall and the instrument is then withdrawn while maintaining the pressure. The major effectiveness of hard tissue removal by filing is in outstroke or withdrawal of the instrument by dragging the flutes on the dentin wall.

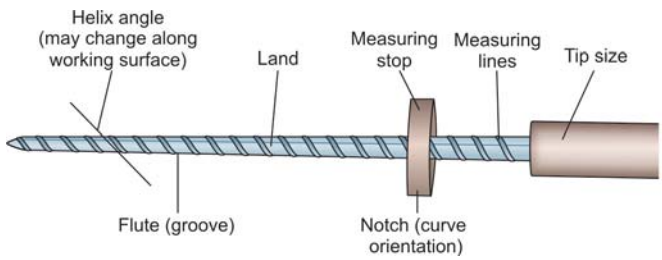


Fig. 10.9: Basic design of file

K-FILE

First manufactured by "KERR MANUFACTURING COMPANY" in 1904, and so it is named as K-files (Fig. 10.10).

Features

- K-file is square in cross-section. Tighter twisting of file spirals increases the number of flutes in file (Fig. 10.11).
- 1 1/2 to 2 1/2 cutting blades per mm of their working end.
- The tip is cutting and pyramidal in shape.
- The angle of the flutes to long axis is about 25-40°.
- It is placed into canal at desired length, pressure is exerted against the wall and it is withdrawn from canal without turning. It is used with *rasping and pulling motion*.
- This is the first instrument used to extirpate the pulp and shape the canal.

Cutting Flutes (Figs 10.12 and 10.13)

Files	1.5 to 2.5 per mm
Reamer	0.5 to 1 per mm

Modifications

K-flex file
Flex R-file
K-flexofile
Flexicut.

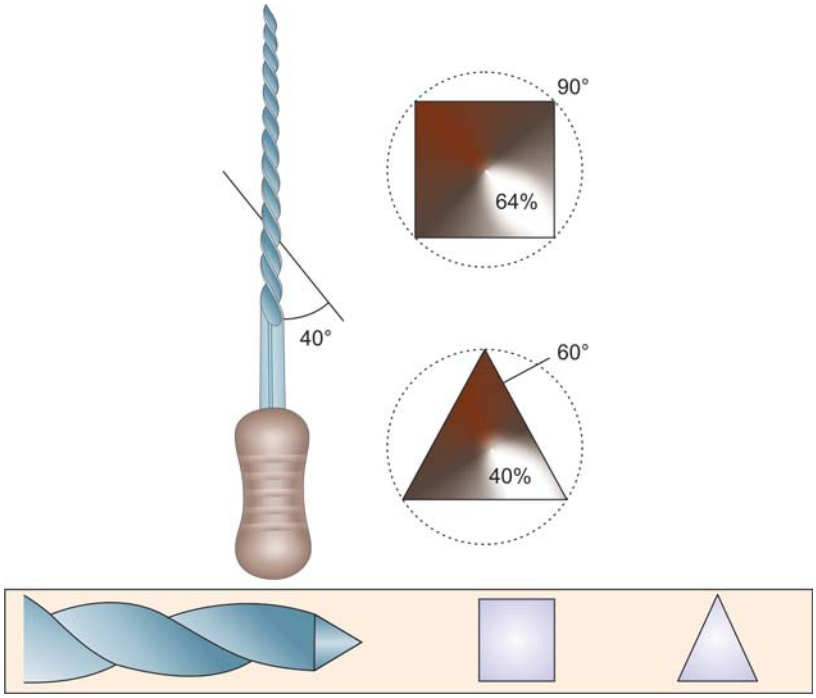


Fig. 10.10: K-file

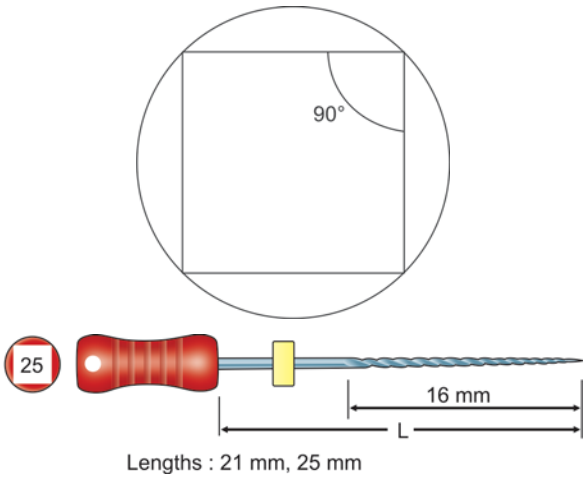


Fig. 10.11: K-file

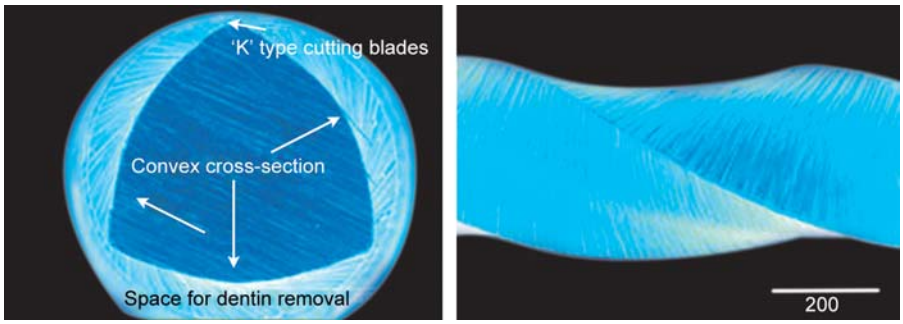
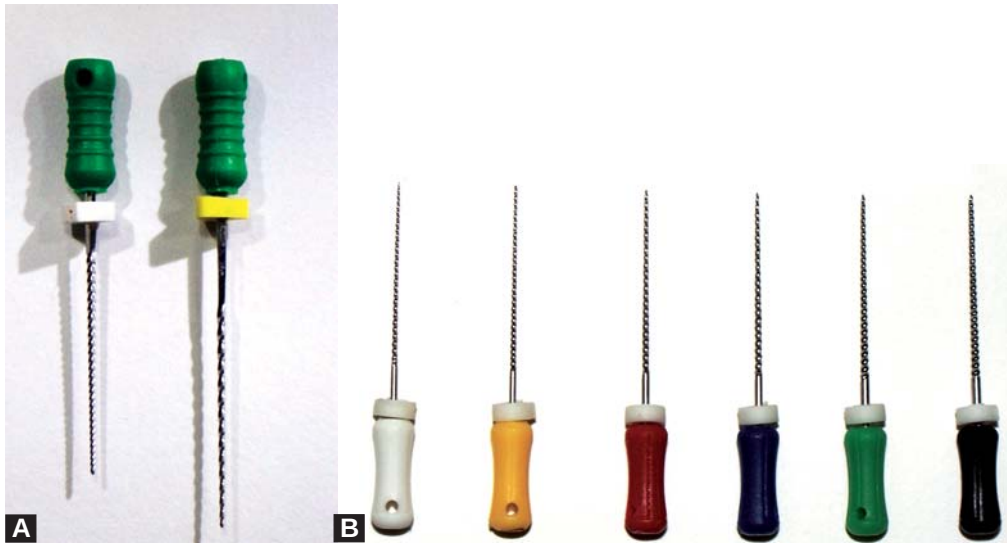


Fig. 10.12: Reamer (microscopic view)



Figs 10.13A and B: (A) K-file (left), reamer (right) (B) Files of different sizes

K-FLEX FILE (D-TYPE FILE)

- K-flex file was introduced by Kerr manufacturing company in 1982. The main purpose of developing this file is to increase the flexibility and cutting efficiency while maintaining the shape of canals, especially curved canals.
- They are rhomboid or diamond shaped in cross-section. Angle at the tip is 25°-50°. It is fabricated from V-4 steel and made from stainless steel by twisting □/Δ cross section.
- It has series of cutting flutes with alternate sharp (<60°) and obtuse noncutting edges. The two acute angles which increase the sharpness and two obtuse angles give more room for efficient removal of debris and also decrease the contact of instrument with canal walls.
- It has greater cutting efficiency, increased flexibility and, because of increased space between the working edges acting as a reservoir, it has increased ability to remove debris.

FLEXO FILE

- Flexo file was first manufactured by "MAILLEFER". It is made up of stainless steel.
- It is same as K-File except that it has triangular cross section. This triangular cross section gives sharper cutting blades and more room for debris than K-FILE.
- Its extremely flexibility helps in resisting fracture. It has noncutting tip (butt).

FLEX-R FILE

Flex-R file was developed in 1985 by Roane's for use in the "BALANCED FORCE TECHNIQUE".

- The file is made by removing sharp cutting surface at the tip of cutting edge so the tip rides along the canal rather than the gauge (stuck) into it. This design eliminates the possibility of ledge (block) formation or canal transportation.
- Triangular in cross section. Flutes are sharper with less negative rake angle.

- Clockwise rotation with anticlockwise rotation and apical pressure create less canal blockage and less extruded apical debris than step back technique.
- It is mainly advocated for curved canals.

H-FILE (HEDSTROM FILE)

- Hedstrom file is manufactured from round wire that is machined to produce spiral flutes resembling a series of intersecting cones. It resembles Wood screw or the Christmas tree (Fig. 10.14).
- It is manufactured by cutting spiral grooves into round, tapered steel wire to produce sharp cutting edges which cuts only on pulling strokes (retraction) (Fig. 10.15).
- It has elevated cutting edges which face towards the handle. They are larger from tip towards handle and they cut on withdrawal stroke only.
- Highest cutting efficiency due to positive rake angle. Cutting edges are turned in the same direction of applied force so they are very aggressive in cutting.
- The angle between the cutting edges and the long axis of the instrument is about 60-65°. Thus, they are designed primarily for a linear filling motion (Figs 10.16 and 10.17).

Other Uses

- It is primarily used to clean and shape the coronal half of canal. It is quite effective in irregular canals (immature teeth) and harbors debris.
- Hedstrom files are strong, aggressive cutters, and as they lack flexibility and are fragile in nature. They are used to machine straight canals or coronal portion of curved canals.
- They are used to flare the canal orifices.
- They are used to remove broken instruments, gutta percha and silver points. The file is placed alongside the material to be removed, then rotated, and pulled towards the occlusal surface.

Drawbacks

- Weak due to smaller shaft diameter and prone to breakage on torquing motion. Furthermore, it is almost impossible to remove the fractured file from the root once

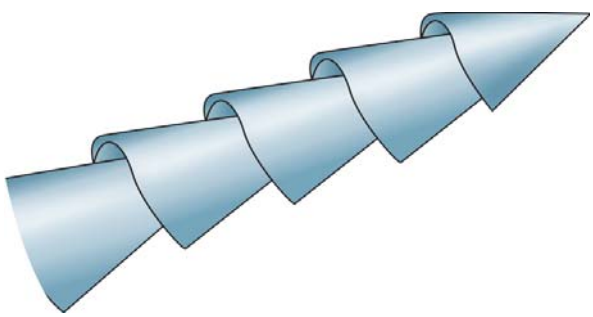


Fig. 10.14: H-file

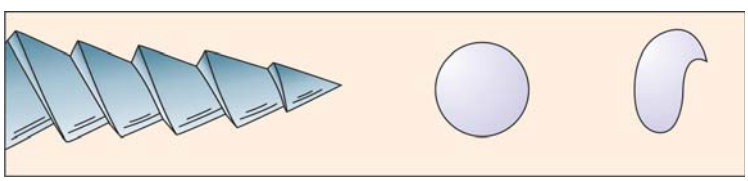


Fig. 10.15: H-file

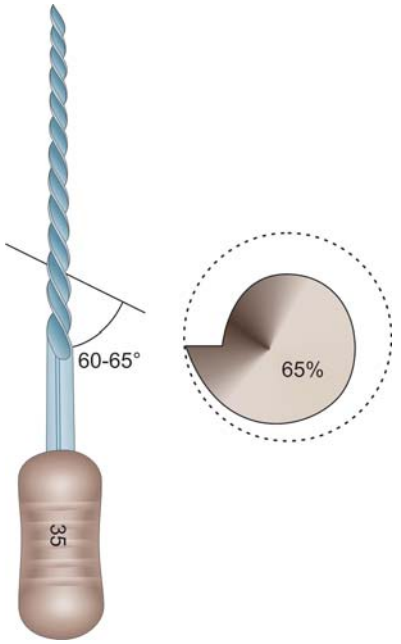


Fig. 10.16: H-file

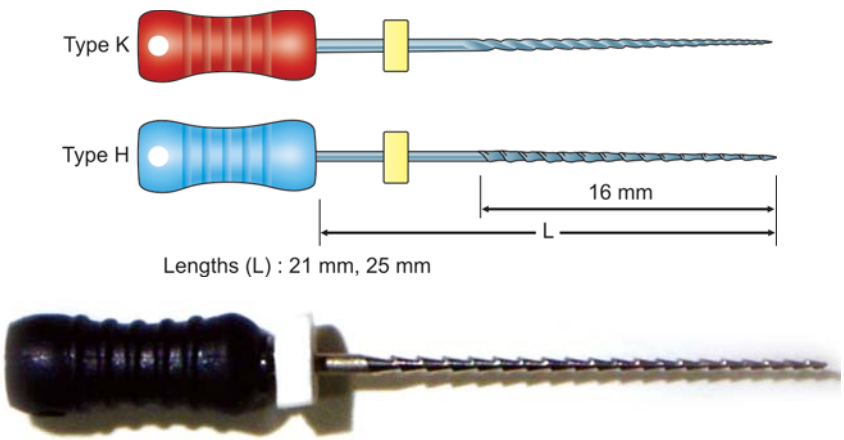


Fig. 10.17: H-file

it is locked in the dentin. It can be withdrawn only by backing off until the flutes are free.

- Screw like configuration further drives the instrument apically and cracks the weakened or stressed root on clockwise rotation.

Modifications

Unifile/ Dynatrak
S-file
Safety H-file.

UNI FILE

Mc-spadden modified the traditional hedstrom file, which were marketed as Burns unifile.

- Unifiles are manufactured by cutting superficial grooves to produces a file in a double helix design.
- Blades present an S-shaped or double helix design rather than the single helix teardrop shape of hedstrom file.
- Unifile has 2 continuous cutting edges.
- Uniform depth of flutes renders the tip more flexible. It is stiff in coronal and middle thirds but bends at apical thirds to form noncutting tip.
- It can be used in both filling and reaming actions hence referred to as "universal file".
- It is less effective in cutting but less subjected to fracture.

S-FILE

- S-file was developed in Sweden. It is machined from solid piece of wire to produce a sharp, double, cutting edge forming double helix design. They are stiffer than H files.
- It is "S" shaped in cross section and has 90° cutting tip. It is available in size of 5 to 40.
- S-file has good cutting efficiency in both filing and reaming actions and thus it is also classified as hybrid design.
- Angle of flute is constant and flute depth increases from tip to handle.
- Its noncutting sides with smoothened edges prevent ledging in curved canal.
- Millimeter scale is etched on the shaft of the file for length control.

SAFETY HEDSTROM FILE

- Kerr manufacturing co. introduced these files in 1998.
- It has a noncutting safety side along the length of blade which reduces the potential for strip perforation.
- Noncutting side is oriented to the side of the canal where cutting is not desired. It is indicated by a *flattened side* of handle (Fig. 10.18).

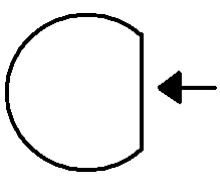


Fig. 10.18: Safety hedstrom file

- It is used with traditional vertical filling technique. When used in a linear filing motion, it helps to prepare curved canals without ledging the outer wall.

CHAPTER

11

Hand Instruments in Conservative Dentistry and Some Considerations for Viva Voce

BASIC CLASSIFICATION OF DENTAL INSTRUMENTS

Dental instruments are classified in many ways, including by the number of working ends, by function, by manufacturer's name and number, and by Black's number formula.

Number of Working Ends

According to the number of working ends, instruments can be divided into two categories; single-ended and double-ended instruments. Single-ended instruments have only one working end and long handles. Double-ended instruments have two working ends in the following combinations.

- The two ends have similar functions, but one end is larger than the other (difference in size).
- The two ends are paired right and left for preparing the right or the left side of the cavity preparation (for example, gingival marginal trimmer).
- The two ends have a combination function in which the ends are used for the same procedure but each end has a different use (for example, plastic filling instrument).

Instruments Classified by Function

Instruments are classified by function. Operative hand instruments are categorized as cutting and noncutting. Other instruments are classified according to a specialty, use with a specific material, or a procedure.

Manufacturer's Number

The manufacturer's number is written on the handle of the instrument. This number, used for ordering the instrument, indicates the instrument's placement in a set of instruments. Some instruments are named or classified by the name of the individual who designed the instrument.

Black's Formula

GV Black developed the formula for standardizing the exact size and angulation of an instrument. This formula minimizes discrepancies in the production of instruments from one manufacturer to other and simplifies the ordering of these instruments.

DENTAL INSTRUMENTS CAN BE GROSSLY DIVIDED INTO FOLLOWING TWO CATEGORIES

Cutting Instruments

Hand cutting instruments are used to assist in the design of the cavity preparation. They define and redefine the cavity walls and margins. There are six main hand cutting instruments; chisels hatchets, hoes, gingival margin trimmers, angle formers and excavators.

Hand	*Rotary*
Hatchets	Burs
Chisels	Stones
Hoe	Disks
Excavators	Others
Others	

Noncutting Instruments

Noncutting instruments include the basic examination instruments and the instruments used to insert and finish amalgam and composite restorative materials. They can be categorized as follow;

1. *Condensing instruments*:
   Pluggers—Hand, Mechanical.
2. *Plastic instruments*:
   - Spatulas
   - Carvers
   - Burnishers
   - Packing instruments
3. *Finishing and polishing instruments*:

Hand	*Rotary*
Hand held wooden stick	Finishing burs
Polishing points	Mounted stones
Finishing strips	Rubber cups
	Integrated disks
	Wheels

4. *Isolation instruments*:
   - Rubber dam kit
   - Salivary ejectors
   - Cotton roll holders
   - Evacuating tips.
5. *Miscellaneous instruments*:
   - Mouth mirror
   - Explorers
   - Probes
   - Tweezers
   - Pliers.

INSTRUMENT NOMENCLATURE

GV Black categorises the operative hand instruments as below:
1. Order: It indicates purpose of instrument (scaler, excavator)
2. Suborder: It indicates position or manner of use (push, pull)
3. Class: It indicates form of working end (hatchet, chisel)
4. Subclass: It indicates shape of shank (monoangle, biangle)

INSTRUMENT FORMULA

GV Black gave the formula describing dimension and angulations of the hand instruments. The formula consists of four units.

1st figure	Width of blade in 10th of millimeter
2nd figure	Angle between the cutting edge and the long axis of the handle (Primary cutting edge angle). It is always greater than 50°
3rd figure	Length of blade in millimeter
4th figure	Angle which the blade forms with the axis of handle. It is expressed in 100th of a circle or centigrade. It is always less than 50°

E.g. 10 – 80 – 10 – 12 indicates,	
10	Blade width in 10th of 1 mm ($^{10}/_{10}$ = 1 mm)
80	Cutting edge angle in hundredths of circle
10	Length of blade in mm (8 mm)
12	Blade angle in centigrade

The basic formula was consisting of three units only. The second figure was added later on. These three basic measurements are sufficient to indicate the majority of operative instruments. However, the instruments with cutting edge and with angle other than the angle of blade to long axis, requires a fourth unit. So it was added to the second place on the basic formula.

EXPLORING INSTRUMENTS

Mouth Mirror

This instrument is used to reflect light onto the field of operation, to view the cavity indirectly, and to retract the cheek or tongue, as necessary. It consists of stainless steel handle with a round mirror which is angulated/straight to handle.

Types

A. Based on material:
   - Stainless steel
   - Plastic (Plastic mirrors are disposable).

B. Based on size:
   - No. 3, No. 4, No. 5 (most commonly used are no. 4 and no. 5)

C. Based on reflecting surface of the mirror:
   - Flat (plane, regular) surface mirrors

     They have reflective surfaces (silver coating) on the back of the glass. This gives the image- "a ghost image"; it reflects only once to give a clear view free of distortion.

- Concave
  They magnify the image.
- Front surface mirrors
  They have reflective coatings (rhodium) on top of the glass. This coating eliminates the "ghost image"; it reflects only once to give a clear view free of distortion.

D. One-sided, double-sided:
In double-sided mirror, one side is used to reflect the cheek or lips and the back side provides illumination. Concave mirrors provide enlarged view of the sight but may be misleading if the clinician is not well oriented with the amount of enlargement of view.

Uses

- For illumination
- For retraction and protection of soft tissue
- For percussion of tooth
- To check tooth mobility
- For indirect vision.

Explorer

Explorers are single-ended or double-ended instruments. The working end is a thin, sharp point of flexible steel. This allows the operator to examine surface of the teeth to detect any irregularities and hardness. It is used to identify where soft carious dentine is present. There is a variety of angles of explorers, and often the ends are different so the operator can access various areas of mouth. Several common shapes include the pig tail, the shepherd's hook, the right angle and the #17.

Care should be taken not to poke the explorer point into very small carious lesions. This may destroy the tooth surface and the caries arrestment process. Also, do not probe into deep cavities where you might damage or expose the pulp.

Parts

Handle, shank, tip.

Types

Straight	*Right angle*	*Arch explorer*	*Interproximal explorer*
Shank is straight except for a single curvature near the explorer tip.	Shank is at right angle to the tip.	Shank is curved in a semi circular shape like an arch, and the exploring tip, at right angle to handle. E.g. Shepherd's hook explorer	Shank has angle, and the tip is pointed towards handle.

Uses

- For diagnosing carious lesion
- For detecting plaque and calculus
- For checking furcation involvement
- For percussion of tooth
- For checking overhanging restoration
- For checking smoothness of walls and floors.

Cotton Pliers (Tweezers)

Cotton pliers have got narrow and angulated working end. They may have serrations on the inner aspect of the beaks for better holding of material. These instruments are used for carrying cotton wool rolls, cotton wool pellets, wedges and articulation paper from the tray to the mouth and back. They are available in locking handles, and the tips may be straight or angled.

Types

A. Locking and non-locking
B. Working ends can be:
   - Straight/curved
   - Serrated/smooth

Uses:

- To carry and hold the cotton rolls, thus aid in isolation.
- To remove any broken fragment of tooth or material from the mouth.
- To make cotton rolls.

INSTRUMENTS FOR TOOTH STRUCTURE REMOVAL

They are of three types:
1. Hand cutting and rotary instruments
2. Abrasive instruments
3. Ultrasonic instruments.

RESTORATIVE INSTRUMENTS

Mixing Instruments

Spatula

It is used for mixing of restorative materials. Some of these spatulas can cause discoloration of the material being mixed. The selection of a mixing spatula is not critical except when preparing a permanent anterior composite restoration. Some composite restoration material discolors easily, so use the spatulas provided by the manufacturer when working with it.

They have flat, wide nibs with blunt edges and straight shank. They are available in different sizes and stiffness. They are made up of stainless steel or plastic.

Plastic Filling Instruments

Plastic filling instruments are used to place and condense pliable restorative materials and to place cement bases in the cavity preparation. It is also called as cement carrier.

They are made up of stainless steel, ivory or plastic, and are usually double ended. They have flat sides with blunt corners. They may have Teflon coating to minimize adhesion and facilitate easy cleaning.

Plugger end: This end is flat, round and nonserrated, and looks like smooth condenser.
Paddle end: It is flat and elongated.

Uses

Paddle end: To carry and handle materials after mixing while material is in plastic/sticky stage.
Plugger end: To condense the material into prepared cavity.

Condensing Instruments

- They have hammer-like working end, which is large enough to compress the soft amalgam without sinking into it. The working end of condenser is called "face", and the blade is called "nib".
- They are available in different shapes, shank angles and curvatures.
- They may be single-ended or double-ended. Double-ended instruments have one end smaller and other larger.
- Amalgam condensers are often called *"pluggers"*.

Types

According to shape:
- Round
- Triangular
- Parallalogram.

According to basis of surface:
- Smooth surface for amalgam
- Serrated surface for gold.

Uses

- For condensing restorative materials
- For root canal obturation with hot gutta percha.

Burnishers

Burnishers are the instruments with smooth-faced nibs, and with different angulations and curvature of shank.

Shapes

- Ball-shaped
- Egg-shaped
- Apple-shaped
- Beaver tail-shaped
- Conical
- Fish tail-shaped
- Bullet-shaped.

Uses

- For initial carving of amalgam.
- To burnish the amalgam before and after condensing so as to adapt amalgam to the margins of restorations, reducing chances of leakage and deficient margins (Pre-carve and postcarve burnishing).
- To contour the matrix band according to contour of tooth.

Carvers

Carvers are usually double-ended instruments and are available in different sizes.

Shapes

Working end is of different shapes.
- Flat (hollenback carver)
- Discoid (disc shape)

- Cleoid (pointed like claw of bird toe)
- Diamond.

Uses

They are used for carving of amalgam restoration. The sharp end is used for carving the occlusal anatomy like grooves and marginal ridge. Operator preference and shape of restoration will dicate which carver is required.

HAND INSTRUMENTS

They are classified as:
A. Excavators
B. Chisels
C. Other cutting instruments.

Excavators

	Excavators			
	Hatchet	*Hoe*	*Angle former*	*Spoon excavators*
General	A dental hatchet resembles a camper's hatchet, except much smaller.	Dental hoes look like a miniature garden hoe.	It is a *four numbered* instrument in which cutting edge is at an angle to the blade. It is combination of GMT and chisel because it encompasses the advantages of both.	The spoon excavator is modified hatchet. It is a double-ended instrument with a spoon, claw, or disk-shaped blade. Spoon excavators are used primarily to remove debris from tooth cavities.
Design	– Hatchet *blades* are set at 45-90° angles from the shank. These instruments have different lengths and widths of blades. – The *width* of the blade of the instrument is approximately 1 mm. An example is the Ash 10-6-12. – The *cutting edge* and long axis of the handle are in the same plane. – It may be *single-ended or double-ended*.	– Hoe *blades* are set at a 45- to 90-degree angle from their handle. (Hoe is the chisel with angle of blade greater than 12.5°) – It can be mesially *beveled* (Edge is beveled towards the shaft) or distally beveled (Edge is beveled away from the shaft) – *Cutting edge* is perpendicular to the long axis of tooth.	– It is a special type of excavator with a *bevel* of 80° to the shaft which forms an acute angle with the long axis of the blade. – Available as right and left sided instrument; single- or double-*ended*.	– It has bin-*angled* or triple angled shank for improved accessibility. – Their tips and sides are designed for cutting action. The most common sizes are the small and the large spoon excavators. – Paired instrument with blade curved to right and left. – Direction of curve of *blade* makes it lateral cutting instrument. – Circumferential *bevel* is sharpened to thin edge. – *Cutting edge* is ground to semicircular shape.

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Sizes				They are of three sizes: *Small*: – The diameter of the spoon is about 1 mm. – An example is the Ash 153-154. It is for use in small cavities and for excavating the caries. As the neck of the instrument is rather fragile, it can break if too much force is applied whilst excavating. *Medium*: – The diameter of the spoon is about 1.5 mm. – An example is the Ash 131-132. It is mainly used for removal of soft caries from larger cavities. The rounded surface of the spoon can also be used to push mixed restorative material into small cavities. *Large*: – The diameter is about 2 mm. – An example is the Ash 127-128. This instrument can be used in large cavities and for removing of excess glass-ionomer material from the restoration. – It has enlarged working blade.

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Shapes				*Discoid* – Circular disk-like shape. – Circular blade placed at an angle to shaft. – Cutting edge extends all around the periphery except where it joins to shank. *Cleoid* – Blades resemble claw of bird
Motion	– It is used with push – pull motion. – Right-sided instrument is moved from left to right and left sided instrument is moved from right to left.	– They are used with a pulling motion to smooth and shape the floor and sides of cavity preparations.	– It works in three motions: Vertical, push, pull	– Right/left lateral motion
Uses	– Hatchets are used on the wall of the cavity preparation to cleave enamel and to cut dentin to prepare a sharp cavity outline. – This instrument is used for widening the entrance to the cavity, for slicing away thin unsupported and carious enamel, left after carious dentin has been removed. – It can be used to prepare retentive undercuts in the anterior teeth cavity preparation.	– Planning the prepared walls and forming line angles. – Sharpening and defining of the line angles. – Delicate cutting during the gold restoration preparation especially class III and V preparations.	– To accentuate the internal line and point angles of the cavity preparation (especially for cavity preparation for gold restoration) – To create retention form in dentin	– To remove caries – To carve amalgam and wax pattern – To remove the temporary crowns – To remove the temporary cements

Chisels

Chisels are designed after ordinary carpenter's tool and used to cut tooth structure. It is beveled on one side and, usually, double-ended; one end with standard bevel on blade and the other end with a reverse bevel.

Uses

Chisels are used to cleave (split) tooth enamel, to smooth cavity walls, and to sharpen cavity preparations.

Types

- Straight
- Mono angle
- Biangle—Cleave/split the undermined enamel
- Triple angle—To flatten the pulpal wall

Chisels			
	Straight	*Bi-angle*	*Wedelstaedt*
Design	• Shank and blade are in the same plane. • Cutting edge is on one side only with bevel of the blade at right angle to the shaft. • Usually available as single-ended instrument.	• Shank has two angulations so called bin-angled chisel. • Single bevel cutting edge: Mesial or distal • May be single- or double-ended instrument	• Unlike the straight chisel, the blade is curved in relation to shank. • Usually it is available as a double-ended instrument. • Cutting edge has single bevel: Mesial or distal
Motion	It can be used in five different movements: Vertical, push, pull, right, left	• Mesially beveled instrument is used with push motion • Distally beveled instrument is used with pull motion	
Use	• Used to cut enamel and dentin margins • To plan the walls of the cavity preparation	• Used to clave and spit the undermined enamel • To cut and plane enamel in cavity preparation	• To clave and plan enamel in the cavity preparation

Enamel Hatchet

Enamel hatchet is similar in design to the ordinary hatchet but the blades are larger and heavier. It is a one-sided instrument. It is used to split undermined enamel in buccal and lingual proximal walls and for placing grooves. Right-sided instrument is identified by ring on shank.

Gingival Marginal Trimmers

- The gingival margin trimmers (GMTs) are modified hatchets that have working ends with opposite curvatures and bevels.
- It is a four numbered instrument with the cutting edge at right angle to the blade and the long axis of the handle. The blade has a curvature.

Uses

- It is primarily used with lateral cutting motion as the cutting edge is in a plane other than that of shaft.
- GMTs are used to trim, to smoothen and to shape the gingival floor of a cavity preparation.
- GMTs are available in double-ended styles and are used in pairs, such as the #26 and #27. This is because the working ends of the even-numbered instruments are designed for use on the distal surfaces, and the odd numbered are used on the mesial surfaces.
- It is also used for beveling and rounding of the axiopulpal line angle.

Number

90 to 100	To bevel distal margin
85 to 75	To bevel mesial margin
100, 75	For inlay/onlay
90, 85	For amalgam preparations

Other Cutting Instruments

Instruments		
Others	*Feature*	*Uses*
Knives	Known as finishing knives or amalgam or gold knives	For trimming excess filling material on gingival, facial and lingual margins of a proximal restoration.
Files	The teeth of the instruments are so positioned that it can be used in both push-pull motion	To trim excess filling material, particularly on gingival margin
Discoid-cleoid instruments	The working ends of the instrument are larger than the discoid or cleoid excavators.	– For carving occlusal anatomy – To burnish inlay-onlay margin

CONSIDERATIONS FOR OPERATIVE DENTISTRY AND ENDODONTIA VIVA VOCE

- **Mercury-free alloys:** These are Gallium-indium alloys. They are made up of Ag-Sn particles in Gallium-indium liquid. Ga-In substitutes for the Hg in dental amalgams. Gallium melts at 28°C and can be used to produce liquid alloys at room temperature.
  Disadvantages
  – Surface roughness
  – Marginal discoloration
  – Tooth fracture due to expansion
  – Difficult manipulation.
- If the adjacent proximal boxes are of different sizes, prepare the larger first so that the preparation for the smaller can be kept more conservative, and conversely restore the smaller first and the larger, last.
- **Bevels**
  Single beveled instruments. It has bevel on only one side.
  E.g. Enamel hatchet, GMT

Bibevelled instruments

It has beveling on both sides.

E.g. Ordinary hatchet, Osteotome

To determine whether the instrument has right or left bevel, the primary cutting edge is held down and pointing away, and if the bevel appears on right side then it is the right instrument of the pair.

Mesial bevel can be determined by observing the inside of the blade curvature. If the primary bevel is visible, it is the mesial bevel. If primary bevel is not visible, it is a distal bevel.

- Sharpness of the instrument can be tested by resting the cutting edge on a hard plastic surface. If the cutting edge digs over the surface, the instrument is sharp. If it slides, then the instrument is dull.

  In other method, observe the primary cutting edge under the light. If the light reflects, it indicates a blunt edge. Sharp edges don't reflect the light.
- **Number of blades:**
  1. Number of blades is always even.
  2. Excavating burs have 6-10 blades.
  3. Finishing and polishing burs have 12-40 blades.
  4. Greater the number of blades, smoother will be the cutting at slow speed.
- Steel burs are mainly used for finishing procedures.
- Cross cuts are needed on fissure burs to obtain adequate cutting efficiency, even at low speeds. They are not used at high speeds as they tend to produce an unduly rough surface.
- Vibrations above 1300 rpm are imperceptible to the patients, so sensations are lost at high speed range.
- Temperature during cavity preparation should not exceed 130°F or 60°C. When using high speed, low pressure and water coolant should be used to prevent overheating.
- Pressure is directly proportional to heat generated. Whenever rpm is increased, pressure has to be reduced. It should not exceed 4 ounces when using high speeds, and not more than 12 ounces, when using low speeds.
- Noise level more than 75db causes hearing damage.
- Glass ionomer bonds best to enamel than dentin and cementum.

Classifications of GIC

According to use by manufacturer:

Type I	Luting cement
Type II	Restorative cement
Type III	Liner and bases
Type IV	Fissure sealant cement
Type V	Orthodontic cement
Type VI	Core build up cement
Type VII	For geriatric and pediatric patients
Type VIII	Esthetic restoration of anterior teeth
Type IX	Posterior packable GIC

According to Application

Type I	Luting cement
Type II	Restorative cement
Type III	Lining cement
Type IV	Fissure sealant
Type V	Orthodontic cement

According to Wilson and McLean (1988)

Type I	Luting cement
Type II	Restorative cement a. Restorative esthetic b. Restorative reinforced

Latest Classification

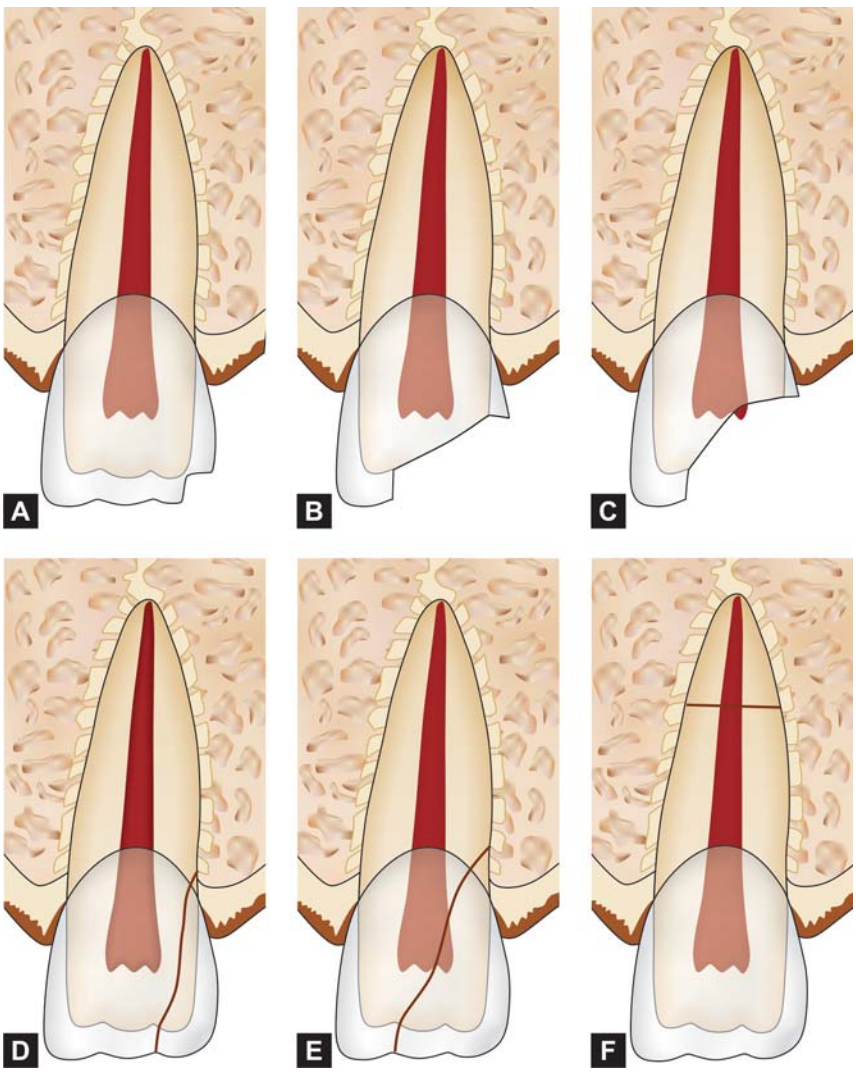
Traditional glass ionomer	Type I – Luting cement Type II – Restorative cement Type III – Liners and base
Metal modified glass ionomer	a. Miracle mix b. Cermet
Light cure glass ionomer	Hydroxy Ethyl Methacrylate is added to liquid
Hybrid glass ionomer/ resin modified glass ionomer	a. Composite resin in which filers substituted with glass ionomer particles b. Precured glasses blended into composites

CLINICAL CLASSIFICATION OF TRAUMATIC DENTAL INJURIES INCLUDING CODES OF WHO INTERNATIONAL CLASSIFICATION

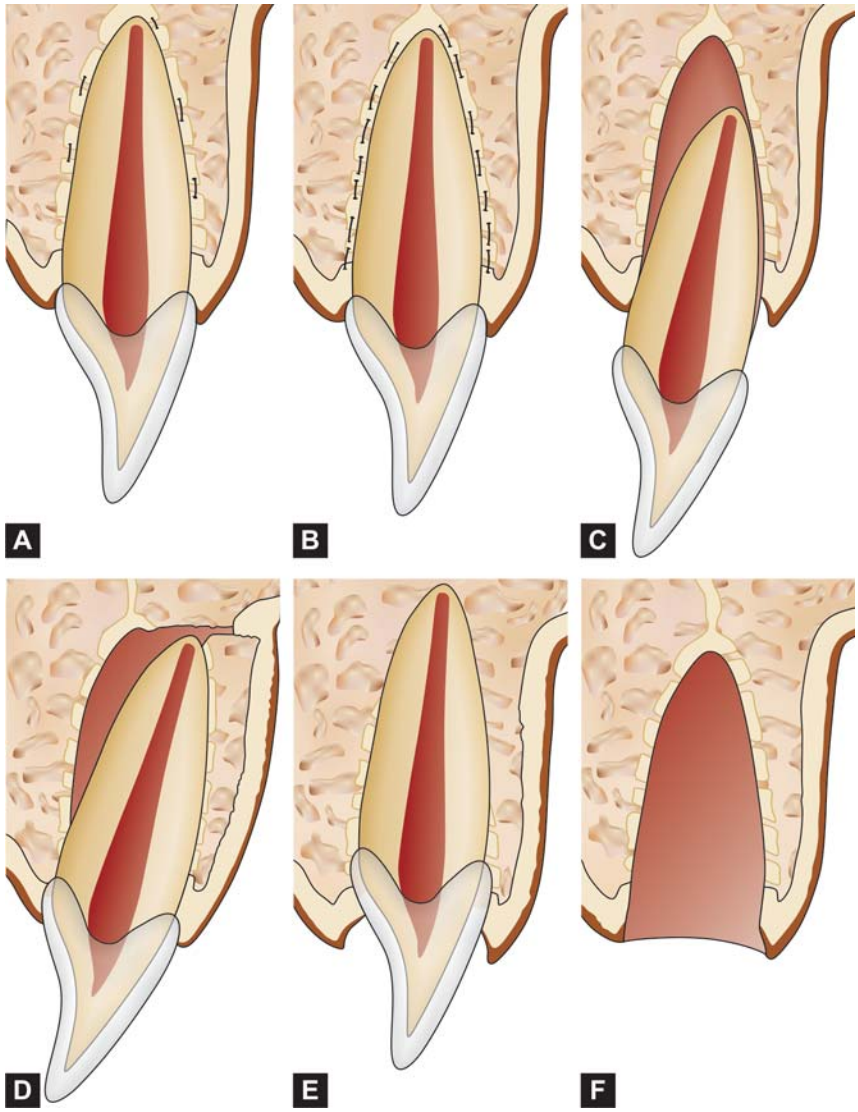
(a) Injuries to the hard dental tissues and the pulp		
Code	*Injury*	*Criteria*
N 502.50	Enamel infarction	An incomplete fracture (crack) of enamel without loss of tooth substance
N 502.50	Enamel fracture (Uncomplicated crown fracture) (Fig. 11.1A)	A fracture with loss of tooth substance confined to the enamel
N 502.51	Enamel-dentin fracture (Uncomplicated crown fracture) (Fig. 11.1B)	A fracture with loss of tooth substance confined to enamel and dentin, but not involving the pulp
N 502.52	Complicated crown fracture (Fig. 11.1C)	A fracture involving enamel, dentin, and exposing pulp
N 502.54	Uncomplicated crown-root fracture (Fig. 11.1D)	A fracture involving enamel, dentin, and cementum but not exposing pulp
N 502.54	Complicated crown-root fracture (Fig. 11.1E)	A fracture involving enamel, dentin, and exposing pulp
N 502.53	Root fracture (Fig. 11.1F)	A fracture involving dentin, cementum and pulp. Root fractures can be further classified according to displacement of the coronal fragments.

(b) Injuries to the periodontal tissues		
Code	*Injury*	*Criteria*
N 503.20	Concussion (Fig. 11.2A)	An injury to the tooth-supporting structures without loosening or displacement of tooth, but with marker reaction to percussion
N 503.20	Subluxation (loosening) (Fig. 11.2B)	An injury to the tooth-supporting structures with abnormal loosening, but without displacement of the tooth
N 503.20	Extrusive luxation (peripheral dislocation, partial avulsion) (Fig. 11.2C)	Partial displacement of the tooth out of its socket
N 503.20	Lateral luxation (Fig. 11.2D)	Displacement of the tooth in a direction other than axially. This is accompanied by comminution or fracture of the alveolar socket.
N 503.21	Intrusive luxation (central dislocation) (Fig. 11.2E)	Displacement of the tooth into the alveolar bone. This injury is accompanied by comminution or fracture of the alveolar socket.
N 503.22	Avulsion (exarticulation) (Fig. 11.2F)	Complete displacement of the tooth out of its socket
(c) Injuries to the supporting bones		
Code	*Injury*	*Criteria*
N 502.40	Comminution of the maxillary alveolar socket	Crushing and compression of the alveolar socket. This condition is found concomitantly with intrusive and lateral luxation (Fig. 11.3A)
N 502.60	Comminution of the mandibular alveolar socket	
N 502.40	Fracture of the maxillary alveolar socket wall	A fracture is confined to facial or oral socket wall (Fig. 11.3B)
N 502.60	Fracture of the mandibular alveolar socket wall	
N 502.40	Fracture of the maxillary alveolar process	A fracture of the alveolar process which may or may not involve the alveolar socket (Figs 11.3C and D)
N 502.60	Fracture of the mandibular alveolar process	
N 502.42	Fracture of maxilla	A fracture involving the base of maxilla or mandible and often the alveolar process (jaw fracture). The fracture may or may not involve the alveolar socket (Fig. 11.3E and F)
N 502.61	Fracture of mandible	

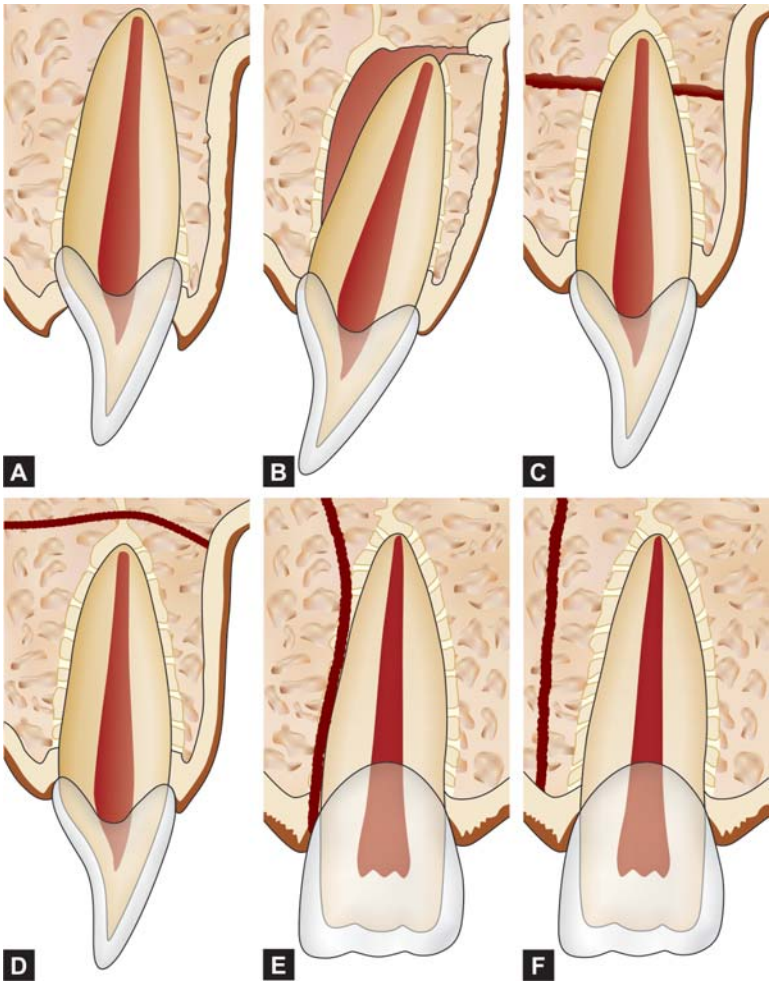
(d) Injuries to gingiva or oral mucosa		
Code	*Injury*	*Criteria*
S 01.50	Laceration of gingiva or oral mucosa (Fig. 11.4A)	A shallow or deep wound in mucosa resulting from tear, and usually produced by a sharp object
S 00.50	Contusion of gingiva or oral mucosa (Fig. 11.4B)	A bruise usually produced by impact with a blunt object and not accompanied by a break in the mucosa, usually causing submucosal hemorrhage
S 00.50	Abrasion of gingiva or oral mucosa (Fig. 11.4C)	A superficial wound produced by rubbing or scraping of the mucosa leaving a raw, bleeding surface.



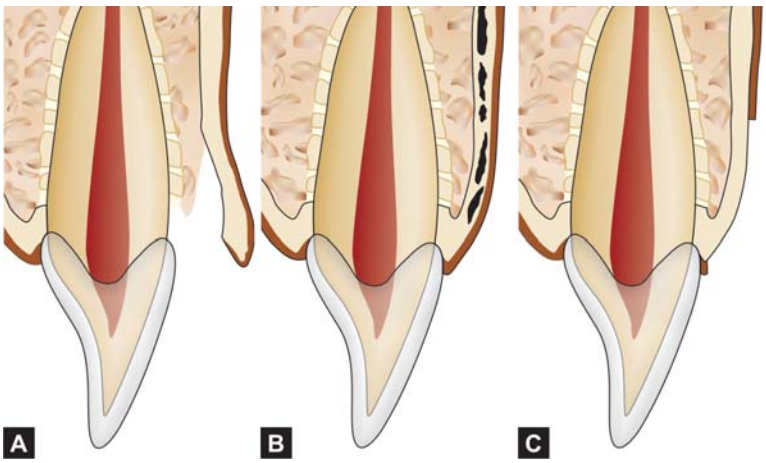
Figs 11.1A to F: Injuries to the hard dental tissues and pulp (A) Crown infarction and uncomplicated fracture without involvement of dentin. (B) Uncomplicated fracture with involvement of dentin. (C) Complicated crown fracture. (D) Uncomplicated crown-root fracture. (E) Complicated crown-root fracture (F) Root fracture



Figs 11.2A to F: Injuries to the periodontal tissues (A) Concussion. (B) Subluxation. (C) Extrusive luxation. (D) Lateral luxation. (E) Intrusive luxation. (F) Exarticulation



Figs 11.3A to F: Injuries to the supporting bone (A) Comminution of alveolar socket. (B) Fractures of facial or lingual alveolar socket wall. (C and D) Fractures of alveolar socket with and without involvement of tooth socket. (E and F) Fractures of mandible or maxilla with and without involvement of the tooth socket



Figs 11.4A to C: Injuries to gingiva or oral mucosa (A) Laceration of gingiva. (B) Contusion of gingiva. (C) Abrasion of gingiva

CHAPTER

12 Gingiva in Health and Disease

Health			Disease	
C/F	*Clinically*	*Histologically*	*Clinically*	*Histologically*
Size	Normal	The size of gingiva corresponds to sum of total bulk of cellular and intercellular elements and their vascular supply.	Size increases in case of gingival enlargement. Types of gingival enlargement are as mentioned below;	There are two types; – **Hypertrophy** Increase in size of cells. – **Hyperplasia** Increase in number of cells. It can be inflammatory or noninflammatory.
			I. Inflammatory enlargement A. Acute	A. Purulent focus in connective tissue surrounded by diffuse inflammation.
			B. Chronic	B. Exudative and proliferative features of chronic inflammation are seen.
			II. Drug-induced enlargement	Pronounced hyperplasia of connective tissue and epithelium
			III. Enlargement associated with systemic diseases and conditions A. Conditioned enlargement	

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Health			Disease	
C/F	*Clinically*	*Histologically*	*Clinically*	*Histologically*
Size			1. Pregnancy	1. Increase in epithelial and connective tissue cells with newly formed capillaries along with chronic inflammatory infiltrate.
			2. Puberty	2. Chronic inflammatory features.
			3. Vitamin C deficiency	3. Chronic inflammatory features.
			4. Plasma cell gingivitis	4. Chronic inflammation with dense plasma cell infiltration.
			B. Systemic diseases causing gingival enlargement	
			1. Leukemia	1. Various degrees of chronic inflammation and isolated surface areas of acute necrotizing inflammation.
			2. Granulomatous diseases	2. Chronic inflammation with scattered **giant cells** and foci of acute inflammation with microabscess.
			IV. Neoplastic enlargements (gingival tumors)	– Depends on type of lesion.
			V. False enlargement	– No abnormal features.
	Marginal and attached gingiva (Coral pink) Alveolar mucosa (Red)	Due to gingival vascular supply. Thickness and degree of keratinized epithelium. Presence	Color changes to 1. Red or bluish red in chronic gingivitis. Changes start in interdental papillae	– Due to vascular proliferation and reduction of keratinization due to epithelium

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	Health		Disease	
C/F	Clinically	Histologically	Clinically	Histologically
Color	Color is lighter in blond individuals compared to dark haired individuals	of pigment containing cells.	and gingival margin and spread to attached gingiva.	compression by inflamed tissue. – Bluish hue is due to venous stasis.
			2. Pale gingiva E.g. Phenytoin induced gingival enlargement.	Due to – ↓ Vascularization – Fibrosis – ↑ Epithelial keratinization
			3. Intensely red in acute gingivitis. Changes may be marginal (in ANUG), diffuse (in Herpetic Gingivostomatitis) or patch like (in acute reaction to chemicals).	Due to – ↓ Vascularization – Gingival infiltration – Red cell diapedesis – Tissue necrosis
			4. Dull whitish gray in severe acute necrotizing gingivitis.	– Tissue necrosis is demarcated from the adjacent gingiva by a thin, sharply defined erythematous zone.
Pigmentation	Normal melanin pigmentations are present. Diffuse, deep, purplish or irregularly-shaped brown and light brown patches are seen. Pigmentations are predominant in blacks and absent or severely diminished in albinos.	Due to presence of melanocytes in basal and spinous layer of gingival epithelium.	Abnormal gingival pigmentations 1. Metallic pigmentation, e.g. heavy metals like bismuth, arsenic, mercury, lead, silver produce black or bluish line in gingiva, which follows contour of margin. May also appear as an isolated black blotches involving interdental marginal and attached gingiva.	– Occurs as a result of perivascular precipitation of metallic sulfides in subepithelial connective tissue. Also due to gingival inflammation, ↑ permeability of irritated blood vessels leads to seepage of metal ions into surrounding tissues.

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	Health		Disease	
C/F	*Clinically*	*Histologically*	*Clinically*	*Histologically*
Pigmentation			2. In Addison's disease isolated patches of discoloration varying from bluish black to brown.	– Due to increased melanin production.
			3. Peutz-Jeghers syndrome and Albright's syndrome produce areas of melanin pigmentation.	
			4. In jaundice, oral mucosa becomes yellowish.	– Deposition of bile pigments in mucous membrane.
			5. In hemochromatosis, blue-gray pigmentation occurs in oral mucosa.	– Deposition of iron pigments in mucous membrane.
			6. Amalgam tattoo produces localized bluish-black areas of pigmentation.	– Implantation of amalgam particles in mucosa.
Consistency	Normal gingiva is firm, resilient and tightly bound to underlying bone (except free gingival margin)	Due to – Collagenous nature of lamina propria – Contiguity of lamina propria with mucoperiosteum of alveolar bone – Gingival fibers	*Acute gingivitis* 1. Diffuse puffiness and softening 2. Sloughing with grayish, flake like particles adhering to the eroded surface. 3. Vesicle formation	1. Acute inflammation (diffuse edema) 2. Necrosis with pseudomembrane formation (composed of bacteria, PMNs, degenerated epithelial cells in fibrinous network) 3. Intercellular and intracellular edema with degeneration of nucleus and cytoplasm, rupture of cell wall.

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	Health		Disease	
C/F	*Clinically*	*Histologically*	*Clinically*	*Histologically*
Consistency			*Chronic gingivitis* Both destructive (edematous) and reparative (fibrotic) changes coexist.	
			1. Soggy puffiness, pitting on pressure (in chronic gingivitis) 2. Marked softness and friability, fragmentation on exploration with probe, pin point surface areas of redness and desquamation. 3. Firm and leathery	1. Due to marked infiltration by fluid and cells of inflammatory exudates. 2. Degeneration of connective tissue and epithelium associated with inflammation, thinning of epithelium. 3. Fibrosis and epithelial proliferation.
Surface texture	Stippled gingival surface (numerous small depressions and elevations) similar to orange peel. Stippling is restricted to attached gingiva predominantly to the subpapillary area with variable extension into interdental papilla.	Stippling is produced by alternate rounded protuberance and depression of gingival epithelium into connective tissue. Papillary layer of connective tissue projects into elevations of epithelium. Prominence of stippling is related to degree of keratinization.	In chronic inflammation, gingival surface texture depends on whether the process is exudative or fibrotic. *Exudative* 1. Smooth and shiny with loss of surface stippling of gingiva occurs (in atrophic gingivitis). 2. Peeling of surface (in chronic desquamative gingivitis) *Fibrotic* Firm, nodular and leathery in consistency (in drug induced gingival overgrowth)	– Due to inflammation leading to epithelial atrophy. – Leathery due to hyperkeratosis and nodular due to gingival overgrowth.
	Marginal gingiva envelops teeth in a collar like fashion and follows scalloped outline on facial and lingual surface.	Contour of overlying gingiva follows the contour of alveolar bone margin and cementenamel junction.	Gingival contour is altered in disease. – The marginal gingiva becomes rolled or rounded and interdental	– Inflammatory changes in marginal gingiva.

Contd....

Contd....

	Health		Disease	
C/F	Clinically	Histologically	Clinically	Histologically
Contour	However, 1. In teeth with relatively flat surface, it forms straight outline. 2. In teeth with pronounced mesiodistal convexity, (e.g. Maxillary Canine) gingiva is located further apically. 3. On teeth with lingual version, gingiva is horizontal or thickened. The gingival appears prominent over the tooth and slightly concave in the interproximal area.		papilla becomes blunt and flat (in chronic gingivitis).	
			– Ballooning of interdental papilla and gingival margin (in inflammatory gingival enlargement).	– Inflammatory changes in marginal gingiva.
			– Flattened or cratered interdental papilla (in ANUG).	– Acute necrotizing inflammation of marginal gingiva.
			– Irregularly-shaped denuded appearance (in chronic desquamative gingivitis)	– Inflammatory changes in marginal gingiva.
			– Stillman's clefts (Apostrophe-shaped indentation of gingival margin for varying distance on facial surface)	– Inflammatory changes in marginal gingiva.
			– McCall festoon (Rolled, thickened band of gingiva) It is a life preserver-shaped enlargement of margin.	
			– Exaggerated scalloping (in gingival recession)	– Depends on the cause of recession.
	Normally absent	Normal histological features of gingiva	1. Chronic and recurrent bleeding. (In chronic gingivitis)	– Dilation and engorgement of capillaries due to gingival inflammation.

Contd....

Contd....

Health			Disease	
C/F	*Clinically*	*Histologically*	*Clinically*	*Histologically*
Bleeding on probing				– Capillaries lie closer to the surface. – Thinning or ulceration of sulcular epithelium. – Cell rich and collagen poor tissue.
			2. Acute episodes of bleeding. (In injury/ spontaneously in ANUG)	– Engorged blood vessels in inflamed connective tissue are exposed by ulceration of necrotic surface epithelium.
			3. Occurs spontaneously or after irritation. (Excessive and difficult to control)	– Failure of hemostatic mechanism.
Position	Normally, marginal gingiva envelops cervical margin of tooth crown in collar like fashion at or near CEJ.	Normal histological features of gingiva	Gingival recession – Exposed root – Root caries – ↑ Hypersensitivity	– Hyperemia of pulp.

CHAPTER

13 Dentogingival Junction

The dentogingival junction is an anatomical and functional interface between the gingiva and the tooth structure. It provides attachment of the gingiva to the enamel surface via hemidesmosomes. Biologic width is the term applied to the dimensions of the dentogingival junction. It was first described by Sicher in 1959. Biologic width is the apicocoronal distance in which the junctional epithelium and supracrestal connective tissue fibers are attached to the tooth.

The body maintains the biologic width as a stable dimension. When the biologic width is encroached upon and injured by extension of restorative preparations and materials into this area, uncontrolled inflammation results as the body tries to reestablish this dimension.

DEFINITION

Gingival apparatus maintains the free gingiva and junctional epithelium in close approximation to tooth. The attachment of the junctional epithelium to tooth is reinforced by the gingival fibers, which brace the gingiva against the tooth surface. So that the gingival fibers along with junctional epithelium is considered as functional unit referred to as "*Dentogingival unit*" (Fig. 13.1).

COMPONENTS

1. *Epithelial component* is derived from reduced dental (enamel) epithelium and oral epithelium. In some references, the epithelial part derived from the reduced dental epithelium is referred as primary attached epithelium or initial junctional epithelium. While in other references, a definitive epithelium replaces this initial one 3-4 years after the tooth erupts.
2. *Connective tissue component* is derived from the lamina propria of the oral mucosa. Gargiulo and colleagues studied the anatomy of the dentogingival junction and quantified the average as a constant 2.04 mm (the epithelial attachment is 0.97 mm, and connective is 1.07 mm). The distance is measured histologically from the most coronal part of the junctional epithelium (base of the sulcus) to the crest of the alveolar bone.

DEVELOPMENT

Just before the reduced enamel epithelium comes in contact with the oral epithelium during tooth eruption, the mitotic activity in the basal cells of the oral epithelium, at the cusp tips of the erupting teeth, decreases.

The reduced enamel epithelium in the basal cell layer, close to the cusp tip, starts displaying high mitotic activity.

After erupting, the cusp tip has become exposed in the oral cavity; it is surrounded by a shallow sulcus ending in the still attached reduced enamel epithelium, and bordered orally by the free gingival margin.

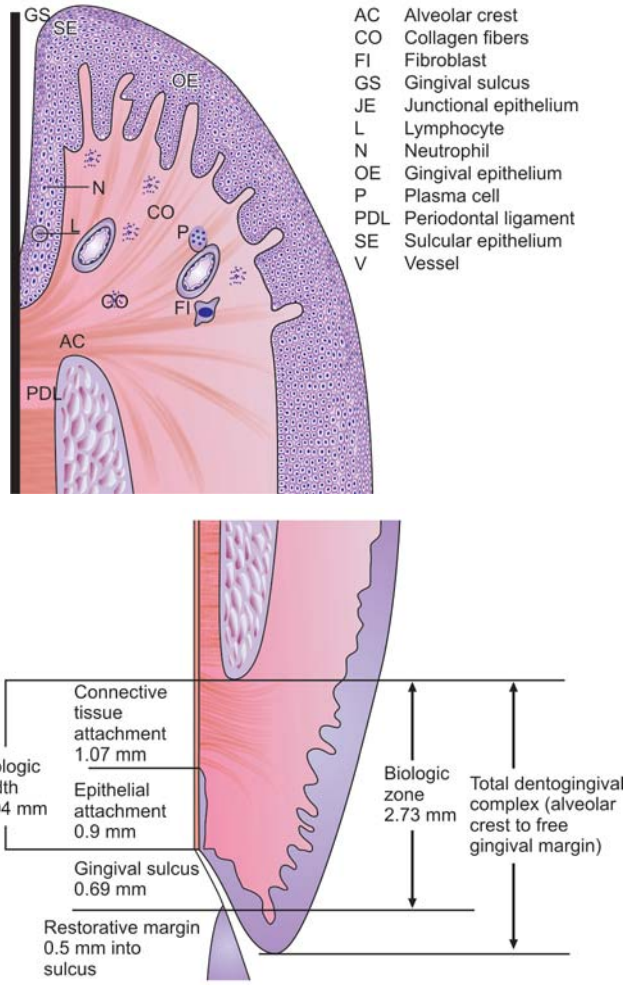


Fig. 13.1: Dentogingival junction

As the tooth erupts, the reduced enamel epithelium is gradually replaced through proliferation of its basal cells, and the new epithelium joining the tooth surface is called *"Junctional epithelium"*. The attachment of this epithelium to the tooth surface is called the *"Epithelial attachment"*.

MECHANISM OF ATTACHMENT OF THE DENTOGINGIVAL JUNCTION

- Electron microscopic findings revealed that the strong union between the soft gingival tissues, namely junctional epithelium and the tooth surface, namely enamel and/or cementum, are dependent on the presence of a basal lamina and hemidesmosomes.
- Junctional epithelium is associated with two basal laminae;
  i. One is present between the junctional epithelium and the tooth surface which is referred to as *internal basal lamina.*
  ii. The other is present between the junctional epithelium and the underlying connective tissue of the lamina propria, and it is called as *external basal lamina.*

Both basal laminae consist of a lamina lucida (clear layer) and lamina densa (dense layer). Though both basal laminae are morphologically similar, their biochemical structure is different. The basal lamina between the junctional epithelium and the tooth surface appears

to lack collagen types IV and VII. These collagen fibers are present at the basal laminae at the epithelial/connective tissue interfaces.

AGE AND INFLAMMATION CHANGES

- The level of the junctional epithelium relative to the tooth surface shifts apically with increasing age, and it is believed that inflammation is an important factor that contributes to this apical migration. This apical shift is gradual and if it is accelerated, this pathological condition is referred to as gingival recession.
- The underlying connective tissue is believed to play a significant role in determining the formation of the junctional epithelium. When connective tissue is destroyed during the periodontal disease, junctional epithelium migrates apically until it reaches intact connective tissue that provides the signal to stop its migration, forming long junctional epithelium.

CLINICAL IMPLICATION

The concept of biological width is used as a decision-making guide to the amount of soft tissue that should be removed during surgical procedure. The decision of surgical flap therapy depends upon the biologic width.

CHAPTER

14 Cementum in Disease

The cementum is a specialized mineralized tissue covering the root surfaces and, occasionally, small portions of the crown of the teeth. It has many features in common with bone tissue. The cementum contains no blood or lymph vessels and has no innervations. It does not undergo physiologic resorption or remodeling, but is characterized by continuing deposition throughout life. Like other mineralized tissues, it contains collagen fibers embedded in an organic matrix.

Exposed cementum is normally covered by a thin acquired pellicle of glycoproteins. The pellicle is believed to play an active part in the selective adherence of bacteria to the tooth surface.

Pathologic changes in exposed cementum are:

- Structural changes
- Cytotoxic changes
- Chemical changes
- Physical changes.

STRUCTURAL CHANGES

Presence of Pathologic Granules

Pathologic granules are 0.1 to 0.2 micrometer spaces between the external apical epithelial cells and cemental surface. It represents the zone of collagen degradation/areas where collagen fibrils have not been fully mineralized. It may represent the zone of bacteria/artifact.

Areas of Increased Mineralization

Exposure of cervical root surface to the oral cavity facilitates exchange of minerals at cementum–saliva interface. Minerals that are increased in diseased root surfaces include Ca, Mg, P, F. These areas of increased mineralization increase the tooth resistance to decay.

Areas of Demineralization

Areas of demineralization are often related to "root caries". Exposure to oral fluid and bacterial plaque results in proteolysis of embedded remnants of sharpey's fibers.

Cementum may become soft and may undergo:

- Fragmentation
- Cavitation.

Root caries progress around the tooth rather than deep into the tooth.

Active carious lesion	Yellowish or light brown areas covered by plaque. It has soft and leathery consistency.
Inactive carious lesion	Well-defined darker lesion with smooth surface and hard consistency on probing.

Dominant organisms are:

- *Actinomyces viscosus*
- *Actinomyces naeslundii*
- *Streptococcus mutans*
- *Streptococcus salivarius*
- *Streptococcus sanguis*
- *Bacillus cereus.*
- Tooth may not be painful. Exploration of the surface reveals presence of a defect and penetration of involved area with probe causes pain. Caries may lead to pulpitis, sensitivity to sweet and thermal changes or severe pain.
- Necrotic cementum must be removed during scaling and root planing.

Clinically, softening of cementum surface is usually asymptomatic, but painful when probe or explorer penetrates the area. They act as a reservoir for bacteria and reinfect area after treatment. Necrotic areas should be removed by root planing until a smooth hard surface reaches (Fig. 14.1).

CHEMICAL CHANGES

Mineral

- Chemical changes are in the form of increased mineral content of exposed cementum.
- Diseased cementum is characterized by either an absorption into or depletion of major mineral components like calcium, magnesium and phosphorus from cementum. By minerals absorption, cementum becomes highly calcified imparting increased resistant to decay compared to normal cementum. Absorption of toxic mineral is harmful.

	Prediseased Root	*Diseased Root*
Ca + Mg	26%	27.5%
Phosphorus	12.3%	12.6%

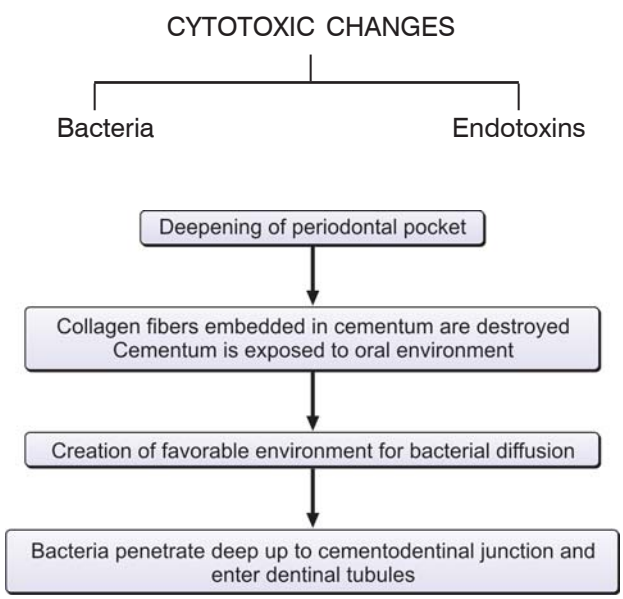


Fig. 14.1: Pathogenesis

I. *Bacteria* present are:
   - *Actinomyces viscosus*
   - *Streptococcus mutans*
   - *Streptococcus sanguis.*
   - *Streptococcus naeslundii*
   - *Streptococcus salivarius*

II. *Bacterial products* such as endotoxins are detected in the cementum wall of pocket. Diseased cementum depresses the fibroblast proliferation and prevents the attachment of fibroblasts. Cavitation and partial calcification as deep as 300 μm, without loss of contour, can harbor endotoxins. These endotoxins serve as a substance for inflammatory exudates.

III. *Exudates* contain
   - Histamine
   - IgG, IgA, IgM
   - Enzymes
   - Collagenase.
   - Bradykinin
   - Complement cementum
   - Hyaluronidase

IV. *Endotoxins* act as an antigen and promote activation of host response leading to:
   - Leukopenia
   - Activate factor XII (Hageman factor) leading to intravascular coagulation
   - Activate complement system
   - Localized Schwartzman phenomenon with tissue necrosis occurs after two more exposures
   - Cytotoxic effects on cells like fibroblasts
   - Bone resorption.

PHYSICAL CHANGES

Ankylosis

- Replacement resorption and ankylosis are often used as synonyms. Replacement resorption describes the active process of resorption of tooth and replacement by bony tissue while ankylosis is a Greek word for immobile.
- Ankylosis is a prerequisite for replacement resorption. It describes the fusion between tooth and bone, which can be permanent or transient depending on size of resorption areas.

Resorption and Repair

- Resorption is not necessarily a continuous process. It may interchange with periods of repair and forms a demarcating line called as “reversal lines”. However, cementum repair requires viable connective tissues.
- Root exposed by pocket formation appears as isolated areas, and from these areas bacteria penetrate into dentin. These areas undergo significant changes because they may perpetuate the periodontal infection and may complicate periodontal treatment.
- Areas of cellular resorption of cementum and dentin are common in root unexposed by periodontal disease. They are usually symptom-free and may undergo repair as long as they are covered by periodontal ligament.

Hypercementosis

Hypercementosis is characterized by cementum formation beyond the physiologic limits of the tooth. It may be localized to one tooth or affect the entire dentition. Excessive proliferation of cementum may be seen in association with a wide range of neoplastic and non-neoplastic conditions, including benign cementoblastoma, cementifying fibroma, periapical cemental dysplasia, and florid cemento-osseous dysplasia.

CHAPTER

15 Tooth Mobility

INTRODUCTION

Tooth mobility is defined as a "degree of looseness of tooth" (Kenry AAP, 1986). Mobility of tooth is one of the important signs as well as symptoms of periodontal disease. It helps in the diagnosis of periodontal disease by determining the disease prognosis. Mobility is a condition demanding immediate attention in order to save the tooth causing discomfort to the patient, affecting speech, mastication and esthetics, and leading to psychological trauma to the patient.

Mobility is most commonly noticed in the mandibular anterior region due to decreased thickness of alveolar bone and comparative less support in this region.

TYPES

The periodontal assessment includes an evaluation of tooth mobility. Tooth mobility should be distinguished between physiologic and pathologic mobility.

Physiological

Physiologic mobility is the normal or expected mobility of a tooth. It is a function of the height of the alveolar bone and the width of the periodontal ligament. It is limited tooth movement or displacement permitted by resiliency of the alveolar bone and cushioning effect of the periodontal ligament. All teeth have a slight degree of physiologic mobility due to cushioning of periodontal ligament. Physiologic tooth movement occurs when moderate forces are applied to the tooth during the oral examination and during the mastication.

Increased tooth mobility is physiologic in nature. It is due to the forces applied to the teeth surrounded by horizontal or angular bone loss and/or increased width of periodontal ligament but of normal composition. It depends on time, number and directions of the root.

Pathological or Abnormal

Tooth mobility beyond the physiologic range is termed as pathologic or abnormal. Only progressively increasing tooth mobility, which may occur in conjunction with inflammatory changes within the periodontal ligament, may be considered "Pathologic". It is the perceptible tooth movement in response to the force.

Other Terminologies

1. Altered tooth mobility
   Transient or permanent changes in the periodontal apparatus supporting the tooth leading to altered tooth movement are termed as altered tooth mobility.

2. Functional mobility
   Movement of tooth during functional and parafunctional forces is termed as functional mobility.

CAUSES

Mobility of tooth is caused by changes in periodontal structures (soft tissue changes, alveolar bone changes, periodontal ligament changes).

1. *Bone loss*
   - Loss of alveolar bone surrounding tooth is chiefly due to extension of the inflammation from gingiva into the periodontal structures. Severity and distribution of bone loss determines the degree of tooth mobility.
2. *Inflammation*
   - Gingival inflammation extending into the periodontal ligament results in degenerative changes that can increase tooth mobility, even in the absence of bone loss. These inflammatory changes are responsible for the increased tooth mobility in case of advanced gingivitis or early periodontal disease. The spread of inflammation from acute periapical abscess produces a temporary increase in tooth mobility in the absence of periodontal disease.
3. *Trauma from occlusion*
   - Tooth mobility is the most common clinical sign of trauma to the periodontium. It is seen in case of primary or secondary trauma from occlusion either in absence or in association with inflammation.
   - Tooth mobility during the injury stage is the increased tooth mobility.
   - Primary trauma from occlusion occurs when teeth in normal periodontium are subjected to excessive occlusal forces that cause pathologic mobility.
   - Secondary trauma from occlusion occurs when teeth in a compromised periodontium become mobile because they cannot withstand normal occlusal forces.
   - Initially, mobility is seen as a result of resorption of cortical plate and subsequently, reduced fiber support, and later on an adaptation phenomenon resulting in widened periodontal space makes the tooth mobile.
4. *Hypofunction*
   Hypofunction of tooth without antagonist widens the periodontal ligament space, and bigger room for tooth displacement increases the tooth mobility.
5. *Periodontal surgery*
   Tooth mobility increases immediately after the surgery but later on it diminishes below the pretreatment level by 4th week.
6. *Hormonal changes*
   Tooth mobility increases during pregnancy, menstruation or during use of hormonal contraceptive. It is associated with pregnancy, particularly in the last weeks before delivery, and it is probably due to increased hydration of connective tissue.
7. *Habits and systemic diseases*
   Abnormal habits, such as grinding (bruxism) and clenching are common causes of tooth mobility. Smokers exhibit increased tooth mobility. Other conditions that reduce periodontal support may lead to mobility. For example, Down's syndrome, Ehlers-Danlos syndrome, cyclic neutropenia.
8. Blunt injury to tooth and crown/root fracture.

FACTORS AFFECTING TOOTH MOBILITY

- Severity and distribution of bone loss at individual root surfaces.
- Number, length and shape of the root.

Single-rooted tooth exhibit more mobility than multirooted tooth. Likely, long-rooted tooth manifests less mobility compared to short-rooted tooth (It is highest in the central and lateral incisors).

- Frequency, duration, velocity and magnitude of occlusal forces. Periodontal ligament can tolerate tension much better than the compression.
- Root size compared to crown -

  A tooth with short-tapered roots is more likely to loosen than one with normal size or bulbous roots with same amount of bone loss (Ratio of crown-to-root length).
- Amount of inter-radicular bone.
- Number and distribution of remaining teeth in the arch.
- History of root amputation.
- Time.

In the morning, it is increased because of slight extrusion of tooth due to limited occlusal contact during sleep and progressively decreases during the day time. During the waking hours, mobility is reduced by effect of chewing and swallowing forces, which intrudes the tooth into socket.

INCREASED VS INCREASING MOBILITY

Increased

It is an adaptation of periodontium to occlusal forces that may not necessarily be considered pathological. Increased tooth mobility is observed in conjunction with trauma from occlusion. However, it may also be the result of reduction of the height of alveolar bone with or without accompanying angular bony defect.

Some of the factors producing increased tooth mobility are as follows:

- Loss of tooth support (bone loss)
- Trauma from occlusion
- Extension of inflammation from the gingiva or from the apex into periodontal ligament
- Periodontal surgery temporarily increases tooth mobility
- Pregnancy, menstrual cycle and hormonal contraceptives (Increases tooth mobility).

Mobile teeth with healthy connective tissue attachment can be maintained in the absence of inflammation. A widened periodontal ligament space may be a manifestation of adaptive changes to increased trauma. It may be accompanied by reduced height of alveolar bone.

Treatment: Only occlusal equilibrium and perhaps splint therapy.

Removal of the excessive occlusal load by occlusal equilibrium and splint therapy can decrease and even eliminate tooth mobility. Periodontal ligament regains its normal width and bone apposition may occur.

Increasing

It is due to pressure or localized infection causing bone destruction, and always pathological.

Treatment: This condition must receive the periodontal therapy, an occlusal analysis and equilibration, and if needed, followed by reevaluation for extraction or splinting of the affected teeth.

Single-time measurements of mobility are of limited value. However, when they are repeated at regular intervals and a change occurs, the alteration may be a significant finding that pinpoints a localized change or indicates an alteration in parafunctional habits (e.g. bruxism) that may affect the overall prognosis greatly.

MILLER'S CLASSIFICATION (1950)

Class I	Physiologic mobility
Class II	Up to 1 mm transverse movement
Class III	More than 1mm in any direction, vertical or horizontal

GLICKMAN'S CLASSIFICATION (1972)

Grade I	Slightly more than physiologic
Grade II	Moderately more than physiologic
Grade III	Severe mobility buccolingually and/or mesiodistally combined with vertical displacement

LINDHE'S CLASSIFICATION

Degree I	Movability of the crown of the tooth 0.2-1 mm in horizontal direction
Degree II	Movability of the crown of the tooth exceeding 1 mm in horizontal direction
Degree III	Movability of the crown of the tooth in vertical direction as well

STAGES OF TOOTH MOBILITY

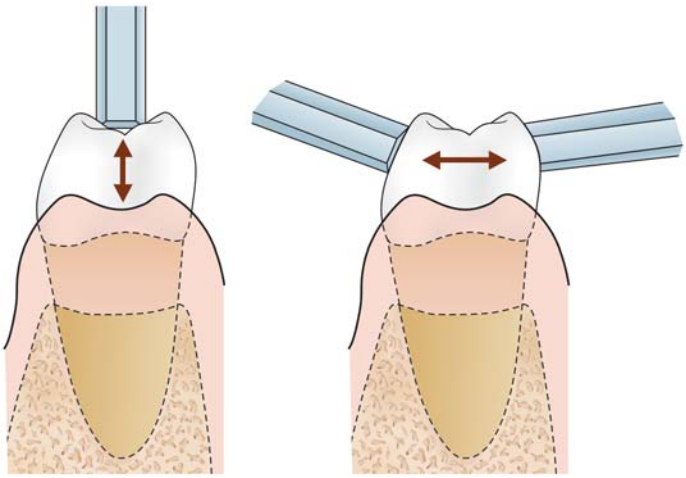
Tooth mobility occurs in two stages.

1. Initial stage/intrasocket stage:
   - It is the result of intraalveolar displacement of root and the movement occurs within confines of periodontal ligament.
   - Tooth moves within the confines of the periodontal ligament. It is associated with viscoelastic distribution of ligament, and redistribution of periodontal fluids so varies from person to person within the range of 0.05 to 0.10 mm (50-100 μm) with the force of about 100 gm.
2. Secondary stage:
   - There is gradual elastic deformation of alveolar bone in response to increased horizontal forces. When a force of 500 gm is applied to the crown, the resulting displacement is about 100 to 200 μm for incisors, 50 to 90 μm for canines, 8 to 10 μm for premolars, and 40 to 80 μm for molars.

MEASUREMENT OF TOOTH MOBILITY

Horizontal tooth mobility is the ability to move the tooth in a facial-lingual direction in its socket. Horizontal tooth mobility is assessed by putting the handles of two dental instruments on either side of the tooth and applying alternating moderate forces (Fig. 15.1).

- Tooth is held firmly between the handles of two metallic instruments. One on buccal/labial side and the other, on the lingual side of the tooth. Alternatively, a metallic instrument at one end and finger, as a substitute for other instrument, can also be used. But it is not recommended, generally, because the soft tissue pads of fingers act as a cushion and mask movement (Fig. 15.2).
- Now effort is made to move tooth in all directions (100 gm force is applied).
- Mobility is measured according to the ease and extent of tooth movement. A normal tooth has minute amount of "give" to it (i.e. is not ankylosed). A tooth that moves more than this minute amount but in total arc of less than 1 mm has class I mobility.



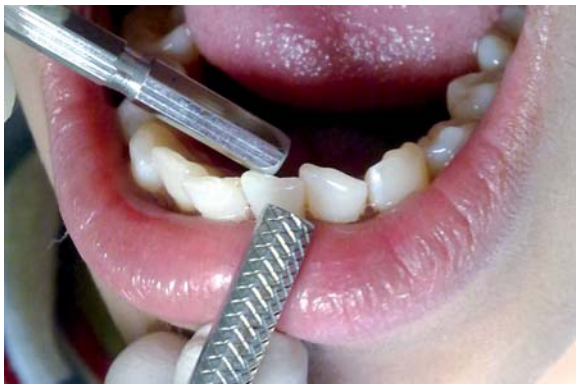


Fig. 15.1

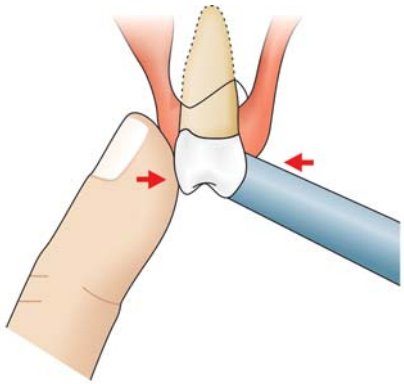


Fig. 15.2

Figs 15.1 and 15.2: Assessment of tooth mobility

- Abnormal mobility, most often, occurs in faciolingual direction. It is graded according to ease and extent of the tooth movement, using the above criteria. Mobility beyond the physiologic range is termed as "pathologic".

Vertical tooth mobility is the ability to get depressed the tooth in its socket, and is assessed using the end of an instrument handle to exert pressure against the occlusal or incisal surface of the tooth.

Instrument used to measure the tooth mobility is called as "*Periodontometer*".

Periotest

A new method for determining tooth mobility was presented by Schulte and coworkers (Schulte 1987, Schulte et al, 1992) using "Periotest" (Siemens AG, Bensheim, Germany) system. A tapping instrument delivers the controlled percussion force to the tooth and the reaction of the periodontium is recorded by the Periotest device. A metal rod is accelerated to a speed of 0.2 m/s with the device, and maintained at a constant velocity. The tooth is deflected, and the rod decelerated by impact with tooth. The contact time between the tapping head and the tooth varies between 0.3 and 2 milliseconds, and is shorter for stable than mobile teeth. Interpretation of Periotest is as mentioned below;

– 8 to +9	Clinically firm teeth
10 to 19	First distinguishable sign of movement
20 to 29	Crown deviates within 1 mm of its normal position
30 to 50	Mobility is readily observed

The Periotest values correlate well with:
1. Tooth mobility assessed with a metric system, and
2. Degree of periodontal disease and alveolar bone loss.

GENERALIZED TREATMENT OF MOBILITY

The primary thing in treatment of tooth mobility is removal of etiological factors. It is followed by treatment of contributing etiologic factors.

CHAPTER

16 Food Impaction

Food impaction is one of the predisposing factors for localized gingival and periodontal disease. It also aggravates the severity of preexistent pathologic changes. Failure to recognize and eliminate food impaction may be responsible for the unsuccessful outcome of thoroughly treated case of periodontal disease. It usually occurs in the interproximal areas but can also be seen in relation to facial or lingual tooth surfaces. Food impaction and subsequent retention may contribute to root caries in individuals who do not perform proper oral hygiene interdentally.

DEFINITION

"Food impaction is forceful wedging of food into the periodontium by occlusal forces (Hirschfeld 1930)."

However, in addition to vertical impaction due to chewing pressure, it also includes horizontal impaction by the forcing food interproximally by tongue or cheek pressure (AAP 2001a).

TYPES

There are two ways of food impactions;

Vertical Food Impaction

It is due to open contacts, irregular marginal ridges and plunger cusps. Open contacts between teeth may be anatomical in origin, iatrogenic in origin, or may be due to caries and pathologic migration of periodontally involved tooth.

Horizontal Food Impaction

Horizontal food impaction occurs due to enlarged gingival embrasures. Lateral pressure from cheeks and lips forces the food particles interdentally.

FACTORS AFFECTING FOOD IMPACTION (FIG. 16.1)

Normally forceful wedging of food is prevented by:

1. *Integrity and location of the proximal contacts*:
   Absence of contact or presence of unsatisfactory proximal tooth relationship favors food impaction and endangers the periodontal health (level of attachment) of adjacent teeth.
   - Intact, firm proximal contact prevents food impaction interproximally.
   - Coronally placed proximal contact reduces the funneling effect and forms smaller occlusal embrasures, and thus prevents food impaction.

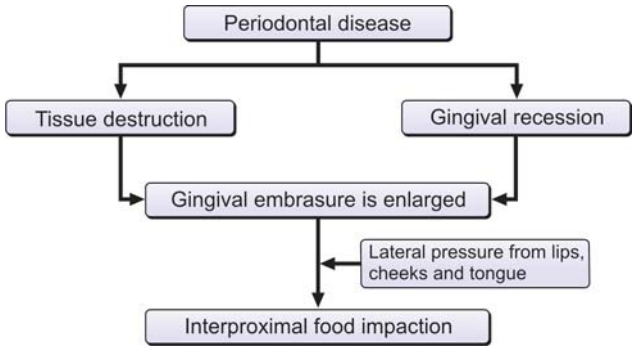


Fig. 16.1: Periodontal disease and food impaction

2. *The contours of the marginal ridges and developmental grooves*:

Occlusal surface loose the convexities and become oblique facets by attrition

↓

Wedging effect of opposing cusp into the interproximal area is exaggerated

↓

Food impaction

Contours of marginal ridges and related developmental grooves deflect the food away from the interproximal surface.

3. *Contour of facial and lingual surfaces*.
4. *Excessive anterior bite*:
   It is a common cause of food impaction on lingual surfaces of maxillary anterior teeth and facial surfaces of opposing mandibular teeth. This can be prevented by restoration of normal bite plane.

PLUNGER CUSP

Cusps that tend to forcefully wedge food interproximally are called plunger cusps. It occurs due to occlusal wear or as a result of shift in tooth position after failure to replace missing tooth (Fig. 16.2).

CLASSIFICATION OF FACTORS CAUSING FOOD IMPACTION

The classical analysis of the factors leading to food impaction was made by Hirschfeld (1928) who recognized the following factors:

- Uneven occlusal wear
- Opening of contact point due to loss of proximal support or from excursion
- Congenital morphological abnormalities
- Improperly constructed restorations.

CLASS I	Occlusal wear
CLASS II	Loss of proximal contact
CLASS III	Extrusion beyond the occlusal plane
CLASS IV	Congenital morphological abnormality
CLASS V	Improperly constructed restorations

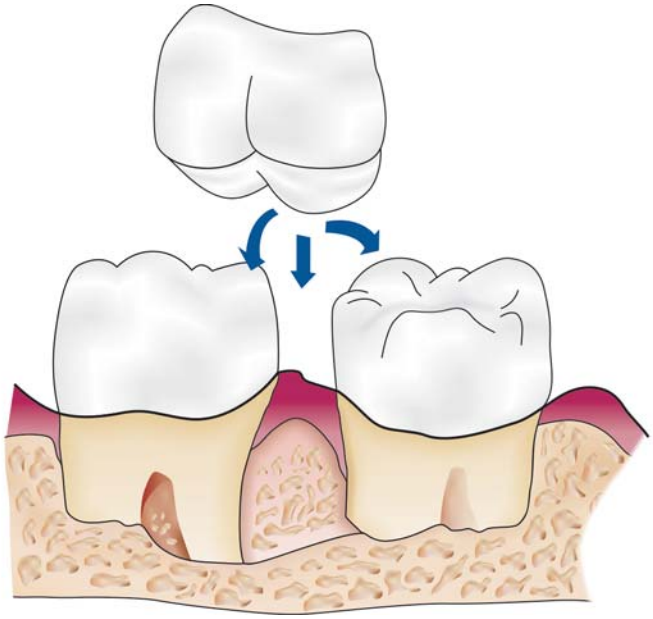


Fig. 16.2: Plunger cusp

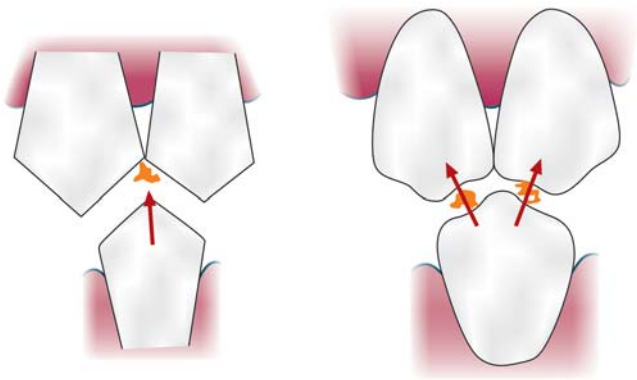


Fig. 16.3: Wedging effect due to occlusal wear

CLASS I: Occlusal Wear

Type A: Wedging action produced by plunger cusp into oblique facets (Fig. 16.3).
Type B: Remaining obliquely worn cusp of a maxillary tooth, overhanging the distal surface of its functional antagonist (Fig. 16.4).
Type C: Obliquely worn mandible tooth, overhanging the distal surface of its functional antagonist (Fig. 16.5).

CLASS II: Loss of Proximal Support

Type A: Loss of distal support through the removal of a distally adjacent tooth (Fig. 16.6).
Type B: Loss of mesial support due to extraction
Type C: Oblique drifting due to nonreplacement of a missing tooth

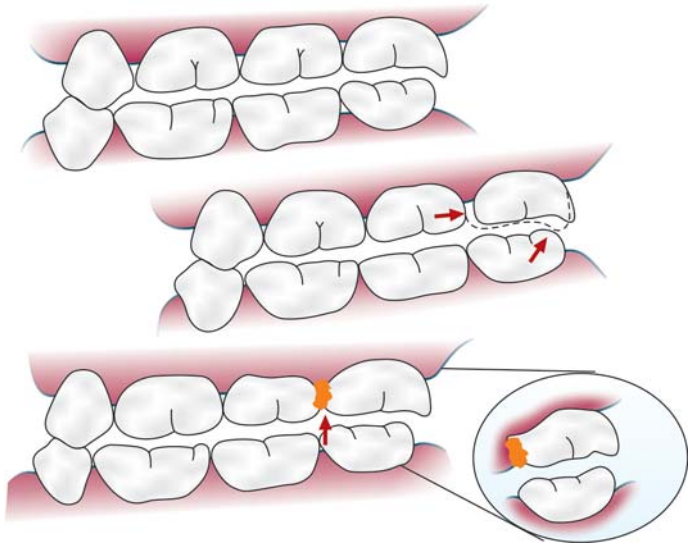


Fig. 16.4: Obliquely worn cusp of maxillary tooth

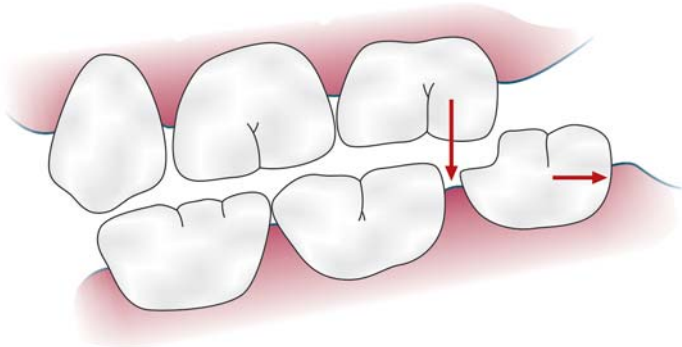


Fig. 16.5: Obliquely worn cusp of mandibular tooth

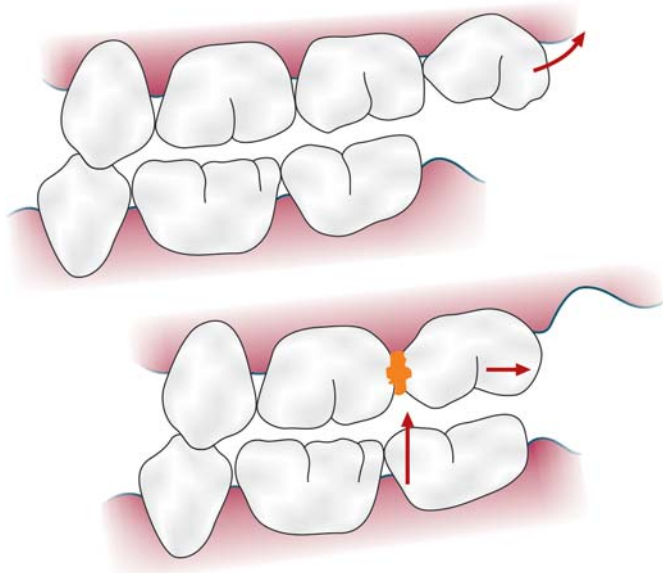


Fig. 16.6: Loss of distal support leading to food lodgement

Type D: Permanent occlusal openings to interdental spaces
i. Drifting after extraction
ii. Habits forcing teeth out of position
iii. Periodontal disease
iv. Caries

CLASS III: Extrusion

CLASS IV: Congenital Abnormalities

Type A: Rotation of tooth
Type B: Emphasized embrasure between thick-neck teeth
Type C: Faciolingual tilting
Type D: Malposition (Facial or lingual).

CLASS V: Improperly Constructed Restoration

Type A: Loss of contact point
Type B: Improper location of contact point
Type C: Improper occlusal contour
Type D: Improperly constructed cantilever restorations
Type E: Scalloped cervical bevels on the tissue-born areas of prosthetic restorations.

SIGNS AND SYMPTOMS

1. Trouble
   - Discomfort (feeling of pressure)
   - Vague pain
   - Root caries
   - Halitosis.
2. Periodontal Changes
   - Gingival inflammation (bleeding)
   - Gingival recession
   - Periodontitis
   - Periodontal abscess formation
   - Vertical alveolar bone loss
   - Crater formation.

PREVENTION AND TREATMENT

1. *Occlusal adjustment*:
   An occlusal adjustment to create adequate food escape grooves as well as to reduce prominent opposing cusps and correct uneven marginal ridges may completely eliminate food impaction (Fig. 16.7).
   a. Plunger cusp: Rounding of the sharp cusp tips especially lingual cusp.
   b. Level the occlusal height of the marginal ridges:
      i. Restoration of marginal ridge depends on (Fig. 16.8);
         1. Relation to opposing tooth structure to the overall plane of occlusion
         2. Degree of discrepancy
         3. Iatrogenic hazards.
      ii. Consider the slope of the marginal ridge (external, internal)
      iii. When a marginal ridge is being leveled, the proximal fossa is generally obliterated which must be recarved.

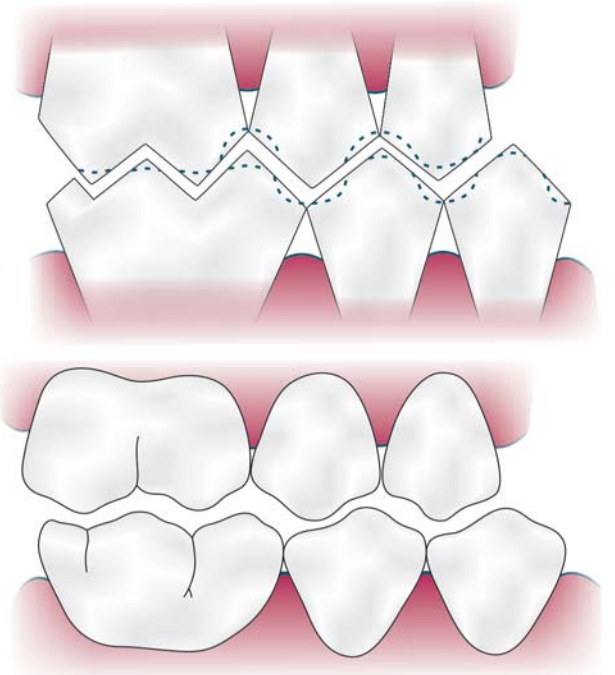


Fig. 16.7: Occlusal adjustment

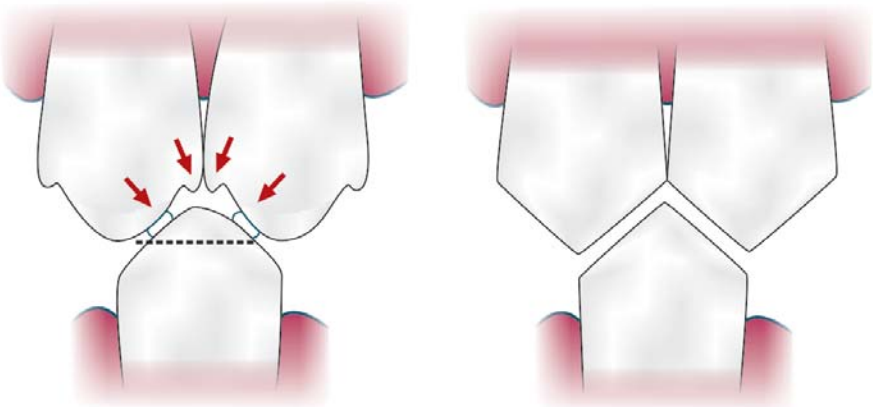


Fig. 16.8: Restoration of marginal ridge

c. Grooves and fossa (Fig. 16.9)
   i. Recarve obliterated or missing fossa.
   ii. Distinct but shallow, saucer-shaped fossa with slightly extended grooves mesially and distally act as spillways or sluiceways to deflect food from interproximal areas to buccal/lingual surface.

2. *Periodontal treatment*:
   Scaling, flossing, curettage and surgical flap therapy, and elimination of bony defects improve the periodontal condition.

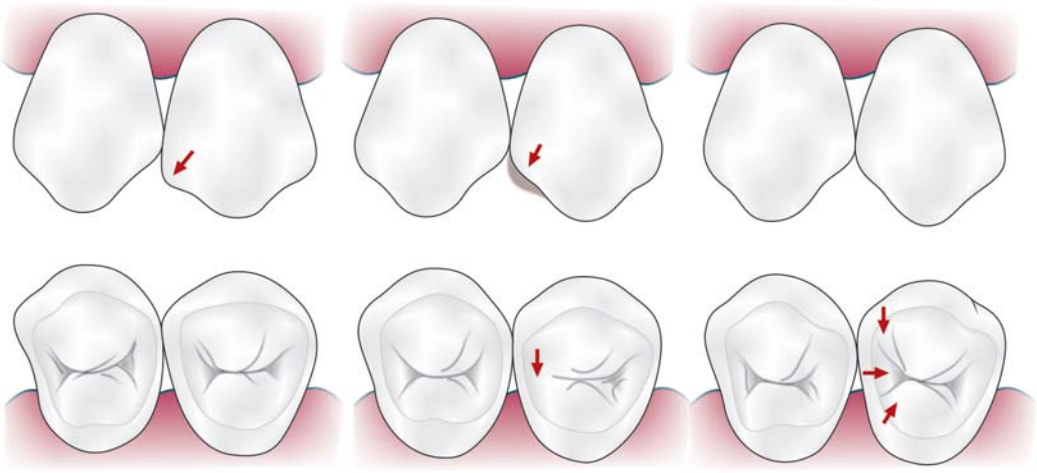


Fig. 16.9: Grooves and fossa

3. Making ideal contact:
   Intact, firm proximal contact relationships prevent forceful wedging of food interproximally and protect interdental papilla.
   Ideal contact should have ideal location, width, height and tightness. It can carefully be checked with dental floss.
4. Permanent restoration and proper prosthesis:
   Deficient areas require permanent restoration with proper
   - Proximal contact
   - Contour of occlusal surface
   - Facial and lingual contour.

   Faulty prosthesis should be replaced with proper proximal and occlusal relationship with other teeth of the oral cavity.

CHAPTER

17 Halitosis

The word halitosis originates from the Latin word *"halitus"* meaning to breathe and the Greek word—*"osis"* meaning abnormal or diseased. Halitosis itself is not a disease but it is a symptom of a disease. It is also known as "fetor ex ore", "fetor oris", and "oral malodor". It was 1st described by "HOWE" in 1874.

Breath odor can be defined as the subjective perception after smelling someone's breath. It can be pleasant, unpleasant or even disturbing, if not repulsive. If unpleasant, the terms breathe malodor, halitosis or bad breath can be applied.

DEFINITION

Halitosis is a foul or offensive odor emancipating from oral cavity and should be considered as a symptom, not as a disease.

CLINICAL FEATURES

Halitosis varies with age, gender, hunger state, and also with the time of the day. Morning is usually the worst. It also varies in intensity and quality.

Sex: More common in male

Age: More common above 40 years (85%).

It is experienced by the patient or by surrounding. It has impact on social environment, business and persons with whom he lives. The distinct odor produced by different diseased states has diagnostic importance. Oral malodor in healthy patients arises from the oral cavity and generally originates on the dorsum of the tongue. Tongue is an excellent site for growth of microorganisms and creates a unique ecological site.

TYPES

Transitory Halitosis

Transitory halitosis lasts between 24 and 72 hours, and is usually caused by foods such as garlic, pepper, onion, or by smoking. Everyone has transitory halitosis. It often disappears following eating, brushing teeth, flossing, and rinsing with specialized mouthwash. Breath malodor should not be confused with these momentary disturbing odors.

Chronic Halitosis

The cause of chronic halitosis is usually oral in nature but 10 percent of cases are reported to be caused by other factors such as a disease or ailment.

Physiological	*Pathological*
Morning Breath Hunger Breath	Not time specific
Less intense	More intense
Transient	Persistent
Nondistinct	Distinct in quality
Generally responds to change in habits	Requires treatment of the underlying cause and improvement of oral hygiene

PRIMARY FACTORS AFFECTING HALITOSIS

- Saliva flow
- pH of saliva
- Gram-negative bacteria
- Presence of protein and food debris.

MICROBIOTA IN HALITOSIS

T. denticola
P. endodontalis
P. intermedia
P. metaninogenica
B. forsythus
R. denticariosca
Fusobacterium.

MECHANISMS

- As the mouth is exposed to less oxygen and is inactive during the night (decrease in salivary secretion), the odor is usually worse upon awakening ("morning breath").
- The most unpleasant odors are known to arise from proteins trapped in the mouth, which are processed by oral bacteria, sheded epithelium and food debris.
- Halitosis is caused by liberation of volatile sulfur compound, specially hydrogen sulfide and methylmercaptun, which results from bacterial putrification of proteins containing sulfur, amino acids. These products could involve in transition from health to gingivitis and then progressively to periodontitis.
- The gaseous substances consist primarily of hydrogen sulfide (H_2S), dimethyl sulfide ($[CH_3]_2S$), methylmercaptan (CH_3SH) and sulfur dioxide (SO_2). The breakdown of cysteine and methionine produce hydrogen sulfide and methyl mercaptan respectively.
- Volatile fatty acids such as valerate, butyrate and proprionate are also malodorous.
- Tongue is the primary site for production of all these volatile substances. These locations in descending prevalence order are: interdental and subgingival niches, faulty dental work, food impaction areas in between the teeth, abscesses and unclean dentures. Irregular surface topography of the tongue is an ideal niche for bacterial adhesion and growth. Tongue coatings comprising accumulated food remnants, exfoliated cells and bacteria cannot be easily removed.

Halitosis is experienced more in the morning because of the following physiological changes during sleep like:

- Decreased salivary flow
- Decreased cleaning effect due to decreased movement of cheeks and tongue
- Decreased BMR (Basal metabolic rate).

ETIOLOGY (FIG. 17.1)

There are more than 300 bacterial species associated with oral malodor. All odor production is a result of gram-negative bacterial metabolism, the gram-positive contributes very little.

The normal pH of the mouth is slightly acidic (6.5-6.9). A pH level of 7.2 or more in the mouth increases the growth of gram-negative bacteria. A pH level of 6.5 or less in the mouth increases the growth of gram-positive bacteria.

Etiologic factors can be categorized into two main categories: intraoral and extraoral.

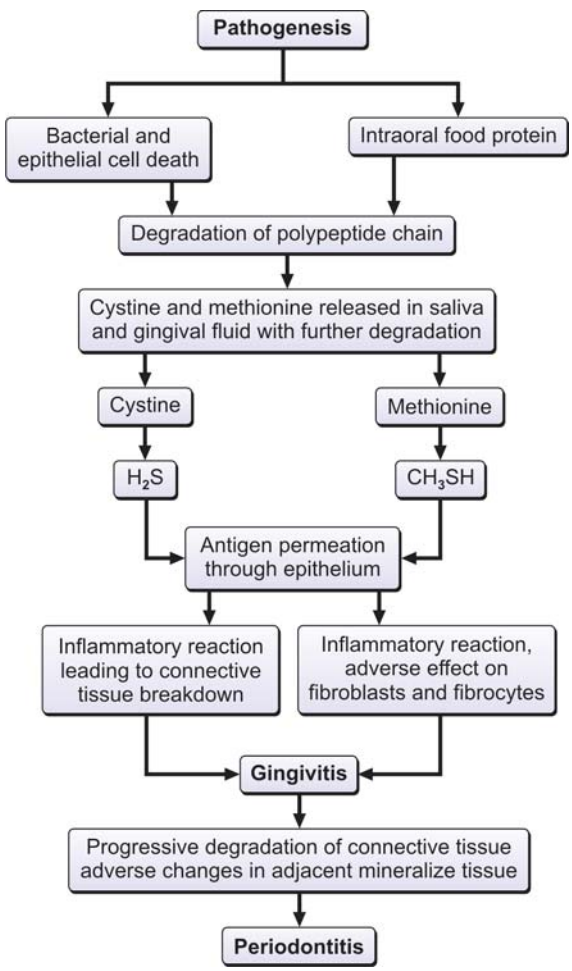


Fig. 17.1: Pathogenesis of bacteria

Intraoral Causes

Nonpathological

1. Heavy smoking (Transient halitosis)
2. Vulcanite denture halitosis (Denture breath)
3. Age-wise.

Children	Sweet odor due to food entrapment at tonsillar crypt.
Old age	Odor due to unclean dentures

Pathological

1. Poor oral hygiene due to any reason.
2. Food lodging areas in mouth resulting from;
   - Malposed teeth
   - Prosthetic, orthodontic or fractured appliance
   - Deep carious lesion
   - Loss of interdental papilla.
3. Periodontal pocket, pus and putrification of content materials.
   Bacteria associated with gingivitis and periodontitis are almost gram-negative and are known to produce VSCs (volatile sulfur containing substances). The low oxygen tension in deep periodontal pockets also results in low pH and activation of the amino acids to cadaverine and putrescine. These VSCs aggravate the periodontitis by increasing the permeability of the pocket and mucosal epithelium, and thereby exposes the underlying connective tissues of the periodontium to bacterial metabolites.
4. Other malodorous pathologies are:
   - Pericoronitis
   - Major recurrent oral ulcers
   - Herpatic gingivitis
   - Necrotizing ulcerative gingivitis.
5. Following surgery associated with oral bleeding and tissue necrosis resulting from various causes like diabetic gangrene, leukemia, and malignant growth.
6. Dry socket
7. *Coated tongue*.
   The dorsum of the tongue has very rough and uneven topography. The depressions created by the filiform and fungiform papillae are ideal niches for adhesion and growth of microorganisms. Food remnants intermingled with bacteria and exfoliated cells are not easy to remove from the dorsum of the tongue, and it is the primary site for oral malodor.
8. Dryness of mouth due to decreased salivary flow is associated with:
   - Mouth breathing
   - Heavy smoking
   - Salivary gland aplasia
   - Sjögren's syndrome, Mikulicz's disease
   - Peritonsillar abscess
   - Radiation therapy
   - Diabetes, lung cancer
   - Menopause in women

- Emotional distress (Vincent disease)
- Aging
- High fever
- Hormonal changes in the women.

  Large amount of deposits increasing the microbial load is frequently been observed in the patients with xerostomia.

9. Postnasal drip at the posterior dorsum of the tongue
10. Cyst with fistula draining in the oral cavity.

Extraoral Causes

Nonpathological

1. *Alcoholic breath*:
   Alcohol consumption causes dryness of mouth and alteration of microbial flora with proliferation of odor fermenting organisms.
2. Hunger odor by putrefaction of pancreatic juice.
3. *Stress*:
   It affects parasympathetic nervous system and decreases salivary secretion. Less cleansing effect by decreased salivary flow along with decrease in amount of oxygen present in oral cavity favors the growth of anaerobic organisms producing temporary bad odor.
4. *Smoking*:
   Liberation of certain chemicals from the smoke decreases salivary flow and also decreases amount of oxygen within oral cavity.
5. *Certain food partictes like*:
   Metabolites from certain ingested food excrete in the breathe air and impart odor to breathe.

   Onion, garlic impart odor to the breath by being absorbed in circulatory system and then liberates volatile agents in air which is ventilated through lung *(Garlic liberates volatile sulfur)*.

   Sugar containing diet is a good source of nutrition for anaerobic bacteria. Growth of anaerobic bacteria is responsible for breath halitosis.

   Dairy products like milk, cheese cause temporary halitosis. (They contain protein lactase which is not digested by humans but broken by anaerobic group of microorganisms).

   Nonvegetarian food.

Pathological

Respiratory	
Sinusitis	Tonsillitis
Pharyngitis	Tumors
Bronchitis	Pulmonary tuberculosis
Pneumonia	Lung abscess (Acid sweet odor)
Bronchiectasis (Foul putrefactive odor)	Gangrene of lung

Liver (Sweetish odor), Kidney (Odor of ammonia, uremic odor)	
Liver cirrhosis	Gallbladder disease
Liver failure	Uremia

Gastrointestinal	
Esophageal reflux	Hiatus hernia
Pyloric stenosis	Stomach cancer
Malabsorption	Diabetes (Acetone odor, fruity)
Zenker's diverticulum	Regurgitation esophagitis

Other
Rheumatic fever (Fluctuant amine odor)
Scurvy
Blood dyscrasias

Drugs causing halitosis	
Chloral hydrate	Antiparkinsonians
Metronidazole	Antidepressants
Amyl nitrate	Anticholinergics
Narcotics	Antihistaminics
Decongestants	Antihypertensives
Antipsychotics	Medication containing female hormones (estrogen, progesterone)

- Infections or lesions of respiratory tract.
  Odor from aromatic substances in bloodstream such as excretory products of cell metabolism is excreted in breathe air through the lung.
- Certain metabolic conditions involving enzymatic and transport anomalies like Trimethylaminuria.
- Mucosa of oral cavity and upper respiratory tract is used to expel volatile compounds from the body, including gases and metabolic end products of the diet (garlic, alcohol) produced in oral cavities as well as extraoral sites.

DIAGNOSIS

1. Organoleptic methods:
   - Breath consists of odor originating from both oral cavity and lungs.
   - Ask the patient to close the nostrils and the smell indicates that of the oral cavity.
   - Ask the patient to close the mouth and the smell indicates respiratory problems.
   - Individuals are instructed to refrain from using any dental products, eating, or using deodorants or fragrances 48 hours prior to visiting the dental clinic.
   - Subject is instructed to close the mouth for 2 minutes and to refrain from swallowing during this period. After 2 minutes, the subject breaths out gently at a distance of 10 cm from the nose of their counterpart.
     Organoleptic grading:

   (Organoleptic rating—Rosenberg and McCulloch)

   0 – No appreciable odor

   1 – Barely noticeable odor

2 – Slight but clearly noticeable odor
3 – Moderate odor
4 – Strong odor
5 – Extremely foul odor.

2. Tongue odor.

   Odor emancipating from the tongue coating is definite indicator of halitosis. Scrape the dorsum of patient's tongue and immediately smell its odor. It is also known as *Spoon test.*

3. Floss odor.

   Smelling wax dental floss after passing interproximally between all posterior teeth is also used for identifying halitosis.

4. Quantitative methods.
5. Dark field/phase contrast microscopy.

   Periodontal diseases like gingivitis and periodontitis are associated with higher incidences of motile organisms and spirochetes. Certain spirochetes have been associated with a specific malodor which can be isolated by dark field/phase contrast microscopy.

6. Gas chromatography.

   Portable machines, such as the oral chroma, are currently being introduced. This technology is specifically designed to digitally measure molecular levels of the three major 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 graphic form via computer interface. Breath odor changes in intensity throughout the day depending on many factors, therefore, multiple testing may be necessary.

7. Zinc oxide thin film semiconductor sensor.
8. Portable sulfide monitor (Helimeter).

   It is a portable sulfide meter with an electrochemical voltmetric sensor which generates signal when exposed to sulfide or mercaptan gases.

   It measures the concentration of H_2S in part per billion. However, it has drawbacks in clinical applications. For example, other common sulfides (such as mercaptan) are not recorded as easily and can be misrepresented in test results. Certain foods such as garlic and onions produce sulfur in the breath for as long as 48 hours and can result in false readings. The Helimeter is also very sensitive to alcohol, so one should avoid drinking alcohol or using alcohol-containing mouthwashes for at least 12 hours prior to being tested. This analog machine loses sensitivity over time and requires periodic recalibration to remain accurate.

9. *BANA test*:
   This test is directed to find the salivary levels of an enzyme indicating the presence of certain halitosis-related bacteria.
10. β-*galactosidase test*:
    Salivary levels of this enzyme were found to be correlated with oral malodor.
11. *Electronic nose*:
    This device is based on sensor technology that can smell and produce unique profiles for distinct odors.
12. Home tests are now available which use a chemical reaction to test for the presence of polyamines and sulfur compounds on tongue swabs.

Specific characteristic of breath odor	
Odor	*Indication*
Rotten eggs smell	Volatile sulfur compound
Sweet (dead mice)	Liver insufficiency
Rotten apples	Insulin dependent-diabetes (accumulation of ketones)
Fish	Kidney insufficiency
Foul odor	Chest infections
Fruity malodor	Oral thrush
Distinct metallic smell	Ulcers

PREVENTION

1. Maintain oral health.
   Gently cleaning the tongue surface twice daily is the most effective way to keep bad breath in control as the tongue is the principle site of accumulation.
2. Eating a healthy breakfast with rough foods helps clean the very back of the tongue.
3. Chewing gum: As dry mouth can increase bacterial buildup and cause or worsen bad breath, chewing sugarless gum can help in production of saliva and thereby help to reduce bad breath.
4. Gargling right before bedtime with an effective mouthwash.
5. Maintain water levels in the body by drinking several glasses of water a day.

MANAGEMENT OF ORAL MALODOR

Accurate labelling and interpretation of different oral malodors contribute to the diagnosis and treatment of the underlying disease. Improved oral hygiene is a key factor in the successful clinical treatment of halitosis.

1. Local chemical/antimicrobial methods
   - Mouth rinses have been used as a chemical approach but most of the commercially available rinses merely mask odors and provide little antiseptic effect as the thick layer of plaque and mucus protect the microbes. Most commercially available products contain alcohol which can dry the oral tissue and again act as a risk factor.
   - Zinc rinses available in chlorine, citrate or acetate form have been found to reduce the oral malodor by preventing bisulfide group reduction to thiols.
   - Chlorine dioxide rinses are also helpful in reducing oral malodor. Chlorine dioxide is a strong oxidizing agent.
   - Triclosan rinses are effective in most types of oral bacteria. However, combined zinc and triclosan has cumulative effect.
   - Hydrogen peroxide reduces the level of salivary thiol precursors.
   - Topical Azulene ointment with a small dose of clindamycin and Breathnol—a propriety mixture of edible flavors are also useful.
2. Oral hygiene maintenance
   - Proper brushing and cleaning of tongue after each meal and use of flossing and other interdental aids help in maintaining the oral hygiene. Scraping tongue reduces oral malodor by approximately 75 percent.
   - Elimination of local factors includes scaling and root planing, elimination of periodontal pockets, restoration of carious lesions and correction of open contacts

between teeth, and extraction of unrestorable teeth. Correction of any defect to minimize the accumulation of food debris and stagnation of saliva.
- Denture should be kept clean by brushing and putting in cleansing and disinfecting solution.
- Patient is asked to maintain the oral hygiene.

3. Salivary stimulation and/or substitutes
   - Patient with xerostomia can use sodium carboxy ethylcellulose to moisturize oral cavity.
4. Nasal mucus control methods
5. Avoidance of food, fluids and medications
   - Sugar-free meal
   - Use of fibrous food
   - Use of plenty of liquid or water
   - Less consumption of dairy products
   - Bioadhesive tablets and lozenges
   - Smoking should be discontinued.
6. Correction of anatomical abnormalities.
7. Medical management of systemic diseases.
8. If etiological factors other than local factors are suspected, laboratory tests like total blood count, urine analysis should be preferred and consultation of physician is required.
9. Systemic antibacterial methods.

CHAPTER

18 Periodontal Probe

The clinical **periodontal assessment** is a fact-gathering process designed to provide a complete picture of a patient's periodontal health status. Much of the information collected during the periodontal assessment involves the use of a periodontal probe. It is the most widely-used diagnostic tool for assessment of connective tissue destruction in periodontitis.

The word probe is derived from the Latin word "Probo", which means "to test". Its use was first described by FV Simonton of the University of California, San Francisco (Simonton 1925), when he and others referred to it as *periodontometer* (Carranza and Shklar 2003). Periodontal probes are used primarily to detect and to measure the periodontal pockets and attachment loss. Orban described probe as "Eye of the examiner beneath the gingival margin".

DEFINITION

It is a slender, blunt-ended, tapering assessment instrument used to evaluate the health status of the periodontal tissues.

The calibrated periodontal probe is a periodontal instrument that is marked in millimeter increments.

DESIGN

Probes have blunt, rod-shaped working ends that may be circular or rectangular in shape. There are many different types of periodontal probes, and each has its own manner of indicating measurements on the tip of the instrument. The working end of the probe is marked by indentations, grooves or color bands at certain millimeter intervals from the tip. There may be some color bands in between two markings for easy identification of measurements.

FUNCTIONS

- It is used to measure the comprehensive findings of periodontal examination to determine the health of the periodontal tissues.
- It is used to measure sulcus and pocket depths, to measure clinical attachment level, to determine the width of attached gingiva, to assess bleeding on probing and suppuration, and to measure the size of oral lesions.
- In addition, they are used to locate calculus, to measure gingival recession, and to locate and measure the furcation involvement.
- *Documenting measurements:*
  It is best to use anatomic references rather than "length" or "width" to document your measurements on the chart (e.g. as the anterior-posterior measurement and the superior-inferior measurement) (Fig. 18.1).

- *Determining the height of a raised lesion*:
  Place the probe tip on normal tissue alongside of the deviation. Imagine a line at the highest part of the deviation, and record this measurement as the height (Fig. 18.2).
- *Determining the depth of a sunken lesion*:
  Carefully place the probe tip in the deepest part. Imagine a line running from edge-to-edge of the deviation. The depth is the distance from this imaginary line to the base of the deviation (Fig. 18.3).

CLASSIFICATION OF PROBES

In 1992, Pihlstorm et al classified probes into three generations for consistency of use and academic purposes. In 2000, Watts extended his classification by adding fourth and fifth generation probes.

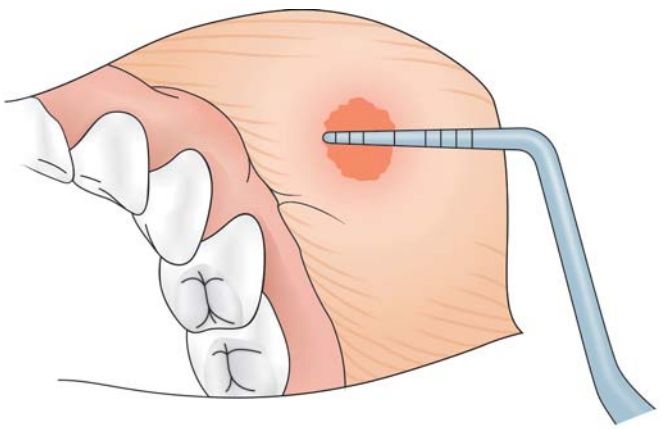


Fig. 18.1: Dimensions of lesion

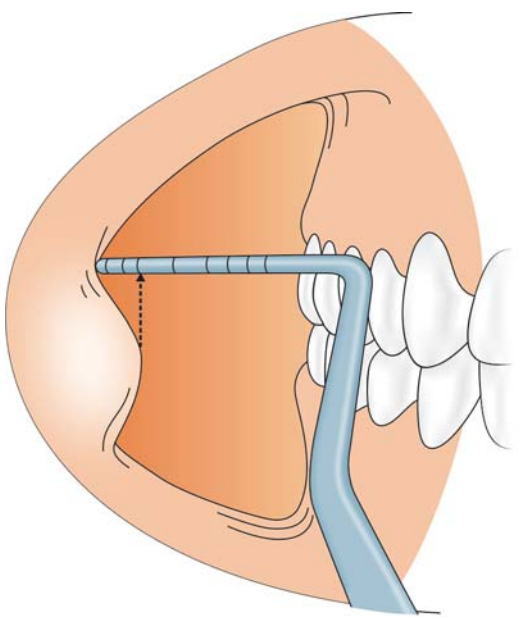


Fig. 18.2: Height of lesion

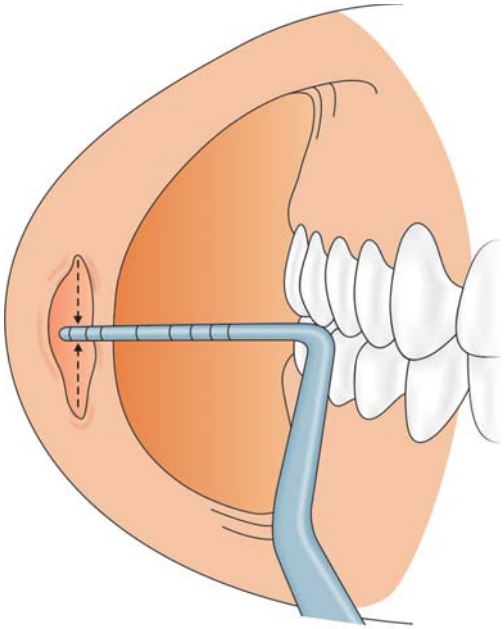


Fig. 18.3: Depth of lesion

1st Generation (Conventional) Probes

- They were invented in 1963 by periodontist Charles HM Williams. The Williams periodontal probe is the prototype or benchmark for all first generation probes.
- Conventional (manual) probe do not control the probing pressure.

Williams probe, University of Michigan O probe, Goldman Fox, Glickman, Merritt A and B	Routine clinical periodontal screening
Nabers probe	Used to measure the horizontal component of furcation involvement
CPITN probe	Used for screening and monitoring of the patients with CPITN index
UNC-15	Used in clinical research, if conventional probes are required

Advantages

- Easy availability and inexpensive
- Tactile sensitivity is preserved
- Tip is rounded to avoid tissue trauma
- Color-coded for easier and faster identification of readings
- Even in presence of subgingival calculus, probe can be inserted with little navigation by the operator.

Disadvantages

- Heavy in weight
- Probing force is not controlled, so the tip of the probe may pass beyond the base of the pocket
- In terms of reproducibility, it has limited value as precision varies from person to person
- An assistant is needed to transfer the readings to the chart
- Errors during visualizing the readings are possible.

2nd Generation (Pressure Sensitive) Probes

- They were introduced by Hunter in 1994.
- They have disposable probe head and hemispheric probe tip with diameter of 0.5 mm.
- These probes have a visual guide and a sliding scale where two indicator lines meet at a specific pressure.
- These are pressure sensitive allowing improved standardization of probing pressure.
- It has shown that with force up to 20 gm, the probe remains within junctional epithelium.
- Force of 50 gm helps in diagnosis of osseous defect.

For example, True pressure sensitive probe, Yeaple probe

Advantages

- Standardization of probing forces
- Comfortable to patient
- Constant pressure.

Disadvantages

- Lacks tactile sensitivity
- Probe tip may pass beyond the junctional epithelium in inflamed tissues
- No computer storage of data
- Readings have to be performed manually, and an assistant is needed to record the same on the patient chart
- It is used with a fixed amount of pressure regardless of the site, presence/absence of inflammation, which may lead to inaccurate measurement and discomfort to patient.

3rd Generation (Automated, Computerized) Probes

They were invented by Gibbs in 1988 in order to help minimize the errors of second generation probes, such as reading errors, calculation errors.

- Probe is attached with the handpiece. It has got a digital read out, foot switch and computer interface, and a computer.
- Probe tip is of 0.45 mm diameter. The probing force is automatically standardized at 15 gram.

Mechanism of Action

- The main mechanism of action is the detection of the cementoenamel junction.
- The ball tip moves over the root surface at a controlled speed and preset pressure. Abrupt changes in the acceleration of the probe movement indicate the reach of cementoenamel junction, and it stops at the base of the pocket.
- The position and acceleration time are analyzed to determine attachment level and pocket depth.
- It reciprocates through sleeve, and measurements are made electronically and transferred automatically to computer on pressing the foot switch.
- The software stores date of attachment loss, recession, pocket depth, furcation depth. For example, Foster miller probe (detects distance from cementoenamel junction so helps in assessing attachment loss) is the prototype of third-generation probes. Florida probe, Toronto probe, InterProbe.

Advantages

- Standardization of probing forces.
- High degree of accuracy and reproducibility of measurement as it detects the cementoenamel junction, which is a better landmark than gingival margin because the latter may change depending on inflammation or recession.
- Errors in reading the probe and transferring the data are eliminated.
- Printout of the data from the computer can be used for patient education.

Disadvantages

- The main disadvantage is that it considers the root roughness or root surface irregularities as the cementoenamel junction.
- Tactile sensitivity is decreased.
- Probe may pass beyond the junctional epithelium in inflamed sites, overestimating the pocket depth.
- After the inflammation has resolved, probe may not penetrate beyond the long junctional epithelium, leading to underestimation of the pocket depth.
- Use of fixed force setting throughout the mouth regardless of the site or inflammatory status may generate inaccurate measurement or patient discomfort.

4th Generation Probes

They are still under investigation. These probes are aimed at recording sequential probe positions along the gingival sulcus.

Advantages

- Three dimensional pocket configuration can be assessed
- Sequential probe positions are measured.

Disadvantages

- Invasive probe.

5th Generation Probes

- They were devised by Hinders and Companion at the NASA Langley Research Center.
- They are aimed to identify the attachment level without penetrating it.
- The probe has an ultrasonic beam projection. They are close enough in size to the width of the periodontal ligament space to give the optimal coupling, and small enough to inspect the area between the teeth, while still delivering sufficient signal strength and depth of penetration to image the periodontal ligament space.
- They are suitable for universities and research institutes.
  For example, Ultrasonographic (US) probe.

Advantages

- There is no possibility of imprecise readings due to the passing of the probe beyond the junctional epithelium as it utilizes the ultrasonic waves to detect, image and map the upper boundary of periodontal ligament.
- A noninvasive probe that provides painless probing to the patient.
- Provides information regarding the condition of gingival tissues.
- Computer storage of data and printout or visuals can be used for patient education.

Disadvantages

- Expensive
- Operator needs training to interpret the image provided by the computer
- Requires learning curve.

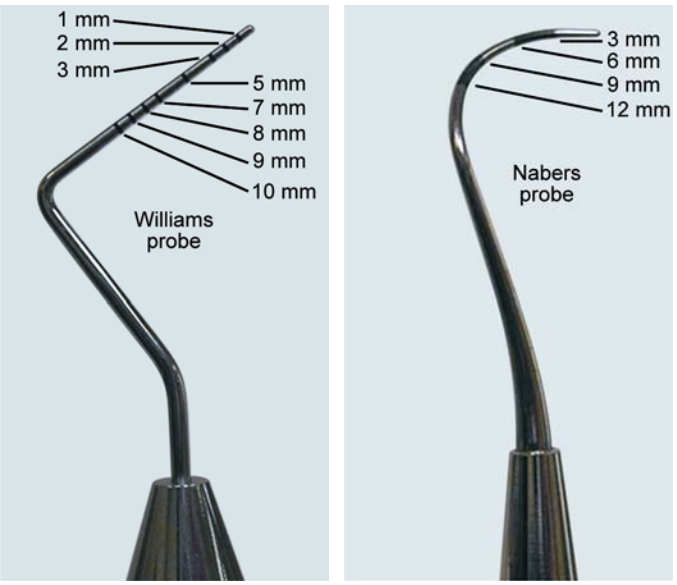


Fig. 18.4: Markings of Williams probe and Nabers probe

Periodontal probes	*Design*
Marquis color-coded probe	Calibrations are in 3 mm sections
University of North Carolina (UNC-15) color-coded probe	– Length of probe is 15 mm – Each mm from 1 to 15 is marked – Black marking at 5 mm, 10 mm, 15 mm
Michigan "O" color-coded probe	– Markings at 3 mm, 6 mm, 8 mm – A modification of the probe with markings of Williams probe is also available – Probe shape is same as that of Williams probe but ends are rounded
Nabers probe (Fig. 18.4)	– Markings are at 3 mm, 6 mm, 9 mm
Williams probe (Fig. 18.4)	– 13 mm in length, tip diameter is 1 mm – Probe tips and handles are enclosed at 130° – Markings at 1 mm, 2 mm, 3 mm, 5 mm, 7 mm, 8 mm, 9 mm, 10 mm (All from 1 to 10 except one number before and one after 5) – 4 mm and 6 mm markings are absent to improve visibility and avoid confusion in reading
WHO probe (CPITN probe)	– 0.5 mm ball at tip and millimeter markings at 3.5 mm, 5.5 mm, 8.5 mm, 11.5 mm – Color coding (black band) from 3.5 mm to 5.5 mm
Florida probe	0.45 mm tip diameter, 0.97 mm sleeve diameter
Goldman fox	Markings are same as Williams probe It is flat while Williams is round, tapered

CHAPTER

19 Probing

DEFINITION

"Probing is act of walking the tip of probe along the junctional epithelium within the sulcus or pocket for the purpose of assessing the health status of periodontal tissue."

Force	*Effect*
0.75 N (25 gram)	Well-tolerated by tissue and considered as an optimal force.
30 gram	Required to penetrate up to junctional epithelium.
50 gram	Required to detect bony defects.

Biological depth	It is the distance between gingival margin and the base of the pocket (coronal end of junctional epithelium). It is a histological evaluation.
Probing depth	It is the distance between gingival margin and the apical end of the periodontal probe penetration. It is a clinical evaluation.

In healthy gingiva, probe tip penetrates to most coronal intact junctional epithelium/ partly within the junctional epithelium/ sometimes up to coronal intact fibers of connective tissue attached to the tooth surface.

In inflamed tissues, the probe passes through the junctional epithelium and to the connective tissue. This means, true histologic depth of the pocket is not being measured. Consequently, the term *probing depth* often replaces the more traditional term *pocket depth* (Fig. 19.1). Pocket depth is the distance between the base of the pocket and the gingival margin. It may change from time to time.

PROBING TECHNIQUE

- Insert the probe parallel to the vertical axis of the tooth surface (both mesiodistally and labiolingually) and then walk circumferentially around each surface of each tooth to detect area of deepest penetration (Fig. 19.2).
- Move the probe up and down in short bobbling strokes and go ahead in 1mm of increments.
- The probe is not completely removed from the sulcus with each upward stroke as repeated act of removal and insertion of probe can traumatize the marginal gingival.
- The probing forces varying from *20-25 gram* have been suggested appropriate. Probe should be activated with light pressure by gentle motion of wrist or finger. Several investigators have found that *0.75 N* of force is well-tolerated and accurate. This probing force can be clinically determined by applying the probe tip to your fingernail bed, where it will initiate blanching (Greenstein 1990, 2005).

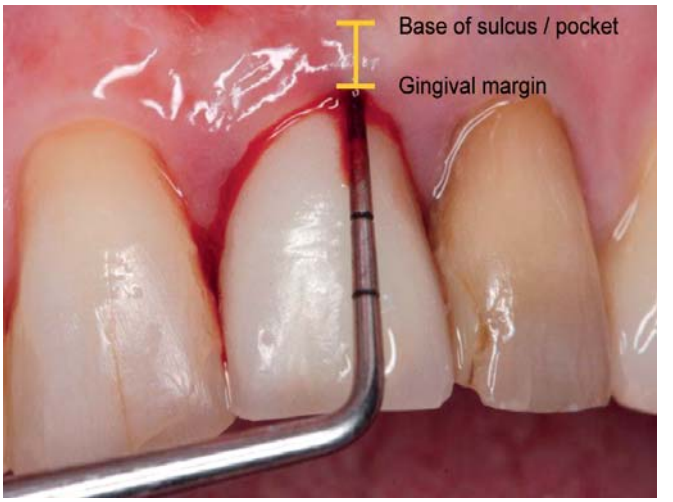


Fig. 19.1: Probing depth

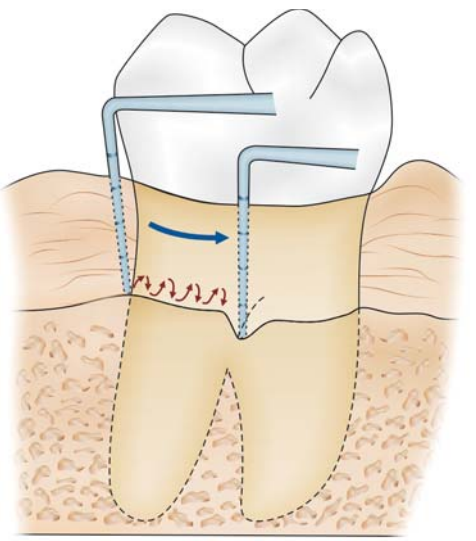


Fig. 19.2: Probing technique

- Probe penetration can vary depending on the force of introduction, the shape and the size of the probe tip, and the degree of tissue inflammation. Improper probing can injure the junctional epithelium.
- The side of the probe tip (1-2 mm) should be kept in contact with the tooth surface. Probe should be kept as parallel as possible to the tooth surface.
- Access the area beneath the contact area by tilting the probe to extend the tip beneath the contact area, and gently press down to touch the junctional epithelium. Probe should be placed from both facial and lingual surfaces to detect the deepest point beneath the contact area (Fig. 19.3).
- Record the six measurements for each tooth and finally the deepest reading is recorded.
- In healthy gingiva, penetration of the probe gives more resistance while in presence of inflammation, the probe tip may go beyond the junctional epithelium.

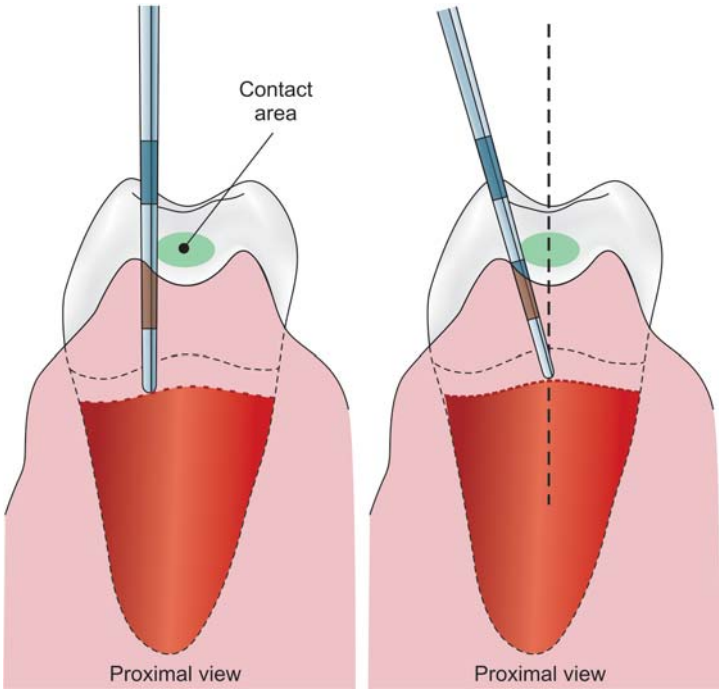


Fig. 19.3: Probing below contact area

INTERPRETATION

The depth of a sulcus or the pocket is determined by measuring the distance from gingival margin to the base of the gingival sulcus with a calibrated periodontal probe (Fig. 19.1).

Healthy gingiva	2/3 of the length of junctional epithelium
Gingivitis	Probe stops 0.1 mm short of apical end of junctional epithelium
Periodontitis	Probe tip consistently passes beyond the most apical cells of junctional epithelium. Probe tip passes 0.3 mm in the connective tissue apical to the junctional epithelium.

Normal Sulcus

In disease-free sulcus, junctional epithelium forms base of the sulcus by attaching to the enamel of the crown near the cementoenamel junction. The probing depth of normal gingival sulcus is 2-3 mm as measured with periodontal probe.

Periodontal Pocket

Periodontal pocket is pathologic deepening of the gingival sulcus due to apical migration of the junctional epithelium. The junctional epithelium forming the base of the pocket is somewhat more apical than normal position at cementoenamel junction. It is due to destruction of periodontal ligament and alveolar bone. The normal sulcus deepens to 5-6 mm usually.

Actual pocket depth cannot be measured as the probe always penetrates the tissue variably in periodontitis. Variables in measuring pocket depth are:

- Accurate probe gradations
- Diameter of probe, angle and force
- Degree of inflammation.

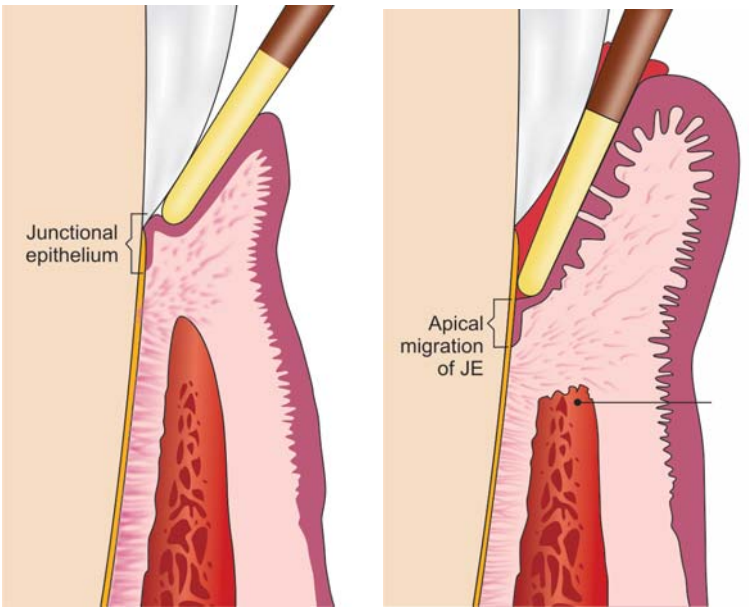


Fig. 19.4: Probing of healthy sulcus and periodontal pocket

Probing depths are not reliable indicators of the extent of bone support because these measurements are made from the gingival margin. The position of gingival margin changes with tissue swelling, overgrowth and recession.

LIMITATIONS

Periodontal probing presents many problems in terms of sensitivity and reproducibility of the measurements.

- Reading error may result from interference by the calculus from tooth and root surfaces, presence of overhanging restorations or abnormal contour of crown.
- Operator's error like incorrect angulations of the probe, misreading of the probe, recording data imprecisely, and miscalculating the attachment loss.
- Clinical pocket depth readings normally do not coincide with the histologic pocket depth because the probe normally penetrates the coronal level of the junctional epithelium, if inserted properly, and the precise location of the probe tip varies depending on the degree of inflammation of underlying connective tissues.

FACTORS AFFECTING PROBING

Various factors, such as probe tip size, probing technique, angle of insertion of probe, probing pressure, precision of probe calibration, and degree of inflammation in underlying periodontal tissues, affect the sensitivity and reproducibility of measurements (Fig. 19.4).

Inflamed tissues offer less resistance to probe penetration, whereas after the subgingival instrumentation, healed gingiva offers increased resistance to probing. Likely, increased fibrosis in smokers offers more resistance to probe tip penetration.

PROBING AT VARIOUS TIMES

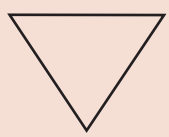
1. Pretreatment probing in moderate or advanced cases may not be an accurate representation of periodontal condition. Interference by abundant calculus and presence

of heavy inflammation hampers the accurate assessment. This pretreatment probing with other clinical and radiographical findings aid to determine whether the tooth can be saved or not.

2. After scaling and adequate plaque control by patient, the major inflammatory changes disappear, and more accurate probing of the pockets reveals the level of attachment and degree of root and furcation involvement. The data from the second time probing provides valuable information for treatment planning.
3. Later in periodontal treatment, probing is done to determine changes in pocket depth and to ascertain healing progress after various procedure.

CHAPTER

20 Scalers and Curettes

Differences between scaler and curette		
	Scaler	*Curette*
Use	Supragingival scaling (primary use) Subgingival scaling (secondary use)	Subgingival scaling, root planing, curettage (primary use)
Blade	Thicker	Finer
Working tip	Converge to pointed tip (Fig. 20.2)	Round toe (Fig. 20.4)
Working edge	2 working edge (Fig. 20.1)	Universal – 2 Area specific – 1 (Outer, convex) (Fig. 20.3)
Design	Heavy	Fine, delicate, vital
Insertion	Only 1 mm subgingivally	More subgingivally
Adaptation	Adequate (Doesn't adapt to root surface properly)	Good (Possible to adapt to deeper areas)
Cross section	Triangular 	Semicircular or spoon shaped 
Stroke	Scaling stroke – Short, powerful, pull	Root planning stroke – Moderate to light, pull
Curvature	Curved in one plane	Curved in two plane
Types	U 15/30, Ball and Indiana Jaquette sickle # 1,2 and 3 Curved 204 sickle Nevi 2 posterior sickle scaler	Two basic types; Universal – Barnhart – Columbia Area specific – Gracey – Langer

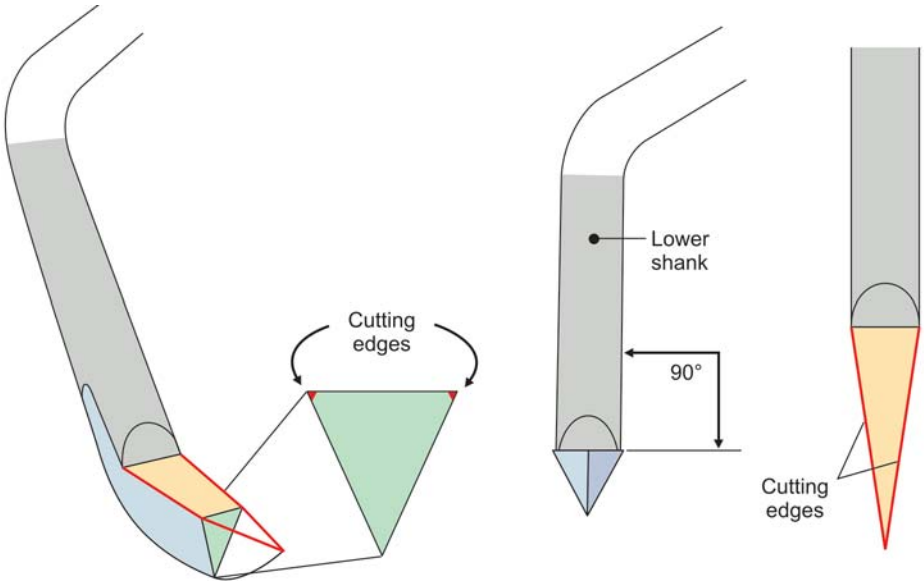


Fig. 20.1: Features of scaler

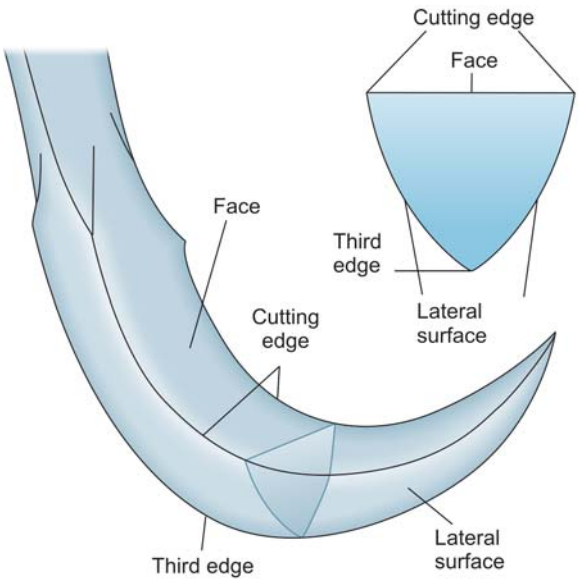


Fig. 20.2: Design of scaler

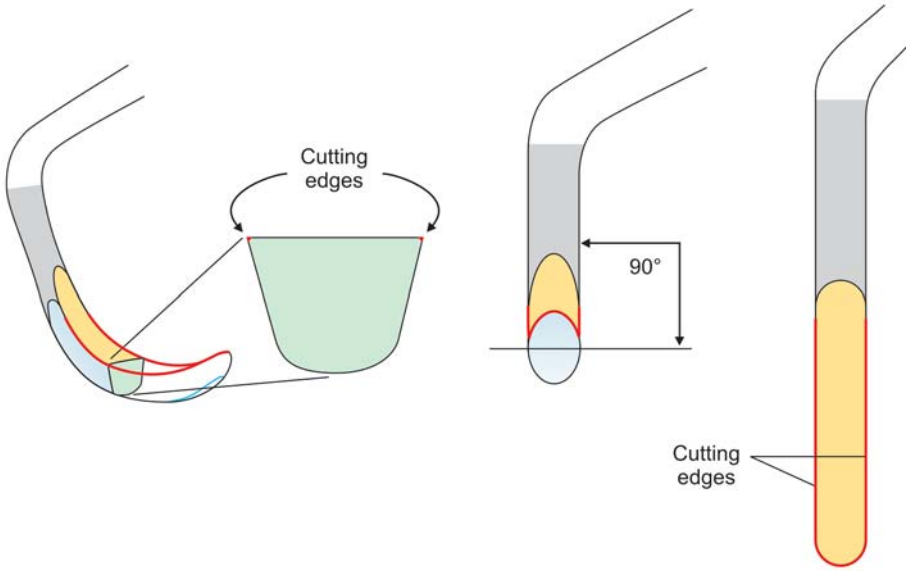


Fig. 20.3: Features of curette

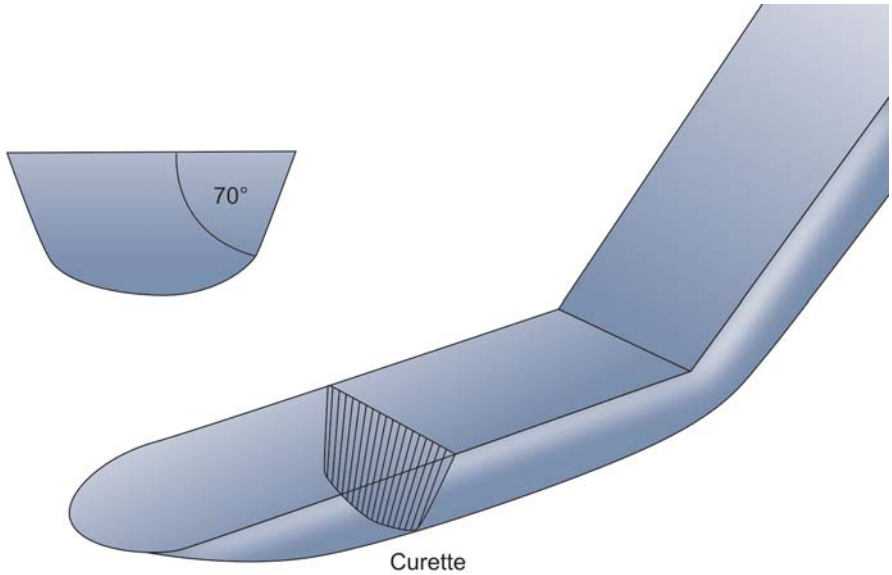
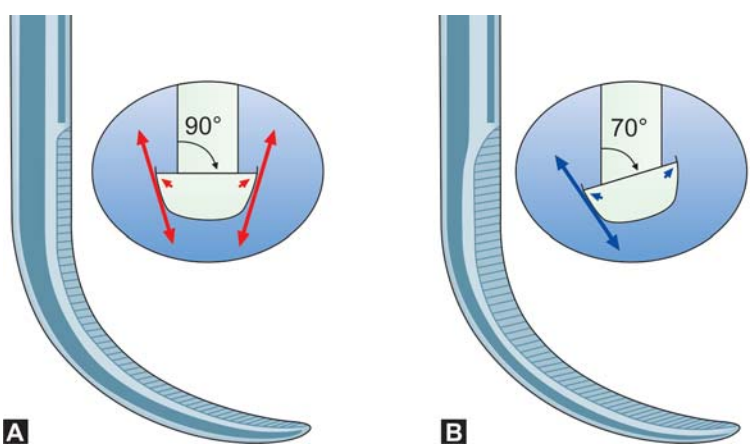


Fig. 20.4: Design of curette

DIFFERENCES OF GRACEY CURETTE AND UNIVERSAL CURETTE (FIG. 20.5A AND B)

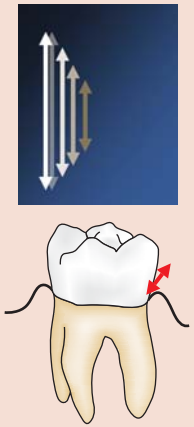
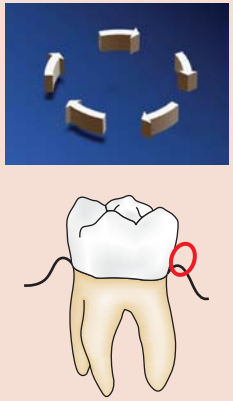
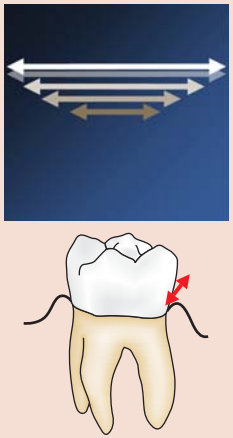
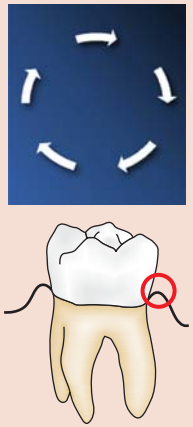
	Gracey curette (Fig. 20.5B)	*Universal curette*
Area of operation	Area and surface specific Different designs for different areas	Universal—Only one curette is used for all teeth by changing position of blade, fulcrum, adaptation and finger rest
Cutting edge	One cutting edge is used, i.e. work with outer edge only	Both cutting edges are used, i.e. work with either inner or outer cutting edges
Blade	Curved in two planes. Blade curves up and to the sides	Curved in one plane. Only upwards and not to the side
Blade angle	Offset blade: face of blade is beveled at 60° to 70° from the lower shank	Not offset blade: face of blade is beveled at 90° to lower shank
Working end	The working end is automatically at the correct angulation when the lower shank is parallel to the tooth surface	The lower shank must be tilted slightly towards the tooth surface to establish correct angulation with tooth

Gracey curettes	
No. 1-2, 3-4	Anterior teeth
No. 5-6	Anterior and premolar teeth
No. 7-8, 9-10	Posterior teeth: facial and lingual
No. 11-12	Posterior teeth: mesial surface
No. 13-14	Posterior teeth: distal surface
No. 15-16	Blade of 11-12 and shank of 13-14
No. 17-18	Blade of 13-14 and shank extended by 3 mm



Figs 20.5A and B: (A) Universal curette, (B) Gracey cure tte

COMPARISON OF SCALERS

	Hand scaler	*Ultrasonic scaler*		*Sonic scaler*
		Magneto strictive	*Piezoelectric*	
Principle	–	The conversation of 60 HZ, 120 V current in an ultrasonic unit, which continually alters the shape of a (magnetostrictive) bimetallic stack of nickel-cobalt alloys, into an elliptical motion.	When electric energy is applied across the piezoelectric substance, measurable changes in form of expansion and contraction of the crystal produces linear motion of tip.	Compressed air over an eccentric rod drives the rod to vibrate.
Tip *- Action* *- Adaptibility*	Vertical Good 	Elliptical Fair 	Linear (scraping) Fair 	Orbital (rotary) Fair 
- Application	Removal of deposits is directly influenced by the angle of tip application onto the tooth surface as only two tip surfaces are active at once.			Removal of deposits is independent from tip application the tooth surface as all sides of tip are active.

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	Hand scaler	Ultrasonic scaler		Sonic scaler
		Magnetostrictive	Piezoelectric	
Oscillation Amplitude	–	50-70 μm approximately Higher pressure damps down the oscillation		100-250 μm approximately Oscillations are independent of contact pressure
Frequency	–	20,000 to 40,000 cycles/sec (Hz)	24,000 to 45,000 cycles/sec (Hz)	2,000 to 6,500 cycles/sec (Hz)
Force	More (around 2N)	Less force is required in comparison of hand scalers (It depends on tip frequency)		
Efficiency	Good, if proper instrumentation technique is used	Excellent (Sulcus lavage due to constant water irrigation have additional effect in removing bio-film)		
Time	Time consuming	Requires less time		
Maintenance	Medium	Relatively high due to complex mechanism		Medium
Cost	Less	High		Medium
Aerosol formation	No	Aerosol formation is same for all		
Health hazards	Least	High due to contagious aerosol formation		
Noise	Least		Medium	High
Heat	No	Metal stack in magnetostrictive scaler	Quartz crystal is piezoelectric scaler generate less heat compared to magnetostrictive scalers	Lower than piezoelectric
Resorption Damage	Less	More (because of high frequency)		Medium
Tissue abrasion	There is no conclusive data. It depends on various factors such as applied force, angle of tip placement, frequency of tip, etc.			

Contd....

	Hand scater	Ultrasonic scaler		Sonic scaler
		Magnetostrictive	Piezoelectric	
Comfort	During removal of tenacious deposits, sonic and ultrasonic instruments produce less tissue trauma, if properly manipulated and therefore less postoperative discomfort to the patient than hand instruments.			
Asepsis	Good	Good but requires meticulous care to remove the debris from the hand piece and to sterilize the core areas		
Contraindication	–	– Patient with infectious and communicable diseases, as it may spread with aerosol – Patient with respiratory and pulmonary diseases or those having difficulty in breathing (e.g. Bronchitis, Asthma). – Patients with metallic (older) cardiac pacemakers as ultrasonic waves may interfere with its proper functioning. Sonic scalers do not produce the same effect. – Patients with titanium implants can't be treated with routine tips (Special titanium tips,Teflon-coated or plastic tips can be used) – Patients with compromised gag reflex – Sometimes pediatric patients can be the relative contraindication as primary teeth have large pulp chambers and the growing tissues are more susceptible to damage by heat generated by instrument.		

AREAS OF INSTRUMENTATION OF GRACEY CURETTES (FIGs 20.6 TO 20.8)

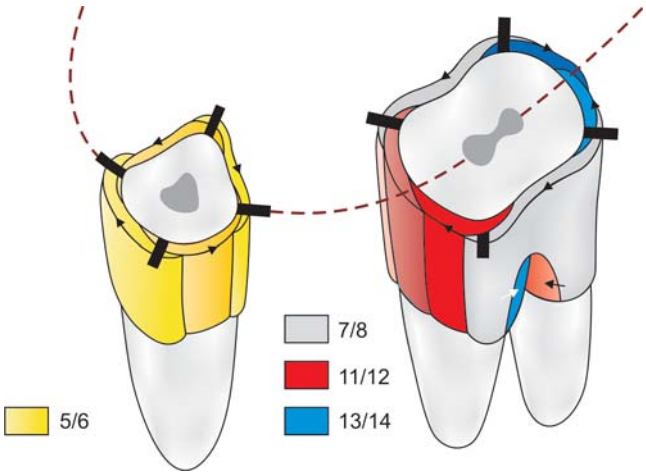


Fig. 20.6: Areas of instrumentation of particular curette

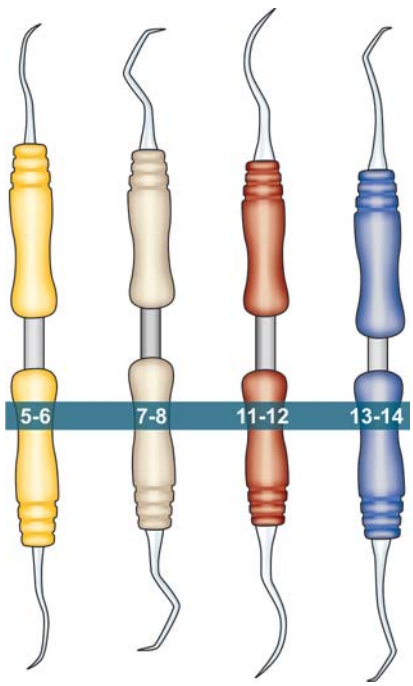


Fig. 20.7: Gracey curettes

Extended shank curettes

The shank is extended by 3 mm than the standard gracey curette which allows the extension into deeper periodontal pockets. They are available in all standard gracey numbers except 9-10.

For example, After five curettes

Miniblade curettes

These are modified after five curettes with the blade length half that of conventional curettes. The shorter blade allows easier insertion and adaption in deep, narrow pockets and furcation.

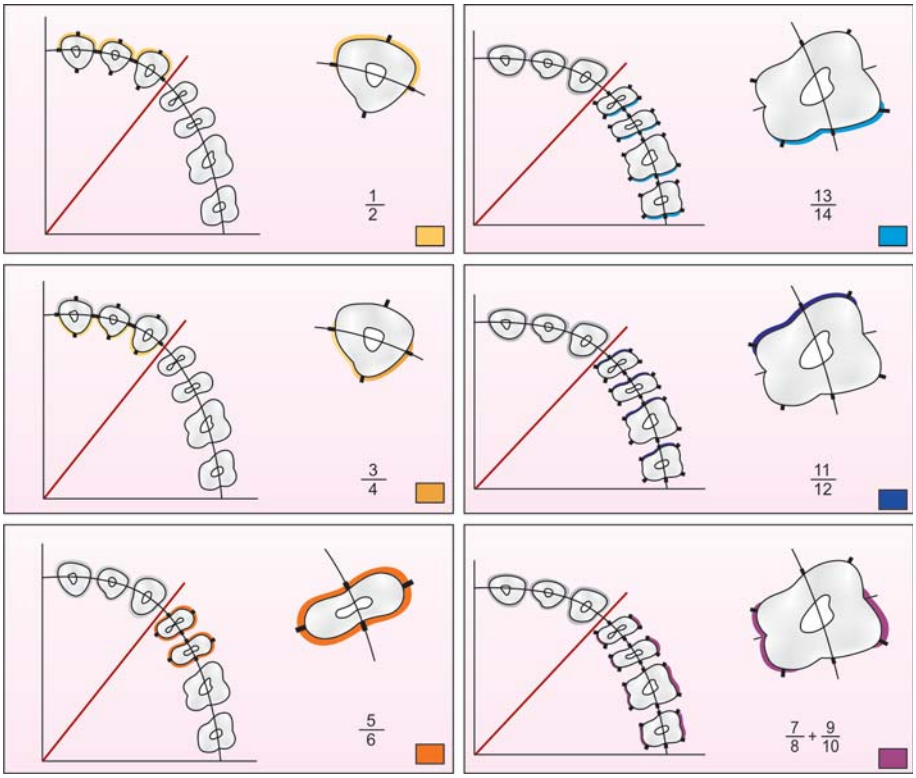


Fig. 20.8: Operational areas of root surfaces for Gracey curettes

For example, Mini five curettes

Angulations in instrumentation	
Angulation for blade insertion	0°
Angulation for scaling and root planing	45-90°
Angulation for curettage	>90°

Angles in instrument	
Blade angle of hoe	90°
Angle of blade with shank in universal curette	90°
Angle of blade with shank in gracey curette	60-70°
Angle between face and lateral surface of the blade	70-80°
Angle for sharpening	100-110°

CHAPTER

21 Gingival Curettage

DEFINITION

"Gingival curettage is the surgical procedure of scraping of gingival wall of periodontal pocket to separate diseased soft tissue (Fig. 21.1)."

Gingival curettage is an older type of periodontal surgical procedure that involves an attempt to scrape away the lining of the periodontal pocket. It is a surgical technique designed to remove, by debridement, the inner aspect of the diseased gingival wall, including the ulcerated and hyperplastic gingival epithelium and the contiguous zone of damaged connective tissue downward and outward to the firm and intact aspect of the gingival corium, thus converting diseased tissue to a surgical wound (Kon et al. 1969; Pollack 1984). Removal of this tissue was assumed to enhance pocket reduction beyond the results achieved by scaling and root planing alone, providing faster healing and the formation of new connective tissue attachment to the root surface. This rationale has been seriously questioned for many years and the procedure is no longer considered as standard treatment.

Gingival curettage is a surgical procedure which consists of removal of inflamed soft tissue lateral wall of pocket while subgingival curettage refers to a surgical procedure performed apical to the epithelial attachment severing connective tissue attachment down to osseous defects to remove the diseased tissues.

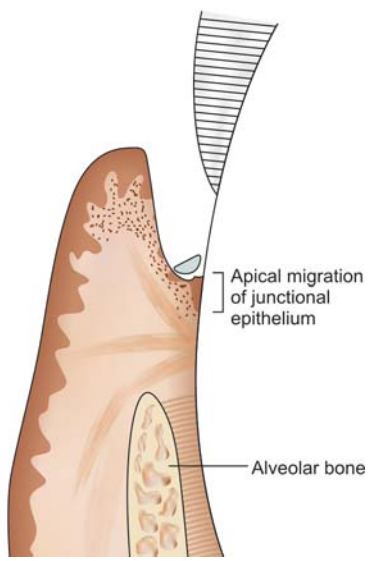


Fig. 21.1: Gingival curettage

Closed crevicular curettage refers to performing soft tissue curettage rather than a flap procedure in treating periodontal pockets. This approach has several advantages. It may be used (1) for initial preparation to obtain predictable soft tissue shrinkage, (2) when surgery is contraindicated for health or medical reasons, (3) when emotional patients cannot tolerate definitive surgery, (4) when esthetic concerns are a consideration (Pollack 1984).

RATIONALE

The goal of therapy is to remove chronically inflamed granulation tissue that forms the lateral wall of periodontal pocket.

Along with granulation tissues, flakes of calculus and bacterial colonies are also removed from root surface leading to shrinkage of soft tissue wall of pocket. By removal of epithelial lining of the pocket and the underlying junctional epithelium by curettage gives chances of new attachment. However, opinions differ regarding the extent of removal of pocket lining and junctional epithelium.

INDICATIONS

1. It is done as nondefinitive surgery to reduce inflammation of lateral wall of pocket before aggressive planned periodontal surgery.
2. It is indicated in patient who cannot be subjected to other surgical pocket elimination procedure due to age or systemic condition as the goal of pocket elimination is compromised and prognosis is marred.
3. It may be performed during recall visit as a part of maintenance therapy in cases treated with pocket elimination surgery earlier.
4. Curettage eliminates suprabony pocket that are located in accessible areas and have an inflammatory edematous pocket wall that shrinks to the sulcus depth after treatment.
5. New attachment attempts in moderately deep infrabony pocket located in accessible areas where a type of closed surgery is advisable.

CONTRAINDICATIONS

1. Furcation involvement.
2. Presence of bony craters, bony deformities.
3. The case of very deep infrabony pocket where instrumentation is not possible.
4. Pocket with fibrotic gingival wall.

LIMITATIONS

1. One should be careful while curetting thin friable gingiva as there is danger of perforating or tearing such tissue.
2. Root planing mobilizes fragments of calculus and cementum that may be forced into tissue during gingival curettage, if the procedures are done simultaneously.
3. Curettage does not eliminate cause of infection, for example, bacteria and plaque deposits, so it should always be preceded by scaling and root planing.
4. Lack of predictability of removing the pocket epithelium, epithelial attachment and subjacent altered connective tissue.
5. It is technically demanding and considered extremely difficult procedure to master.
6. It is often a “blind procedure” rather grossly inexact and depends solely on tactile sensitivity.

PROCEDURES

- It is usually done under local anesthesia after scaling and root planing with the help of curettes.
- As curettage does not eliminate the causes of inflammation, it should always be preceded by scaling and root planning.
- Curettage can be done with help of curettes like Universal Columbia Curettes / a Specified Gracey Curette.
- The instrument is inserted in such a way as to engage the inner lining of pocket wall and the instrument is carried along with the soft tissue.
- Usually, the horizontal stroke is applied and at the same time the pocket wall may be supported by a gentle finger pressure externally.
- The curette is placed under the cut edge of junctional epithelium to undermine it.
- In subgingival curettage, the tissues attached between bottom of pocket and alveolar crest are also removed with a scooping movement of curette.
- The area is flushed to remove the flakes of calculus from root surface. By flushing the pocket wall, debris and tags of tissue come out and periodontal dressing is applied.
- Suturing the papillae and application of periodontal pack may be indicated.

OTHER TECHNIQUES

ENAP (Excisional New Attachment Procedure)

Subgingival curettage is performed with knife (15/11 no. blade) is known as ENAP (Excisional new attachment procedure). It is an approach to reestablish periodontal attachment and reducing pocket depth. It is tempted by surgically removing sulcular and junctional epithelium, the transseptal and gingival crest fibers, root calculus, through an internal beveled incision without detachment of mucogingival complex. It was first presented and evaluated experimentally and clinically in 1976 (Yuka et al 1976; Yuka 1976).

- After anesthetizing the area, an internal bevel incision is placed from free gingival margin to a point below the bottom of the pocket on all the sides of tooth (facial, lingual and interproximal).
- With preservation of as much interproximal tissues as possible, the inner portion of soft tissue wall of the pocket is excised.
- Curette is used to remove the excised tissues and all exposed cementum is thoroughly root planed. Connective tissue fibers are preserved on the root surface for better healing.
- Wound edges are approximated and wound is sutured. Bone contouring may be performed, if required.

Ultrasonic Curettage

Ultrasonic devices deliver the vibrations that disrupt tissue continuity, separate collagen bundles and lift off the epithelium. Morse scaler-shaped and rod-shaped ultrasonic instruments are used for this purpose.

Some studies found ultrasonic devices equally effective as manual instruments and at the same time resulted in less inflammation and less removal of underlying connective tissue.

Chemical Curettage

Caustic drugs such as sodium hypochlorite, sodium sulfide and phenol have been used for chemical curettage. However, the effect of these agents is not limited to the epithelium and depth of penetration cannot be controlled. Thus, inability to control the extent of tissue

destruction and increase in amount of tissues to be removed by enzymes and phagocytes has proved it ineffective.

HEALING AFTER CURETTAGE

- Immediately after curettage, blood clot fills the pocket area which is partly/totally devoid of epithelial lining.
- Hemorrhage is present in tissue with dilated capillaries and abundant polymorphonuclear leukocytes which is gradually followed by rapid proliferation of granulation tissue.
- Decrease in number of blood vessels is observed as healing progresses.
- Epithelialization of sulcus generally required 2-7 days.
- Restoration of junctional epithelium occurs within 5-7 days. Immature collagen fibers reappear and establish within 3 weeks.
- Healing results in long, thin junctional epithelium and no new thin connective tissue attachment.
- Tissue usually shrinks and takes its position apical to normal.

CHAPTER

22 Infrabony Pocket

Periodontal pocket is defined as "Pathological deepening of gingival sulcus due to apical migration of junctional epithelium".

Periodontitis starts as an inflammation of the gingiva in response to bacterial change. The transformation of gingival sulcus into a periodontal pocket creates an area where plaque removal becomes impossible. The pathogenesis of periodontal destruction involves a complex interplay between bacterial pathogens and the host tissues. Inflammatory and immune reactions extending deeper into the connective tissue beyond the base of the pocket may also include alveolar bone loss in this destructive process. Periodontal pocket shows following signs and symptoms.

SIGNS

- Change in morphology
  Bluish red, thickened, rolled out marginal gingiva and blunted interdental papilla.
- Change in color
  A bluish red, vertical zone extending from gingival margin to alveolar mucosa; change in color of gingiva varies according to severity of inflammatory involvement.
- Smooth, shiny gingiva.
- Puffy, flaccid and edematous gingiva with loss of stippling and pitting on pressure.
- Discontinuity of interdental papilla, from both labial and lingual aspect.
- Bleeding by gently probing soft tissue wall of pocket.
- Suppuration may be present in many cases and pus may be expressed by applying digital pressure.
- Loose, extruded tooth and tooth mobility may be present.
- Diastema formation.
- When explored with probe, inner aspect of pocket is generally painful.

SYMPTOMS

Periodontal pockets are generally painless but may give rise to following symptoms:

- Gnawing type of pain which may radiate to deeper periodontal structures. Severity of pain varies according to severity of periodontal destruction.
- Bleeding from gingival tissue.
- Pus discharge even on digital pressure on attached gingiva.
- Food lodgment in localized region.
- Urge to dig with a pointed instrument and resultant bleeding gives relief.
- Feeling of itching in the gums.
- Foul taste in localized areas.
- Sensitivity with hot and cold, toothache in absence of caries.

CLASSIFICATIONS OF POCKETS

On the basis of position of epithelial attachment on tooth surface as well as position of marginal gingiva, pockets can be classified as follows:

- Gingival pocket or pseudo pocket
  It occurs due to gingival enlargement without destruction of underlying periodontal tissues. Coronal proliferation of marginal or papillary gingiva without any change in epithelial attachment gives rise to deepening of gingival sulcus as a result of an increase in size of gingiva, i.e. in case of chronic gingivitis.
- Periodontal pocket or true pocket
  Periodontal pocket shows the change in position of epithelial attachment and destruction of supporting periodontal tissues.

CLASSIFICATIONS OF PERIODONTAL POCKETS

Periodontal pockets are further classified as follows:

Depending on the Level of Bottom of Pocket

- Suprabony, supracrestal or supra-alveolar
  The bottom of pocket and the junctional epithelium are coronal to underlying alveolar bone. Deepening of gingival sulcus occurs with destruction of adjacent gingival fibers, periodontal ligament fibers, and crestal alveolar bone and it is associated with apical migration of the junctional epithelium. They are associated with horizontal bone loss.
- Intrabony, infrabony, subcrestal or intra alveolar
  Deepening of gingival sulcus to a level at which the bottom of the pocket and the junctional epithelium are apical to the crest of alveolar bone. They are associated with the vertical bone loss.

Depending on Nature of Soft Tissue Wall

- Edematous pocket
- Fibrotic pocket

Depending on Disease Activity

- Active
- Passive

According to Number of Tooth Surfaces Involved

- Simple (involving one tooth surface)
- Compound (involving two or more surfaces)
- Complex or spiral pocket (periodontal pocket on one side may travel spirally, mesially or distally involving one or more additional surfaces). They are most commonly seen in furcation areas.

CLASSIFICATIONS OF INFRABONY DEFECTS

According to Number of Walls (Goldman and Cohen, 1958)

- One walled (Hemiseptum)
- Two walled
- Three walled (Infrabony)
- Combined osseous defect (The number of walls in the apical portion of the defect are greater than that in its occlusal portion)

According to the Depth and Width of the Underlying Osseous Defect

Type I – shallow narrow
Type II – shallow wide
Type III – deep narrow
Type IV – deep wide

Goldman and Cohen Classification of Intrabony Defects

According to by the number of walls around the lesion (JP, 58):

1. *Three osseous walls*
   A three-walled intrabony defect is surrounded by three bone walls, with the root surface as the fourth wall (Fig. 22.1). The walls may be at different level coronally. It occurs most frequently in the interdental area. They may be called intrabony defect and they are frequently associated with food impaction. They may also be seen on facial and lingual surfaces having enough bone to support the formation of walls, e.g. defects on facial and lingual of mandibular posterior teeth and palatal of maxillary teeth. These 3-walled defects are sometimes called *wells.*
   a. Proximal, buccal, and lingual walls
   b. Buccal, mesial, and distal
   c. Lingual, mesial, and distal.
2. *Two osseous walls*
   A two-walled intrabony defect (crater) is the most common osseous defect in the interdental area (Fig. 22.1). Usually buccal and lingual walls are present and bone loss occurs on the proximal surfaces of adjacent teeth. Two-walled defects with either facial or lingual wall and a proximal wall are less common.
   a. Buccal and lingual walls (crater)
   b. Buccal and proximal walls
   c. Lingual and proximal walls.
3. *One osseous wall*
   One-walled intrabony defect usually exists in the interdental area. However, most intrabony defects are of mixed types; e.g. the entrance has one wall or two walls but the bottom has three walls (Fig. 22.1).
   a. Proximal wall (hemiseptal)
   b. Buccal wall
   c. Lingual wall.
   If the remaining bone wall is on the proximal surface, it is called a *hemiseptal defect* and that on facial or lingual surface is called as *a ramp*. Shallow one-walled defects may be managed by osseous surgery.
4. *Combined osseous defect*
   In combined osseous defect, numbers of walls in the apical portion of the defect are greater than those in its occlusal portion (Fig. 22.2). Such defects are more complex apically than coronally. The depth, width, topography, number of remaining osseous walls, and the configuration of the adjacent root surfaces are all important in determining the therapeutic approach.
   a. 3 walls + 2 walls
   b. 3 walls + 2 walls + 1 wall
   c. 3 walls + 1 wall
   d. 2 walls + 1 wall

In the original classification by Cohen defects are also classified as follows:

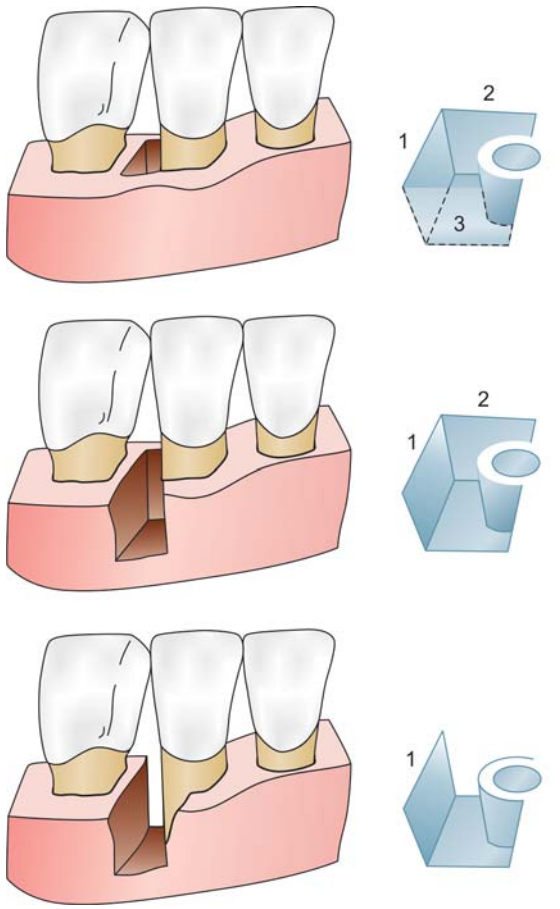


Fig. 22.2: 1-wall defect, 2-wall defect, 1-wall defect

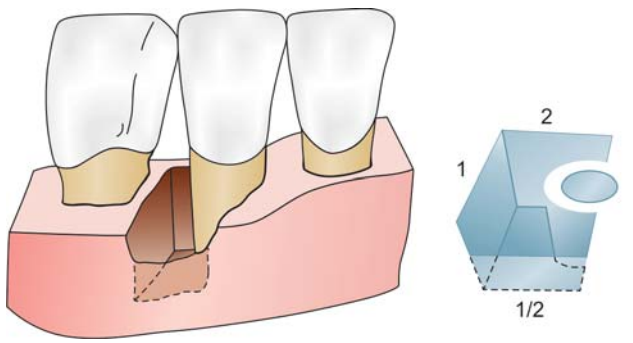


Fig. 22.2: Combination defect

Zero-walled Defects

These are alveolar dehiscences and fenestrations found on facial and lingual surfaces of teeth where the alveolar housing is typically thin or where tooth is abnormally inclined or malpositioned. They are not seen on radiographs. Fenestrations in the presence of

marginal periodontitis may convert to dehiscence. Osseous surgery is not treatment of choice for zero-walled dehiscence.

Four Osseous Walls (Circumferential)

They are usually present with buccal, lingual, mesial and distal wall. Because they sometimes encircle an entire tooth, four-walled defects have been called circumferential or moat defect. Osseous surgery is not treatment of choice with four-walled defect.

But, later on they are removed from this classification.

ETIOLOGY OF INFRABONY POCKET AND INFRABONY DEFECT

Both suprabony and infrabony pockets are the result of plaque; however, there are some differences of opinions for the factors that influence the formation of the infrabony pocket. Most agree that vertical bone loss and subsequent infrabony pocket formation can occur whenever there is direct extension of inflammation into the periodontal ligament, in the presence of sufficient thickness of bone.

Bacterial plaque can induce bone loss within the radius of action of 1.5 to 2.5 mm and there is no bone effect beyond 2.5 mm. Radius of action is principal factor for formation of infrabony defect. Angular bony defects can appear only in the spaces wider than 2.5 mm because narrow spaces would be destroyed entirely. In addition to the important role of local factors, that are plaque, calculus and material alba, trauma from occlusion plays a major role.

1. Trauma from occlusion facilitates the spread of an inflammatory lesion from the zone of irritation directly down into the periodontal ligament (i.e. not via the interdental bone). This alteration of the "normal" pathway of spread of the plaque-associated inflammatory lesion results in the development of angular bony defects. Trauma from occlusion is found as an etiologic factor (codestructive factor) of importance in situations where angular bony defects are combined with infrabony pockets. It may add to the effect of infection by causing bone resorption lateral to periodontal ligament and leads to creation of osseous defect. (Anatomic characteristics of area such as wide bone margin may favor the production of angular lesion and infrabony defect).
2. It was stated that the forceful wedging of the food into the interproximal region may result in unilateral destruction of the attachment apparatus and down growth of the epithelial attachment. Food impaction and infrabony pocket often occurs together but yet it is not established whether food impaction produces pockets or aggravates pockets caused by other factors.

INCIDENCE

Vertical defects can appear on any surface of tooth. Angular defects increase with age. They are found most often on the distal and mesial surfaces. Three-walled defects are more frequently found on the mesial surfaces of second and third maxillary and mandibular molars.

DIAGNOSIS OF INFRABONY DEFECT

1. *Radiograph*

   Vertical defects occurring interdentally can generally be seen on the radiograph, although thick bony plates sometimes may obscure them. Defects appearing on facial and lingual or palatal surfaces are not seen on radiographs.

   X-rays can reveal existence of angular bone loss in interdental spaces but it will not show the number of bony walls of defect. Radiographic marker (Hirschfeld points, silver points, gutta-percha points) placed in the bony defects demonstrate the extent

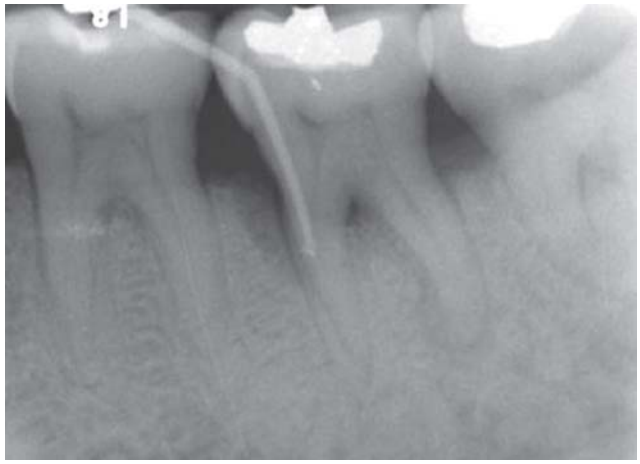


Fig. 22.3: Gutta-percha point in bony defect

of bone loss. Gutta-percha packed around the tooth can be helpful to identify the configuration of defect (Fig. 22.3).

2. *Clinical examination*

Probing can determine the presence and depth of periodontal pockets around any surfaces of any tooth. Both clinical and radiographic examination may suggest presence of infrabony defect, if one or more of following are found.

i. Angular bone loss
ii. Irregular bone loss
iii. Pockets of irregular depth.

Under local anesthesia, osseous defect or morphology is detected by probing from bottom of the pocket, both apically and laterally to alveolar bone. It is called as *"Transgingival probing or Sounding".* It is the process of walking the periodontal probe along the tissue-tooth interface so as to examine, and predict the underlying osseous topography. Typical finding of presence of interdental infrabony defect is sudden increase in the probing depth compared to that of adjacent proximal surface.

However, the three-dimensional morphology of a defect cannot be determined until the defect is visualized at the time of surgery. Surgical exposure and visual examination provide the most definitive information regarding the bone architecture.

TREATMENT

Aims

1. Elimination of the periodontal pocket.
2. Reattachment of periodontal ligament to tooth surface and achievement of a tissue shape which will allow the patient to carry out efficient plaque control.
3. Filling of osseous defect and improve tooth support.

Basic treatment consists of:

- Elimination of local irritants and inflammatory conditions.
- Correction of factors that are responsible for inflammation and that aggravate the effects of trauma and food impaction leading to formation of infra bony pockets.
- To shape the bone in such a way that after healing and remodeling the resultant alveolar architecture will allow effective oral hygiene measures to be carried out. This procedure, osteoplasty, must be undertaken with great care.

- Make an attempt to obtain some fill-in of the bone defect. This may be achieved with or without bone graft.
- To obtain new connective tissue regeneration.

Treatment plan is divided into:

1. Soft tissue phase
2. Hard tissue phase
3. Functional phase
4. Maintenance phase.

Soft Tissue Phase

Management of soft tissue of pocket:

- The soft tissue wall of pocket consists of epithelial lining and granulation tissue. These epithelial structures must be removed to make room for new connective tissue fibers to attach to tooth surface.

Management of periodontal fibers adhering to bone surface:

- Periodontal fibers adhering to the bone must be removed to permit the flow of blood and osteogenic cells into osseous defect.

Hard Tissue Phase

Initial periodontal therapy or basic treatment involving the removal of both sub and supragingival plaque creates an environment conducive for periodontal regeneration. Guided tissue regeneration (GTR) helps in acquiring new attachment on the root surface covered by a membrane, and bone regeneration is expected in the osseous defect area. However, in wide and deep osseous defects, osseous defects in which space making is difficult, and osseous defects with furcation involvement, bone grafts may be used for regeneration.

Management of root surface

Root surface is scaled and planed to remove all deposits, softened tooth structures, adherent remnant and epithelium to make the root surface "hard" and "smooth". It removes not only soft and hard deposits from the root surface but also small amounts of tooth substance (thin layer of altered cementum) as tiny extensions of the subgingival calculus into the root surface hamper the new attachment procedure.

Management of wall of osseous defect

Bony defect is curetted thoroughly to form a clean surface. The debridement of the exposed root surfaces in the defect area is comprehensive. Since the location of the defect and width of the bony defect entrance may limit the access of curettes for proper debridement. Surgical flap therapy offers better visualization and eases the process of debridement. Granulation tissues from the osseous defect are thoroughly removed to provide room for tissue attachment.

Furthermore, at the time of surgery, previously undiagnosed defects may be recognized or some defects may have a more complex outline than initially anticipated. Exposed bone surface of the defect is perforated with a 1/2 round bur after complete removal of granulation tissue from the osseous defect. This facilitates the formation of blood coagulum on the bone surface and accelerates the healing. This is known as *"Regional acceleratory phenomena"*.

Three-walled infrabony defect often provides a better mould for bone repair than two-walled or one-walled defects. Attachment gain can be achieved by flap curettage in a three-walled defect area. New attachment can be achieved even in one-walled and two-walled defects by using a barrier membrane. However, this approach is limited to deep osseous defects due to requirements of space making. Important factors in determining

therapy for intrabony defects include depth of defect, width, position, number of remaining bone walls, and adjacent root morphology.

1. One-walled angular defects usually require the bone to be reduced to the level of the most apical portion of the defect.
2. Three-walled defects, if narrow and deep, can be successfully treated by new attachment and bone reconstruction therapy.
3. Two-walled defects can be treated with either of above method, depending on depth, width and configuration of defect.
4. Ochsenbein proposed surgical therapy for combination defect that combined regenerative and resective procedures. In combination defect, the wall coronal to a three-walled combined-type intrabony defect has no hope for regeneration. The osseous defect then is reshaped to three walls, and a barrier membrane is placed over the osseous defect to facilitate regeneration.

Selection of Method

The method of achieving regeneration is selected after careful probing and clinical and radiographic examination. The final decision is based on morphology of osseous defect (depth and width), degree of furcation, and the anatomic condition of the root as observed clinically after flap reflection (Fig. 22.4).

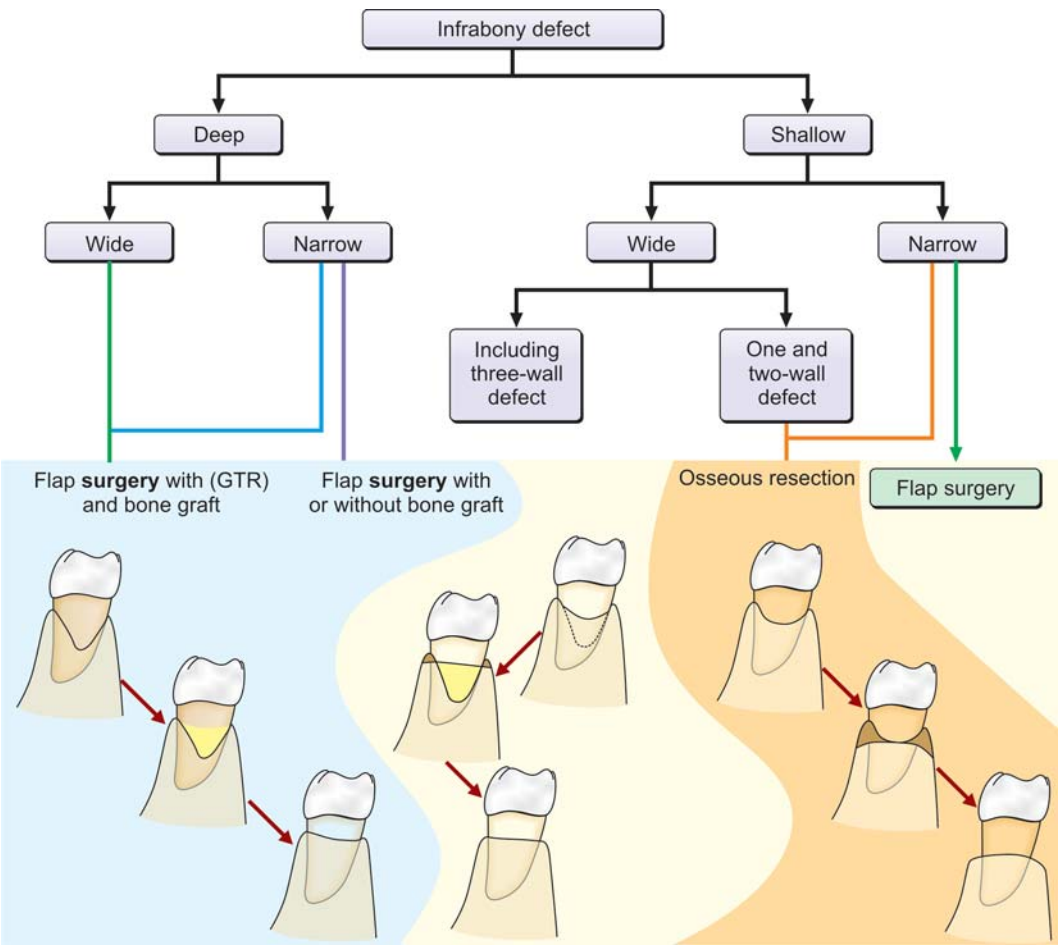


Fig. 22.4: Treatment planning

In shallow osseous defects, a resective procedure should be selected because bone regeneration cannot be expected. However, morphology is not the only factor to consider when selecting a method for treating the osseous defect.

The prognosis for successful resolution of infrabony defects is influenced by the:

1. Number of remaining osseous walls
2. Size of the osseous defect (depth, width)
3. The proximity of the defect to important anatomical landmark
4. Number of root surfaces involved
5. Extent of bony destruction
6. Amount of bone that need to be removed to achieve positive bony architecture
7. Presence or absence of furcation involvement
8. Ability to effectively detoxify and debride the defect and tooth
9. The predictability of alternate form of therapy.

Segments of the periodontium with generalized horizontal patterns of bone loss and multiple shallow interproximal osseous defects with less than three walls are traditional indications for osseous surgery. As a general rule, defect with greater number of osseous walls and also narrower in width, better the prognosis for regeneration. Defects that conceivably will hold water offer excellent opportunities for bone graft containment and periodontal regenerative procedures.

Regenerative procedures using a barrier membrane is another choice. However, it is not applicable in the esthetic zone because remarkable postoperative gingival recession occurs, if complete membrane coverage is not achieved.

Of the regenerative procedures, GTR is the method that requires preservation of the interdental papilla and thick gingiva. Therefore, GTR cannot be used where there is thin gingiva or gingival recession. Sufficient width of keratinized gingiva is necessary for flap surgery without a barrier membrane. If there is insufficient keratinized gingiva in the surgical area, increasing the keratinized gingiva with a free autogenous gingival graft is needed as pretreatment (Fig. 22.5).

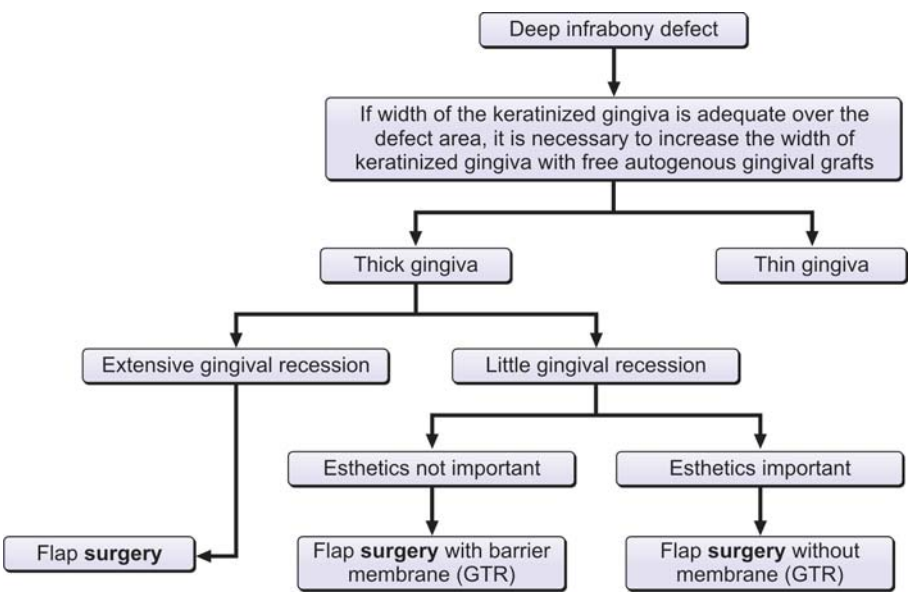


Fig. 22.5: Selection of method

Functional Phase

It includes following steps for correction of occlusal trauma to restore balanced occlusion,

- Selective reshaping of occlusal surfaces with the goal of establishing a stable, nontraumatic occlusion is called as "*Occlusal equilibrium or coronoplasty*". It eliminates premature contacts and reduces the loading of teeth that have lost bone due to periodontal disease. As occlusal adjustment is an irreversible intervention, clinician should be prudent to plan and to execute the cuspal reshaping.
- Removal of plunger cusp causing food impaction is an imperative procedure in treating infrabony defect.
- Grinding of high point contact of restoration and marginal ridges alters contour of the unfavorable, faulty restorations and helps in establishing stable functional relationships favorable to the patient's oral health.
- Occlusal rehabilitation by prosthesis in the form of crowns and bridges restores the plane of occlusion and establishes the full mouth equilibration to the degree that maximum intercuspation is coincident with centric relation. Building proper contacts between adjacent teeth prevents food impaction.

The role of occlusal adjustment in the management of periodontal disease is more complex because both periodontitis and trauma from occlusion can lead to tooth mobility.

Maintenance Phase

Patient should be advised to rinse with 0.12 percent chlorhexidine gluconate immediately after surgical procedure and twice daily thereafter until normal plaque control technique can be resumed. Plaque and food accumulation impair the healing so patient is advised to keep the area as clean as possible by the gentle use of soft toothbrush. Vigorous brushing is not feasible during the first few weeks after the surgical procedure. Proper brushing technique is explained to the patient as the daily mechanical removal of plaque by the patient is the only practical mean of maintaining oral health.

Suprabony pocket	*Infrabony pocket*
Base of the pocket is coronal to alveolar bone.	Base of the pocket is apical to alveolar bone (i.e. pocket wall lies between bone and tooth).
Pattern of bone destruction is horizontal.	Pattern of bone destruction is vertical.
The transseptal fibers are arranged horizontally in the space between base of pocket and alveolar bone.	The transseptal fibers are oblique rather than horizontal. They extend from cementum beneath base of pocket along the alveolar bone and over the crest to cementum of adjacent tooth.
On facial and lingual surfaces, periodontal ligament fibers beneath pocket follow their normal course between tooth and bone.	On facial and lingual surfaces, periodontal ligament fibers follow angular pattern of adjacent bone.

CHAPTER

23 Bone Replacement Grafts

Numerous therapeutic grafting modalities for restoring periodontal osseous defects have been investigated. Bone replacement grafts include autografts, allografts, xenografts, and alloplasts. Bone replacement grafts are the most widely-used treatment options for the correction of periodontal osseous defects. It has been proved that bone replacement grafts provide clinical improvements in periodontal osseous defects compared with surgical debridement alone. For the treatment of infrabony defects, bone grafts have been found to increase bone level, reduce crestal bone loss, increase clinical attachment level, and reduce probing pocket depths compared with open flap debridement procedures.

Periodontal defects as sites for transplantation differ from osseous cavities surrounded by bony walls. Saliva and bacteria may easily penetrate along the root surface, and epithelial cells may proliferate into the defect, resulting in contamination and possible exfoliation of the grafts. Therefore, the principles established to govern transplantation of bone or other materials into closed osseous cavities are not fully applicable to transplantation of bone into periodontal defects.

Bone graft materials are generally evaluated based on their osteogenic, osteoinductive, or osteoconductive potential. *Osteogenesis* refers to the formation or development of new bone by cells contained in the graft. *Osteoinduction* is a chemical process by which molecules contained in the graft (bone morphogenetic proteins) convert the neighboring cells into osteoblast, which in turn form bone. *Osteoconduction* is a physical effect by which the matrix of the graft forms a scaffold that favors outside cells to penetrate the graft and form new bone.

It would seem that graft materials which lack osteogenic potential act simply as a replacement for the blood clot which usually breaks down, or as an inert scaffold on which some bone formation takes place prior to the resorption of the graft. This is because cellular events of periodontal regeneration involve the controlled integration of a number of cell signaling systems for bone, cementum and periodontal ligament. Unless these are present in the graft material and/or in the adjacent tissues in the right proportions, controlled regeneration cannot take place. However, regeneration of new cementum, periodontal ligament and alveolar bone can be achieved to some degree in intrabony defects with some grafting techniques.

EXTRA AND INTRAORAL DONOR SITES FOR AUTOGENOUS BONE GRAFTS

If autogenous bone is considered the gold standard, its osteogenic potential is one feature that differentiates it from the rest of the graft materials. The primary reason for its superiority is its capacity to support osteogenesis in conjunction with its endogenous osteoinductive and osteoconductive properties. Due to their osteogenic potential, autogenous bone grafts of extra- and intraoral sources have been used in periodontal therapy.

The major drawback of the autogenous bone graft is that a donor site is required to harvest the bone, which can lead to increased time, cost, and morbidity for patient. The donor site and its individual variability limit the amount of bone that can be harvested.

Cancellous bone and marrow can be obtained from a number of sites in the mouth such as the tuberosity, extraction sockets or the edentulous ridge, bone trephined from within the jaw without damaging the roots. The maxillary tuberosity or a healing extraction site is typically the donor choice for intraoral cancellous bone with marrow grafts *(Cancellous bone marrow transplant)*. They are generally easy to manipulate, and revascularize rapidly. It is important to remember that cancellous bone imparts no mechanical strength; when it is used to reconstruct large continuity defects, additional rigid fixation is required. In the oral cavity, cancellous grafts are used to fill bony defects, alveolar clefts, maxillary sinus, and other similar scenarios where bone can be placed into an area and retained. The corticocancellous graft usually produces the best results by combining the attributes of both cortical and cancellous forms. It allows for mechanical stabilization while providing good revascularization.

Iliac grafts have been used fresh or frozen. One common complication is fresh marrow tissue *(Iliac autografts)* often produces root resorption and ankylosis. Successful bone fill has been demonstrated using iliac cancellous bone with marrow in furcations, dehiscences, and intraosseous defects of various morphologies. But they have had only limited use because of the difficulty in obtaining the graft material, morbidity, postoperative infection, varying rates of healing, the possibility of root resorption and rapid recurrence of the defect. In addition to these, it increases patient expense and is also found difficult to procure the donor material so the technique is no longer in use.

Good clinical results have been achieved with the use of cancellous autogenous bone grafts from an adjacent edentulous site. Other techniques report bone fill using cortical bone chips and osseous coagulum or bone blend–type grafts. Studies report histologic evidence of regeneration and new connective tissue attachment and the presence of a long junctional epithelium following these procedures.

OSSEOUS COAGULUM

Osseous coagulum is a technique described by R. Earl Robinson using a mixture of bone dust and blood. Small particles ground from cortical bone are used. The advantage of the particle size is that it provides additional surface area for the interaction of cellular and vascular elements. Graft materials can be taken from the lingual ridge on the mandible, exostoses, edentulous ridges, the bone distal to a terminal tooth, bone removed by osteoplasty or ostectomy, and the lingual surface of the mandible or maxilla.

BONE BLEND

Bone is removed from a predetermined site, triturated in the capsule to a workable, plastic-like mass, and packed into bony defects.

BONE SWAGING

This technique requires an edentulous area adjacent to the defect, from which the bone is pushed into contact with the root surface without fracturing the bone at its base.

ALLOGENIC BONE GRAFTS

Allogenic bone is nonvital, osseous tissue taken from one individual and transferred to another of the same species. Iliac cancellous bone and marrow, freeze-dried bone allograft (FDBA), and decalcified freeze-dried bone allograft (DFDBA) are the types of bone allografts widely available from commercial tissue banks. Grafts are taken from cadaver bone, typically freeze-dried and treated to prevent disease transmission. They are obtained from cortical bone within 12 hours of the death of the donor, defatted, cut in pieces, washed in absolute alcohol, and deep-frozen. The material may then be demineralized, and subsequently

ground and sieved to a particle size of 250 to 750 μm and freeze-dried. Finally, it is vacuum-sealed in glass vials.

Numerous steps are also taken to eliminate viral infectivity. These include exclusion of donors from known high risk groups and various tests on the cadaver tissues to exclude individuals with any type of infection or malignant disease. The material is then treated with chemical agents or strong acids to inactivate the virus, if still present.

Freeze-dried Bone Allograft

FDBA works primarily through osteoconduction. The graft does not activate bone growth but acts like a scaffold for natural bone to grow into. Eventually the graft is resorbed and replaced by new bone. Freeze-drying the bone decreases the antigenicity of the allograft. Radiographically, FDBA appears radiopaque because it is not demineralized. When using FDBA to treat periodontal defects, trials indicate bone fill ranging from 1.3 to 2.6 mm. A combination of FDBA with tetracycline has also shown promising results in the treatment of defects resulting from juvenile periodontitis.

Demineralized (Decalcified) Freeze-dried Bone Allografts

Urist showed that DFDBA is osteoinductive. Demineralization in cold, diluted hydrochloric acid exposes the components of bone matrix, which are closely associated with collagen fibrils and have been termed bone morphogenetic proteins (BMPs). DFDBA is believed to induce bone formation due to the influence of these bone-inductive proteins (BMPs) exposed during the demineralization process. DFDBA is therefore thought to be osteoinductive and osteoconductive.

DFDBA has demonstrated periodontal regeneration in controlled human histologic studies. Superior gains in bone fill with DFDBA compared with openflap debridement have consistently been reported. It has been observed in several reentry studies that grafting with DFDBA is always superior to debridement alone when used for the correct indications. Laboratory studies have found that DFDBA has a higher osteogenic potential than FDBA and is therefore preferred. Studies have demonstrated that preparation of allograft material can differ from one distributor to another and that the material may differ in its biologic activity. DFDBA may also vary from batch to batch. Commercial bone banks do not verify the specific amount of BMPs or the levels of inductive capacity in any graft material. The development of stricter bone bank standards that evaluate the potency of their preparations, including (1) using bones from individuals under a specific age, using bones from individuals free of bone diseases, or using fresh bone, and (2) developing assays that can test the inductive capacity of the material before sales, may lead to more consistent and reliable clinical results.

Comparison of freeze-dried bone allograft and demineralized freeze-dried bone allograft

FDBA	*DFDBA*
Not demineralized	Demineralized
Better space maintenance	More bone morphogenetic protein expression potential
Slower resorption rate compared with DFDBA	Possible osteoinduction
Osteoconductive	Osteoconductive
More radiopaque	More radiolucent
Breakdown by way of foreign body reaction	Rapid resorption
Primary indication: bone augmentation associated with implant treatment (e.g. guided bone regeneration, sinus grafting, ridge augmentation)	Primary indication: periodontal disease associated with natural tooth

ALLOPLASTIC MATERIALS

Alloplastic materials are synthetic, inorganic, biocompatible, or bioactive bone graft substitutes. Alloplastic materials are believed to promote bone healing through osteoconduction. Currently, six types of alloplastic materials are commercially available: hydroxyapatite cement, nonporous hydroxyapatite, porous hydroxyapatite (replamineform), beta tricalcium phosphate, polymethylmethacrylate/hydroxyethylmethacrylate (PMMA/HEMA) calcium-layered polymer, and bioactive glasses and ceramics. Ideally, alloplast bone substitutes should have the following properties: (1) biocompatibility, (2) minimal fibrotic reaction, (3) the ability to undergo remodeling and support new bone formation, (4) similar strength comparable to cortical/cancellous bone, and (5) similar modulus of elasticity comparable to bone to prevent fatigue fracture under cyclic loading.

Calcium phosphate biomaterials have excellent tissue compatibility and do not elicit any inflammation or foreign body response. These materials are osteoconductive, not osteoinductive. Two types of calcium phosphate ceramics have been used, as follows:

1. Hydroxyapatite (HA) has a calcium-to-phosphate ratio of 1:67, similar to that found in bone material. It is generally nonbioresorbable.
2. Tricalcium phosphate (TCP), with calcium-to -phosphate ratio of 1:5, is mineralogically B-whitlockite. It is at least partially bioresorbable.

Bioactive glass is made from calcium salts, phosphate, sodium salts, and silicon. It is used in the form of irregular particles measuring 90 to 170 pm (PerioGlas, Block Drug, Jersey City, NJ) or 300 to 355 pm (BioGran, Ortho Vita, Malvern, Pa). When this material comes into contact with tissue fluids, silicon forms a silica gel layer on the surface of these particles which promotes formation of a hydroxycarbonate-apatite layer and incorporates organic ground proteins such as chondroitin sulfate and glycosaminoglycans. On this layer of hydroxycarbonate-apatite, osteoblasts are claimed to proliferate and form bone. These materials have the ability to truly integrate or chemically bond with bone, because of its capability to exchange ions or molecules with osseous tissue. Overall, histologic evaluation of bioactive glass shows limited regenerative potential, with minimal bone regeneration and no signs of new cementum or periodontal ligament. These bioactive glass materials also appear to be encapsulated by collagen. Tricalcium phosphate and bioactive glass are absorbable.

Porous and nonporous hydroxyapatite materials and PMMA/HEMA polymer are nonabsorbable. Histologically, however, alloplast grafts tend to heal by encapsulation with connective tissue with minimal or no bone formation. Some histologic evidence shows that a very limited amount of regeneration may be possible following PMMA/HEMA polymer grafts.

Coral-Derived Materials have been used in clinical periodontics. There are two different coralline materials: natural coral and coral-derived porous hydroxyapatite. Both are biocompatible, but whereas natural coral is resorbed slowly (several months), porous hydroxyapatite is not resorbed or takes years for resorption.

Overall, the effect of alloplast material has been inconsistent. It appears that alloplastic materials function as nonirritating fillers.

XENOGRAFTS

Xenograft (heterograft) is osseous tissue that is harvested from one species, processed, and then transferred to a recipient site of a different species. They are bone from a different species treated with ethylene diamine to remove the organic and antigenic fraction and referred to as inorganic bone. These proprietary processes remove all cells and

proteinaceous material. It leaves behind inert, absorbable bone scaffolding. It is on this scaffolding that revascularization, osteoblast migration, and woven bone formation supposedly occur. Signs of periodontal regeneration have been reported with xenografts; however, most data support a bone fill or repair of bone for guided bone regeneration around implants, sinus lift procedures, and ridge augmentation.

Calf bone treated by detergent extraction, sterilized, and freeze-dried, has been used for the treatment of osseous defects. Kiel bone is calf or ox bone denatured with 20 percent hydrogen peroxide, dried with acetone, and sterilized with ethylene oxide. These materials have been tried and discarded for various reasons.

Currently, an inorganic, bovine-derived bone marketed under the brand name Bio-Oss (OsteoHealth) has been successfully used both for periodontal defects and in implant surgery. It is an osteoconductive, porous bone mineral matrix from bovine cancellous or cortical bone. The organic components of the bone are removed, but the trabecular architecture and porosity are retained.

CHAPTER

24 Hypersensitivity

Dental hypersensitivity is a major patient problem in dentistry and several explanations for its mechanism have been given. It is a symptom complex rather than a disease.

DEFINITION

"It is a brief, sharp painful sensation in response to heat, cold, tactile and osmotic stimuli".

A more specific definition was given by Holland and colleagues, who stated that dentin hypersensitivity is a "short, sharp pain arising from exposed dentin in response to stimuli typically thermal, evaporation, tactile, osmotic or chemical and which cannot be ascribed to any other form of dental defect or pathology."

It is the most common dental problem characterized by severe acute shooting pain. Dental hypersensitivity is the particular problem in the patient after periodontal surgery.

BASIC CONCEPTS OF TOOTH SENSITIVITY

The most widely accepted explanation of dentin hypersensitivity is **"Hydrodynamic theory"** by Brännstörm and Orchardson and Gillam.

According to this theory, various thermal, evaporation, tactile, osmotic stimuli causes rapid outward flow of fluid in the dentinal tubules. Rapid flow causes a pressure changes across the dentin, stimulating pulpal A-δ nerve fibers and resulting in perception of pain. Cold is most common stimuli for dentin hypersensitivity.

Dentin hypersensitivity is more likely to occur when tubules are open and in communication with the oral environment. Hypersensitive dentin contains larger and more numerous tubules than normal nonsensitive dentin. Greater and larger tubules lead to substantial increase in dentinal fluid flow and thereby amplify the severity of response to external stimuli which may not be able to produce pain in normal dentin. *The increase in fluid flow with increasing tubule size is greater than might be expected because the area of circle increases by square of radius.*

PAIN MEDIATORS

- Nociceptors
- Sensory nerve terminals for pain
- A-δ nociceptors which mediates pain.

NEUROPHYSIOLOGY

The rich network of dental pulp is highly innervated by both myelinated and unmyelinated nerve fibers (Fig. 24.1). Nerve fiber in pulp contains Aδ and c fibers which are responsible for conduction of stimuli.

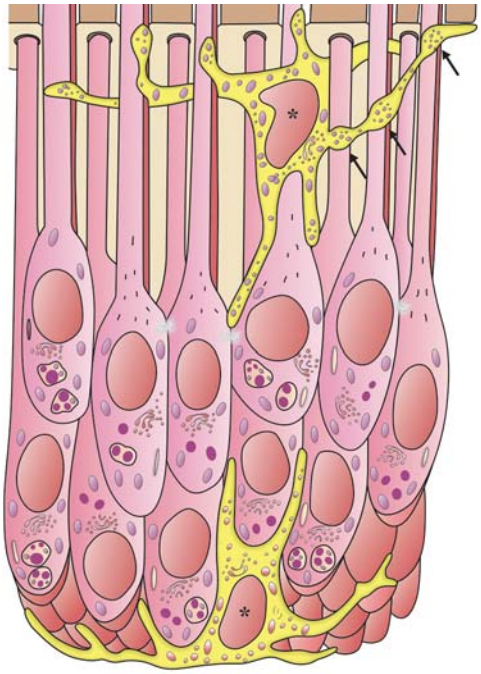


Fig. 24.1: Dental neural structure

Aδ and Aβ fibers	Are myelinated and conduct fast pain
C fibers	Are unmyelinated and conduct slow pain. They are very sensitive to local anesthesia

Myelinated Aδ and Aβ fibers seem to be responsible for dentin sensitivity, whereas C fibers are concerned with dull radiating pain sensation connected with pulpal inflammation.

OTHER THEORIES OF DENTIN HYPERSENSITIVITY

Odontoblastic Transduction Theory

This theory is based on the synaptic like relationship between the terminal nerve endings and the odontoblastic processes. Such synaptic relation, if present, then it can be the way for transmission of the stimuli from the tubules to the brain. It requires the acetylcholine as a neurotransmitter.

Modulation Theory

Strong stimuli to the dentin may injure the odontoblasts which subsequently release variety of neurotransmitting agents as well as vasoactive and pain producing amines and proteins.

Gate Control Theory

When dentin is irritated, all the pulpal nerves become activated from the stimuli-like vibration and leads to hypersensitivity.

PREVALENCE

- 4-57 percent of adult population experience cervical dentin hypersensitivity in one or

more teeth.

- It is commonly seen in 25–45 years of age group due to the general increase in exposed root surfaces of the teeth from periodontal diseases, toothbrush abrasion or cyclic loading fatigue of the thin enamel near cementoenamel junction (CEJ).
- Generally, slightly higher incidence of hypersensitivity is reported in females than in males.
- It is substantially higher in periodontal patients (60-98 percent).

HIGHEST INCIDENT SITE

The most common locations for dentin hypersensitivity are cervical region of facial surfaces of canines > premolars > incisors > molars and commonly on opposite side of the patient's dominant hand.

Regarding the side of mouth, in right-handed toothbrushers dentin hypersensitivity is greater on the left-sided teeth.

PATHOPHYSIOLOGY

- Teeth sensitivity occurs more frequently in cervical area of the root where the cementum is extremely thin. Scaling and root planing procedures may remove the thin layer of cementum leading to hypersensitivity.
- Transmission of stimuli from surface of dentin to nerve endings located in dental pulp/ in pulpal region of dentin may occur through odontoblastic processes by displacement of dentinal fluid.
- Sensitivity is encountered when physical, chemical, thermal, bacterial, and traumatic stimuli are transmitted through tubule which initiates pain (Fig. 24.2).
- Dentin hypersensitivity is primarily dependent on remaining dentine thickness (effective depth) and diameter of tubules.
- The tubules are shorter, more numerous and increase in diameter near the pulp. Deep dentin is less effective pulpal barrier than superficial dentin near the dentinoenamel junction

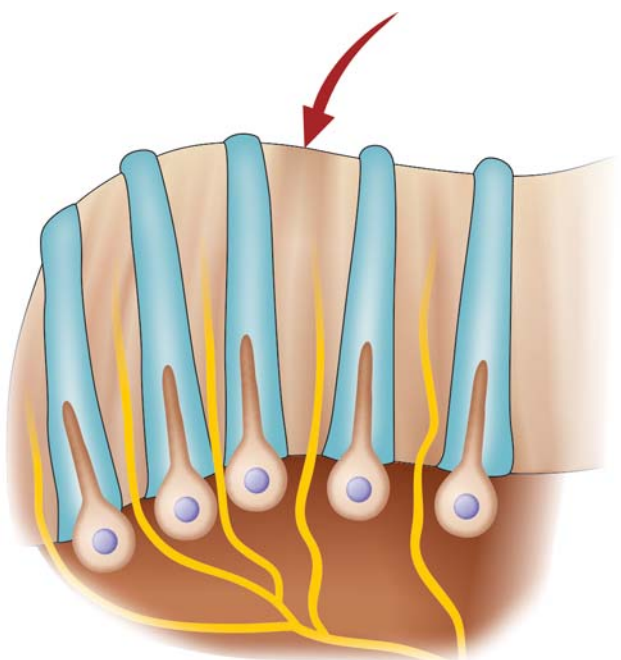


Fig. 24.2: Pathophysiology

or dentinocementum junction.

CAUSES

The primary etiological factor responsible for dentinal hypersensitivity is exposed dentinal tubules and tubules must be open. Exposed dentin is usually covered by a smear layer made up of loosely bound organic and calcified debris that clogs the dentinal tubules so that they are not open to the environment. When the smear layer is removed, the tubule openings become exposed and are then susceptible to external stimuli, which can lead to dentin hypersensitivity. The smear layer is easily removed by dietary acids, gastric acids, and also detergents found in toothpaste and rinses.

Dentin may become exposed by two processes; either by loss of covering periodontal structures (gingival recession) or by loss of enamel (tooth wear). The most common clinical cause for exposed dentinal tubules is gingival recession.

Dental hypersensitivity is perhaps a symptom complex rather than a disease and results from stimulus transmission across exposed dentin. A number of dental conditions are associated with dentin exposure and therefore may produce the same symptoms.

Such conditions include:

- Gingival recession (Gingival recession doesn't cause cervical hypersensitivity but rather sets the stage for it by exposing root surfaces to the oral environment)
- Attrition, abrasion, erosion, abfraction
- Postperiodontal treatment (Scaling, root planing, periodontal surgery)
- Traumatic injury
- Chipped tooth
- Fractured restoration
- Restorative treatments
- Dental caries
- Cuspal grinding (during chewing)
- Crown preparation
- Aging.

CLINICAL IMPLICATIONS

Dental hypersensitivity manifests as short and sharp pain in response to certain stimuli. Patient with hypersensitivity refrains from brushing because of pain and this leads to reduced oral hygiene and vicious cycle of plaque accumulation. This aggravates the periodontal problems and further leads to increase in hypersensitivity.

DIAGNOSTIC METHODS

According to a recent review, "Dentin hypersensitivity is usually diagnosed after other possible conditions have been eliminated" and may be considered as a diagnosis of exclusion.

Thermal Stimuli

Sensitivity to thermal stimuli, especially to cold appears to be the most prevalent feature. Stimulus can be delivered to the tooth simply by gentle blow of the room temperature air. Cold water testing method utilizes cold water (7°C) to provoke the sensitivity. Sometimes heat can also act as stimulus.

Tactile Sensation

This is a simplest method in which the tooth is gently touched with sharp explorer over the suspected site (of attrition, abrasion) to grade the response on a severity scale. Toothbrushing and flossing can also serve as stimuli.

Osmotic Stimuli

Hyperosmotic salt solutions like sodium chloride, calcium chloride or sweet solutions like glucose, sucrose are used to elicit the sensitivity.

Electric Stimuli

It can be delivered by using electric pulp tester. Its use is limited as it may act as harsh stimuli and lead to severe hypersensitivity.

CONDITIONS TO RULE OUT HYPERSENSITIVITY

As many conditions are associated with dentinal hypersensitivity, a careful history together with a thorough clinical and radiographic examination is necessary before arriving to definitive diagnosis. Sometimes, there may be more than one condition coexist which makes it difficult to approach a perfect diagnosis.

- Pulpal pathology
- Undisplaced palatal cusp
- Tooth with cracked cusp
- Fractured tooth with exposed dentin
- Fracture of restoration
- Areas of root involvement
- Areas of tooth in bruxism patient.

WAYS TO RULE OUT

- Careful history
- Clinical examination
- Radiographic examination.

OCCURRENCE OF PAIN

- Pain on brushing
- Pain on flossing
- Hot/warm beverages
- Eating of cold food especially ice cream
- Cold drinks
- Breathing of cold air
- Eating sour acidic food
- Eating sweet/sugary liquids.

PEOPLE AT RISK

- Over enthusiastic brushers (Hard bristle brushes)
- Periodontally treated patients
- Bulimics
- Patients with xerostomia
- Acidic food/drink consumers
- Older patients with gingival recession

– Age-associated changes also produces sensitivity.

TREATMENT STRATEGIES

For long-term management, in addition to pain control, treatment strategies should include the elimination or reduction of contributing factors. Hypersensitivity can resolve without the treatment or may require use of desensitizing agents for several weeks. Once diagnosed, trace out the reason for the exposed dentinal tubules and any other etiological factor causing hypersensitivity. Remove the etiological factors and educate the patient about the dietary habits.

Treatment of hypersensitivity is challenging for both patient and clinician because:

1. The pain threshold differs from patient-to-patient.
2. It is difficult for patient to change the habit causing problem.

Single treatment modality may not be consistently effective in treating the hypersensitivity therefore dentist must be familiar with alternative methods of treatment.

The principal treatment strategies are:

1. Desensitize the nerve, making it less responsive to stimuli.
2. Occlude tubules preventing the outward fluid flow.

This can be addressed by:

1. Home care with dentifrices
2. In office treatment modalities
3. Patient education.

MANAGEMENT OF HYPERSENSITIVITY

1. Home care with dentifrices
   a. Potassium nitrate dentifrices (5%)
   b. Strontium chloride dentifrices (10%)
   c. Fluoride dentifrices (Sodium monofluorophosphate)
   d. Bioactive glass (Calcium sodium phosphosilicate).
2. Professional/In office treatment modalities
   a. Tubule sealants
      – Dentin bonding agents
      – Restorative resins.
   b. Cavity varnishes
   c. Treatments that partially blocks the tubules
      – Iontophoresis
      – Fluoride compounds: (Sodium fluoride, sodium silicofluoride, stannous fluoride)
      – Tubule blocking agents (Potassium oxalate, Strontium chloride)
      – Calcium compounds (Calcium hydroxide, Dibasic calcium phosphate)
      – Silver nitrate
      – Zinc chloride (Potassium ferrocyanide).
   d. Antiinflammatory drugs
   e. Miscellaneous
      – Lasers
      – Dental restorations.
3. Patient education

   Patient education is of prime importance to control of etiological factors.
   – Plaque control
   – Proper toothbrushing technique with soft brushes
   – Control of xerostomia

– Diet counseling.

Hypersensitivity sometimes resolves without any kind of treatment. This can be attributed to decreased dentin permeability because of natural mechanisms.

Natural mechanisms contributing to desensitization includes;

1. Formation of reparative dentin by pulp
2. Obturation of dentinal tubules by mineral deposition.

Several factors can reduce dentin permeability and subsequently contribute to the spontaneous remission of dentin hypersensitivity. For example, a dentin smear layer often covers the exposed surface of dentin and may occlude the dentinal tubules, thereby reducing dentin hypersensitivity. Adherence of salivary proteins to the outer dentinal surface and adherence of plasma proteins to the inner dentin surface may also reduce hypersensitivity, as can the formulation of less permeable reparative dentin by the pulp.

Home Care with Dentifrices

Medicated dentifrices are most widely advocated home care treatment modality for dentinal hypersensitivity. It is the simple and inexpensive way to treat dentinal hypersensitivity. However, the duration of relief is highly variable.

Advantages of using dentifrices

- Noninvasive
- Easy to use
- Inexpensive
- Treat multiple teeth simultaneously.

Disadvantages of using dentifrices

- Compliance
- Difficulty to deliver to specific sites
- Slow onset of action
- Requirement of continuous use.

Potassium Nitrate Dentifrices (5%)

The first approach to treat the dentinal hypersensitivity is to treat the tooth with a chemical agent such as potassium nitrate or potassium chloride. The increased level of potassium ions into the dentinal tubules reduces the sensitivity by inactivating voltage-gated sodium channel, thereby blocking active potential generation. Therefore the tubules remain patent and rapid fluid flow still occurs in response to stimuli, but the nerves are not activated.

Strontium Chloride Dentifrices (10%)

The second approach to treat the tooth with a chemical or physical agent such as strontium chloride that creates a deposition layer and mechanically occludes dentin tubules, which reduces sensitivity by prevention of pulpal fluid flow. Potassium nitrate is a diffusible-free radical of nitric oxide that can reach the nerve endings easily. Hypersensitivity may reappear due to toothbrush abrasion, the presence of acid challenges in the mouth or by degradation of coating.

Fluoride Dentifrices (Sodium Monofluorophosphate)

Fluoride helps in remineralization of dentin and works in three ways to reduce dentinal hypersensitivity.

- It reduces the ability of bacteria to make acids.
- It reduces the acid dissolution of enamel.

- It remineralizes the areas of tooth that have been attacked by acids from bacteria.

Bioactive Glass (Calcium Sodium Phosphosilicate)

Calcium sodium phosphosilicate is a highly biocompatible bioactive glass. In presence of body fluids, it deposits hydroxycarbonate apatite (HCA) on to the exposed dentinal tubules and mechanically occludes the dentinal tubules.

Mechanism (Fig. 24.3)

Calcium sodium phosphosilicate exchanges sodium ions (Na^+) with hydrogen cations (H^+ or H_3O^+) ions on exposure to aqueous environment

↓

Rapid release of (Ca^+) and phosphates (PO_4^{3-}) which blocks the dentinal tubules by forming Ca – P layer on the dentin surface

↓

Multiple layers crystallize into biologically stable hydroxylapatite crystal

Professional/In Office Treatment Modalities

According to hydrodynamic theory, a rapid movement of fluid from dentinal tubules activates the intradental sensory nerves and the hypersensitivity can be treated effectively by occluding the dentinal tubules, reducing the diameter of the tubules or by covering the tubule orifice. In office treatment modalities works mainly by obturating the dentinal tubules.

Tubule Sealants

Dentin bonding agent and restorative resins impregnate the dentinal tubule, form a polymeric coating on the tooth surface and prevents the pain producing stimuli reaching the pulp. Usually this is not employed for generalized root sensitivity but it can be an effective alternative when other forms of therapy fail. Gluma desensitizer containing 30 percent HEMA (Hydroxyethyl methacrylate) and 5 percent glutaraldehyde is an effective desensitizing agent. Strong bonds of these agents to the dentin can provide relief from sensitivity when other form of modality fails. They are relatively thin and susceptible to abrasion.

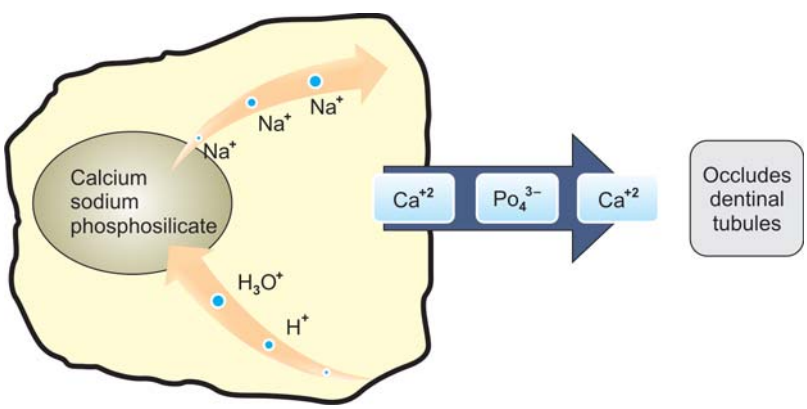


Fig. 24.3: Mechanism of action of bioactive glass

Cavity Varnishes

Cavity varnishes when applied to the exposed dentinal surfaces, it forms a thin film and blocks the stimuli reaching the pulp. It provides temporary relief however; varnishes containing fluoride can be more effective due to sustain release of fluoride. They adhere to the dry or wet tooth surfaces and sets in contact with saliva. 5 percent sodium fluoride contains 22600 ppm of fluoride and forms a protective layer of calcium fluoride that inhibits fluid flow within tubules, which has been suggested to be effective treatment.

Treatments that Partially Blocks the Tubules

Iontophoresis

Iontophoresis works by forcing the fluoride ions deeply into the dentin which cannot be achieved with topical fluoride application. It uses the electrical potential which transfers the ions into the dentin. If the drug is negative, treatment electrode is also negative and if drug is positive, treatment electrode is also positive. By completion of the circuit with a second indifferent electrode, the drug is forced into the tissue to be treated.

Fluoride Compounds

Fluoride is well known desensitizing agent that works by precipitation of calcium and phosphates in the dentinal tubules and reduces the lumen diameter.

NaF forms layer of CaF_2 (Dominant reaction product) which occludes the tubules.

Acidulated sodium fluoride has acidic pH so it demineralizes the dentin and therefore leads to deeper penetration of fluoride.

Sodium silicofluoride forms a gel by reacting with calcium of tooth and forms barrier on the tooth surface. 0.6 percent sodium silicofluoride is highly potent desensitizing agent.

Stannous fluoride releases tin and fluoride and forms highly insoluble tin-fluoride phosphate complex on dentinal surface that is more resistant to dissolution by acid. 10 percent solution of stannous fluoride also blocks the tubules.

Strontium Chloride

Topical application of concentrated strontium chloride on the exposed dentin surface penetrates up to the depth of 10–20 μm and plugs the tubules.

Oxalates

Oxalate ions react with the calcium ions in the dentinal tubules and form insoluble calcium oxalate crystals that are deposited in the tubules. They are inexpensive, easy to apply and well tolerated by patient. Potassium oxalate and ferric oxalate solutions are most commonly used. It has been shown that potassium oxalate has both dentinal tubule obturation properties and inhibitory effects caused by the potassium ions actions on nerve activity. Oxalate ions react with calcium to form insoluble calcium oxalate crystals that bind tightly to dentin and obturate the dentinal tubules.

Calcium Compounds (Calcium Hydroxide, Dibasic Calcium Phosphate)

These are very primitive and well known agents used for treatment of hypersensitivity. The exact mechanism of action is unknown but they may work by:

- Blocking the dentinal tubules

- Inducing peritubular dentin mineralization
- They may reduce the excitability of nerves at higher concentration.

The paste of calcium hydroxide is applied to root surface and allowed to remain there for 3-5 minutes.

Silver Nitrate

It blocks the tubules by precipitation of constituents of dentinal tubules.

Zinc Chloride (Potassium Ferrocyanide)

It forms a stable and highly crystalline precipitate which covers the exposed dentin.

Miscellaneous

Laser Treatment

There are a number of reports that suggest that laser treatment may be useful in the treatment of dentin hypersensitivity, although definitive trials are lacking. Combination of dental laser with other modes of treatment of dentinal hypersensitivity is an effective and lasting dentinal tubule obturation procedure.

Patient Education

Plaque Control

The role of plaque as an etiological factor in dentin hypersensitivity is not resolved, although it has been suggested that dental plaque promotes and sustains dentin hypersensitivity, and that plaque control is important in preventing its development. However, it has been noted that hypersensitive dentin is found in toothbrush abrasion areas that are almost plaque-free.

Proper Toothbrushing Technique with Soft Brushes

Faulty brushing technique can lead to tooth wear resulting in hypersensitivity. Loss of dentin can be prevented by use of proper brushing technique and selection of soft brush.

Control of Xerostomia

Reduced salivary secretion may lead to plaque accumulation and increase in intraoral pH and subsequently hypersensitivity. Salivary substitutes can reduce the pH and prevent the exposure of dentin.

Diet Counseling

Diet with high acidic content leads to erosion of tooth structure especially cementum of exposed root surface leading to opening of dentinal tubules. Diet counseling should focus on quantity and frequency of acid intake occurring in relation to the brushing time. Any treatment may fail, if these factors are not controlled. The best way is to monitor diet chart of the patients with dentinal hypersensitivity in order to advise those concerning eating habits.

Smear layer formed immediately after the scaling and root planing prevents the sensitivity of teeth. However, fruit juices containing citric acid removes smear layer and leads to hypersensitivity.

Patient should be cautioned not to brush immediately after exposure of tooth surfaces to dietary acid which greatly increased loss of dentin.

Reduce or Eliminate Parafunctional Habits

Parafunctional habits such as clenching and grinding deliver eccentric occlusal forces to the tooth and leads to tooth flexure resulting in an abfraction and hypersensitivity. The patient should be encouraged to change any parafunctional oral habits that are contributing to recession.

Anti-inflammatory Drugs

It has been suggested that corticosteroids reduce the pulpal inflammation which may be responsible for hypersensitivity. However, more information is needed regarding the relationship between these two conditions.

CONCLUSION

Dentin hypersensitivity is a significant and prevalent issue affecting dental profession. Two conditions-gingival recession and erosive tooth wear—most commonly predispose a patient to suffer the symptoms of dentin hypersensitivity. Differential diagnosis is critically important, followed by a clinically appropriate management plan that also addresses any predisposing conditions. The currently recommended management/treatment continuum for persistent dentin hypersensitivity, depending on severity and extent of pain, should involve the least invasive intervention. In some cases of long-term dentin hypersensitivity, gingival recession and tooth surface loss involve more invasive periodontal and restorative treatments. In all cases with an initial early diagnosis, minimally invasive options should be tried first.

CHAPTER

25 Definitions and Questionnaire for Periodontia Viva Voce

GINGIVA

It is the part of mucosa that covers the alveolar processes of jaws and surrounds the necks of the teeth. A modern definition states that it is the fibrous investing tissue covered by keratinized epithelium that immediately surrounds a tooth and is contiguous with its periodontal ligament and the mucosal tissue of the mouth (AAP2001a).

Marginal Gingiva

Marginal gingiva or unattached gingiva is the terminal edge or border of the gingiva surrounding teeth like a collar (Fig. 25.1).

It not directly attached to the tooth forming the soft tissue wall of the sulcus. It extends 1.0-1.5 mm on facial and lingual from gingival margin to free gingival groove. It has a dull surface and firm consistency.

Free Gingival Groove

It is a fine groove running parallel to the gingival margin (at a distance of 0.5 to 1.5 mm) dividing the free gingival from the attached gingiva, present in 30-40 percent teeth. It appears as a shallow line or depression and often but does not necessarily corresponds to the location of the bottom of the gingival sulcus. It is believed that it arise from functional impacts on the movable free gingiva back on the attached and immovable gingiva (Orban 1948a).

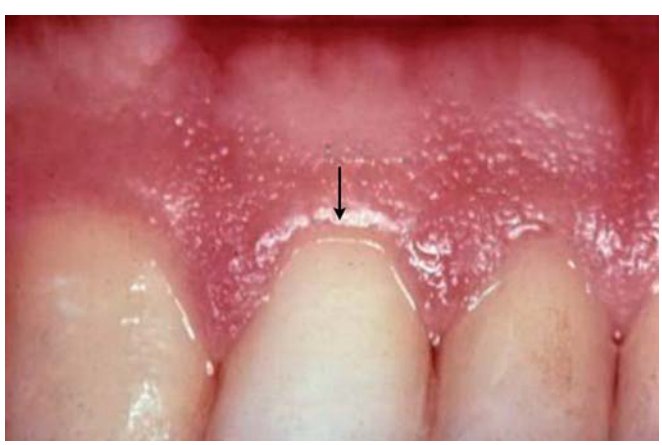


Fig. 25.1: Marginal gingiva

Gingival Sulcus

It is the shallow crevice or space around tooth bounded on one side by surface of tooth and on other side by epithelium lining-free gingiva margin. The depth of the gingival crevice varies according to location, state of eruption, and presence and absence of disease. The average depth of the gingival crevice is usually less than 3 mm; however, it should not be considered abnormal if greater than 2 or 3 mm (Ramfjord and Ash 1979c; AAP2001a).

Attached Gingiva

It is the part of the gingiva that is continuous with marginal gingiva, which is firm, resilient and tightly bound to the periosteum of underlying alveolar bone (Fig. 25.2).

It is frequently stippled, firm, and thick, lacking a submucous layer; and it has no glands. It is located from free gingival groove to mucogingival junction. It is attached to tooth directly in case of dehiscence. The width of facial attached gingival ranges from 0 to 10 mm. It is greatest on the facial surface of the maxillary lateral incisor and narrowest over the facial surface of the mandibular canine and premolars. The dimensions of marginal and attached gingiva are intimately related to each other. In fact, it may be considered as a gingival unit.

Functions

- It acts as an anchor between free gingival groove and alveolar mucosa.
- It dissipates forces from muscle fibers through alveolar mucosa.

Width of Attached Gingiva

It is the distance between the mucogingival junction to the projection on the external surface of the bottom of the gingiva sulcus or periodontal pocket.

The width of attached gingiva is greater in adults than in children (Bowers, 1963). This widening is not linear since the attached gingiva in newly erupted permanent tooth is significantly narrower than that of its deciduous predecessor. It can be concluded that the width of attached gingiva;

– changes concomitantly to changes in the sulcus and crevice depth during eruption and shedding.

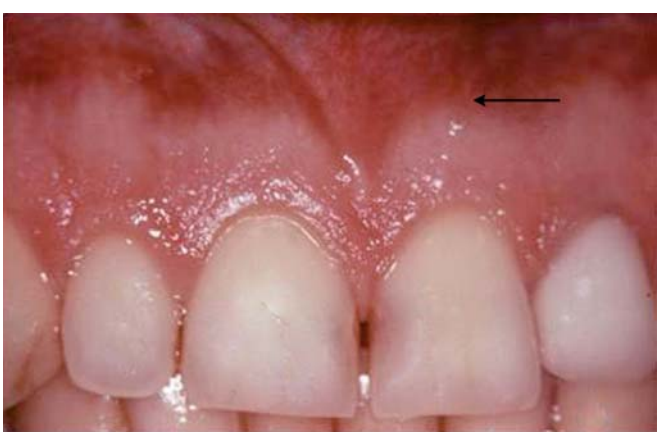


Fig. 25.2: Attached gingiva

- increases with age in the primary dentition.
- is significantly narrower in newly erupted permanent teeth than in their deciduous predecessors.
- increases gradually with the eruption of the permanent teeth—however, in certain teeth it may take many years.

Interdental Gingiva

It is the part of the gingiva, which occupies the gingiva embrasures, e.g. The interproximal space beneath the area of tooth contact.

Interdental Groove

Vertical groove parallel to long axis of adjacent teeth in interdental area of attached gingiva is called as interdental groove.

Desmosomes

Keratinocytes are interconnected by structures on cell periphery called desmosomes.

Keratinosomes or Odland Bodies

Upper most cells of stratum spinosum contain numerous dense granules known as keratinosomes.

Oral or Outer Epithelium

The epithelium, which covers the outer surface and crest of the marginal gingiva and the outer surface of attached gingiva, is called as oral epithelium.

Alveolar Mucose

It has thin, nonkeratinized epithelium lacking retepegs. Its connective tissue is consisting of thin lamina propria and vascular submucosa. It has predominantly elastic fibers, so is loosely bound to underlying bone.

Sulcular Epithelium

Sulcular epithelium lines the gingival sulcus extending from upper limit of junctional epithelium to the crest of gingiva margin.

Junctional Epithelium

It is a stratified squamous nonkeratinized epithelium that extends apically from bottom of sulcus to form color like band around each tooth.

(It is approximately 1 mm in health. Proline/hydroxyproline secreted by epithelial cells bind junctional epithelium to enamel or cementum).

Dentogingival Unit

Attachment of junctional epithelium to the tooth surface is reinforced by gingiva fibers, which firmly braces marginal gingiva against tooth surface. Thus, gingiva fibers and junctional epithelium works together as a functional unit called "DENTOGINGIVAL UNIT".

Cuticle

It is a thin, acellular structure which a homogenous matrix sometimes enclosed with in a clearly demarcated linear borders.

Coronal Cementum

It is a thin layer of homogenous organic material of variable thickness overlying the enamel surface.

Gingiva Fluid or Sulcular Fluid

Fluid that seeps into gingiva sulcus from gingiva connective tissue through the thin sulcular epithelium is called as sulcular fluid. As early as 1817, it was believed that this tissue fluid was secreted by gingival glands, later called Serres glands (Serres 1817). The fluid was soon found to be an inflammatory exudate (Bodecker 1933, 1945).

Gingiva Fibers

The connective tissue of marginal gingiva is densely collagenous, containing a prominent system of collagen fibers bundles called gingiva fibers.

It stabilizes the attached gingiva to alveolar process and to the tooth and it is important in postorthodontic relapse.

Stippling

The portion of gingival outer epithelium appears to be elevated and between them there are shallow depressions corresponding to extensions of connective tissue papilla into epithelium retepegs. This appearance is referred to as stippling. It appears as "matted" in texture, just distance from the gingival margin; being associated with numerous regularly distributed small depressions and giving the tissue "orange peel appearance" (Fig. 25.3) (King 1945; Orban 1948a; Greene 1962; AAP2001a). The degree of stippling varies with individuals and; it had been thought to be an expression of functional adaptation to mechanical impacts. It has been described as "fine" or "coarse", as variable between individuals and ages, and as being finer in females than in males. (Wentz et al., 1952, Rosenberg and Massler, 1967).

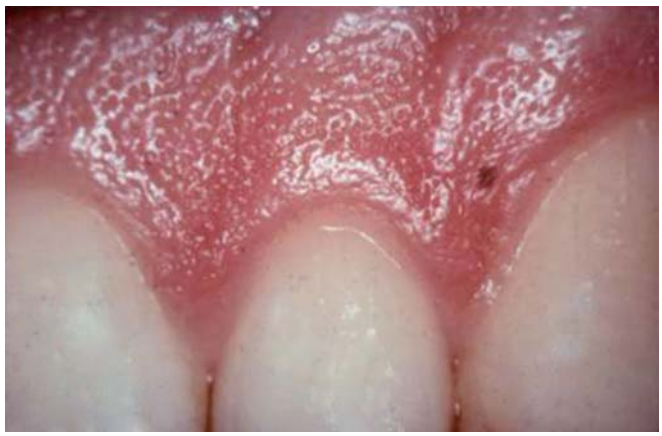


Fig. 25.3: Stippling (Orange peel appearance)

Melanin

Melanin is nonhemoglobin derived brown pigment, which is responsible for normal pigmentation of the skin, gingiva and remainder of the oral mucous membrane.

Anatomic Crown

The portion of tooth, which is covered by enamel is called anatomic root.

Clinical Crown

The part of tooth that has been denuded of its gingiva and projects into oral cavity is known as clinical crown.

Anatomic Root

Anatomic root is the portion of the tooth, which is covered by cementum.

Clinical Root

Clinical root is the portion of tooth, which is covered by periodontal tissue.

Lamina Propria

The connective tissue beneath the gingiva that is made up of two layers: the papillary layer immediately beneath the epithelium, which consists of papillary projections between the retepegs; and the reticular layer which extends to the periosteum.

It is approximately 1 mm between CEJ and crest of bone. The primary cells of lamina propria are the fibroblast.

Papillary Layer

It is the part of lamina propria subjacent to epithelium.

Reticular Layer

It is the part of lamina propria, which is continuous with periosteum of alveolar bone.

Active Eruption

The movement of teeth in the direction of occlusal plane is called active eruption.

Passive Eruption

The exposure of tooth by apical migration of gingiva is called passive eruption.

Gingival Recession

Gingival recession is exposure of tooth by apical migration of gingiva.

Physiologic Recession

Root exposure with increasing age is considered normal and referred to physiologic recession.

Col

Col is a valley-like depression at the tip of the papilla that connects the facial and lingual gingiva just beneath the contact point (AAP 2001a). This term is borrowed from mountaineering, where it is used to describe a ridge-shaped depression adjoining two adjacent peaks (Fish 1961). This is considered as a site of least resistance (locus minoris resistance) to invasion by microorganisms and/or their toxins. (Stahl 1963; Jablonski 1992)

THE TOOTH-SUPPORTING STRUCTURE

Periodontal Ligament

Periodontal ligament is a soft specialized connective tissue situated between the cementum covering the root of tooth and bone forming the socket wall.

It has "hour glass" shape and it is narrowest at mid-root. Fibers run mainly in "*apicocoronal direction*" from cementum to bone inserting as Sharpey's fibers.

Sharpey's Fibers

Terminal portions of the principle fibers of periodontal ligament that insert into cementum and bone are termed as Sharpey's fibers.

Collagen

Collagen is the protein composed of different amino acids, the most important of which are glycine, proline, hydroxylysine and hydroxyproline.

Indifferent Fiber Plexus

In addition to principle fiber types, small collagen fibers also run in all directions forming a plexus that is termed indifferent fiber plexus.

Cementicles

Small foci of calcified masses, which are adherent to or detached from root surface, are called as cementicles.

Cementum

Cementum is a specialized, calcified avascular connective tissue that covers the anatomic root of the tooth and gives attachment to the periodontal ligament.

Acellular Cementum

Acellular cementum is primary cementum and covers approximately the cervical third or half of the root.

Cellular Cementum

Cellular cementum is secondary cementum formed after the tooth reaches the occlusal plane and is more irregular containing cells in individual spaces.

Hypercementosis

It is a regressive change of teeth characterized by the deposition of excessive amounts of secondary cementum on root surface.

Reversal Line

Newly formed cementum is demarcated from the root by a deeply staining irregular line known as reversal line.

Ankylosis

Fusion of cementum and alveolar bone with obliteration of the periodontal ligament is known as ankylosis.

Alveolar Bone Proper

Inner wall of tooth socket is composed of thin compact bone, also known as lamina dura or cribriform plate.

Osteoid

The bone matrix that is laid down by osteoblasts and which is not mineralized is known as osteoid.

Bundle Bone

Bone adjacent to periodontal ligament that contains a great number of Sharpey's fibers is called as bundle bone.

Periosteum, Endosteum

The tissue covering the outer surface of bone is called as periosteum while endosteum is the tissue lining the internal bone cavities.

Fenestrations

Isolated areas in which the root is denuded of bone and the root surface is covered only by periosteum and overlying gingiva are called fenestrations. The term is derived from Latin word fenestra, meaning "window". In periodontology, it has been described as a window-like aperture or opening in alveolar bone over the root of tooth with intact marginal bone (AAP 2001a). Fenestration is more common in the maxilla.

Dehiscence

The absence of alveolar cortical plate sometimes exceeding more than half of the root length and often results in a denuded root surface (Elliott and Bowers 1963; Larato 1970d). When denuded areas extend through the marginal bone, the defect is called dehiscence.

Dehiscences are more common in the mandible (usually bilateral). Do not perform scaling and root planing in the particular area with dehiscence. Sharpey's fibers are not removed and left in their intact position, if the area is healthy.

Pre-cementum or Cementoid

A meshwork of irregularly arranged collagen fibrils sparsely distributed in an irregularly arranged ground substance or matrix is called precementum.

Intermediate Plexus

Individual fibers of periodontal ligament, rather than being continuous, consist of two separate parts spliced together midway between cementum and bone in a zone called intermediate plexus.

Physiologic Mesial Migration

Physiologic mesial migration is mesial movement of tooth due to flattening of proximal areas with aging and wear.

Trauma From Occlusion

Injury results when forces exceed adaptive capacity of periodontium are called trauma form occlusion.

Dental Epidemiology

It is the study of the pattern and dynamics of dental diseases in human population.

Epidemiologic Indices

Indices is a numerical value describing relative status of population on a graduated scale with definite upper and lower limit, which is designed to permit, and facilitate the comparison between same group/other groups described by same criteria and method.

EPIDEMIOLOGY OF PERIODONTAL DISEASE AND GINGIVAL DISEASE

Dental Plaque

It is a soft deposit that forms the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity, including removable and fixed prosthesis.

It is the highly variable structural entity resulting from the colonization and growth and sequential maturation of microorganisms on the surfaces of teeth, restorations, and oral soft tissues and consisting of a number of microbial species and strains embedded in an extracellular matrix.

It is defined by the AAP as 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. Other components include an organic polysaccharide protein such as enzymes, food debris, desquamated epithelial cells, and inorganic components such as calcium and phosphate (AAP 2001a).

Materia Alba

Materia alba is the yellowish or grayish-white, soft sticky deposits containing food debris, desquamated tissue cells, bacteria, leukocytes and salivary proteins which lacks the organized structure of plaque and can be easily displaced with a spray of water. It serves as a medium of bacterial growth.

PERIODONTAL MICROBIOLOGY

Supragingival Plaque

Plaque found at or above the gingival margin is called as supragingival plaque.

Marginal Plaque

Supragingival plaque that is in direct contact with the gingival margin is called as marginal plaque.

Subgingival Plaque

Plaque found below gingival margin between tooth and gingival sulcular tissues is called as subgingival plaque.

Co-aggregation

Ability of different species and genera of plaque microorganism to adhere to one another by a process known as co-aggregation.

Chemotaxis

It is the direct movement of a cell along chemical gradient.

Opsonization

Opsonization refers to process of coating a particle with recolonization molecules to enable phagocytic ingestion.

DENTAL CALCULUS

Calculocementum

Calculus embedded deeply in cementum may appear morphologically similar to cementum termed as calculocementum.

Stains

Pigmented deposits on the tooth surface are called stains.

ROLE OF IATROGENIC AND OTHER LOCAL FACTORS

Food Impaction

Food impaction is the forceful wedging of food into the periodontium by occlusal force.

Plunger Cusps

Cusps that tend to forcibly wedge food interproximally are known as plunger cusps.

DENTAL OCCLUSION

Occlusion

Occlusion is the contact relationship of teeth in their functional state under the neuromuscular control of the masticatory system (Musculature, TMJ, mandible, periodontium).

Physiologic Occlusion

It is an occlusion that exists in an individual who has no signs of occlusion-related pathosis.

Traumatic Occlusion

It is an occlusion associated with traumatic lesions or disturbances in supporting structures of teeth, muscles and TMJ.

Therapeutic Occlusion

It is an occlusion used to counteract structural interrelationships-related to traumatic occlusion.

Border Movements

Border movements are the limits to which the mandible can move in any direction and are not common during mandibular function.

Contact Movements

Contact movements are the movement of the mandible with one or more of the opposing occlusal surfaces in contact.

Intraborder Movements

Any mandibular movement within the perimeter of border movement is called as intraborder movements.

Excursions

Excursion is the movement of the mandible with the teeth in contact.

Bennett Movement

Lateral shift of condyle of mandible is called Bennett movement.

Physiologic Rest Position

When the teeth are not in contact in mastication, swallowing or speech, the lips are at rest and jaws are apart. This is called physiologic rest position.

Free Way Space

The space between the mandible and maxillary teeth when the mandible is in the postural position is called as free way space.

Masticatory or Chewing Cycle

The pathway of mandible in chewing is referred to masticatory cycle.

Supracontact

It is general term for any contact hindering the remaining occlusal surfaces from achieving many pointed, stable contacts.

There are two types:

1. Intercuspal supracontacts
2. Retrusive supracontacts

Vertical Dimension of Occlusion

Vertical dimension is the distance between maxilla and mandible when teeth are in intercuspal position.

Attrition

Attrition is the physiologic wearing of tooth surface as a result of tooth-to-tooth contact, as in mastication. It primarily occurs on the incisal and occlusal edges of the teeth as wear from tooth-to-tooth functional contact (e.g. buccal and lingual surfaces of anterior teeth in deep overbite relationship). Parafunctional attrition is often related to excessive grinding motions. Reduced salivary flow may contribute to tooth wear as a result of increased tooth-to-tooth friction.

It is characterized by:

- Matching wear of the occluding surfaces of teeth
- Shiny facets on amalgam contacts
- Enamel and dentin wear at the same rate
- Possible fracture of cusps of restorations.

Facets

Occlusal or incisal surfaces worn by attrition are called facets.

Erosion

Erosion is the loss of tooth structure by means of some chemical action that does not involve known as bacterial action. It is an apparent chemical dissolution of enamel and dentin, unrelated to caries, causing a cavity that has hard, smooth base. It is most commonly produced by excessive and frequent intake of an acidic diet and less commonly by the reflux of hydrochloric acid from the stomach. Acids demineralize and soften the enamel and dentin surfaces, making them more susceptible to abrasion, particularly by toothbrushing. Dietary acids and gastric acids are two most common causes of dental erosion. Dietary erosion is most commonly observed as moderate, generalized erosion of the cervical and buccal surfaces of the maxillary teeth and the occlusal and buccal surfaces of the mandibular teeth. Gastric erosion is more severe than dietary erosion. It is commonly observed on the lingual aspects of anterior and posterior maxillary teeth, buccal aspects of mandibular posterior teeth.

It is characterized by:

- Broad cavities within dull, smooth surface enamel
- Cupping of occlusal surfaces (incisal grooving) with dentin exposure
- Increased incisal translucency
- Wear on nonoccluding surfaces
- “Raised” occlusal restorations
- Clean, nontarnished appearance of amalgams
- Loss of surface characteristics of enamel in young children
- Preservation of enamel “cuff” in gingival crevice is common
- Dentin hypersensitivity
- Pulp exposure in deciduous teeth.

Frictional Ablation

It is the process caused by juxtaposition of natural and artificial dental surfaces and hyperfunctional oral soft tissues.

Abrasion

Abrasion is the pathologic wearing of tooth surface by some mechanical forces such as hard bristle toothbrush, coarse tooth powder, etc. Clinically, they are ‘V’ or wedge-shaped defects and notches at or apical to the CEJ.

It is characterized by:
- Lesions that are more wide than deep
- Premolars and cuspids are commonly affected
- Usually located at cervical areas of teeth

Some factors affecting the tooth abrasion are:
- Brushing technique
- Frequency of brushing
- Amount of time spent for brushing
- Force applied during the brushing
- Type of toothbrush
- Abrasiveness and amount of dentifrices used
- Personal habits, such as finger nail of thread biting and holding foreign objects with the teeth (pipe smoking, seed shelling), may cause incisal or occlusal abrasion.

Abfraction

Abfraction is a noncarious cervical lesion hypothesized to be the result of microfractures in enamel and dentin caused by eccentrically applied occlusal parafunctional forces. For example, during bruxism, the changing back and forth direction of the occlusal forces bends the tooth, causing side to side fatigue and microfracturing at the most flexed area in the cervical region of the tooth.

It is characterized by:
- Deep, narrow, V-shaped notch
- Affects buccal and labial cervical areas of teeth
- Commonly affects single tooth with excursive interferences or eccentric occlusal loads.

Bruxism

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

INFLUENCE OF SYSTEMIC DISEASES ON PERIODONTIUM

Scurvy

It is the disease characterized by hemorrhagic diathesis and retardation of wound healing caused by severe vitamin C deficiency.

Leukemia

It is a disease characterized by the progressive overproduction of WBC, which usually appear in the circulatory blood in an immature form.

AIDS

It is the disease indicative of defect in cell-mediated immunity occurring in a person with no known cause for immunodeficiency other than positive of HIV.

CLINICAL FEATURES OF GINGIVITIS

Acute Gingivitis

Acute gingivitis is a painful condition that comes on suddenly and is of short duration. Subacute gingivitis is the less severe phase of acute condition.

Chronic Gingivitis

Chronic gingivitis is of long duration which is slowly progressing and is painless unless complicated by acute or subacute exacerbations.

Recurrent Gingivitis

Gingivitis which having been eliminated by treatment and then reappears again is called as recurrent gingivitis.

Gingival Clefts

It is a narrow type of gingival deformity encountered on the marginal aspects of the gingiva and extending regularly or irregularly from the gingival margin in an apical or lateral direction, with margins that are usually thick and often rolled inward (Moskow and Bressman 1965).

Stillman's Clefts

The gingival clefts have been defined as "small apostrophe-shaped fissures extending apically from gingival margin for varying distances (usually 5 to 6 mm), usually on the vestibular surface of teeth (Jablonski 1992). They were originally considered to be due to regional circulatory disturbances within the gingiva initiated by traumatic occlusion (Stillman 1921, Stillman and Mc Call 1937). However, researches have shown that there is no evidence that such defects are brought about by occlusal disharmonies (Posselt 1959). They may be considered as epithelial down growths leading to splitting of the gingiva (AAA2001a).

McCall's Festoons

They are 'life preserver'-shaped enlargements of the marginal gingival that occur primarily on facial surfaces of anterior teeth and bicuspids. These semilunar enlargements were named after John Oppie McCall, who along with Paul R Stillman, believed occlusal trauma as an etiological factor (Stillman and McCall 1922). But later studies couldn't substantiate or show evidence that festoons was initiated by occlusal disharmonies (Posselt 1959).

GINGIVAL ENLARGEMENT

Gingival enlargement is increase in the size of gingiva so that soft tissue overfills the interproximal spaces balloons out over the teeth and protrudes into the oral cavity.

Granuloma Pyogenicum

It is the tumor-like gingival enlargement that is considered as an exaggerated conditioned response to minor trauma.

Epulis

It is a generic term used clinically to designate all discrete tumors or tumor-like masses of the gingiva.

ACUTE GINGIVAL INFECTIONS

Acute Necrotizing Gingivitis (ANUG)

It is an inflammatory destructive disease of the gingiva that presents characteristic signs and symptoms.

Vincent's Angina

It is a fusospirochetal infection of the oropharynx and throat.

Pericoronitis

It refers to inflammation of gingiva in relation to crown of an incompletely erupted tooth. It is also known as operculitis.

DESQUAMATIVE GINGIVITIS AND ORAL MUCOUS MEMBRANE DISEASES

Desquamative Gingivitis

It is a unique condition of gingiva characterized by intense redness and desquamation of the surface epithelium.

Lichen Planus

Lichen planus is an inflammatory disease of skin and mucous membranes characterized by the eruption of papules.

Leukoplakia

Leukoplakia is a nonscrapable white lesion occurring in oral cavity, which cannot be classified clinically or histopathologically under any other. It is usually more than 5 mm in size.

Pemphigus

Pemphigus is a serious chronic autoimmune disease involving skin and mucous membrane characterized by the appearance of vesicle and bulla, small or large fluid filled blisters that develop in cycles.

Erythema Multiforme

It is an acute inflammatory eruptive disease of unknown etiology involving the skin and oral cavity.

Scleroderma

Scleroderma is the multisystem connective tissue disease that involves hardening of skin and mucosa smooth muscle atrophy and fibrosis of internal organs.

PERIODONTAL POCKET

Eruption Gingivitis

It is gingivitis associated with tooth eruption.

Periodontal Pocket

Periodontal pocket is a pathological deepened gingival sulcus due to coronal growth of gingiva or apical migration of junctional epithelium.

Gingival Pocket (False/ Pseudopsocket)

Gingival pocket is a pocket, which is formed by gingival enlargement without any destruction of underlying periodontal tissue.

True Periodontal Pocket

It is a type of pocket occurs with destruction of the supporting periodontal tissue.

Suprabony Pocket

Pocket with the bottom coronal to the underlying alveolar bone is also called supracrestal or supraalveolar pocket.

Infrabony Pocket

If the bottom of the pocket is apical to the level of the adjacent alveolar bone, it is also called subcrestal or intraalveolar pocket.

Spiral Pocket

Pocket which is originating on one tooth surface and twisted around the tooth to involve one or more additional surfaces.

Periodontal Abscess

Periodontal abscess is a localized accumulation of pus in periodontal tissue. It is also known as lateral or parietal abscess.

BONE LOSS AND PATTERN OF BONE LOSS

Exostoses

Exostoses are over growths of bone of varied size and shape.

Buttressing Bone Formation

Bone formation sometimes occurs in an attempt to buttress bone trabeculae weakened by resorption.

Two Types

CENTRAL: when occurs within jaw.
PERIPHERAL: when occurs on external surface.
When it causes bulging of the bone contour, it is termed as "*LIPPING*".

Osseous Craters

Osseous craters are concavities in the crest of the interdental bone confined to the facial and lingual walls.

Bulbous Bone Contours

They are bony enlargement caused by exostoses, adaptation to function or buttressing bone formation.

Reversed Architecture

A morphological relationship when the interdental gingival or bone is located apical to midfacial and midlingual margins (AAP 2001a).

Ledges

They are plateau like bone margins caused by resorption of thickened bony plates.

Furcation Involvement

Invasion of the bifurcation and trifurcation of multirooted teeth by periodontal diseases is known as furcation involvement.

PERIODONTAL RESPONSE TO EXTERNAL FORCES

Pathologic Migration

Pathologic migration refers to the tooth displacement those results when the balance among the factors that maintain physiologic tooth position is disturbed by periodontal disease.

Refractory Periodontitis

Cases that for unknown reasons therapy and/or recur soon after adequate treatment has been referred to as refractory periodontitis.

PREPUBERTAL AND JUVENILE PERIODONTITIS

Juvenile Periodontitis

The term is used to describe disease of periodontium in the preadult years (11-19).

Papillon-Lefevre Syndrome

This syndrome is characterized by hyperkeratotic skin lesion; server destruction of the periodontium and in some cases calcification of the dura.

Down's Syndrome

Down's syndrome is a congenital disease caused by chromosomal abnormality and characterized by mental deficiency and growth retardation.

OTHER

Diagnosis

Diagnosis is determination of the nature of a case of a disease.

Lamina Dura

Radiopaque appearance of an alveolar bone proper on radiograph is called as lamina dura.

Prognosis

It is a prediction of the duration course and termination of a disease and its response to treatment.

Treatment Plan

It is the blue print for a case a management.

RATIONALE FOR PERIODONTAL TREATMENT

Regeneration

Regeneration is the growth and differentiation of new cells and intracellular substances to form new tissues or parts by growth from the same type of tissue that has been destroyed (Jablonsky 1992). It is the biologic process by which the architecture and function of lost tissue is completely restored.

It results in regrowth of precise tissue that were present before the damage or disease occurred.

Reattachment

The modern definition for reattachment is "to attach again, the reunion of connective tissue with a root surface on which viable periodontal tissue is present, the area of reattachment is not affected by bacterial contamination" (Mellonig 1992; AAP 2001a). It is the reunion of the connective tissue and root that have been separated by incision or injury but *not by disease*. It is frequently necessary to move healthy tissue away from the tooth root or bone temporarily during some types of periodontal surgery to allow access to damaged parts of the periodontium on adjacent teeth. The expected healing in this type of incision is by reattachment.

New Attachment

New attachment is the term used to describe the union of a pathologically exposed root with connective tissue or epithelium.

New attachment occurs when the epithelial and connective tissues are newly attached to a tooth root *where periodontitis had previously destroyed the attachment.* It differs from the reattachment because new attachment must occur in an area formerly damaged by disease, whereas reattachment occurs when tissues are separated in the absence of disease.

Repair

Repair is healing of wound by formation of tissue that does not truly restore the original architecture or original function. It is healing by scar tissue formation. Certainly the healing is complete following formation of scar during the healing of scar, but the tissue is not precisely the same tissue in appearance or function that excised on the part before the wound.

PRINCIPLE OF PERIODONTAL INSTRUMENTATION

Adaptation

Adaptation refers to the position of the first 1-2 mm of the lateral surface of instrument in contact with the tooth.

Angulations

Angulations refer to the angle between the face of a bladed instrument and tooth surface.

Lateral Pressure

Lateral pressure refers to the pressure created when force is applied against the surface of a tooth with the cutting edge of bladed instrument.

Scaling

Scaling is the process by which plaque and calculus are removed from both supragingival and subgingival tooth surfaces.

Root Planing

Root planing is a periodontal process by which residual embedded calculus and portion of cementum contaminated with microorganisms and toxins are removed from the root to produce a smooth hard clean surface.

Probe

Probe is a slender, blunt ended, tapering assessment instrument used to evaluate the health status of the periodontal tissues.

Probing

Probing is the act of walking the tip of probe along the junctional epithelium within the sulcus or pocket for the purpose of assessing the health status of the periodontal tissues.

Probing Depth

It is the measurement of the depth of a sulcus or periodontal pocket. It is determined by measuring the distance from the gingival margin to the base of the sulcus or pocket with a calibrated periodontal probe.

PLAQUE CONTROL

Plaque control is the removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival surface.

Dentifrices

It is the therapeuticomechanical aid for cleaning and polishing tooth surfaces.

Disclosing Agent

These are the solution or wafers capable of staining bacterial deposits on the surface of teeth, tongue and gingiva for instruction, evaluation and research.

Cavitation

Cavitation refers to formation of tiny bubbles in the water exiting form the electronically powered instrument tip. When these tiny bubbles collapse, they produce shock waves that destroy the bacteria by tearing the bacterial cell walls.

Periodontal Debridement

It refers to the removal or disruption of bacterial plaque, its products, and the plaque retentive calculus deposits from coronal surfaces, root surfaces, and from within the pocket. Periodontal debridement includes instrumentation of every square millimeter of root surface for plaque and calculus removal, but does not include the deliberate, aggressive removal of cementum.

QUESTIONNAIRE FOR PERIODONTIA VIVA VOCE

Q.1. What is the action of gingival massage?

Ans: Gingival massage increases the mitotic activity in the epithelium and connective tissue. The increased keratinization occurs only on the oral gingiva not on the areas vulnerable to microbial attack like sulcular epithelium and interdental areas (gingival col). Epithelial thickening, increased keratinization and increased blood circulation have not been shown to be beneficial for restoring gingival health. Improvement in gingival health is more likely due to plaque control rather than gingival massage.

Q.2. Can gingival massage with gum paint be performed in presence of local deposits?

Ans: No, gingival massage with gum paints can't be carried out for longer duration or regularly because in the presence of calculus or plaque massaging will cause friction between the calculus and the gingiva underneath deposits causing the laceration.

Q.3. What does gum paint contain?

Ans: Contents of traditional gum paint are:

	W/V
Potassium iodide	0.8%
Iodine	0.6%
Tannic acid	2.0%
Menthol	0.5%
Thymol	0.5%
Camphors	0.5%
Zinc phenol sulphonate	0.8%
Menthe oil	0.3%
Clove oil	0.3%
Phenol	0.2%
Glycerin	Q.S

Q.4. At which stage you will say that gingivitis has progressed to periodontitis?

Ans: Theoretically, in the advance stage of gingivitis, the inflammatory changes cause degeneration of connective tissue wall of the gingival sulcus. As consequences, collagen fibers just apical to junctional epithelium are destroyed and apical cells of junctional epithelium proliferate on the root. With migration of the apical portion of the junctional epithelium, coronal portion detaches from the root. This stage of periodontal breakdown marks the transition from normal gingival sulcus to pathogenic pocket.

Q.5. What is the interaction if bactericidal and bacteriostatic drugs are prescribed together?

Ans: Depending on mechanism of antibiotic action, combinations of antibiotics may have an additive, synergistic, antagonistic effect. Static antibiotics used in combinations generally exhibit an additive effect, bactericidal antibiotics exhibit a synergistic effect, and combination of bactericidal and bacteriostatic agents exhibits antagonistic effect.

Bactericidal drugs are effective only during the multiplication of the bacteria which is inhibited by bacteriostatic drug. So when we are prescribing a bacteriostatic drug, multiplication of bacteria will ultimately stop and it will make bactericidal drug less effective.

Q.6. How to find lesion is primary of endodontic or periodontal origin in endoperio-lesion and what are the treatment strategies?

Ans: The differential diagnosis and treatment strategies of the endoperio lesion are demanding.

Differential diagnosis:

Factors to be considered are:

- Vitality of pulp
- Sequencing of clinical symptoms like pain and swelling and clinical signs like probing depth.
- Location, form and extension of the radiolucency (Radiographic observation).

Pulp vitality test is of prime importance in differential diagnosis.

i. Presence of deep carious lesion, nonvital pulp and periapical radiolucency with pocket not approximating the apex indicates the endodontic cause as a prime factor.

ii. Deep periodontal pocket approximating the apex, vital pulp and no carious involvement of tooth indicates the lesion is of primarily periodontal origin.

However, in many cases, the overlapping signs and symptoms may create dilemma of accurately assessing the contribution of endodontic or periodontal cause.

	Pulpal lesions	*Periodontal lesions*
Vitality	Nonvital	Vital
Usual area of swelling	Vestibule	Attached gingiva
Pain	Intermittent throbbing	Dull, Continuous
Probing	Narrow, isolated defect	Non-isolated defect
Sinus tract location	Mucosa	Attached gingiva, Sulcus
Radiograph	Localized bone loss	Generalized bone loss of area
Local factors	Variable	Calculus
Etiology	Deep caries or restoration	Possibly non-restored

Treatment Strategies

It is clinically not possible to determine the extent to which endodontic or periodontal lesion has affected the supporting tissues. Therefore, the treatment strategy for combined endoperio lesion must be first focused on the pulpal infection. Thorough endodontic treatment (phase-I) will resolve a part of lesion contributed by pulpal infection. So the phase-I is followed by the period of observation (phase-II) to observe the healing resulting from the endodontic treatment. Improvement in probing depth can be observed within couple of weeks, while bone regeneration may require several months. So, the periodontal treatment (phase-II) is postponed until the proper evaluation of endodontic treatment.

If the periodontal disease is solely responsible for the combined endoperio lesion, the endodontic treatment will not contribute to the periodontal healing as an endodontic lesion being secondary involvement. In this case, the primary approach is periodontal therapy. If primary lesion is of endodontic origin, the endodontic treatment should be delivered first.

Whenever, there is a doubt regarding the source of origin (endodontic/periodontal) of combined endoperio lesion, following guidelines should be followed.

Cause	*Condition of pulp*	*Treatment*
Endodontic	Nonvital	Endodontic
Periodontal	Vital	Periodontal
Endodontic/periodontal	Nonvital	First – endodontic treatment Second – phase of observation Third – periodontal treatment, if necessary

Q.7. What are the different methods of diagnosis of infrabony defect?
Ans: There are three main methods.
- Transgingival probing
- Radiographs with radiopaque markers
- Surgical opening of flap is best to reveal the configuration of defect.

Q.8. Can the actual configuration of bony defect whether 3-walled or 2-walled be diagnosed radiographically?
Ans: No. Sometimes radiographs can make it possible to predict the bony defect but its accurate diagnosis by visual examination requires surgical exposure of flap.

Q.9. Difference between acanthosis and acantholysis?
Ans: Acanthosis—Thickening of spinous cell layer.
Acantholysis—Intraepithelial clefting by autoantibody to desmosomes, which connects two cells. It is seen in pemphigus.

Q.10. What is the pathologic potential of calculus?
Ans: Calculus acts as a contributing factor for plaque formation and promotes its retention on teeth. Also, the rough surface of calculus is usually covered by a layer of plaque. It interferes with the efforts of improved plaque control and keeps plaque in the vicinity of the periodontal tissues all the time. The persistent inflammation around the free calculus itself is the unambiguous evidence of the pathologic effect of calculus.

Q.11. What does pus indicate?
Ans: There is tendency to overemphasize the importance of purulent exudates and to associate it with severity of periodontal disease. Pus indicates ongoing suppurative process. It is the evidence that no necrosis has taken place and cells are living which exudates pus. Pus is a common feature of periodontal disease but it is only a secondary sign. *Pus merely reflects the nature of the inflammatory changes in the pocket wall. It is not an indication of the depth of pocket or severity of destruction of the supporting tissues. Extensive pus may occur in shallow pockets, whereas deep pockets may exhibit little or no pus*. Pus formation indicates the period of exacerbation while its absence reflects the period of quiescence.

Clinically, the presence of the pus can be determined by gently applying digital pressure by ball of index finger along the lateral aspect of marginal gingiva in a rolling motion from base of pocket towards the crown.

Visual examination without digital pressure may be insufficient as the purulent exudate is formed from the lateral wall of pocket.

Q.12. How to determine mucogingival junction?
Ans: Various methods to determine the mucogingival junction are:
- Attached gingiva is tightly bound to underlying bone which extends to the relatively loose and movable alveolar mucosa. The side of instrument (probe tip) is passed from marginal gingiva apically, a feel of tag or stuck of instrument marks the mucogingival junction (Fig. 25.4).

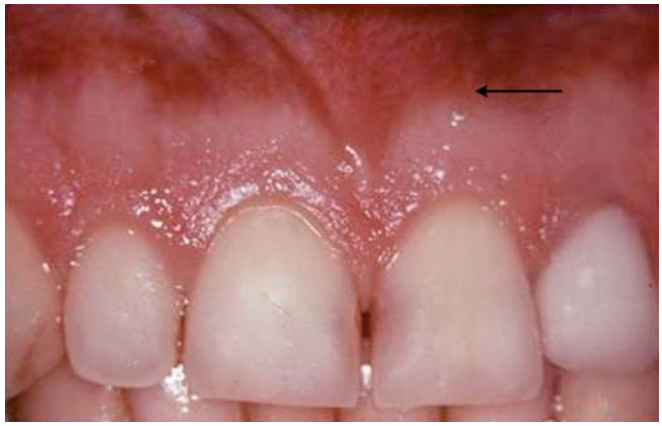


Fig. 25.4: Mucogingival junction

- By using "Schiller's solution" containing potassium iodide, keratin can be stained. Iodine binds to protein of keratinized surface. As being keratinized, iodine stains the attached gingiva and demarcates it from nonkeratinized alveolar mucosa.
- Another method to demarcate the mucogingival junction is pushing the lip (cheek) coronally.

Q.13. What is red complex and what is its significance?
Ans: Red complex consists of *B. forsythus*, *P. gingivalis,* and *T. pallidum* [BPT]. This red complex is most commonly related to bleeding on probing. (Remember other complexes also).

Q.14. What is the reason for high prevalence of horizontal bone loss in anterior region and vertical defects in posterior region?
Ans: Bacterial plaque can induce bone loss within the radius of 1.5-2.5 mm. There is no effect beyond 2.5 mm *(Radius of action)*. For that reason, interproximal angular defects can appear only in spaces that are wider than 2.5 mm because narrow spaces would be destroyed completely. Defects extending a distance of 2.5 mm from the tooth surface may be due to presence of bacteria in tissue. In anterior region, interdental septum is not as wide as that in posterior segment. This results in horizontal type of bone loss in anterior segment and vertical defects in posterior segment.

Q.15. What is native bone level?
Ans: Native bone level is the alveolar crest level in the absence of any pathologic loss. The constant distance between the lower edge of junctional epithelium and the alveolar crest is 1.07 mm.

Q.16. What are the basic parameters for selection of antibiotics in periodontia?
Ans: Selection of antibiotic is customized for patient-to-patient and it depends on many factors. But the empirical therapy for the patient with gingival and periodontal problems is as below:
If gram-positive organisms are suspected, penicillin is preferred antibiotic group. If the patient is allergic to penicillins, macrolides are second choice followed by fluoroquinolones. If gram negative organisms are anticipated, imidazole derivatives (metronidazole/tinidazole) are effective.

Q.17. What are Human leukocyte antigens (HLA)?
Ans: Human leukocyte antigens found on chromosome-6 and transmitted by heredity. They regulate immune responses and have been associated with various autoimmune diseases.

Q.18. How the Fremitus test is performed?
Ans: Periodontal fremitus (vibrations or tremors) occurs in either of the alveolar bones when an individual sustains trauma from occlusion. Fremitus test is the measurement of the vibratory pattern of teeth when teeth are placed in occlusion and also during movements. It is a result of teeth exhibiting slight mobility against the adjacent walls of their sockets. An index finger (alternatively two fingers can also be placed vertically) is placed horizontally along the labial vestibule against the alveolar bone to detect the fremitus. Fremitus may be detected both in centric occlusion and in lateral excursive movements (lateral fremitus). Patient is asked to close his or her mouth into maximum intercuspation and is asked to grind his or her teeth slightly. The volume of vibrations is expanded by inflammatory responses, bone resorption or both. If trauma from occlusion is present, it leads to abnormal mobility; vibrations are felt at finger. Assessment of fremitus involves measurement of vibratory patterns of teeth when teeth are placed in contacting position and movements. It is easier to detect fremitus of maxillary teeth than the mandibular teeth. Fremitus is recorded as $\pm$. There is no numerical fremitus scale.

Class I	Mild vibration
Class II	Easily palpable vibration but not visible movement
Class III	Movement visible with naked eye

Q.19. Should any commercially available alcohol-based mouthwash be prescribed in periodontitis?
Ans: Ethyl alcohol is commonly used both to stabilize certain active ingredients and to improve the shelf-life of the product. In periodontitis, alcohol-based preparations causes drying of mucosa and pocket content and aggravates the condition and halitosis.
(Mouthwashes containing more than 25 percent alcohol could increase the risk of oral and pharyngeal (throat) cancer by 50 percent. Most, if not all, mouthwashes in the market contain less than 15 percent alcohol.)

Q.20. Are stains harmful?
Ans: Yes. Stains are harmful as they produce rough surface and serve as a plaque retentive factor.

Q.21. What do you mean by cleft and what's its significant?
Ans: Cleft is break in continuity of gingival margin. Presence of cleft definitely indicates recession.

Q.22. Why mobility increases immediately after surgery?
Ans: Body responds to an infection and trauma by developing inflammation. Immediately after surgery, mo bility increases temporarily as a result of inflammation of the periodontal ligament fibers in response to the surgical trauma. Raising flap detaches the connective tissue fibers from tooth surface. Resolution of inflammatory changes and subsequent bone condensation contribute to a significant extent. Also, regeneration and reformation of connective tissue fibers during healing along with decrease in the inflammation diminishes the tooth mobility below the pre-treatment level by fourth week.

Q.23. What is the change in the marginal gingiva in inflammation?
Ans: Normal knife-edge gingival margin becomes rolled out due to inflammatory changes making oral hygiene practice more difficult to perform.

Q.24. What is the cause of spontaneous or increased bleeding at morning in absence of systemic diseases but in the presence of gingival inflammation?
Ans: Salivary flow carrying anticoagulant factors as well as antimicrobial agents is reduced during the night. In addition, the absence of mechanical cleansing by tongue exacerbates bacterial activity in presence of inflammation. All these together aggravate the existing inflammation and undisturbed growth of bacteria produces vascular engorgement and thinning of epithelium leading to bleeding.

Q.25. What is the observation in pre- and post-treatment radiograph in periodontal therapy?
Ans: Periodontal reconstruction without use of bone graft shows little less bone level compare to pretreatment radiograph. During surgery, raising the mucoperiosteal flap from the bone, temporarily decreases blood supply and increases osteoclastic activity. As the main blood supply for alveolar bone is the mucoperiosteum, cessation of blood supply leads to decrease (little) in bone level. As healing progresses, it comes to normal level. In order to determine whether the bone loss is progressing or not, standardized radiographs are taken at regular interval and analyzed. Clinical evaluations by radiographs at 6 to 8 months help to decide the disease course.

While is case of periodontal flap surgery with bone grafting, the posttreatment change in bone level depends on the amount of periodontal regeneration by graft material.

Q.26. What is the fate of regenerative bone graft procedure in case of horizontal and vertical bone loss?
Ans: Bone grafting in horizontal bone loss have not responded well to bone fill regenerative procedures. Lack of stability of the grafting material and the susceptibility to wash out makes the procedure futile. These areas are best treated with pocket elimination flap procedure with or without osseous resective surgery.

Vertical defects especially those with two or three walls, respond well to bone fill regenerative procedure. When used in intrabony defects, easy adaptation of bone graft material in angular defect provides more resistance to washing off effect of expensive material by saliva and maximum benefit of grafting material is achieved.

Q.27. What are the limitations of radiographs as a diagnostic aid in periodontia?
Ans:
- Inorganic content of bone should be decreased by 30 percent to 60 percent to become radiographically evident. So after initiation, any bone changes require around 6 to 8 months to be radiographically evident.
- Actual destruction is more than that appear on the radiograph so it may lead to misinterpretation of actual condition.
- Radiographs are two-dimensional representation of a three-dimensional anatomy.
- Radiograph cannot determine the activity of disease.

Q.28. Effects of unilateral chewing habit.

Ans: Patients who are unilateral chewer, more function on one side and less function on another side. The used side shows increased bone density (trabeculation), widened periodontal ligament space and increased wearing of tooth whilst the unused side shows decreased in bone density, narrow ligament space and more plaque and calculus accumulation.

Q.29. Effect of cigarette smoking on periodontium.

Ans: Smoking is an important established environmental risk factor in periodontitis. It can affect the vascular, inflammatory, immune and healing responses. Nicotine is main factor responsible for the dependence-forming properties of tobacco smoking. Cotinine is a metabolite of nicotine. The word 'cotinine' is an anagram of 'nicotine'. The degree of exposure to tobacco smoking can be measured in pack years or by measuring the serum nicotine and cotinine levels.

Effects on vasculature: Smoking induced vasoconstriction could contribute to impaired gingival flow and decrease the amount of oxygen and blood constituents that reach the gingiva, whilst also reducing the capacity to remove tissue waste products, leading to damage and / or compromised immune response. It alters the ability of hemoglobin to transport the oxygen and creates the serious environmental risk factor for periodontal diseases. Smoking suppresses the gingival inflammation and simultaneously the underlying periodontal disease activity continues without manifestations of inflammation. Healing is also impaired because of depleted nutrition and oxygen by diminished blood supply due to vasoconstriction.

Effects on immune system: Smoking has major effect on the protective elements of the immune response, resulting in an increase in the extent and severity of periodontal disease. Alteration in neutrophil number or function affects the critical functions such as chemotaxis and phagocytosis. Impaired response of neutrophils to periodontal infection and increased release of tissue-destructive enzymes reduces the protection against periodontal infections.

It has been shown that the suppressed inflammatory response is reversible on smoking cessation. In addition, reduction in smoking may have a beneficial effect on associated periodontal disease. It is been observed that periodontal destruction is more in smokers compared to non-smokers or former smokers. The extent of smoking exposure and the dose may also increase the smoking associated risk of periodontal disease.

Q.30. Why necrosis starts with interdental papilla in case of ANUG?

Ans: Interdental papilla contains end arteries which are the terminal branches of artery and there is no collateral circulation at the tip of papilla. So any interference in the blood supply makes the area deprived of nutrition and makes it prone to necrosis.

Q.31. What are the indications of surgical pocket therapy?

Ans: In moderate and deep pockets this surgery is indicated for:

- To increase the accessibility to the root surface, making it possible to remove all irritants.
- Reduce or eliminate pocket depth, making it possible for the patient to maintain the root surfaces free of plaque
- Reshape soft and hard tissues to attain a harmonious topography.

Q.32. How will you classify periodontal surgeries?
Ans: Periodontal surgeries are classified as follows:

Pocket reduction surgery	
Resective surgeries	Gingivectomy, apically displaced flap and undisplaced flap with or without osseous resection
Regenerative surgeries	Flaps with grafts and membranes, etc.
Correction of anatomical/Morphologic defects	
Plastic surgery techniques to widen attached gingiva	Free gingival grafts, and other techniques, etc.
Esthetic surgery	Root coverage, recreation of gingival papillae
Preprosthetic techniques	Crown lengthening, ridge augmentation, vestibular deepening
Placements of dental implants, including techniques for site development for implants	Guided bone regeneration, sinus grafts

Q.33. Probes
Ans: Following points should be mentioned to describe a probe;
WHO (CPITN) probe:

Parts	Handle, shank, calibrated working end, ball tip
Markings	0.5 mm diameter of ball tip Markings at 3.5, 5.5, 8.5,11.5 (in mm) Color coding (black marking) from 3.5 to 5.5 mm
Interpretation	If black band is visible, it indicates need for scaling and root planing. If black band is submerged or not visible, it shows need for complex surgical procedure.
Use	Screening of large number of patients

Williams probe:

Parts	Handle, shank, calibrated working end.
Markings	1, 2, 3, 5, 7, 8, 9, 10 (in mm)
Interpretation	Up to 3 mm – Normal, nonsurgical treatment. Up to 4 mm – Gingivitis Up to 5-7 mm- Beginning of destructive periodontitis (Treatment depends on level of attachment) More than 7 mm – Advanced destruction, requires surgical treatment.

Nabers probe:

Parts	Same as Williams probe but the end is curved.
Markings	3, 6, 9, 12 (in mm, at 3 mm interval)
Interpretation	3-6 mm: First black band (incipient furcation) 9-12 mm: Second black band If bands submerged partially – grade II furcation involvement If more (totally) – grade III/IV
Use	To determine the horizontal component of furcation involvement

Severity of furcation involvement	
Horizontal (Grade 0-3)	*Vertical (Subclass A-C)*
F0 – Normal	A – Up to 3 mm
F1 – Up to 3 mm	B – 4 - 6 mm
F2 – > 3 mm	C - ≥ 7 mm
F3 – Through and through between 2 roots	

Working end selection: The correct working-end of the probe has been selected if the lower (terminal) shank is positioned parallel to the tooth surface being examined. The incorrect working-end has been selected if the lower shank is perpendicular to the long axis of the tooth surface being examined.

Method to use furcation probe: Position the probe at the gingival line at a location near where the furcation is suspected (Fig. 25.6). Direct the probe beneath the gingival margin. At the base of the pocket, rotate the probe tip towards the tooth to fit the tip into the entrance of the furcation (Fig. 25.5).

Q.34. How to diagnose a case of furcation involvement?

Ans: Furcation involvement can be diagnosed by two methods;

1. *Clinical examination:* A thorough clinical examination is the key to diagnosis and treatment planning. Careful probing is performed by using Nabers probe. Transgingival probing further helps to determine the anatomy of the furcation defect.
2. *Radiographic examination:* Radiographs can also be useful 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	Radiographs show a radiolucent area in the crotch of the tooth
Grade IV	A clear radiolucent area is seen

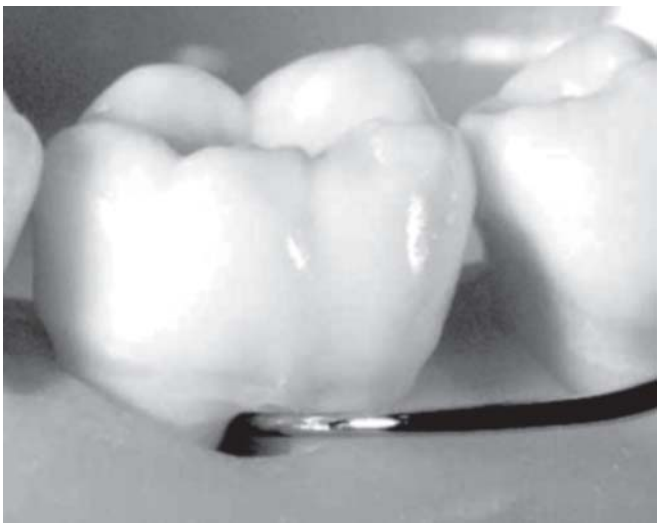


Fig. 25.5: Furcation measurement

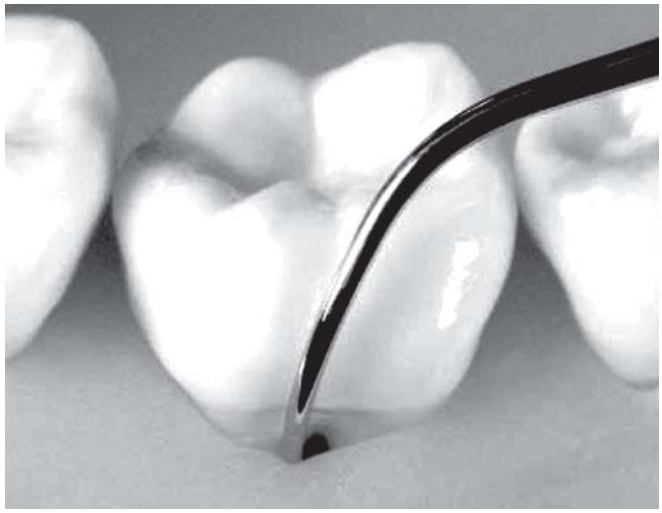
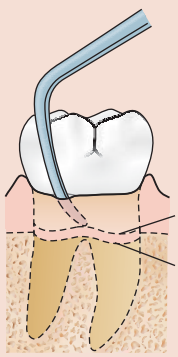
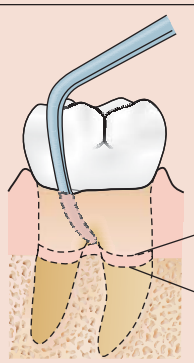


Fig. 25.6: Furcation measurement

Q.35. Furcation classifications

Ans: Irving Glickman was the first one to classify furcation invasion based on the degree of lateral periodontal destruction under the roof of the furcation in 1953.

Glickman's furcation classification (1953)			
Grade		*Description*	*Symbol*
I		• The concavity—just above the furcation entrance—on the root trunk can be felt with the probe tip; however, the furcation probe cannot enter the furcation area. • It is early-incipient stage of furcation involvement which primarily affects the soft tissues only. • Bone loss may have occurred but can't be evident on radiographic evaluation. • No horizontal component of furcation can be probed.	^
II		• The probe is able to partially enter the furcation—extending approximately one-third of the width of the tooth—but it is not able to pass completely through the furcation. It is known as *cul-de-sac* effect. (Dead end/ blind valley) • Partial bone loss with definite horizontal component • Radiographs may or may not show furcation involvement. • Both vertical and horizontal components are present. • One or more furcation of the same tooth can be affected.	Δ

Contd....

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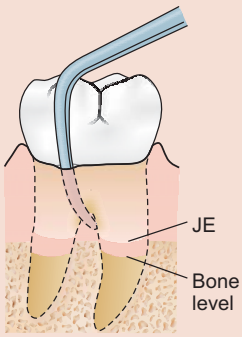
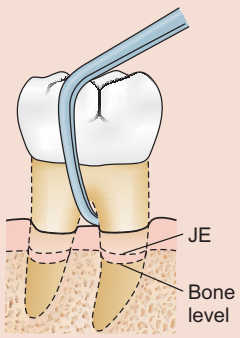
Glickman's furcation classification (1953)			
Grade		*Description*	*Symbol*
III		• The probe may pass completely through the furcation between the mesial and distal roots. • Bone is not attached to the dome of the furcation but the furcation may be filled with soft tissue and may not be visible. • On radiograph, there is a radiolucent defect is visible at furcation area.	▲
IV		• Same as a class III furcation involvement except that the entrance to the furcation is visible clinically owing to tissue recession. • Destruction of interdental bone and recession of soft tissue. • Periodontal probe passes through and through.	◆

In 2000, Fedi et al. modified Glickman's classification to include two degrees of a grade II furcation defect:

Grade II degree I - exists when furcal bone loss possesses a vertical component of >1 but <3 mm.

Grade II degree II - exists when furcal bone loss possesses a vertical component of >3 mm, but still does not communicate through-and-through.

Other classifications:

i. According to Goldman and Cohen (1968) –
   1. Grade I – Incipient lesion
   2. Grade II – Cul-de-sac lesion
   3. Grade III – Through and through lesion

ii. According to Hamp et al (Hamp, Nyman and Lidhe, 1975).
   The amount of furcation involvement in this classification is measured in millimeters. He considered horizontal component of bone loss.
   1. Degree I – Horizontal loss of periodontal tissue less than 3mm.
   2. Degree II – Horizontal loss of periodontal tissue exceeding 3mm but not encompassing the total width of furcation area.
   3. Degree III – Horizontal through and through destruction of the periodontal tissue in furcation.

iii. According to Tarnow and Fletcher (1984)
   This system records vertical loss from the roof of the furcation apically to the level of radicular bone in millimeter. It may be combined with any acceptable grade I, II, III or other classifications.

1. Subclass A – vertical loss of 0-3 mm of interradicular height
2. Subclass B – vertical loss of 4-6 mm of interradicular height
3. Subclass C – vertical loss of interradicular height of 7 mm or greater.

Q.36. What are the treatment guidelines for furcation involvement?
Ans:

	Traditional procedures	*Regenerative procedures*
Grade I	Scaling, root planing, curettage, gingivoplasty, odontoplasty.	–
Grade II	Scaling, root planing, curettage, gingivoplasty, odontoplasty, osteoplasty with limited ostectomy, in shallow grade II invasion. In more severe furcation involvements root resection/ hemisection is done.	Bone grafting, GTR, root conditioning, coronally-displaced flap and combination of procedures.
Grade III	Tunneling, root sectioning, hemisection, extraction.	GTR and combination of procedures.
Grade IV	Maintenance	–

Q.37. What is the reason for absence of pocket or gingivitis in association with trauma from occlusion?
Ans: Trauma form occlusion mainly affects the supporting tissues and does not affect the gingiva. The marginal gingiva is unaffected by trauma from occlusion because its blood supply is not affected, even when the vessels the periodontal ligament are obliterated by excessive occlusal forces. Thus, trauma from occlusion does not cause pocket or gingivitis.

Q.38. What are the differences in compositions of calculus at different regions of mouth?
Ans: Supragingival calculus most commonly contains hydroxyapatite and octacalcium phosphate. Subgingival calculus contains same hydroxyapatite, more magnesium whitelockite.

Hydroxyapatite is the major crystallite in both supra and subgingival calculus.

Brusite	More common in mandibular anterior region as the principal source of saliva for mandibular anterior region is submandibular and sublingual glands though Warthin's and Stensen's duct respectively.
Magnesium whitelockite	More common in posterior regions as the source of mineral is saliva from the parotid gland.

Q.39. Can mouthwash eliminate bad breath?
Ans: No, mouthwash alone cannot eliminate bad breath for long term. Mouthwash can be used as a last step in the routine oral hygiene maintenance procedure as an added regimen. Most of the mouthwashes with volatiles mask the bad breath for a few hours only.

First, the food particles that can rot must be removed from in between the teeth immediately after each meal. Brushing the teeth at least 2 times a day is essential. Apart from these basic oral hygiene maintenance procedures, other etiological factors responsible for halitosis should be treated accordingly. An effective way is to increase the secretion of saliva or to lower the pH of saliva which increases the solubility of malodorous compounds.

Q.40. How far does a toothbrush penetrate below the free gingival margin?
Ans: It varies from person to person depending on anatomical variations, type of bristles and method of brushing but the average value found is 0.9 mm.

Q.41. Few facts about plaque control.
Ans:

- Flossing is 80 percent effective in removing interdental plaque in the presence of an interdental papilla.
- In the absence of interdental papilla, brushing/flossing is 55 percent, Perio-Aid is 80 percent, and interdental brush is 95 percent effective.
- Mouth rinses penetrate 0.2-1.2 mm into the sulcus. Surface tension and outflow of gingival crevicular fluid decrease ability to penetrate without force.

Q.42. Chlorhexidine as a chemical plaque control agent (Fig. 25.7).
Ans: Chlorhexidine is a cationic bisbiguanide and was first established as an antimicrobial agent in 1954. It exists as acetate (diacetate), gluconate, and hydrochloride salts. Chlorhexidine gluconate is commonly used. It is a broad spectrum antiseptic and widely used as preventive and therapeutic agent. It is the dicationic nature of chlorhexidine, making it extremely interactive with anions, which is relevant to its efficacy, safety, local side effects and difficulties with formulation in products.

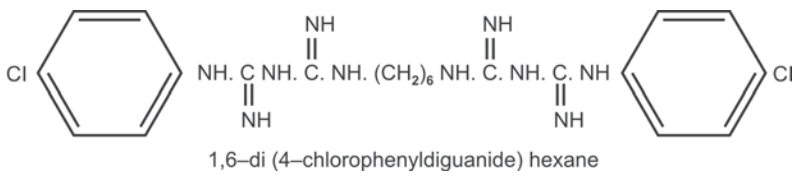


Fig. 25.7: Structure of chlorhexidine

Dose

The patient is informed to rinse twice a day for 30 seconds with 10 ml of 0.2 precent solution. To maintain the almost optimum 20 mg doses derived from 10 ml of 0.2 precent rinses, 0.12 precent is recommended as a 15 ml rinse (18 mg dose). The studies revealed equal efficacy for 0.2 precent and 0.12 precent rinses when used at appropriate similar doses (Segreto et al. 1986). 0.12 precent concentration of chlorhexidine gluconate solution is freely available in United States. In the unavailability of 0.12 precent solution, 1:1 dilution of 0.2 precent solution gives 0.10 precent concentration which is nearer to 0.12 precent concentration.

Clinical Efficacy

Reduction in salivary bacterial counts	80 to 90%
Reduction of plaque	45 to 60%
Reduction in severity of gingivitis	27 to 67%

Adverse Drug Reactions

- Brownish discoloration of teeth, restoration and dorsum of tongue. Most likely mechanisms proposed for tooth staining are:
  1. Degradation of chlorhexidine molecule release of parachloraniline.
  2. Catalysis of Maillard reactions.

3. Protein denaturation with metal sulfide formation.
4. Precipitation of anionic dietary chromogens.

- Transient impairment of taste perception (disgeusia): Chlorhexidine affects primarily salt/sweet tastes and intensities which return to normal after discontinuing the rinse.
- Mucosal erosions (hypersensitivity) are rarely seen as an idiosyncratic reaction and lesions are superficial.
- Unilateral/bilateral parotid swelling due to stenosis of parotid duct. It is very rare and explanation is not available.
- Supragingival calculus promotion:

Chlorhexidine promotes supragingiva calculus formation by precipitation of salivary protein to the tooth surface, thereby increasing pellicle thickness and/or precipitation of inorganic salts on to the pellicle layer. It does not seem to decrease effectiveness and is easily removed.

Mechanism of Action (Flow chart 25.1)

Chlorhexidine exhibits both antiplaque and antibacterial properties. The main target of chlorhexidine is the bacterial cytoplasmic membrane. After chlorhexidine has caused extensive damage to the cytoplasmic inner membrane, precipitation or coagulation of proteins and nucleic acids occurs. Damage also occurs to the outer membrane in gram-negative bacteria and the cell wall in gram-positive cells.

Chlorhexidine acts as bacteriostatic at low concentration and bactericidal at high concentration. These concentrations vary between bacterial species.

At lower concentrations, chlorhexidine has a bacteriostatic effect against most gram-positive bacteria (e.g. at 1 μg/ml), many gram-negative bacteria (e.g. at 2 to 2.5 μg/ml). It strongly binds to the bacterial cell membranes leading to increased permeability of cytoplasmic membrane and subsequently leakage of intracellular components including potassium. At high concentration, it causes precipitation of bacterial cytoplasm and cell death. At chlorhexidine concentrations of 20 μg/ml or more, a bactericidal effect as well as activity against yeasts can be expected.

The superior antiplaque activity of chlorhexidine is due to its property of sustained availability—"Substantivity". The antiseptic has a broad antimicrobial action, including a wide range of gram-positive and gram-negative bacteria (Wade and Addy,1989). It is also effective against some fungi and yeasts including candida, and some viruses including HBV and HIV. Chlorhexidine inhibits the plaque by:

- Preventing pellicle formation by blocking acidic groups on salivary glycoproteins and thereby reducing glycoprotein adsorption on to the tooth surface.
- Preventing adsorption of bacterial cell wall onto the tooth surface by binding to the bacteria.
- Preventing binding of mature plaque by precipitating agglutination factors in the saliva and displacing calcium from the plaque matrix.

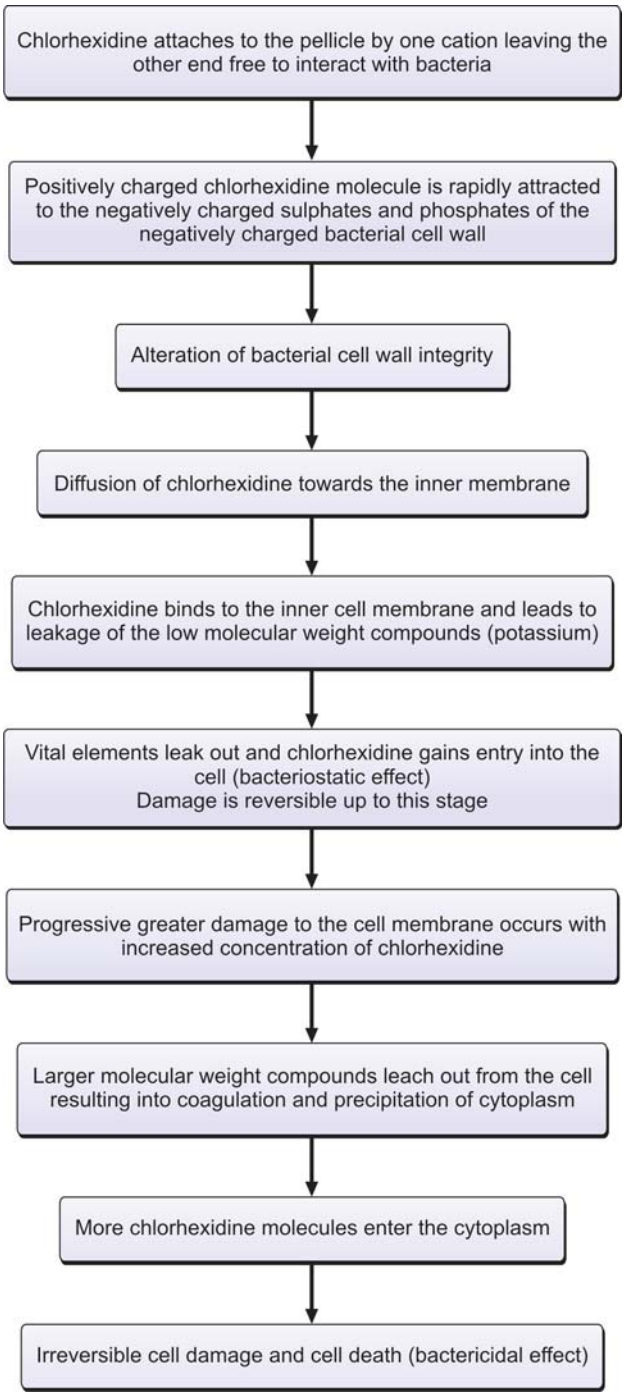
Chlorhexidine has strong affinity for binding to skin and mucous membrane. It adsorbs to the surfaces including pellicle coated teeth and slow release of the chlorhexidine from these reservoir surfaces, resulting into "Bacteriostatic milieu" in the oral cavity. After a single rinse with chlorhexidine, saliva itself exhibits antibacterial activity for about 5 hours and suppresses salivary bacterial counts for over 12 hours (so the frequency of use is twice a day). Following several rinses of chlorhexidine, the number of aerobic and anaerobic species in saliva can be reduced by 80-90 percent. Chlorhexidine is potent antifungal agent.

This mechanism is similar to that associated with tooth staining. (This explains why the anionic substances such as sodium lauryl sulfate of toothpaste and also the food containing anionic dietary chromogens especially beverages like tea, coffee and red wine reduce the

effectiveness of chlorhexidine, if used shortly after the rinse). Patient should be instructed not to take tea or coffee during the immediate period after rinse. Gargling at the night is recommended as no beverages will be consumed at night and the sustained release throughout the night gives protection when it is most sought because of decreased salivary secretion.

Pin-cushion Effect

Flow chart 25.1: Mechanism of action of chlorhexidine



Safety

1. Chlorhexidine has very low systemic toxicity: Chlorhexidine is poorly absorbed in the GI tract. Over 90 percent of swallowed chlorhexidine is excreted unchanged in the feces.
2. It has not produced any appreciable resistance of oral microorganisms.
3. It has not associated with teratogenic alterations.

Clinical Applications

1. Pretreatment rinse
2. Immunosuppressed patients
3. Postperiodontal surgery: Chlorhexidine used postoperatively reduces bacterial risk factors associated with wound healing
4. Postextraction
5. Subgingival irrigation
6. Necrotizing ulcerative periodontitis
7. Aphthous stomatitis
8. Caries control
9. Oral malodor.

Q.43. Listerine as a chemical plaque control agent.
Ans:

- Listerine is an essential oil mouth rinse. It is available over-the-counter (nonprescription) and is composed of a fixed combination of phenol-related essential oils: thymol (0.064%), methyl salicylate (0.060%), eucalyptol (0.092%), and menthol (0.042%) in 26.9 percent ethanol.
- At this concentration, the ethanol serves to dissolve the active ingredients. Contrary to persistent myths, methanol (which is frequently confused with menthol) is not an ingredient. Thymol is an antiseptic, methylsalicylate is a cleaning agent, and menthol is a local anesthetic.
- Listerine has with the highest alcohol content. There has been concern that the use of alcohol-containing mouthwash such as listerine may increase the risk of developing oral pharyngeal cancer but there is now sufficient evidence to accept the proposition that developing oral cancer is increased or contributed to by the use of alcohol-containing mouthwashes. Whilst many of these products may have been shown to be effective in penetrating oral microbial biofilms *in vitro* and reducing oral bacterial load, it would be wise to restrict their use to short-term therapeutic situations if needed. Perhaps the use of mouthwashes that do not contain alcohol may be equally effective. Further, mouth rinses should be prescribed by dentists, like any other medication. There may well be a reason for the use of alcohol-containing mouth rinses, but only for a particular situation and for a limited and controlled period of time. It should be restricted to adults for short durations and specific, clearly defined reasons.
- Chlorhexidine mouth rinse produces more extrinsic tooth staining and higher calculus formation compared to essential oils containing mouth rinse. This may limit the patient compliance for long-term use of chlorhexidine and as a long-term plaque-control adjunct for moderate- to high-risk patients, essential oil mouth rinse is better.
- Listerine is mouth rinse of choice for the patient allergic to the chlorhexidine. It gives 25-35 percent reduction in gingivitis.
- It is very strong and may lead to burning sensation in the mouth. Also high content of alcohol may exacerbate the xerostomia.
- The recommendation for use is twice a day for 30 seconds with 20 ml.

Q.44. Tetracycline-containing fibers (Actisite).
Ans:

- Actisite is a tetracycline fiber—a polymer fiber and ethyl/vinyl acetate 25 percent saturated with tetracycline. It is placed without anesthesia using a blunt instrument like retraction cord packer and leave in place 1-2 weeks. Adhesive (in kit) can also be used to hold in place.
- It is an ethylene/vinyl acetate copolymer fiber (diameter 0.5 mm) containing 12.7 mg of drug per 9 inches and it maintains 1300 μm/ml of concentration of tetracycline in GCF for 10 days.
- It is not used at initial and antiinfective phase of therapy but it is used only after traditional methods have been employed.
- Predominantly used in refractory cases, ailing implants, or the periodontal abscess. No tetracycline resistance has been found with fiber therapy.

Q.45. Subgingival doxycycline (Atridox).
Ans:

- Atridox is a 10 percent formulation of doxycycline in a bioabsorbable, "flowable" poly-DL-lactide and *N*-methyl-2-pyrrolodone mixture delivery system that allows for controlled release over 7 days.
- It is available in gel form and it is the only local drug delivery system accepted by ADA.

Q.46. Periochip.
Ans:

- It is an orange-brown, biodegradable, rectangular chip rounded at one end. It has an active ingredient of chlorhexidine gluconate (2.5 mg) that is released into the pocket over a period of 7 to 10 days.
- It is a resorbable delivery system consists of a small biodegradable hydrolyzed gelatin chip (4.0 x 5.0 x 0.35 mm).

Q.47. Subgingival minocycline (Arestin).
Ans:

- Arestin is an ointment of 2 percent (wt/wt) minocycline hydrochloride. The 2 percent minocycline is encapsulated into bioresorbable microspheres in a gel carrier.
- It is applied once every 2 weeks for four applications due to insufficient sustained-release properties.

Q.48. What is the treatment guideline for ANUG?
Ans:

	Treatment sessions for ANUG
First visit	• Removal of pseudomembrane and nonattached debris after the application of topical anesthetics. • Amoxicillin 500 mg orally every 6 hours for 10 days (For patients allergic to amoxicillin, erythromycin 500 mg every 6 hours is good alternative) and metronidazole 500 mg twice daily for seven days is given to patients with systemic manifestations. • Deep scaling should be avoided at first visit. Supragingival scaling without touching the marginal gingiva can be performed. • Patient is advised to rinse with a glass full of equal dilution of 3 percent H_2O_2 with warm water every 2 hours and/or twice daily with 0.12 percent chlorhexidine solution.

Contd....

Contd....

	Treatment sessions for ANUG
Second visit	After 1-2 days of the 1st visit, scaling is performed and oral hygiene instructions are given.
Third visit	Approximately 5 days after 2nd visit, thorough scaling and root planing is performed. The H_2O_2 rinses are substituted with chlorhexidine rinses which are continued for 2-3 weeks.
Subsequent visits	Patients without other gingival diseases are dismissed for one week. Tooth extraction or periodontal surgery should be postponed for 4 weeks. Contouring of the gingival margin can be done by gingivoplasty.

SCALING AND ROOT PLANING

Q.1. How much time does plaque takes to form on tooth surface after scaling and polishing?
Ans: Plaque starts forming immediately within nanoseconds after scaling and polishing.

Q.2. In how many sittings scaling should be completed ideally and why?
Ans: Ideally, scaling should be completed in one sitting because after first sitting the inflammatory infiltrate decreases in size and the pocket wall becomes fibrous. Dentogingival tissue becomes more resistant to penetration of instrument making it difficult to access the deeper calculus subsequently. Patients with small amount of calculus and relatively healthy tissues can be treated in one appointment. However, in patients with heavy deposits confined by deep edematous pockets, severe inflammatory changes limit the reach of the instrument deep into the pocket at the first sitting. On subsequent sittings, gradual reduction in inflammation and shrinkage of pocket wall make the unreachable areas accessible. Most of the patients require several treatment sessions for the dentist to perform a complete debridement of tooth surfaces. The selection of optimal therapy varies from patient-to-patient depending on the oral condition.

Q.3. After what duration and why should patient be recalled for follow-up after scaling and root planing?
Ans: Clinical evaluation of the soft tissue response to scaling and root planing, including probing should not be conducted earlier than 4 weeks postoperatively. This permits sufficient time for both epithelial and connective tissue healing and also allows the patient sufficient practice with oral hygiene skills to achieve maximum improvement. Gingival inflammation usually substantially reduced or eliminated within 3 to 4 weeks after removal of calculus or local irritants.

Q.4. What is the difference between polishing by rubber cup and brush?
Ans: Polishing brush is more abrasive than the cup. Brush is advocated for cleaning pit and fissures and stubborn stains. It should be avoided near cervical areas as it could lacerate the gingiva and may abrade the cementum while rubber cup is safer to use near the cervical region, root surface and less susceptible to cause abrasion.

Q.5. At what speed should the polishing tools be used?
Ans: Polishing tools should be rotated with lowest possible speed, only enough to keep them rotating and high enough that removes the stains.

HISTORY

Q.1. What is history?
Ans: It is a systematic approach to patient for diagnosis and treatment.

FEW POINTS ABOUT INTRAORAL EXAMINATION

General oral hygiene.
Oral hygiene can be evaluated from the accumulated deposits such as plaque, calculus, food debris stains. Poor oral hygiene leads to periodontal diseases.

Oral (good, poor, fair) is related to the OHI score of particular patient.

(In examination of soft tissue, better to write is no pathological changes are found rather than writing normal).

Stillman's clefts.
It is a special type of recession consisting of narrow triangular-shaped gingival recession. These are apostrophe-shaped indentations extending from and into gingival margin for varying-distance. It varies in length from slight break in gingival margin to 5-6 mm of depth.

McCall's festoon.
These are life preserver shaped enlargement of marginal gingiva that occurs most frequently on the facial surfaces in canine and premolar areas.

Surface texture.
Gingival surface is textured with stippling. Stippling is present only on center of interdental papilla and attached gingiva while it is absent on marginal gingiva. It is produced by alternate elevations and depressions representing connective tissue retepegs projecting into epithelium. It is the form of adaptive specialization or reinforcement for function. It is viewed by drying the gingiva.

Changes occurring in disease: There is loss of stippling in inflammation due to epithelial atrophy and edema of gingiva.

Surface: It is smooth and shiny when exudative process is predominant and it is firm and nodular when fibrotic process is predominant. Leathery texture is seen in hyperkeratosis. Peeling of surface occurs in chronic desquamative gingivitis. (It can be different in different areas of oral cavity depending on changes in gingiva and is noted respectively in the history form).

Width of attached gingiva (Fig. 25.8).
Width of attached gingiva can be measured by following ways:

i. Measurement approach
   Width of attached gingiva = Distance between margins of gingiva to mucogingival junction – Distance between margin of gingiva and free gingival groove (Sulcus/pocket depth).
ii By using a solution called as "Schiller's potassium iodide solution".
   Potassium iodide solution stains only the keratinized epithelium (marginal gingiva, attached gingiva, interdental papilla). After staining the gingiva, width of attached gingiva can be easily measured by measurement approach.
   Width of attached gingiva = Stained gingiva – Pocket depth
iii. *Tension test*: It is a simple clinical determination of the quantity of attached gingival surrounding tooth. Lip or cheek is stretched to demarcate the mucogingival junction and to see any movement of free marginal gingiva. Any movement or blanching of

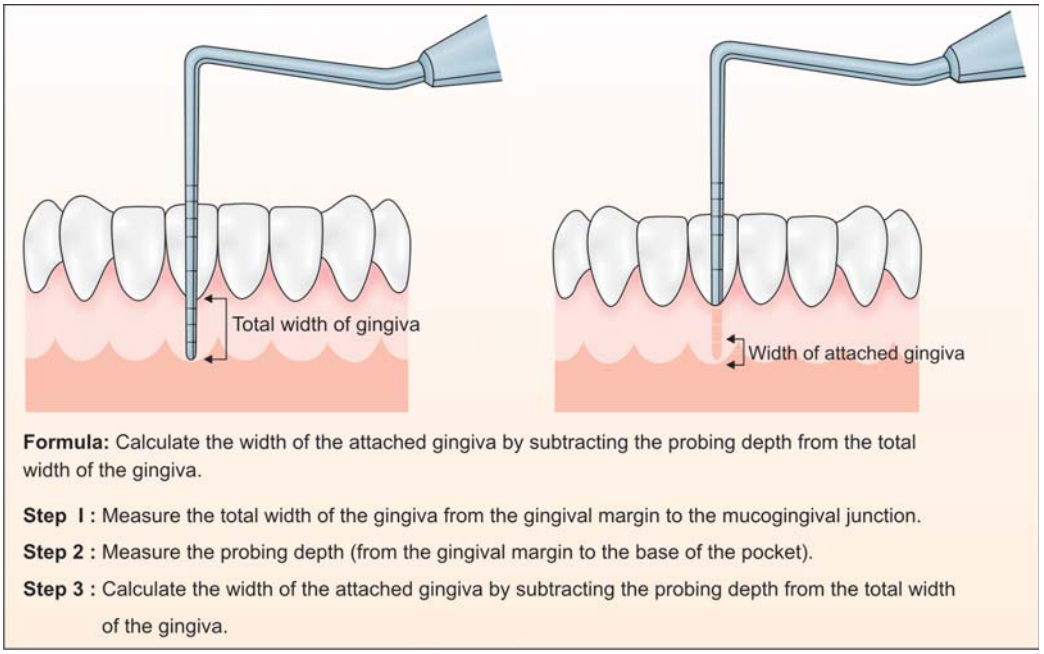


Fig. 25.8: Width of attached gingiva

free marginal gingival during stretching of lips indicates inadequate width of attached gingival (Kopczyk and Saxe 1974; Vincent et al. 1976).

Width of attached gingiva may increase with increasing the age due to coronal movement of tooth (However, the gingiva must be healthy). The mucogingival junction remains stationary. Increase in the width attached of gingiva occurs due to changes at the coronal level.

Position.

The position of gingiva refers to the level at which the gingival margin is attached to the tooth.

a. *Actual position of gingiva*: It is the level of epithelium attachment on the tooth.
b. *Apparent position*: It is the level of the crest of the gingival margin.

Recession: "Gingival recession is the exposure of the root surface by an apical shift of gingiva."

Severity of recession is determined by the actual position of the gingiva, not by apparent position. The prevalence, extent and severity of gingival recession increase with age and more prevalent in male. Recession refers to the location of gingiva, not its condition. Receded gingiva can be inflamed but may be normal. It can be localized or generalized.

Recession is the cumulative effect of minor pathologic involvement and repeated minor direct trauma to the gingiva. Susceptibility of recession is also influenced by the position of tooth in the arch, the root-bone angle, and the mesiodistal curvature of the tooth surface. The rotated, tilted or facially displaced tooth may represent thinned or reduced buccal bone plate. The effect of the angle of root in the bone on recession is often observed in the maxillary molar area. Flaring of buccal and palatal roots thins the cervical bone and repeated trauma results in recession.

Types:

i. Visible—Recession that is clinically observable.
ii. Hidden—Recession that is covered by gingiva can be measured only by inserting probe into the level of epithelium attachment.

The total amount of recession is the sum of the two.

Causes:

Etiological factors include, but are not limited to (Figs 25.9 to 25.14):

- Gingival inflammation
- Tooth malposition, deep overbite

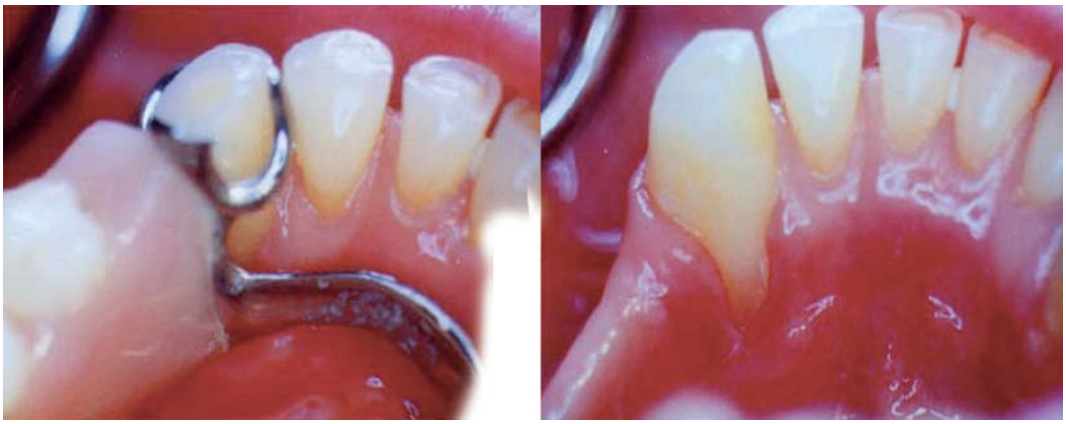


Fig. 25.9: Appliance

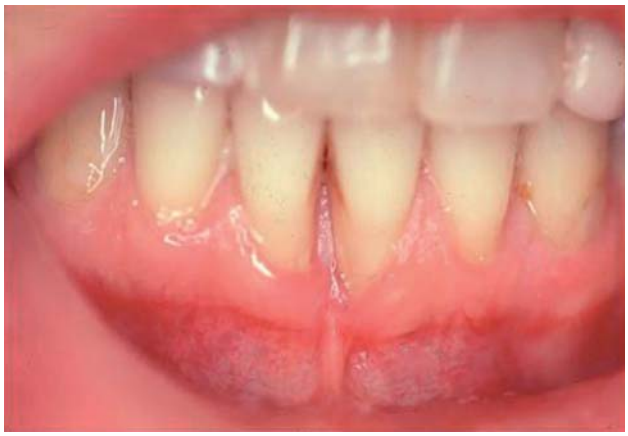


Fig. 25.10: High frenum attachment

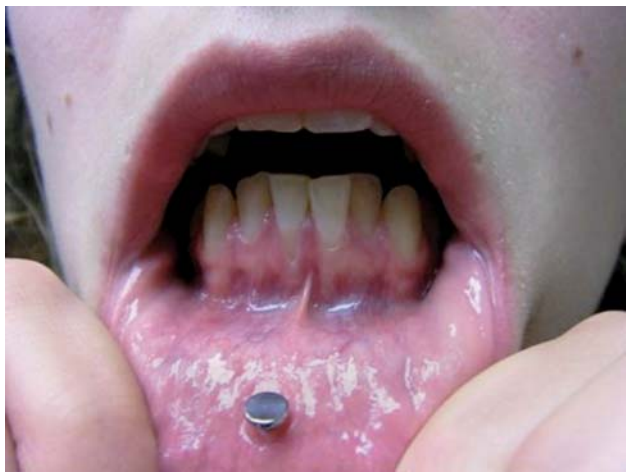


Fig. 25.11: Oral piercing

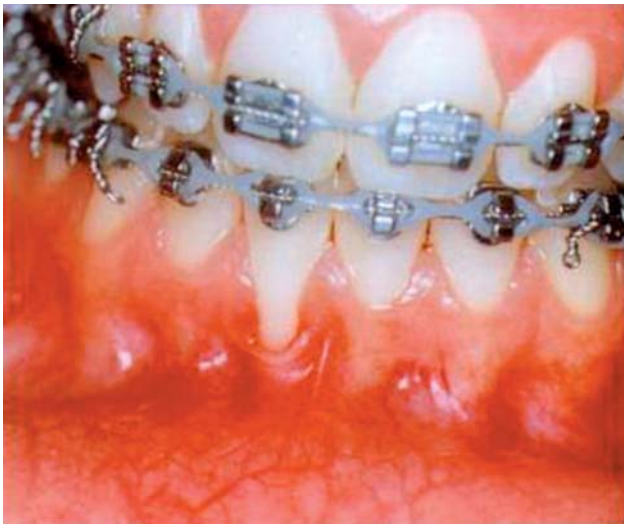


Fig. 25.12: Orthodontic appliance

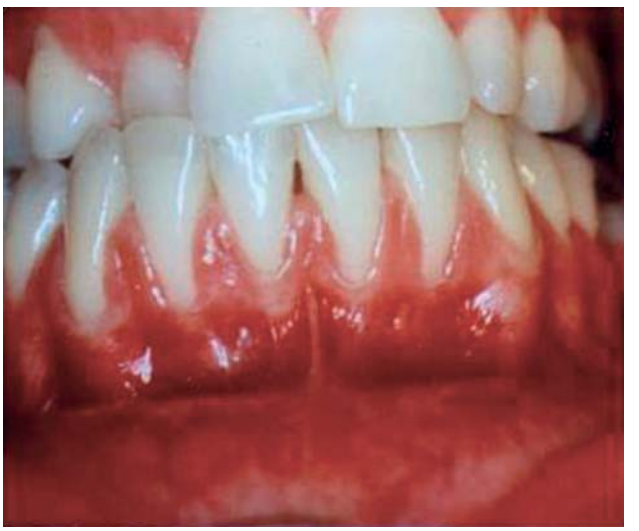


Fig. 25.13: Prominent roots

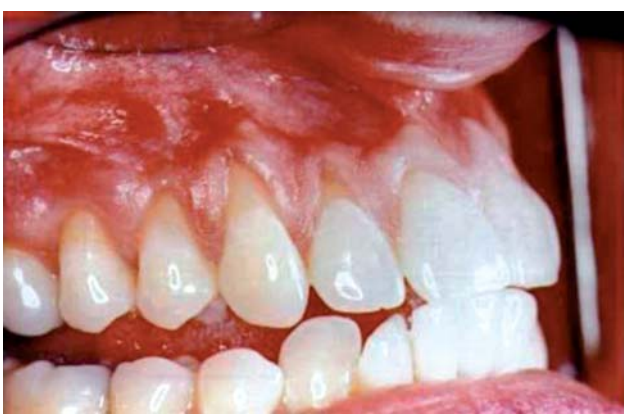


Fig. 25.14: Toothbrush trauma

- Toothbrushing injury (gingival abrasion)
- Friction from soft tissue (gingival ablation)
- Anatomical abnormalities (High frenum attachment, dehiscence, thin bony plates)
- Trauma from occlusion
- Iatrogenic causes (Damage by orthodontic band, faulty restorations, faulty prosthesis, oral piercing).

Significance:

- Exposed root surfaces are more susceptible to caries, abrasion, and erosion.
- Exposed accessory canals are vulnerable to infection.
- Wearing away of expose cementum causes dentinal sensitivity.
- Interproximal recession creates spaces, in which plaque accumulation aggravates inflammation and also makes oral hygiene maintenance difficult.

Classifications:

i. *Sullivan and Atkins classification (1968)*: Many authors have defined gingival recession by its degree of severity and extent in order to help clinician to define treatment options and prognosis. In 1968, Sullivan and Atkins defined gingival recession as exposure of the root surface by an apical shift in the position of the marginal tissue. The classification system describes the type of recession and expected amount of root coverage utilizing the free gingival graft procedure. Deep-wide defects were thought not to be able to be covered.
   - Shallow-narrow
   - Shallow-wide
   - Deep-narrow
   - Deep-wide.

ii. *Guinard and Caffesse (1978)*:
   - Visible recession: It is the amount of clinically observable root measured from the cemento-enamel junction to the crest of the soft tissue margin.
   - Hidden recession: It is the depth of the sulcus or pocket as measured from the soft tissue margin to the epithelial attachment (junctional epithelium)

iii. *Miller's classification (1985)*:
   PD Miller combined the four classes of Sullivan and Atkins into two (shallow and deep) and then created two additional classes, all related to height if the interproximal bone and the relation of the gingival recession to the mucogingival junction. This classification helps clinicians to determine the degree of root coverage expected during periodontal plastic surgery (Figs 25.15 to 25.18).

Class I	Marginal tissue recession **does not extend to** the mucogingival junction. There is no loss of bone or soft tissue in the interdental areas. This type of recession can be narrow or wide.		
Class II	Marginal tissue recession **extends to or beyond** the mucogingival junction. There is **no loss of bone or soft tissue** in the interdental areas. This type of recession can be narrow or wide.	To memorize	extends to or beyond
Class III	Marginal tissue recession **extends to or beyond** the mucogingival junction. There is **bone and soft tissue loss** in the interdentally or **malpositioning** of tooth.	bone and soft tissue loss	
Class IV	Marginal tissue recession **extends to or beyond** the mucogingival junction. There is **severe bone and soft tissue loss** interdentally or **severe tooth malposition**.		

Grade	Treatment
Class I	Complete, 100 percent coverage of this type of recession can be achieved. For example, by use of free connective tissue grafts
Class II	Complete coverage of root surface can still be achieved. With such deep recession, guided tissue regeneration (GTR) with membrane can be employed instead of connective tissue grafts.
Class III	Complete regeneration of such defects is not possible; the facial root surface can, at best, be partially covered and rebuilding the papillae is hopeless.
Class IV	Regeneration of lost tissue by surgical procedure is rarely possible.

Prognosis of class I and II is good to excellent. Only partial coverage can be expected in Class III. Class IV has poor prognosis.

Level of the gingival margin: The level of the gingival margin can change over the time period in response to trauma, medications, or disease. Three possible relationships exist between the gingival margin and the cementoenamel junction (CEJ) of the tooth.

1. Gingival margin significantly covers the CEJ (Fig. 25.19).
   a. In this instance, the gingiva covers a significant portion of the tooth crown.

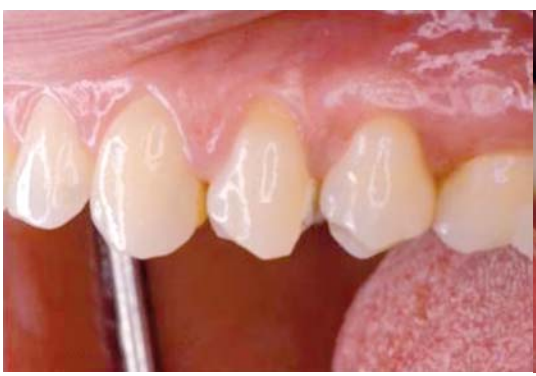


Fig. 25.15: Class I

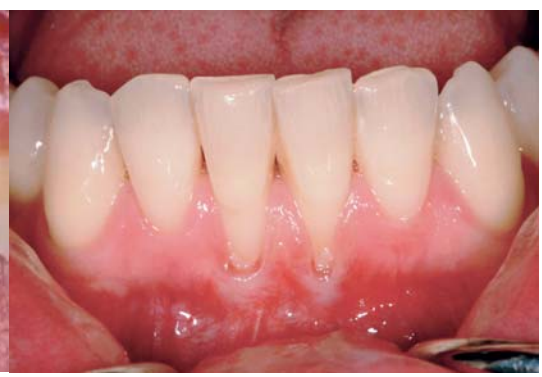


Fig. 25.16: Class II

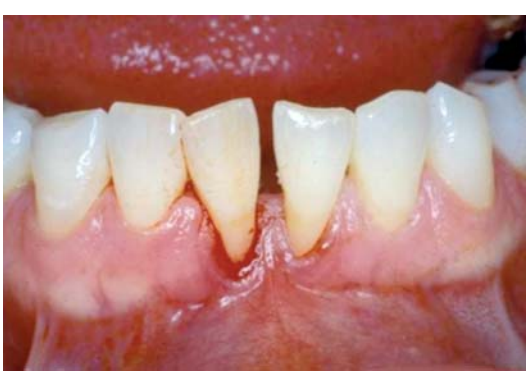


Fig. 25.17: Class III

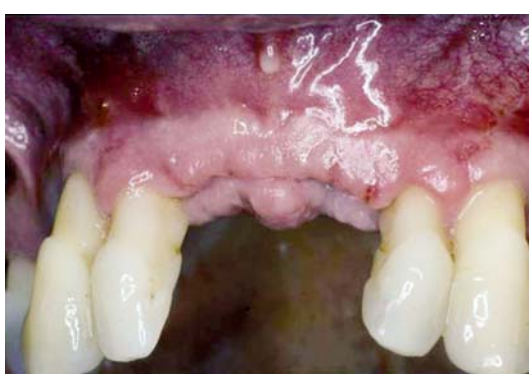


Fig. 25.18: Class IV

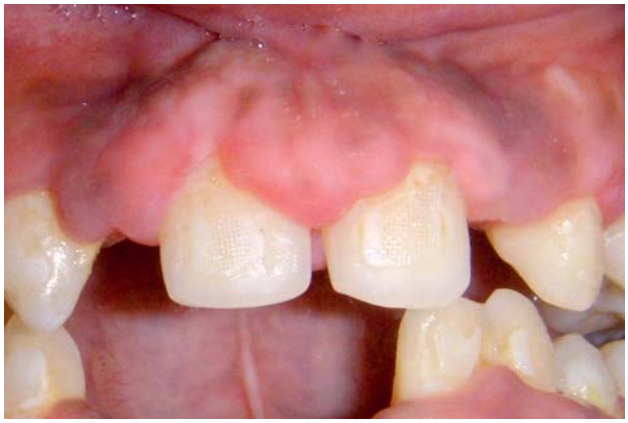


Fig. 25.19: Gingival margin significantly covers the cementoenamel junction (CEJ)

b. The position of the gingival margin may be coronal to the CEJ owing to following reasons;
   - Swelling (edema)
   - An overgrowth of the gingival tissues caused by certain medications taken by patients as a part of treatment of medical condition
   - An increase in the fibrous connective tissue of the gingiva caused by a long-standing inflammation of the tissue

2. Gingival margin is at the CEJ (Fig. 25.20).
   This is the natural position of the gingival margin.
3. Gingival margin is significantly apical to the CEJ (Fig. 25.21).
   a. When the gingival margin is significantly apical to the CEJ, a portion of the root surface is exposed in the mouth. This relationship is known as gingival recession.
   b. Gingival recession is the movement of the gingival margin from its normal position—usually with underlying loss of bone—resulting in the exposure of a portion of the root surface. In recession, the gingival margin is apical to the CEJ and the papillae may be rounded or blunted.

Technique to determine the gingival margin level:
When tissue swelling or recession is present, a periodontal probe is used to measure the distance that the gingival margin is apical or coronal to the CEJ.

1. *For gingival recession*: If gingival recession is present, the distance between the CEJ and the gingival margin is measured using a calibrated periodontal probe. This distance is recorded as the gingival margin level (Figs 25.22 and 25.23).
2. *When the gingival margin covers the CEJ*: If the gingival margin covers the CEJ, the distance between the margin and the CEJ is estimated using the following technique:
   a. Position the tip of the probe at a 45-degree angle to the tooth.
   b. Slowly move the probe beneath the gingival margin until the junction between the enamel and cementum is detected.
   c. Measure the distance between the gingival margin and the CEJ. This distance is recorded as the gingival margin level.

Calculating clinical attachment level:
A competent clinician must understand the procedure for determining the Clinical Attachment Level for the three possible relationships of the gingival margin to the CEJ.

1. The gingival margin may be apical to the CEJ, cover the CEJ, or be at the CEJ.

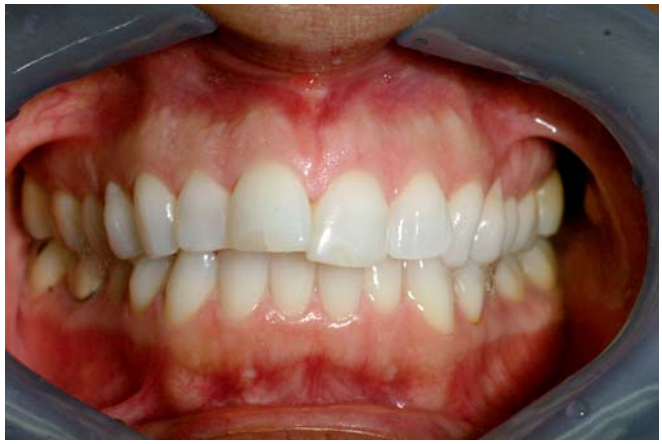


Fig. 25.20: Gingival margin at the cementoenamel junction (CEJ)

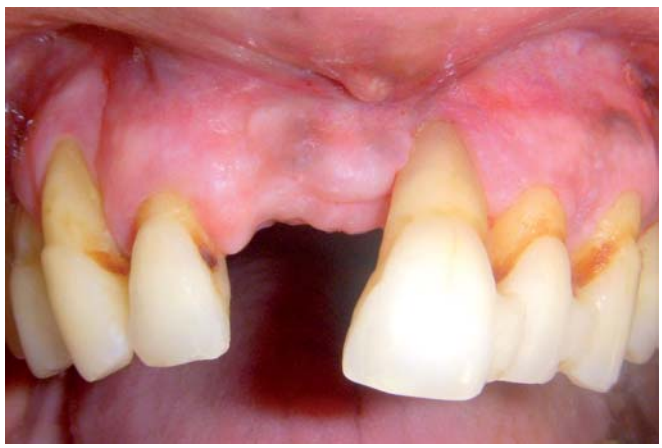


Fig. 25.21: Gingival margin significantly apical to the cementoenamel junction: It is known as recession, this relationship leads to exposure of the root surface

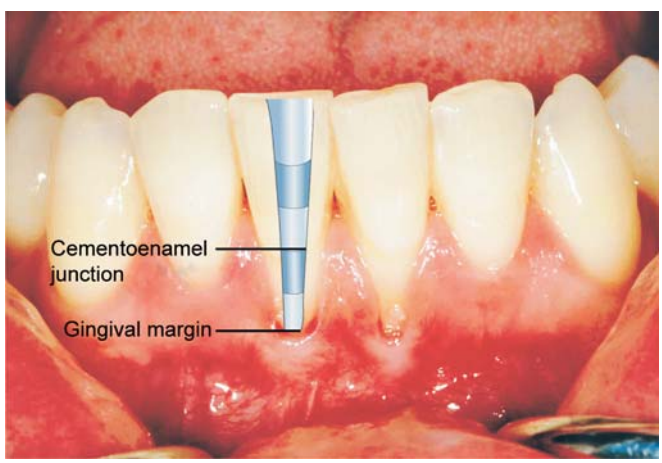


Fig. 25.22: Calculating the clinical attachment level

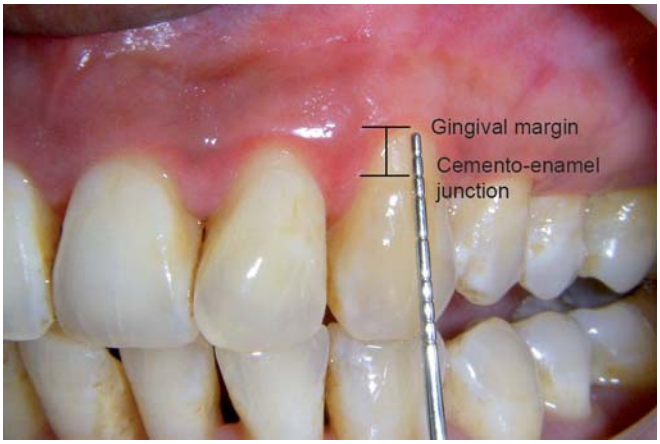


Fig. 25.23: Loss of attachment

2. Two measurements are used to calculate the clinical attachment level:
   a. The probing depth and
   b. The level of the gingival margin (Distance from CEJ to gingival margin)

Note that both of these measurements are routinely taken and documented on a periodontal chart.

Calculating clinical attachment level in the presence of gingival recession:

When recession is present, the CAL is calculated by adding the probing depth to the gingival margin level (Fig. 25.24).

For example:

Probing depth measurement: 4 mm
Gingival margin level: 2 mm
Clinical attachment loss: 6 mm

Calculating clinical attachment level when the gingival margin covers the CEJ:

When the gingival margin is coronal to the CEJ, the clinical attachment level is calculated by subtracting the gingival margin level from the probing depth (Fig. 25.25).

For example:

Probing depth measurement: 9 mm
Gingival margin level: 3 mm
Clinical attachment loss: 6 mm

Calculating clinical attachment level when the gingival margin is at the CEJ:

When the gingival margin is at the CEJ, no calculations are needed because the probing depth and the clinical attachment level are equal (Fig. 25.26).

For example:

Probing depth measurement: 6 mm
Gingival margin level: 0 mm
Clinical attachment loss: 6 mm

BLEEDING ON PROBING (Fig. 25.27)

Gingival bleeding helps in clinical evaluation of degree of gingival inflammation. It is related to persistent presence of plaque on the teeth. Bleeding on probing appears before other signs of gingivitis requiring subjective estimation like color and texture changes and it serves as an important objective sign of inflammation. Therefore, it is of value for the early diagnosis and prevention of more advanced gingivitis. Gingival bleeding varies in severity, duration, and ease of provocation. Severity of gingival bleeding increases with increase

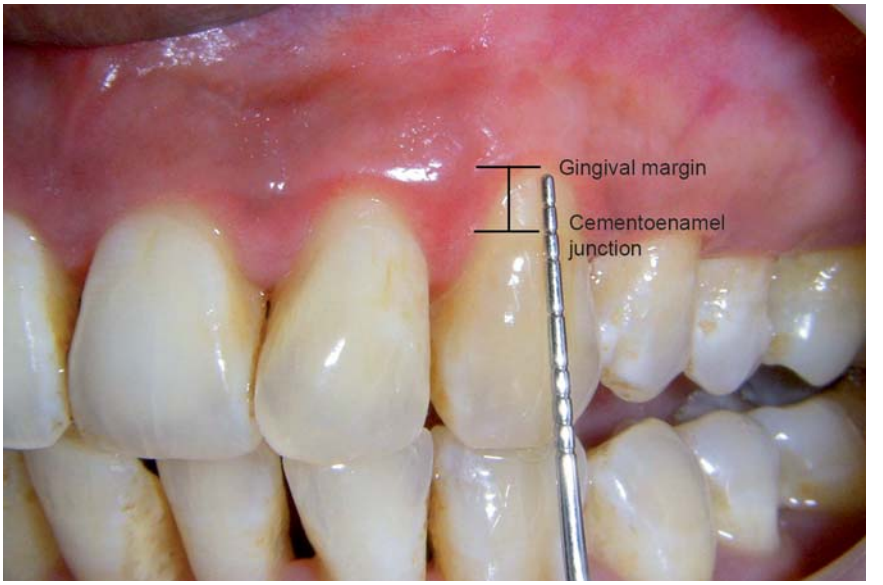


Fig. 25.24

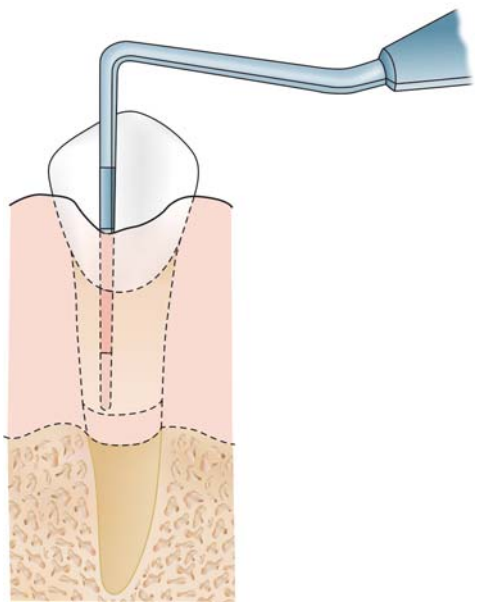


Fig. 25.25

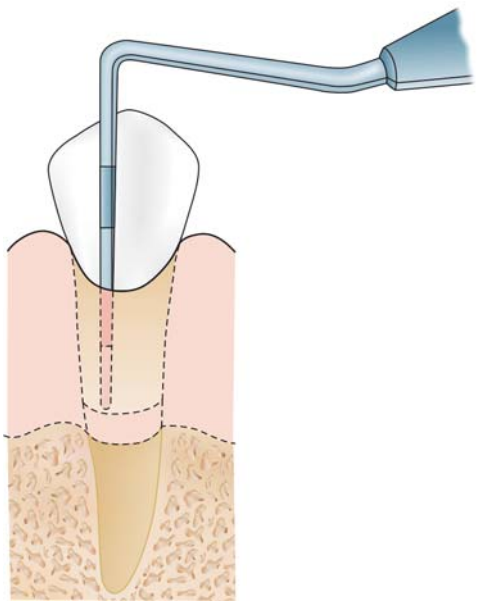


Fig. 25.26

Figs 25.24 to 25.26: Calculating clinical attachment level

in size of inflammatory infiltrate in the connective tissue at the base of sulcus. Bleeding on gentle skimming examines the health of marginal gingiva and bleeding on probing examines the health at the base of sulcus and pocket.

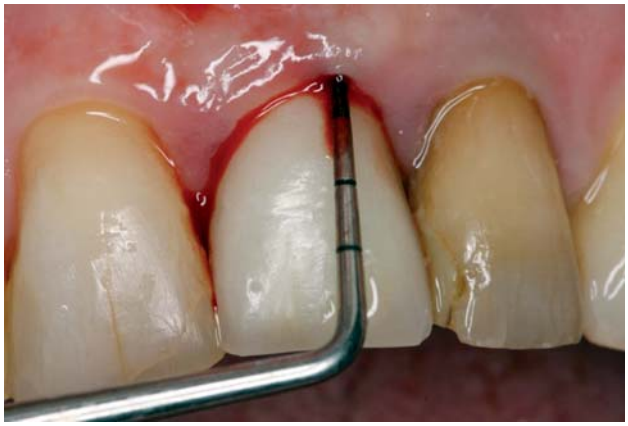


Fig. 25.27: Bleeding on probing

Bleeding on gentle probing is a sign of inflammation. The insertion of a probe to the bottom of the pocket elicits bleeding, if the gingiva is inflamed and the pocket epithelium is atrophic or ulcerated. *Bleeding on probing is an earlier sign of inflammation than gingival change in color and other signs of inflammation.* Depending on severity of inflammation, bleeding can vary from a tenuous red line along the gingival sulcus to profuse bleeding. In acute inflammation, it occurs as soon as probe is inserted into the sulcus while in chronic inflammation; it occurs few seconds after the probe is removed. Therefore clinician should recheck for bleeding 30 to 60 seconds after probing. *The severity of bleeding (amount of bleeding) and the ease of its provocation (the time between stimulation and appearance of blood) depend on the intensity of the inflammation.* The sulcus bleeding index by Mühlemann provides an objective, easily reproducible assessment of the gingival status. It is extremely useful for detecting early inflammatory changes and the presence of inflammatory lesions located at the base of the periodontal pocket, an area inaccessible to visual examination.

Bleeding on probing is an important diagnostic factor for clinicians to use in planning periodontal therapy. The presence of bleeding is an indicator of active gingival inflammation, and until it is controlled, the patient is at risk of continuing periodontal disease and tissue destruction. Bleeding on probing is widely used by clinicians to measure outcomes of treatment, and to motivate patients with their home care.

Limitations:

1. Longitudinal studies fail to demonstrate a significant relation between bleeding on probing, other clinical signs and subsequent attachment loss.
2. Bleeding on probing from healthy site with intact periodontium further limits the use of bleeding as an inflammatory parameter.

Conclusion:

Even after the limited predictive value of bleeding on probing for disease progression (attachment loss), its absence indicates periodontal stability. It is an excellent negative predictor of future attachment loss and therefore the absence of gingival bleeding on probing is desirable and implies low risk for future clinical attachment loss. The documentation of bleeding points during probing is important and should be included in the patient's periodontal records.

SUPPURATION

The clinical assessment of suppuration, formation of pus within the periodontal pocket is performed by gently pressing the ball of index finger along the lateral aspect of marginal

gingiva and rolling it towards the crown of tooth with pressure. Suppuration is formed on inner pocket wall, but the external appearance of pocket wall is unchanged. Therefore, the visual examination with digital pressure is required. Suppuration doesn't occur in all periodontal pockets. However, suppuration may be present, although not visibly detected, and therefore its clinical absence is not an indicator of periodontal stability. Clinically, detectable suppuration should be documented in the patient's periodontal records.

Q.1. What are the differences between gingival and periodontal abscess?
Ans:

	Gingival	*Periodontal*
Definition	Abscesses localized in the gingiva, caused by injury to the outer surface of the gingiva, and not involving the supporting structures are called gingival abscess.	Periodontal abscess is localized purulent inflammation in the periodontal tissues.
History	It occurs in previously disease-free gingiva. It is usually an acute inflammatory response.	Generally, it occurs in the course of chronic destructive periodontitis. It can be acute or chronic.
Etiology	Gingival abscess if the localized, acute inflammatory lesion that may arise from variety of sources, including microbial plaque infection, trauma, and foreign body impaction. When a foreign substance is forcefully embedded into the gingiva, bacteria carried deep into the tissues produce acute inflammatory gingival enlargement.	It results from extension of the infection into the still intact periodontal tissues and lack of proper drainage. The principal ways of abscess formation are; a. Extension of infection from a periodontal pocket deeply into the supporting periodontal tissues. b. Lateral extension of inflammation from the inner surface of the periodontal pocket into the connective tissue of the pocket wall. c. Formation abscess in a pocket with a tortuous course around the root. d. Incomplete removal of calculus during treatment followed by shrinkage of gingival wall leading to marginal closure of a deep periodontal pocket. e. Trauma to the root and accidental root perforation during endodontic therapy.
Location	Gingival abscess generally remains limited to the marginal gingiva or interdental papilla and does not involve the supporting periodontal tissue.	According to location they are classified as follows; a. Periodontal abscess involves the supporting periodontal structures. It is seen along the lateral aspect of the root. It is not necessarily located on the same surface of root from which it is formed. b. Abscess in the soft tissue wall of deep periodontal pocket.

Contd....

Contd....

	Gingival	*Periodontal*
Clinical course	In the early stages, it appears as a red swelling with a smooth, shiny surface. Within 24 to 48 hours, the lesion usually becomes fluctuant and pointed with a surface orifice from which a purulent exudate may be expressed.	a. Acute: Appear as an ovoid elevation of gingiva along the lateral aspect of the root. Pus may be expressed from gingival margin on gentle digital pressure. b. Chronic: Usually it presents a sinus that opens onto the gingival mucosa somewhere along length of root. Patient may present history of intermittent exudation.
Signs and symptoms	The adjacent teeth are often sensitive to percussion.	Acute – Throbbing, radiating pain, tenderness of gingiva on palpation, tooth mobility, lymphadenitis, and systemic effects like fever, fever, leukocytosis, malaise. Chronic – Usually it is asymptomatic but there may be dull gnawing pain, slight elevation of tooth, and a desire to bite down on and grind the tooth. It presents a sinus tract with difficult-to-detect pinpoint opening on gingiva.
Diagnosis	Based on history and clinical findings.	Based on careful exploration of history, clinical and radiographic findings. Continuity of abscess with gingival margin is the evidence of periodontal abscess.
Treatment	Treatment of gingival abscess is directed to reverse the acute phase and immediate removal of the cause. Scaling and root planing under local anesthesia ensures the removal of microbial deposits. Fluctuant abscess is incised and purulent material is drained. Any foreign material, if present, is removed. In case of large abscess, scaling and root planing and surgical therapy should be delayed until the major signs have abated. In these patients, use of adjunctive antibiotic therapy is recommended. Gingivectomy is performed and the fluctuant area is incised with scalpel blade. The exudate may be expressed by gentle digital pressure.	Acute – Treatment is aimed to alleviate symptoms, control the spread of infection, and establish drainage. Chronic – Scaling and root planing and surgical therapy is recommended. Antibiotic therapy may be indicated.

Q.2. Which clinical parameters may be used to judge the success of periodontal treatment?

Ans: There are several clinical and radiographical parameters that may be used to judge the success of periodontal therapy. The principle parameters are;

i. *Reduction or absence of bleeding on probing*:
Bleeding on gentle probing is still the best prognostic indicator of the potential for future attachment loss. Absence of bleeding on probing is a 98 percent negative predictor

that the site will lose attachment in future. Since it is impossible to predict exactly which site will lose attachment, the thrust of therapy is to control inflammation at all sites.

ii. *Reduction of probing depth and gain in periodontal attachment*:
Periodontal therapy is focused on removal of etiological agents and contributing factors and subsequent maintenance of health. One way to improve this possibility for both the patient and practitioner is to reduce the probing depths. Persistence of periodontal pathogens and progressive loss of attachment is associated with deeper pockets.

iii. *Positive radiographic changes*:
Positive radiographic changes related to the success of periodontal therapy include the reappearance of a crestal lamina dura at the interproximal osseous crests, evidence of bone fill in areas of regenerative therapy, narrowing of periodontal ligament space in relation to teeth subjected to periodontal trauma and the absence of calculus on coronal and root surfaces.

iv. *Occlusal stability*:
The main factors responsible for tooth mobility are presence of edema in gingival and periodontal tissues, loss of attachment, and the effects of occlusal forces on the attachment apparatus. After proper inflammatory control, teeth often exhibit decreased mobility. Elimination of edema and reformation of supragingival connective tissue fibers contribute to tooth stability, particularly in case of tooth with attachment loss. Judicial occlusal adjustment by selective grinding also contributes to increased tooth stability.

Q.3. How will you manage a case of moderate to severe generalized periodontitis? Describe the most preferred method for treating pockets in anterior segment?

Ans: Moderate to severe periodontitis constitutes symptoms and signs like:

- 4-5 mm or more attachment loss
- Moderate to through and through furcation involvement
- Moderate to excessive tooth mobility

Periodontitis is diagnosed based on disease activity and rate of attachment loss

Three models of periodontitis

1. Continuous paradigm (traditional concept)
   It is a plaque-induced, slowly progressive disease.
   - Continuous deepening of pockets
   - Gradual loss of attachment.
2. Random burst (newer concept)
   It is bacteria-induced disease.
   - Destruction of attachment apparatus progresses by recurrent acute episodic bursts of activity.
3. Asynchronous multiple burst
   - Destruction occurred during a defined time
   - Disease goes into remission for an indefinite time.

Health vs. disease:

There are 3 stages;

1. Periodontal health.
2. Periodontitis inactive: inflammation but no attachment loss.
3. Periodontitis active: inflammation with attachment loss.

On examination:

1. Considerations in diagnosis of periodontal disease are:
   Active or inactive
   Sulcus vs. pocket
   Sulcus (healthy attachment, plaque < threshold)
   Pocket (diseased attachment, plaque > threshold)

2. Assessment methods:
   Clinical: Clinical methods of direct observations are:
   - Subgingival plaque and calculus
   - Gingival inflammation (color changes)
   - Bleeding on probing
   - Suppuration
   - Loss of form
   - Pocket depth/probing depths (attachment level changes).

   Histological:
   It is difficult sometimes because of invasive nature of disease and it shows different activities at multiple sites.
   Microbiological:
   Culture and sensitivity test
   Organisms associated with different tissue conditions are;

Healthy sulcus	Gram-positive organisms predominate
Gingivitis	Shift from gram-positive organisms to gram-negative organisms
Adult periodontitis	Gram-negative anaerobic rods, 30-50 percent motile rods and spirochetes

Immunological:
PMNs, lymphocytes, antibody titers, complement fractions, lymphokines.
Steps for treatment are:
1. *Preliminary phase*:
   It includes treating emergency conditions like drainage of periodontal abscess, extraction of hopeless teeth.
2. *Removal of local irritants (phase I):*
   Local deposits like plaque and calculus can be removed by scaling and root planing. Subgingival scaling is done along with root planing for final smoothening and planing of the root surface.

Patient's education
- Motivating patients to perform effective plaque control is one of the most critical and difficult elements of long-term success in periodontal therapy.
- Proper brushing technique is explained and patient is instructed to maintain oral hygiene. The brushing technique should emphasize access to the gingival margins of all accessible tooth surfaces and extension as far onto the proximal surfaces as possible.
- Dental floss and other interdental aids like toothpicks, interdental brushes are advised, if required.
- If dictated by the oral hygiene, patient is advised chemical plaque control agent for aided benefits.

3. *Evaluation of response to Phase I therapy.*
   Final decision on the need of periodontal surgery should be made only after thorough evaluation of the effects of Phase I therapy. The assessment is usually made no less than 1-3 months and may extend up to 9 months after the completion of Phase I therapy. In the reevaluation of Phase I, look for:
- Persistence of calculus
- Root caries
- Defective restorations
- Pocket depth
- All signs of persistent inflammation.

Evaluation of phase I therapy determines whether any surgical intervention is required or not. Necessary surgical and restorative procedures are performed to retain the degree

of the health attained after Phase I. In moderate to severe periodontitis surgery is usually needed because of one or more factors like:

- Presence of bone defects
- Deep pockets limiting the calculus removal
- Persistent inflammation.

The transformation of the initial deep, active pocket into a shallower, inactive, maintainable pocket requires some form of definitive pocket therapy and constant supervision thereafter.

4. *Surgical phase.*

Criteria for the selection of one of the different surgical techniques for pocket therapy are based on clinical findings in the soft tissue pocket wall, tooth surface, underlying bone, and attached gingiva. Clinician should determine morphological feature, thickness, and topography of the soft tissue wall of pocket, presence of deposits, altered cementum, accessibility of the root surface to instrumentation, morphology and architecture of underlying bony defect, and presence or absence of an adequate width of attached gingiva when selecting a pocket therapy.

Before planning for surgical phase, routine blood investigation and other tests, if necessary, are carried out to evaluate patient's systemic condition. Surgery for moderate to severe periodontitis requires flap elevation. Flap surgery differs for anterior segment and for posterior segment depending on variety of factors such as age of the patient, esthetic requirement of patient, clinical findings etc.

For Anterior Segment

Selection of flap technique is crucial task and it depends on the deliberate examination and prudent clinical judgment. If interdental space is wide enough, considering the esthetic requirement of the patient, papillary preservation flap is preferred. It retains the entire papilla covering the lesion. If interdental space is narrow making it impossible to perform a papilla preservation flap, conventional flap with only crevicular incision is the better option. If some degree of gingival enlargement is present and recession is permissible, modified Widman flap can be used. This technique doesn't remove the pocket wall, but it does eliminate the pocket lining.

For Posterior Segment

The two important landmarks to determine the amount of attached gingiva and subsequently dictating surgical flap technique are pocket depth and location of mucogingival junction. Undisplaced flap and modified Widman flap are commonly used depending upon situation.

The modified Widman flap eposes the root surface for meticulous instrumentation and also removes the pocket lining. It is not intended to remove the pocket wall and the reduction in pocket depth is merely due to healing by tissue shrinkage. Undisplaced flap is an Excisional procedure and it is used only if the sufficient attached gingiva remains apical to the incision. It removes the pocket wall, thereby reduces or eliminates the pocket.

If widening of the zone of attached gingiva is required along with the pocket eradication, the apically displaced flap technique is used. It preserves and increases the width of attached gingiva by transforming the previously unattached keratinized pocket wall into attached gingiva. Attempts are directed towards maintaining the as much possible attached gingiva along with pocket elimination.

For regenerative or reconstructive procedures like bone grafting, flap is elevated accordingly. The conventional flap using only crevicular retain maximum amount of gingival tissue, including the papilla. Unlike the resective surgeries, the flap is not thinned. The thick flap is necessary to prevent exposure of the graft or the membrane.

5. *Maintenance phase.*

Patient must understand the importance of the maintenance program, and the dentist must emphasize that preservation of teeth depends on the maintenance therapy. Regular follow

up is must to maintain the periodontal structures in healthy state. The interval between the visits is set at 3 months but it may be varied according to the patient's needs.

Recall visit during maintenance phase starts with examination and evaluation and checking of the plaque control followed by treatment. At the recall examination changes that have occurred since last evaluation are observed and noted. Radiographic examinations are carried out depending on the initial therapy to check the bone height and repair of the osseous defects. Patient is asked to perform the plaque control immediately before recall visit for review and correction of plaque control. Scaling and root planing are performed as a prophylaxis, if required, after thorough review of medical and dental history, intraoral and extraoral soft tissue examinations, a dental examination and a periodontal examination.

Some points to memorize

Diseases	*Principal immune response*
ANUG	Elevated antibody titers to intermediate sized spirochetes and *P. intermedia*
Adult periodontitis	Elevated antibody titers to *P. gingivalis*
Localized aggressive periodontitis	Elevated antibody levels to *A. atinomycetamcomitans*
Generalized aggressive periodontitis	Elevated antibody titers to *P. gingivalis*
Refractory periodontitis (RPP)	Elevated antibody levels to *P. gingivalis, T. forsythia, F. nucleatum, P. micros, E. corrodens, Streptococcus intermedius*

Condition	*Brushing Technique*
Gingival recession and root exposure	Modified stillman
Healing wound after surgery	Charters technique
Routine technique in patient with or without periodontal disease	Bass or sulcular technique
Recommended technique in patient with periodontal disease	Sulcular technique
Recommended for children	Fones technique
Least effective	Roll technique
Most commonly performed	Scrub technique

Complexes		
Initial colonizers	Yellow complex	*S. mitis* *S. sanguis* *S. oralis*
	Purple complex	*V. parvula* *A. odontolyticus*
Secondary colonizers	Red complex (BPT)	*P. gingivalis* *T. denticola* *B. forsythus*
	Orange complex	*P. intermedia* *C. rectus* *F. nucleatum*
	Green complex	*A. antinomycetamcomitans*

Red complex is important as it is associated with bleeding on probing which is important sign of periodontal destruction.

CHAPTER

26 Ameloblastoma

TUMORS

New growth of abnormal tissue of body is called tumor. It is defined as, "A mass of tissues formed as a result of abnormal, excessive, uncoordinated, autonomous and purposeless proliferation of cells."

It can be malignant or benign.

ODONTOGENIC TUMORS

Odontogenic structures are formed by inductive interactions between epithelium and mesenchyme. It begins during 5th and 6th week of intrauterine life and because of that, during this period, there is possibility for occurrence of odontogenic lesions resulting into malformations, hamartomas and neoplasms.

CLASSIFICATION

Classification of epithelial odontogenic tumors is based on the principle of embryonal inductive influence of epithelial cells on the connective tissue cells.

Epithelial Odontogenic Tumors

1. Tumors producing minimal inductive changes in connective tissue:
   a. Ameloblastoma (adamantinoma)
   b. CEOT (Calcifying epithelial odontogenic tumor/Pindborg tumor)
   c. OAT (Adenoameloblastoma) odontogenic adenomatoid tumor.
2. Tumors producing extensive inductive changes in connective tissue:
   a. Ameloblastic fibroma
   b. Ameloblastic fibro-odontoma
   c. Odontoameloblastoma
   d. Odontoma
      – Compound composite odontoma
      – Complex composite odontoma.

Mesodermal Odontogenic Tumors

1. Central odontogenic fibroma
2. Odontogenic myxoma

3. Cementoma
   - Peripheral cemental dysplasia
   - Cementifying fibroma
   - Benign cementoblastoma
4. Dentinoma

Tumors of Unknown Origin

Melanotic neuroectodermal tumor of infancy.

Malignant Odontogenic Tumors

1. Odontogenic carcinoma
   - Primary intraosseous carcinoma
   - Malignant ameloblastoma.
2. Odontogenic sarcoma
   - Ameloblastic fibrosarcoma
   - Ameloblastic odontosarcoma.

AMELOBLASTOMA

Ameloblastoma is the most common odontogenic tumor exhibiting minimal inductive changes in connective tissue. It is a true neoplasm and most controversial lesion. Generally, it is considered as a benign but it is a persistent and locally malignant in nature. There are different names given to this tumor. It is also called as adamantinoma, adamantinoblastoma, epithelial odontoma and multilocular cyst.

HISTORY

1827	Cuzack	First recognized
1879	Falksson	Follicular cystoids tumor
1885	Malassez	Adamantinoma
1934	Ivy and Churchill	Ameloblastoma

DEFINITION

Robinson defined as "Usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent."

WHO defined as "It is a true neoplasm of enamel organ type tissue which doesn't undergo differentiation to a point of enamel formation."

It is a benign but locally invasive polymorphic neoplasm consisting of proliferating odontogenic epithelium which is usually in a follicular or plexiform pattern, lying in fibrous stroma.

ETIOLOGY

- *Irritation*: It might be considered as one of the etiological factors as it often occurs in the posterior region of the mandible which is most susceptible to irritation.
- *Infection*: Robinson found that 1/3rd of the cases have history of oral infection, extraction of teeth and injuries to teeth.
- *Trauma*: Trauma can be a causative factor for ameloblastoma.

- *Dietary deficiency*: Dietary deficiency has been considered as a possible factor for, e.g. pronounced defect in development of tooth germ as seen in rickets may lead to irregularity in the ameloblastic layer.
- *Virus*: Infections from polyoma viruses have been shown to produce ameloblastoma like lesions in animals.

CLASSIFICATION

On pathological basis:
– Peripheral ameloblastoma
– Pituitary ameloblastoma
– Adamantinoma of long bones.

On histological basis:
– Follicular ameloblastoma
– Plexiform ameloblastoma
– Acanthomatous ameloblastoma
– Basal cell ameloblastoma
– Unicystic ameloblastoma
– Plexiform unicystic ameloblastoma
– Granular cell ameloblastoma
– Papilliferous ameloblastoma
– Hemangioameloblastoma
– Desmoplastic ameloblastoma
– Clear cell ameloblastoma
– Dentinoameloblastoma
– Melanoameloblastoma
– Keratoameloblastoma

Other classifications
1. Central/intraosseous
2. Peripheral/extraosseous.

PATHOGENESIS

According to Thomas and Wilms tumor arises from:
1. Developing enamel organ
2. Cell rests of enamel organ
   – Remnants of dental lamina
   – Remnants of Hertwig's sheath/Epithelial rests of Malassez.
3. Basal cells of surface epithelium of jaws
4. Hetropic epithelium in other parts of body especially pituitary gland
5. Epithelium of odontogenic cysts particularly dentigerous cyst and odontomas.

INCIDENCE

1 percent of all oral tumors
18 percent of all odontogenic tumors.

AGE

It is common between 20–50 years.
It is mostly seen in individuals younger than 40 years old.
Average age is 32.7 years.
Unicystic ameloblastoma is more common in 2nd and 3rd decade of life and the extraosseous form is more common in older age group.

SEX

No sex predilection.

SITE

It is more frequent in mandible than maxilla (2:1).

It develops in the molar-ramus area (approximately 3/4th of cases) in the mandible and also occurs in maxilla in 3rd molar area, followed by maxillary sinus and floor of the nose. The right side of the mandible is affected slightly more as compared to the left side.

SIZE

Size varies from 1 to 16 cm.

RACE

It is more common in Africans.

PRECEDING FACTORS

Neoplasm is frequently preceded by extraction of teeth, cystectomy or some other traumatic episode.

ONSET

It begins as a central lesion of the bone which is slowly destructive but tends to expand the bone rather than perforate it.

CLINICAL FEATURES

- It is generally asymptomatic in early stages but later on, it produces pain and swelling that is slow growing, hard, nontender, and ovoid in shape.
- Other features includes:
  - Mobility of teeth
  - Exfoliation/Root resorption
  - Ill fitting dentures
  - Malocclusion
  - Ulceration.
  - Nerve involvement in later stage leads to sensory changes like lower lip paresthesia.

  Tumor may produce expansion and thinning of cortex leading to "Egg shell crackling" (this shell of bone cracks when palpated).
- In maxilla, it leads to nasal obstruction, bleeding, trismus and maxillary sinus involvement. Maxillary lesions are more dangerous than mandibular lesions due to tendency of the former lesion to spread more extensively in the porous maxillary bone and possibility of the involvement of the cranial base.
- Central ameloblastoma has following features:
  - Grows in all direction and thins the surrounding bone.
  - Invasion of medullary space is the first feature.
  - As being nonencapsulated, it enlarges and invades the neighboring tissues rather than pushing them as seen in cysts.
  - Destroys the bone by pressure and distention or by osteoclastic resorption.
  - Locally invasive so compresses the vital structures, obstructs airway, and impairs swallowing.
  - May invade middle cranial fossa and causes gross facial deformity.

- Peripheral ameloblastoma has following characteristics:
  - It produces a small nodule with swelling entirely within the soft tissue.
  - It grows slowly without massive growth potential of intraosseous lesion.

SPREAD

Distant metastasis may occur in later stages.

Most common site is lung due to aspiration of tumor cells. Other sites are regional lymph nodes, liver, kidney, long bones, skull, vertebra, etc.

RADIOGRAPHIC FEATURES

- *Radiodensity*:
  In the early stages, there is area of bone destruction which is well-defined and is indicative of slow growth with hyperostotic borders.
- *Margins*:
  Outline is smooth, scalloped, well-defined and well-corticated. The walls of the cavity are coarse. In some cases, the margins of the tumor are devoid of bony covering.
- *Internal structure*:
  Usually it is multilocular but may be unilocular. Coarse or fine trabeculae may be present within the tumor and it is common for the free margins of the tumor to be devoid of bony covering.
- *Appearance*:
  There is presence of the septa in the lesion. In some cases, number and arrangement of the septa may give the area "Honey comb appearance" (numerous small compartments) or a "Soap bubble appearance" (larger compartments). In advanced stages, perforated cortical plate may contribute to a multilocular appearance.
- *Progress*:
  In the early stage ameloblastoma presents a bubble-like appearance with fairly large, round and distinct compartments. As the tumor grows and expands, the compartment may coalesce and fuse. With further increase in size of lesion, cortex expands and is destroyed.
- *Subclinical lesion*:
  A small subclinical lesion usually presents in radiographs as many small rounded cavities in the bone having sharply defined and sometimes corticated borders.
- Effect on surrounding structures:
  1. The jaws are likely to be enlarged, depending on overall size of the tumor.
  2. Extensive root resorption may occur.
  3. Thickening of membrane, cloudiness and destruction of walls are the findings when the sinus is involved.
  4. Expansion and thinning of cortical plate occurs leaving thin eggshell of bone.
  5. Perforation of bone is the late feature.
- Radiographic appearance can be of four types:
  1. Multiloculated multicystic (Figs 26.1 and 26.2)
  2. Solid trabeculated
  3. Unilocular
  4. Separate.
- Desmoplastic ameloblastoma (a variant) is almost radiopaque due to dense connective tissue content.
- Radiographic appearance is merely an adjunct to diagnosis. Occlusal view shows buccal and lingual expansion of the tumor.
- New imaging techniques like computerized tomography and MRI can demonstrate osseous and soft tissue pathology respectively.
- Definitive diagnosis is possible by microscopic examination.

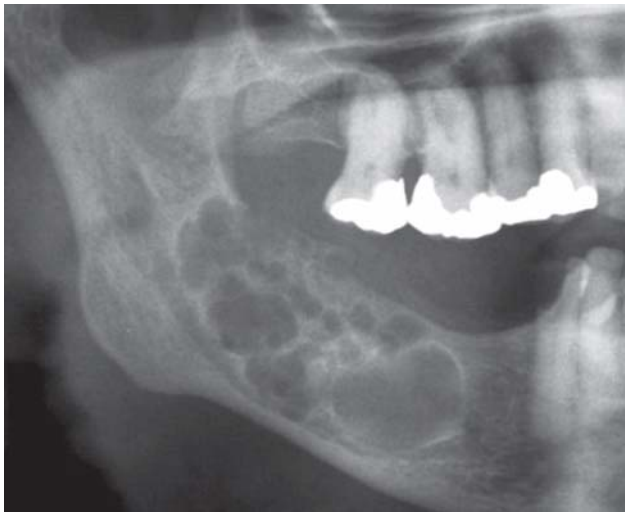


Fig. 26.1: Multiloculated appearance

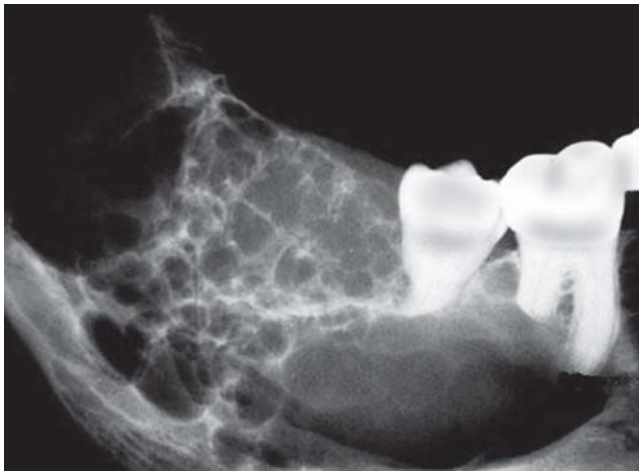


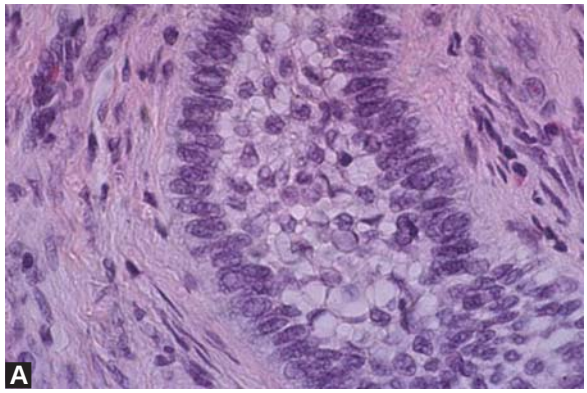
Fig. 26.2: Multiloculated appearance

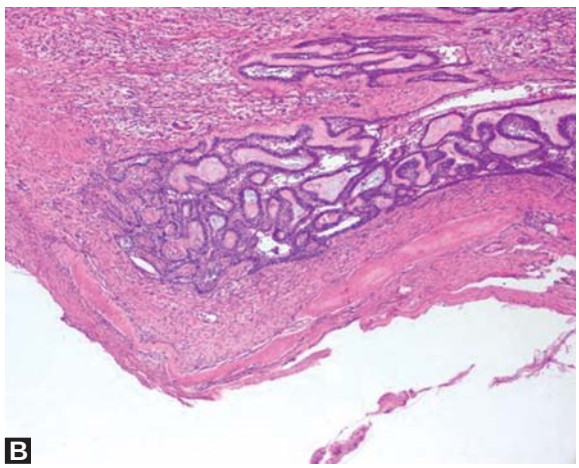
DIAGNOSTIC AIDS

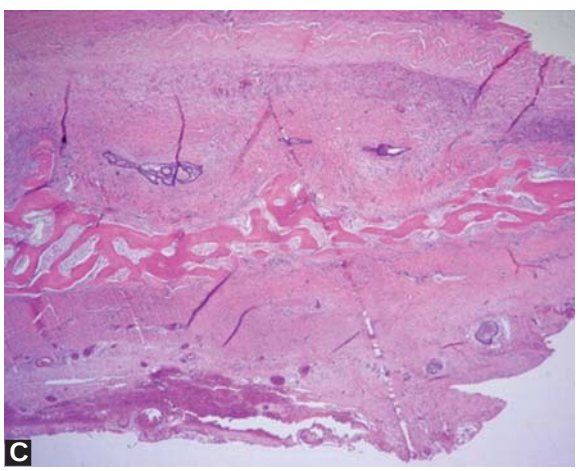
- History
- Age
- Signs and symptoms
- Bimanual palpation
- Radiographs (OPG, Lateral view, CT, MRI)
- Incisional biopsy including piece of normal bone tissue

HISTOLOGICAL FEATURES

- *Follicular type* is the most common. Central portion has loose network of polyhedral or spindle-shaped cells surrounded by tall columnar cells with nuclei polarized (responsible for high recurrence rate) and away from basement membrane (Figs 26.3A to C).
- Cystic degeneration of central portion leaves clear space lined by flat, stellate cells.







Figs 26.3A to C: Follicular type

- *Plexiform pattern* shows cords of columnar epithelial cells in double columns. Central stellate cells are arranged in periphery of areas of cystic degeneration.
- *Intraluminal ameloblastoma* grows into the lumen without violating the wall of cyst.
- *Mural ameloblastoma* is one which grows into the wall of cyst and remains limited within the wall.
- *Invasive ameloblastoma* extends beyond the wall of the cyst and into the adjacent bone or soft tissues.

MANAGEMENT

1. Treatment must be considered on the basis of behavior and potential of trauma.
   - Growth characteristic, if various physical forms are present
   - Anatomical site, if recurrence is evident
   - Clinical extent and size
   - Histological assessment.
2. Surgical (age must be taken in consideration)
3. First operation should be definitive and offering best opportunity for cure and with least psychological distress imposed on patient in future.
4. Curettage has highest recurrence rate.
   Intraosseous multicystic – 55 to 100 percent
   Intraosseous unicystic – 18 to 25 percent
5. Cancellous bone is easily invaded so there is more microscopic invasion into bone than that seen in radiograph.
6. Dense cortices – buccal, lingual and inferior borders are temporary barriers and can be easily destroyed by tumor.
7. Safe margin of normal bone of approximately 2 cm is resected.
8. Resorption of cortical plate permits growth into periosteum and once it is perforated soft tissue spread occurs.
9. Periapical connective tissue stroma whether dense/loose may circumscribe the lesion but does not encapsulate and tumor can grow through it.

SPECIFIC PRINCIPLES

Solid Multicystic Lesion

- Resection with or without continuity defect.
- Inferior alveolar nerve can be sacrificed, if within the lesion. Nerve grafting is planned and it is best to graft at the time of resection.
- Resection is always performed away from the tumor. Thin inferior border should be preserved, if possible. (It has high recurrence rate after previous operation due to reverse polarity of the cells).
- Sharp resection instruments permit radiologic assessment of resection margins.
- Immediate reconstruction should be planned, if soft tissue involvement is not there and if soft tissue involvement is present, then secondary reconstruction should be planned.
- Adequate soft tissue coverage in the tissue bed should be available to cover the graft. (If soft tissue invasion is present, second stage treatment is done otherwise bone graft gets separated and there is also less soft tissue to cover the bone).
- Maxillary lesion should be resected aggressively because of ease with which tumor spreads.

Intraosseous Unicystic Ameloblastoma

- It is diagnosed clinically and radiographically as cyst and often treated without biopsy.
- Careful X-ray examination may show solitary cyst.
- Complete enucleation is the key treatment and since it may impact deeply, adjunct treatment of bone bed is done by chemical fixation with Carnoy's solution.
- Curettage is discouraged.

Peripheral (Extraosseous)

- Excision biopsy is done with 1.5–2 cm of safety margin excision.

CHAPTER

27 Properties of X-rays

RADIATION

Radiation is mission and propagation of energy through a space or substance in the form of wave or particles.

X-RADIATION

X-rays can be defined as the weightless bundles of photons (energy) that are without an electrical charge and travel in waves with a specific frequency at the speed of light.

PROPERTIES OF X-RAYS

1. *Appearance*:
   X-rays are electromagnetic radiation. They are invisible and cannot be detected by any of the senses.
2. *Mass and charge*:
   X-rays have no mass or weight and no charge.
3. *Speed*:
   X-rays travel at the same speed of light in free space, i.e. 3×10^8 m/sec (1,86,000 miles/sec) and are not affected by magnetic field.
4. *Dualistic behavior*:
   X-rays show dualistic behavior, i.e. wave and particle. They are electromagnetic waves. According to quantum theory, X-rays are made up of discrete units of energy called quantum of photon. Each wave pocket is equivalent to its energy and is called photon. X-rays are made up of millions of photons of different energy.
5. *Propagation*:
   No medium is required for propagation of X-rays. The electric and magnetic fields are perpendicular to each other and perpendicular to the path of propagation.
6. *Frequency*:
   Their frequency ranges from 2×10^{16} sec^{-1} to 3×10^{19} sec^{-1}.
7. *Wavelength*:
   X-rays travel in waves and have short wavelength with a high frequency. Wavelength of X-ray used in diagnostic radiography is 0.1 to 0.5 Angstrom (Å). X-rays with shorter wavelength have greater penetrating power and are called hard rays. X-rays having relatively longer wavelength, possessing less energy and less penetrating power are called Grenz rays.
8. *Production*:
   X-rays belong to gamma rays group in the spectrum and are produced by interaction of particulate radiation (cathode rays) with matter. So, X-rays are produced by the conversion of electrical energy into radiation.

Wavelength	*X-ray*
1-2 Å	Grenz or Super soft X-rays
1-0.5 Å	Soft X-rays
0.5-0.1 Å	Medium X-rays
0.1 Å	Hard X-rays
0.001 Å	Gamma rays

9. *Path of travel*:
   X-rays travel in straight lines in wave motion and can be deflected or scattered.
10. *Diffraction and interference*:
    When a wave hits an object, the portion of wave that just missed the object spreads in a circle or sphere, into the space behind the object. The edges of the object allow the wave to 'curl round' behind the object (Figs 27.1A and B).

    Close to the object is an area in shadow where no wave exists. Further away the diffracted waves have spread until the waves from each edge begin to interfere with each other. Waves are also diffracted by narrow slits (of width less than wavelength). A wavelet is formed from the initial wave at the obstacle (Figs 27.2 and 27.3).
11. *Focusing capability*:
    X-rays cannot be focused to a point and they always diverge from a point.

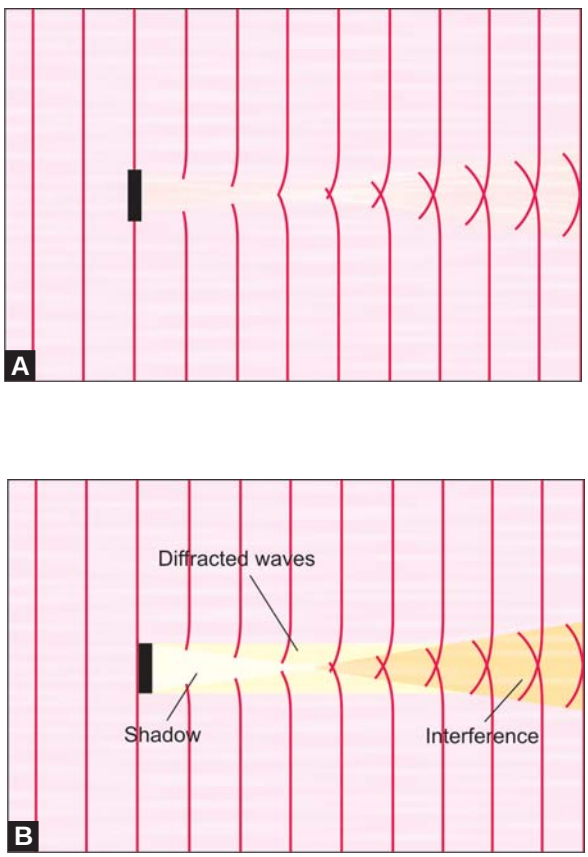


Fig. 27.1A and B: Diffraction

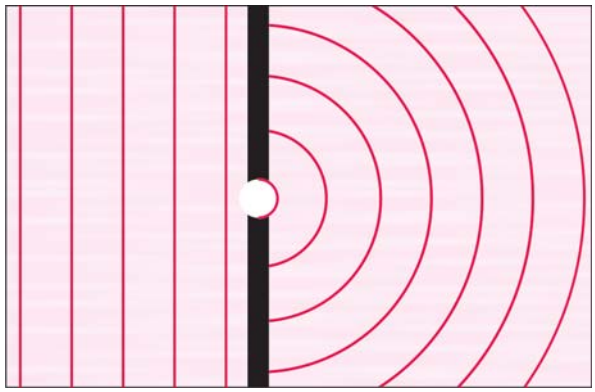


Fig. 27.2: Curling of wave

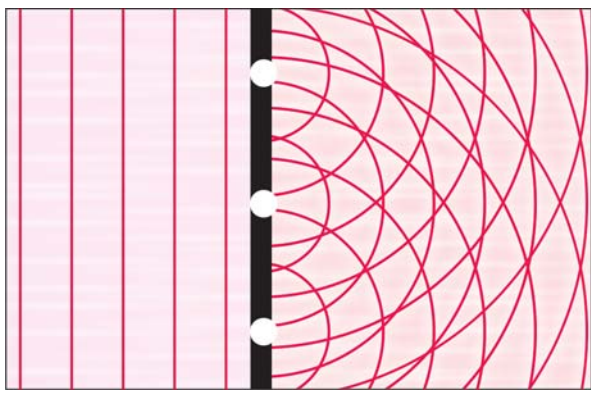


Fig. 27.3: Curling of multiple waves

12. *Energy*:
X-rays consist of wave pockets and its energy is equivalent to energy of electromagnetic radiation that originated at the atomic level. The energy level varies from 25 to 125 keV. Thus, energy of an X-ray depends on its wavelength. Shorter the wavelength, the more is the energy and higher the penetration power.
13. *Attenuation*:
X-rays shows selective attenuation. When passing through the matter, the intensity of radiation is reduced (attenuation) because the radiation energy is taken up by the material (absorption) and some deflection from the original path, to travel in a new direction (scattering).
Attenuation depends upon thickness of object, atomic number or density of object, etc.
14. *Scattering:*
When X-rays strike an object, some are scattered in all directions by atoms of the object. This secondary radiation is called scattered radiation. X-rays produce different types of scattered and secondary radiations. It is undesirable both for the operator (decreases the image quality) and for the patient (increases the radiation dose). Scattering depends upon the kilovoltage applied, the size and area to be examined, and the size of X-rays beam.
An X-ray which reflects from the surface of a substance travels less distance than an X-ray which reflects from a plane of atoms inside the crystal. The penetrating X-ray travels down to the internal layer, reflects, and travels back over the same distance before being back at the surface. The distance travelled depends on the separation

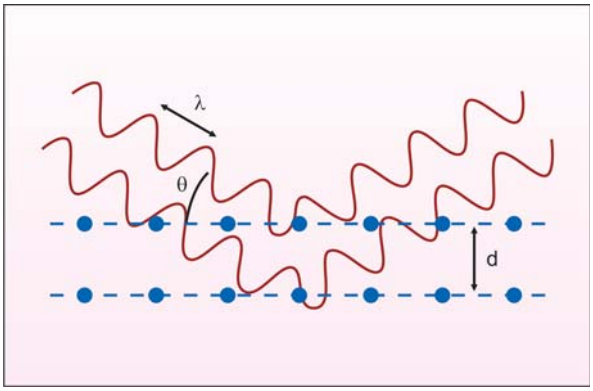


Fig. 27.4: Bragg's law

of the layers and the angle at which the X-ray entered the material. This is expressed by *Bragg's Law* (Fig. 27.4).

15. *Penetrating power (quality)*:
X-rays can penetrate a structure depending on its energy. More is the energy, more is the penetrating power. Penetrability also depends on the composition of structure, which determines whether X-ray beam will penetrate (pass through) or be absorbed. The quality of X-ray beam is determined by the kilovoltage, milliampere, distance between target and the object, time or length of exposure, filtration and target material. X-rays can penetrate and pass through many solids that are opaque to light but cannot penetrate through heavy metals and bones, and therefore cast a shadow of these objects on film. This property forms foundation for diagnostic property.

$$I \propto \frac{1}{D^2}$$

16. *Intensity*:
In free space, they obey the inverse square law, which states that for a point source of radiation, the intensity (I) at any given place varies inversely as the square of the distance (d) from the source to the place at which intensity are being considered.
17. *Ability to produce fluorescence*:
X-rays can cause certain substances to emit radiation or fluorescence (immediate emission of visible light after exposure to radiation). For example, visible light is emitted, when it is exposed on certain chemicals like phosphorus. Delayed emission of light after exposure to radiation is called phosphorescence. This property is useful in diagnostic radiography.
E.g. a) Intensifying screens
b) Fluoroscopy.
18. *Effect on film:*
They affect photographic film. For example X-rays can produce an image on photographic film.
19. *Ionization capability*:
X-rays interacts with materials they penetrate and cause ionization. They are used in photochemical for ionization of chemicals.
20. *Biologic effect*:
X-rays cause somatic or genetic changes in living tissue so it requires precautions in radiography. It produces biological damage in the living tissues. The effect manifests as either damage or death of cells.

CHAPTER

28 Considerations for Oral Medicine Viva Voce

Congenital disease
The disease which is present at or before the birth, but it is not necessarily inherited (i.e. transmitted through genes).

Hereditary conditions
Conditions that are apparent at birth but do not become evident for number of years after the birth are called hereditary conditions.

Agnathia
Agnathia is a lethal anomaly, characterized by hypoplasiaor absence of the mandible, with abnormally positioned ears having an autosomal recessive mode of inheritance.

Micrognathia
Micrognathia is an anomaly characterized by a small jaw, and either maxilla or mandible is affected.

Macrognathia
Macrognathia is a condition of abnormally large jaws.

Hemihyperplasia
Hemihyperplasia is a rare developmental anomaly characterized by asymmetric overgrowth of one or more body parts.

Fordyce's disease/granules
Heteropic collections of the sebaceous glands at various sites in the oral cavity (especially on the cheek mucosa opposite the molar region) are known as Fordyce's granules.

Cheilitis glandularis
It is an uncommon poorly, understood inflammatory disorder of lip, characterized by progressive enlargement and eversion of lower labial mucosa that results in obliteration of mucovermilion interface.

Median rhomboid glossitis
It is the congenital abnormalities of the tongue due to failure of tuberculum impair to retract before the fusion of the lateral halves of the tongue.
- It is seen as an ovoid, diamond, rhomboid-shaped smooth erythematous patch on the dorsal surface of the tongue immediately anterior to the circumvallate papillae.
- It has a strong relationship with the localized chronic infection especially Candidiasis.

Benign migratory glossitis

(Geographic tongue, Wandering rash of the tongue, Glossitis areata exfoliativa, Erythematic migrans).

– Unknown etiology (Emotional stress).
– Multiple areas of the desquamation of the filiform papillae of the tongue in a circinate pattern are seen.

Dentinogenesis imperfecta:

Type	*Characteristic*	
Type I	Dentinogenesis imperfecta without osteogenesis imperfecta Opalescent dentin Shields type II Capdepont teeth	Obliteration of pulp chamber
Type II	Bradywine type Shields type III Multiple pulp exposure in deciduous teeth	Large pulp space (Shell tooth)

Dentin dysplasia (Rootless teeth)

It is a rare disturbance of dentin formation characterized by normal enamel but atypical dentin formation with abnormal pulpal morphology.

Type	*Pulp characteristic*
Type I – Radicular dysplasia (Dentin dysplasia)	Deciduous – Completely obliterated Permanent – Crescent-shaped remnant
Type II – Coronal dysplasia (Anomalous dysplasia of dentin)	Deciduous – Obliteration Permanent – Large pulp chamber, Thistle tube appearance

Papilloma

It is an exophytic growth made, up of numerous, small finger-like projections, which results in a lesion with a roughened, verrucous or "cauliflower like" surface.

Nevus

It is a congenital, developmental tumor-like malformation of the skin or mucous membrane.

1. Congenital
2. Acquired
   – Intradermal nevus (Common mole)
   – Junctional nevus
   – Compound nevus
   – Spindle cell and/or epitheloid cell nevus
   – Blue nevus.

Leukoplakia

It is a white patch/plaque which cannot be characterized clinically/pathologically as any other condition.

Bowen's disease

It is a special form of intraepithelial carcinoma occurring particularly in patients who had an arsenical therapy.

Oral submucous fibrosis

It is an insidious chronic disease affecting any part of the oral cavity and sometimes the pharynx. Although occasionally preceded by and/or associated with vesicle formation,

it is always associated with a juxtaepithelial inflammatory reaction followed by a fibroelastic change of the lamina propria, with epithelial atrophy leading to stiffness of the oral mucosa and causing trismus and inability to eat.

Fibroma

It is a benign soft tissue neoplasm of the oral cavity characterized by excessive proliferation of fibroblast and synthesis of large amount of collagen.

Central giant cell fibroma of bone

It is a central neoplasm of the bone which is well demarcated, expansile, encapsulated and composed of cellular fibrous tissue containing spherical calcifications and randomly oriented bony structures.

Peripheral giant cell fibroma of bone

It is the common giant cell lesion which arises from tooth bearing areas of jaw and appears as a purplish—red nodule.

TNM Classification and Staging

The TNM classification, applicable to squamous cell carcinoma of the oral cavity, is based entirely on clinical findings and is developed by the American Joint Committee on Cancer and the UICC in 1978.

T—Tumor size

N—Regional lymph nodes

M—Distance metastasis

Surface dimensions of the primary tumor are included as the most important parameter for determining primary tumor staging in the oral cavity. The N staging system takes into account the size and multiplicity as well as the unilateral or bilateral presence of metastatic nodes as the parameters.

	T—Primary tumor
TX	Inability to meet the minimum requirements to assess the primary tumor
TO	No evidence of primary tumor
Tis	Carcinoma *in situ* (Preinvasive carcinoma)
T1	Tumor 2 cm or less in diameter (<2 cm)
T2	Tumor greater than 2 cm, not greater than 4 cm (>2 cm, <4 cm)
T3	Tumor greater than 4 cm in diameter (>4 cm)
T4	Tumor with extension to bone, muscle, skin, antrum neck, etc.
	N—Regional lymph nodes
NX	Inability to meet minimum requirements to assess the regional lymph nodes
N0	Clinically palpable lymph nodes, metastasis not suspected
N1	Clinically palpable homolateral cervical lymph nodes, not fixed, metastasis suspected
N2	Clinically palpable contralateral or bilateral lymph nodes, not fixed, metastasis suspected
$N2_a$	Metastasis in single ipsilateral lymph node, >3 cm but <6 cm in its greatest dimension.
$N2_b$	Metastasis in multiple ipsilateral lymph node, none >6 cm
$N2_c$	Metastasis in multiple bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
N3	Clinically palpable lymph nodes, **Fixed**, Metastasis suspected, >6 cm
	M—Distant metastasis
M_0	No distant metastasis
M_x	Inability to meet the minimum requirements to assess the presence of distance metastasis.
M1	Clinical and/or radiographic evidence of metastasis other than to cervical lymph nodes.

1. Pretreatment clinical classification (cTNM)
   Based on clinical data before treatment
2. Postsurgical histopathological classification (pTNM)
   Based on cTNM and surgical findings plus examination of the respected specimen.
   The stage groupings for the TNM system classification for oral cancer are:

Stage I	$T_1N_0M_0$
Stage II	$T_2N_0M_0$
Stage III	$T_3N_0M_0$, $T_1N_1M_0$ $T_2N_1M_0$ $T_3N_1M_0$
Stage IVA	$T_4N_0M_0$ $T_4N_1M_0$ Any T N_2M_0
Stage IVB	Any T N_3M_0
Stage IVC	Any T Any N M_1

New Classification

A newer staging system that includes the exact site (S) in the oral cavity and histopathologic grading (P). However, little is still known about the possible advantages of this S-TNM-P classification over the internationally accepted TNM classification.

S-TNM-P Classification

S – Site
T – Tumor dimension
N – Lymph node involvement
M – Distance metastasis
P – Histopathology.

Histopathological Grading (G)

G_X	Grade of differentiation cannot be assessed
G1	Well-differentiated
G2	Moderately-differentiated
G3	Poorly-differentiated
G4	Undifferentiated

Presence/absence of Residual Tumor

R_X	Presence of residual tumor cannot be assessed
R0	No residual tumor
R1	Microscopic residual tumor
R2	Macroscopic residual tumor

Reference: Rapidis AD, Langdon JD, Patel MF, et al.Clinical classification and staging in oral cancer. J Maxillofac Surg 1976,4:219-26.

Rapidis AD, Langdon JD, Patel MF, et al. A new system for the clinicopathological classification and identification of intraoral carcinomata. Cancer 1977,39:204-09).

Cyst
Cyst is a pathological cavity having fluid, semifluid, and gaseous contents which aren't created by accumulation of pus frequently and may or may not be lined by epithelium (Krumer 1974).

Dentigerous cyst
It can be defined as an odontogenic cyst that surrounds the crown of an impacted tooth; caused by fluid accumulation between the reduced enamel epithelium and the enamel surface, resulting in a cyst in which the crown is located within the lumen.

Epstein's pearls
These are cystic, keratin filled nodules along the midpalatine raphe, probably derived from entrapped epithelial remnants along the line of fusion.

Odontogenic keratocyst
A cyst derived from the remnants of the dental lamina, with a biologic behavior similar to a benign neoplasm, with a distinctive lining of six to ten cells in thickness, and that exhibits a basal cell layer of palisaded cells and a surface of corrugated parakeratin.

Dental lamina cyst of newborn
They are multiple, occasionally solitary, superficial raised nodules on edentulous alveolar ridges of infants that resolve without treatment; derived from rests of the dental lamina and consisting of keratin-producing epithelial lining.

Gingival cyst of adult
A small developmental odontogenic cyst of the gingival soft tissue derived from the rests of the dental lamina, containing a lining of embryonic epithelium of cuboidal cells and distinctive focal thickenings similar to the lateral periodontal cyst.

Lateral periodontal cyst
A slow growing, nonexpansile developmental odontogenic cyst derived from one or more rests of the dental lamina, containing an embryonic lining of one to three cuboidal cells and distinctive focal thickenings.

Eruption cyst
It is defined as an odontogenic cyst with the histologic features of a dentigerous cyst that surrounds a tooth crown that has erupted through bone but not through soft tissue and is clinically visible as a soft fluctuant mass on the alveolar ridges.

Bohn's nodules
They are keratin filled cysts scattered over the palate, most numerous along the junction of hard and soft palate and apparently derived from palatal salivary gland.

Ameloblastoma (Adamantinoma)
It is a true neoplasm of enamel organ type tissue which does not undergo differentiation to the point of enamel formation.

Robinson defined it as
"Usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent" *(Key to memorize - UNIABC).*

Attrition
It is the physiologic wearing of tooth surface as a result of tooth-to-tooth contact, as in mastication.

Abrasion
It is the pathologic wearing of tooth surface by some mechanical forces such as hard bristle toothbrush, coarse tooth powder, etc.

Erosion
Erosion is the loss of tooth structure by means of some chemical action that does not involve bacterial action.

Papule
Papule is *raised* solid lesion smaller than 1 mm in diameter.

Plaque
Plaque is *raised* solid lesions greater than 1mm diameter.

Macule
Macule is well-circumscribed, *flat* lesions that are noticeable because of change from normal skin.

Nodules
Nodules are the small tumor-like lesions present deep in dermis.

Vesicle
Vesicle is elevated blister-like lesions containing clear fluid that are less than 1 cm in diameter.

Bulla
Bulla is elevated blister-like lesions containing clear fluid that are over 1 cm in diameter.

Ulcer
Ulcer is a brick or discontinuity in the surface epithelium – skin or mucous membrane, which appears as a well-circumscribed depressed lesion. Generally, they have fibrotic margin and a bed of granulation tissue.

Carcinoma
Carcinoma is a malignant tumor of epithelial tissue origin.

Epidermoid carcinoma (Squamous cell carcinoma)
It is defined as, "a malignant epithelial, neoplasm exhibiting squamous differentiation as characterized by the formation of keratin and/or the presence of intercellular bridges" (Pindborg JJ, et al. 1977). It is flat, slowly growing, mildly malignant and rapidly infiltrated tumor of skin.

Sarcoma
It is a rare cancerous tumor arising from connective tissues such as muscle/ bone.

Dysplasia
Abnormal development of tissue is called as dysplasia. It represents the earliest changes of neoplastic transformation than can be detected at the microscopic level.

Anaplasia
Loss of differentiation and function is called anaplasia. Anaplastic lesions are composed of cells, which resemble those of the tissue from which they arise.

Parasitic Infections

Protozoa (Unicellular)	
Plasmodroma	Chilophora

Helminths (Multicellular)		
Nemathelminthes (roundworms)nematoda	*Platyhelminthes*	
	Cestoda (tapeworms)	Trematoda (flukes)

Dental caries

It is an irreversible, infectious microbial disease of the calcified tissues of the teeth characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth, which often leads to cavitation.

Zone of enamel caries:

Zone 1: *Translucent zone* lies at advancing front of enamel lesion
Zone 2: *Dark zone* lies adjacent and superficial to the translucent zone
Zone 3: *Body of the lesion* is greatest area of demineralization
Zone 4: *Surface zone* which is relatively unaffected because of greater degree of mineralization

Zone of advanced dental caries:

Zone 1: Zone of *fatty degeneration* of tomes fibers.
Zone 2: Zone of *dentinal sclerosis* characterize by deposition of Ca^{++} in dentinal tubules
Zone 3: Zone of *decalcification* of dentin, a narrow zone preceding bacterial invasion
Zone 4: Zone of *bacterial invasion* in decalcified but intact dentin
Zone 5: Zone of *decomposed dentin*

Cellulitis

It is a diffuse inflammation of soft tissues which is not circumscribed or confined to one area, but which, in contradistinction to the abscess, tends to spread through tissue spaces and along fascial planes.

Ludwig's angina

By Archer,

It is bilateral, acute, rapidly spreading, septic, inflammatory, indurated, wooden hard cellulites of floor of mouth.

By thoma,

It is a gangrenous cellulitis of loose alveolar tissue which originates in the submandibular space and spread rapidly towards the floor of mouth.

By Killey, Seward and Kay,

It is a clinical diagnosis and the name given to the massive brawny cellulitis occurring bilaterally at the submandibular region which also involves sublingual space.

Bruxism

It is a habitual grinding of the teeth either during sleep or as an unconscious habit during waking hours.

Biopsy

It is the removal of tissue from the living organism for the purpose of microscopic examination and diagnosis.

Porphyria

It is inborn error of porphyrin metabolism characterized by over production of uroporphyrin and related substances.

Juxtaoral organ of Chievitz
Clusters of nests of squamous epithelial cells found microscopically in tissue taken from approximate site used by dentist for injection of inferior alveolar nerve.

TYPES OF GEOGRAPHIC TONGUE

Type I
"Normal" geographic tongue, in which the lesion on the tongue change from time to time, without lesions of the mucosa elsewhere in the mouth.
Type II
Similar to type I but associated with similar lesions elsewhere in the mouth.
Type III
Lesions on the tongue not so typical as in type I and may or may not be associated with lesions elsewhere in the oral cavity.

There are two subtypes:

a. Fied form—Only a few areas of the tongue are affected. The lesions come and go, but always appear in the same place.
b. Abortive form—The lesions start as white-yellow spots, but disappear before taking the classical appearance of geographic tongue.

Type IV
No lesion on the tongue—Lesions elsewhere in the oral mucosa resemble those of geographic tongue.
(Reference: Hume WJ, Smith CJ, Franklin DC. Verruciform xanthoma. Br J Oral Surg 1980, 8:157-61).

Disease	*Etiology*
Actinomycosis	*Actinomycosis israeli*
Acute lymphonodular pharyngitis	Coxsackie virus A10 group
Candidiasis	*Candida albicans*
Coccidioidomycosis	*Coccidioides immitis*
Diphtheria	Cornybacterium diphtheria Klebs-Loeffler bacillus
Gonorrhea	*Neisseria gonorrhoeae*
Granuloma inguinale	*Donovania granulomatis*
Herpangina	Coxsackie virus A group Type 1, 6, 8, 10, 16, 22
Histoplasmosis	*Histoplasma capsulatum*
Hand-foot-mouth disease	Coxsackie virus A16 group, A5 and A6, occasionally B2,B5
Leprosy	*Mycobacterium leprae*
Mumps	*Paramyxovirus*
North American blastomycosis	*Blastomyces dermatitidis*
Rhinoscleroma	*Klebsiella rhinoscleromatis*
Scarlet fever	*β-hemolytic streptococci*
Syphilis	*Treponema pallidum*
South American blastomycosis	*Blastomyces brasiliensis*
Tularemia	*Pasteurella tularensis*
Tetanus	*Clostridium tetani*
Tuberculosis	*Acid-fast Mycobacterium tuberculosis*

Disease	Synonyms
Acute necrotizing ulcerative gingivitis	Vincent's infection Trench mouth
Agranulocytosis	Granulocytopenia
Amelogenesis imperfecta Ameloblastoma	Hereditary enamel hypoplasia, Hereditary opalescent teeth Adamantinoma
Auriculotemporal syndrome	Frey's syndrome Gustatory sweating
Recurrent aphthous minor	Canker sore
Recurrent aphthous major	Mikulicz's aphthae, Sutton's disease
Basal cell carcinoma	Rodent ulcer
Burkitt's lymphoma	African jaw lymphoma
Bifid rib-Jaw cyst-Basal cell nevus syndrome	Gorlin-Goltz syndrome, Basal cell nevus syndrome
Benign migratory glossitis	Geographic tongue, Wandering rash, Aveata exfoliativa, Erythema migrans
Chelitis granulomatosa	Miescher's syndrome
Candidiasis	Thrush, Moniliasis
Cylindroma	Adenocystic carcinoma
Calcifying odontogenic cyst	Gorlin cyst, Calcifying epithelial odontogenic cyst
Calcifying epithelial odontogenic tumor	Pindborg's tumor
Cementoma	Periapical cemental dysplasia
Chronic focal sclerosing osteomyelitis	Condensing osteitis
Chronic osteomyelitis with proliferative periostitis	Chronic focal sclerosing osteomyelitis
Cherubism	Familial fibrous dysplasia of jaw
Cleidocranial dysplasia	Marie and Sainton's disease
Craniofacial dysostosis	Crouzon disease
Cyclic neutropenia	Periodic neutropenia
Dental lamina cyst of newborn	Epstein's pearls, Bohn's nodules, Gingival cyst of newborn
Developmental lingual salivary gland depression	Stafne's cyst, Latent bone cyst, Static bone cavity, Static bone cyst
Dentinal dysplasia	Rootless teeth
Dentinogenesis imperfecta	Hereditary opalescent dentin
Dens in dente	Dens invaginatus

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Disease	*Synonyms*
Dens evaginatus	Occlusal enamel pearl, Leongs premolar
Dentigerous cyst	Follicular cyst
Developmental lingual mandibular salivary gland depression	Static bone cyst, Latent bone cyst, Static bone
Down syndrome	Trisomy 21
Encephalotrigeminal angiomatosis	Sturge-Weber disease
Epidermoid carcinoma	Squamous cell carcinoma
Erythroplakia	Erythroplasia of Queyrat
Ewing's sarcoma	"Round cell" sarcoma
Facial hemiatrophy	Parry Romberg syndrome
Fibromatosis gingivae	Elephantiasis gingivae
Fissured tongue	Scrotal tongue
Focal epithelial hyperplasia	Heck's disease
Focal dermal hyperplasia syndrome	Goltz-Gorlin syndrome
Generalized cortical hyperostosis	van Buchem disease
Herpes simplex	Acute herpetic gingivostomatitis, Herpes labialis, Fever blister, Cold sores
Herpangina	Apthous pharyngitis
Hemophilia	Bleeder's disease, Disease of Kings, Disease of Haspburgs
Herpes zoster	Shingles, Zona
Histoplasmosis	Darling's disease
Hereditary hemorrhagic telangiectasia	Rendu-Osler-Weber syndrome
Hereditary intestinal polyposis syndrome	Peutz-Jeghers syndrome
Horner's syndrome	Sympathetic ophthalmoplegia
Hypercementosis	Cementum hyperplasia
Internal resorption	Pink tooth of mummery Odontoclastoma
Intraepithelial carcinoma	Carcinoma *in situ*
Infantile cortical hyperostosis	Caffey's disease
Infectious mononucleosis	Glandular fever
Jaw cyst-Basal cell nevus-Bifid rib syndrome	Gorlin and Goltz syndrome
Jaw-winking syndrome	Marcus Gunn phenomenon
Juvenile periodontitis	Periodontosis
Kaposi's sarcoma	Angioreticuloendothelioma

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Disease	*Synonyms*
Keratoacanthoma	Self-healing carcinoma
Keratosis follicularis	Darier's disease
Leprosy	Hansen's disease
Massive osteolysis	Vanishing bone disease
Mandibulofacial dysostosis	Treacher Collins syndrome
Median anterior maxillary cyst	Nasopalatine duct cyst, Incisive canal cyst
Median rhomboid glossitis	Central papillary atrophy of tongue
Mumps	Endemic perotitis
Measles	Rubeola
Mucormycosis	Phycomycosis
Multiple myeloma	Plasma cell myeloma, Plasmacytoma Kahler's disease
Mikulicz's disease	Benign lymphoepithelial lesion
Nasoalveolar cyst	Klesdat's cyst
Neurolemmoma	Schwannoma, Neurolemmoma
North American Blastomycosis	Gilchrist's disease
Noma	Cancrum oris, Gangrenous stomatitis
Osteogenesis imperfecta	Brittle bones, Osteopsathyrosis
Osteopetrosis	Marble bone disease Albers-Schonberg disease
Osteitis deformance	Paget's disease
Pernicious anemia	Addison's disease
Pits of lower lip and cleft of lip or palate	vander Woude's syndrome
Periapical granuloma	Apical periodontitis
Palatal cyst of neonate	Epstein pearls, Bohn's nodule
Peripheral cemental dysplasia	Cementoma, Perapical fibrous dysplasia
Peripheral ossifying fibroma	Peripheral odontogenic fibroma, Peripheral cementifying fibroma
Peripheral giant cell granuloma	Giant cell epulis, Reparative granuloma
Pleomorphic adenoma	Mixed tumor
Rubella	German measles
Recurrent aphthous stomatitis	Canker sores
Regional odontodysplasia	Ghost teeth, Odontogenesis imperfecta, Odontodysplasia

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Disease	*Synonyms*
Radicular cyst	Apical periodontal cyst, Periapical cyst, Dental root end cyst
Sjögren's syndrome	Sicca syndrome
Small pox	Variola
Chicken pox	Varicella
South American Blastomycosis	Lutz's disease
Syphilis	Lues
Sphenopalatine neuralgia	Lower-half headache, Horton's syndrome, Cluster headache, Periodic migrainous neuralgia, Atypical facial neuralgia, Vidian nerve neuralgia
Stomatitis nicotina	Pipe smoker's palate
Squamous cell carcinoma	Epidermoid carcinoma
Systemic sclerosis	Scleroderma
Thalassemia	Cooley's anemia Mediterranean disease
Tetanus	Lock jaw
Traumatic neuroma	Amputation neuroma
Traumatic cyst	Solitary bone cyst, Hemorrhagic cyst, Unicameral bone cyst, Simple bone cyst, Idiopathic bone cavity
Trigeminal neuralgia	Tic douloureux, Trifacial neuralgia, Fothergill's disease, Six inch syndrome
Uveoperotid fever	Heerfordt's syndrome
von Willebrand's disease	Pseudohemophilia
van Buchem syndrome	Generalized cortical hyperostosis
Warthin's tumor	Papillary cyst adenoma lymphomatosum
White sponge nevus	Canon's disease

Cells	*Histology*	*Seen in*
Arbiskov cells	Modified monocytes	Myeloblastoma
Asteroid bodies	–	Spirotrichosis
Anitschkow cells	Modified epithelial cells with elongated nuclei containing linear bar of chromatin with radiating processes of chromatin extending towards the nuclear membrane	Aphthous ulcer Sickle cell disease Iron deficiency anemia
Angulate body cells	–	Granular cell myeloblastoma
Corp, ronds, grains	–	Benign dyskeratotic cells
Civatte/cytoid/hyaline /colloid bodies	–	Lichen planus

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Cells	*Histology*	*Seen in*
Döhle bodies	–	Chédiak-Higashi syndrome
Downey cells	–	Infectious mononucleosis
Foam cells	Lipid laden cells	Periapical granuloma
Gaucher's cells	Pale cells containing eccentric nucleus and a wrinkled or "crumpled silk" cytoplasm	Gaucher's disease
Ghost cells	Eosinophilic swollen epithelial cells without nucleus	Compound odontoma Ameloblastic fibroodontoma Craniopharyngioma Gorlin cyst
Hyaline cells	Modified myoepithelial cells	Pleomorphic adenoma
Howell Jolly bodies Cabot's ring	–	Pernicious anemia
Handerson Peterson bodies	Eosinophilic intracytoplasmic	Molluscum contagiosum
Molluscum bodies	inclusion bodies	
Hurler cell/Clear/ Gorgyle cells	Fibroblasts	Hurler's syndrome Mucopolysaccharidoses
Lipschutz bodies	Eosinophilic, ovoid, homogenous material which displace nuclear chromatin peripherally	Primary herpetic stomatitis
Lacunar cells	B-lymphocyte (Histologic variant of Reed-Sternberg cells)	Nodular sclerosis type of Hodgkin's disease
LE cells	Rosette of neutrophils surrounding pale nuclear mass derived from lymphocyte	Systemic lupus erythematosus
Lepra cells	Vacuolated macrophages or histiocytes	Lepromatous leprosy
Niemann-Pick cells	Lipid laden cells distributed throughout reticuloendothelial system	Niemann-Pick disease
Negri bodies	–	Rabies
Pale- dark cells	–	Odontogenic myxoma
Reed-Sternberg cell	Multinucleated cells, derived from B-lymphocyte or monocyte machrophage	Malignant cells of Hodgkin's Disease
Racquet cells Ribbon cells	Modified spindle cells	Rhabdomyosarcoma
Reily bodies	Metachromatic granules in lymphocyte	Hurler's syndrome
Russel bodies	Immature plasma cells unable to produce antibody	Seen in chronic inflammatory disease like periapical granuloma, multiple myeloma

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Cells	*Histology*	*Seen in*
Rushton bodies	–	In lining epithelium of odontogenic cyst
Tzanck cells	Multinucleated giant cell. Characterized by degenerative changes which include swelling of nuclei and hyperchromatic staining. Clumps of epithelial cells	Herpes simplex, Varicella (Chicken pox), Pemphigus vulgaris, Granular cell myoblastoma
Target cells, Safety pin cells	RBC	Thalassemia
Verocay bodies	–	Neurilemmoma/Schwannoma

Characteristic Signs	*Disease*
Auspitz's sign- Dry papule covered by silver scales which on removal leaves tiny bleeding points	Psoriasis
Blue sclera	Osteogenesis imperfect Marfan's syndrome Ehlers-Danlos syndrome Osteopetrosis
Bence Jones protein-Unusual protein which coagulates when urine is heated to 40°C to 60°C and disappear when urine is boiled.	Multiple myeloma
C-shaped/ Chinese character trabeculae	Monostotic fibrous dysplasia
Carpet tack extension of lesion	Discoid lupus erythematosus
Café-au-lait spots (Brownish melanin pigmentation)	Polyostotic fibrous dysplasia Neurofibroma, Peutz-Jegher's syndrome Hypothyroidism
Crowe's sign (Axillary freckling)	von Recklinghausen's neurofibromatosis
Centrifugal growth pattern of bone (tumor grows by expansion equally in all directions resulting in a round tumor mass)	Central ossifying fibroma Central cementifying fibroma Central cement-ossifying fibroma
Cotton wool appearance	Paget's disease
Egg cell crackling	Ameloblastoma, Osteosarcoma
Hypermobility of joints	Ehlers-Danlos syndrome Marfan's syndrome Osteogenesis imperfect Down syndrome
Herald spot	Ptyriasis rosea
Koplik's spots	Measles
Liesegang rings	Pindborg tumor

Contd....

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Characteristic signs	*Disease*
Mouse eaten furrowed ulcer	Oral tuberculosis
Mosaic pattern of bone Jig saw puzzle appearance of bone	Paget's disease
Monro's abscess	Psoriasis, Benign migratory glossitis
Nikolsky's sign-Epithelium readily strips off from connective tissue on nonulcerated area on pressure leaving raw sensitive surface which readily bleeds.	Pemphigus, Desquamative gingivitis, Haily-haily disease Epidermolysis bullosa
Palmar plantar keratosis	Gorlin-Goltz syndrome Papillon-Lefèvre syndrome Follicular keratosis
Pseudoepitheliomatous hyperplasia	Granular cell myoblastoma
Pink tooth	Internal resorption
Punched out lesion-Crest of interdental papillae destroyed forming hallow out area	ANUG
Target lesion, Iris, Bull's eye	Erythema multiforme

Appearance	*Disease*
Angelic look/Eyes towards heaven	Cherubism
Bird-like face	Mandibulofacial dysostosis, Pierre Robin's syndrome Bilateral TMJ ankylosis
Buffalo hump, Moon's face Butterfly distribution of lesion	Cushing's syndrome Discoid or systemic lupus erythematosus
Bumpy lips	Type III MEN syndrome
Cart wheel/Checker board appearance	Multiple myeloma
Claw hand	Scleroderma, Hurler's syndrome
Coup-de-sabre appearance	Scleroderma
Cobblestone appearance	Keratosis follicularis, Pyostomatitis vegetance
Fish face appearance	Treacher Collins syndrome
Herculean appearance	Congenital myotonia
Hebra nose	Rhinoscleroma
Honey comb appearance, Swiss cheese pattern	Cylindroma
Mask-like face	Scleroderma, Bell's palsy
Parrot's beak	Craniofacial dysostosis, Syphilis
Petrified man	Generalized myositis ossification
Rubber man	Ehlers-Danlos syndrome
Risus sardonicus, Opisthotonus	Tetanus
Starry sky appearance	Burkitt's lymphoma

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Appearance	*Disease*
Saw tooth appearance	Lichen planus
Sarrowful appearance	Myasthenia gravis
Sailor's skin/Farmer's skin	Solar elastosis
Spidery fingers	Marfan's syndrome
Swan neck	Dystrophic myotonia

Skin disease	*Characteristic features*
Ectodermal dysplasia	- Congenital dysplasia of one or more ectodermal structures - Hypohydrosis, hypotrichosis, hypodontia
Lichen planus	- Whickham's stria, bilateral symmetrical distribution of lesion with female predilection - Saw tooth retepegs, liquefaction degeneration of basal layer - Colloid/ civatte bodies - Grinspan's syndrome
Psoriasis	- Auspitz's sign, painless nonpruritic papules - No involvement of oral mucosa and Monro's abscess
Pityriasis rosea	- Herald spot
Erythema multiforme Keratosis follicularis (Darier disease)	- Target iris/Bull's eye lesion - Recent history of herpes simplex infection - Stevens-Johnson syndrome, Toxic epidermal necrolysis - Corps, ronds and grains - Leafing out pattern of parabasal cells - Cobblestone appearance - Characteristic nail changes - Typical suprabasal cleavage
White sponge nevus	- Hyperparakeratosis, acanthosis - Thickened and folded mucosa with soft, spongy texture, peculiar white opalescent hue
Pemphigus	- Nikolsky's sign, Tzank cells, Suprabasilar spilt
Cicatrial pemphigoid/ Benign mucous membrane pemphigoid	- Separation of basement membrane with epithelium from the underlying connective tissue - Lesion of skin are rare except on genitalia
Familial benign chronic pemphigus (Hailey-hailey disease)	- Nikolsky's sign is positive - Dilapidated brick wall defect
Lupus erythematosus	- Butterfly distribution of lesion - Carpet tack extensions - LE phenomenon, lupus band test
Systemic sclerosis of hands	- Mask-like appearance of the face and claw-like appearance - CREST syndrome - Extreme widening of periodontal ligament
Ehlers-Danlos syndrome	- Hyperelasticity of skin (rubber man), hyperextensibility of joints, defective healing - Cutaneous fragility

Contd....

Bone disease	*Characteristic features*
Osteogenesis imperfecta	- Extreme fragility and fractures of bones, blue sclera, brownish or yellowish discoloration of teeth - Abnormality of phosphorylase enzyme
Infantile cortical hyperostosis	- Hyperostosis, tender, deeply placed soft tissue swellings - Seen mainly in infants - Mandible and clavicle are more frequently affected
Cleidocranial dysplasia	- Brachycephalic skull, abnormalities of clavicle - Presence of wormian bones, open fontanels - Sagittal sutures remains sunken - Hypermobility of shoulder joint due to absence of clavicle - Multiple unerupted supernumerary teeth - Absence of cellular cementum
Craniofacial dysostosis	*Cranial* Early synostosis of sutures, small maxilla, triangular frontal defect *Facial* Hypoplasia of maxilla (Class III malocclusion) Parrot beak appearance, Hypertelorism
Mandibulofacial dysostosis	- Antimongoloid palpebral fissure, Hypoplasia of malar bones and mandible, facial clefts, malformation of ears - Fish/bird like face, deficiency of eyelashes
Pierre Robin syndrome	- Cleft palate, micrognathia, glossoptosis, bird face
Marfan syndrome	- Defective organization of collagen - Excessive length of tubular bones, long and narrow skull and face - Cardiovascular complications - Multiple odontogenic cyst
Down syndrome	- Trisomy of 21 - Mongoloid faces, subnormal mentality, large mandible, macroglossia - Low caries incidence than in normal people and high incidence of periodontal disease
Osteopetrosis	- Endosteal bone formation along with lack of normal bone resorption - Pathologic fracture of bones - Elevated level of acid phosphatase - Medullary cavities replaced by bone, thickened cortex predisposing to osteomyelitis
Achondroplasia	- Dwarfism, disturbance of endochondral bone formation - Brachycephalic skull, bowed legs, limited joint motion - Premature fusion of bones - Mandibular prognathism (CL III)
Paget's disease	- Enlargement of skull and jaw bones, predilection for maxilla - Compression of nerves in their foramina - Headache, deafness, blindness, bone pain, facial paralysis - Both osteolytic and osteoblastic lesion - "Mosaic and jigsaw puzzle" appearance of bone on histological examination, reversal lines are seen - "Cotton wool appearance" on radiograph

Contd....

Bone disease	*Characteristic features*
	- Isolated lesions are called as "osteoporosis circumscripta" - Hypercementosis, loss of lamina dura - Elevated alkaline phosphatase level - Increased susceptibility to development of osteosarcoma
Massive osteolysis	- Also called vanishing disease of bone - Resorption of bone with ultimate total disappearance of bone
Fibrous dysplasia of bone	*Polyostotic* (Jaffe's type/ Albright syndrome) - Lesion involving more than one bone - Albright type is associated with endocranial disturbances, Café-au-lait spots *Monostotic* - Lesion involving one bone - Chinese letter shaped trabeculae - Mottled, ground glass or peau d'orange appearance
Cherubism	- Chubby face, angelic look of eyes towards of heaven appearance - Premature exfoliation of deciduous teeth in 3 years of age - Teeth appear as "floating in cystic spaces" on radiograph - Presence of giant cells in histologic examination

Radiographic appearance	*Disease*
Crew cut Hair on end appearance	Thalassemia, Sickle cell anemia
Crescent-shaped pulp remnants Thistle tube pulp chamber	Dentinal dysplasia
Cherry blossom	Sjögren's syndrome
Driven snow appearance	Pindborg tumor
Floating teeth	Cherubism
Ghost teeth	Regional odontogenic dysplasia
Ground glass appearance	Monostotic fibrous dysplasia Hyperparathyroidism
Heart-shaped radiolucency	Median anterior maxillary cyst
Honey comb appearance	Ameloblastoma Pindborg tumor Odontogenic keratosis Aneurysmal bone cyst Central giant cell granuloma
Hypercementosis	Paget's disease Hyperpituitarism
Match band	Incipient caries
Mirror image-shaped vertical bone loss	Localized juvenile periodontitis
Moth eaten appearance Orange peel appearance Onion skin appearance	Chronic osteomyelitis Monostotic fibrous dysplasia Ewing's sarcoma Garre's osteomyelitis Caffey's disease

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Radiographic appearance	*Disease*
Periapical radiolucency Irregular Regular	 Periapical abscess Periapical granuloma/Periapical cyst
Pear-shaped radiolucency between lateral incisor and canine	Globulomaxillary cyst
Punched out radiolucent area in bone, regular Punched out radiolucent area in bone, irregular	Multiple myeloma Eosinophilic granuloma
Soap bubble appearance	- Ameloblastoma - Aneurysmal bone cyst - Central giant cell granuloma - Hemangioma - Odontogenic myxoma - Odontogenic keratocyst
Sunray appearance	Osteosarcoma Central hemangioma Ewing's sarcoma
Shell tooth	Dentinogenesis imperfecta Type III (radicular)
Tennis racket appearance	Odontogenic myxoma
Thistle tube appearance of tooth	Dentinogenesis imperfecta Type II (coronal)
Widening of periodontal ligament	Osteosarcoma Scleroderma Juvenile periodontitis
Obliteration of periodontal ligament space	Paget's disease Hypercementosis Ankylosis Hypofunction of tooth
Onion peel appearance	Ewing's sarcoma
Orange peel appearance	Monostotic fibrous dysplasia

SYNDROMES WITH CHARACTERISTIC FEATURES

Albright syndrome:

Polyostotic fibrous dysplasia
Café-au-lait pigmentation
Precocious puberty.

Aldrich's syndrome:

Thrombocytopenic purpura, eczema, increased susceptibility to infection (immune deficiency), bloody diarrhea (secondary to thrombocytopenia).

(It is also sometimes called as *eczema-thrombocytopenia-immunodeficiency syndrome* in keeping with Aldrich's original description in 1954).

Ascher's syndrome (ABN)

A—Acquired double lip
B—Blepharochalasis
(Drooping of the eyelid between eyebrow and the edge of the upper eyelid caused by relaxation of the supratarsal fold a result of the atrophy and thinning of the skin)
N—Nontoxic thyroid enlargement

Apert's syndrome

Crouzan's disease with syndactyly

Behçet's syndrome

Recurrent *oral* ulcers, recurrent *genital* ulcers, *ocular* inflammation.
[Behçet disease (Behçet's syndrome, Morbus Behçet, Silk Road disease) is a form of vasculitis that can lead to ulceration and other lesions. It can be interpreted as a chronic disturbance in the body's immune system. This system, which normally protects the body against infections through controlled inflammation, becomes overactive and produces unpredictable outbreaks of exaggerated inflammation. This extra inflammation affects blood vessels, usually the small ones. As a result, symptoms occur wherever there is a patch of inflammation, and can be anywhere where there is a blood supply].

Beckwith's hypoglycemic syndrome

Macroglossia
Neonatal hypoglycemia
Mild microcephaly
Umbilical hernia
Fetal visceromegaly
Postnatal somatic gigantism.

B-K mole syndrome

Large pigmented nevi and high risk of development of *melanoma.*

Bowen's disease

Special form of intraepithelial carcinoma occurring on skin particularly in patients who have had arsenic therapy.

(It is also known as "Familial atypical multiple mole–melanoma (FAMMM) syndrome", "Familial melanoma syndrome," and " Dysplastic nevus syndrome". It is a cutaneous condition described in certain families, and characterized by unusual nevi and multiple inherited melanomas).

Chédiak-Higashi Syndrome

Malignant lymphoma, albinism, nystagmus, recurrent infections, gingivitis, glossitis.

Costen's syndrome

Tinnitus, otalgia, headache, dizziness, burning tongue and throat.

Cowden syndrome (Multiple hamartoma and neoplasia syndrome)

Facial trichilemmoms associated with GI tract, thyroid, CNS and musculoskeletal abnormalities, *oral papillomatous* lesion.

(It is a disease with impaired bacteriolysis due to failure of phagolysosome formation. As a result of disordered intracellular trafficking there is impaired lysosome degranulation with phagosomes, so phagocytosed bacteria are not destroyed by the lysosome's enzymes. In addition, secretion of lytic secretory granules by cytotoxic T cells is also affected. The

disease is characterized by large lysosome vesicles in phagocytes (neutrophils), which thus have poor bactericidal function, leading to susceptibility to infections, abnormalities in nuclear structure of leukocytes, anemia, and hepatomegaly).

CREST syndrome

Calcinosis cutis
Raynaud's phenomenon
Esophageal dysfunction
Sclerodactyly
Telangiectasia.

(The limited cutaneous form of systemic sclerosis scleroderma is often referred to as CREST syndrome).

Crouzon's syndrome (craniofacial dysostosis—early fusion of cranial sutures):

Prognathic mandible, hypoplastic maxilla, high-arched palate, parrot beak appearance, hypertelorism.

(Crouzon syndrome is a genetic disorder known as a branchial arch syndrome. Specifically, this syndrome affects the first branchial (or pharyngeal) arch, which is the precursor of the maxilla and mandible. Since the branchial arches are important developmental features in a growing embryo, disturbances in their development create lasting and widespread effects).

Down's syndrome (Trisomy 21, Mongolism, Trisomy G)

Hypermobility, macroglossia, flat face, large anterior fontanelle, sexual underdevelopment, cardiac abnormalities.

Eagle's syndrome

Elongation of styloid process (more than 30mm) or ossification of stylohyoid ligament leading to sore throat, otalgia and glossodynia.

[Two forms of eagle syndrome exist: The classic form and the vascular one.

Patients with the classic "Eagle syndrome" can present with unilateral sore throat, dysphagia, tinnitus, unilateral facial and neck pain, and otalgia.

In patients with the vascular form of "Eagle syndrome", the elongated styloid process is in contact with the extracranial internal carotid artery. This can cause a compression (while turning the head) or a dissection of the carotid artery causing a transient ischemic accident or a stroke].

Ehlers-Danlos syndrome (Rubber man)

Hyperelasticity of skin (rubber man), hyperextensibility of joints, defective healing, cutaneous fragility.

[The syndrome is named after two doctors, Edvard Ehlers of Denmark, and Henri-Alexandre Danlos of France, who identified it at the turn of the 20th century. It is a group of inherited connective tissue disorders, caused by a defect in the synthesis of collagen (a protein in connective tissue). The collagen in connective tissue helps tissues to resist deformation (decreases its elasticity). In the skin, muscles, ligaments, blood vessels, and visceral organs collagen plays a very significant role and with increased elasticity, secondary to abnormal collagen, pathology results].

Fanconi syndrome

Aplastic anemia, microcephaly, hypogenitalism, olive brown pigmentation.

Frey's syndrome (Auriculotemporal syndrome)

Gustatory sweating.

Floppy infant syndrome
Generalized weakness due to hypotonia, inability to sit, stand, and walk.

(The term "floppy infant syndrome" is used to describe abnormal limpness when an infant is prone. Floppy babies are hypotonic infants who are unable to maintain flexed ligaments, and are able to extend them beyond normal lengths).

Grinspan's syndrome
Lichen planus
Hypertension
Diabetes mellitus
(Lichen planus with two most common diseases).

Gardener's syndrome
Multiple unerupted supernumerary teeth
Multiple polyposis of large intestine
Osteomas of the bones
Multiple epidermoid or sebaceous cysts
Desmoid tumors.

Goltz-Gorlin syndrome (Focal dermal hypoplasia)
Multiple papillomas, atrophy of skin, polydactyly, sunken eye, sparse hair.

Hutchinson's triad
Interstitial keratitis, 8th nerve deafness, mulberry molars

Horner's syndrome
Ptosis, anhydrosis, myosis, enophthalmos, loss of ciliospinal reflex (Mnemonic to memorise is PAMELa).

Horton's syndrome (Sphenopalatine Neuralgia)
It is characterized by unilateral paroxysms of intense pain in the region of the eyes, the maxilla, the ear, and the mastoid, base of the nose, and beneath the zygoma.

Jaw cyst—Basal cell nevus—Bifid rib syndrome (Basal cell nevus syndrome, Gorlin and Goltz syndrome)

Cutaneous anomalies	*Basal cell carcinoma*, other benign dermal cysts and tumors, palmar pitting, palmar and plantar keratosis, dermal calcinosis
Dental and osseous anomalies	*Odontogenic keratocysts*, mild mandibular prognathism, *rib anomalies* (bifid), vertebral anomalies, brachymetacarpalism
Ophthalmologic anomalies	Hypertelorism with wide nasal bridge, dystopia canthorum, congenital blindness, internal strabismus
Neurological anomalies	Mental retardation, dural calcification, agenesis of corpus callosum, congenital hydrocephalus, medulloblastoma
Sexual abnormalities	Hypogonadism in males, ovarian tumors

Jaw-Winking syndrome
Ptosis, rapid elevation of ptotic eyelid on movement of mandible to contralateral side.

Marfan syndrome
Long thin extremities, hyperextensibility of joints, spidery fingers, arachnodactyly, bifid uvula, CVS complication.

Myofacial pain dysfunction syndrome (MPDS)

Masticatory muscle tenderness, pain, limitation of motion, clicking noise.

Multiple endocrine neoplasm syndrome

MEN I ($PPPA_CU$)	MEN II (PA_MT) *Sipple's syndrome*	MEN III (NA_MTO)
• Hyperplasia of pituitary, parathyroid, pancreatic islets and adrenal cortex • Peptic ulcer and gastric hypersecretion	• Also called Sipple's syndrome • Parathyroid hyperplasia Pheochromocytoma of adrenal medulla • Medullary carcinoma of the thyroid *(Placed black strike through to differentiate from MEN I)*	• Mucocutaneous neuromas, Pheochromocytomas of adrenal medulla • Medullary carcinoma of the thyroid Marfanoid habitus

Median cleft-face syndrome

Median cleft of premaxilla and palate (Cleft)
Hypertelorism, Cranium bifidum occultum (Face)

Melkerson-Rosenthal syndrome

Cheilitis granulomatosa
Scrotal tongue
Facial paralysis
(Mnemonic to memorise is CSF).

Möbius syndrome

Partial or complete facial paralysis, drooling of saliva, congenital facial diplegia, difficulty in mastication.

[Möbius syndrome (also spelled Moebius) is an extremely rare congenital neurological, disorder which is characterized by facial paralysis and the inability to move the eyes from side-to-side. Most people with Möbius syndrome are born with complete facial paralysis and cannot close their eyes or form facial expressions. Limb and chest wall abnormalities sometimes occur with the syndrome. Most people with Möbius syndrome have normal intelligence, although their lack of facial expression is sometimes incorrectly taken to be due to dullness or unfriendliness. It is named for Paul Julius Möbius, a neurologist who first described the syndrome in 1888].

Orofacial digital syndrome

Cleft of tongue and mandibular alveolar process.

Papillon-Lefèvre syndrome

Juvenile periodontitis, palmar plantar keratosis, calcification of Falx cerebri.

Pierre Robin syndrome

Cleft palate, micrognathia, glossoptosis.

[At some time during the stage of the formation of the bones of the fetus, the tip of the jaw (mandible) becomes 'stuck' in the point where each of the collar bones (clavicle) meet (the sternum), effectively preventing the jaw bones from growing. When the fetus begins to move, the movement of the head causes the jaw to "pop out' of the collar bones. From this time on, the jaw of the fetus grows as it would normally, with the result that, when born, the jaw of the baby is much smaller (micrognathia) than it would have been with normal development, although it does continue to grow at a normal rate until the child reaches maturity].

Peutz-Jeghers syndrome

Familial generalized intestinal polyposis (hamartomatous).

Pigmented spots on the face, oral cavity, hand and feet (mucocutaneous melanotic macules)

Plummer-Vinson syndrome

Triad of Iron deficiency anemia, carcinoma of hypopharynx (post cricoids carcinoma), koilonychia.

Ramsay Hunt Syndrome
Herpes zoster infection of the geniculate ganglion.

[Ramsay Hunt syndrome type 2 also known as **herpes zoster oticus** is a disorder that is caused by the reactivation of pre existing herpes zoster virus in a nerve cell bundle in the head (the geniculate ganglion). The neurons in this ganglion are responsible for the movements of facial muscles, the touch sensation of a part of ear and ear canal, the taste function of the frontal two-thirds of the tongue, and the moisturization of the eyes and the mouth. The syndrome specifically refers to the combination of this entity with weakness of the muscles activated by the facial nerve. In isolation, the latter entity would be called Bell's palsy].

Reiter's Syndrome
Urethritis, arthritis, conjunctivitis, mucocutaneous lesion.

Reader's Syndrome (Paratrigeminal syndrome)
Severe headache, sign of ocular sympathetic paralysis.

Rubinstein-Taybi syndrome
Talon cusp, developmental retardation, broad thumbs and great toes, delayed/incomplete descend of testis in males, stature, head circumference and bone age below 50th percent.

[The CREBBP gene makes a protein that helps control the activity of many other genes. The protein, called CREB binding protein, plays an important role in regulating cell growth and division and is essential for normal fetal development. If one copy of the CREBBP gene is deleted or mutated, cells make only half of the normal amount of CREB binding protein. A reduction in the amount of this protein disrupts normal development before and after birth, leading to the signs and symptoms of Rubinstein-Taybi syndrome. It is also known as **Broad Thumb-Hallux syndrome**].

Stevens-Johnson syndrome
Severe bullous form of erythema multiforme involving skin, eyes, oral cavity, genitalia.

[Stevens-Johnson syndrome (SJS) is a form of erythema multiforme which is a life-threatening condition affecting the skin in which cell death causes the epidermis to separate from the dermis. The syndrome is thought to be a hypersensitivity complex affecting the skin and the mucous membranes. Although the majority of cases are idiopathic, the main class of known causes is medications, followed by infections and (rarely) cancers].

Sjögren's syndrome
Rheumatoid arthritis, Keratoconjunctivitis sicca, Xerostomia.

(The hallmark symptoms of the disorder are dry mouth and dry eyes)

Treacher Collins syndrome (Mandibulofacial dysostosis)
Hypoplasia of mandible and malar bone, macrostomia, malformation of external ear, high arched palate, bird/fish like face and antimongoloid slant with coloboma of lower eyelids.

Trotter's syndrome
Tumors of nasopharynx producing pain similar to trigeminal neuralgia.

Wallenberg's syndrome
Sensational loss on opposite side of the body due to vascular occlusion of posterior cerebellar artery affecting V, IX and X cranial nerves.

Waterhouse-Friderichsen syndrome
Bilateral adrenal hemorrhage, purpura, death due to septicemia caused by meningostrepto-pneumococci.

[Waterhouse-Friderichsen syndrome (WFS) or hemorrhagic adrenalitis is a disease of the adrenal glands most commonly caused by the bacterium *Neisseria meningitidis*. The

infection leads to massive hemorrhage into one or (usually) both adrenal glands. It is characterized by overwhelming bacterial infection meningococcemia, low blood pressure and shock, disseminated intravascular coagulation (DIC) with widespread purpura, and rapidly developing adrenocortical insufficiency].

Projection	*Film placement*	*Head position*	*Projection of central ray*	*Exposure*	*Important features*
PA view of skull	Vertical	Centered in front of the cassette with the canthomeatal line parallel to the floor. Canthomeatal line is 10° above the horizontal plane and Frankfort plane is perpendicular to the same	Posterior to anterior direction perpendicular to the plane of film	70 kVp 30-50 mAs	• Used to examine skull for disease, trauma and sinus· • PA view with 10° tilt is called *"Caldwell projection"*. • Good for visualization of facial structures • Best for viewing coronoid process
Lateral skull or cephalometric view	Vertical	The left side of the face should be positioned near the cassette and the midsagittal plane should be parallel with the plane of the film	Is directed towards the external auditory meatus perpendicularly to the plane the film and mid sagittal plane	70 kVp 15-25 mAs	• Cephalometric view shows soft tissue profile. • Used to assess facial structures and skull for disease and trauma and facial growth. • Right-sided structures are more clearly visible.
Water's projection (Occipito-mental projection)	Vertical	The sagittal plane of head should be perpendicular to the plane of film. *Chin is raised such that canthomeatal line is 37° above horizontal plane*	Central rays passes through the maxillary sinus	70 kVp 100 mAs	• Best for viewing zygoma fractures, paranasal sinus (esp. maxillary) and nasal cavity· • It's variation of PA view.
Reverse-Towne's projection	Vertical	Canthomeatal line is 25-30° downwards	Central rays is directed towards the occipital bone	70 kVp 100 mAs	• Best for diagnosing condylar neck fractures (esp. medially displaced) • Reveals Posterolateral wall of the maxillary antrum.

Contd....

Contd....

Projection	*Film placement*	*Head position*	*Projection of central ray*	*Exposure*	*Important features*
Submento-vertex	Vertical	Patient's head and neck should be extended backwards such that the vertex of the skull is on the center of the cassette	Central rays is directed from below the mandible towards the vertex of skull	70 kVp 100 mAs	• Contraindicated in patient of cervical spondilities • Best for viewing fracture of base of skull and zygomatic arch • Frankfort plane is vertically oriented and parallel to film. • For viewing zygomatic arch exposure time is reduced to $^1/_3$rd than that used for visualization of skull.

Projection	*Film placement*	*Head position*	*Projection of central ray*	*Exposure*	*Important features*
Lateral oblique mandibular body projection	Placed against patient's cheek over the 1st molar. Parallel to lower border of mandible. 2cm below the lower border of mandible.	Tilted towards the side being examined Mandible is protruded.	Directed towards 1st molar region, 2 cm below the angle of mandible	65 kVp 10 mAs	• Best for viewing inferior border of mandible· • Shows premolar and molar region.
Lateral oblique mandibular ramus projection	Placed over the ramus far posteriorly to include condyle. Parallel to lower border of mandible. 2cm below the lower border of mandible.		Directed posteriorly towards the ramus, 2cm below the inferior	65 kVp 10 mAs	• Used often for examining 3rd molar region of maxilla and border mandible. • Gives view of the ramus from the angle to condyle.

Radiographic projection	*Synonyms*
Occipitomental projection	Water's view
Submentovertex projection	Base view, Full axial view, Jug handle view
Transpharyngeal projection	PARMA view, Infracranial projection, Mcqueen projection
Transorbital projection	Transmaxillary view, Zimmer projection

Structure	*Best radiographic projection*
Fracture of zygomatic arch	Submentovertex (Jug handle view, 1/3rd exposure)
Base of skull	Submentovertex
Maxillary sinus Nasal septum Fractures of zygoma	Water's view
Condylar neck fracture	Reverse-Towne's view
Medially displaced condylar fracture	PA view
Coronoid process of mandible	PA view of skull
Midmandibular ramus to condylar apex	Transpharyngeal view
Bony ankylosis of TMJ	CT scan
Fibrous ankylosis of TMJ Internal derangement of disk	MRI
Disk perforation	Arthrography

CHAPTER

29 Orbital Blow Out Fracture

Orbital fractures are often associated with ocular injuries and midfacial fractures. Fractures involving solely the floor of orbit are termed as blow out fractures and represent the third most common isolated midfacial fracture. In 1957, Smith and Regan coined the term "blow out fracture".

DEFINITION

"It is a depressed fracture of orbital floor with herniation and incarceration of periorbital soft tissues in maxillary sinus".

ETIOLOGY

1. The most widely accepted theory concerning the cause of blow out fracture is that the globe is struck by an object of greater transverse diameter than itself. The kinetic energy with which the object strikes the globe is suddenly converted into hydraulic energy by fluid-filled globe. This is then transmitted uniformly to the orbital walls.

Direct trauma to the orbit with a larger object like fist/cricket ball.
↓
Increased hydraulic pressure within the orbit
↓
Compression of orbital content and
Fracture of orbital floor due to its upwards inclination and thinness (Fig. 29.1)

2. Many times a direct blow to the inferior orbital rim leads to buckling of the floor.

TYPES (FLOW CHART 29.1)

In 1960 Converse and Smith introduced the concept of "pure" (isolated floor) "impure" (floor and rim) blow out fractures.

Pure fractures are thought to be caused by a sudden instantaneous increase in intraorbital pressures from direct blunt trauma to the globe itself. Impure fractures are caused by direct trauma and compression of the bony rim and collapse of the surrounding facial bones, and result in the disruption of internal orbital walls (Fig. 29.2).

SITES

Blow out fracture involving the floor of orbit most commonly occurs in the posterior area of the floor medial to the infraorbital fissure, the thinnest portion of the orbit.
Note: Along with floor the weak medial wall also fractures (20 to 50% cases).

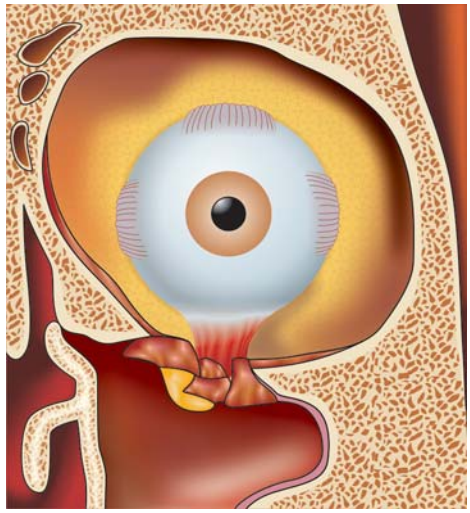
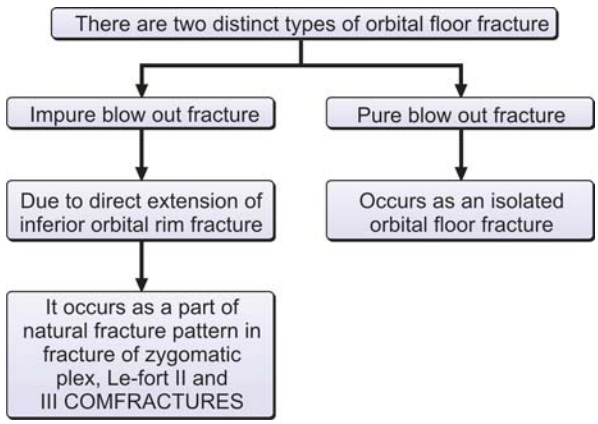


Fig. 29.1: Fracture of orbital floor

Flow chart 29.1: Types of orbital floor fracture



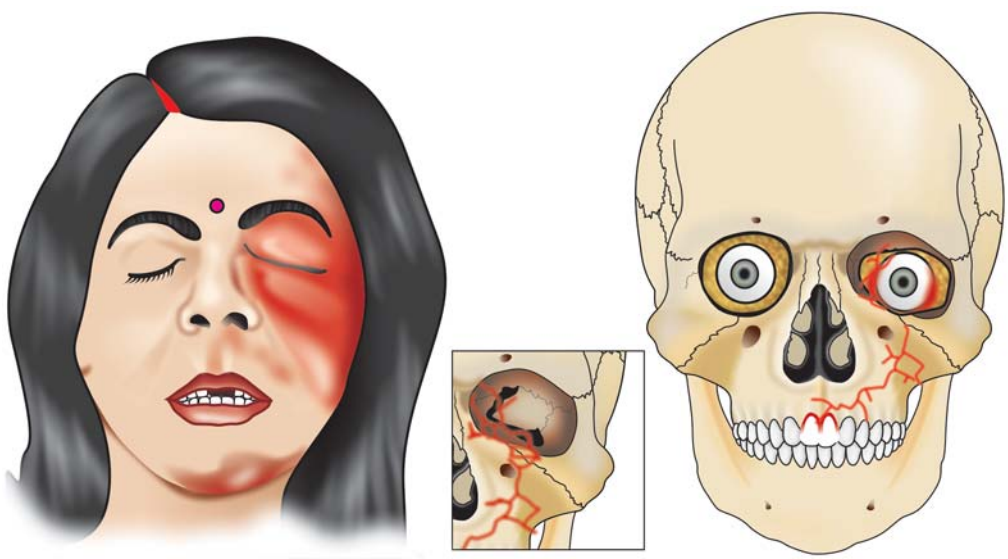


Fig. 29.2: Facial features and fracture line

CLINICAL SIGNS

- Signs and symptoms are masked by edema and ecchymosis during the immediate posttraumatic phase.
- They become clinically evident after increase in orbital volume of 1.5-2.0 ml or when diplopia occurs.
  1. *Circumorbital and subconjunctival ecchymosis:* It may be associated with surgical emphysema due to leakage of air from the paranasal air sinuses.
  2. Limitation of eye movement in upward gaze due to tethering of inferior muscles.
  3. *Enophthalmos:* It occurs because of the enlargement of the orbital volume leading to herniation of orbital contents (fat) through floor of orbit.
  4. Pseudoptosis and deepening of the supratarsal fold accompany the enophthalmos. If the eyelid covers the pupil, the patient will attempt to elevate both eyelids, and lid retraction will be seen on the opposite side.
  5. *Double vision (Diplopia):* It may be due to restricted ocular motility from proplapse of the periorbital contents into the underlying maxillary sinus. Such diplopia may also be due to entrapment or direct impingement on the fine suspensory ligamentous system of the orbit or, less frequently, of the extraocular muscles. It is commonly due to interference in acting of inferior oblique and inferior rectus muscles.

     It usually takes 7 to 10 days for the initial edema or bleeding, or both, to disappear and resorb. It is therefore difficult to ascertain during that period whether the diplopia noted is of a transitory nature.
  6. Infraorbital paresthesia due to damage to infraorbital nerve.

DIAGNOSIS

It is difficult to make a clinical diagnosis of an isolated blowout fracture. On occasion, these fractures are not recognized until the diplopia is noted. Early on, visual inspection is often of limited value because edema may mask any enophthalmos and also makes palpation difficult.

Diagnosis is mainly by:

a. Force duction test
b. Radiographic examination.

a. Force-duction test:

The eye should be anesthetized topically and the test is performed.
Grasp the tendon of inferior rectus muscle with a small
tissue holding forcep (Adson's forcep)
↓
Patient is asked for the entire range of movement
↓
Inability to rotate the globe superiorly
signifies entrapment of muscles in floor.

b. Radiographic examination:

- Waters view

  It allows visualization of the orbital roof and floor so it is particularly useful for evaluation of the orbital floor blowout fractures (Fig. 29.3). With this view, the petrous portion of the temporal bones is projected below the maxillary sinuses and indirect signs of fracture can be noted such as "teardrop formation" or air-fluid levels. It shows "hanging drop sign" due to proposal of orbital fat into maxillary sinus (Fig. 29.4).

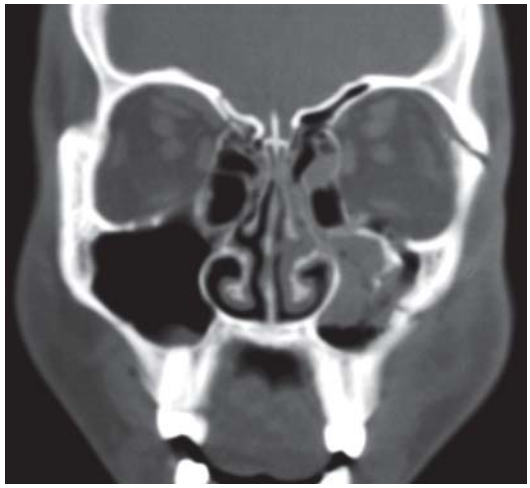


Fig. 29.3: Blow out fracture on left side

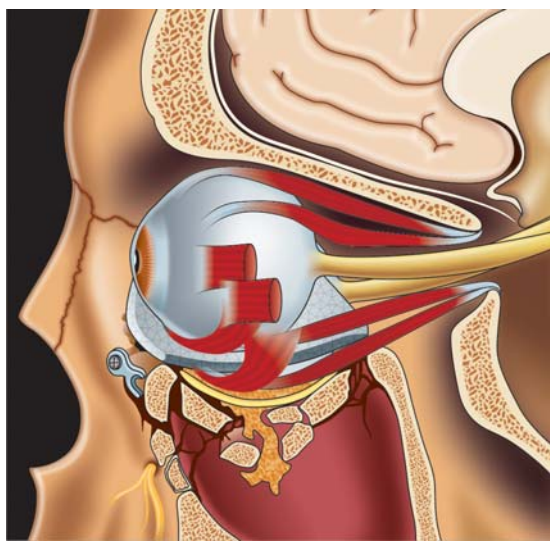


Fig. 29.4: Fracture of orbital floor

- Caldwell-Luc view
- CT scan
  Sagittal CT scans are the most accurate diagnostic aid. It shows "Trapdoor deformity" with mechanical impingement of orbital structures indicating blow out fracture.

MANAGEMENT

Surgical exploration and reconstruction of orbital floor.

Indications for Surgery

The goals of acute or primary reconstruction of primary orbital fractures are to alleviate any functional deficit and to restore the facial esthetics by restoring the configuration of the orbital walls, return prolapsed orbital contents to the orbit proper, and eliminate any impingement or entrapment of orbital soft tissues.

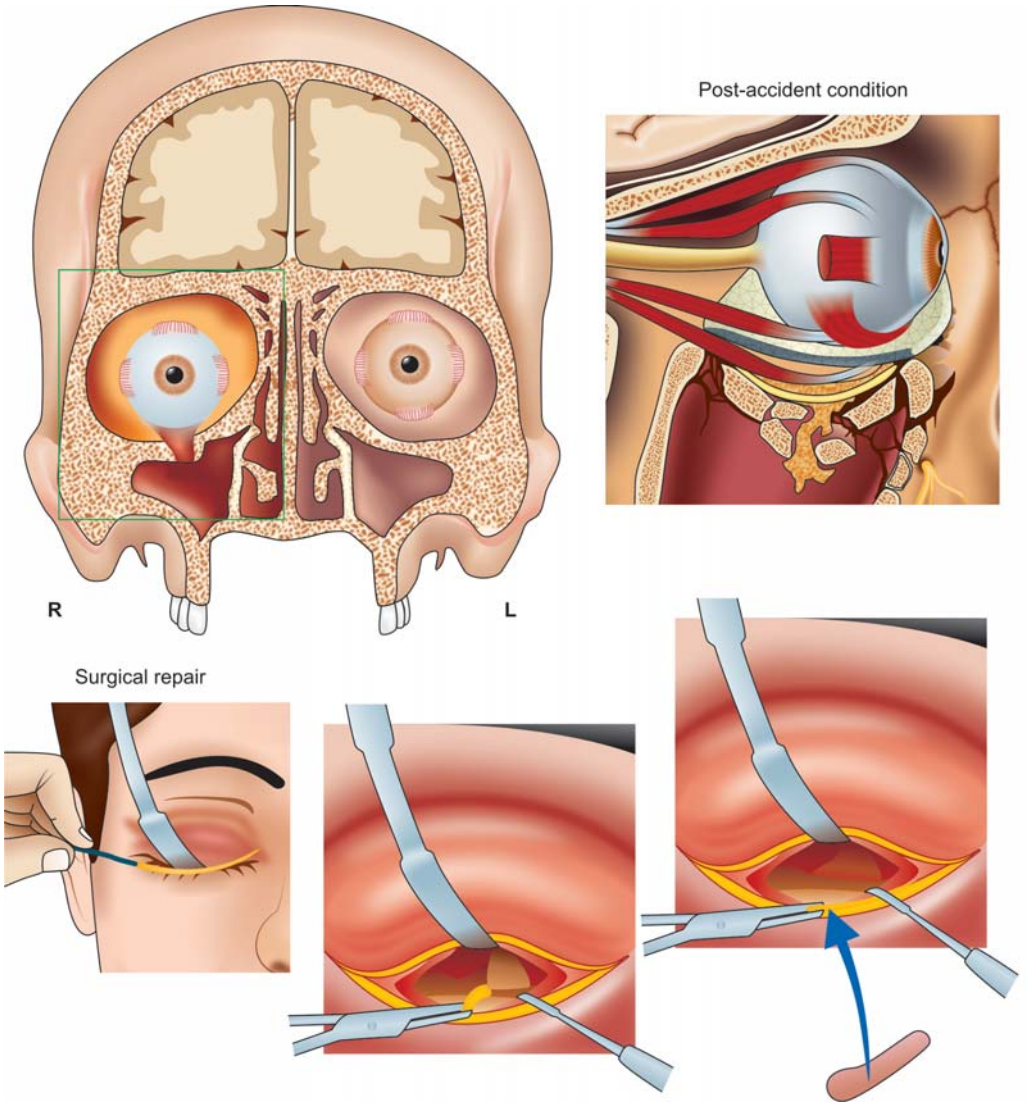


Fig. 29.5: Surgical repair

Indication	*Aim of treatment*
Diplopia not resolving during first 10 days after injury	Restoration of ocular motility
Herniation of soft tissue into antrum	Repair of floor and correction of enophthalmos
Incarceration of tissue which causes retraction of globe in upward direction	Reconstruction, prevention of fibrosis
Enophthalmos > 3 mm	Restoration of the orbital volume

Decision of surgery is taken only after 7-10 days while edema subsides. Volume changes account for abnormal globe positioning. The goal of reconstruction (Fig. 29.6) is to restore the anatomic position of the bony rim and associated facial bones and to reapproximate the normal bony volume with reconstructive materials (Fig. 29.7).

Incisions (Flow chart 29.2)

There are three basic incisions used for accessing the orbital floor (Fig. 29.5):

1. Transconjunctival with/without lateral canthotomy
2. Subciliary
3. Infraorbital.

Subciliary and transconjunctival incisions are the most popular due to superior esthetics and generous access.

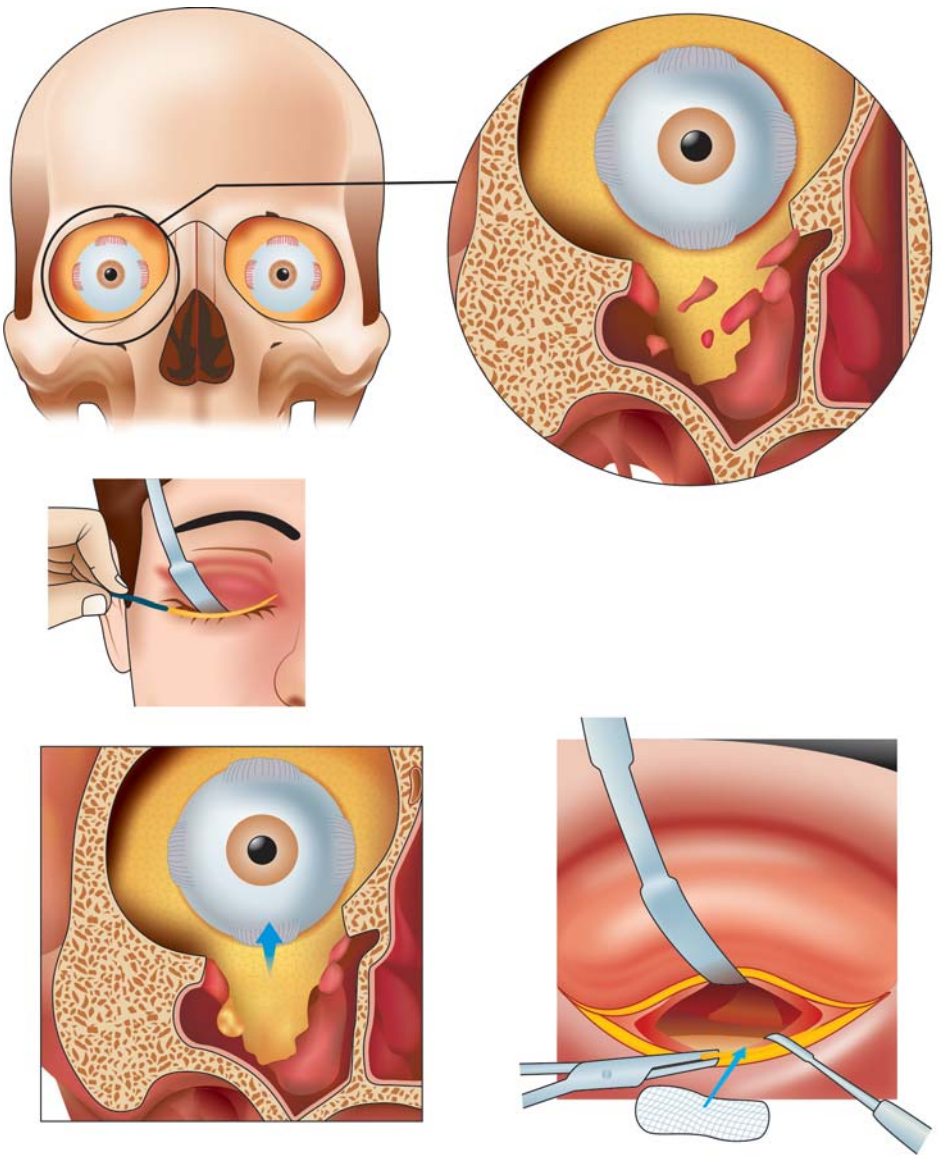
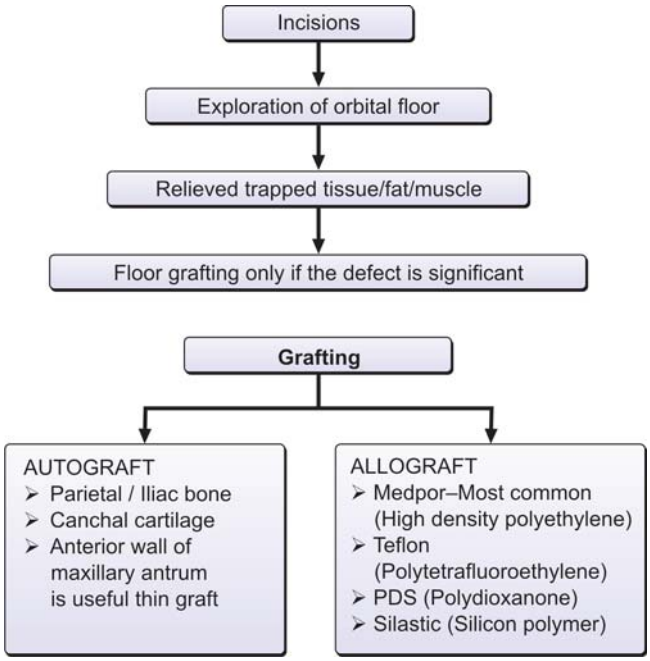
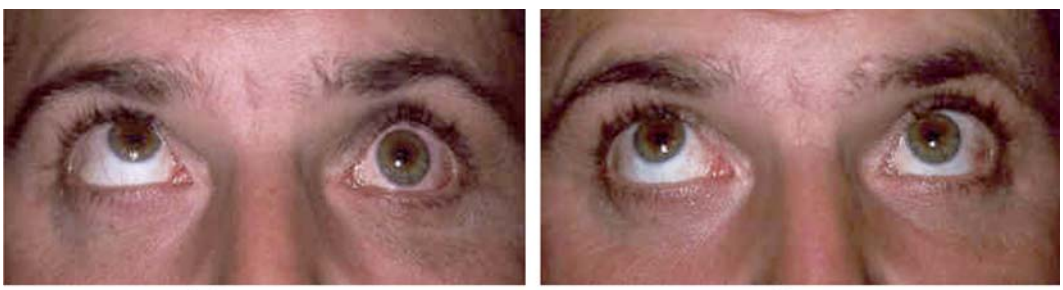


Fig. 29.6: Surgical reconstruction

Flow Shart 29.2: Steps of incisions and grafting





Before treatment After treatment

Fig. 29.7: Before and after treatment

Materials

Numerous materials have been described for the reconstruction, such as porous polyethylene, bioresorbable polydioxanone, nylon, marlex mesh, gelatin film, titanium mesh, and autogenous bone grafts (Fig. 29.8). Each has its own pros and cons related to strength, application, reactivity, infection rate, biointegration, and complication rate.

Many surgeons prefer autogenous graft as allograft has risk of extrusion and becoming encapsulated in cyst like swelling. However, porous polyethylene sheet is extremely biocompatible and nonresorptive with sufficient tensile strength and flexibility.

Autogenous grafts are perhaps most favored for significant floor defects. They are highly biocompatible but require second surgical procedure. The donor sites that are usually harvested have been the inner aspect of anterior and posterior iliac crest or unicortical or bicortical calvarial bone.

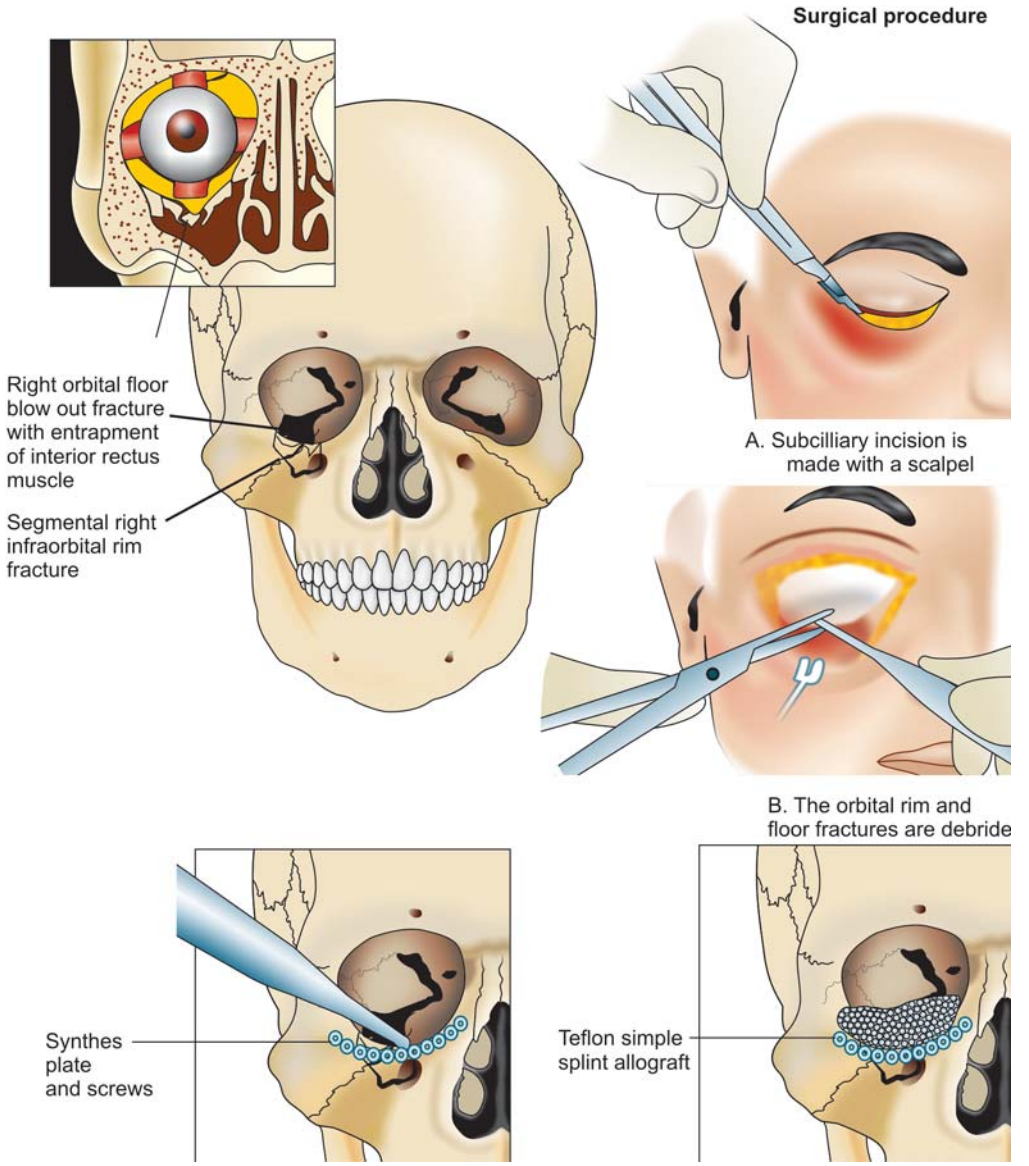


Fig. 29.8: Reconstruction of orbital floor

Titanium mesh or orbital floor plates with screw fixation and autogenous graft are quite useful in severe or comminuted injuries. Graft can be stabilized by simple wiring/by using microplates if required.

(**Note:** Lesser wing of sphenoid surrounds the optic foramen and protects optic nerve from injury so there is no blindness in maxillofacial trauma).

CHAPTER

30 Considerations for Oral Surgery Viva Voce

LEUKOPLAKIA (WHO—1975)

Leukoplakia is any white patch or plaque that cannot be characterized clinically or pathologically as any other disease.

ORAL SUBMUCOUS FIBROSIS (OSMF)

It is an insidious chronic disease affecting any part of oral; cavity and sometimes pharynx although occasionally preceded by and/or associated with vesicle formation, it is always associated with juxtaepithelium inflammatory reaction followed by fibroelastic change of lamina propria/with epithelial atrophy leading to stiffness of oral mucosa and causing trismus and inability to eat.

SIALOSIS

Sialosis is noninflammatory, nonneoplastic enlargement of salivary gland.

SIALADENITIS AND SIALODOCHITIS

Inflammation of salivary gland arising from various infectious and noninfectious causes is called sialadenitis and inflammation of salivary duct is called as sialodochitis.

SIALADENOSIS

Sialadenosis is an unusual noninflammatory, nonneoplastic disorder characterized by salivary gland enlargement, particularly involving parotid gland.

XEROSTOMIA

Xerostomia refers to a subjective sensation of a dry mouth; it is frequently, but not always, associated with salivary gland hypofunction.

SIALOLITHIASIS

Calculi or stone within ductal system of major and minor salivary gland is known as sialolithiasis.

SIALECTASIS

Sialectasis is atrophy of part or total salivary gland.

SIALORRHEA/PTYLISM

Sialorrhea is excessive secretion of saliva greater than 4 ml/min.

TRIGEMINAL NEURALGIA

It is a sensory disturbance of 5th cranial nerve characterized by sudden, spontaneous, sharp, shooting, lancinating, paroxysmal, intense pain that is usually confined to the specific branches of trigeminal nerve.

PAIN

Pain is an unpleasant emotional experience usually initiated by noxious stimuli and transmitted over a specialized neural network to the central nervous system where it is interpreted as such.

LOCAL ANESTHESIA

Loss of sensation in circumscribed area of body caused by depletion of excitation in nerve ending or by inhibition of conduction process in peripheral nerve without inducing loss of consciousness is known as local anesthesia.

LUDWIG'S ANGINA

Given by Archer,

It is bilateral, acute, rapidly spreading, septic, inflammatory, indurate, wooden hard cellulites of floor of mouth.

By Thoma,

It is a gangrenous cellulites of loose alveolar tissue which originates in the submandibular space and spread rapidly towards the floor of mouth.

By Killey, Seward and Kay,

It is a clinical diagnosis and the name given to the massive brawny cellulites occurring bilaterally at the submandibular region which also involves sublingual space.

BELL'S PALSY

It is an idiopathic lower motor neuron facial paralysis of sudden onset and unrelated to disease elsewhere in the body.

OSTEOMYELITIS

Osteomyelitis is inflammation of medullary portion of bone marrow/cancellous bone.

It is defined as an inflammatory condition of bone that begins as an infectian of medullary cavity and haversian system of the cortex and extends to involve the periosteum of the affected area.

CYST

Cyst is a pathological cavity having fluid, semifluid, and gaseous contents which are not created by accumulation of pus frequently and may or may not be lined by epithelium (Krumer 1974).

MARSUPIALIZATION

It refers to the procedure of creating a surgical window in the wall of the cyst and evacuation of the cystic content. This process decrease the intracystic pressure promotes the shrinkage of the cyst and bone fill.

NEURALGIA

It is an intense paroxysmal pain that is confined to the specific branches of the nerves of the head and neck.

TUMORS/NEOPLASM

New growth of abnormal tissues of the body is known as tumors.

SOME CONSIDERATIONS FOR VIVA VOCE

- Spellings of all ester type of local anesthetics have only one "i", e.g. tetracaine, cocaine, while that of all amide type of local anesthetics have two "i", e.g. lidocaine, bupivacaine.
- Heart rate is 72 and respiratory rate is 16 per minute, so ratio of chest compression and mouth breathing is 4:1 in CPR.
- Antibiotics should be given preoperatively as the incised wound is most susceptible to infection during first six hours. It takes six hours to activate immunity after placing incision, so preoperative antibiotics protects wound during this period and prevent bacteremia.
- Odontotomy – Tooth division or tooth splitting
  Odontectomy – Transalveolar extraction.

Principle	*Mechanical advantage*
Wheel and axle	4.6
Wedge principle	2.5
Lever principle	3

Some Numerical about Local Anesthesia

1. Two percent lignocaine means – 2 gm (2000 mg) in 100 ml, i.e. 1 ml contains 20 mg of lignocaine. Same way, 3 percent means, 1 ml contains 30 mg of lignocaine.
2. Increasing concentration of local anesthetic solution from 2 to 5 percent causes rapid onset of action due to availability of more amount of nonionic local anesthetic molecules (RN). But the duration of binding to receptor is not dependent on concentration. So duration is not affected.
3. 1:2,00,000 adrenalin means – 1 g (1000 mg) of adrenalin in 2, 00,000 ml of local anesthetic solution. So 1 ml of local anesthetic solution contains 0.005 mg of adrenalin. For example, 1:1000 adrenalin means 1 ml solution contains 0.005 mg of adrenalin.
4. How many cartridge of 2 percent xylocaine without adrenalin can be given safely to the patient? Safe dose of local anesthetic without adrenalin – 300 mg
   In 2 percent xylocaine, 1 ml solution contains 20 mg of lignocaine. So 300/20 = 15 ml or 7 cartridge. (1 cartridge contains 2 ml approximately).
5. How many cartridges of local anesthetic with 1:2,00,000 adrenalin can be given to a normal healthy patient?
   1:2,00,000 means 1 ml contains 0.005 mg of adrenalin.
   Safe dose of adrenalin in healthy patients is 0.2 mg so 0.2/0.005= 40 ml or 20 cartridges (Approximately)
6. How many cartridges of local anesthetic with 1:1,00,000 adrenalin can be given for a cardiac patient?
   1:1,00,000 means 1 ml solution contains 0.001 mg of adrenalin.
   Safe dose of adrenalin on cardiac patient = 0.004 mg. So 0.04/0.01 = 4 ml or 2 cartridges.

Aδ fibers	Myelinated and conduct fast pain
C fibers	Unmyelinated and conduct slow pain. They are very sensitive to local anesthesia.
Natural local anesthetic agent	Cocaine
Local anesthetic with intrinsic vasoconstriction property	Cocaine
Local anesthetic with most potent vasodilating property	Procaine
Local anesthetic with least vasodilating propertyLocal anesthetic for patients in whom vasoconstrictors are not recommended	Mepivacaine
Safest local anesthetic for children (Due to short duration and less toxicity), Local anesthetic with shortest duration of action	2-chloroprocaine
Most toxic of all local anesthetics	Propoxycaine
Local anesthetic which causes methemoglobinemia	Prilocaine, Articaine
EMLA (Eutectic mixture of local anesthesia)	Lidocaine + Prilocaine
Local anesthetic with high pKa value	Procaine

- The receptors for pain are free nerve endings. The loss of sensation after the administration of local anesthetic is in the order of pain, temperature, touch and proprioception. The return of sensation is in the reverse of preceding order.
- Mepivacaine has least vasodilating effect. 3 percent mepivacaine without vasoconstrictor is ideal when vasoconstrictor is contraindicated as in hyperthyroidism.
- Mepivacaine is the most commonly used local anesthetics in pediatric dentistry.
- Methylparaben is most commonly used preservative in local anesthetic solution. It has bacteriostatic, fungistatic and antioxidant properties.
- Sodium bisulfate is the most commonly used antioxidant in local anesthetic solution. It prolongs the self-life of solution by rendering it more acidic and thus prevents deterioration of epinephrine.
- RN (lipophilic) – nerve penetration
  RNH^+ (hydrophilic) – connective tissue penetration.

Low pH (more H^+)	↑ diffusion, ↓ penetration
High pH (less H^+) (In case of infection)	↓ diffusion, ↑ penetration
Low pKa (Benzocaine)	↑ penetration, ↓ diffusion
High pKa	↓ penetration, ↑ diffusion

- The gauge of needle used in dental syringes refers to internal diameter of the lumen. Larger the gauge, smaller the diameter.
- Main barrier for diffusion of local anesthetic is the perineurium. Endoneurium is the actual site of action and rate of diffusion depends on lipid solubility of drug.
- The maxillary tooth that is difficult to anesthetize by infiltration is first molar due to the presence of thick zygomatic buttress.
- After the removal, tissue should be immediately placed in 10 percent formalin (4% glutaraldehyde). Volume should be at least 20 times the volume of specimen.
- One unit of platelet rich plasma raises the platelet count approximately by 7000–10000 per μl.
- One unit fresh blood raises the hemoglobin concentration by 1 gm percent.

- 1 unit (150 ml) of fresh frozen plasma contains 200 μ factors VIII, 200 μ factor XI and 400 mg fibrinogen. It is stored at 30°C.
- Cryoprecipitate contains factor VIII (100 μ), factor XIII, von Willebrand factor and fibrinogen (250 mg).
- Hypovolemic shock develops after loss of 40 percent of blood. Initial resuscitation should be done with crystalloids such as Normal saline or Ringer lactate.
- Fluids to replace the 1 liter of blood are:
  1:3 = Blood: Colloidal fluid
  1:1 = Blood: Crystalloid fluid
- Antidote for narcotic analgesics is *naloxone*.
- The drug of choice in treating the anaphylaxis is 0.2–0.5 ml of 1:1000 solution of adrenalin by IM or SC route.
- Mental foramen opening is directed backward and lateral direction.
- Phentolamine, an alpha-adrenergic blocking agent is used to reverse the effect of local anesthesia.

However, it does not reverse anesthetics themselves. Instead, it competes with epinephrine for receptors. Epinephrine or neocobefrin are included in most dental anesthetic formulations as vasoconstrictors to keep the anesthetic in the targeting area. Dilation of blood vessels washes the anesthetic agent away. By blocking the vasoconstrictors, phentolamine accelerates this clearance. It accelerates the recovery of normal sensations.

Phentolamine has been used since the 1950s as an antihypertensive in a 5- to 10-mg dose, and can cause fainting and other side effects in these doses. A 1.7-ml cartridge of *OraVerse* contains only 0.4 mg of phentolamine. Dentists can inject OraVerse as a cartridge in the same manner and at the same site of injection of anesthetics.

The drug could be used with any minor procedure, such as a restoration, in which long-term anesthesia is not needed.

Instrument	*Use*
Ash forceps	To extract lower incisor
Rowe's disimpaction forceps	To reduce the tooth bearing portion of upper jaw
Walsham or Asche's forceps	To reduce nasal complex fracture
Bristow's elevator	To elevate fractured zygomatic bone
Osteotome (bibeveled)	To split tooth
Chisel (monobeveled)	To remove bone
Bone rongeur	To trim bone
Bone gauge	To make window in maxillary antrum
Gigli saw (Surgical saw)	To cut bone
No. 16 cowhorn forceps	To extract mandibular molars

Glasgow Coma Scale

	Motor response	*Best verbal response*	*Eye opening*
5	Obeys commands	Well-oriented	-
4	Localized pain	Confused conversation	Spontaneous
3	Flexion response to pain	Inappropriate speech	On request
2	Extension response to pain	Incomprehensible	As response to pain
1	None	None	None

SOME COMMONLY USED COMPOSITIONS

White head varnish	Iodoform – 10 mg Benzoin – 10 gm Prepared storax – 7.5 ml Balsam of tolu – 5 gm Solvent ether – 100 ml It consists of a number of aromatic resins which slowly broken down to produce benzoic acid, a potent antiseptic.
Carnoy solution (Used for fixing the tissues in the tumor bed)	Absolute alcohol – 6 ml Chloroform – 3 ml Glacial acetic acid – 1 ml
Bone wax (To reduce bleeding)	7 parts yellow wax 2 parts olive oil 1 part phenol
Tabot's solution (Used as astringent in pericoronitis)	Iodine Zinc iodine Glycerin water
McInns solution (Bleaching of vital tooth)	1 part anesthetic ether 5 part HCL (36%) 5 parts of H_2O_2 (30%)
Monsel's solution	It contains ferric sulphate. It acts by precipitating proteins. It is effective in arresting the capillary bleeding and post-extraction bleeding in medullary bone.

Important Signs

Colman's sign	Hematoma in floor of mouth due to rupture of dorsal lingual vein in symphyseal/body fracture.
Guardsman's/Parade ground fracture	Symphysis fracture associated with bilateral subcondylar fracture. Commonly seen in epileptics and soldiers.
Battle's sign	Hematoma below and behind the mastoid process associated with fracture of base of skull. Commonly seen in mandibular condylar fracture.
Guerin's sign	Hematoma at greater palatine foramen. Commonly seen in Le Fort I fracture
Bucket handle fracture	Downward and backward displacement of the anterior part of the mandible leading to respiratory distress.

CLASSIFICATIONS

Dentoalveolar Fracture

Andreasen and Andreasen, 1994

1. *Dental hard tissue injury*
   a. Crown infraction (crack of enamel or incomplete fracture)
   b. Crown fracture—enamel only
   c. Crown fracture—enamel + dentin

d. Crown fracture—enamel + dentin + pulp
e. Crown—root fracture (vertical fracture)
f. Crown—root fracture (oblique fracture)
g. Root fracture.

2. *Periodontal injury*
   a. Concussion (no displacement of tooth but tender to percussion)
   b. Subluxation (loosening of tooth without displacement)
   c. Intrusion
   d. Extrusion
   e. Lateral luxation (loosening of tooth with displacement)
   f. Avulsion.
3. *Alveolar bone injury*
   a. Intrusion of tooth with comminution of socket
   b. Fracture of single wall of socket or alveolus
   c. Fracture of both walls of socket or alveolus
   d. Fracture of mandible or maxilla involving the alveolus and/or tooth socket.
4. *Gingival injury:*
   a. Contusion
   b. Abrasion
   c. Laceration.
5. *Combination of above*

Mandibular Fracture

I. Dingman and Natvig

a. Symphyseal
b. Parasymphyseal
c. Body
d. Angle
e. Ramus
f. Condylar process
g. Coronoid process
h. Alveolar process.

II. Kazanjian and Converse

Depending upon presence or absence of serviceable teeth in relation to line of fracture:

Class I	Teeth present on both sides of fracture line
Class II	Teeth on one side of fractured line
Class III	Edentulous patient

III. Rowe and Killey

Those involving basal bone	**Those not involving basal bone**
Single unilateral fracture	Alveolar process
Double unilateral	
Bilateral	
Multiple	

IV. Kruger

- Simple
- Compound
- Comminuted.

V. Kruger and Schilli

a. *Relation to external environment*
   - Simple
   - Compound.

b. *Types of fracture*
   - Incomplete
   - Complete
   - Greenstick
   - Comminuted.

c. *Dentition of jaw within reference to use of splints*
   - Sufficiently dentulous jaw
   - Edentulous or insufficiently dentulous jaw
   - Primary and mixed dentition.

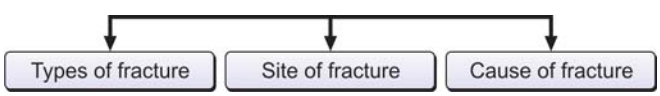
d. *Localization*
   - Fracture of symphysis region between canines
   - Fracture of canine region
   - Fracture of body of mandible between canine and angle
   - Fracture of angle in third molar region
   - Fracture of mandibular ramus between angle and sigmoid notch
   - Fracture of coronoid process
   - Fracture of condylar process.

VI. Mandibular Angle Fracture

(Depending upon direction of fracture line and effect of muscle action on fracture fragment)

i. Vertically favorable or unfavorable (Seen on occlusal view)
ii. Horizontally favorable or unfavorable (Seen on OPG).

VII. Other Classification



A. Types of fracture

a. *Simple fracture*
These are linear fractures which are not in communication with exterior. Usually, they are found in the region of condyle, coronoid process, ascending ramus, edentulous mandible, in the angle posterior to 3rd molar tooth. Green stick fracture seen in children is a variant of simple fracture.

b. *Compound fracture*
Fracture of the tooth bearing portion of the mandible, with extraoral or intraoral wound involving fracture line is present. Such fracture communicates to the site with potential of infection through mouth via periodontal ligament or through skin.

c. *Comminuted fracture*
Comminuted fractures are characterized by two or more fragments of bone at the fracture site. The main etiological factor for comminuted fracture is direct violence

to mandible from penetrating sharp objects or missiles delivering high degree of kinetic energy upon limited area. They are usually compound by nature and complicated to manage due to bone, soft tissue loss.

d. *Pathological fracture*
These are spontaneous fractures of the bone as a result of a normal degree of muscular contraction, or following minimal trauma. They result from minimum trauma to mandible which is already weakened by pathological condition such as osteomyelitis, neoplasm or generalized skeletal disease.

e. *Complicated fracture*
Complicated fractures can be defined as those fractures that either directly or indirectly involve surrounding major vessels nerves, or joints. As mandible itself contains the inferior alveolar neurovascular bundle, most mandibular fractures can be thought of as complicated fractures.

f. *Impacted fracture*
Impacted fractures are those in which the fractured fragments interdigitate to such an extent that there is little or no movement at the fracture site. They are very rare in mandible but are commonly seen with midfacial fractures.

g. *Greenstick fracture*
In children, elasticity of bone allows it to bend instead of fracturing full thickness of bone. Only the one sided cortex fractures while that on another side remains intact in position.

B. Site of fracture

- Dentoalveolar
- Condyle
- Coronoid
- Ramus
- Angle
- Body
- Parasymphysis
- Symphysis.

C. Cause of fracture

- Direct violence
- Indirect violence
- Excessive muscular contraction.
  (Coronoid fracture because of sudden reflex contraction of temporalis).

Condylar Fracture

I. Comprehensive Classification (Lindahl 1977)

a. Depending upon level of fracture

1. *Condylar head or intracapsular*
   It is difficult radiographically to differentiate the exact anatomical confines of the head. By definition, a condylar head fracture is within the capsule and is therefore termed 'intracapsular'. It may be further divided into vertical, compression and comminuted.
2. *Condylar neck*
   The radiographic constriction representing the condylar neck corresponds anatomically to the region of the inferior attachment of the joint capsule.
3. *Subcondylar*
   This is the region below the neck extending down to the most inferior point on the sigmoid notch anteriorly, while its posterior limit is situated more inferiorly

corresponding with the point of maximum curvature of the natural concavity of the posterior border of the mandible in that region.

b. Relation of condylar fragment to mandible
   1. Undisplaced
   2. Deviated
      This is a simple angulation of the condylar process in relation to the main mandibular fragment without overlap.
   3. Displaced with medial overlap of condylar segment.
   4. Displaced with lateral overlap.
   5. Anterior posterior overlap (infrequently seen).
   6. No contact between the fragments.
c. Relation of condylar head to fossa
   1. Not displaced (the joint space appear normal).
   2. Displaced (the joint space is increased but the condyle is still related to the glenoid fossa).
   3. Dislocation (the condylar fragment is completely out of the fossa).

II. Clinical Classification (MacLennan 1952)

a. No displacement
b. Fracture deviation: There is simple angulation of the condylar process to the major fragment. This is the green-stick fracture of childhood.
c. Fracture displacement: There is overlap of the condylar process and major mandibular fragments.
d. Fracture dislocation: The head of the condylar process has been completely disrupted from the articular fossa.

III. Classification of Condylar Neck Fractures (Spiessl and Schroll)

Type I	Condylar neck fracture without serious dislocation
Type II	Deep seated condylar neck fracture with dislocation
Type III	High condylar neck fracture with dislocation
Type IV	Deep-seated condylar neck fracture with luxation
Type V	High condylar neck fracture with luxation
Type VI	Head or intracapsular fracture

IV. Other

Temporomandibular joint fractures are also classified according to the height of the fracture.

a. *Intracapsular fractures*: These fractures run irregularly, usually diagonally through the head. Because of anatomic variations in the capsular attachment, they can lie within the capsule and outside it.
b. *High temporomandibular joint fractures*: These fractures are below the capsule and muscle attachment but above the sigmoid notch.
c. *Low temporomandibular joint fractures*: These fractures run from the sigmoid notch to the back edge of the mandibular ramus.

Fracture of Zygomatic Complex and Arch

I. Knight and North (1961)

Based on Water's Radiograph,

Group 1: Undisplaced fracture
Group 2: Isolated displaced arch fracture
Group 3: Displaced body fracture might rotate
Group 4: a. Medially rotated but at malar buttress
b. Inward at frontozygomatic suture
Group 5: a. Lateral rotation: up at infraorbital margin
b. Out at frontozygomatic suture
Group 6: Additional fracture line (Comminuted)

II. Rowe and Williams

1. *Fracture stable after elevation*
   a. Arch only (medially displaced)
   b. Rotation around vertical axis.
      – Medially
      – Laterally.
2. *Fracture unstable after elevation*
   a. Arch only (inferior displacement)
   b. Rotation around horizontal axis
      – Medially
      – Laterally.
   c. Dislocation en-bloc
      – Inferiorly
      – Medially
      – Posterior laterally.
   d. Comminuted fractures.

Fracture of Middle-third of Face

I. Rowe and Williams (1985)

A. Fracture not involving occlusion

1. *Central region*
   a. Fracture of nasal bone and/or nasal septum
      i. Lateral nasal injury
      ii. Anterior nasal injury.
   b. Fracture of frontal process of maxilla
   c. Fracture of types (a) and (b) which extend into the ethmoid bone (nasoethmoidal injury)
   d. Fracture of types (a), (b) and (c) which extends into the frontal bone (fronto-orbital-nasal dislocation)
2. *Lateral region:*
   Fracture involving zygomatic bone, arch and maxilla (zygomatic complex) excluding dentoalveolar component.

B. Fracture involving occlusion

1. Dentoalveolar fractures
2. Subzygomatic fracture
   - Le Fort I (low-level or Guerin)
   - Le Fort II (pyramidal).

3 Suprazygomatic
   - Le Fort III (high level or craniofacial dysjunction).

Nasoethmoidal Orbital (Noe) Fracture

1. *Isolated nasoethmoid injury without other fractures of the midface*
   a. Bilateral
   b. Unilateral.
2. *Combined nasoethmoid injury with other fractures of the midface*
   a. Bilateral
   b. Unilateral.

Frontal Sinus Fracture

I. First Classification

A. Anterior wall fracture
   1. Frontonasal drainage intact.
   2. Frontonasal drainage compromised.

B. Combined anterior and posterior wall fracture
C. Posterior wall fracture (B), (C) almost always involve frontonasal duct.

II. Second Classification

A. Anterior table fracture
   1. Linear (Undisplaced)
   2. Displaced.

B. Posterior table fracture
C. Outflow duct injury

TOOTH IMPACTION

A tooth is entitled as impacted only if the root formation is complete and yet it has not erupted fully up to the final position.

Pell and Gregary Classification

1. *Relation of tooth to ramus of the mandible and 2nd molar*

Class I	Sufficient space to accommodate the mesiodistal diameter of crown of the 3rd molar.
Class II	The space between distal surface of 2nd molar and ramus is less than mesiodistal diameter of the crown of the 3rd molar.
Class III	The 3rd molar is located within the ramus

2. *Based on relative depth of 3rd molar*

Position A	The highest portion of tooth at the level of occlusal plane or above it.
Position B	The highest portion of tooth is below the occlusal plane but above the cervical line of 2nd molar.
Position C	The highest portion of the tooth is below the cervical line of the 2nd molar.

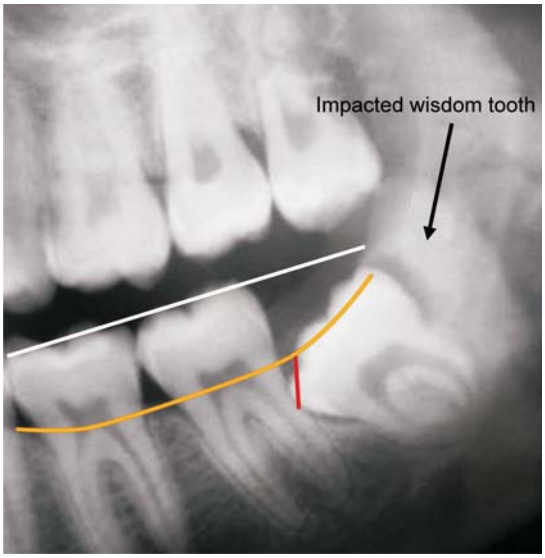


Fig. 30.1: Winter's lines

3. *Based on position of long axis of 3rd molar in relation to the long axis of 2nd molar (Winter's classification)*
   Vertical, horizontal, inverted, mesioangular, distoangular, linguoversion, unusual positions.

The exact position and depth of the tooth can be ascertained by three imaginary lines called as *Winter's Lines* (Fig. 30.1).

1. *White line:* Line drawn touching the occlusal surfaces of 1st and 2nd molars is extended posteriorly over the 3rd molar region. It indicates the difference in occlusal level of 2nd and 3rd molars.
2. *Amber line:* Runs at the level of the crest of interdental septum between the molars and represents the bone level covering the impacted tooth.
3. *Red line:* It is drawn perpendicular from the amber line to an imaginary point of application of elevator. It indicates the depth of the tooth in the bone and the difficulty encountered in removing the tooth.

Condition	*Position of the patient*
During recovery form syncope positions	Semireclined and Trendelenburg
During CPR	Supine position
A patient with suspected cervical fracture	Body and neck extended
Preferable patient position in recovery room after ambulatory general anesthesia	Lateral
Pregnant patient in syncope	Left lateral position
Patients affected with orthostatic hypertension	Upright position or with her trunk slightly to one side
Congestive heart failure patient	Upright position, (Supine position should be avoided)

CHAPTER

31 Morphological Differences between Primary and Permanent Teeth

Primary Teeth	*Permanent Teeth*
General	
Develops directly from dental lamina.	Develops as a lingual or distal extension of dental lamina.
Usually they are not covered with bony crypts.	They are completely covered with bony crypts.
All the primary teeth erupt into oral cavity at the age of $2^1/_2$–3 years. For root completion, it takes 1-$1^1/_2$ years after eruption. i.e. Root formation of primary teeth is completed by 3-4 years of age.	All the permanent teeth except 3rd molar erupt into oral cavity at the age of 12–13 years. For root completion, it takes 2-3 years after eruption. i.e. Root formation of primary teeth is completed by 14-16 years of age.
All primary teeth develop and erupt almost in the horizontal plane and occupy a more vertical position in the jaws.	Permanent teeth develop in different planes.
Duration of deciduous dentition ranges from 12-14 years (Fig. 31.1).	Average duration of permanent teeth is about 60 years of life time (Fig. 31.2).
Deciduous teeth are smaller in all **dimensions** (about 1/2) than that of corresponding permanent teeth except deciduous 2nd molar (Figs 31.3 to 31.6).	Larger in all dimensions than that of corresponding primary teeth.
Relation between upper and lower teeth is tooth-to-tooth relation. (Edge-to-edge contact)	Relation between upper and lower teeth is intercuspal relation.
Number of teeth: 20 Premolars are absent. 5 in each quadrant. 2 incisors, 1 canine, 2 molars.	Number of teeth: 32 Premolars are present. 8 in each quadrant. 2 incisors, 1 canine, 2 premolars, 3 molars.
Key to memorize: Development (4 points), Duration, Dimensions,	Relation, Number.

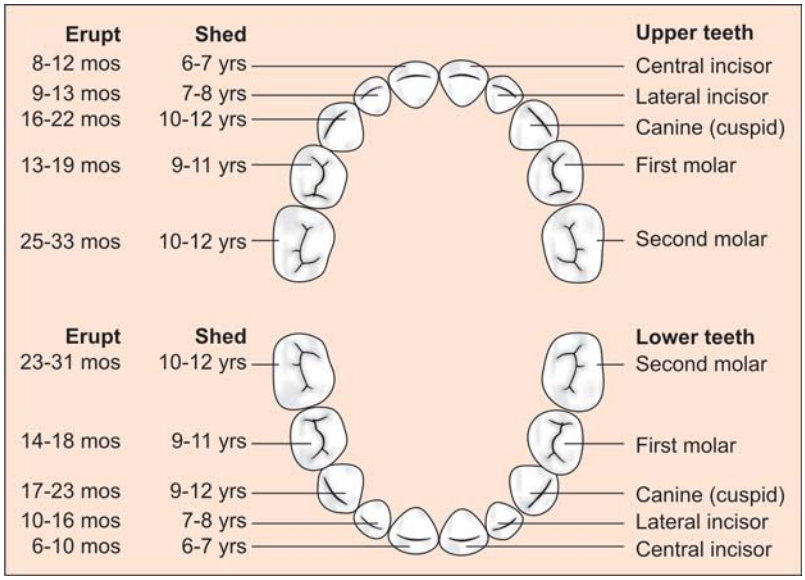


Fig. 31.1: Deciduous dentition

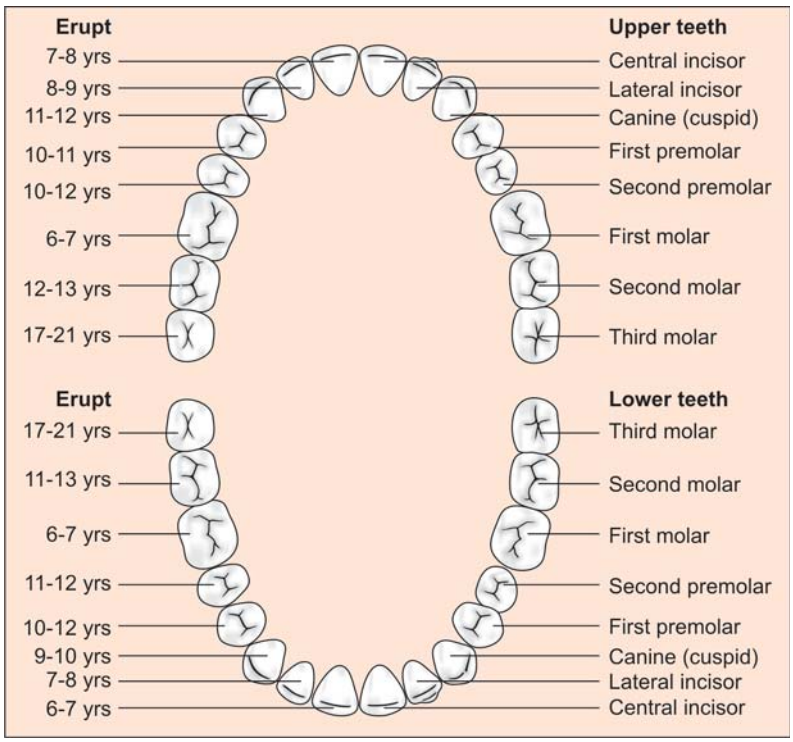


Fig. 31.2: Permanent dentition

The crown	
Lighter in **color**—bluish white also called as milk teeth as its refractive index is same as milk (1)	Darker in color—grayish or yellowish white.
The **contact areas** between molars are broader, flatter and situated gingivally.	The contact areas between molars are situateds occlusally.
Cervical ridges are more pronounced especially on the buccal aspect of the first molar.	Cervical ridges are more flat.
Cuspids are slender and tend to be more conical.	Cuspids are less conical.
Crowns are wider in mesiodistal dimensions in relation to the cervicoocclusal height. This gives a cup shaped appearance to anterior teeth and squat shape to molars.	Crowns of anterior teeth are larger in cervico-occlusal dimensions than the mesiodistal. This gives a longer appearance to anterior teeth.
The **enamel** is thinner and has a more consistent depth of about 1 mm thickness throughout the entire crown.	The enamel is thicker and has a thickness of about 2-3 mm.
The **enamel road** at the cervical slopes occlusally from the DEJ.	The rods are oriented gingivally.
Supplemental grooves are more.	Supplemental grooves are less.
Mammelons are absent.	Mammelons are present at the incisal edges of newly erupted teeth.
Occlusal plane is relatively flat.	Occlusal plane has curved contour.
Buccal and lingual surfaces of molars converge towards occlusal surface so that they have narrow **occlusal table** in a buccolingual plane.	There is less convergence of buccal and lingual surfaces of molars towards occlusal surface.
1st molars are more bulbous and are sharply constricted (bell shaped) cervically. They are smaller in dimension than 2nd molar.	They have less constriction of neck. They are larger in dimension than 2nd molar.

Key to memorize:
C – Color, Contact area, Cervical ridge, Cuspids
D – Dimensions
E – Enamel, Enamel rods, Supplemental grooves, Mammelons.
Occlusal plane, Occlusal table, 1st molar.

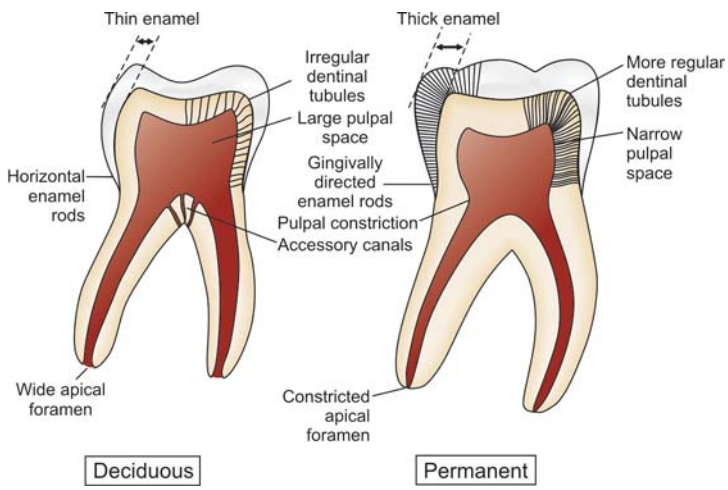


Fig. 31.3

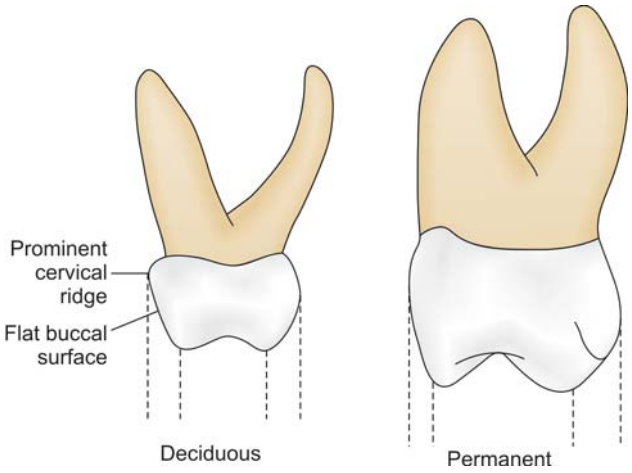


Fig. 31.4

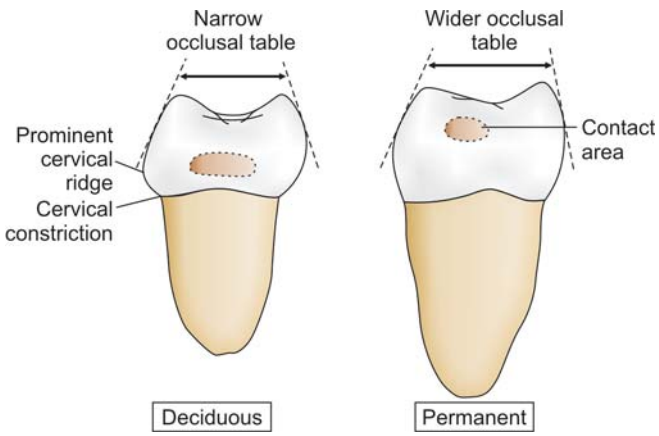


Fig. 31.5

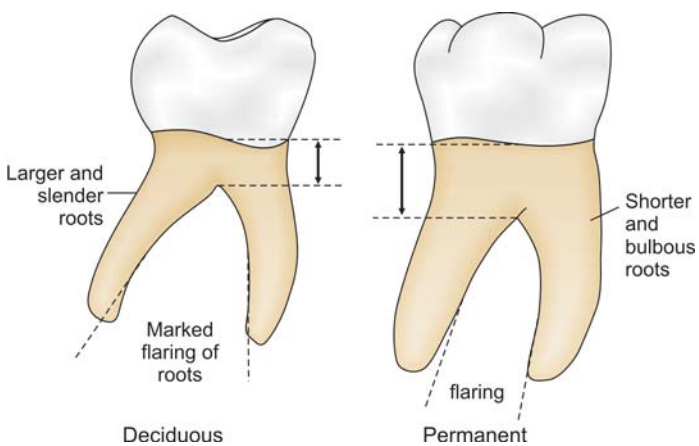
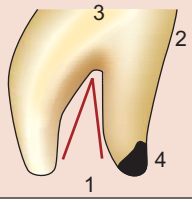
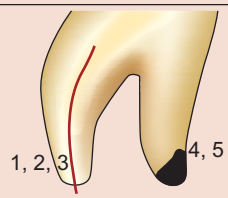


Fig. 31.6

Figs 31.3 to 31.6: Differences between deciduous and permanent teeth

Root	
1. The roots are larger and more slender in comparison to crown size.	1. The roots are shorter and bulbous in comparison to crown size.
2. Roots of the primary molars are more divergent and flare outwards as they have to accommodate the permanent tooth bud.	2. Roots are less divergent and do not flare to a great degree.
3. Furcation is more towards cervical area so that root trunk is smaller.	3. Placement of furcation is apical thus the root trunk is larger.
4. Undergo physiologic resorption during shedding of primary teeth so position of apical foramen varies with age.	4. Physiologic resorption is absent so position of apical foramen and length of root canal is fixed.
Key to memorize: 	

Pulp	
Pulp volume is large compared to tooth volume.	Pulp volume is less compared to tooth volume.
High degree of cellularity and vascularity of tissue at least in stages prior to advanced physiologic resorption of roots.	Comparatively less degree of cellularity and vascularity in tissue.
High potential for repair.	Comparatively less potential for repair.
Pulp outline follows DEJ more closely and pulp horns are closer to outer surface.	Pulp outline doesn't follow DEJ exactly and pulp horns are away from the outer surface.
No marked cervical constriction between coronal pulp and root canal.	Marked cervical constriction between coronal pulp and root canal.
Comparatively less tooth structure around pulp.	More tooth structure surrounding pulp offers more protection and increases potential for repair.
Greater thickness of dentin over the pulpal wall at the occlusal fossa of molars.	Lesser thickness of dentine over the pulpal wall at the occlusal fossa of molars.
Ratio of thickness of dentin in primary in permanent teeth is 1:2.	
Numbers of dentinal tubules are less.	Number of dentinal tubules are more about 50,000–90,000 per mm^2.
Root canals are more ribbon-like. Radicular pulp is thin and tortuous.	Root canals are well-defined and less branching.
Floor of pulp is porous and accessory canals from the floor of pulp chamber leads directly to inter-radicular furcation.	Floor of pulp chamber usually doesn't have any accessory canals.
Nerves terminate at pulpodental junction and their branches enter the dentinal tubules but end away from the dentinoenamel junction.	Nerves terminals transverse the whole length of dentinal tubules and end in the dentinoenamel junction.

Histological differences	
Roots have wider apical foramina, and resultant abundant blood supply demonstrates a more typical inflammatory response.	Foramina are restricted, and reduced blood supply favors calcific response.
Incidence of reparative dentin formation beneath carious lesion is more extensive and more irregular.	Reparative dentin formation is less.
Localization of infection and inflammation is poorer in pulp.	Infection and inflammation is usually local.
Pulp nerve fibers pass to the odontoblastic area, where they terminate as free nerve endings.	Pulp nerve fibers terminate mainly among odontoblasts and even beyond the predentin.
Density of innervations is less so primary teeth are less sensitive to operative procedure. Neural tissue degenerated first during root resorption.	Density of innervations is more, making tooth more sensitive to operative procedures.
Dentinoenamel junction is relatively flat.	Dentinoenamel junction is scalloped.
Key to memorize: 3 for foramina 2 for innervations 1 for DEJ  	

Mineral content	
Enamel and dentin are less mineralized, so acid etching time is more for primary teeth.	More mineralized so acid etching time is less.
Organic content is more.	Organic content is less.
Neonatal lines are present.	Neonatal lines are present only in 1st permanent molar.
Enamel: Stria of Retzius are less common which may be responsible for bluish white color of enamel.	Stria of Retzius are more common.
Dentin: *Tubules* are less regular. *Thickness* is half than that of permanent teeth. Dentin forming *cells* are active functionally for 360 days. *Interglobular dentin* is absent. Dentin is less dense. This can be observed clinically by resistance offered by it to bur while cutting. Dentin is cut more easily and abrades rapidly.	Dentin: *Tubules* are more regular. *Thickness* is double than that of deciduous teeth. Dentin forming *cells* are functionally active by 700 days. *Interglobular dentin* is present. Dentin is more dense and difficult to cut.

Periodontal ligament	
Area of periodontal ligament is less.	Area of periodontal ligament is more.
Lamina dura is relatively thick.	Lamina dura is relatively thin.

Other	
Cementum is very thin and of the primary type.	Secondary cementum is present.
Alveolar atrophy is rare.	Alveolar atrophy occurs.

CHAPTER

32 Definitions in Pedodontia

PEDODONTICS

By American Academy of Pediatric Dentistry

Pediatric dentistry is an age-defined specialty that provides both primary and comprehensive preventive and therapeutic oral healthcare for infants and children through adolescence, including those with special healthcare needs.

PSYCHOLOGY

Psychology is the science dealing with human nature, function and phenomenon of his own soul in the main.

CHILD PSYCHOLOGY

Child psychology is the science that deals with the mental power or an interaction between the conscious and subconscious element in a child.

CHILD MANAGEMENT

Child management is defined as the means by which a course of treatment for a young patient can be completed in the shortest possible period, while at the same time ensuring that he will return for the next course willingly (PJ Holloway, JN Swallow)

EMOTION

It is an effective state of consciousness in which joy, sorrow, fear, hate or the likes are expressed. Emotion is a state of mental excitement characterized by physiological, behavioral changes and alteration of feelings.

BEHAVIOR

Behavior is any change observed in the functioning of the organism.

FEAR

Fear is a reaction to a known danger (augmenting the flight or fright response). It may be defined as unpleasant emotion or effect consisting of psychological changes in response to realistic threat or danger to one's own experience.

ANXIETY

Anxiety is an emotion similar to fear but arising without any objective sources of danger.

PHOBIA

Phobia is an irrational fear resulting in the conscious avoidance of a specific feared object, activity or situation.

BEHAVIORAL SCIENCE

Behavior science is the science which deals with the observation of behavioral habits of man and lower animals in various physical and social environments including behavior pedodontics, psychology, sociology and social anthropology.

BEHAVIOR MANAGEMENT

Behavior management is the means by which dental health team effectively and efficiently performs treatment for a child and at the same time instills positive dental attitude. (Wright 1975)

BEHAVIOR SHAPING

Behavior shaping is the procedure which slowly develops behavior by reinforcing a successive approximation of the desired behavior until the desired behavior comes into being.

BEHAVIOR MODIFICATION

Behavior modification is defined as the attempt to alter human behavior emotion in a beneficial way and in accordance with the laws of learning (Mathewson).

NURSING CARIES

It is a unique pattern of dental decay in young children due to prolong nursing habit.

RAMPANT CARIES

It is suddenly appearing, widespread, rapidly spreading, burrowing type of caries resulting in early involvement of pulp and affecting those teeth, which are usually regarded as immune to decay (Massler, 1945).

PLUNGER CUSP

Cusp that tends to forcefully wedge food interproximally are called plunger cusp. It occurs due to wear, as a result of shift in tooth position after failure to replace missing tooth.

PIT AND FISSURE SEALANTS

It is "A material that is introduced into the pits and fissures of caries susceptible teeth, thus forming a micromechanically bonded, protective layer cutting access of caries producing bacteria from their source of nutrients" (Simonsen).

INDIRECT PULP CAPPING

It is defined as procedure where in small amount of carious dentin is retained in deep areas of cavity to avoid exposure of pulp. Followed by placement of a suitable medicaments and restorative material that seals off the carious dentin and encourages pulp recovery.

DIRECT PULP CAPPING

It is defined as the placement of a medicament or nonmedicated material on a pulp that has been exposed in course of excavating the last portions of deep dentinal caries or as a result of trauma (Kopel, 1992).

PULPOTOMY

It can be defined as complete removal of the coronal portion of the dental pulp, followed by placement of a suitable dressing or medicament that will promote healing and preserve vitality of the tooth (Finn, 1995).

PULPECTOMY

It is defined as complete removal of the necrotic pulp from the root canals of primary teeth and filling them with an inert resorbable material so as to maintain the teeth in the dental arch (Mathewson, 1995).
Pulpectomy is removal of all pulpal tissues from the coronal and radicular portions of the tooth (Finn).

APEXOGENESIS

It is defined as the treatment of a vital pulp by capping or pulpotomy in order to permit continued growth of the root and closure of the open apex.

APEXIFICATION

It is the method of inducing the development of root apex in an immature pulp less tooth by formation of osteocementum or other bone like tissue (Cohen).

PREVENTIVE ORTHODONTICS

Preventive orthodontics is defined as the actions taken to preserve the integrity of what appears to be normal occlusion at a specific time (Grabber, 1966).

INTERCEPTIVE ORTHODONTICS

It is that phase of science and art of orthodontics employed to recognize and eliminate the potential irregularities and malpositions in the developing dentofacial complex (American association of Orthodontists, 1969).

SPACE CONTROL

It is defined as careful supervision of the developing dentition; it reflects an understanding of the dynamic nature of occlusal development (Gainsforth, 1955).

SPACE MAINTENANCE

It is defined as the process of maintaining a space in a given arch previously occupied by a tooth or a group of teeth.

SPACE MAINTAINER

It is a fixed or removable appliance designed to preserve the space created by the premature loss of a primary tooth or a group of teeth (Boucher).

HABIT

It can be defined as a fixed or constant practice established by frequent repetition. It is defined as a tendency towards an act or an act that has become a repeated performance, relatively fixed, consistent, easy to perform and almost automatic (Boucher OC).

THUMB SUCKING

Thumb sucking is defined as placement of the thumb in varying depths into mouth.

TONGUE THRUSTING

Tongue thrusting is a forward placement of the tongue between the anterior teeth and against the lower lip during swallowing.

MOUTH BREATHING

Mouth breathing is defined as habitual respiration through the mouth instead of nose (Sassouni, 1971).

BRUXISM

Bruxism is defined as the habitual grinding of teeth when an individual is not chewing or swallowing. (Ramfjord, 1966).

SELF-INJURIOUS HABIT

Self-injurious habit is repetitive act that results in physical damage to the individual. These habits are seen with increased incidence in the mentally retarded population.

HANDICAPPED PERSON

WHO has defined a handicapped person as " One who over an appreciable period is prevented by physical or mental conditions from full participation in the normal activities of their age group including those of a social, recreational, educational and vocational nature."

MENTAL RETARDATION

It is a general term is applied to persons whose intellectual development is significantly lower than that of normal persons and whose ability to adapt to their environment is consequently limited.

It is defined as subaverage general intellectual functioning which originates during the developmental period and is associated with impairment in adaptive behavior (American Association of Mental Deficiency).

CEREBRAL PALSY

It is defined as a nonprogressive lesion which occurs in the developing brain before, during, after birth, leaving the child with a variety of neurological problems.

Instructions to the Reader

Some important things to be kept in mind before appearing for the final year examination are mentioned below. Most of the students tend to forget the same during hectic exam schedule.

- Final year exam for dental undergraduate students is very tiring. It exhausts all the energy by the last paper. Most of the time it lasts for almost a month. One should not stretch himself/herself more for the initial papers otherwise; he/she will be completely exhausted by last papers.
- Keep yourself relaxed and tension free. Take enough sleep and have healthy food. Do remember that you can do better only if you are physically and mentally fit. Peace of mind is absolutely mandatory while writing papers.
- Avoid junk food and irregularities in sleep and take daily meal regularly at least two months before the exam. Falling ill during this time can spoil your year's hard work.
- Do not forget to keep your exam identity card/no and other armamentarium like pen, pencil, scale, rubber, sharpener and colors ready with you; it will reduce wastage of time during precious exam hours.
- It is always advisable to keep all instruments for practical exams ready in sets well in advance. This will avoid missing of instruments during exam rush time.
- Keep your journals and work records ready in certified form.
- Be in touch with your patients whom you have planned for practical exams.
- Be through with all definitions as each viva starts with definition. Viva may not move further if first question is wrongly answered.
- Answer only that you know well. Answers to initial questions draw your viva. It depends on the candidate to direct viva in a desired direction. Do not speak anything you are not confident about or you do not know correctly. Speak only what is necessary.
- Do not try to impress examiner by throwing excessive information. Be precise and to the point.
- Decent attire, body language and facial expression are very important. Professional dress up reflects your sincerity. Aprons should be tidy and clean, do remember that everybody likes pleasant personality. First impression has major impact on your viva, so make your efforts accordingly.

With the hope that this information would be useful to all the doctors who are going to face the most challenging task of their life—the final exam.

I wish all the best to them.

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